Exploring the Role of Personality, Expectancies and Coping Strategies in Co-occurring Social Anxiety Disorder and Substance Use Disorder

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Author Note
Thesis submitted in partial fulfilment of the requirements for the degree of Professional Doctorate of Clinical Psychology
I hereby declare that this thesis does not contain any material that has been accepted for the award of any other degree or diploma, or any material previously published or written by another person, except where due reference is made in the text.
I further declare that the ethical principles and procedures specified in the Faculty of Health, Arts and Design Human Research Ethics Committee document have been adhered to in the process of conducting this research.

Name: Annette Raber

Signed:
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Dedication

This thesis is dedicated to my father Paul (Pinek) Raber from whom I get my curiosity about people and love of learning, and to my mother Giza Raber from whom I get my belief that anything is possible.
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Social anxiety disorder (SAD), and substance use disorders (SUD) are highly prevalent disorders that also frequently co-occur. The comorbid condition often presents with more severe symptoms, contributing to a further detrimental impact on the individual’s quality of life, with the consequences affecting the community more generally. Despite the high rate of the SAD-SUD comorbidity, and the considerable research generated in relation to it, inconsistencies in findings remain. This has led to the investigation of the role of various factors which are thought to influence this relationship. Most research has focused on the relationship between social anxiety and alcohol use in non-clinical samples, and methodological differences may at least partly account for some of the contradictory findings. In addition, more recently, evidence has emerged of the importance of cannabis use in its relationship with social anxiety.

This research sought to increase understanding about the SAD-SUD comorbidity by simultaneously exploring and comparing the roles of personality factors (sensitivity to reward and punishment), alcohol outcome expectancies (positive and negative), and coping strategies (adaptive and maladaptive) in both non-clinical (Study 1) and clinical (Study 2) samples. Previous research supports the individual importance of these factors on the relationship between social anxiety and substance use. In addition to the individual difference variables, other factors measured included demographic characteristics, social anxiety, fear of negative evaluation, depression, risky alcohol use and risky cannabis use. The same measures for all variables (except depression) were utilised in both studies.

Using a dimensional framework, Study 1 examined an analogue sample of 294 community and student participants between 18 and 63 years of age ($M = 24.67; SD = 10.22$). As indicated consistently in previous research, the advantage of using a non-clinical cohort is that a large sample size can be obtained relatively easily in order to conduct correlational and regression analyses and identify targeted factors that can be examined in clinical cohorts. Participants completed an online questionnaire. Results from Study 1 confirmed the importance of the six individual difference variables in the relationship between social anxiety and risky alcohol use. Only one of the variables, positive alcohol expectancies was associated with risky cannabis use, and extends prior
findings confirming the similarities between expectancies for alcohol and other substances. Also, the results from Study 1 demonstrated the importance of fear of negative evaluation, which was associated with all six of the individual difference variables, as well as playing a critical role with positive alcohol expectancies mediating the relationship between fear of negative evaluation and risky alcohol use.

The second study used both clinical and non-clinical cohorts in order to observe similarities and differences on the same personality, cognitive and behavioural factors as tested in Study 1. With limited past research using clinical samples, it was anticipated that this approach would inform intervention strategies, with respect to the SAD-SUD comorbidity. Study 2 recruited a total of 84 participants aged between 18 and 59 years of age ($M = 32.83; SD = 8.56$), all of whom were clinically diagnosed with SAD and some of whom were also clinically diagnosed with a SUD (alcohol abuse, alcohol dependence, substance abuse and/or substance dependence). The diagnoses of SAD and SUD were made via clinical interviews. All clinical participants completed a pen and paper version of the questionnaire. Participants were divided into three groups: participants with SAD but no diagnosed SUD (the SAD group; $n = 27$), participants diagnosed with both SAD and SUD who scored in the high risk category for alcohol use on the substance use measure (the SAD-SUD Alcohol group; $n = 18$), and all other participants with a diagnosis for both SAD and SUD (the SAD-SUD Other group; $n = 39$). A fourth group comprising healthy controls was added to Study 2 (the CONTROL group; $n = 30$). The CONTROL group comprised Study 1 participants who scored below the cut-off for social anxiety on the social anxiety measure, and who also scored below the relevant threshold scores for risky alcohol use, risky cannabis use and risky use of all other substances (apart from tobacco) on the alcohol and substance use measure.

Results from Study 2 demonstrated the important contribution in particular of personality (drive sensitivity), and alcohol expectancies to the SAD-SUD comorbidity in clinical populations. High levels of both reward sensitivity and punishment sensitivity are associated with the comorbid condition, and likewise, high levels of both positive and negative alcohol expectancies are held by those with co-occurring SAD and SUD, especially those who are heavy and problematic users of alcohol. The significance of cannabis in its role with social anxiety was also shown. Findings from both studies
confirmed the importance of personality, alcohol expectancies and coping strategies on the spectrum of severity of symptoms of co-occurring social anxiety and substance use disorder. It is recommended that the tension created by having high levels of both types of drive sensitivity and alcohol expectancies be factored into an integrated treatment protocol for people with the comorbid condition. An “enhanced CBT treatment for the SAD-SUD comorbidity” is proposed and future research directions discussed.
Exploring the Role of Personality, Expectancies and Coping Strategies in Co-occurring Social Anxiety Disorder and Substance Use Disorder

Chapter 1: Social Anxiety Disorder

Introduction

Social Anxiety Disorder (SAD) is a highly prevalent mental disorder (Kessler, Chiu, Demler, & Walters, 2005b). It can negatively impact the affected individual in many spheres of life including level of education attained, work (productivity, opportunities for promotions and income level), social relationships including marital status, and health status. The detrimental effect of social anxiety can be further exacerbated when it is comorbid with other psychological conditions including anxiety, mood, and substance use disorders (Katzelnick & Greist, 2001). The purpose of this chapter is to provide an up-to-date description of research on SAD including its nature, diagnostic criteria, prevalence, age of onset and course, gender distribution, impairment and quality of life, aetiology, treatment and treatment limitations, and comorbidities.

The Nature of Social Anxiety Disorder

The main characteristic of social anxiety disorder (SAD), previously known as social phobia is an intense fear or anxiety about being scrutinized by others in social situations (American Psychiatric Association, 2013) (Rapee & Sanderson, 1998). More specifically the fear or anxiety is severe and enduring and usually relates to being negatively evaluated by others in social or performance situations (Morris, Stewart, & Ham, 2005). This fear is essentially comprised of cognitions (e.g., “I’m making a fool of myself”), behaviour (e.g., poor eye contact) and somatic symptoms (e.g., sweating, blushing). Individuals with social anxiety either tolerate such social situations with significant distress or else they seek to avoid them. The strategy of avoidance usually adds to the level of interference and suffering as it prevents or limits occasions in which the person can become experienced and confident in social situations (Bandelow & Stein, 2004). Examples of feared and avoided social situations include meeting new people, entering a room full of people, attending or speaking up at meetings, dating, speaking to authority figures, public speaking, performing (generally or specifically) in front of others,
being assertive, and facing test situations (Rapee & Sanderson, 1998). Given the many ways in which social contact with other people occurs, social anxiety can be extremely pervasive across multiple areas of the affected person’s life.

Social anxiety has been conceptualized as both a unitary construct with quantifiable differences (on a continuum from shyness or social anxiety with little or no impact on daily living to extreme impairment and avoidance of most social activities), and as consisting of two or more qualitatively different constructs (the most commonly characterised as interaction anxiety and performance anxiety) (Hook & Valentiner, 2002). Interaction anxiety involves fear or anxiety related to conversing with others in a variety of social situations, for example, dating, at social gatherings, at school or work; whilst performance anxiety involves fear of being scrutinized during engagement in specific tasks, for example, public speaking, eating, writing (Mattick & Clarke, 1998). This qualitative distinction has been adopted in some measures of social anxiety including the instrument used in this study, the Liebowitz Social Anxiety Scale (Liebowitz, 1987), although the totals of such measures can also be used to measure the existence of, and overall severity of social anxiety.

Some authors contend that there are differences in symptoms, course, aetiology, prognosis, and response to treatment between performance anxiety and interaction anxiety (Hook & Valentiner, 2002). There is some evidence for this distinction, especially for the category of performance fears/anxiety (Bögels et al., 2010; Hook, Valentiner, & Connelly, 2013). People who are anxious/fearful only when performing for an audience (especially public speaking), do tend to develop the fear at a later age, are not generally shy or behaviourally inhibited, have more intense psychophysiological reactions (and relate their fear to the reaction), are more likely to respond to specific medications (beta blockers), and their fear is not familial (Bögels et al., 2010).

To some extent, the qualitative distinction between interaction and performance anxieties has also been incorporated into the diagnostic criteria for SAD (via specifiers) in previous and current editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), with interaction anxiety underlying generalised SAD and performance anxiety underlying specific SAD (a history of diagnosis of SAD in the DSM follows in the next section of this paper). Rather than focusing on the performance/interaction distinction, the
majority of research tends to support a continuum theory with regard to the severity of SAD based on the number of fears rather than the content of the fears (Heimberg et al., 2014). Using the continuum model, at one end of the spectrum are people without any symptoms of social anxiety, followed by individuals with subclinical symptoms, people with one or very few specific social fears, people with many social fears which impact on several domains and people with the most severe social fears who may also have avoidant personality disorder (APD) (Hook et al., 2013). This thesis takes the continuum approach and uses a dimensional framework in relation to the nature of social anxiety partly due to the weight of evidence in favour of the dimensional model, as well as due to the focus of this study which is on the comorbidity with substance use disorders. This issue is better characterised in the diagnostic and dimensional literature. While this research utilises a dimensional framework, it is noted that there may be a need to consider qualitative differences when interpreting the results.

Diagnostic Criteria

Although the first edition of the Diagnostic and Statistical Manual of Mental Disorders (“DSM”) was published in 1952, social anxiety disorder (SAD) was not classified as a distinct disorder until the third edition (DSM-III), in 1980 (American Psychiatric Association, 1980; Bögels et al., 2010; Carrigan & Randall, 2003). In DSM-III, social anxiety was classified as a phobic disorder and was called social phobia. There were three diagnostic criteria; first, a persistent, irrational fear and/or compelling desire to avoid a situation of being exposed to possible scrutiny by others and acting in a humiliating or embarrassing way; second, significant distress and recognition that the fear is excessive or unreasonable; and third, that the disturbance is not due to another mental disorder, such as avoidant personality disorder (APD). Generally a socially phobic individual was assumed to have only one social phobia and the examples given were subsequently considered specific or performance social phobias (American Psychiatric Association, 1980; Bögels et al., 2010). More generalised forms of social anxiety were covered in APD.

The specification of a generalised subtype of social phobia was first included in the next edition of DSM, the DSM-III-R. In addition, social phobia and APD could be diagnosed together (American Psychiatric Association, 1987; Bögels et al., 2010). In the following (fourth) edition of DSM, social phobia was called “Social Phobia (Social Anxiety
Disorder)” and its definition was widened requiring eight diagnostic criteria to be met (American Psychiatric Association, 1994). These included a significant ongoing fear of social or performance situations in which the social phobic was exposed to unfamiliar people or possible scrutiny by others, and where that individual feared they would act in an embarrassing or humiliating way (or display anxiety symptoms). Furthermore, the exposure needed to invoke anxiety in the individual who also recognised the fear as excessive or unreasonable. In addition, the feared situations had to be avoided or endured with dread, the social phobia needed to significantly interfere with the individual’s life, and the fear could not be due to the direct physiological effects of a substance or general medication. Finally, if the person had a medical condition, the social phobia could not be related to it, and if the individual was less than 18 years old, the social phobia must have continued for a period of at least 6 months. The social phobia could be specified as ‘generalised type’ if the fear included most social situations. If the fear involved fear of one or several but not most social situations, it could be considered specific or non-generalised (although there was no specifier for this distinction). It was noted that APD could be a more severe variant of “Social Phobia, Generalised” (Bögels et al., 2010). In addition, both childhood diagnoses from the previous editions were removed, and the diagnosis of SAD for children was restricted to children who were capable of establishing social relationships and displayed social anxiety with peers and not only with adults (Bögels et al., 2010).

At the time of collection of data for this thesis, the DSM-IV-TR (published in July 2000) was the current edition of the DSM. Changes were mostly confined to text revisions that accompanied each disorder (American Psychiatric Association, 2000). Social anxiety was still called Social Phobia (Social Anxiety Disorder) and contained the same eight diagnostic criteria. There were notations relating to children displaying some of the stated signs of social phobia. Generalised social anxiety disorder was still classified the same way. There was also a notation to consider the additional diagnosis of APD.

In 2013, the DSM-5 was published (American Psychiatric Association, 2013). It contains a number of changes to the way SAD is defined. These can be summarized as follows: “social anxiety disorder” has become the primary name with “social phobia” in brackets after it; there is an increased emphasis on the fear of negative evaluation (the
definition for negative evaluation now also includes behaviour/anxiety symptoms that will lead to rejection or will offend others, in addition to being humiliating or embarrassing), and on cultural considerations; a minimum duration of 6 months is required regardless of age; if the affected person has a medical condition the social fear/anxiety/avoidance cannot be related to it, or must be excessive; and the “generalised” specifier has been replaced with a “performance only” specifier to cover situations where the fear is restricted to speaking/performing in public (Heimberg et al., 2014). Reference is also made to the more generalised social anxiety disorder (but not “social anxiety disorder, performance only”) being frequently comorbid with APD (American Psychiatric Association, 2013). Over time SAD has become more broadly defined, and can therefore capture a wider range of affected individuals who may benefit from treatment.

Prevalence

Prevalence rates for social anxiety vary for a number of reasons including differences in diagnostic criteria applied, and differences in the threshold or definition of “diagnosis” (Furmark, 2002). However, overall, social anxiety or SAD is a highly prevalent mental disorder.

Social anxiety has been found to be the third most common mental disorder, after depression and alcohol use disorders (Morris et al., 2005). In the United States of America (U.S.A.), based on the collection of data between 2001 and 2003 for the National Comorbidity Survey Replication (NCS-R) and using DSM-IV criteria, SAD has an estimated lifetime prevalence of 12.1% and an estimated 12 month prevalence of 7.1% (Ruscio et al., 2008; Stein & Stein, 2008). Out of all mood and anxiety disorders, SAD has the third highest lifetime and 12 month prevalence rates, after depression and specific phobias (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Other Western nations have similar prevalence rates (Stein & Stein, 2008).

In Australia, based on the collection of data for the 2007 National Survey of Mental Health and Wellbeing (NSMHW), and using DSM-IV criteria, SAD has an estimated lifetime prevalence of 8.4%, and an estimated 12 month prevalence of 4.2% (McEvoy, Grove, & Slade, 2011). Out of all anxiety disorders surveyed (agoraphobia, SAD, panic disorder, generalised anxiety disorder, obsessive-compulsive disorder and post-traumatic stress disorder), SAD has the highest lifetime prevalence and the second highest 12 month
prevalence (after post-traumatic stress disorder), and the third highest lifetime and prevalence rates after affective disorders (depression, dysthymia, bipolar disorder type I or II), and substance use disorders (alcohol and drug abuse and dependence) (McEvoy et al., 2011).

**Age of Onset and Course**

It is difficult to deduce a typical age of onset for SAD although children as young as 8 years of age often meet SAD criteria (Rapee & Sanderson, 1998). A significant amount of data collected for treatment-based studies is from children age 8 and higher, whilst fewer studies include data for children down to age 6. Generally it is agreed that school age children can be reliably and validly diagnosed with SAD by the age of 9 (Bögels et al., 2010). Statistics from the U.S.A. NCS-R show that phobias, including SAD have earlier median ages of onset compared to other anxiety and mood disorders (Kessler et al., 2012). In Australia, results from the 2007 NSMHW demonstrate that SAD is the earliest onset anxiety disorder with a median age of onset of 13 (McEvoy et al., 2011).

Retrospective studies with clinical populations have found that there are two common periods of onset of SAD; childhood (before age 10), and during adolescence (ages 14 – 17). Childhood onset of SAD has been shown to be associated with higher levels of current psychopathology, functional impairment and emotional disorder vulnerabilities compared to later age of onset (Rosellini, Rutter, Bourgeois, Emmert-Aronson, & Brown, 2013). Onset of SAD during adolescence coincides with increased pressure to socialize at this period of life.

Most information about the course of SAD comes from retrospective self-reports from community participants. These studies indicate a chronic course with long duration and low recovery rates. In addition, risk factors for chronicity from self-reports include younger age of onset and comorbidity with medical and/or other psychiatric conditions (Beard, Moitra, Weisberg, & Keller, 2010). Less information is available from prospective studies. One study which collected data from participants that had entered treatment facilities, found that the probability of recovery from SAD after 12 years was around one person in three (Bruce et al., 2005). These researchers also found that all anxiety disorders had a high probability of recurrence, and the course of the illness was worsened by the presence of comorbid disorders such as depression and substance use (including alcohol).
disorders. Another study that collected data from participants attending family medicine clinics for primary care, found the probability of recovery from SAD after 5 years was between one person in three and one person in two, with lower rates of recovery predicted for those with lower psychosocial functioning, a longer duration of SAD episode, and comorbidity with panic disorder with agoraphobia (Beard et al., 2010).

Contrary to the above findings, a few prospective studies have indicated a less chronic course, evidenced by socially anxious individuals no longer meeting diagnostic criteria for SAD at follow-up. However, the majority of these individuals continued to endorse symptoms of SAD at four year follow-up (Beard et al., 2010). A recent prospective study of adolescents and young adults (ages 14 to 24), which collected data over a 10-year period, showed that the course of SAD was enduring with symptoms fluctuating over this period (Beesdo-Baum et al., 2012).

In summary, the course of SAD appears to be chronic and long, with a low probability of recovery and a high likelihood of recurrence particularly in those with comorbid disorders including substance use disorders. It is hoped that the results of this study, which examines the co-occurrence of social anxiety and substance use, will increase our understanding of this comorbidity, so that more targeted interventions can be developed.

Gender Distribution

Epidemiological and survey studies indicate a higher proportion of women meet criteria for SAD. For example, in the U.S.A., data from the NCS-R showed significant gender differences, with a higher prevalence rate in women over men in the 13-17 age group and the 18-64 age group (Kessler et al., 2012). Another large U.S.A. community sample who participated in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) which also used DSM-IV criteria for diagnoses of mental disorders, identified the lifetime prevalence of SAD was 4.20% for men and 5.67% for women (Xu et al., 2012). In Australia, results from the 2007 NSMHW demonstrated that SAD is more prevalent amongst women at all age groups between 16 and 85 (McEvoy et al., 2011).

On the other hand, treatment studies have indicated equal numbers and sometimes even more men with SAD. This may be related to gender roles which generally require men to initiate dating and problems in this area may lead them to seek treatment (Rapee &
Sandeerson, 1998). In one clinical study, no gender differences were evident on history or subtype of SAD, or on comorbidity with other anxiety disorders, mood disorders or APD. However, women had more severe social fears compared to men (Turk et al., 1998). A more recent study involving clinical participants indicated higher initial symptom severity in men compared to women (Bezerra De Menezes, Fontenelle, & Versiani, 2008). There seems to be a higher proportion of women compared to men with social anxiety in non-clinical populations. The results with clinical populations are not so clear.

**Impairment and Quality of Life**

DSM criteria for SAD include significant functional impairment in daily, social, occupational/academic or other important life domains (American Psychiatric Association, 2000; 2013). Such impairment can affect quality of life which can be broadly defined as a person’s subjective assessment of their sense of well-being, perceived via their social relationships, health status, life satisfaction, economic status and external life situations (Wong, Sarver, & Beidel, 2012).

A number of studies have demonstrated that people diagnosed with SAD have impaired quality of life with around half of socially anxious persons reporting at least one severe functional limitation in their life in terms of role impairment, professional help seeking or use of medication (Wong et al., 2012). SAD has been shown to negatively affect education, career and romantic relationships (Wittchen, Fuetsch, Sonntag, Müller, & Liebowitz, 2000). People diagnosed with generalised SAD have been found to be more affected in terms of negative impact on perceived quality of life compared to people diagnosed with non-generalised SAD (Wong et al., 2012). Although people with anxiety disorders including panic disorder, generalised anxiety disorder, obsessive compulsive disorder and SAD generally report overall impairment with quality of life, SAD has been shown to significantly affect social and leisure domains in quality of life measures compared to other anxiety disorders (Barrera & Norton, 2009; Lochner et al., 2003). In summary, both community and clinical studies demonstrate that the quality of life experienced by people with social anxiety is reduced. People with SAD and comorbid conditions are likely to have their quality of life further reduced. Comorbidities with SAD are discussed in the last section of this chapter as this study focuses on one comorbidity in particular, that of SAD and substance use disorders. A discussion of aetiological models
follows next and sets up further consideration of factors that might make socially anxious individuals vulnerable to substance use comorbidities.

**Aetiology**

A number of biological (e.g., genetic and temperamental), psychological (e.g., cognitive and behavioural) and social (environmental) factors have been associated with the development (and maintenance) of SAD, although it is not fully understood how these biopsychosocial factors interact such that no comprehensive model of understanding exists. This section includes a general summary of these factors, emphasizing the ones that are most relevant to this study.

**Genetic factors.** Several studies support the view that genetic factors play a moderate but important role in the development and maintenance of SAD in children and adults, via family studies and twin studies (Rapee & Spence, 2004). Family studies show a higher rate of social anxiety amongst family members of individuals affected by SAD compared to non-clinical controls (Fyer, Mannuzza, Chapman, & Martin, 1995; Hughes, Furr, Sood, Barmish, & Kendall, 2009). Meta-analyses of twin studies identify heritability estimates for SAD as high as 0.65 (Beatty, Heisel, Hall, Levine, & La France, 2002). However other studies report lower heritability indices for SAD of, for example, 30% in female twins (Kendler, Neale, Kessler, & Heath, 1992). Another study found a heritability estimate of 0.48 in twins for fear of negative evaluation, a more general trait and related construct of SAD (Stein, Jang, & Livesley, 2002). However, despite these results, most of the literature indicates that the genetic component to SAD is non-specific and is shared with other anxiety disorders as well as with mood disorders (Mosing et al., 2009).

**Neurobiological factors.** Genetic research has also investigated the possibility of neurobiological vulnerabilities such as genes that may be specifically involved in the development of social anxiety. However mixed findings have been reported. For instance one study involving second grade children found a significant relationship between shyness/social anxiety and the long form of the serotonin transporter promoter region polymorphism genotype (Arbelle et al., 2003). This finding was in contrast to other studies which did not find such an association (Schmidt, Fox, Rubin, Hu, & Hamer, 2002). More recently, reduced binding of a specific serotonin-1A receptor (5-HT) related to SAD has
been identified (Lanzenberger et al., 2007). Future research in this area may lead to more definitive findings to help explain the genetic component of SAD.

**Temperamental factors.** It has been suggested that early temperamental styles precede the development of SAD and thus reflect common genetic and/or environmental causal mechanisms. Early temperamental styles may also represent initial manifestations of the same construct which is later labelled a disorder when its manifestation becomes more severe (Rapee & Spence, 2004). Behavioural inhibition (BI) is a characteristic of temperament and can be defined as a relatively consistent pattern of responses, both emotional and behavioural, to new or unfamiliar situations and people. Inhibited children are described by their parents as quiet, withdrawn, shy, watchful, vigilant, cautious and sensitive (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). Evidence, including a recent meta-analysis of studies measuring SAD and BI, indicates an increased risk for developing SAD in behaviourally inhibited children (Gauss & Blackford, 2012; Kagan et al., 1984; Neal, Edelmann, & Glachan, 2002). Another recent study found significant differences on both symptom severity and degree of BI in early onset social anxiety (onset prior to age 18 with the higher levels of BI) compared to later onset SAD (over age 18) outpatients (Lim et al., 2013). However, there is also evidence that BI is a risk factor for other problems including anxiety problems in adolescence (Prior, Smart, Sanson, & Oberklaid, 2000), and depression, alcohol abuse, violence, and suicide in adulthood (Caspi, Moffitt, Newman, & Silva, 1998). Such data are consistent with genetic studies which more strongly support a common genetic link across several mental disorders, rather than a specific genetic marker for SAD.

One explanation as to why some children high on BI may not later develop SAD is the proposal that BI can be separated into two constructs: social and physical wariness (Rapee & Spence, 2004). The social dimension of BI in childhood has been linked to SAD in adulthood (Neal et al., 2002), whilst the physical dimension of BI has been linked to anxiety disorders in general (Van Ameringen, Mancini, & Oakman, 1998), although Neal et al. (2002) did not find the physical BI component to be linked to SAD, depression, agoraphobia or general anxiety/panic symptoms. However, both these studies found an association between the social dimension of childhood BI and later depression. These results are consistent with past findings that although all anxiety disorders and depression
share high negative affect (neuroticism), only SAD and depression both also show low positive affect (extraversion/sociability). The construct BI is relevant to this study, which measures related concepts (i.e., sensitivity to reward and punishment) and examines their role in the comorbidity between social anxiety and substance use. More detail about the inclusion of the reward sensitivity construct is contained in chapter 3 of this thesis, which details the comorbidity of social anxiety and substance use.

**Environmental factors.** Environmental influences on the aetiology of social anxiety include parent/child interaction, aversive social experiences and negative life events. Although studies have shown generally there is a relationship between insecure attachment of parent/caregiver and infant, and the development of anxiety, there have been mixed findings with respect to SAD specifically (Higa-McMillan & Ebesutani, 2011). One longitudinal study which followed children from age 15 months to ages 8-9 years, found that children exhibiting secure attachment styles had less SAD, were more popular at school, and were more socially active, compared to children with anxious-ambivalent or anxious – avoidant attachment styles (Bohlin, Hagekull, & Rydell, 2000). Another study examined attachment styles in socially anxious adults and found they fell into one of two main groups of attachment styles, those anxiously attached and others securely attached, with the latter group being less dysfunctional as a result of SAD. Also of note was that SAD mediated the relationship between attachment insecurity and depression (Eng, Heimberg, Hart, Schneier, & Liebowitz, 2001). Other studies have not found a relationship between insecure attachment and SAD (Roelofs, Meesters, Ter Huurne, Bamelis, & Muris, 2006). Overall, findings are not definitive with respect to the relationship between attachment style and SAD.

The issue of parent/child interaction is a complex one, one reason being that both parental and children’s anxious behaviours can have a reciprocal effect (Ollendick & Hirshfeld-Becker, 2002). However, retrospective studies from adults with SAD generally point to greater parental control and less parental warmth (Rapee & Spence, 2004). After a review of the parenting literature, Rapee and Spence (2004) concluded that, although parental overprotection is also associated with other anxiety disorders, its relationship with SAD might be stronger.
Another way parents can influence the development of SAD in their children is through modelling social avoidance themselves (Higa-McMillan & Ebesutani, 2011). Retrospective self-reports by children of socially anxious parents indicate the parents tend to isolate their children from social interactions, place a lot of importance on other people’s opinions, and use shame as a form of discipline (Bruch & Heimberg, 1994; Rapee & Melville, 1997). Overall, while further research is needed to understand more precisely how parental influence may contribute to the development of SAD, there does seem to be a relationship between parent/child interactions, including modelling, and SAD.

The main aversive social experiences that date from childhood and have been related to the development of social anxiety include teasing, bullying, humiliation, ridicule and rejection (Hackmann & Clark, 2000). A recent study of nearly 600 students over age 23, found that two subtypes of childhood bullying in particular, social exclusion (being ignored or left out on purpose) and relational victimisation (the damaging of the victim’s relationships with others by the bully), predicted social anxiety in adulthood (Boulton, 2013). However the direction of causality in the relationship between SAD and adverse social experiences remains unclear given the findings that early unpleasant experiences can contribute to the development of extremely negative images of the social self which are activated in subsequent negative social situations, and which ignore more favourable experiences (Hackmann & Clark, 2000).

Other more general forms of early traumatic life events such as divorce, parental conflict, parental psychopathology, sexual abuse, illness, and separation from parents, have also been proposed as a contributing factor to the development of SAD (Bandelow et al., 2004). However, these types of events have also been shown to increase the risk of development of other psychopathologies and are not unique to SAD. Further research in this area, for instance via longitudinal studies is needed to clarify the relationships of these types of events with the onset or development of SAD. Research is also needed to understand how the above-described environmental factors contribute to the development of SAD in some individuals but not in others.

**Cognitive factors.** Of major relevance to this study is the contribution of cognitive factors to the development of SAD. Research shows that anxious individuals interpret ambiguous stimuli or events as threatening or negative. Socially anxious individuals
evaluate social situations as threatening and believe they will be seen as socially incompetent, be criticised or disapproved of, and that catastrophic results will follow (Ollendick & Hirshfeld-Becker, 2002). These biases and distortions in thoughts, attitudes and beliefs about various or specific types of social situations trigger and maintain the social anxiety. The detailed cognitive model of Clark and Wells (1995) which still informs treatment today, delineated four processes that prevent affected individuals from disconfirming their negative beliefs: self-focused attention which occurs when a socially anxious person enters a feared situation and thinks they are in danger of being negatively evaluated; engaging in “safety behaviours” intended to reduce the risk of negative evaluation but having the opposite effect as they prevent the affected person from having their distorted beliefs from being disconfirmed; behaving in a less friendly, warm or outgoing manner as a result of being preoccupied with monitoring their performance; and anticipatory and post-event processing.

Rapee and Heimberg (1997) emphasised external cues a socially anxious individual attends to as well as internal ones. Cues of external threats could include frowns and signs of boredom from “audience” members. Rapee and Heimberg (1997) use the term “audience” to refer to one or more persons who may potentially observe the socially anxious individual’s behaviour (including speech) or appearance. The individual then determines whether they are meeting the presumed standard of the audience in the given situation. The difference between the individual’s perception of the audience’s appraisal of their appearance and/or behaviour and the individual’s perception of the audience’s standard for evaluating their appearance and/or behaviour, determines the perceived likelihood of negative evaluation from the audience. This perception leads to further anxiety which is demonstrated somatically, behaviourally and cognitively, thereby perpetuating the cycle (Rapee & Heimberg, 1997).

As can be seen from these two cognitive models, an important component of social anxiety is the cognitive vulnerability of fear of negative evaluation. It is included in DSM criteria for SAD. This fear was initially conceptualized much earlier (Watson & Friend, 1969) than the first DSM classification of social phobia in 1980. Its definition included fear, distress, and avoidance in relation to the expectation of negative evaluation by others, and a scale measuring it was developed by Watson and Friend (1969). The measure was
subsequently revised and abbreviated (Leary, 1983), and has been recently refined (Carleton, McCreary, Norton, & Asmundson, 2006). It correlates highly with social anxiety measures (Carleton, Collimore, & Asmundson, 2007).

**Social skills deficits.** The final relevant component that may play a role in the development of and maintenance of social anxiety is a lack of social skills. There have been mixed findings in relation to this contention. For example, some researchers have found that children with SAD demonstrate poorer social skills compared to normal or non-anxious controls on a behaviour assessment task (Beidel, Turner, & Morris, 1999); via reduced non-verbal communication skills, for example reduced general facial activity (Melfsen, Osterlow, & Florin, 2000); and via less competence in identifying emotions in facial expressions of others (Melfsen & Florin, 2002). However, other researchers have not found that children with SAD demonstrate poorer social skills compared to other children, but rather that such children believe they have social skills deficits (Cartwright-Hatton, Tschernitz, & Gomersall, 2005). Even if it is accepted that social skills deficits are related to SAD, more research is needed, for example to clarify what comes first (actual social skills deficits or the belief a socially anxious individual has about having them). It may well be that socially anxious children have social skills deficits which play a more significant role in the development and maintenance of SAD in children compared to adults (Rapee & Spence, 2004).

In summary, there are a number of possible risk factors for the development and maintenance of SAD including genetic, temperamental, environmental, cognitive, and social components. Further research is needed to clarify how these factors may interact to lead to the onset of SAD in childhood, adolescence or adulthood, and to eventually put forward a comprehensive aetiological model of understanding about social anxiety.

**Treatment and Treatment Limitations**

Since the initial classification of SAD as a distinct disorder in the DSM in 1980, research into the effectiveness of various treatments has significantly increased (Bandelow & Stein, 2004, p. 235). This section provides a summary of the current treatments used to manage and overcome social anxiety disorder, including cognitive-behavioural therapy (CBT), pharmacotherapy, and other psychotherapies. The summary ends with a short description of treatment limitations.
Cognitive-behavioural therapy (CBT). The most widely used evidence based treatment for social anxiety is CBT derived from the cognitive and cognitive-behavioural models proposed by Clark and Wells (1995) and Rapee and Heimberg (1997) respectively (both these models have been described in the previous section of this chapter). CBT aims to sever the interdependence of the socially anxious person’s negative and dysfunctional belief system, which is accompanied by avoidance and/or safety behaviours. The two main features of CBT are cognitive restructuring (to alter perceptions of threat and fear of negative evaluation), and exposure to feared social situations, via a ranked hierarchy from less anxiety provoking to more anxiety provoking (Hofmann, 2004). Other features such as relaxation and social skills training are sometimes also included in CBT treatment (Bogels et al., 2014). While CBT can be delivered in individual, group or online formats, treatment needs to be tailored to the specific thoughts, feelings and behaviours and the interactions of these components experienced by the affected individual, in social situations that make them fearful or anxious.

More recent research into the efficacy of CBT components has focused on judgmental bias in the cognitions of socially anxious persons. Judgmental bias has been categorised in two ways: firstly a probability bias (i.e., tending to link feared stimuli or responses with an unrealistic high threat of harm); and secondly, a social cost bias (i.e., tending to exaggerate the negative effects of a harmful event) (Otto, Hearon, & Safren, 2010). For instance, socially anxious individuals are likely to demonstrate high probability perceptions of negative evaluation and to consider the outcomes of negative evaluation to be catastrophic. One study found that changes in social cost bias accounted for a significant amount of variance in treatment outcome (Hofmann, 2004), whilst another one found this did not occur when probability bias was statistically controlled (McManus, Clark, & Hackmann, 2000). Other researchers have found differences in the timing of these bias types, with reductions in probability bias preceding a reduction of fear, and reduced social cost bias occurring after fear reduction (Smits, Rosenfield, McDonald, & Telch, 2006). Further research on these types of biases could increase the effectiveness of cognitive restructuring and the interrelationship between thoughts and feelings in feared social situations.
**Pharmacotherapy.** Overall, studies investigating treatment outcomes for SAD support the efficacy of both CBT (Heimberg, 2002), and pharmacotherapy (Otto et al., 2010). With pharmacological treatments, monoamine oxidase inhibitors (MAOIs), selective serotonin reuptake inhibitors (SSRIs) and benzodiazepines are considered to be the most effective treatments (Van Ameringen, Mancini, & Patterson, 2009). A recent review of pharmacology for SAD focused on published randomised control trials (RCTs) and meta-analytic reviews and considered the efficacy of different medications (Blanco, Bragdon, Schneier, & Liebowitz, 2013). Blanco et al. (2013) concluded that, for the majority of patients, SSRIs should constitute the first line of medical treatment of SAD as they have been shown to have a moderate effect size, are generally well tolerated and are also efficacious for common comorbid conditions such as depressive disorders and other anxiety disorders. For patients that do not respond or do not respond well to SSRIs, benzodiazepines are an alternative. However, studies for this medication group show a range of results (some differ from placebo whilst others do not). In addition, given the high comorbidity of SAD and alcohol abuse/dependence, care should be taken when prescribing benzodiazepines to SAD patients. Blanco et al. (2013) further concluded that a third alternative pharmacological treatment for SAD is the use of MAOIs although their use requires strict diet requirements (a low tyramine diet) to reduce the risk of hypertensive crisis. In summary, SSRIs appear to be efficacious in the treatment of SAD for most sufferers, especially those with comorbid depressive or anxiety disorders.

Other treatments for SAD that have been evaluated include interpersonal/psychodynamic therapies, and more recently, acceptance and commitment therapy (ACT).

**Interpersonal/psychodynamic therapies.** These treatment modes have the potential to allow clients to re-evaluate their negative cognitions and related anxiety symptoms in social situations by way of finding historical explanations for their social anxiety, increasing their awareness of their thoughts and feelings, and exploring/addressing the transference (i.e., how the client relates to the therapist) (Busch & Milrod, 2004; Otto et al., 2010). Psychodynamic therapy may additionally encompass other psychological constructs including defence mechanisms and unconscious conflicts or fantasies (Busch & Milrod, 2004).
In a RCT comparing cognitive therapy with interpersonal psychotherapy, both types of therapy were found to lead to considerable improvements that were maintained one year after treatment. However, cognitive therapy was found to be more efficacious in terms of the reduction of SAD symptoms (Stangier, Schramm, Heidenreich, Berger, & Clark, 2011). A more recent and larger RCT comparing CBT with psychodynamic therapy, established that although both treatments were effective in treating SAD, there were significant differences between the two modes of therapy in favour of CBT (Leichsenring et al., 2013). Another recent study compared CBT with psychodynamic therapy, and reported no significant differences between the two treatments for SAD (Bogels et al., 2014). However, the psychodynamic therapy consisted of a higher average number of sessions (31.4) compared to the CBT treatment (19.8). Overall, current research indicates that CBT is a more efficient treatment for SAD compared to interpersonal/psychodynamic therapies.

**Acceptance and Commitment Therapy (ACT).** ACT is part of the third wave of cognitive-behaviour therapy which focuses on acceptance of difficult or negative thoughts and feelings, rather than challenging them (Hayes, Strosahl, & Wilson, 1999). This form of therapy guides the client away from experiential avoidance by refocusing their behaviour to align with important goals and values, despite their feelings of anxiety (Otto et al., 2010). ACT uses various techniques to accept uncomfortable thoughts and feelings, including mindfulness (living moment to moment). Mindfulness has been successfully incorporated into treatments for other mental disorders including mindfulness-based cognitive therapy for depressive disorders and dialectical behaviour therapy (DBT) for borderline personality disorder (Dalrymple & Herbert, 2007). Mindfulness and ACT have also been used to treat SAD. A recent review of studies reported a significant reduction of SAD symptoms with these forms of treatment (Norton et al., 2015). However, benefits of such treatment were equal to, or less than, benefits obtained from CBT. Nevertheless, ACT is emerging as a promising alternative treatment for socially anxious individuals who do not respond well to CBT. More research needs to be conducted to compare psychological therapies for SAD and also to assess their effectiveness at later follow-ups after treatment has ended.

**Combining treatment modes.** Research has also been conducted on the combined use of pharmacotherapy and CBT, however, results have been mixed (Dalrymple & Herbert, 2007). In one randomized double blind study in general practice, patients with
SAD symptoms were either given sertraline (an SSRI) or placebo, and were also randomized into receiving exposure therapy or general medical care. Results indicated that the combined sertraline and exposure therapy treatment as well as sertraline treatment alone, were both significantly superior to placebo, although there was no significant difference between patients treated with exposure therapy compared to those who weren’t. As general practitioners were trained for this study to provide exposure therapy only (and not cognitive therapy), this research only looked at treatment combining medication with one component of CBT. More recent research into a therapy-enhancing medication, d-cycloserine (DCS), has indicated its use with exposure-based therapies enhances behavioural learning for SAD patients (when compared to SAD patients receiving the same therapy with a placebo) (Guastella et al., 2008).

Other research has shown no increased benefit in combining pharmacotherapy and CBT to treat SAD (Davidson et al., 2004). The main contention against the combination of medication with CBT as treatment for SAD is that whilst it may be beneficial at the start of treatment, the continued use of medication may become another safety behaviour, and may lead to a higher risk of relapse when treatment is concluded (Otto et al., 2010). It appears that more research is needed to investigate the benefits or detriments of combining pharmacotherapy and CBT to treat socially anxious individuals.

Treatment limitations. Although treatment modes such as CBT and pharmacotherapy have been shown to improve symptoms, management and course of social anxiety in affected individuals, outcomes can vary. Treatment outcomes for CBT can be affected by a number of factors including homework compliance, degree of severity/impairment of SAD, expectancy for improvement at commencement of treatment (amongst individuals receiving group CBT), anger (greater suppression of angry feelings is linked to greater post-treatment SAD and depressive symptoms), and comorbidity with other conditions, for example depression (Holaway & Heimberg, 2004).

Generally, outcomes for all modes of treatment are poorer for socially anxious people with comorbid conditions such as other anxiety disorders, depression/mood disorders, avoidant personality disorder and substance use (including alcohol) disorders. The following section provides a summary of these comorbidities ending with the social anxiety/substance use comorbidity, as this comorbidity is the focus of this thesis.
Co-morbidities

Social anxiety disorder frequently co-occurs with other psychological disorders. In Canada, of people with lifetime SAD, 52% met criteria for at least one other lifetime mental disorder, while 27% met criteria for three or more lifetime mental disorders (Chartier, Walker, & Stein, 2003). In the U.S. between 63% and 90% of people (depending on the number of social fears) with lifetime SAD, were found to meet criteria for at least one other lifetime mental disorder. Respondents were assessed on SAD based on their endorsement of between one and 14 social interactional and/or social performance fears (Ruscio et al., 2008). In terms of strength of association or probability of comorbidity, SAD is most strongly associated with other anxiety disorders, followed by mood disorders and then substance use disorders (Fehm, Beesdo, Jacobi, & Fiedler, 2008). Age of onset of social fears has been found to precede age of onset in 32% of cases of comorbidity with other anxiety disorders, in 71% of cases with comorbid mood disorders and in 80% of cases with comorbid substance use disorders (Chartier et al., 2003). As previously noted in this chapter, SAD also can co-occur with APD. It also co-occurs less often with other mental disorders such as eating disorders and body dysmorphic disorder (Fehm & Wittchen, 2004). This section provides a synopsis of the most frequent comorbidities with SAD.

Other anxiety disorders. General population surveys in Western countries like the U.S.A. and Australia, demonstrate that there is a high rate of comorbidity amongst anxiety disorders (McEvoy et al., 2011). Up to 50% of people with SAD have reported at least one other anxiety disorder (Fehm & Wittchen, 2004). Results from studies considering prevalence rates or odds ratios (likelihood of occurrence) of SAD comorbidities with other anxiety disorders have varied due to differences in structure and content of surveys/interviews (Chartier et al., 2003). Earlier surveys demonstrated that simple phobia has the highest prevalence rate (25.4%) with SAD, followed by agoraphobia (13.2%), generalised anxiety disorder (7.4%) and lastly panic disorder (5.8%) (Chartier et al., 2003). More recent information from the NCS-R conducted in the U.S.A. indicates that co-occurring SAD with agoraphobia (without panic) has the highest odds ratio, followed by co-occurring SAD and specific phobia, then generalised anxiety disorder, panic disorder, separation anxiety disorder, and lastly post-traumatic stress disorder (Ruscio et al., 2008). Overall, the comorbidity of SAD with other anxiety disorders is associated with greater
impairment in social, educational and occupational domains (Filho, Freitas, Martin-Santos, de Souza Crippa, & Nisihara Chagas, 2013). The increased level of suffering and interference with daily life is more likely to lead people with SAD and a co-occurring anxiety disorder to seek treatment.

**Mood disorders.** SAD is also frequently comorbid with mood disorders. Community studies indicate that around 41% of participants with SAD also have a mood disorder with strong associations existing between SAD and co-occurring major depressive disorder, dysthymia, and bipolar disorder (Fehm & Wittchen, 2004). Earlier surveys indicated that major depression was the most prevalent comorbid disorder with SAD (18.8%), followed by dysthymia (8.8%) and lastly bipolar disorder (4.0%) (Chartier et al., 2003). More recent population survey results indicate the most likely co-occurring mood disorder with SAD is dysthymia, followed by an equal probability of SAD co-occurring with major depressive disorder, and also with bipolar disorder (Ruscio et al., 2008).

The onset of a depressive disorder for a socially anxious individual can be partly contributed to by having less social support compared to other people, as a result of having fewer friends (Rapee & Sanderson, 1998). Adding to the depressed state can also be the socially anxious individual’s negative cognitions resulting from low self-esteem. This can lead the SAD individual to conclude that any negative evaluation is accurate which can worsen the depressed state. This can result in low motivation and hopelessness and interfere with treatment. On the other hand, if the individual feels sufficiently motivated to commence treatment, mood and motivation are likely to improve when treatment gains become apparent to the affected individual (Rapee & Sanderson, 1998).

Recent research has indicated that there are different treatment mechanisms at work for people affected by both SAD and major depression (Moscovitch, Hofmann, Suvak, & In-Albon, 2005). In the Moscovitch study, people diagnosed with SAD participated in group-based CBT and self-reported levels of depression. Although both SAD and depression improved with treatment, only reductions in SAD fully mediated reductions in depression, but not vice versa, that is, 91% of the variance in reduced depression could be accounted for by reductions in SAD symptoms, but only 6% of reduced depressive symptoms accounted for reduced SAD symptoms. These results indicate that SAD is improved via group CBT by distinct mechanisms from those that reduce symptoms of
depression in individuals affected by this comorbidity. As with other comorbidities, socially anxious individuals with a co-occurring mood disorder are more likely to seek treatment due to increased levels of suffering and interference with their daily lives.

**Avoidant personality disorder (APD).** SAD frequently also co-occurs with APD (Rapee & Sanderson, 1998). There is arguably considerable overlap between the diagnostic criteria for SAD and APD in the DSM (American Psychiatric Association, 2000). In preparation for publication of the DSM-5, it was recommended that APD retain its separate status as a personality disorder (Heimberg et al., 2014). In addition, the APD criteria were not changed from the previous DSM edition. APD requires a persistent pattern of feelings of inadequacy, social inhibition and hypersensitivity to negative evaluation which has been apparent in a variety of settings indicated by at least four out of a possible seven criteria. These criteria include avoiding occupational activities with others due to fears of criticism or rejection; unwillingness to become involved with others unless certain of being liked; showing restraint in close relationships due to fear of being ridiculed or shamed; being preoccupied with thoughts of being criticised or rejected in social situations; being inhibited in new interpersonal situations due to feelings of inadequacy; seeing oneself as socially inept, unappealing or inferior to others; and being unusually reluctant to take risks or to engage in new activities due to fears they may prove embarrassing (American Psychiatric Association, 2000).

Studies investigating the comorbidity between SAD and APD usually examine individuals with one of these diagnoses to see if there is overlap with the other diagnosis (Bögels et al., 2010). In people with APD, an average comorbidity prevalence rate of 42% for SAD has been reported with somewhat higher rates for generalised SAD (Alden, Laposa, Taylor, & Ryder, 2002). Socially anxious individuals who have also been diagnosed with APD are generally more severe on a number of indices. However, higher comorbidity prevalence rates would be expected if APD were simply a more severe form of SAD and recent studies have challenged such a hypothesis (Hummelen, Wilberg, Pedersen, & Karterud, 2007). Thus, although SAD is typically more comorbid with APD than with other personality disorders, and APD is more comorbid with SAD than with other anxiety disorders, there is not enough evidence to conclude that these two disorders are exclusively related, or to support a simple severity hypothesis (Bögels et al., 2010).
Reaching such a conclusion could also compromise the treatment of someone with significant deficits in the normal development of interpersonal relationships (a core feature of a personality disorder), by categorising that person as someone simply with severe social fears (Bögels et al., 2010). In order to ensure that SAD (and not APD) was assessed in this research study, a highly reliable measure, the Liebowitz Social Anxiety Scale (Liebowitz, 1987) was used. Furthermore, fear of negative evaluation, a core feature of SAD, was also measured, by the Brief Fear of Negative Evaluation scale (Leary, 1983).

**Substance use disorders (SUD).** A sizeable proportion of people with SAD will also have problems with drug or alcohol use (Fehm & Wittchen, 2004). Most research in this area has focused on the relationship between social anxiety and alcohol use. However, more recently research has also considered other substances/drugs both legal and illicit, including nicotine, cannabis and cocaine (Buckner, Heimberg, Ecker, & Vinci, 2013b; Fehm & Wittchen, 2004). This thesis focuses on co-occurring problematic alcohol use and problematic cannabis use with SAD. For instance, from a longitudinal cohort over a period of 14 years with a mean starting age of 16.6 years, Buckner et al. (2008a) found SAD to act as a unique and significant risk factor (compared to other anxiety disorders) for subsequent alcohol and cannabis dependence. Earlier epidemiological studies demonstrated that the prevalence rate of co-occurring alcohol abuse and/or alcohol dependence with SAD was 16.3%, followed by co-occurring alcohol dependence only (9.3%), alcohol abuse (7.2%), and lastly drug abuse and/or dependence (6.7%) (Chartier et al., 2003). More recent population survey results in the U.S.A. indicate that amongst respondents with SAD, 27.3% also have alcohol dependence, and 20.9% also have alcohol abuse (Schneier et al., 2010). These results indicate that current prevalence rates for co-occurring SAD and alcohol use disorders are quite high. Other recent survey results indicate that co-occurring substance abuse or dependence has a slightly higher odds ratio (3.0) than alcohol abuse or dependence (2.8) (Ruscio et al., 2008).

Co-occurring SAD and substance use disorders (SUD) produces greater impairment compared to either disorder on its own (Buckner et al., 2013b). Using data from a large U.S.A. population survey collected between 2001 and 2002 and then again from 2004 to 2005, drinking to self-medicate anxiety (including social anxiety) has been found to be associated with the subsequent occurrence or persistence of alcohol dependence in 12.7%
and 33.4% of cases respectively (Crum et al., 2013). Furthermore, co-occurring SAD and SUD may be more difficult to treat although there are efficacious treatments for each disorder alone.

Limited research has been carried out to inform treatment for the SAD-SUD comorbidity. One reason for this is that research investigating treatment for SAD has typically excluded people with co-morbid substance use problems, so that the efficacy of the intervention for SAD cannot be assumed to generalise to people with substance use problems (Baillie et al., 2013). Only one study to date has specifically examined the impact of a co-morbid SUD on SAD treatment outcomes (McEvoy & Shand, 2008). The severity of pre-treatment alcohol use significantly predicted change in social interaction anxiety (with less alcohol use being associated with more improvement), but not social performance anxiety. This finding is consistent with the idea of anxiety in social interaction situations being maintained by socially anxious individuals who drink to cope with their anxiety in such circumstances. Thus it may be more difficult to treat socially anxious people who drink to cope with their anxiety in social interaction situations.

**Summary**

This chapter has provided an overview of social anxiety, its features and current treatments. It has presented social anxiety as operating within a continuum framework starting with mild symptoms with little or no effect on the affected individual’s life, through extreme interference in many domains which can significantly affect the social anxious person’s quality of life. Although much research has been conducted to understand the nature, development and maintenance of social anxiety, results are insufficient to develop a fully comprehensive model of understanding of SAD in the context of common comorbidities such as SUD. Further research is needed to achieve this goal and to ultimately lead to better prevention strategies and improved treatment outcomes. Deepening our understanding of the comorbidity of social anxiety and SUDs forms the basis for this thesis. The next chapter considers SUDs.
Chapter 2: Substance Use Disorder

Introduction

Problematic substance use is rife in many countries and can contribute to a variety of problems for the individual as well as for communities, societies and countries in which they live. Substance use disorders (SUDs) are the fourth most prevalent class of mental disorders, after (starting from most common) anxiety, mood and impulse control disorders (Kessler et al., 2005b). They comprise alcohol abuse, alcohol dependence, drug abuse and drug dependence disorders with alcohol abuse followed by drug abuse being the most common within the category of substance use disorders (Kessler et al., 2005b). SUDs are often comorbid and sometimes precipitate the development of other psychological disorders. In many instances, the problematic use of substances develops as a way of managing or coping with stress or symptoms associated with other mental health problems.

The purpose of this chapter is to provide an overview of substance use disorder (SUD) including its nature, diagnostic criteria, prevalence, age of onset and course, gender distribution, impairment and quality of life, aetiology, treatment and treatment limitations, and comorbidities. The focus is on two psychoactive substances, alcohol and cannabis as these are the substances researched as part of this thesis.

The Nature of Substance Use Disorder

In the nineteenth century, alcoholism was conceptualized as a moral or character flaw. This idea was reflected in early editions of the DSM in which alcoholism and drug addiction were classified within the personality disorders (Saunders, 2006). Another early formulation of substance use problems was as a biologically determined disease process resulting in the affected individual’s predictable idiosyncratic response (Jellinek, 1960). This notion was adopted by self-help movements such as Alcoholics Anonymous. There was also the public health or epidemiological position whereby substance use problems were seen to have occurred due to their societal use, with the level of use being influenced by cultural traditions, availability of the particular substance, its ease of manufacture, distribution and price factors (Saunders, 2006). By the 1970s, many had dismissed the concept of SUDs as a disease process. This was partly due to the rise of social cognitive
theory which explained the development of problematic alcohol and drug use by way of many influences (e.g., social) on behaviour, and also by way of cognitions about consequences of use (Bandura, 1977b). Recently the emphasis on the biological viewpoint has re-emerged with developments into understanding substance use problems from new knowledge about neurobiological processes and related findings from genetic research (Saunders, 2006).

Despite these competing models of substance use, alcohol and drug use disorders have come to be defined in a practically useful and empirically supported way via the concept of the “dependence syndrome” first coined by Edwards and Gross in 1976 in relation to alcohol use (Edwards & Gross, 1976). Rather than being aetiological in nature, the dependence syndrome was more descriptive of experiences, behaviours and symptoms related to repetitive alcohol use, clustered in time, and occurring repeatedly (Saunders, 2006). The notion of the dependence syndrome has been adopted for other substances which also have the potential for reinforcement of use. These include cannabis, opioids (prescribed and illicit), benzodiazepines, cocaine, amphetamines, nicotine, caffeine and anabolic steroids (Saunders, 2006). The dependence syndrome was later supplemented by four other types of drug use which also reflect repetitive substance use but which do not fulfil the requirements of substance dependence (Saunders, 2006). These were “unsanctioned use” (does not conform to societal norms or mores); “dysfunctional use” (leads to impaired psychological or social functioning); “hazardous use” (increases the risk of harmful consequences for the user); and “harmful use” (causes damage to physical or mental health of the user) (Edwards, Arif, & Hodgson, 1981). The dependence syndrome concept has been adopted for the classification of psychoactive substance use disorders in DSM editions starting from DSM-III-R, published in 1987.

**Diagnostic Criteria**

A high level of detail is contained in each (relevant) DSM edition with respect to substance use disorders, related mental disorders, classes of substance, subtypes and course of disorder specifiers. As DSM-IV criteria (American Psychiatric Association, 1994) were used to diagnose substance use disorders when data was collected for this thesis, this section focuses on those criteria, as well as the changes that have come into effect as a result of the publication of DSM-5 (American Psychiatric Association, 2013).
As with social anxiety disorder, substance use disorders were not classified separately until the third edition of the DSM in 1980 (American Psychiatric Association, 1980; Saunders, 2006). In DSM-III, pathological use of substances was divided into “Substance Abuse” and “Substance Dependence”. Substance dependence was conceptualised as a more severe form of substance use disorder, and required physiological dependence, evidenced by either tolerance or withdrawal.

DSM-III-R (American Psychiatric Association, 1987) expanded the definition of dependence significantly in accordance with the Edwards and Gross conceptualisation referred to above. It effectively reduced the term of “substance abuse” to a residual diagnosis that would only apply to individuals who did not fulfil the broad definition of dependence (Saunders, 2006).

DSM-IV (American Psychiatric Association, 1994) continued with the classification of substance use disorders into substance abuse or substance dependence, consistent with the conceptualisation in DSM –III-R. There were 11 classes of substances: alcohol, amphetamines, caffeine, cannabis, cocaine, hallucinogens, inhalants, nicotine, opioids, phencyclidine (PCP) and sedatives. Dependence was defined as three or more of the following criteria occurring at any time in the same 12 month period: tolerance; withdrawal; using the substance in larger amounts or over longer time periods than intended; desire or unsuccessful efforts to reduce or control use; spending a lot of time obtaining, using or recovering from substance use; stopping or reducing important social, occupational or recreational activities; and continued substance use despite recognising its contribution to a psychological (e.g., depression) or physical problem (e.g., organ damage).

DSM-IV Substance Abuse referred to a maladaptive pattern of use leading to significant impairment or distress as evidenced by at least one of the following criteria occurring within the same 12 month period: recurrent use resulting in a failure to fulfil major role obligations at school, work or home; recurrent use in situations in which it is physically hazardous; recurrent substance-related legal problems; or continued use despite persistent or recurrent social or interpersonal problems contributed to by the effects of the substance. In addition, the symptoms must have never met criteria for Substance Dependence for the particular class of substance for the diagnosis of Substance Abuse to
apply. Overall, DSM-IV dependence criteria captured more people than the DSM-III version, and abuse was seen as a less serious condition (Saunders, 2006).

In DSM-5 (American Psychiatric Association, 2013), there are 9 classes of drugs which can be diagnosed as a SUD: alcohol; tobacco; cannabis; phencyclidine (PCP); inhalants; opioids; sedatives, hypnotics, and anxiolytics; stimulants (including amphetamine-like substances and cocaine); and other (or unknown) substances. There is no further classification into dependence and abuse categories amongst the substance use disorders. There are some differences in diagnostic criteria for different SUDs depending on the substance but for alcohol and cannabis use disorders (the drugs investigated in this thesis), the diagnostic criteria are the same.

To diagnose either alcohol use disorder (AUD) or cannabis use disorder (CUD), there must exist a pattern of problematic substance use during a 12 month period, which leads to significant distress or impairment indicated by at least two out of the following eleven criteria: using the substance in larger amounts or over a longer period than intended; a persistent desire or unsuccessful attempts to cut down or control substance use; a lot of time spent obtaining, using or recovering from the effects of the substance; a craving or strong desire to use the substance; chronic use of the substance resulting in failures to fulfil major obligations relating to school, work or home; continued use of the substance despite recurring social or interpersonal problems contributed to by the effects of the substance; ceasing or reducing important social, occupational or recreational activities due to substance use; persistent substance use in physically hazardous situations; continued substance use despite knowledge of a frequent physical or psychological problem likely to be contributed to or exacerbated by use of that substance; tolerance symptoms; and lastly, withdrawal symptoms (American Psychiatric Association, 2013). Cannabis withdrawal was non-existent in DSM-IV but has been included in DSM-5. In summary, the criteria comprising substance abuse and substance dependence in earlier DSM editions have now been combined for each substance use disorder, providing a more coherent way of diagnosing problematic substance use.

**Prevalence**

The use and abuse of substances is common throughout the world with tobacco and alcohol use being respectively the fourth and fifth greatest contributors to the international...
burden of disease (Reid, Lingford-Hughes, Cancela, & Kalivas, 2012). Cannabis is the most widely used illicit drug throughout the world (Khan et al., 2013). In the U.S.A., results from a large population survey, the 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) indicate 12 month prevalence rates based on DSM-IV criteria for alcohol use disorders are 4.65% for abuse and 3.81% for dependence (Grant et al., 2006a). In Australia, based on data collected in the 2007 National Survey of Mental Health and Wellbeing (NSMHW), which also used DSM-IV criteria, lifetime and 12 month prevalence rates are 18.3% and 2.9% respectively for alcohol abuse and 3.9% and 1.4% respectively for alcohol dependence. The prevalence rate for any lifetime alcohol use disorder is 22.1% (Teesson et al., 2010). Differences between the Australian and U.S.A. results can be explained by use of different interview measures and procedures to determine the presence of DSM-IV criteria, as well as by cultural differences (McBride et al., 2009).

Cannabis data in the U.S.A. based on the 2001-2002 NESARC indicate lifetime and 12-month prevalence rates of 8.5% and 1.5% respectively for any cannabis use disorder. Cannabis abuse has lifetime and 12-month prevalence rates of 7.2% and 1.1% respectively, whilst cannabis dependence has lifetime and 12-month prevalence rates of 1.3% and 0.3% respectively (Compton, Thomas, Stinson, & Grant, 2007). In Australia, results from the 2007 NSMHW indicate that lifetime and 12 month prevalence rates for cannabis use are 18% and 6% respectively, and prevalence rates for lifetime and 12 month cannabis use disorders are 6.2% and 1% respectively. The proportion of people who have ever used cannabis who have a lifetime cannabis use disorder is 32.2%, and the proportion of people who have ever used cannabis, and who had a cannabis use disorder in the last 12 months (at the time of the survey) is 14.3% (Teesson et al., 2012). As for alcohol use disorders, differences between U.S.A. and Australian results with respect to prevalence of cannabis use disorders may be explained by different interview measures and procedures used in the respective surveys, in addition to cultural influences.

One example of a cultural difference is that parents of Australian youth demonstrate more favourable attitudes to substance use compared to parents of American youth, and this is reflected in policy differences. The U.S.A. has a more conservative policy towards substance use of zero tolerance, compared to Australian policy which favours harm minimisation (McBride et al., 2009).
**Age of Onset and Course**

Many adolescents try substances as part of the developmental process which includes experimenting with adult roles and behaviour, establishing their individuality, and participating in similar activities with peers (Dodgen & Shea, 2000). However, only a small proportion continues using substances like alcohol and cannabis in a problematic way such that they develop a substance use disorder (SUD). In Australia, results from the 2007 NSMHW population survey indicate the mean ages of onset for alcohol abuse and alcohol dependence are respectively 22.7 (median 20) and 24.3 (median 20) years (Teesson et al., 2010). In contrast, the mean ages of onset of cannabis use and cannabis use disorders are respectively 16.9 (median 17) and 19.8 (median 18) years (Teesson et al., 2012). Empirical studies (cited in Henry et al., 2011) show that adolescents who commence drinking alcohol at age 14 or younger are at higher risk of developing subsequent problematic alcohol use and alcohol dependence (as defined by DSM-IV criteria). Similarly, Odgers et al. (2008) (cited in Henry et al., 2011) report that early use of other drugs is linked to later drug related problems including adult substance dependence.

With regard to research findings about later ages of onset, most research has been conducted in relation to alcohol, due to the illegal nature of many other drugs. Some people commence (or re-commence) using alcohol and/or other substances problematically at later life stages including the start of tertiary education (contributed to by exposure to peer pressure, additional opportunities for drinking for those who have moved out of home, and academic pressures); and the onset of retirement (contributed to by boredom, lack of structure and no adverse work consequences) (Jung, 2010). Overall, however, most studies suggest that problematic substance use (including alcohol) declines with age.

Another group of people that may develop problematic alcohol or substance use at different ages are those that do so in response to life stresses such as divorce, or job loss; and/or in response to the development of other psychopathology, for example anxiety disorders, mood disorders, post traumatic stress disorder. Whilst some studies have shown that substance use disorders precede the onset of other mental disorders, the majority of studies demonstrate that mental disorders precede the onset of substance use disorders (Glantz et al., 2009). Thus, although many people drink alcohol or use other substances, a
small minority may develop problematic alcohol or substance use in response to the onset of other mental disorders.

The course of a SUD can vary significantly depending on a number of factors. For example, studies indicate that over long periods of time, the number and types of symptoms associated with problematic alcohol use can vary, as can the severity of the disability and the treatment response (Vaillant, Gale & Milovsky, 1982). The course of other substance use disorders is likely to be similar. In general SUDs follow a chronic course, with a worsening progression for the more severely symptomatic, whilst others’ use of substances can lead to abuse at one point in time and “normal” or controlled use/abstinence during other periods. (Dodgen & Shea, 2000). Thus, the course of substance use disorders can vary significantly, so that where it co-occurs with another psychological disorder like social anxiety disorder, a careful assessment must be made of the course and/or severity of both disorders.

**Gender Distribution**

Population survey studies in the U.S.A., like the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) which used DSM-IV criteria for diagnoses of mental disorders, have found that men have a significantly higher 12 month prevalence rate of alcohol abuse (6.93%) compared to women (2.55%), as well as a significantly higher 12 month prevalence rate of alcohol dependence (5.42%) compared to 2.32% for women (Grant et al., 2006a).

In Australia, results from the 2007 NSMHW also indicate that men have a significantly higher 12 month prevalence rate of alcohol abuse (3.9%) compared to women (1.9%), and a significantly higher 12 month prevalence rate of alcohol dependence (2.1%) compared to 0.8% for women (Teesson et al., 2010). From the same survey in Australia, current cannabis use disorders are twice as common in men compared to women, with comparable results in the U.S.A. (Teesson et al., 2012).

With regard to gender differences in treatment for alcohol use disorders, men are more likely than women to have had treatment. This may be partly contributed to by barriers and biases to treatment access (Jung, 2010). It is also possible that more men receive treatment if the percentage of men needing treatment is higher, which seems to be
borne out by results from population surveys outlined above. With regard to cannabis use disorders, both genders exhibit low treatment-seeking rates (Khan et al., 2013).

It may be that with generational changes in the roles of men and women in society, gender differences in terms of prevalence rates and treatment will reduce. Some evidence for this comes from the findings of Grant and her colleagues in relation to U.S.A. population survey results (Grant et al., 2006a). When comparing differences between data collected in 1991-1992 and 2001-2002, the prevalence of alcohol dependence was found to have significantly decreased for men but remained unchanged for women.

**Impairment and Quality of Life**

There has been a long-standing awareness that people with alcohol or drug dependence are affected in a number of ways including physically, emotionally and in their various functional roles (Morgan, Morgenstern, Blanchard, Labouvie, & Bux, 2003), and therefore experience impaired quality of life. People with alcohol or other drug-related use problems can also impact others negatively not just through declines in interpersonal relationships but due to the increased risk of having car accidents. This negative impact on others can further impair the quality of life of people with SUDs.

People that have been treated for a substance use disorder and/or are in remission from a SUD, may never recover the same level of quality of life they previously enjoyed. Results from a large U.S.A. population survey (with data collected in 2001-2002 and 2004-2005) indicated that people who had remitted from alcohol dependence had lower quality of life scores compared to people with no lifetime history of this disorder, and people who had remitted from alcohol abuse or a cannabis use disorder did not report significant improvement of quality of life scores (Rubio et al., 2013). Thus, remission of alcohol or cannabis use disorders may not result in full restoration of health-related quality of life in people with one of these disorders.

Another study focused on data collected in 2001-2002 in relation to cannabis use and cannabis use disorders, from the same large U.S.A. population survey (Lev-Ran et al., 2012). Results demonstrated that cannabis users had significantly lower quality of life scores compared to non-users, as did people with a cannabis use disorder, compared to people without a cannabis use disorder. These findings indicate that people who have
problems with alcohol or cannabis use may have a reduced quality of life even during periods when they are not using these substances in a problematic way.

In clinical samples, overall quality of life has been found to be more affected by addiction severity and psychiatric comorbidity, rather than the type of substance dependence (Colpaert, De Maeyer, Broekaert, & Vanderplasschen, 2013). People with substance use disorders often have co-occurring mental disorders which further reduces their quality of life by increasing the impairment of psychological, cognitive, social, physical and occupational functioning. Comorbidities with substance use disorders are reviewed in the last section of this chapter due to the focus of this thesis on co-occurring SUDs with SAD. Aetiology of SUDs is discussed in the next section to clarify factors that might contribute to comorbid SUD with SAD.

**Aetiology**

As with other mental disorders, there are various genetic and environmental influences that interact to result in problematic substance use; however, there is no clearly enunciated explanatory model. This section describes the main biopsychosocial factors that may contribute to the development of SUDs focusing on those most relevant to this thesis.

**Genetic factors.** There is considerable evidence that a predisposition to substance abuse which can be genetically transmitted exists in some people. Early research via family, adoptee and twin studies focused on alcohol abuse as it was legal and prevalent (Collins & deFiebre; and Crabbe, McSwigan & Belknap, 1985, both cited in Dodgen & Shea, 2000). Studies demonstrate that about 25% of the sons of alcoholics became alcoholics, whilst almost one-third of alcoholics had one parent who abused alcohol. In addition, sons of alcoholics adopted by nonalcoholic parents were just as likely as sons raised by their biological alcoholic parents, to be alcoholic. A weaker but significant correlation has also been reported for biological mothers and daughters. Studies comparing monozygotic (identical) and dizygotic (fraternal) twins demonstrate a higher similarity for alcoholism in monozygotic twins (Dodgen & Shea, 2000).

More recent research indicates that up to 70% of the variance associated with the diagnosis of a substance use disorder (abuse or dependence) may be heritable (Kendler, Myers, & Prescott, 2007). Furthermore, 100% of genetic variance and 80% of phenotypic variance are shared among the different substance use disorder categories (Tsuang et al.,
1998, cited in Sloboda, Glantz, & Tarter, 2012). Thus, there is strong evidence for a genetic vulnerability to alcohol and other substance use disorders.

**Neurobiological factors.** Recent outcomes from research on the neurobiology of addiction have ascertained that psychoactive drugs activate dopamine release/transmission in corticolimbic brain regions albeit via differing mechanisms depending on the substance type (Reid et al., 2012). The release of dopamine otherwise naturally occurs when an environmental stimulus considered biologically important is encountered (e.g., food or sex). Its release has the effect of preparing brain neurons to more efficiently undergo the required neuroplasticity to learn how to obtain (or avoid) the important stimulus (Reid et al., 2012). Neuronal plasticity also underpins feelings (e.g., craving) and memories. Thus, the use of the psychoactive drug is reinforced, substance use can be perpetuated, and substance dependence can evolve (Self & Tamminga, 2004). In particular, people who are vulnerable to problematic substance use may have a heightened sensitivity to reward which may increase the need for dopamine release. Sensitivity to reward is one of the shared vulnerability factors being considered in this thesis in terms of its contribution to the SAD–SUD comorbidity. This issue is discussed in more detail in Chapter 3.

Other neurobiological research points to the importance of connections from the frontal cortex to the hypothalamus via the hypothalamic pituitary adrenal (HPA) and hypothalamic pituitary gonadal (HPG) axes. For example, the HPA axis regulates a person’s response to environmental threat via the release of glucocorticoids from the adrenal gland. Ongoing stress (e.g., from family conflict) affects homeostatic regulation resulting in low cortisol levels. This may increase the predisposition for substance use due to diminished physiological activation of cortisol (Sloboda, Glantz, & Tarter, 2012). Further research could enable a more comprehensive understanding of the biological mechanisms underlying psychological risk for problematic substance use.

**Temperamental factors.** Some temperamental factors have been implicated with increased risk for SUDs, including impulsivity or lack of self-control, novelty/sensation-seeking, hyperactivity, and aggression (Kassel, Weinstein, Skitch, Veilleux, & Mermelstein, 2005; Sloboda et al., 2012). Childhood mental disorders such as conduct disorder and attention deficit hyperactivity disorder are significantly heritable and contain the same characteristics which precede drug use, (i.e., inattention, behaviour undercontrol,
and overactivity). Adult personality disorders such as borderline personality disorder in women and antisocial personality disorder in men also incorporate undercontrolled behaviour and commonly co-occur with drug use (Sloboda et al., 2012). Thus, young children exhibiting such temperament traits (which have a strong genetic basis) may be at increased risk for problematic substance use. Interestingly, these traits appear to be at the opposite end of the same spectrum, with the behavioural inhibition end (described in Chapter 1 of this thesis), increasing the risk of young children developing SAD. Gray’s (1970) biologically-based theory of personality, which he called “reinforcement sensitivity theory” (RST), comprises two primary systems: (a) the Behavioural Approach System (BAS) which is characterised by a sensitivity to reward stimuli; and (b) the Behavioural Inhibition System (BIS), characterised by a sensitivity to potentially threatening stimuli. This theory is explored in greater detail in Chapter 3 of this thesis, which describes important theories (including RST) and factors relevant to the SAD-SUD comorbidity.

Psychopathology (other mental disorders) is a widely recognized risk factor for problematic substance use (Sloboda et al., 2012). Specific comorbidities with substance use disorders are discussed in more detail in the last section of this chapter. It may be that it is not mental disorders themselves that increase the risk for problematic drug use but rather common underlying conditions, dysfunctions or deficiencies (Sloboda et al., 2012).

**Developmental factors.** From a developmental perspective, there are a number of psychological (e.g., cognitive and behavioural), social and other environmental factors which may contribute to the risk of developing a SUD. During the prenatal period and early childhood, these factors include exposure to teratogens (e.g., viruses, bacteria, chemicals, drugs) during foetal development, which can affect normal development including physical, cognitive, emotional and behavioural functioning of the affected child. In turn, these injuries can present challenges to parenting abilities which can then negatively influence the quality of parent-child attachment and bonding (Sloboda et al., 2012). This influence can subsequently affect the establishment and consolidation of basic psychological competencies including acquiring appropriate social skills to form friendships. Other early childhood factors which can increase the risk of subsequent substance use include socioeconomic disadvantage, exposure to parental modelling of substance use, community violence and limited parental guidance (Sloboda et al., 2012).
In middle childhood, as more time is spent away from the family and at school, risk factors for problematic use include permissive parenting, low parental warmth, harsh discipline, tolerance of substance use, school failure, peer rejection, low school commitment, deviant peer group, and accessibility/availability of substances. At an individual level, risk factors include poor impulse control, lack of behavioural/self-control regulation, aggression, antisocial behaviour, sensation seeking. In addition, mental disorders such as anxiety disorders, impulse control disorders, and conduct disorder which have their onset during this period of development, can further impede healthy attachment to school and peers, adaptive learning, and self-regulation (Sloboda et al., 2012).

Adolescence is a unique period of vulnerability to substance use. Significant changes to the adolescent’s brain occur during a time when more adult roles and behaviours are being assumed. Evidence shows that changes occur in the mesocorticolimbic dopaminergic systems (which can positively reinforce drug use as described earlier in this section), as well as via the maturation of the prefrontal cortex and amygdala (Kassel et al., 2005). The prefrontal cortex is the last region of the brain to fully mature and drives executive cognitive functions, including decision-making, self-monitoring, abstract thinking, and goal-directed behaviour (Sloboda et al., 2012). It also oversees behavioural and affective regulation whilst the amygdala is involved in emotional reactivity and emotional processing. Thus, the adolescent can be exposed to extreme emotional and drive states and the likelihood of using and abusing substances is increased (Steinberg et al., 2004, as cited in Kassel et al., 2005). In addition, drug use can hamper neurodevelopment and delay the acquisition of psychological self-regulation (Sloboda et al., 2012). These changes during adolescence occur in addition to other ongoing vulnerabilities the adolescent may have developed or may continue to be exposed to from an earlier developmental phase (individual, family/contextual, school/peers, community risk factors mentioned in preceding paragraphs).

Another vulnerability factor for the development of substance use in adolescence is a cognitive one, that is, the beliefs or expectancies one holds about the effects of using alcohol or other substances. Alcohol expectancies are formed in childhood largely based on parental modelling and before actual use. However, expectancies during adolescence can be significantly affected by the individual’s first experiences of substance use. There is
strong evidence to show that adolescents who hold positive expectancies about the effects of alcohol consumption are at a higher risk of problematic alcohol use (Kassel et al., 2005). The role of expectancies on the relationship between social anxiety and problematic substance use is being investigated in this study and is considered in more detail in the next chapter of this thesis.

A final vulnerability factor for adolescents is their greater risk of developing problematic drug use as a way of coping with stress, and in the absence of alternative coping repertoires (Kassel et al., 2005). Over the last 20 years, the stress-coping model of drug abuse has become quite influential, partly due to its integrative framework (Wills, 1990). Past research has shown that maladaptive coping styles are related to problematic drug use (Cooper, Frone, Russell, & Mudar, 1995), that strong associations exist between various indices of subjective stress and problematic drug use, and that individuals’ motives to use drugs are related to substance-use behaviour and consequences (Kassel et al., 2005). One two year longitudinal study found that behavioural coping (an adaptive form of coping) was inversely associated with the initial level of adolescent substance use, as well as with growth over time of substance use with peers (Wills, Sandy, Yaeger, Cleary, & Shinar, 2001). The role of coping strategies in the SAD-SUD relationship is considered in Chapter 3 of this thesis.

Prevalence rates for substance use are highest for the late adolescence/early adulthood period (age 18 to 25) (Sloboda et al., 2012). From a contextual point of view, this period coincides with greater independence and moving out of home in addition to the other risk factors mentioned previously in this section. Adulthood is usually associated with a reduction in prevalence of drug use, although adults who have already experienced problematic substance use and/or are going through difficult transitions (e.g., getting married, having children, changing jobs), are likely to be more vulnerable. Other contextual risk factors at this time include detachment from family and associating with drug-using peers (Sloboda et al., 2012).

In summary, a number of genetic, neurobiological, cognitive, behavioural, social, and environment factors can interact and contribute to the development, onset, and course of problematic substance use. Given the complexity of SUDs, which can be further complicated by the type of substance being used or abused, this study focuses on two
prevalent substances, alcohol and cannabis, and their relationship to SAD. The roles of three factors relevant to both SAD and SUD, that is personality (via sensitivity to reward and punishment), alcohol expectancies and coping strategies, will be investigated to assist in further understanding and potentially improving treatment for people with this co-morbidity.

**Treatment and Treatment Limitations**

Treatment of SUDs varies considerably as it takes into account factors such as type of substance, and stage, course, and severity of substance use problem/s. In addition, differences exist when considering treatment for different populations, including people with comorbid conditions, people in various settings including hospitals (inpatients, outpatients, day treatment), prisons, and residential treatment within specialised drug treatment facilities. This section outlines current treatments for SUDs focusing on alcohol and cannabis use disorders, and also summarises the main treatment limitations. There are two key forms of treatment for SUD: pharmacotherapy and psychosocial/psychotherapy treatments.

**Pharmacotherapy.** The first main form of treatment for substance use disorders (including both alcohol and cannabis) is pharmacotherapy. Pharmacotherapy is primarily used to reduce withdrawal symptoms and to ensure the physical safety of people with substance dependence symptoms. Some medications can also prevent intoxication, reduce cravings and cause substance aversion (Jung, 2010). For alcohol dependence, the ultimate goal of pharmacotherapy treatment is to stabilize abstinence via relapse prevention after detoxification, whilst an interim goal is to reduce alcohol consumption in order to diminish its harmful effects (Franck & Jayaram-Lindström, 2013). The first phase of treatment is usually detoxification with sedatives, for example benzodiazepines, to treat or to prevent withdrawal symptoms. This is followed by pharmacotherapy to prevent relapse to heavy drinking, as well as to provide support for abstinence and psychosocial interventions (Navarro, Shakeshaft, Doran, & Petrie, 2012).

The three main drugs approved for use for alcohol dependence are disulfiram, naltrexone and acamprosate. Disulfiram (“Antabuse”) is designed to deter alcohol use by causing unpleasant effects, naltrexone prevents the reinforcing effects of alcohol and reduces cravings, and acamprosate (“Campral”) is believed to reduce withdrawal associated
distress and to alter learning responses to alcohol cues. The overall effect size of these three drugs is moderate (Franck & Jayaram-Lindström, 2013). In Australia, acamprosate and naltrexone are the two most common drugs used to treat alcohol dependence (Navarro et al., 2012).

Unlike alcohol dependence, there are no established pharmacotherapy interventions for cannabis use disorders (Vandrey & Haney, 2009). Medications that can potentially reduce withdrawal symptoms (e.g., dysphoric mood, loss of appetite, insomnia), the likelihood of relapse and reinforcing effects are being investigated. Potential pharmacotherapy which has undergone preliminary investigation via controlled laboratory studies, and small clinical studies but which still requires controlled clinical trials to establish efficacy, include buspirone, dronabinol, fluoxetine, lithium and lofexidine (Vandrey & Haney, 2009).

Alcoholics Anonymous. The second main form of treatment for substance use disorders (including both alcohol and cannabis) is psychotherapy/psychosocial treatments. In the U.S.A. the main approach used to treat people with problematic substance use is to encourage participation in a 12-step support group like Alcoholics Anonymous (AA) (Randall, Book, Carrigan, & Thomas, 2008). Both meta-analytic reviews and prospective studies have shown that AA provides positive but modest benefits to problem substance users (Tonigan et al., 2010).

Cognitive-behavioural treatments (CBT). Most other psychosocial treatments are cognitive-behavioural in nature (CBT) and include variations such as harm minimization, motivational interviewing, relapse prevention, and contingency management. Harm minimization (HM), also called harm reduction, employs methods such as short-term and long-term pharmacological substitution, education about safer substance use, and treatment goals such as reduced or moderate substance use (Towsley, 2013). Motivational interviewing (MI) is designed to motivate the substance user to change their substance use (reduce or cease using) by increasing their awareness of the effects of the substance on them, as well as exploring and resolving ambivalence about changing their substance use behaviour (Apodaca & Longabaugh, 2009; Miller & Rollnick, 2013). Relapse prevention (RP) is a cognitive-behavioural strategy designed to teach individuals trying to change their addictive behaviours such as drinking or substance use, how to anticipate and cope with the
problem of relapse (Marlatt & Donovan, 2008). Contingency management (CM) is based on operant conditioning principles and provides monetary or non-monetary rewards contingent on negative toxicology screens which indicate abstinence from drug use (Dutra et al., 2008).

Psychosocial interventions for substance use disorders (including cannabis). A recent meta-analytic review of psychosocial interventions for SUDs identified 34 randomised controlled trials investigating the efficacy of psychosocial treatments for SUDs excluding alcohol and nicotine use disorders (Dutra et al., 2008). Five of these trials were for cannabis. Treatments evaluated included CM, RP, general CBT, and CBT combined with CM. The results suggested that the benefits of psychosocial treatments are of moderate effect size, with these interventions being the most efficacious for cannabis use, and least efficacious for polydrug use (Dutra et al., 2008).

Another review of the literature on cannabis treatment found that generally there was no difference in efficacy with respect to different psychotherapies (mostly CBT or CBT-related), although psychotherapy treatments were more efficacious compared to no treatment (Nordstrom & Levin, 2007). The only exception was the use of vouchers (contingency management) which was more efficacious compared to other psychotherapy treatments in two of the reviewed studies. In addition, there were no differences in efficacy depending on length of psychotherapy treatment. Overall, there appear to be modest benefits of using CBT and CBT-related treatments for cannabis use disorders.

Psychosocial interventions for alcohol use disorders (AUD). With regard to psychosocial interventions for alcohol use disorders, a review of treatments for adolescents concluded that the most efficacious treatments were Multidimensional Family Therapy (MDFT) and group administered CBT (Perepletchikova, Krystal, & Kaufman, 2008). MDFT focuses on four areas: individual characteristics of the adolescent (perceptions about use, using behaviour, coping strategies, emotion regulation processes); the parents (personal issues, parenting practices); family interaction patterns; and outside influences such as school, legal system. However, more research is needed in this area given the high rates of relapse and the lack of research into the efficacy of pharmacotherapy for adolescents with alcohol use disorders.
The most effective psychosocial treatments for alcohol use disorders based on evidence from multiple trials and/or meta-analyses are CBT, two CBT-related treatments (MI and CM, described above), the brief intervention and the community reinforcement approach (Arias & Kranzler, 2008). The brief intervention treatment is targeted at hazardous and/or harmful drinkers with the intention of reduced alcohol use. It usually consists of CBT-related strategies including MI, psychoeducation, alternate coping strategies/behaviours, provision of self-help materials and counselling. The intervention can be as short as one 5 minute session or a total of 3 or 4 sessions including follow up sessions. Several reviews of brief interventions conducted in the last 25 or so years have found efficacy for substantial reductions in alcohol use which have been maintained for up to 12 months following the intervention (Funderburk, Maisto, Sugarman, Smucny, & Epling, 2008). Another meta-analytic review of 22 studies on the efficacy of self-help material for problem drinkers found modest support for reduced harmful and risky drinking (Apodaca & Miller, 2003). The community reinforcement approach is multidimensional and incorporates marital/family counselling, relapse prevention, employment counselling, and social-recreational counselling (Dodgen & Shea, 2000, p.123). Overall, CBT and CBT-related treatments have shown modest benefits for people with problematic alcohol use. In addition, MDBT has been shown to be efficacious in treating adolescents with alcohol problems. However, more research is needed to investigate the efficacy of psychosocial treatments for people with alcohol use disorders.

Other psychosocial interventions established as efficacious in reviews of treatment approaches for alcohol use problems include additional variations of CBT such as behavioural couples therapy, behavioural self-control training (which combines self-monitoring of alcohol use with behavioural strategies to reduce alcohol use), and social skills training (behavioural activities to enhance social skills, refuse alcohol and/or cope with stressful situations). There have been mixed findings for cue exposure, (the use of deconditioning procedures to reduce urges to drink and cravings). Overall, meta-analyses for MI, CBT and some of the other psychosocial interventions described above in relation to alcohol use disorders, indicate typical effect sizes to be in the low to moderate range (Martin & Rehm, 2012)
Combining treatment modes. There is also some evidence for the efficacy of combining psychosocial and pharmacological treatments for alcohol dependence. Vaughn and Howard (2004) conducted a systematic review of 14 controlled trials which integrated psychosocial interventions with opioid antagonist medication (naltrexone in 13 studies and nalmefene in 1 study) for participants diagnosed with alcohol dependence (according to DSM-IV criteria). They found that relapse rates, craving levels and post-treatment consumption levels were significantly lower in the group of participants receiving both treatments compared to the group of participants receiving psychosocial treatment only (combined with placebo). However, long-term efficacy (beyond 12-14 weeks) of combining pharmacological and psychosocial treatments for alcohol use disorders was not evident.

More recently, 1,383 alcohol dependent (according to DSM-IV criteria) participants were randomised into one of eight groups receiving naltrexone, acamprosate, both or placebo, with or without behavioural interventions (the COMBINE study) (Anton et al., 2006). Whilst at the end of the 16 week trial all patients showed reduced drinking, only patients receiving naltrexone, behavioural interventions or both these treatments combined, had significantly better outcomes on reduced drinking measures compared to the group receiving placebos. There was no difference for patients receiving acamprosate either on its own, combined with naltrexone, behavioural intervention or both, compared to those receiving placebo. Although group differences were similar one-year post treatment, they were no longer significant. Thus, more research in needed to determine or clarify the efficacy of combining pharmacological with psychosocial treatment for alcohol dependence.

As outlined earlier in this section, there is a lack of evidence at this stage to support specific pharmacological treatments for cannabis use disorders. Therefore, there is also a deficiency of evidence for the combined use of medication with psychosocial treatments for cannabis use although efficacy for some psychosocial treatments has been described earlier in this section. Much more research is required to develop treatment for cannabis use disorders, especially as to the efficacy of medications and the combination of pharmacotherapy with psychosocial treatments.
**Interpersonal therapy.** There is also limited research on other types of psychotherapy such as interpersonal/psychodynamic therapies and ACT, in treating people with substance use problems. The underlying assumption in using interpersonal therapy (IPT) to treat substance users is that the substance is being abused to make up for deficiencies in interpersonal skills, and this type of therapy aims to solve/alleviate interpersonal problems and develop new skills to alleviate distress, which in turn leads to a reduced reliance on the substance (Weissman, Markowitz, & Klerman, 2000, as cited in Brache, 2012). Very few controlled trials have been conducted comparing IPT with evidence-based interventions on substance abusers without comorbid conditions. These trials were conducted 20 or more years ago. One finding that did emerge is that IPT is comparable to RP in treating low-severity cocaine addiction (Najavits & Weiss, 1994, as cited in Brache, 2012). No trials have been conducted comparing IPT with other treatments for alcohol or cannabis abusers. However, IPT has influenced other treatments for substance abusers including group therapy, 12-step programs (such as AA) and MI. Suggested adaptations to improve the use of IPT as a treatment for SUDs include helping the patient find a meaningful place in society, encouraging social bonding with non-users, developing a strong therapeutic relationship to increase the patient’s capacity to self-soothe, and incorporating elements of MI, for example exploring ambivalence about substance use (Brache, 2012).

**Psychodynamic psychotherapy.** The underlying assumption for using psychodynamic psychotherapy as a treatment for SUDs is that people use substances to cope with difficult, overwhelming emotions. This is also known as the self-medication hypothesis (Khantzian & Albanese, 2008, as cited in Gottdiener, 2013). The self-medication hypothesis is described in more detail in relation to the SAD-SUD comorbidity in Chapter 3 of this thesis. The psychodynamic model suggests that self-medication occurs due to deficiencies in defence mechanisms including self-deception, denial and rationalizations. People with SUDs are thought to have a much lower tolerance for dysphoric emotions compared to non-users. They are also thought to be more impulsive and therefore need to immediately gratify their painful emotions with substance use (Gottdiener, 2013). Research has established that psychodynamic psychotherapy improves the functioning of defence mechanisms in people with varying psychopathologies including
treatment of men with alcohol use disorders (Gottdiener, 2013). However, the studies cited by Gottdiener (2013) in relation to alcohol use disorders were completed 30 to 40 years ago. No research appears to have been conducted more recently to determine the efficacy of psychodynamic psychotherapy treatments for people with SUDs exclusively (without comorbid conditions). Integrating psychodynamic psychotherapy with other evidence-based interventions including CBT and 12 step programs is suggested to increase its efficacy (Gottdiener, 2013). Overall, there appears to be very little evidence as to the efficacy of interpersonal or psychodynamic therapies in treating people with substance use disorders (and without comorbid disorders). Future research could include the integration of these therapies with existing evidence-based interventions to determine if treatment outcomes can be improved.

**Acceptance and commitment therapy (ACT).** Due to the relatively recent development of ACT as a treatment for various psychopathologies (see description of ACT in Chapter 1, Treatment section of this thesis), there is limited empirical data as to its efficacy including the treatment of SUDs. A stage 1 pilot study investigating the use of ACT for methadone detoxification found that 37% of people randomized to ACT had successfully detoxified after the 6 month methadone reduction dose program, compared to 19% of a drug counselling treatment condition. In addition, fear of detoxification was reduced across time in the ACT treatment compared to the drug counselling treatment group (Stotts et al., 2012). A case study reports the use of ACT to treat a middle-aged alcohol dependent client via “acceptance” of what he couldn’t change (the urge to drink) and changing what he could, by clarifying his values and applying them to goals in several life domains. By the end of treatment, the client reported improved quality of life and almost 100% sobriety. Furthermore, these results were maintained nine months after treatment (15 weekly therapy sessions) ended (Heffner, Eifert, Parker, Hernandez, & Sperry, 2003). Another study involved three cannabis-dependent adults who were individually treated with ACT (Twohig, Shoenberger, & Hayes, 2007). At post-treatment, all three reported zero cannabis use (this was confirmed through testing with oral swabs). At three-month follow-up, one was still abstinent and the other two were using (self-reported) at reduced levels (compared to baseline). Despite methodological concerns with the cannabis study, the use of ACT as a treatment for alcohol and cannabis use disorders
shows early signs of promise. Further research is required to clarify the efficacy of ACT compared to other current treatments, and to establish longer-term follow-up outcomes for all substance use disorder treatments.

**Treatment limitations.** As previously mentioned in this chapter, there are numerous factors that can affect the onset, course, maintenance and treatment of substance use disorders. To date, research has supported modest efficacy for psychosocial treatments for both alcohol and cannabis use disorders, as well as modest support for pharmacological treatments for alcohol use disorders. No empirically supported pharmacological treatments for cannabis use disorders exist at the present time. More research is required to ascertain supportive and effective treatment approaches for people with SUDs.

Another factor which can impact treatment outcome with respect to psychosocial/psychotherapeutic interventions for SUDs not mentioned thus far is the impact of the therapist. Martin and Rehm (2012) cite various studies in support of the contention that therapist effects account for more variance in treatment outcome compared to specific treatment types. The ability of clients with SUDs to form a strong therapeutic alliance with their therapist is another critical factor impacting on treatment outcome.

Finally, a significant treatment limitation for people with SUDs is the high level of comorbidity with other substances (i.e., polydrug users), as well as comorbidities with other mental disorders, for example, anxiety, mood, and psychotic disorders. Such co-morbidities further complicate treatment for people with a substance use disorder. There is limited evidence as to the efficacy of treatment for comorbid SUDs with other psychological disorders. For example, only a few studies have examined the impact of comorbid anxiety disorders and/or mood disorders on substance treatment outcomes. Some studies have reported lower rates of abstinence and higher risk of relapse in alcoholics with co-occurring anxiety or depression compared to alcoholics with no comorbidity (Driessen et al., 2001; Kushner et al., 2005). Kushner and colleagues (2005) found that amongst people with alcohol use disorders and co-occurring anxiety disorders or depression, SAD was the strongest predictor (out of other anxiety disorders and depression) of returning to drinking after treatment for the alcohol problem. In contrast, no greater risk of relapse or shorter period of abstinence was found in people with alcohol use disorders and co-occurring social phobia or agoraphobia (Marquenie et al., 2006).
The limitations of existing treatment of SUDs are generally amplified when considering treatment of SUDs with comorbid mental conditions. Overall, treatment outcomes for people with problematic substance use and comorbid conditions such as poly-substance use, mood disorders, anxiety disorders and psychotic disorders are worse compared to those with one SUD. The following section summarises comorbidities with SUD and ends with the alcohol use/social anxiety and cannabis use/social anxiety comorbidities, the subjects of this thesis.

Co-morbidities

Substance use disorders (SUDs) are highly comorbid with each other, as well as with other psychiatric disorders. Furthermore, psychopathology is a widely recognized risk factor for problematic substance use (Sloboda et al., 2012). As this thesis focuses on alcohol and cannabis, this section examines co-occurring alcohol and cannabis use disorders, as well as comorbidities of each of these substances with other mental disorders.

Other substance use disorders (SUDs). Past population studies conducted between the early 1980s and the late 1990s in the U.S.A., Canada and Australia have demonstrated that, for people with an alcohol use disorder, there is a higher likelihood of also having another substance use disorder compared to people without an alcohol use disorder, and vice versa (Stinson et al., 2006). More recently, from data collected in Australia for the 2007 NSMHW, Teesson et al. (2010, 2012) found significant associations between current alcohol use and other drug use disorders, as well as strong associations between current cannabis use disorders and alcohol use disorders. Alcohol use disorders had 12-month prevalence rates of 11.4% with any cannabis use disorder, and 4.6% with any other drug use disorder. Comorbidity rates were higher for alcohol dependence, which had a 12-month prevalence rate of 17.9% with any cannabis use disorder and a 12-month prevalence rate of 7.2% with any other drug use disorder (Teesson et al., 2010). Even higher 12-month prevalence rates were found for cannabis use disorder comorbidities, 45.1% with alcohol abuse, and 26.6% with alcohol dependence (Teesson et al., 2012).

From data collected in the U.S.A. for the 2001-2002 NESARC, Stinson et al. (2006) reported the 12-month prevalence rate of respondents with an alcohol use disorder who also had any drug use disorder was 55.17%, and the 12-month prevalence rate of respondents with an alcohol use disorder who also had a cannabis use disorder was 57.63%.
Prevalence rates of respondents with any drug use disorder who also had an alcohol use disorder was 13.05%, and prevalence rates of respondents with a cannabis use disorder who also had an alcohol use disorder was 9.89%. Overall, in both Australia and U.S.A., comorbidity rates between alcohol use disorders and cannabis use disorders, as well as between alcohol use disorders and other drug use disorders are quite high. These results highlight the need to conduct further research to improve prevention and treatment strategies for individuals with problematic polydrug use.

**Psychotic and personality disorders.** Evidence supports high comorbid rates between substance use disorders and serious psychiatric disorders including psychotic and personality disorders. Prevalence rates as high as 56% for comorbid SUDs and bipolar I disorder, and 33.7% for comorbid SUDs and schizophrenia spectrum disorders have been reported in some studies (Ralevski, Gianoli, McCarthy, & Petrakis, 2014). Data collected in the U.S.A. for the 2002 NESARC indicated that people with a lifetime diagnosis of schizophrenia, psychotic illness or episode (SPIE) had a diagnosis of alcohol dependence in 38.2% of cases, alcohol abuse in 16.6% of cases, drug dependence in 18.9% of cases, and drug abuse in 27.6% of cases. The most highly reported comorbid drug use disorder with lifetime SPIE was cannabis with a prevalence rate of 28.6% (McMillan, Enns, Cox, & Sareen, 2009).

Data from the NESARC also provided evidence of highly co-occurring SUDs and personality disorders. Results revealed that 28.6% of respondents with a current alcohol use disorder had at least one personality disorder, and an even higher rate of 47.7% of respondents with a current drug use disorder had at least one personality disorder. In addition, 16.4% of respondents with at least one personality disorder had a current alcohol use disorder, and 6.5% of respondents with at least one personality disorder had a current drug use disorder (Grant et al., 2006b). In summary, there are high comorbidity prevalence rates for SUDs and serious mental illnesses including psychotic and personality disorders.

**Major depressive disorder.** Epidemiological data from the U.S.A. and Australia, indicate that out of all mood disorders, major depressive disorder has the highest comorbidity amongst people with SUDs, alcohol and cannabis use disorders, with prevalence rates of 14.5%, 15.7% and 32.4% respectively (Grant et al., 2004; Teesson et al., 2010; Teesson et al., 2012). In addition, epidemiological surveys have found that SUDs
have earlier median ages of onset (age 20 years) than mood disorders (age 30 years) (Kessler et al., 2005a). Although in some cases depression subsequent to drug dependence may be due to the effects of intoxication or withdrawal, research has shown that in other cases, depression occurs well after successful treatment for substance dependence (Hasin & Grant, 2002). Further research is required in order to better understand the aetiology underlying this comorbidity.

A recent study which explored the relationship of various aspects of negative emotionality and impulsiveness on the comorbidity between depressive/anxiety disorder and alcohol dependence, found that some aetiological pathways between these two conditions are shared (all negative emotionality aspects measured, i.e., neuroticism, hopelessness, rumination, worry and anxiety sensitivity; as well as some aspects of impulsivity measured, i.e., disinhibition and boredom susceptibility), whilst other pathways exclusively occurred for each of the two disorders (namely high negative emotionality and low thrill and adventure seeking for depressive/anxiety disorders, and disinhibition for alcohol dependence) (Boschloo et al., 2013). These results provide some support for there being a number of shared vulnerability factors, as well as factors expressly related to one or the other condition. The specific pathways for each condition could explain why sometimes depressive/anxiety disorders precede alcohol dependence, whilst in other cases the onset of disorders is the opposite. One limitation of the Boschloo et al. (2013) study is that there were small numbers of research participants with depressive disorder only and with alcohol dependence only. The shared vulnerabilities factors model (i.e., the same factors independently contribute to the development of two co-occurring conditions) forms the basis for the further understanding of the SAD-SUD comorbidity being explored in this thesis. More detail about the theoretical bases for the studies in this thesis is set out in Chapter 3.

Treating comorbid depression and substance dependence disorders requires a careful assessment to ascertain the chronology of symptoms of both conditions. Where symptoms are comorbid, there is an increased risk of suicidality which also needs to be assessed. Treatment usually involves psychopharmacology for the substance use disorder and/or antidepressant medication, combined with relevant aspects of cognitive-behavioural
therapy, an evidence-based treatment for both conditions in their own right (Dongier, 2005).

**Anxiety disorders (excluding SAD).** Australian population surveys indicate that amongst those with alcohol dependence, generalised anxiety disorder (GAD) has the highest comorbidity rate of 23%, slightly higher than the PTSD rate of 22% (Teesson et al., 2010). Obsessive-compulsive disorder (OCD) is the most prevalent comorbid anxiety disorder with cannabis use disorders with a prevalence rate of 19.9% (Teesson et al., 2012). In the U.S.A., the NESARC survey demonstrated that specific phobia had the highest comorbidity prevalence rate with SUD (10.5%). However, rates of PTSD and OCD were not measured in the NESARC (2001-2002) survey (Grant et al., 2004). Amongst people with PTSD in “Wave 2” of the NESARC survey in the U.S.A. conducted in 2004-2005, 46.4% also had a SUD (Pietrzak, Goldstein, Southwick, & Grant, 2011). Population survey results indicate that age of onset of anxiety disorders (median age 11 years) usually precedes age of onset of SUDs (median age 20 years) (Kessler et al., 2005a). As with other comorbidities, co-occurring anxiety disorders with SUDs results in greater impairment and distress compared to having one of these disorders on its own.

**Social anxiety disorder (SAD).** In Australia, co-occurring SAD with an alcohol use disorder has the equal second highest (together with GAD) prevalence rate (11.2%) out of all the anxiety disorders. SAD’s comorbidity prevalence with alcohol dependence of 13.6% is even higher (Teesson et al., 2010). SAD is also the second highest co-occurring anxiety disorder with cannabis use disorder, with a prevalence rate of 14% (Teesson et al., 2012). In the U.S.A., the prevalence rate for people with alcohol dependence who also have SAD is slightly lower than in Australia at 10.9% (Schneier et al., 2010).

This thesis examines differences on three biopsychosocial factors, between people with SAD and people with co-occurring SAD and SUD, focusing on alcohol and cannabis use disorders due to their high prevalence and existing literature on research in this area. This comorbidity has been briefly discussed at the end of Chapter 1 in terms of prevalence rates for people with SAD who also have SUDs. Those rates are higher than the rates presented in this chapter which focus on prevalence rates of SAD in people with alcohol or cannabis use disorders. Nevertheless, for people with an alcohol use disorder or a cannabis use disorder, SAD still commonly co-occurs. Furthermore, prevalence rates of the SAD-
SUD comorbidity are even higher in clinical populations. For, example, within a clinical sample of 150 drug dependent patients who completed the French version of the Liebowitz Social Anxiety Scale, 62.6% were found to also have social anxiety (Zimmermann et al., 2004). In a larger clinical sample of alcohol-dependent and poly-drug dependent participants in Norway, social anxiety was assessed by clinical interview (the Composite International Diagnostic Interview and the Millon Clinical Multiaxial Inventory), and a comorbidity prevalence of 42% was found. The SAD comorbidity rate was higher for the poly-drug dependent patients, 51% compared to 34% for the alcohol dependent group (Bakken, Landheim, & Vaglum, 2005), suggesting that comorbid substance use disorders may be more important in their relationship with SAD compared to alcohol use disorders.

The literature cited in this section demonstrates that SUDs are highly comorbid with several different categories of psychological disorders and personality disorders. Most of the prevalence rates provided relate to population studies. Comorbidity rates amongst clinical samples are usually higher as demonstrated by the high comorbidity rates of SAD in clinical SUD samples described in the previous paragraph. These comorbidities, including the SAD-SUD comorbidity, generally result in poorer quality of life for affected individuals and are more difficult to treat, resulting in poorer treatment outcomes.

Summary

This chapter described the nature, various features and treatment of SUD, focusing on alcohol and cannabis use disorders. As outlined, SUD is a very complex mental disorder. The complexity is enhanced by the high comorbidity of SUD with many other psychological disorders including other SUDs, psychotic disorders, personality disorders, mood, and anxiety disorders. For comorbid conditions, careful assessments need to be conducted with regard to the chronology of all symptoms from SUDs and other disorders, to maximize optimal treatment outcomes. This thesis focuses on the comorbidity of SAD and SUD in order to increase understanding and inform treatment. It does so by considering the role of three biopsychosocial factors that influence this comorbidity, namely personality, alcohol expectancies and coping strategies. Features of the SAD-SUD comorbidity, descriptions of models of understanding, the proposed theoretical basis for this study, and literature reviews of the three factors being examined, follow in Chapter 3 of this thesis.
Chapter 3: Co-morbidity between Social Anxiety Disorder and Substance Use Disorder

Introduction

As described in Chapters 1 and 2 of this thesis, both social anxiety disorder (SAD) and substance use disorder (SUD) are highly prevalent and disabling disorders in their own right. They are also highly comorbid (Buckner et al., 2012b; Schneier et al., 2010). This comorbidity influences treatment outcomes and is associated with higher levels of disability for affected individuals (Buckner et al., 2013b). Some authors propose that there may be causal relationships between SAD and SUD (Buckner et al., 2013b; Fergusson, Boden, & Horwood, 2011). For example, an individual with SAD may be more likely to use substances problematically. Likewise, a person with a SUD may be more likely to develop SAD. Given that these relationships have been ascertained, irrespective of whether the temporal relationships of these comorbidities is complex, it is imperative to ascertain biopsychosocial factors that influence this comorbidity in order to better inform treatments.

With respect to SUD, the focus in this chapter is on alcohol use disorders (AUD) and cannabis use disorders (CUD), the substances investigated in their relationship with SAD. A vast majority of research about the SAD-SUD comorbidity has focused on alcohol. More recently research into cannabis as the substance co-occurring with SAD has developed. Therefore much of the content of this chapter is on the relationship between SAD and alcohol use. Where available, relevant literature about the comorbidity between SAD and cannabis use is included.

This chapter first describes features of the SAD-SUD comorbidity, followed by a literature review of current treatment and treatment limitations. Third, current models of understanding are described, including the theoretical basis for this thesis. Finally, this chapter reviews the literature relating to three biopsychosocial factors which influence the relationship between SAD and problematic substance use; personality (sensitivity to reward and punishment), alcohol expectancies, and coping strategies.
Prevalence

Epidemiological studies have shown that 48% of individuals with a lifetime diagnosis of SAD also meet criteria for a lifetime diagnosis of an alcohol use disorder (AUD) (Grant et al., 2005). Such surveys indicate a similar pattern of comorbidity rates for SAD and cannabis use disorders (CUD), with the lifetime rate of SAD in people with cannabis dependence reported to be 15.5% (Buckner et al., 2012b). Comorbidity rates amongst clinical populations are even higher. For example, 19.8% of outpatients with SAD have been found to also have comorbid CUD (Tepe, Dalrymple, & Zimmerman, 2012). Overall, comorbidity rates of SAD and AUD, as well as rates of SAD and CUD are high whether the data is collected from socially anxious individuals with a substance use disorder, or from problematic substance users with social anxiety.

Order of Onset and Course of the SAD-SUD Co-morbidity

SAD typically precedes AUD in comorbid cases (Morris et al., 2005), suggesting that SAD is a risk factor for AUD (Buckner, Timpano, Zvolensky, Sachs-Ericsson, & Schmidt, 2008b; Randall, Thomas, & Thevos, 2001). Longitudinal studies also support the contention that SAD is a risk factor for AUD (Buckner & Turner, 2009; Crum & Pratt, 2001). A recent epidemiological survey (Schneier et al., 2010) demonstrated that among people with co-morbid SAD and alcohol dependence, the mean age of onset of SAD (14.3 years) significantly preceded the mean age of onset of alcohol dependence (21.7 years). Similar findings have resulted from examining the order of onset of SAD and CUD. For instance, among people who have a comorbid cannabis use disorder with SAD, 81.5% reported that the onset of SAD preceded the onset of CUD, 15% reported that CUD occurred before SAD, and 3.5% reported that the two disorders occurred in the same year (Buckner et al., 2012b). Longitudinal studies confirm this order of onset for the two disorders in the majority of cases (Buckner et al., 2008a).

The course of the SAD-SUD comorbidity can be influenced by a number of factors. In the majority of cases where SAD symptoms appear earlier, subsequent problematic substance develops initially as a way of alleviating the anxiety. As a result, SUD may maintain or worsen SAD for a number of reasons (Morris et al., 2005). First, the fear or anxiety may not abate as the effect of the substance is to anaesthetise the feelings, thus preventing or retarding the learning or use of other more adaptive coping skills to deal with
the social anxiety. This may also affect the possibility of altering unproductive thoughts about the affected individual’s social adequacy and may perpetuate the view that they only coped with the social situation by using the substance. Second, the substance use may lead to additional anxiety symptoms for physiological reasons (withdrawal symptoms) and/or in relation to worries about the affected individual’s behaviour in social situations whilst intoxicated. This could in turn worsen that person’s social anxiety. Thus it is quite plausible that a vicious cycle may be established between SAD and SUD with both of them interacting, as well as mutually maintaining each other. Further research is needed to understand the possible reciprocity of symptoms of both SAD and SUD ideally by way of longitudinal studies.

**Gender Distribution**

Population surveys indicate that among people with alcohol dependence, the odds of having comorbid SAD are significantly higher for women compared to men, with similar gender difference patterns for people with alcohol abuse (Schneier et al., 2010). Amongst people with SAD, the odds of having CUD are significantly higher for men, while for people with CUD, population surveys indicate that the odds of having comorbid SAD are significantly less for men (Buckner et al., 2012b). Overall, findings indicate that women with either an AUD or CUD have a higher risk of having co-occurring SAD compared to males with either an AUD or CUD. However further research is needed particularly with clinical or treatment-seeking populations to further clarify gender differences within these comorbidities (SAD-AUD and SAD-CUD).

**Impairment and Quality of Life**

Population surveys indicate that people with co-occurring SAD and AUD are more likely to have attained less education and to have a lower income compared to people with only one of these conditions (Schneier et al., 2010). Individuals with SAD and a history of AUD have more severe symptoms of SAD, more health problems and more interpersonal stress (Buckner et al., 2008b). People with co-occurring SAD and CUD are likely to have lower incomes, to have attained less education, and to have poorer physical health. They are also more likely to use other illicit drugs (apart from cannabis) and less likely to be married, compared to people without this comorbidity (Buckner et al., 2012b). In addition,
people with co-occurring SAD and AUD, or SAD-CUD are more likely to have other psychological comorbidities, compared to people with only one of these conditions.

**Other Co-morbidities**

Aetiological research indicates that 97% of respondents with co-occurring alcohol dependence and SAD have at least one other psychological disorder. Excluding nicotine dependence disorder, the most prevalent comorbid disorder with co-occurring SAD and alcohol dependence is specific phobia (46%), followed by obsessive-compulsive personality disorder (38.9%) and major depressive disorder (35.4%) (Schneier et al., 2010). Data from the same population indicates that 99% of respondents with lifetime cannabis use disorder and SAD have at least one other psychiatric disorder (Buckner et al., 2012b), with alcohol dependence being the most prevalent (57%), followed by specific phobia (47%), and obsessive-compulsive personality disorder (44%). Comorbid major depressive disorder had a prevalence rate of 30.95% amongst this cohort. Although not all anxiety, mood, personality or substance use disorders were included in this survey, it is clear that major depressive disorder highly co-occurs with both the SAD-AUD and SAD-CUD comorbidities.

In addition to aetiological research, treatment research indicates that socially anxious substance abusers present with more psychopathology (e.g., mood disorders, other anxiety disorders) compared to non-socially anxious substance users (Bakken et al., 2005; Book et al., 2009). In summary, in both clinical and non-clinical populations, social anxiety and substance use disorders are highly comorbid with other psychological disorders.

**Treatment and Treatment Limitations**

Although efficacious treatments exist for people with SAD or SUD separately, there are treatment limitations for socially anxious individuals with a substance use problem, as for substance users who also have SAD. There is no established treatment for people suffering from both these disorders at the same time, and there is a paucity of research into concurrent treatment of SAD and SUD. As this thesis focuses on developing psychosocial treatment for the SAD-SUD comorbidity, this section is limited to describing research into
psychotherapeutic/psychosocial treatment. Pharmacological treatment is only referred to if it was relevant to any of these studies.

Early research compared cognitive-behavioural therapy (CBT) with Twelve Step Facilitation Therapy (TSFT) for people with alcohol use disorders (AUD) and co-occurring SAD (Thevos, Roberts, Thomas, & Randall, 2000). No significant differences were found with respect to treatment outcomes for men. However, women with SAD and AUD were found to be slower to relapse when treated with CBT compared to TSFT. One explanation for this gender difference may be the higher ratio of men compared to women who attend AA meetings. For socially anxious women this could make them uncomfortable about attendance. This poorer treatment outcome for socially anxious women in TSFT has been supported in more recent research in this area (Tonigan et al., 2010). Findings from the Tonigan study demonstrated a trend for socially anxious women to attend less AA meetings compared to non-socially anxious women, and a significantly lower likelihood of socially anxious women finding an AA sponsor (one of AA’s requirements).

Only two randomly-controlled trials (RCTs) have been conducted to investigate treatment outcomes using combined treatments for people with co-occurring SAD and SUD. Randall et al. (2001) randomised people with comorbid SAD and alcohol dependence (according to DSM-III-R criteria) into one of two treatment groups. Participants were required to have consumed alcohol in the 30 days prior to screening for the trial. One group received manualised CBT (delivered individually) for alcohol dependence only, whilst the second group received manualised CBT (delivered individually) for both SAD and alcohol dependence. Treatment sessions for the dual treatment group were 30 minutes longer than treatment sessions for the alcohol only group. At post treatment (12 weeks) and three-month follow-up, the group that received the dual CBT treatment had worse outcomes on three out of four drinking measures, compared to the group that received CBT treatment for alcohol dependence only,. There were no significant differences between the groups on social anxiety indices, although overall both groups improved on drinking and social anxiety measures from baseline to end-of-treatment. (Randall et al., 2001).

In the second RCT, Schade et al. (2005) randomly assigned patients with alcohol dependence (according to DSM-IV criteria) and comorbid SAD or agoraphobia (with or
without panic attacks), to one of two treatment groups. The “alcohol treatment group” received a relapse-prevention program, and the “alcohol and anxiety treatment group” received the relapse-prevention program plus CBT for anxiety as well as optional pharmacotherapy consisting of an SSRI. The majority of patients in the combined treatment group refused the offer of the SSRI. Both treatments were conducted over a 32 week period. All patients had been abstinent for 6 weeks prior to commencement of treatment. The primary outcome measure was the percentage of patients who relapsed during the treatment period. Although the combined treatment reduced anxiety symptoms, there was no significant effect on alcohol relapse rates.

Despite differences in methodology in both RCTs, the findings indicate that combining treatments (sequentially or in parallel) for alcohol use and SAD does not improve, and may even worsen drinking outcomes, whilst such treatment may improve anxiety symptoms. More research needs to be done in this area and currently there is a RCT in progress investigating whether the provision of CBT for socially anxious substance abusers prior to their entry into a residential rehabilitation facility improves retention rates (Staiger et al., 2014).

In a relatively recent pilot study, patients with concurrent SAD and SUDs were treated with manualised group CBT treatment for SAD which specifically addressed the link between social anxiety and substance use (Courbasson & Nishikawa, 2010). The results demonstrated a significant reduction in social anxiety symptoms as well as significantly reduced negative affect across treatment. There was no significant change in level of positive alcohol expectancies and level of substance use was not measured across treatment. The results from this study indicate that psychosocial therapy that integrates treatment for problematic substance use and SAD may result in better treatment outcomes for both disorders. One advantage of integrated treatment could be the reduction of cognitive demands on patients being treated for both SAD and SUD compared to separate treatments operated in parallel or sequentially. At this time, no research has been conducted to investigate an integrated approach to treat the SAD-SUD comorbidity although one trial is in the process of being developed (Stapinski et al., 2014).

It may be that an integrated approach rather than a sequential approach to treating these co-occurring disorders could produce more beneficial outcomes for affected
individuals. It is hoped that this thesis will contribute to the development of an integrated treatment approach by examining the roles of important biological, psychological and social factors (personality, alcohol expectancies and coping strategies) in the SAD-SUD relationship. In summary, there has been limited research into treatments for comorbid SAD and SUD individuals at the present time. Treatment needs to be informed by understanding. The following section describes the current understanding of the SAD-SUD comorbidity based on a review of current theoretical models.

**The Nature of the Relationship between SAD and SUD**

Dating back 30 years, but more prolifically in the last 20 years, significant research has been generated in attempting to explain the relationship between social anxiety and alcohol use in both clinical and non-clinical populations. One reason for the extent of this research is that much about this relationship remains unclear due to inconsistent findings, despite evidence as to high comorbidity rates amongst surveyed or studied populations.

Studies investigating the relationship between social anxiety and cannabis use are more recent and fewer in number compared to research examining social anxiety and alcohol. However, this appears to be a growing area of research. Contradictory findings have also emerged from examination of the relationship between social anxiety and cannabis use. Conflicting results about the relationship between social anxiety and alcohol or cannabis-related variables support the need for further research cognizant of the complex interplay of various factors.

This research study aims to increase understanding of the relationship between social anxiety and substance use disorders by examining its association with three biopsychosocial factors: personality (drive sensitivity), alcohol expectancies, and coping strategies. Ultimately, this improved understanding will better inform treatment of SAD with and without SUD. As the focus of this research is to identify important factors to target in the prevention and treatment of co-occurring social anxiety and problematic substance use, this thesis does not speak directly to causation with respect to the SAD-SUD comorbidity. However, some of the theories that discuss causation may still be able to identify significant factors that are important in treatment.

From past research, three possible pathways emerge when considering the SAD-SUD relationship: (a) the “self-medication” model, which proposes people drink alcohol or
use drugs to alleviate social anxiety (SAD precedes SUD); (b) the “precipitation” model, that is, drinking alcohol or using drugs leads to social anxiety (SUD precedes SAD); and (c) the “shared vulnerabilities” model, whereby specific factors or vulnerabilities which are common to both disorders (SAD and SUD) influence the comorbidity.

**Self-medication model.** The self-medication model is the predominant perspective used currently to explain the relationship between SAD and problematic substance use. This theory relies on the knowledge of the anxiolytic properties of alcohol and other substances and implies that people drink alcohol or use drugs including cannabis, to relieve anxiety caused by their fear of being negatively evaluated in social situations.

**History and development of the self-medication model.** The self-medication model evolved from tension-reduction theory which was developed by Conger (1956). Conger conducted animal studies and demonstrated that the release of stress-related hormones was significantly lower in animals which had consumed alcohol and were involved in stress-inducing tasks. However, “tension” was broadly defined and did not take into account individual differences, leading to contradictory findings. This criticism led to the development of the “stress response dampening model” (Sher & Levenson, 1982), which expanded on the previous model by allowing for individual differences in terms of anxiety sensitivity. It proposed that people with social anxiety are more sensitive than others to the “stress response dampening effects” of alcohol consumed both before and during social interactions and social performance situations (Morris et al., 2005). However, this theory remained too general as it only took into account one individual difference factor, and could not adequately explain other inconsistencies in this research area.

The self-medication hypothesis further advanced this line of theory in relation to the SAD-SUD comorbidity. It was first proposed by Khantzian (1985) and assumes the following: (a) that the (social) anxiety develops before the problematic substance use; (b) that the substance use relieves the anxiety symptoms; and (c) that this relief leads to continued problematic substance use in social (anxiety provoking) situations (Chutuape & de Wit, 1995).

Assumption (a) is strongly supported with the majority of findings demonstrating that SAD precedes SUD (Buckner et al., 2008a). However research findings with respect to assumption (b) are less clear. Carrigan and Randall (2003) found that socially anxious
people do use alcohol to alleviate anxiety; however, there was less certainty as to whether alcohol actually reduces social anxiety. The authors also pointed out that most of the studies they reviewed excluded anyone with a substance use disorder, thereby weakening the conclusions that could be drawn about the comorbid condition. Furthermore, a relatively recent large-scale epidemiological survey, concluded that reports of drinking to ease anxiety (sub-clinical symptoms as well as diagnosed anxiety disorders), was related to the subsequent occurrence of alcohol dependence (Crum et al., 2013). The results of this study combined with the findings of Carrigan and Randall (2003) suggest that it may be perceived relief, rather than actual (physiological) changes as a result of substance use, that reduce anxiety.

The findings of both sets of researchers (Carrigan & Randall, 2003; Crum et al., 2013) also support assumption (c). The lack of clarity with respect to assumption (b) has led to a significant amount of research in this area. Various research methods have been utilised to explore the relationship between social anxiety and substance use (predominantly alcohol use) based on the self-medication model. A literature review of these studies follows.

Research methods used to examine the relationship between social anxiety and alcohol use. Three published experimental studies to date have directly investigated the effects of alcohol on socially anxious individuals, using a placebo versus alcohol group design and a speech challenge as the social anxiety provoking situation.

Two early experimental studies (Himle et al., 1999; Naftolowitz, Vaughn, Ranc, & Tancer, 1994) investigated the effects of alcohol and placebo on socially anxious individuals according to DSM-III-R and DSM-III criteria respectively. Neither of these studies supported the hypothesis that alcohol consumption reduces social anxiety. However both these studies had several limitations including the amount of alcohol consumed (.03% BAC) which may have been too low to produce anxiolytic effects, small sample sizes (too small to detect medium effect sizes), a speech challenge not producing sufficient anxiety, and 25% of participants being on anxiolytic medication which may have reduced the effect of alcohol for those individuals (Tran & Smith, 2008).

The third study conducted by Abrams and colleagues (Abrams, Kushner, Medina, & Voight, 2001) addressed these limitations and also improved the design with three
groups: alcohol - expected and received alcohol; placebo – expected alcohol but received placebo (disguised as alcohol with a few alcohol drops on the surface of the drink); and control – expected and received non-alcoholic beverage. In addition, those receiving alcohol consumed .05% BAC, each group had 20 or 21 participants, a more anxiety provoking speech challenge was conducted (participants had mean heart rates of 119 beats per minute compared to 87 beats per minute for participants in the Himle study), and potential participants were excluded if they were taking psychiatric medication. Inclusion criteria were meeting all DSM-IV criteria for current SAD, and being a social drinker (non-drinkers or light drinkers were excluded as were people with an AUD). Participants completed a speech task both before and after drinking (alcohol or non-alcoholic beverages) at an interval. Findings demonstrated that both the pharmacologic and expectancy effects of alcohol significantly reduced performance anxiety. However it is of note that participants were recruited from the community by way of advertisements seeking people with public-speaking (performance) fears and who were light social drinkers. Furthermore, only about half of eligible recruited community members participated in the study (N=61). Thus only people with less severe SAD appear to have been represented in this study sample. Finally, whilst there was a significant reduction in anxiety between the alcohol and control groups, the reduction in anxiety between the alcohol and placebo groups approached significance, and there was no significant difference in reduction of anxiety between the placebo and control group. In summary, these experimental studies do not conclusively support the contention that the effects of alcohol directly reduce social anxiety.

Another research method has involved investigating the comorbidity via subcategories of the SAD construct. Researchers in one study investigated differences between socially anxious individuals with public speaking fears only, and socially anxious persons with this type of fear as well as other social fears (Kessler, Stein, & Berglund, 1998). The latter group, being analogous to the “generalised” subtype of SAD, had significantly higher rates of comorbid AUD compared to the group with speaking fears only. Researchers in another study investigated the behaviour of drinking alcohol to cope with anxiety in social situations, and found that a group of socially anxious individuals were more likely to drink to cope in social interaction situations, than in social performance
situations (Thomas, Randall, & Carrigan, 2003). Notably, the matched control group in this study was also more likely to drink in social interaction rather than performance situations. However, when the two groups were compared on overall drinking to cope, the socially anxious group was more likely to drink to cope in a higher number of both types of social situations compared to the control group. These two studies provide some support for the view that certain social anxiety subtypes (especially generalised SAD) are at increased risk for developing a comorbid AUD. The latter study (Thomas et al., 2003) also supports the continuum theory with respect to the severity of SAD, as the non-socially anxious group were comprised of participants whose scores on the two SAD scales were below the empirical cut-off scores for SAD and all participants (both groups) were drinkers.

There is also an area of research showing that the strength of the relationship between social anxiety and alcohol use is moderated by severity of the other condition (Tran & Smith, 2008). Two longitudinal studies provide support for a positive association between subclinical levels of social anxiety and AUD (Crum & Pratt, 2001; Merikangas, Avenevoli, Acharyya, Zhang, & Angst, 2002). Crum and Pratt (2001) conducted a 12.6 year follow-up among three large groups of adult survey respondents: current or lifetime history of SAD, subclinical SAD (social fears without avoidance or impairment), and no SAD. They found that the subclinical group was more than twice as likely to develop heavy drinking (2.41 odds ratio) or an AUD (2.30 odds ratio), compared to the group without SAD. Furthermore, there was no greater risk of heavy drinking in the SAD group (risk of AUD in the SAD group was not estimated as there were no cases of AUD) compared to the group without SAD. Merikangas et al. (2002) found similar results in their study with a large group of 18-19 year olds who were subsequently interviewed on five occasions spanning a 15-year period. Only the sub-threshold group (one or more SAD symptoms plus avoidance) had a significantly higher risk of developing an AUD (3.2 odds ratio) compared to the “symptoms” group (one or more SAD symptoms only) and the control group (no SAD symptoms). There was no significant risk of developing an AUD for the SAD group (two or more SAD symptoms plus avoidance plus significant subjective distress). Results support the view that people with severe SAD are likely to avoid most social situations and therefore do not need to drink to cope with their anxiety in social situations, whilst people with less severe symptoms don’t avoid social situations but use
alcohol if available in those situations to cope with their social anxiety. However, the findings from these two studies are inconsistent with those of both Kessler et al. (1998) and Thomas et al. (2003) which indicated that the generalised subtype of social anxiety increases the risk for alcohol use problems.

With respect to severity of alcohol use, two studies provide support for the contention that people with SAD have higher rates of alcohol dependence compared to those without SAD despite there being little difference in frequency or quantity of alcohol consumption (Ham, Hope, White, & Rivers, 2002; Thomas, Thevos, & Randall, 1999). Thomas et al. (1999) recruited treatment-seeking alcoholics from Project Match, a large multi-site study investigating treatments for alcoholics. Of the entire group, 23% met DSM-III criteria for lifetime SAD. These people comprised one group and a comparison group was created from other Project Match participants, with equal numbers and matched for age, gender and treatment variables. The group with SAD were significantly higher on two measures of alcohol dependence but had similar results to the comparison group on measures of frequency and quantity of alcohol use. However, the group with SAD also had significantly higher levels of depression and psychiatric problems, both of which could have contributed to the higher levels of alcohol dependence.

Ham et al. (2002) compared treatment seeking socially anxious individuals and dysthymics with normal controls and found no significant difference in drinking levels between these groups. This study adds support to the contention that socially anxious individuals have higher levels of alcohol dependence rather than level of alcohol use. In both studies the social anxiety groups also had significantly higher positive alcohol expectancies compared to the other groups, which supports the claim that socially anxious individuals with a comorbid AUD are more psychologically dependent on alcohol compared to alcoholics without SAD. Overall, there are difficulties in reconciling the findings of studies showing on the one hand that people with subclinical symptoms of social anxiety are more likely to have an AUD (compared to individuals with a full diagnosis of SAD), with findings from other studies indicating that people with SAD have higher rates and more severe symptoms of alcohol dependence compared to people with alcohol dependence who don’t have SAD. These inconsistent results add weight to the
contention that the SAD-AUD comorbidity is complex and further research is needed in this area to amplify our understanding of this relationship.

Other research methods that have been used to explore the relationship between SAD and AUD include investigating whether there is a genetic predisposition to both SAD and AUD, and examining the neurobiology of the co-morbidity (Tran & Smith, 2008). Both twin and family studies have been conducted to investigate the genetic influence in the relationship between SAD and AUD. Twin studies that have examined the comorbidity of SAD or SAD symptoms and AUD, provide some support for a shared genetic vulnerability (Knopik et al., 2004; Nelson et al., 2000). Nelson et al. (2000) studied the relationship between SAD, AUD and major depressive disorder with 2431 pairs of female adolescent twins. They found that the three disorders shared a common additive genetic risk factor to varying degrees, but AUD also had a disorder-specific genetic component. Knopik et al. (2004) examined the genetic effects on alcohol dependence risk in a study with 2723 pairs of monozygotic and dizygotic twins and found that social anxiety problems (determined by a non-diagnostic measure) played only a minor role in mediating the genetic risk of alcohol dependence. These studies indicate that genetics may play a role in the relationship between social anxiety and alcohol use. More research is required to determine the extent of the role genetics may contribute.

A family study (Merikangas et al., 1998) investigated patterns of familial aggregation in the comorbidity of alcoholism and anxiety disorders (panic disorder and SAD) in the relatives of 165 affected probands (individuals with one of these disorders) selected from treatment settings or at random from the community. Findings indicated that there was a significant risk for SAD among relatives of probands with SAD, and there was a significant odds ratio (2.4) for alcoholism among socially anxious relatives of socially anxious probands. Findings also suggested that these two disorders did not share common genetic risk factors and supported findings of other clinical studies indicating that SAD typically precedes AUD. Overall there are mixed findings as to whether there is a shared genetic component to both SAD and AUD.

Researchers who have examined the neurobiology of co-occurring SAD and AUD have found that serotonin, gamma amino butyric acid (GABA) and dopamine are all potential contributors to this comorbidity. Tran et al. (2008) describes studies that suggest
that serotonin dysfunction is involved in both SAD and AUD, as well as studies that
demonstrate that alcohol consumption enhances both GABA and dopamine activity,
resulting in reduced anxiety/social anxiety levels. Thus it appears that these three
neurotransmitter systems are involved in the SAD-AUD comorbidity although further
research is needed to understand the roles they play. However, as such factors have limited
relevance to psychological treatments relative to the cognitive and behavioural variables of
interest in this thesis, they are not mentioned further.

The final and currently predominant research method used to investigate the SAD-
AUD comorbidity, focuses on the contributions of other relevant variables to the
relationship including for example, their moderating and/or mediating roles. This thesis
uses this last approach and focuses on the roles of such three variables within a
biopsychosocial framework: personality in terms of drive sensitivity (a biological factor),
alcohol expectancies (a cognitive factor) and coping strategies (a cognitive and behavioural
factor). This thesis considers the role these three variables play in the relationship between
social anxiety and alcohol use, and between social anxiety and cannabis use.

To a large extent this research method has evolved as a result of inconsistent
findings as to the existence of a significant relationship between social anxiety and alcohol-
related variables, as well as contradictory findings as to the direction of the relationship.
Possible explanations for these inconsistencies include limitations due to self-reporting by
participants in some study designs (e.g., due to recall biases or other memory problems);
use of different scales, subscales, and subtypes of social anxiety; different measures of
drinking variables (e.g., measures of consumption versus measures of negative drinking
consequences); different study designs; and differences in statistical analyses applied. For
instance, in one study, both social avoidance and distress (two indicators of social anxiety)
were negatively correlated to drinking frequency, but fear of negative evaluation (another
social anxiety construct) was positively related to drinking problems (Stewart, Morris,
Mellings, & Komar, 2006). These findings support the idea that people who avoid social
situations drink less, but socially anxious people that attend social functions drink to reduce
their fear of being negatively evaluated and may thus develop drinking problems.

A pattern of findings has emerged indicating a positive association between social
anxiety and alcohol problems/alcohol use disorders (AUDs) in both clinical (Grant et al.,
Social Anxiety and Substance Use (Grant et al., 2005; Ham et al., 2002; Thomas et al., 1999) and non-clinical samples (Buckner & Heimberg, 2010; Buckner, Schmidt, & Eggleston, 2006b; Gilles, Turk, & Fresco, 2006; Lewis & O'Neill, 2000; Lewis et al., 2008; Stewart et al., 2006). There have been two studies in which no significant relationship between social anxiety and alcohol-related problems was demonstrated (Ham, Bonin, & Hope, 2007; Ham & Hope, 2005).

The latter of these two studies can be distinguished from others as participants were mostly male students required to attend an alcohol skills program after receiving their first warning in relation to alcohol use on campus.

Data about the relationship between social anxiety and alcohol use (volume/frequency of drinking) is less clear. Most research in this area has utilised non-clinical samples with the majority of these comprised of university students (Battista, Stewart, & Ham, 2010). Schry and White (2013) recently conducted a meta-analysis to examine the relationship between social anxiety and alcohol-related variables in student populations, and found that social anxiety was negatively associated with alcohol use. However, in their review of the SAD-SUD comorbidity, Buckner et al. (2013b) concluded there was no significant relationship between SAD and alcohol use from findings in the majority of studies. Overall, findings with regard to the relationship between SAD and alcohol use in non-clinical studies appear to be mixed, with some studies finding a negative relationship (Eggleston, Woolaway-Bickel, & Schmidt, 2004; Ham et al., 2007; Ham & Hope, 2005; Myers, Aarons, Tomlinson, & Stein, 2003; Stewart et al., 2006), others finding no relationship (Anderson, Tomlinson, Robinson, & Brown, 2011; Buckner et al., 2006b; Gilles et al., 2006; Lewis et al., 2008), and one study a positive relationship (Neighbors et al., 2007). However, the participants recruited by Neighbors and his colleagues were undergraduate students living in residence halls, where a higher level of drinking has been found to occur compared to first year students living at home (O'Hare, 1990).

Limited research has specifically examined alcohol use (volume and frequency) in clinical samples. Two studies (Ham et al., 2002; Thomas et al., 1999) demonstrated no significant relationship between SAD and alcohol use. A third study (Holle, Heimberg, Sweet, & Holt, 1995), involving socially anxious treatment-seekers being compared to a group of community controls, revealed a negative relationship between SAD and alcohol
use. Thus mixed findings have also resulted from clinical research investigating the relationship between SAD and alcohol use.

Despite inconsistencies in defining the nature and direction of the relationship between SAD and alcohol in the literature, one can conclude that alcohol-related problems/AUDs are positively associated with SAD, whereas the nature of the relationship between SAD and alcohol use (levels) is less apparent. This lack of clarity, combined with inconsistent findings found in the SAD-AUD relationship by use of other research methods described in this section, makes it difficult to exclusively rely on the self-medication hypothesis as the theoretical basis for this comorbidity. In addition, even if SAD precedes AUD (as implied by this theoretical model), it does not take into account treatment complications that can arise due to this comorbidity, nor the mechanisms by which it can be maintained or worsened, as described earlier in this chapter. Thus it appears that a more comprehensive approach is required to examine the SAD-SUD relationship, for example, the simultaneous consideration of the roles of three important variables such as personality factors, alcohol expectancies and coping strategies, within a biopsychosocial framework. This is the approach being taken in this thesis.

**Research methods used to investigate social anxiety and cannabis use.** Some of the same research methods have been used to investigate the relationship between SAD and cannabis use disorders (CUD) as described above for the SAD-AUD comorbidity. Investigations into the SAD-CUD comorbidity have also predominantly relied on the self-medication model.

As with subclinical levels of social anxiety and AUD, the same longitudinal study (Merikangas et al., 2002) with the group of 18-19 year olds interviewed over a 15-year period, provides some support for a positive association between subclinical levels of social anxiety and CUD. Merikangas and her colleagues found that subthreshold socially anxious individuals had a higher risk of developing a CUD (2.0 odds ratio) compared to the SAD group, the group with fewer social anxiety symptoms, and the control group. However, the association between subthreshold SAD and CUD was not significant, as was the association between subthreshold SAD and AUD. As with the findings for the SAD-AUD relationship, this result provides some support for the view that the more severe socially anxious
individuals completely avoid social situations, whilst the less severely affected may use cannabis to cope with their anxiety in social situations.

The effects of particular cannabis constituents such as cannabidiol (CBD) and tetrahydrocannabinol (THC) on anxiety and social anxiety have been investigated in experimental research. As with the alcohol literature, studies that investigate the effects of cannabis on social anxiety are based on the premise that the onset of SAD precedes the onset of CUD. Past research has shown that CBD, a key non-psychotomimetic ingredient of the cannabis plant, has anxiolytic effects (Bergamaschi et al., 2011). In a double-blind randomized design, Bergamaschi and colleagues compared the effects of a public speaking test on three groups. Two groups were comprised of individuals who fulfilled DSM-IV criteria for SAD. One of these groups received CBD, the other received a placebo. The third group was comprised of healthy controls. The SAD-Placebo group was significantly more anxious with greater cognitive impairment, discomfort and alert compared to the healthy controls during the test. The SAD-CBD group had similar results to the healthy controls on these measures during the test. These findings demonstrate the anxiolytic effect of CBD on socially anxious individuals.

Although some cannabis users have reported a reduction in anxiety, others, especially those who are not regular users, have reported acute anxiety occurring after taking cannabis. Studies have shown that THC, the other key ingredient of cannabis, is also anxiolytic when taken in low doses, but in high doses, can cause intense fear and anxiety and even panic attacks in a small minority of people (Crippa et al., 2009). Thus cannabis can be both anxiolytic, which supports the self-medication model of understanding the SAD-CUD relationship, and anxiogenic, which supports the “precipitation” model (CUD precedes SAD) of understanding of the relationship.

Two lab-based studies have looked at cannabis cravings among regular cannabis users, some of whom also had social anxiety. In the earlier of these studies (Buckner, Silgado, & Schmidt, 2011), participants were randomly assigned into one of two groups: speech task or reading task. The socially anxious individuals during the speech task reported significantly greater cravings compared to socially anxious individuals during the reading task, suggesting that social anxiety precedes cannabis-related behaviours. In the more recent study (Buckner, Ecker, & Vinci, 2013a), participants were randomly assigned
to a social interaction activity or a reading activity. Cannabis craving increased from before the social interaction task to during it, but there was no such increase of craving for the reading task group. However, there were fewer socially anxious participants in this study (13.4% of the total sample). Nevertheless, these studies support the self-medication hypothesis in relation to increased cannabis cravings amongst regular cannabis users during potentially anxiety-provoking social situations.

As with research investigating the SAD-AUD relationship, due to inconsistent findings in the literature, the role of mediating or moderating variables in the SAD-CUD relationship has also been examined (Buckner et al., 2013b; Thompson, Goldsmith, & Tran, 2011). Similar to the SAD-AUD literature, there appears to be a fairly robust relationship between SAD and cannabis-related problems/CUD (Buckner, Bonn-Miller, Zvolensky, & Schmidt, 2007; Buckner & Schmidt, 2008), whilst the literature pertaining to the relationship between SAD and cannabis use (frequency) is more mixed, at least amongst student populations. Some studies report no relationship between SAD and cannabis use (Buckner et al., 2007; Buckner & Schmidt, 2008; Myers et al., 2003) whilst others have found a positive relationship (Oyefeso, 1991). However, the Oyefeso study can be distinguished from the others as participants were all male undergraduate students compared to the other studies where participants were predominantly female.

Research investigating the SAD-CUD comorbidity is far less developed compared to the SAD-AUD comorbidity. Nevertheless, the literature in this area also demonstrates conflicting findings with respect to the relationship between SAD and cannabis use. Clearly, the natures of both the SAD-AUD and SAD-CUD comorbidities have not as yet been fully explained by the existing literature. Exclusive reliance upon the self-medication model does not elucidate the inconsistent findings in these research areas. In order to more comprehensively understand the nature of these comorbidities, the use of other models as a theoretical basis may advance knowledge in this field.

Precipitation model. The second proposed pathway, the “precipitation” model (SUD before SAD), suggests that SAD develops after problematic substance use. This is based on the premise that anxiety results as a physiological consequence of chronic substance use and social withdrawal (Thompson et al., 2011). In addition, intoxication with particular drugs including cannabis can be anxiogenic (Nunes & Blanco, 2009).
Although this order of onset of co-occurring SAD and SUD occurs in a minority of people, the precipitation model may be important for this group. One longitudinal study (Merikangas et al., 2002) compared order of onset of SAD or varying degrees of SAD symptoms with both alcohol and cannabis abuse. Findings demonstrated that one in five people with co-occurring SAD and a cannabis use disorder (CUD) develop a CUD prior to SAD, whilst one in eight people with co-occurring SAD and an alcohol use disorder (AUD) develop an AUD prior to SAD. Furthermore, a systematic review of studies (dating back to 1970) dealing with comorbid substance use and other psychological disorders, ascertained that for people with an AUD, substance-induced conditions represented 25% of social phobias (Schuckit, 2006).

Despite these statistics, it appears that much less research has evolved based on the precipitation model with respect to these two SAD-SUD comorbidities. Using data from a large-scale longitudinally epidemiological survey (the NESARC), Robinson, Sareen, Cox, and Bolton (2011) tested the precipitation model by examining whether self-medication (with alcohol or other drugs) to relieve subthreshold levels of anxiety, fear and/or avoidance, led to the development of comorbid anxiety disorders. They found significant odds ratios for the development of subsequent SAD after self-medication with both alcohol and other drugs. Robinson and her colleagues offer two possible explanations for this result; first, that some people self-medicate due to subthreshold levels of SAD which then escalates to SAD; and second, that social unacceptability of substance use may lead to avoidance of social situations which then escalates to SAD. Furthermore, SAD that is secondary to alcohol dependence (its symptoms are less severe) has been found to be an independent condition in alcohol-dependent inpatients which does not completely remit after cessation of drinking (Olgiati et al., 2007).

The precipitation model seems less applicable to the SAD-SUD comorbidity compared to the self-medication model based on research findings to date. However, as has been outlined above, further investigation is required to better understand the nature of this comorbidity and this includes further research into substance-induced SAD. It is beyond the scope of this thesis to consider the precipitation pathway in more detail.

**Shared vulnerabilities model.** The third pathway which could explain the SAD-SUD comorbidity is the “shared vulnerabilities” model. This model proposes that
individual differences or vulnerability factors contribute to the development of, or increase the risk of co-occurring disorders (Crippa et al., 2009). This model does not specifically speak to the timing of onset of the disorders but focuses on the genetic or environmental factors in common to both of them. This pathway suggests that individual difference characteristics in the form of biological/genetic, psychological and social vulnerabilities, predispose people to develop both SAD and SUD. The biopsychosocial model comes to the fore here as by examining biopsychosocial individual difference factors in people with SAD only, and in people with the SAD-SUD comorbidity, differences detected between the groups could inform treatment especially for the comorbid group which is more difficult to treat.

Most of the literature relating to co-occurring SAD and SUD (alcohol or cannabis use disorders) is based on the self-medication model. A lesser amount is based on the precipitation model which has been outlined in the preceding subsection. There does not appear to be any literature directly investigating the shared vulnerability model as a theoretical basis for the SAD-SUD comorbidity, although there is some indirect evidence for a shared genetic vulnerability via twin studies mentioned earlier in this chapter (Knopik et al., 2004; Nelson et al., 2000). This is despite the recent preponderance of studies which have looked at individual difference variables to try to explain inconsistencies in the SAD-SUD relationship.

One recent study that focused on the shared vulnerability model, looked more generally at comorbid anxiety/depressive disorders co-occurring with problematic opioid use, using epidemiological data (Martins et al., 2012). Martins and her colleagues found evidence of all three pathways (self-medication, precipitation and shared vulnerabilities). They concluded that the shared vulnerability model could not be ruled out as there was a similar strength of degree of associations in both directions between problematic opioid use and mood/anxiety disorders. Based on these findings, the shared vulnerability model may be more applicable to the SAD-CUD relationship (compared to the SAD-AUD relationship), where findings have indicated a closer degree of strength between the relationship in both directions (in terms of order of onset of the two disorders). In any event, other studies have supported the use of the shared vulnerability model in cases where
the temporal association of the comorbid conditions has not been researched, for example, in comorbid posttraumatic stress disorder (PTSD) and SAD.

Collimore, Carleton, Hofmann, and Asmundson (2010) reviewed literature pertaining to biological and psychological vulnerabilities in co-occurring posttraumatic stress disorder (PTSD) and SAD. One of the psychological vulnerabilities examined was fear of negative evaluation. They also explored the association of both disorders with other factors including depression (another important disorder in terms of its high comorbidity with both SAD and SUD). They concluded that there is evidence demonstrating interplay between genetic, environmental and psychological vulnerability factors in the PTSD-SAD comorbidity although further research is needed to delineate this relationship. A second relatively recent study has focused on the shared vulnerability model in relation to comorbidities where one of the disorders was SAD. Reichborn-Kjennerud et al. (2007) examined co-occurring SAD and avoidant personality disorder (APD) in young adult female twin pairs using the shared vulnerability model. They concluded that there were shared genetic vulnerabilities but the environmental factors were unique to each disorder.

One line of research that has indirectly relied upon the shared vulnerabilities model is that involving the classification of social anxiety into subtypes in an attempt to explain a subset of socially anxious individuals who display atypical behaviours and self-regulation strategies. These characteristics including risk taking, impulsive and approach behaviours, one example of which is substance misuse (Kashdan, McKnight, Richey, & Hofmann, 2009). Thus, a subset of socially anxious individuals exists which has the same core features of social anxiety but also displays some differences.

Researchers have also examined comorbidities using the shared vulnerabilities model where one of the disorders was a SUD. Alloy et al. (2009) tested whether personality vulnerabilities are shared in bipolar disorders and co-occurring substance use problems by comparing a clinical group (on the bipolar spectrum) with a group of control participants in a longitudinal study. They concluded that high BAS (behavioural approach system) sensitivity (also termed “reward sensitivity” which is an aspect of a broad personality theory, “RST”, based on the biological bases of learning and motivation- to be delineated further later in this chapter) and impulsiveness may represent shared vulnerabilities in both disorders. This thesis explores drive sensitivity (sensitivity to reward
and punishment) as one key individual difference factor which may play a role in the SAD-SUD comorbidity. Drive sensitivity has been shown to be an important factor in both social anxiety (e.g., Kashdan & Roberts, 2006) and substance use (e.g., Franken & Muris, 2006).

Hruska and Delahanty (2014) reviewed the literature pertaining to potential common biological mechanisms that may underlie co-occurring PTSD-SUD. They could not definitively determine which of the three pathways best describe the role the biological mechanisms play in the development of this comorbidity, but did conclude that the self-medication model seemed to apply to all these mechanisms suggesting this model may be the best available at the present time.

In summary, it is possible that additional factors which have been shown to play a role in the SAD-SUD relationship, might be common vulnerabilities to both disorders, and/or play a role in the self-medication or precipitation models previously described. Alternatively the shared vulnerability model may be viewed as a mega-theory which includes or subsumes both other pathways to explain the SAD-SUD comorbidity. In any event, irrespective of the pathway which leads to the onset of the SAD-SUD comorbidity, once present, both disorders may maintain or exacerbate each other creating a vicious cycle and making it difficult to treat one or both of them (Stewart & Conrod, 2008). Thus it is important to understand the covariates of SAD, SUD and their shared relationships in order to identify factors that need to be targeted in comorbidity presentations. The following sections describe three factors, personality (drive sensitivity), alcohol expectancies, and coping strategies, and combinations of these individual factors, that are important in the relationship between SAD and SUD, and which form the basis for furthering our understanding of the SAD-SUD comorbidity in this study.

**Personality**

Personality is a broad construct comprised of both biological and environmental influences. One major biologically based theory of personality attempts to explain the influence of two drives, sensitivity to reward and sensitivity to punishment. This theory, “Reinforcement Sensitivity Theory” (R.S.T.) was developed by Gray (1970) and has been applied to research investigating both substance use and anxiety. Research findings consistently demonstrate positive associations between social anxiety and punishment.
sensitivity, as well as positive associations between substance use and reward sensitivity. Thus RST is a highly relevant theory of personality that can contribute to understanding the SAD-SUD comorbidity. This section of Chapter 3 outlines the development and features of RST, explains its significance to the SAD-SUD comorbidity, and provides a literature review of research that has examined RST in relation to SAD, SUD and the SAD-SUD comorbidity.

**Reinforcement sensitivity theory (RST).** Gray (1970) developed RST as a response to concerns with some aspects of the prevailing biological theory of personality at that time, that is, Eysenck’s (1967) arousal/activation theory of personality. Eysenck’s theory was comprised of two dimensions, introversion-extroversion and neuroticism. This theory emphasised the difference between introversion and extroversion in terms of ease of conditioning, with introverts who are highly arousable being easy to condition compared to extroverts (low arousal, more difficult to condition). As highly neurotic people had been observed to be also introverted, and behaviour therapy (based on conditioning principles) was effective with a number of neurotic disorders, the theory was widely accepted (Corr, 2009). With further research, problems became apparent, for example, the finding that at high levels of stimulation, introverts were worse than extroverts at conditioning. Gray dismantled Eysenck’s theory, in particular the idea of one system of drive arousal. He proposed that there are two innate drive systems, one that controls aversive behaviour and is sensitive to punishment, and the other that controls appetitive behaviour and is sensitive to reward (Corr, 2009).

Gray’s RST was consistent with part of Eysenck’s theory, that is, that introverts are more easily aroused than extroverts and thus more sensitive to punishment. Gray obtained the strongest support for his theory from animal experiments in which he observed that barbiturates including alcohol have an extroverting effect on behaviour. He theorised that this was because barbiturates reduced the sensitivity of the punishment mechanism (Gray, 1970). Gray’s modification to Eysenck’s theory specified that the reward and punishment sensitivity drives are primary motivational/learning systems, with highly impulsive individuals being sensitive to reward, and highly anxious people being sensitive to punishment. In comparison, the two dimensions proposed by Eysenck (introversion-extroversion and neuroticism) were considered to be secondary factors of these two more
fundamental processes (Corr, 2009). It is important to note that according to RST, the reward and punishment sensitivity drives share much in common in terms of their purpose and effects (Alloy et al., 2009).

Gray coined the terms “Behavioural Inhibition System” (BIS) and “Behavioural Activation System” (BAS) to depict the sensitivity to punishment and sensitivity to reward drives respectively. BIS was postulated to be responsible for organising behaviour in response to conditioned aversive stimuli, whilst BAS was postulated to be responsible for organising behaviour in response to conditioned appetitive stimuli (Bijttebier, Beck, Claes, & Vandereycken, 2009). Gray further developed RST to include a third major system, the Fight-Flight System (FFS) which was postulated to respond to unconditioned aversive stimuli, by way of unconditioned defensive aggression (fight) or escape behaviour (flight) (Gray, 1987, as cited in Bijttebier et al., 2009).

In applying a biopsychosocial framework to investigations in this thesis, the personality construct of drive sensitivity was selected as it potentially plays a key role in social anxiety, drinking/drug-taking behaviour, and also in the relationship between SAD and SUD. The importance of the temperamental characteristic Behavioural Inhibition (BI) and its role as a risk factor for social anxiety has been discussed in Chapter 1. Punishment sensitivity is a related construct to BI. Neurobiological research has highlighted the importance of the effects of psychoactive substances on the neurotransmitter dopamine. Dopamine’s relationship to reward sensitivity has been outlined in Chapter 2. In addition, behavioural disinhibition has been found to be related to problematic substance use. Thus, both the punishment and reward sensitivity drives would appear to have important functions in the aetiology of both SAD and SUD. Considering the role of both these processes ought to increase our understanding of the SAD-SUD comorbidity in order to better inform prevention and treatment. The roles of reward and punishment sensitivity in the SAD-SUD comorbidity have been poorly explored according to existing literature. RST can assist in further understanding this comorbidity by way of individual differences in reward and punishment sensitivity.

**RST revision.** RST was substantially revised in 2000 by Gray and McNaughton. Three systems are still proposed but different stimuli are supposed to activate them. BAS is postulated to organise behaviour to respond to all appetitive stimuli (both conditioned
and unconditioned). The Fight-Flight-Freeze System (FFFS) is postulated to organise behaviour to respond to all aversive stimuli (both conditioned and unconditioned), and thus takes the role of BIS from the original RST. The FFFS is believed to underlie fear and panic. The role of the original BIS is the most changed in the revised RST. BIS is postulated to be responsible for goal conflict in general and is thought to underlie anxiety (Bijttebier et al., 2009). The role of BIS in resolving goal conflict is theorised to occur by increasing the negative power of stimuli, that is, by activating the FFFS until the conflict is resolved either in favour of avoidance or approach. Thus there is a close relationship between FFFS and BIS in revised RST, and they can still be classified together as a punishment sensitivity factor of personality (Corr, 2009; Gray & McNaughton, 2000). In any event, at the present time, most measures used in research still reflect these two primary drives (BAS/reward and BIS/punishment sensitivity), as research continues to develop psychometrically sound measures to reflect the revised RST drives.

For the purpose of the literature review that follows, it is important to note that two frequently used measures in research examining these two innate drives are the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) (Torrubia, Ávila, Moltó, & Caseras, 2001), and the BIS/BAS scales (Carver & White, 1994). The BAS scale in addition to being a primary measure, is comprised of three subscales which have also been examined in some of the studies. The “Drive” scale measures the active or persistent pursuit of desired goals. The “Fun-Seeking” scale measures the desire for new rewards as well as willingness to approach potentially rewarding events spontaneously. The “Reward Responsiveness” scale measures positive responses to an occurring or anticipated reward (Carver & White, 1994; Corr, 2009).

Social anxiety and personality (drive sensitivity) in non-clinical populations. A number of studies (ten to date) have examined the relationship between social anxiety and personality as represented by RST in non-clinical samples. The literature demonstrates that punishment sensitivity/BIS is positively associated with social anxiety. Findings with respect to an association between social anxiety and reward sensitivity/BAS are less consistent.

Sensitivity to punishment (SP) or BIS, has consistently been found to be positively associated with SAD symptoms in various non-clinical populations. These include children
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The literature pertaining to sensitivity to reward (SR) or BAS is less consistent. SAD symptoms have generally been found to be either negatively related, or unrelated, to SR or BAS. Furthermore, both types of findings have been obtained in the same study in some cases, where more than one measure/subscale of BAS and/or social anxiety symptoms have been used. For example, in a study involving children, Coplan et al. (2006) reported a negative relationship between fear of negative evaluation and BAS, and also between a measure of generalised social avoidance/distress and BAS. However, no relationship was apparent between a measure of avoidance/distress in new situations/meeting new people and BAS. In another example, Booth and Hasking (2009) demonstrated a negative relationship between social anxiety and both BAS Drive and BAS Fun-seeking. However social anxiety was not related to BAS Reward Responsiveness. Findings from both these studies appear to favour a negative relationship between SAD symptoms and SR, as two of the three relevant subscales in each of these studies that were used to measure the strength of the relationship between social anxiety and reward sensitivity, found negative associations between them.

Findings from a number of studies have demonstrated that social interaction anxiety is negatively related to reward sensitivity/BAS (e.g., Kashdan, 2002), while social performance anxiety is unrelated to SR (e.g., Kimbrel et al., 2008). Kimbrel et al. (2010) built on the results obtained by Kashdan (2002) by using a number of both social anxiety and drive sensitivity measures. The authors concluded that the results from their study provide further support for the notion that low reward sensitivity/BAS is related to high social anxiety. More recently, Levinson et al. (2011) obtained results consistent with the findings of Kimbrel et al. (2010), providing additional support for the notion that high (more severe) SAD is associated with low SR. Such results are consistent with the continuum theory in relation to SAD, outlined in Chapter 1 of this thesis. Findings suggest
that social interaction anxiety which is thought to underlie generalised SAD (or more severe social anxiety symptoms), may be related to low reward sensitivity, whilst performance anxiety which is thought to underlie specific SAD and is characterised as a milder form of social anxiety is not related to SR.

Three other studies highlight the inconsistent findings with the afore-mentioned research into the relationship between SR/BAS and social anxiety symptoms in non-clinical studies. First, Kimbrel et al. (2012) obtained results indicating a negative association between social performance anxiety and SR. Second, no relationship at all was found between BAS and social anxiety symptoms by Kashdan and Roberts (2006). Notably, Kashdan and Roberts (2006) measured social anxiety by combining interaction and performance anxiety scores into a unitary measure. Third, and most recently, in an attempt to clarify inconsistencies with regard to negative versus no associations between reward sensitivity and social anxiety, Ly and Gomez (2014) utilised several measures of both social anxiety and BAS/reward sensitivity. Only one of the reward sensitivity measures, BAS Fun-seeking, was negatively related to both performance and interaction anxiety. All other measures of SR were unrelated to social anxiety measures. Thus, the attempts so far to clarify the nature of the relationship between SAD symptoms and SR have not produced consistent findings. At least in part, these inconsistencies may be attributable to methodological differences.

There is only one study (Kingsbury et al., 2013) that indicated a positive association between SR/BAS and social anxiety symptoms. This study involved children but can be distinguished from the study conducted by Coplan and his colleagues, as the BIS/BAS scales used by Kingsbury et al. (2013) were specifically adapted for children. A positive relationship was found between social anxiety and BAS Reward, whilst no relationship was found between social anxiety and either BAS Drive or BAS Fun-Seeking. Notably, no relationship was demonstrated between two of the three BAS subscales and social anxiety symptoms.

In summary, in non-clinical studies, there is a positive relationship between sensitivity to punishment/BIS and social anxiety symptoms. However at the present time, no conclusion can be reached with respect to the relationship between sensitivity to reward/BAS and social anxiety symptoms.
Social anxiety disorder (SAD) and personality (drive sensitivity) in clinical samples. Only one study appears to have been conducted with clinical samples investigating the relationship between, or group differences in relation to SAD and drive sensitivity symptoms. Morgan et al. (2009) compared a group of 23 patients with diagnosed SAD (generalised subtype according to DSM-IV criteria), recruited from a psychiatric outpatient clinic, with a group of 48 controls matched for age, gender and education level. All participants had no other psychopathology and all SAD participants took no psychotropic medication for at least 4 weeks prior to the study. There was a significant group effect on total BIS scores (SAD patients had higher BIS scores). No significant difference was initially found between the groups on total BAS subscale scores. However, a one-way exploratory analysis of the BAS subscales revealed a significant effect for BAS Fun-Seeking only (SAD patients had lower BAS Fun-Seeking scores).

The findings of Morgan et al. (2009) are consistent with those of one of the afore-described non-clinical studies, Ly and Gomez (2014), where only BAS Fun-Seeking was negatively related to both social performance and interaction anxiety. However, caution must be applied to the Morgan et al. (2009) findings due to the low numbers of SAD group members as well as the difference in size between the two groups. It is not clear that all assumptions were tested before data analysis was performed on the information presented. At this time, there is insufficient evidence to indicate that a significant association exists between SAD and reward sensitivity in clinical samples or in non-clinical samples. However, the results with regard to BIS scores confirm a positive relationship exists between SAD and punishment sensitivity in both clinical and non-clinical samples.

Substance use and personality (drive sensitivity). A large number of studies have examined the relationship between personality as defined by RST and substance use in both clinical and non-clinical populations. In experimental research, reward sensitivity has been shown to be a significant predictor of reactivity to alcohol cues (responses to sight, taste and smell of alcohol) and craving in both clinical (Franken, 2002) and non-clinical populations (Franken, 2002; Kambouropolos & Staiger, 2009; Palfai & Ostafin, 2003; Zisserson & Palfai, 2007). Punishment sensitivity has been shown to be unrelated to craving and reactivity to alcohol cues amongst clinical and non-clinical samples (Franken, 2002; Palfai & Ostafin, 2003; Zisserson & Palfai, 2007). As would be expected with an
innate drive that is sensitive to reward without regard for negative consequences, most research in this area (using the SPSRQ and/or the BIS/BAS scales) demonstrates a positive relationship between sensitivity to reward (SR) or the behavioural activation system (BAS) and substance use, abuse and dependence (Bijttebier et al., 2009). However, research considering the association between substance use and sensitivity to punishment (SP) or the behavioural inhibition system (BIS) is less conclusive.

**Substance use and reward sensitivity in non-clinical populations.** In non-clinical studies, there is strong evidence that reward sensitivity/BAS is positively related to alcohol or substance use/abuse (including cannabis) amongst various populations. These include adolescents (van Leeuwen, Creemers, Verhulst, Ormel, & Huizink, 2011), students (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010; Gullo, Ward, Dawe, Powell, & Jackson, 2011; Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Hundt, Williams, Mendelson, & Nelson-Gray, 2013; Jonker, Ostafin, Glashouwer, van Hemel-Ruiter, & de Jong, 2014; Lyvers, Duff, Basch, & Edwards, 2012; O'Connor & Colder, 2005; Pardo, Aguilar, Molinuevo, & Torrubia, 2007), and community members (Ivory & Kambouropoulos, 2012; Kabbani & Kambouropoulos, 2013).

In a number of studies, where BAS subscales have been utilised, BAS Fun-Seeking has shown the strongest association with alcohol or substance use/abuse (Booth & Hasking, 2009; Franken & Muris, 2006; Jorm et al., 1999; Wood, Dawe, & Gullo, 2013). As BAS Fun-Seeking has been shown to be correlated to impulsivity measures (Dawe & Loxton, 2004), this suggests that it is the impulsivity and spontaneity of fun-seeking within the ambit of reward sensitivity that is most strongly related to alcohol and substance use.

In contrast to the majority of non-clinical studies which have demonstrated a positive relationship between reward sensitivity and alcohol use, Hasking (2006) found no relationship between any of the BAS subscales and hazardous drinking in an adolescent sample. However, there may be methodological issues with Hasking’s finding. The average age of the participants was 14 years old, four years younger than the legal drinking age in Australia. Furthermore, the results demonstrated older participants reporting hazardous alcohol use. In addition all participants were recruited from private schools. Hasking (2006) commented that insufficient variance in drinking behaviour and type of
schooling may have contributed to the insignificant finding with respect to the relationship between reward sensitivity and drinking behaviour.

The majority of non-clinical studies examining the relationship between reward sensitivity and cannabis use have demonstrated a positive relationship among adolescents and students (Simons, Dvorak, & Batien, 2008; van Leeuwen et al., 2011). Nevertheless, one study involving a student population, found sensitivity to reward to be unrelated to cannabis use (Simons & Arens, 2007). One explanation for this inconsistent result may be the low usage of cannabis in the sample. Only 28% of the students reported cannabis use in the previous 6 months, and non-use of cannabis was strongly associated with negative cannabis expectancies. In addition, reward sensitivity had an indirect effect on cannabis use as it attenuated the (negative) relationship between punishment sensitivity and cannabis use. In summary, it is clear that reward sensitivity is positively related to alcohol and cannabis use in non-clinical populations.

Substance use and reward sensitivity in clinical populations. Experimental and clinical studies also demonstrate a positive relationship between reward sensitivity/BAS and alcohol/substance use among various populations. These include adolescents (Colder et al., 2013), young adults (Johnson, Turner, & Iwata, 2003) and adults (Dissabandara et al., 2014; Franken, Muris, & Georgieva, 2006; Lyvers et al., 2014; Perry et al., 2013).

With respect to the relationship between reward sensitivity and alcohol use disorders, two exceptions to these findings have been reported. Franken et al. (2006) compared differences on BIS/BAS scales between three groups: drug addicts (cocaine and/or heroin dependent: \(N = 71\)), alcoholics \(N = 39\) and controls \(N = 96\). Drug addicts had significantly higher BAS Drive, BAS Fun-Seeking and BAS Total compared to controls. However, no significant differences were found on BAS between controls and alcoholics (although the mean scores of alcoholics were higher than those of controls on all BAS measures). The researchers suggested that these findings make sense as these two BAS measures reflect risk-taking and novelty-seeking, and such behaviours are more strongly associated with obtaining and using illegal drugs, compared to drinking activities.

The second exception to the afore-mentioned clinical studies was in research involving treatment seekers at a residential rehabilitation program for alcohol and drug dependence. No relationship was detected between sensitivity to reward and hazardous
alcohol use (Gullo et al., 2010). This result was inconsistent with the finding of a positive relationship between sensitivity to reward and hazardous alcohol use with a student population in Study 1 of the same research, and using the same alcohol use measure. However, a separate measure of impulsivity was also utilised, as the focus of the study was on investigating cognitive pathways between impulsivity and alcohol misuse. Impulsivity was positively related to hazardous alcohol use in the treatment seeker group. This finding is consistent with those from non-clinical studies which showed that BAS Fun-Seeking (which measures impulsivity) has the strongest association with substance use. In addition, hazardous alcohol use was measured via a theoretical cut-off score for alcohol dependence on the alcohol use measure. These participants may have been polydrug users which could have confounded the results. In summary, the evidence from clinical studies points to a positive relationship between alcohol/substance use disorders and reward sensitivity.

Substance use and punishment sensitivity in non-clinical populations. Research investigating the relationship between punishment sensitivity and alcohol/substance use in non-clinical populations is less consistent. About half the research indicates a negative relationship between punishment sensitivity and alcohol/substance use, while the other half reports no relationship between sensitivity to punishment and risky substance use. For example, among university students, punishment sensitivity has been found to be negatively related to alcohol use (Franken & Muris, 2006; Jonker et al., 2014; O'Connor & Colder, 2005; Pardo et al., 2007), cannabis use (Simons & Arens, 2007; Simons et al., 2008) and risky substance use (Hundt et al., 2008). Among adolescents, punishment sensitivity has been found to be negatively related to both lifetime and repeated cannabis use (van Leeuwen et al., 2011).

On the other hand, BIS/punishment sensitivity has been found to be unrelated to alcohol and/or substance use in an adolescent sample (Hasking, 2006), a predominantly student population (Booth & Hasking, 2009), student samples (Hundt et al., 2008; Hundt et al., 2013; O'Connor & Colder, 2005), and community samples (Feil & Hasking, 2008; Jorm et al., 1999).

One exception to these findings is a recent non-clinical study that demonstrated a positive relationship between punishment sensitivity and risky alcohol use amongst social drinkers, (Ivory & Kambouropoulos, 2012). Ivory and Kambouropoulos (2012) suggest
that this positive association may be explained by alcohol use being motivated to reduce tension. Another methodological reason for this exception may be that a different self-report scale (measuring the “Fight-Flight-Freeze System”) was used to measure punishment sensitivity as conceptualized in the revisions made to RST (Gray & McNaughton, 2000).

Inconsistent findings with respect to the relationship between punishment sensitivity and alcohol/substance use has led to research investigating the relationship between the punishment and reward drives, partly because interactions between reward and punishment sensitivity are a major tenet of RST (Corr, 2009). With respect to cannabis use, two studies which demonstrate a negative relationship between punishment sensitivity and cannabis use, also found the association was attenuated by reward sensitivity (Simons & Arens, 2007; Simons et al., 2008). With respect to alcohol use, punishment sensitivity has been found to have a positive association with alcohol use/problems only if reward sensitivity levels are high (Keough & O'Connor, 2014; Wardell, O'Connor, Read, & Colder, 2011). At this time, all relevant findings indicate that no conclusion can be reached as to the significance of, and/or direction of the relationship between punishment sensitivity and substance use in non-clinical populations.

**Substance use and punishment sensitivity in clinical populations.** Experimental and clinical studies show no relationship between punishment sensitivity/BIS and alcohol/substance use in adolescents (Colder et al., 2013) and young adults (Johnson et al., 2003). Furthermore, amongst adults, Franken et al. (2006) found no significant differences on BIS across three groups (drug addicts, alcoholics and controls), and Perry et al. (2013) found no difference between two groups (substance dependent inpatients in residential treatment, compared to community members with no alcohol/substance dependence). However, in a recent study, substance dependent inpatients from residential rehabilitation facilities had significantly higher scores on punishment sensitivity compared to a group of community social drinkers (Lyvers et al., 2014). Thus, although some evidence suggests a positive relationship between punishment sensitivity and substance dependence, the majority of research on clinical samples shows no association between these two variables.

**Reward sensitivity and punishment sensitivity in the SAD-SUD relationship.** Only two studies to date appear to have considered drive sensitivity (using either the BIS/BAS scales or the SPSRQ measure), simultaneously with social anxiety and substance
use. Booth and Hasking (2009) directly examined the roles of reward and punishment sensitivity in the relationship between social anxiety and drinking behaviour. Self-report measures were completed by 454 participants of whom the majority (83.2%) was university students. Social anxiety was positively related to BIS and negatively related to BAS Drive and BAS Fun-Seeking. Annual alcohol intake was negatively related to the fear subscale of social anxiety and positively related to BAS Fun-Seeking. After controlling for age and gender, only BAS Fun-Seeking (out of all social anxiety and BIS/BAS subscales) predicted alcohol consumption. These results are consistent with findings in previously discussed non-clinical studies with regard to the relationships amongst personality, social anxiety and substance use.

Nicholls, Staiger, Williams, Richardson, and Kambouropoulos (2014) used latent class analysis to classify 351 community members into four social anxiety subgroups. Type One was characterised by subclinical social anxiety with low punishment sensitivity and risk taking (including low risk alcohol/substance use); Type Two had subclinical social anxiety with high reward sensitivity and risk taking; Type Three had clinical social anxiety with high punishment sensitivity and low risk taking; and Type Four had clinical social anxiety with high punishment sensitivity, reward sensitivity and risk taking. These findings point to socially anxious individuals with problematic substance use, being highly sensitive to both punishment and reward.

In summary, research with respect to drive sensitivity and its relationships to social anxiety and substance use demonstrate strong positive relationships between social anxiety and punishment sensitivity, and between substance use and reward sensitivity. High levels of both reward and punishment sensitivity are associated with the SAD-SUD comorbidity.

**Alcohol Expectancies**

Alcohol expectancies are important cognitions which influence drinking behaviour. They have been extensively researched since the 1970s (e.g., see review by Monk & Heim, 2013). More recently the significance of their role in the relationship between social anxiety and alcohol has been recognised (Burke & Stephens, 1999) and continues to be researched (see recent review by Buckner et al., 2013b). Such research tests the moderating or mediating role of expectancies in the SAD-SUD relationship to advance our understanding and explain inconsistent findings described earlier in this chapter.
Expectancies represent a key psychological factor/vulnerability within the biopsychosocial framework being applied to this research study. This section outlines the role of expectancies in alcohol/substance use behaviour, and then provides a review of the literature on the role of alcohol expectancies in drinking behaviour, and the SAD-SUD comorbidity.

**Expectancies and alcohol use.** Social cognitive theory first proposed by Bandura (1977a, 1997), emphasises the importance of both external influences (one’s environment) and internal influences (one’s cognitions and personality) on the individual’s behaviour and development over time. Thus social cognitive theory recognises the importance of individual differences, and has been applied in research to factors such as personality, expectancies and coping strategies (the three individual difference variables being investigated in this thesis) in relation to drinking behaviour.

Alcohol expectancies are beliefs one has about the effects of alcohol on their behaviour, emotions, cognitions, and physiological state (Burke & Stephens, 1999). Such effects can be desirable or undesirable and are referred to as positive and negative expectancies respectively (Leigh & Stacy, 1993). Therefore, positive expectancies about alcohol will result in increased motivation to drink whilst negative expectancies will inhibit drinking behaviour (Burke & Stephens, 1999).

Alcohol expectancies have been demonstrated to exist from as young as six years of age (Miller, Smith, & Goldman, 1990), indicating that beliefs about the effects of alcohol are initially obtained from social learning experiences rather than from direct pharmacological experience. Children generally have more negative than positive expectancies about alcohol, but, after drinking onset, youth demonstrate positive expectancies (Noel & Thomson, 2012). Longitudinal studies demonstrate that positive expectancies (e.g., expectancies of social facilitation) among adolescents significantly predict both the commencement of drinking behaviour as well as subsequent levels of alcohol consumption (Smith, Goldman, Greenbaum, & Christiansen, 1995). Alcohol expectancies have also been shown to be related to, and to predict problematic alcohol use (Goldman, Del Boca, & Darkes, 1999). After adolescence, subsequent drinking experiences influence the development of expectancies so that the relationship between expectancies and drinking can vary with age. Generally negative expectancies are
predicted to increase with age due to the increasing likelihood of direct experiences with negative outcomes as one gets older, and negative expectancies may be a better predictor of alcohol consumption in those over age 35 (Leigh & Stacy, 2004). Given the changes at different stages of life with respect to the influence of positive versus negative expectancies, it is important to examine both in order to obtain a comprehensive picture of alcohol expectancies of research participants.

**Expectancies and cannabis use.** Alcohol expectancies have an important function with respect to alcohol use and can also influence or predict other substance behaviour, for example cannabis use (Stacy, Newcomb, & Bentler, 1991). Similar cognitive processes (that underlie expectancies) have been shown to be involved in both alcohol and cannabis use (Stacy, 1997). Furthermore, positive alcohol expectancies correlate positively with positive cannabis expectancies, and negative alcohol expectancies have a positive association with negative cannabis expectancies (Aarons et al., 2001). In both clinical (Brown, 1993) and non-clinical (Buckner & Schmidt, 2008) samples, frequent or high cannabis use is associated with positive cannabis expectancies. Thus, there are similarities in findings between clinical and non-clinical groups with respect to alcohol expectancies and use, as well as cannabis expectancies and use.

**Positive alcohol expectancies.** Early research in the 1980s focused solely on the role of positive alcohol expectancies in drinking behaviour for a number of reasons: first, the notion that positive expectancies are brought to mind more quickly as positive consequences are considered more immediate, and are thought to influence drinking behaviour more strongly compared to delayed negative effects; and second, the majority of the early questionnaires designed to measure expectancies only considered positive expectancies (Jones, Corbin, & Fromme, 2001). Early research found that clinical samples (patients in alcohol treatment programs) had stronger (positive) alcohol expectancies compared to non-clinical groups (e.g., students and hospitalized patients without an alcohol use disorder), and that across and within groups, expectancies strengthened with increased frequency and quantity of alcohol use (Brown, Goldman, & Christiansen, 1985). Thus, positive expectancies were found to mediate alcohol consumption in both clinical and non-clinical samples (e.g., Cooper, Russell, & George, 1988) in these early studies.
**Negative alcohol expectancies.** These findings from initial alcohol expectancy research led to questioning as to how the perception of alcohol’s positive reinforcement potential is maintained despite the negative consequences for some people, in particular those with alcohol dependence. It was considered that the strong influence of positive expectancies may increase vulnerability to relapse (Brown, Millar, & Passman, 1988). Therefore, researchers investigating techniques to improve treatment for alcoholics also started examining the role of negative alcohol expectancies. The premise behind the potential importance of negative expectancies was that they may limit the amount of alcohol consumed and/or motivate problem drinkers to reduce or cease drinking (Jones et al., 2001).

When negative expectancy measures were first used, they demonstrated that for clinical samples negative expectancies predict relapse behaviour at least as well as positive expectancies that is, the higher the level of negative alcohol expectancies at the time of admission for treatment, the greater the likelihood of remaining abstinent on discharge (Jones & McMahon, 1994a, 1994b). However, early findings with respect to non-clinical samples were contradictory in that either negative or positive expectancies were associated with alcohol consumption measures, but not both. As an explanation for these inconsistencies, Jones et al. (2001) proposed that the two main initial questionnaires measured different types of negative expectancies (one measured both proximal and distal effects, whilst the other tapped only proximal effects). Research using the questionnaire which considered both proximal and distal effects, found that negative expectancies were associated with alcohol consumption in social drinkers, whilst only positive expectancies were related to alcohol use in a student sample, using an expectancy measure that only measured proximal alcohol effects (studies cited by Jones et al., 2001). At the time of their review, Jones et al. (2001) concluded that positive expectancies may motivate initial use of alcohol, whilst negative expectancies may more strongly influence cessation of use.

**Recent expectancies research.** At the present time, although research on the role of expectancies in drinking behaviour and other substance use has continued, inconsistencies remain. These are contributed to by methodological differences (e.g., in measures used for both expectancies and alcohol/substance use outcomes, and statistical techniques applied), as well as sample differences (e.g., clinical, community, students)...
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(Hasking & Oei, 2008). In addition, research generally remains focused on the role of positive alcohol expectancies, which continue to demonstrate positive associations with risky alcohol use in both non-clinical and clinical populations (e.g., Gullo et al., 2010).

**Non-clinical samples.** Recent research investigating the roles of both positive and negative expectancies has established that students with high levels of positive expectancies but low levels of negative expectancies reported frequent and heavy drinking, whilst students with high levels of both positive and negative expectancies had difficulties with self-control and were at a higher risk of developing drinking problems (Leeman, Kulesza, Stewart & Copeland, 2012). In community samples, both positive and negative alcohol expectancies have been found to be positively related to drinking behaviours including alcohol volume, frequency of use, binge drinking frequency and problematic/hazardous alcohol use (Hasking & Oei, 2004; Pabst, Kraus, Piontek, Mueller, & Demmel, 2014). Thus, with respect to non-clinical samples, research indicates positive associations between both positive and negative expectancies and drinking behaviour (volume and frequency of use and hazardous use).

**Clinical samples.** There are similarities in findings in clinical samples. Alcohol and drug-dependent patients demonstrate that positive alcohol expectancies predict both quantity of alcohol consumption and alcohol problems (Galen, Henderson, & Coovert, 2001). Furthermore, significantly higher scores on total expectancies (both positive and negative added together) have been found for alcohol-dependent participants compared to community participants, with positive associations between total expectancies and alcohol use (frequency and/or volume) in both groups (Hasking & Oei, 2002a). Similar results were obtained by Connor, Gudgeon, Young, and Saunders (2007) with alcohol-dependent patients in treatment for detoxification. The strength of alcohol expectancies (total score of both positive and negative expectancies) was positively associated with dependence severity, as well as frequency and quantity of alcohol use. However, in another study conducted around the same time, neither positive or negative expectancies were associated with or predicted frequency and quantity of alcohol use among a cohort of alcohol-dependent adults who were recruited one week after completing detoxification programs (Hasking & Oei, 2007). Methodological differences may account for these contradictory findings.
Recent research has confirmed the majority of findings showing significant associations between both positive and negative expectancies with alcohol use. Li and Dingle (2012) compared mean scores on both positive and negative expectancy scales between students and a clinical sample. The clinical sample more strongly endorsed three of the four positive expectancy subscales, as well as the negative expectancy subscale. Thus the current evidence from clinical samples seems to point to such cohorts having stronger positive and negative expectancies compared to non-clinical samples, as well as both positive and negative expectancies being positively related to alcohol use disorders. In summary, despite the focus of research on positive expectancies in alcohol use, both positive and negative expectancies are important cognitions among both clinical and non-clinical populations in terms of their influence on drinking and other substance use behaviour.

Social anxiety, expectancies and alcohol/substance use. Two influential reviews have been conducted with respect to the relationship between social anxiety and alcohol use. Burke and Stephens (1999) highlighted the importance of alcohol expectancies in the relationship between social anxiety and alcohol use, particularly in student populations, in the first review of the literature on this topic. The review covered experimental (de Boer, Schippers, & van der Staak, 1994), longitudinal and cross-sectional studies up to that time. The experimental study demonstrated that (self-reported) socially anxious women who believed they had consumed alcohol and had high social facilitation expectancies, were less anxious than socially anxious women with low social expectancies. Burke and Stephens (1999) concluded that social anxiety was an important motivating factor for alcohol use in at least a subset of university students. They found that higher levels of social anxiety were related to higher levels of positive expectancies which in turn were related to higher drinking levels. Burke and Stephens (1999) further concluded that expectancies moderate the relationship between social anxiety and drinking behaviour in that socially anxious students who hold high positive expectancies are more likely to use alcohol, whilst those with high negative expectancies are less likely to drink.

Shortly after the Burke and Stephens (1999) literature review, experimental studies (Abrams et al., 2001; Himle et al., 1999) discussed earlier in this chapter, examined the role of beliefs about alcohol in the relationship between social anxiety and alcohol use.
disorders. Findings from these studies suggested that reduced anxiety in socially anxious individuals may be principally due to a positive anxiety-reducing expectancy of the effect of drinking. Thus these studies supported positive expectancies as a mediator in the SAD-AUD relationship. Another experimental study (Abrams & Kushner, 2004) supported a moderating role for positive expectancies (tension-reduction) in the relationship between alcohol consumption and social anxiety disorder. Men with stronger tension reduction expectancies reported less anticipatory social anxiety compared to men with weaker expectancies (participants had a SAD diagnosis and were social drinkers).

Morris et al. (2005) reviewed the moderating role of alcohol expectancies in the relationship between social anxiety and alcohol use in research using self-report measures. Their review covered the literature up to that time with both clinical and non-clinical samples, and aimed to explain inconsistencies in the literature with respect to the relationship between social anxiety and alcohol-related variables. In two studies using student samples (Eggleston et al., 2004; Lewis & O’Neill, 2000), no moderator effects were found for either positive or negative expectancies in the relationship between social anxiety and alcohol use or alcohol problems. Eggleston et al. (2004) also found no mediator effects, although there were positive associations between both positive and negative expectancies and social anxiety.

In contrast, moderator effects were evident for positive expectancies (for social facilitation) in a study which recruited (undiagnosed) student hazardous drinkers divided into (undiagnosed) high and low social anxiety groups (Tran, Anthenelli, Smith, Corcoran, & Rofey, 2004). The hazardous drinkers with high SAD had more social facilitation expectancies in social drinking situations, than hazardous drinkers with low SAD. Moderator effects were also evident for positive (social facilitation and assertiveness) expectancies in a study with community participants who were compared on (undiagnosed) high and low social anxiety (Ham, Carrigan, Moak, & Randall, 2005). Community members with high social anxiety had more social facilitation and assertiveness expectancies than non-socially anxious community members, although there were no differences between the two community groups on other positive expectancies (that were not specific to social situations). In summary, there was some evidence for moderator
effects of positive alcohol expectancies relating to social facilitation in both student and community samples at the time of the review by Morris et al. (2005).

Moderator and/or mediator effects were also found in two clinical studies conducted during the same period (Ham et al., 2002; Tran & Haaga, 2002). Tran and Haaga (2002) compared three groups on positive and negative alcohol expectancies: socially anxious individuals (SADs), SADs with alcohol use disorders (comorbids), and normal controls. SADs were chosen on the basis of a DSM-IV (American Psychiatric Association, 1994) diagnosis of SAD, no lifetime alcohol use disorder (AUD), and no current drug dependence. Comorbids were required to have current SAD, a current AUD, and no current drug dependence. Tran and Haaga (2002) found that the comorbid group had higher positive (social facilitation and tension reduction) expectancies compared to both other groups, and the SADs had higher social facilitation expectancies compared to the normal controls. These results suggest moderation in that SADs who are high in these positive expectancies may develop the comorbid condition (SAD and AUD). Interestingly with respect to negative expectancies (of cognitive and behavioural impairment), both social anxiety groups were higher on these than controls. This was in contrast to the prediction that the comorbid group would have lower negative expectancies compared to the two non-alcohol abusing groups. The actual result suggests that with prolonged problematic or high alcohol use, there is an increased likelihood that negative consequences may occur, so that comorbid individuals may develop higher levels of negative expectancies.

The second clinical study, Ham et al. (2002) compared three groups on positive alcohol expectancies only: socially anxious individuals (SADs), individuals with dysthymia (dysthymics), and normal controls. SADs and dysthymics were chosen on the basis of clinical interview measures consistent with DSM-IV (American Psychiatric Association, 1994) criteria. Individuals with alcohol/substance use disorders in the previous 6 months were excluded although this only resulted in the removal of one potential participant. The SADs were higher than both other groups on one positive expectancy (social assertiveness). The SADs were also higher than controls on two other positive expectancies (global positive change and tension reduction), whilst the dysthymics did not differ from either group on these two expectancies (they were placed in between the two other groups on their
mean scores for these two expectancies). There were no differences of alcohol consumption between the three groups. Further analysis indicated the relationship between social anxiety and alcohol consumption was both direct and mediated by alcohol expectancies. In summary, the clinical studies at this time found evidence for moderation and mediation of positive expectancies, and there were significant differences between clinical groups and controls on both positive and negative expectancies.

Overall, findings from all studies examining the role of expectancies in the relationship between social anxiety and alcohol use up to the time of the review by Morris et al. (2005), suggested that more positive expectancies were held by socially anxious individuals and/or problem drinkers, compared to individuals without social anxiety and/or without alcohol use problems (Morris et al., 2005). Furthermore, in both clinical and non-clinical samples there was strong evidence of a positive association between social anxiety and social facilitation expectancies, and less strong evidence for a positive relationship between social anxiety and negative alcohol expectancies. There was also evidence for moderation of a wider range of positive alcohol expectancies in the relationship between social anxiety and alcohol use in clinical samples compared to non-clinical (community) samples where the evidence for moderation of positive expectancies was mixed. There was no evidence of moderation of negative alcohol expectancies among non-clinical samples.

**Recent developments in social anxiety and expectancies research.** More recently, strong evidence has emerged for the existence of positive relationships between both positive (e.g., social facilitation, assertiveness, tension reduction) and negative alcohol expectancies and social anxiety in student samples, (findings from a meta-analysis conducted by Schry & White, 2013), and in community samples (Carrigan, Ham, Thomas, & Randall, 2008). In regard to moderation of alcohol expectancies in the relationship between social anxiety and alcohol use/problems, evidence especially in relation to student samples remains somewhat mixed. There is some evidence that positive alcohol expectancies moderate the association between social anxiety and alcohol consumption (Booth & Hasking, 2009; Gilles et al., 2006). However, in both these studies, third (alcohol-related) variables were also involved in the interactions between social anxiety and drinking behaviour. In contrast, other studies with student samples have not found evidence of positive expectancies moderating the relationship between social anxiety and
risky alcohol use (Ham, 2009). However, Ham (2009) did find evidence of one type of positive expectancy (social facilitation) partially mediating this relationship.

Another developing line of research in this area has investigated (directly or indirectly) the moderating role of alcohol expectancies with either more specifically defined drinking or expectancy variables. This research provides evidence supporting the role of alcohol expectancies as moderating variables. In a recent study with a student sample, neither positive expectancies, negative expectancies, or social anxiety, predicted risky alcohol use. However, a positive (social assertiveness) and a negative (cognitive impairment) alcohol expectancy, interacted with social anxiety to predict “drinking due to social anxiety”. This dependent variable was classified as a drinking motive in this study (Cludius, Stevens, Bantin, Gerlach, & Hermann, 2013). Similarly, in another study, two groups of community members were compared on “drinking to cope” status (either high or low level). This variable measured drinking to cope with anxiety generally, and with social anxiety specifically. The findings suggested that both positive and negative alcohol expectancies play a moderating role in the SAD-AUD relationship as both expectancy types were found to be associated with high levels of social anxiety, alcohol consumption, and problematic alcohol use (Carrigan et al., 2008). Finally, another study with a student sample identified the moderating role of both positive and negative alcohol expectancies specifically relating to “convivial contexts” (e.g., at a party), in the relationship between social anxiety and risky drinking (Ham, Zamboanga, & Bacon, 2011).

In summary, at the present time, there is evidence of positive associations between both positive and negative expectancies and social anxiety symptoms particularly in student populations. As research continues, there is also emerging clearer evidence of the moderating role of both positive and negative alcohol expectancies in the relationship between social anxiety and alcohol use in other non-clinical samples. Clinical studies to date have demonstrated significant differences between clinical groups (comorbid social anxiety and alcohol use disorders, and social anxiety disorder only) and control groups with respect to both positive and negative alcohol expectancies. Comorbid groups have significantly higher positive expectancies compared to socially anxious individuals, who generally have higher levels of positive expectancies compared to controls, and both clinical groups have higher negative expectancies compared to controls.
Social anxiety, expectancies and cannabis use. Negative cannabis expectancies have been found to moderate and/or mediate the relationship between social anxiety and problematic cannabis use in both non-clinical student (Buckner & Schmidt, 2008) and clinical samples (Buckner & Schmidt, 2009). As discussed in the Expectancies section of this chapter, expectancies for one behaviour (e.g., drinking) can influence expectancies for other substance behaviours (e.g., cannabis use). Furthermore, expectancies about the effects of other substances such as cannabis, are also thought to be important predictors of other substance use (Stacy, Newcomb & Bentler, 1991). Buckner and Schmidt (2009) studied two groups of current cannabis users (those with SAD and those without SAD). The SAD group was significantly higher on negative cannabis expectancies compared to the group without SAD. Thus, with respect to clinical studies, Buckner and Schmidt’s (2009) findings are consistent with those of Tran and Haaga (2002) where higher negative alcohol expectancies in both the comorbid (SAD and AUD) and SAD groups were found, compared to the control group (no SAD or AUD).

With respect to non-clinical studies, findings regarding cannabis expectancies in the relationship between social anxiety and cannabis use, are inconsistent with findings about alcohol expectancies in their association with social anxiety and alcohol use. Buckner and Schmidt (2008) found that positive cannabis expectancies were negatively related to social anxiety, and negative cannabis expectancies moderated the relationship between social anxiety and cannabis use such that increased negative expectancies predicted increased use. In addition, negative cannabis expectancies partially mediated the relationship between social anxiety and problematic cannabis use. There appears to be some differences in the relationship between social anxiety and cannabis use in non-clinical studies investigating cannabis expectancies, relative to such studies examining alcohol expectancies in the relationship between social anxiety and alcohol use.

Coping Strategies

Coping strategies have been researched in relation to stress for many decades. They represent an influential cognitive and behavioural factor within the biopsychosocial framework being applied in this thesis to investigate the relationship between social anxiety and substance use. Coping strategies are also derived from social learning theory. Specifically Burke and Stephens (1999) proposed that coping strategies are an important
variable in SAD-SUD comorbidity. They theorised that in addition to those socially anxious individuals who hold positive alcohol expectancies (especially the belief that alcohol facilitates social interactions), those who lack other coping strategies, are likely to drink heavily and/or problematically (Tran & Smith, 2008). This section outlines the theory and conceptualisation of coping and coping strategies, distinguishes coping strategies from a related variable (the coping motive in relation to substance use), and then reviews the literature relating to their role in alcohol/substance use behaviour, social anxiety and the SAD-SUD comorbidity.

**Theory of coping and coping strategies.** A significant proportion of the literature pertaining to the use of coping strategies in response to stress relies on the theory proposed by Lazarus and Folkman (1987). They defined coping as comprising cognitive and behavioural efforts to manage a stressful person-environment encounter. The stress is appraised as relevant to the person’s wellbeing and demanding or exceeding that person’s resources. The appraisal is comprised of primary and secondary components which are interdependent on each other. Primary appraisal occurs when the person assesses whether the encounter is stressful, benign/positive or irrelevant. Stressful encounters are categorised as threat (the potential for harm or loss), challenge (the potential for mastery or gain) and/or harm-loss (the injury has already occurred). Secondary appraisal occurs when the person considers their coping options and resources (Folkman & Lazarus, 1985).

Lazarus and Folkman (1987) distinguished between two coping functions in stressful encounters: (a) emotion-focused coping where efforts are directed at managing one’s emotional distress; and (b) problem-focused coping where efforts are directed at altering the relationship between the environment and oneself. Both these forms of coping were likely to be used in any stressful situation. In addition, the researchers showed that problem-focused coping was used more often in situations appraised as being changeable, whereas emotion-focused coping was used more in situations appraised as requiring acceptance. Furthermore, coping is a process such that as a situation changes, so does/can the coping response.

Approach and avoidance coping is another frequently used broad framework to categorise coping strategies particularly in relation to substance use (Forys, McKellar, & Moos, 2007). Approach coping is characterised by attending to the source of the stress and
attempting to deal with it (e.g., active planning), while avoidance coping involves minimizing the threat of the stressor via cognitive or behavioural strategies (e.g., self-distraction). Thus problem-focused coping may include approach strategies whilst emotion-focused coping may include avoidance strategies.

Lazarus and Folkman (1987) also suggested that a coping process may lead to favourable or unfavourable results, raising the possibility that some types of coping may be functional whilst others may be dysfunctional. More specifically, they proposed that functional versus dysfunctional coping may depend on the goodness of fit between what is actually happening and the individual’s appraisal of what is happening, and the individual’s appraisal of their coping options and coping actions. As previously discussed, the predominant explanatory model for the relationship between social anxiety and substance use is the self-medication hypothesis, that is, alcohol or cannabis is used to cope with social anxiety and this is presumably, or can become, a dysfunctional coping strategy. Thus, the relevance of investigating other coping strategies used by socially anxious individuals with and without problematic substance use becomes apparent. By examining coping strategies for stress as a shared vulnerability factor, excluding the use of substances as a coping strategy, it is hoped that our understanding of the SAD-SUD comorbidity with be increased. The present study relies on this classification of functional (adaptive) versus dysfunctional (maladaptive) coping strategies used by both clinical and non-clinical samples in response to stress, to further understand the relationship between social anxiety and substance use.

**Coping strategies and the coping motive.** In addition to coping strategies, another alcohol or substance-related variable that has been researched in relation to substance use behaviour and can be confused with maladaptive coping strategies, is the coping motive. Motives in general (i.e., reasons to drink or use drugs) have been determined to be the most proximal of variables in relation to drinking behaviour, that is, the reason to drink is usually the last factor involved before the actual decision is made (Raber & Hasking, 2010). The coping motive is one type of drinking/substance use motive that has been found to be strongly associated with alcohol/substance use in both clinical and non-clinical populations (Galen et al., 2001; Hasking, Lyvers, & Carlpio, 2011). The coping motive has also been
found to be significantly positively related to social anxiety in a recent meta-analysis of student samples (Schry & White, 2013).

There is a body of research that has looked at whether socially anxious individuals drink to cope or use cannabis to cope, specifically with their social anxiety. Thomas et al. (2003) examined drinking to cope with anxiety in social situations by comparing a group of highly socially anxious individuals with a group of individuals with none or low social anxiety (social anxiety was measured according to cut-off scores for social anxiety on two commonly used scales). They showed that individuals with high levels of SAD drank to cope with social situations, and avoided social situations where alcohol was not available. This finding was replicated by Buckner and Heimberg (2010) who also found that drinking to cope in social situations and avoiding social situations where alcohol was unavailable, mediated the relationship between SAD and alcohol-related problems. Highly socially anxious cannabis users have also been found to use cannabis to cope in social situations and avoid social situations if cannabis is unavailable. In addition, the use of cannabis to cope with social situations can mediate the relationship between highly socially anxious students and cannabis-related problems (Buckner, Heimberg, Matthews, & Silgado, 2012a). More recent research has confirmed the importance of using cannabis to cope with social anxiety in understanding problematic cannabis use (Buckner & Zvolensky, 2014). In summary, findings from research investigating the general coping motive as well as more specific drinking or using cannabis to cope variables, demonstrate strong relationships between such variables and both alcohol/substance use and social anxiety.

Maladaptive coping strategies have been found to be associated with and to interact with coping motives in relation to substance use (Gregg, Haddock, Emsley, & Barrowclough, 2014). In both clinical (Hasking & Oei, 2007) and non-clinical studies (Hasking & Oei, 2004), coping strategies interact with alcohol expectancies, another of the three important biopsychosocial variables being explored in this thesis in relation to the SAD-SUD comorbidity. There was no additional capacity to also consider the role of motives without potentially compromising the strength of findings in this thesis. The following literature reviews pertain specifically to the role of coping strategies, first with substance use behaviour, second with social anxiety, and third in the relationship between social anxiety and substance use.
Coping strategies and substance use. There is an abundance of literature which has examined coping strategies and alcohol or other substance use. This literature review focuses on associations between coping strategies and substance use, as well as the role of coping strategies in the relationship between stress variables and substance use in both clinical and non-clinical populations. It also summarises the literature pertaining to coping strategies in relation to the treatment of alcohol and cannabis use disorders.

Maladaptive coping and substance use in non-clinical samples. Non-clinical studies consistently show a positive association between maladaptive (avoidant and emotion-focused, e.g., self-blame, venting) coping strategies and alcohol use/problems among youth and adolescents (Cooper et al., 1995; Hasking, 2006; Rafnsson, Jonsson, & Windle, 2006), students (Hundt et al., 2013), and community samples (Cooper et al., 1988; 1992; 1995; Feil & Hasking, 2008; Ivory & Kambouropoulos, 2012; Veenstra et al., 2007). Studies with student samples also demonstrate a positive relationship between maladaptive coping strategies and substance use (Hundt et al., 2013). Vargas and Trujillo (2012) established that emotion-oriented (and not task-oriented or avoidance-oriented) coping predicted cannabis consumption in a sample of female students, although it is not clear from this research as to what coping strategies comprised emotion-oriented coping but it may well have included both adaptive and maladaptive strategies.

Some non-clinical studies also show a positive association between maladaptive coping strategies and stress (stressful life events) among youth (Rafnsson et al., 2006), and community adults (Cooper et al., 1992). Cooper et al. (1992) has been replicated in a longitudinal population-based study which looked at the relationships and moderating effects of emotion coping, cognition coping and active coping on stressful life events and alcohol use (Veenstra et al., 2007). Emotion coping style was characterised as inactive, indulgent, submissive and self-blaming (i.e., synonymous with a maladaptive coping style). Only emotion coping moderated the relationship between stressful life events and alcohol use such that people with high emotion coping used more alcohol to cope with a stressful life event, while people with a low emotion coping style used less alcohol in relation to a stressful life event. In summary, there is strong evidence of positive associations between maladaptive coping strategies and alcohol/substance use, as well as between maladaptive
coping strategies and stressful life events. Maladaptive coping styles have also been found to moderate the relationship between stress and drinking behaviour.

**Adaptive coping and substance use in non-clinical samples.** There is some evidence in non-clinical samples for a negative association between adaptive coping strategies and hazardous substance use. Evidence also supports, to some extent, adaptive coping as a moderator in the relationship between stress and drinking behaviour. In contrast to the afore-mentioned study Veenstra et al. (2007), restraint and suppression of competing activities (two adaptive coping strategies) were found to moderate the relationship between stress and alcohol use (but not alcohol problems) in a student sample in a more recent study (Corbin, Farmer, & Nolen-Hoekesma, 2013). However, maladaptive coping strategies were not measured by Corbin and his colleagues, and stress was operationalized as perceived stress with respect to unpredictability, lack of control and over-loading (Corbin et al., 2013). Nevertheless, these findings provide some support for the moderating role of adaptive coping strategies in the relationship between stress and alcohol use.

Although there is some evidence for a negative relationship between adaptive coping strategies and substance use in non-clinical studies, the findings are not as consistent as those of maladaptive coping strategies and substance use. Cooper et al. (1992) reported a negative relationship between adaptive coping strategies (active coping and social support) and both alcohol consumption and alcohol problems in a sample of community members. However, Hasking (2006) only reported a negative relationship between problem-solving (adaptive coping) and hazardous drinking in an adolescent sample after controlling for age and gender. In addition, Feil and Hasking (2008) only found a negative association between risky alcohol use and problem-focused (adaptive) coping in a community sample after controlling for age, depression, anxiety and stress. Different coping measures used by Cooper and colleagues compared to the studies conducted by Hasking (2006) and Feil and Hasking (2008) might explain the different findings.

Unlike Feil and Hasking (2008), Ivory and Kambouropoulos (2012) found a negative relationship between risky alcohol use and both emotion focused and problem focused coping among a group of community (social) drinkers, despite both sets of researchers using the same coping measure. Both emotion focused and problem focused
coping were comprised of adaptive coping strategies including seeking emotional and instrumental support, active coping, planning and suppression of competing activities. The stronger findings obtained by Ivory and Kambouropoulos (2012) may be explained by their use of a larger sample, compared to Feil and Hasking (2008).

Other research has identified both positive and negative relationships between avoidant (maladaptive) coping strategies and alcohol use (volume and frequency) in a community sample (Hasking & Oei, 2004). Three avoidant subscales (i.e., seeking social support, venting and substance use), were chosen for the study, based on the results from a factor analysis on the COPE measure conducted by the authors in 2002 (cited in Hasking & Oei, 2004). Seeking social support (classified as an adaptive coping strategy in other research as well as in this thesis), was negatively related to both volume and frequency of alcohol use, whilst drug/alcohol use was positively related to both alcohol measures. Thus, among non-clinical populations, evidence for a negative relationship between adaptive coping strategies and risky substance use appears to be stronger when other variables (e.g., age and gender) are controlled.

**Coping strategies and substance use in clinical samples.** Research with clinical samples also demonstrates significant relationships between coping strategies and substance use disorders. In a study comparing adolescents with and without cannabis dependence (Cascone, Zimmermann, Auckenthaler, & Robert-Tissot, 2011), withdrawal/avoidant coping strategies significantly predicted cannabis dependence. Neither active nor internal coping (adaptive coping strategies) predicted cannabis dependence.

In a study by Hasking and Oei (2002a), significantly higher scores on total coping strategies (all adaptive and maladaptive strategies added together), were found for alcohol-dependent participants recruited one week after completing detoxification programs (some of whom remained in a treatment program), compared to community participants. Total coping strategies was positively related to frequency of alcohol use in the alcohol-dependent participants group. There were no significant correlations between total coping strategies and volume or frequency of alcohol use in the community group.

In another study conducted by Hasking and Oei (2007), only one of four avoidant (maladaptive) coping strategies, denial, was negatively associated with frequency of
alcohol use, but all four coping strategies (religion, venting, denial and drug/alcohol use) predicted frequency of alcohol use (but not volume) among a cohort of alcohol-dependent adults who were recruited one week after completing detoxification programs. One coping strategy (denial) negatively predicted alcohol frequency, and the other three (religion, venting and drug/alcohol disengagement) positively predicted alcohol frequency. These four subscales were classified as avoidant by the authors according to a previous factor analysis on the COPE measure conducted by them in 2002 (cited in Hasking & Oei, 2007). Overall, findings from clinical studies demonstrate more consistently a positive relationship between maladaptive coping strategies and substance use disorders, compared to findings as to a negative relationship between adaptive coping strategies and substance use disorders.

**Coping strategies, substance use and treatment outcomes.** Other research has focused on coping strategies in the context of treatment outcomes for substance use disorders. Studies have generally shown that the greater the number and frequency of adaptive coping strategies used, the lower the risk of relapse. In a longitudinal study conducted over a 16 year period involving participants with alcohol use disorders (but who had not entered treatment at baseline), those that relied more on approach coping (adaptive coping) were more likely to recover (Moos & Moos, 2007). Furthermore, higher levels of both approach and alcohol-specific (adaptive) coping and lower levels of avoidance coping have been shown to be related to less alcohol and drug use and fewer drinking problems one year after treatment (Forys et al., 2007). Notably, the findings of the Forys study are limited to male veterans with substance use disorders who voluntarily chose to attend a residential treatment facility. Nevertheless, these results point to a negative relationship between adaptive coping strategies and substance use disorders.

One line of research comprises studies comparing treatments for alcohol or cannabis use disorders, where one of the treatments (usually CBT) involves training participants in the use of various adaptive coping strategies (comprised of both active and avoidant strategies) specifically designed to lower the risk of relapse. In a study comparing alcohol use disorder treatments (Litt, Kadden, Cooney, & Kabela, 2003), participants were randomly assigned to either CBT treatment which focused on developing coping skills, or interactional treatment which focused on examining interpersonal interactions. Only the
CBT treatment specifically included training in adaptive coping strategies to prevent relapse. Coping skills and drinking outcomes were assessed both pre and post-treatment and at various times up to 18 months after commencement of treatment. Both treatment groups significantly increased their use of coping strategies which predicted significant treatment outcomes. However, there were no differences between the groups on the use of coping strategies.

In a study with 450 treatment seekers for cannabis dependence (Litt et al., 2005), various outcomes for three treatment groups were compared for up to 15 months from baseline. One group had combined CBT with motivational therapy (MT) and was trained to use adaptive coping strategies (i.e., comprising active and avoidant coping strategies specifically for modifying cannabis use behaviour). The second group had MT only, and the third group was comprised of controls (delayed treatment). There were no significant differences on coping skills between the two active treatment groups at any follow ups. These results were replicated in a more recent study which also compared contingency management (CM) treatment to CBT and MT (Litt, Kadden, Kabela-Cormier, & Petry, 2008). In addition, both these studies demonstrated that the greater the quantity or frequency of use of adaptive coping strategies to prevent relapse, the greater the likelihood of remaining abstinent.

Similarly, in a study using a community sample (Rooke, Norberg, & Copeland, 2011), less use of adaptive coping strategies was related to unsuccessful attempts to quit cannabis. Successful quitters used coping strategies more frequently than unsuccessful quitters. Strategies used included active planning, and alternative methods for relaxing, or coping with distressing emotions. In summary, these treatment studies focusing on the role of using specific coping strategies to prevent relapse in alcohol and cannabis use disorders, demonstrate the importance of using adaptive coping strategies generally in terms of number and frequency. However the findings do not support specific training of adaptive coping skills as a component of a treatment intervention, for example CBT. It may well be that simply commencing treatment leads to the (increased) use of (previously learned) adaptive coping strategies for substance dependent individuals. In particular, more research is needed examining the use of coping strategies for longer follow up periods post-treatment. It may be more challenging for individuals being treated for substance use
disorders to continue using alternative adaptive coping strategies to deal with their distress over the long term, if they have significantly relied upon alcohol or substances to cope with stressors in their lives. Treatment for such individuals may be more effective by simultaneously focusing on the source(s) of distress as well as the use of alternative productive coping strategies.

**Coping strategies and social anxiety.** Cognitive behavioural models of social anxiety propose that socially anxious individuals use avoidant strategies to cope with their fear and anxiety in relation to social (interaction and performance) situations, in addition to using avoidant safety behaviours (Clark & Wells, 1995; Rapee & Heimberg, 1997). More specifically, the socially anxious individual exaggerates the likelihood of a social situation having a negative outcome. This is contributed to by their negative self-views with respect to their ability to control their anxiety in the situation, as well as of themselves as a social object. This leads to their expectation of the occurrence of social mishaps, or the overestimation of possible social costs, resulting in avoidant or safety behaviours (Hofmann, 2004).

The literature considering coping strategies and social anxiety is more limited compared to the amount of research involving coping strategies and alcohol/substance use problems/disorders. The association between the use of alcohol or cannabis to cope with social anxiety has been discussed earlier in this section. For practical reasons as well as the potential for shared variance with other similar factors, the use of alcohol or cannabis specifically as a coping strategy for social anxiety is not being considered in this thesis. This literature review focuses on associations between social anxiety and other coping strategies, as well as the role of coping strategies in relation to treatment of social anxiety disorder.

With respect to the relationship between coping strategies and social anxiety among children and adolescents, findings are mixed. Among children (mean age 10.1 years; age range 7-13 years) diagnosed with SAD (DSM-IV criteria), avoidant coping strategies (e.g., pretending to be sick) have been found to be used in response to 35% of socially distressing situations (Beidel et al., 1999). Among early adolescents, no significant relationship between either adaptive or maladaptive coping strategies and social anxiety has been found (Erath, Flanagan, & Bierman, 2007). Coping strategies were assessed by responses to
open-ended questions about a social interaction task. Responses were coded “problem-focused” or “self-directed”. The self-directed responses (e.g., distraction, relaxation) were hypothesised to be maladaptive due to the disruption to experiencing sensitive interpersonal responding. Social anxiety and self-directed coping strategies were each directly associated with increased peer victimisation for boys, indicating the relevance of maladaptive strategies early in life together with social anxiety symptoms being risk factors for ill-treatment by male peers, which in turn could be a risk factor for social anxiety disorder and other psychopathology. In summary, there is limited evidence for a direct association between maladaptive coping strategies and social anxiety in childhood and adolescence.

In student and community samples, a positive relationship has been shown to exist between maladaptive coping strategies and social anxiety. There is also some evidence for a negative relationship between adaptive coping strategies and social anxiety. Avoidant or dysfunctional coping strategies have been found to be strongly associated with social anxiety in student samples (Mairet, Boag, & Warburton, 2014; Thomasson & Psouni, 2010) as well as amongst members of social anxiety organisations (Thomasson & Psouni, 2010). However, it is important to note that Mairet and her colleagues were investigating coping strategies specifically used to cope with early maladaptive schemas experienced by students with high levels of social anxiety.

Thomasson and Psouni (2010) divided participants (students and individuals who were members of social anxiety organizations) into high and low social anxiety groups according to documented cut-off scores on strongly psychometric social anxiety measures. Dysfunctional coping strategies (denial, mental disengagement, behavioural disengagement and substance use) were positively related to the social anxiety scales. Problem-focused (adaptive) coping strategies (active coping, planning, positive reinterpretation and growth, suppression of competing activities, restraint coping) were negatively related to the social anxiety measures. Maladaptive coping strongly predicted both social anxiety measures. Problem-focused strategies were weaker negative, but still significant predictors of social anxiety. In summary, a positive association exists between maladaptive coping strategies and social anxiety in student and community samples, and a negative relationship between adaptive coping strategies and social anxiety also appears to exist.
Coping strategies, social anxiety disorder and treatment outcomes. As previously outlined in Chapter 1 of this thesis, cognitive-behavioural therapy (CBT) is the current preferred method of treatment for social anxiety disorder (SAD). Studies described in Chapter 1 of this thesis have demonstrated the superiority of CBT for SAD compared to other treatments including exposure group therapy (Hofmann, 2004), interpersonal therapy (Stangier et al., 2011), and psychodynamic therapy (Leichsenring et al., 2013).

Major components of CBT for SAD include cognitive restructuring (to change perceptions of threat and fear of negative evaluation), and removal of avoidance and safety behaviours (both forms of maladaptive coping) via repeated exposures to feared social situations of the affected individual (Hofmann, 2004; 2007). Furthermore, relaxation training (an adaptive coping strategy for managing anxiety symptoms, particularly somatic symptoms) is an optional component of CBT for SAD (Bogels et al., 2014). Thus, CBT incorporates the reduction of maladaptive coping strategies, as well as training in the use of adaptive coping strategies directly into its structured treatment for individuals with SAD.

A more specific field of research has developed in the last 10 years with respect to increasing our understanding of the use of, as well as problems with emotion regulation. This research has compared adaptive and maladaptive emotion regulation strategies in the prediction of various psychopathologies including social anxiety disorder. Some emotion regulation strategies can be characterised as emotion-focused coping strategies with specific strategies pertaining to social anxiety symptoms. Therefore, this research contains useful information about adaptive and maladaptive emotion-focused coping strategies for people with SAD.

Aldao et al. (2014) investigated the role of emotion regulation strategies in predicting social anxiety symptoms in a group of participants undergoing CBT treatment for SAD. They found that the use of adaptive engagement strategies (cognitive reappraisal and acceptance) had a weaker (negative) association with social anxiety symptoms compared to the use of maladaptive avoidance strategies (situational avoidance, situation modification, self-distraction, attempted suppression of visible signs of anxiety). In addition, the use of both types of strategies interacted and the direction of this interaction varied as a function of treatment phase. The 16 week treatment consisted of two phases of CBT; psychoeducation and cognitive restructuring training in weeks 1-7; and in vivo
exposures and continued use of cognitive restructuring skills in weeks 8-16. There was also a follow-up phase when every three months for one year post-treatment, participants provided ratings of their social anxiety symptoms for the previous seven days. More specifically, the findings indicated that social anxiety symptoms and the use of maladaptive avoidance strategies decreased during the two treatment phases, but they plateaued during the follow-up phase. A similar pattern was observed for the use of adaptive engagement strategies (i.e., they increased during the treatment phases, and plateaued during the follow-up phase). The results suggested that the optimal effects of adaptive strategies may arise when the socially anxious individual is using relatively equal levels of both maladaptive and adaptive strategies. The results also supported previous findings of Aldeo and her colleagues (cited in Aldeo et al., 2014) demonstrating a weaker association between the use of adaptive engagement strategies with SAD symptoms, compared to the use of maladaptive avoidance strategies. Thus future CBT treatment of social anxiety disorder may be enhanced by increasing the affected individual’s awareness of specific adaptive and maladaptive emotion-focused coping strategies relevant to social anxiety symptoms. Further research may assist in enhancing the efficacy of CBT. In summary, there appears to be a strong association between maladaptive emotion-focused strategies and social anxiety symptoms in individuals undergoing treatment and post-treatment. There also appears to be a weaker (negative) association between adaptive emotion-focused strategies and social anxiety during treatment and post-treatment for up to 12 months.

Coping strategies, social anxiety and substance use. Tran and Haaga (2002) previously considered in Chapter 3 with respect to the role of expectancies in the relationship between social anxiety and alcohol use disorders remains the only clinical study which also directly considered the role of coping strategies in this relationship. Three groups were compared: socially anxious individuals (SADs), SADs with alcohol abuse, and normal controls. On the self-reported measure of adaptive (problem-focused) coping strategies, both social anxiety groups had less adaptive coping strategies compared to controls in situations where alcohol was not accessible. In situations where alcohol was accessible, the comorbid group had the least adaptive coping strategies, followed by the other social anxiety group, and the controls had the highest adaptive coping strategies. These findings indicate that both clinical groups have less adaptive coping strategies
compared to normal controls. These findings also suggest that adaptive coping strategies moderate the relationship between social anxiety and alcohol use in situations where alcohol is accessible, in that SADs who have low levels of adaptive coping strategies may develop the comorbid condition (SAD and AUD).

In a non-clinical cross-sectional study, the role of perceived stress (to a specific noise-based stressor) was examined in the relationship between social anxiety disorder (SAD) symptoms and cannabis use disorder (CUD) symptoms (Buckner, Schmidt, Bobadilla, & Taylor, 2006a). Perceived stress moderated this relationship such that individuals with high SAD symptoms and low perceived (adaptive) coping in response to an unpredictable noise stressor were associated with higher CUD symptoms, compared to individuals with high SAD symptoms and high perceived coping. However, the relationship between SAD and CUD symptoms did not differ significantly for individuals with high perceived coping. The authors noted that the findings were limited by the use of a coping measure specifically designed for the particular stressor used in the study. Nevertheless, these two studies provide some support for the importance of adaptive coping strategies in the relationship between social anxiety and alcohol/cannabis use.

A more recent study (Gregg et al., 2014) examined the role of coping strategies as a mediator in the relationship between various psychopathologies and substance use in a student population. An inclusion criterion was problematic alcohol or substance use. Maladaptive coping (comprised of behavioural disengagement, venting, denial, self-distraction, and self-blame) mediated the relationship between anxiety and substance use. Although social anxiety was not specifically measured, the findings from this most recent research combined with findings described earlier which associate both social anxiety and substance use with maladaptive coping, suggest that maladaptive coping strategies also play an important role in the relationship between social anxiety and substance use behaviours.

Finally, there is some indirect evidence that replacing maladaptive cognitions about the use of substances to cope with stressors, with alternative adaptive coping strategies in the treatment of the SAD-SUD comorbidity, will at least reduce the severity of social anxiety symptoms (Courbasson and Nishikawa, 2010).
Interactive Roles of Personality, Alcohol Expectancies and Coping Strategies

This thesis examines the roles of three variables (drive sensitivity, alcohol expectancies and coping strategies) simultaneously in both a clinical and non-clinical sample. Some previous empirical research has considered the roles of two of these three variables simultaneously in particular in relation to drinking behaviour. Fewer studies have concurrently examined the roles of two of these variables in the relationship between social anxiety and substance use. A literature review of research that investigates interactions between these factors is relevant to this thesis to deepen our understanding of SAD-SUD comorbidity.

**Personality and alcohol expectancies.** To date, only one study appears to have simultaneously examined the roles of personality and alcohol expectancies in the relationship between social anxiety and drinking behaviour, in a predominantly student population (Booth & Hasking, 2009). The authors reported that social anxiety (fear) interacted with tension reduction (positive alcohol expectancy) to predict alcohol consumption. A negative relationship existed between social anxiety (fear) and drinking where tension reduction expectancies were low. Three three-way interactions also predicted drinking behaviour: a negative relationship existed between social anxiety (fear) and drinking for those with high BAS Drive and low tension reduction expectancies; a negative relationship existed between social anxiety (avoidance) and drinking for those with high BAS Drive and high tension reduction expectancies; and a positive relationship existed between social anxiety (fear) and drinking for those with low BAS Reward and high increased confidence expectancies. Notably, although these interactions were all significant, they did not significantly improve the regression model in this study (Booth & Hasking, 2009). Thus positive expectancies appear to moderate the relationship between social anxiety and alcohol use, whilst both positive expectancies and reward sensitivity interact with each other to moderate the relationship between social anxiety and alcohol use.

**Personality, alcohol expectancies and substance use.** Recently, the development of alcohol expectancies in childhood (mean age of children was 11 years) was examined using the revised RST model (Lopez-Vergara et al., 2012). Findings suggested that both reward and punishment sensitivity were associated with positive alcohol expectancies,
which predicted an increased likelihood of alcohol use. There was also evidence that one aspect of reward sensitivity (BAS Drive) was associated with negative alcohol expectancies which reduced the likelihood of alcohol use.

The roles of personality and cannabis expectancies have also been investigated simultaneously in a student population. Cannabis users reported higher positive cannabis expectancies and lower negative cannabis expectancies compared to student non-users (Simons & Arens, 2007). Simons and Arens (2007) also found that punishment sensitivity attenuated the relationship between positive expectancies and probability of cannabis use.

Other recent research with both clinical (treatment-seeking substance abusers) and non-clinical (student and young adult) cohorts, has shown that positive alcohol expectancies mediate the relationship between reward sensitivity and risky alcohol use (Gullo et al., 2010; Harnett, Lynch, Gullo, Dawe, & Loxton, 2013). With both student and treatment-seeking substance abuser cohorts, Gullo et al. (2010) demonstrated that high reward sensitivity predicted high positive alcohol expectancies, which in turn predicted low drink refusal self-efficacy, which in turn predicted risky drinking. Harnett et al. (2013) used a student sample and reported that high reward sensitivity predicted high positive expectancies which in turn predicted risky drinking. In summary, there is evidence that reward and punishment sensitivities interact with positive and negative alcohol expectancies in their relationships with both alcohol and cannabis use.

**Personality and coping strategies.** These two variables have not been considered simultaneously in research investigating social anxiety or research investigating the relationship between social anxiety and substance use. However, they have been examined simultaneously in their relationships directly with alcohol and substance use. The research shows that coping strategies play moderating and mediating roles in the relationship between drive sensitivity factors and risky alcohol or substance use.

Using a community sample, Feil and Hasking (2008) demonstrated that after controlling for age, depression, anxiety and stress, emotion-focused coping moderated the relationship between both BIS and BAS Fun-Seeking with risky alcohol use. A positive relationship between BAS Fun-Seeking and drinking existed for those with high levels of emotion coping, while a negative relationship existed between BIS and drinking for those with high levels of emotion-focused coping. In addition, Feil and Hasking (2008) reported
that avoidant coping interacted with BAS Fun Seeking to predict risky alcohol use. A positive relationship existed between BAS Fun Seeking and drinking for those with low avoidant coping. Notably, emotion-focused coping was comprised of both adaptive (e.g., seeking emotional support) and maladaptive (e.g., venting) coping strategies. This may explain how emotion-focused coping moderated both reward and punishment sensitivities in their relationships with risky alcohol use.

Ivory and Kambouropoulos (2012) simultaneously examined the roles of sensitivity to reward and punishment, coping strategies and alcohol use in a larger non-clinical sample of social drinkers. The relationship between punishment sensitivity (the “Fight, Flight, Freeze System” from revised Reinforcement Sensitivity Theory) and risky alcohol use was found to be mediated by both emotion-focused (adaptive) and avoidant (maladaptive) coping strategies (Ivory & Kambouropoulos, 2012). The researchers found that high punishment sensitivity predicted both emotion-focused coping and avoidant coping, and both forms of coping predicted risky drinking. In summary, both emotion-focused and avoidant coping strategies have been found to play mediating and moderating roles in the relationship between drive sensitivity and risky alcohol use.

A more recent study using a student population investigated the role of coping strategies in the relationship between drive sensitivity and both risky alcohol and risky substance use. Hundt et al. (2013) found that both emotion-focused (comprising both adaptive and maladaptive coping subtypes) and avoidant (maladaptive) coping mediated the relationship between punishment sensitivity and risky alcohol use (replicating the findings of Ivory and colleagues), whilst only avoidant coping mediated the relationship between punishment sensitivity and hazardous substance use.

These studies highlight the importance of emotion-focused and avoidant coping strategies, both of which mediate the relationship between punishment sensitivity and risky alcohol use, and avoidant coping which also influences the relationship between punishment sensitivity and hazardous substance use. Both coping strategies have also been found to interact with reward and punishment sensitivity to influence drinking behaviour. These findings highlight the need to assist people with high punishment sensitivity (e.g., people with social anxiety) and also high reward sensitivity (e.g., people with substance use
problems) to learn to use more adaptive coping strategies to reduce the likelihood of the development of substance use problems.

**Alcohol expectancies and coping strategies.** Only one study (Tran & Haaga, 2002) has simultaneously considered the roles of alcohol expectancies and coping strategies in the relationship between social anxiety and alcohol use with both clinical and community samples. The results from this study have been described earlier in this chapter (in the “Expectancies” and “Coping Strategies” sections). Overall, findings indicated moderating effects of positive alcohol expectancies and adaptive coping strategies, suggesting that socially anxious individuals who held high positive alcohol expectancies and low adaptive strategies were at risk of using alcohol problematically with the potential of also developing an alcohol use disorder.

**Alcohol expectancies, coping strategies and alcohol use.** The literature shows that alcohol expectancies and coping strategies have moderating or mediating roles in their relationship with drinking behaviour. Two groups of researchers have looked at the interactions between alcohol expectancies and coping strategies in relation to drinking behaviour. The earlier group of researchers led by M. Lynne Cooper examined positive expectancies in this relationship, as well as adaptive and maladaptive coping strategies. Cooper et al. (1988) established that maladaptive (avoidant) coping strategies were important variables contributing to alcohol use and abuse, but only for people who also held strong positive alcohol expectancies. These results were replicated by Cooper, Russell, Skinner, Frone, and Mudar (1992) who also found that stressors (stressful life events and or recent life problems) predicted alcohol use and abuse in individuals with high levels of both positive expectancies and maladaptive coping. In their research investigating motivational pathways to alcohol use and problems, Cooper et al. (1995) replicated these results once more with respect to positive expectancies, maladaptive coping strategies and alcohol use and problems, for both adolescent and adult groups.

A second group of more recent studies considered interactions between alcohol expectancies and coping strategies in relation to drinking behaviour (alcohol volume and frequency of use per annum) (Hasking & Oei, 2002a, 2004, 2007). In their studies, Hasking and Oei also controlled for pre-existing psychopathology and severity of alcohol dependence for all samples in the regression analyses.
In the first study, Hasking and Oei (2002a) used TOTAL expectancies (positive and negative) and TOTAL coping strategies (adaptive and maladaptive) with both community and clinical samples. There was no interaction between expectancies and coping strategies in predicting drinking behaviour in the community sample. Both variables interacted to predict frequency of alcohol consumed in the clinical sample, with both high and low levels of coping combined with high alcohol expectancies being related to higher frequencies of alcohol consumption (indicating that expectancies were the more salient factor in this relationship). These results suggest that both expectancies and coping strategies play a more important role together in predicting drinking behaviour for alcohol-dependent individuals. However, as the researchers failed to examine more specific aspects of expectancies and coping, the findings are limited.

In their next study with a sample of community drinkers, Hasking and Oei (2004) found that positive expectancies interacted with an avoidant coping strategy (seeking social support for emotional reasons) to predict alcohol volume, with this relationship being stronger for those with lower positive expectancies. However, the avoidant (or maladaptive) coping strategy was classified as such by the authors based on confirmatory factor analysis they had conducted with both community drinkers and alcohol-dependent samples (Hasking & Oei, 2002b), whereas this coping strategy is usually categorised as adaptive (e.g., Ivory & Kambouropoulos, 2012).

In the third study with an alcohol-dependent sample, conducted by Hasking and Oei (2007), both volume and frequency of annual alcohol use were predicted by a maladaptive coping strategy (venting) which interacted with negative alcohol expectancies. A strong positive relationship existed between both volume and frequency of alcohol use and venting, for those with strong negative alcohol expectancies.

More recent research has demonstrated that positive alcohol expectancies (increased confidence and tension reduction) mediate the relationship between avoidant coping strategies and drinking behaviour (Hasking et al., 2011). In summary, the literature which examines alcohol expectancies and coping strategies simultaneously in relation to drinking behaviour indicates that one or both of these two variables act as moderators or mediators with the other in their relationship with alcohol use. These relationships confirm that
substance use behaviour is at least partly comprised of a complex interplay between alcohol expectancies and coping strategies.

**Personality, alcohol expectancies and coping strategies.** As far as can be ascertained, no studies appear to have simultaneously examined the roles of these three biopsychosocial variables in the relationship between social anxiety and alcohol/substance use. The importance of drive sensitivity, alcohol expectancies and coping strategies with respect to social anxiety and problematic substance use has been described in this chapter. By considering their roles simultaneously in both non-clinical and clinical samples, it is hoped to gain further understanding about social anxiety disorder (SAD) and about co-occurring SAD and substance use disorder (SUD), that is, the SAD-SUD comorbidity, as well as ideas as to how to improve treatment outcomes for affected individuals.

**Chapter 3 Summary**

The SAD-SUD comorbidity is highly prevalent with SAD preceding SUD in the majority of cases. Affected individuals may experience serious impairment with respect to education, income, health, and interpersonal relationships. Despite the high co-occurrence rates, no established treatment exists. Emerging findings suggest that an integrated treatment may be the most efficacious. The SAD-SUD comorbidity has also been extensively researched partly as a result of inconsistent findings. Most research relies on the self-medication model to explain the nature of the comorbidity, yet contradictions remain. This thesis relies on the shared vulnerability model, and utilizes one of the current methods of researching the SAD-SUD comorbidity, that is, exploring the roles of other important factors. Three such factors, personality (drive sensitivity), alcohol expectancies and coping strategies may also be shared vulnerabilities of both social anxiety and substance use disorders. These factors also represent a biopsychosocial framework within which this research is being conducted. Literature reviews of each of the three factors and their combinations, confirm their importance in relation to social anxiety and substance use. However, inconsistencies remain and causal relationships are hard to extrapolate. Gaps in the literature lead to the need to simultaneously examine the strengths of relationships of these three factors, as well as their moderating (and mediating) effects in the relationship between social anxiety and substance use in order to inform more effective treatments.
Chapter 4: This Thesis

Rationale for This Research

There have been numerous findings regarding the significant associations of personality (sensitivity to reward and punishment), alcohol expectancies and coping strategies with social anxiety and with substance use, particularly alcohol use. However, there is a paucity of literature examining one or more of these three factors in the relationship between social anxiety and substance use in non-clinical samples, and in the SAD-SUD comorbidity in clinical samples. In addition, most research has focused on the relationship between social anxiety and alcohol use, or SAD and alcohol use disorder (AUD) where clinical samples have been used. The importance of cannabis use by socially anxious individuals has emerged in more recent research, for example, Buckner et al. (2008a).

Only two non-clinical studies have looked at personality in the relationship between social anxiety and alcohol or substance use (Booth & Hasking, 2009; Nicholls et al., 2014). Booth and Hasking (2009) found that after controlling for age and gender, only one type of reward sensitivity (BAS fun-seeking) predicted alcohol consumption (but no other personality or social anxiety variables did). Using latent class analysis, Nicholls et al. (2014) found that socially anxious individuals with problematic alcohol use were highly sensitive to both reward and punishment.

Non-clinical studies investigating the role of expectancies in the relationship between social anxiety and alcohol use have generally produced inconsistent findings, at least in part due to methodological differences. Emerging evidence indicates that both positive and negative alcohol expectancies moderate the relationship between social anxiety and substance use (Carrigan et al., 2008). Only two clinical studies have investigated the role of expectancies in the SAD-AUD comorbidity (Ham et al., 2002; Tran & Haaga, 2002). Significant differences were found between clinical and control groups with the clinical groups having higher levels of positive and negative alcohol expectancies.

Only one clinical study has investigated the role of coping strategies in the SAD-SUD relationship and that study only examined adaptive coping strategies (Tran & Haaga, 2002). Findings suggested that adaptive coping strategies moderate the relationship between SAD and AUD. That finding was supported in a non-clinical study which
examined symptoms of cannabis use disorder (CUD) and SAD (Buckner et al, 2006a). However, the findings of Buckner and her colleagues are limited due to the use of a specific coping scale designed to measure the specific stressor used in the study.

Only two of the above studies simultaneously considered the roles of two of the three important factors in the SAD-AUD relationship. Booth and Hasking (2009) examined the roles of personality and expectancies in a non-clinical sample, whilst Tran and Haaga (2002) considered the roles of expectancies and adaptive coping with a clinical sample. No studies have simultaneously considered the roles of personality, expectancies and coping strategies in the SAD-SUD relationship despite the importance of all three factors to both SAD and SUD as demonstrated above.

Overall Aims of This Research

There were two overall aims of this research:

1. To increase understanding of the relationship between SAD and SUD by considering the roles of personality, alcohol expectancies and coping strategies in both clinical and non-clinical samples.
2. To inform treatment for individuals with the SAD-SUD comorbidity, as well as improve prevention and education strategies in relation to this comorbidity.

The roles of the three biopsychosocial variables: drive sensitivity; alcohol expectancies; and coping strategies, in the SAD-SUD relationship, were investigated in both a clinical and a non-clinical sample. The two study approach was utilised to address shortcomings in the existing literature, including different methodologies used in non-clinical studies (resulting in lack of clarity about conclusions that can be drawn), and a dearth of information about the SAD-SUD comorbidity in clinical samples. Using a non-clinical sample provided the opportunity to obtain data from a large group of people, and combined with collection of data from a clinical sample, allowed for enriched conclusions to be drawn from meaningful findings.
The present research was unique in the following ways:

1. It examined the relationship between social anxiety and risky alcohol use as well as the relationship between social anxiety and risky cannabis use in a non-clinical sample (Study 1).

2. It simultaneously examined three important shared vulnerability factors (drive sensitivity, alcohol expectancies, and coping strategies) in the relationship between social anxiety symptoms and risky alcohol use, as well as the roles of these three factors in the relationship between social anxiety symptoms and risky cannabis use in a non-clinical sample (Study 1).

3. It simultaneously examined the same three important shared vulnerability factors in the SAD-SUD comorbidity with a clinical sample (Study 2).

4. It compared SAD with the SAD-SUD comorbidity by examining differences between groups on the three shared vulnerability factors (Study 2).

5. The same measures for the three shared vulnerability factors, social anxiety and risky alcohol and cannabis use, were used in both studies, that is, with both clinical and non-clinical samples in order to increase our understanding about the relationship between social anxiety and substance use.

6. By way of exploration, factors that predict SAD in both studies (with both clinical and non-clinical samples) were examined in order to further our understanding of the nature of SAD.

In addition to the above-mentioned variables (social anxiety, risky alcohol use, risky cannabis use, reward and punishment sensitivity, alcohol expectancies, and coping strategies), two other important variables were included in both studies. First, depressive symptoms were measured given the high comorbid prevalence rates of depression with both SAD and SUD (Fehm et al., 2008; Grant et al., 2006a), as well as the high comorbidity between major depressive disorder and both the SAD-AUD comorbidity and the SAD-CUD comorbidity (Buckner et al., 2012b; Schneier et al., 2010). Second, fear of negative evaluation, a core feature of social anxiety was measured. This aspect of social anxiety consistently demonstrates a positive association with alcohol problems (Morris et al., 2005), and its significance has been reflected in the current DSM’s definition of SAD (American Psychiatric Association, 2013). However, no hypotheses in relation to
depression or fear of negative evaluation were put forward. The inclusion of these two variables was intended to further our understanding of the nature of the relationship between SAD and SUD.

Finally, it would have been useful to also assess the role of cannabis expectancies in relation to the SAD-CUD comorbidity. However, for practical reasons this was not feasible. In order to obtain sufficient numbers of clinical participants, and in order to be able to use the same measures for the variables in both clinical and non-clinical samples, compromises had to be made when selecting the instruments. The development of alcohol expectancies in early childhood (Miller, Smith, & Goldman, 1990) contributed to the decision to assess the role of alcohol expectancies in both the SAD-AUD and SAD-CUD relationships/comorbidities.

The next two chapters contain the aims and hypotheses, method, results and discussion of Study 1 and Study 2 respectively.
Chapter 5: Study 1 – The roles of personality, expectancies and coping strategies in the relationship between social anxiety and substance use in a non-clinical sample.

Aims and Hypotheses

Study 1 focused on the relationships between personality factors (drive sensitivity), alcohol expectancies and coping strategies with social anxiety and risky substance use in a non-clinical sample. Previous studies have shown that these three variables contribute to the relationship between SAD and SUD. Most research in this area has focused on the relationship between social anxiety and alcohol use, with some recent research also considering the relationship between social anxiety and other substance use, in particular cannabis use. Research about the moderating roles of each of the three variables is insufficient or mixed. In any event, findings indicate that each of these variables on their own, are insufficient in explaining the relationship between social anxiety and substance use.

Aim 1. To increase understanding of the nature of the SAD-SUD relationship in non-clinical samples.

Due to inconsistencies in the literature about the existence of and/or direction of this relationship amongst non-clinical samples, no hypotheses were offered concerning the relationship between social anxiety and alcohol use, or about the relationship between social anxiety and cannabis use. However, these relationships were explored in order to increase our understanding of the nature of them in accordance with the first aim of this study.

Research question 1. Is there a significant relationship between social anxiety and alcohol use?

Research question 2. Is there a significant relationship between social anxiety and cannabis use?

Aim 2. To increase understanding of the roles of personality (reward and punishment sensitivity), alcohol expectancies and coping strategies in the relationship between SAD and SUD.
Hypotheses. Two sets of hypotheses were put forward. The first set focused on relationships among the variables, including predictors of risky alcohol use, risky cannabis use, and severity of social anxiety. The second set focused on the interactions of personality, alcohol expectancies and coping strategies with social anxiety to predict risky alcohol use and risky cannabis use.

With respect to the relationship between social anxiety and predictor variables, as well as between substance use and predictor variables, it was hypothesised that:

(1) Social anxiety will be positively related to sensitivity to punishment, both positive and negative alcohol expectancies, and maladaptive coping strategies, but negatively related to adaptive coping strategies, and that all these factors will predict severity of social anxiety, in addition to depression, risky substance use and fear of negative evaluation.

(2) Both risky alcohol use and risky cannabis use will be positively associated with sensitivity to reward, both positive and negative alcohol expectancies and maladaptive coping strategies, but negatively related to adaptive coping strategies, and that, in addition to depression and social anxiety phenomena, all these factors will predict severity of risky alcohol use and risky cannabis use.

With respect to interactions between the moderating variables and social anxiety in predicting risky alcohol use and risky cannabis use, it was hypothesised that:

(3) High levels of social anxiety will be associated with an increased likelihood of risky alcohol/cannabis use when both sensitivity to reward and punishment are high; both positive and negative alcohol expectancies are high, maladaptive coping strategies are high, and adaptive coping strategies are low.

(4) High levels of social anxiety will be associated with a decreased likelihood of risky alcohol/cannabis use when sensitivity to reward is low, sensitivity to punishment is high, negative alcohol expectancies are high, and maladaptive coping strategies are lower/adaptive coping strategies are higher.
Method

Participants. Participants comprised 298 individuals recruited from first year psychology students and general community members. Due to the anonymity of the questionnaire, precise numbers of students and community members could not be ascertained. However, some information identifying students could be obtained in two ways: first, the online questionnaire was initially posted with two separate links – one for first year psychology students at Swinburne University and one for other community members; and second, from responses to two demographic questions which both had a response option of “student”. The questions were: “What is your usual occupation?” and “What is your current employment status?” From these two sources of information, at least 225 participants (75.5% of the total number of participants) could be classified as students. From the student participants at least 164 (72.9%) could be definitively classified as first year students.

Age. All participants were aged between 18 and 63 years of age ($M = 24.59; SD = 10.17$). The commonest age of participants was age 19 (28.9%) followed by age 18 (20.1%).

Gender. There were 82 male (27.5%) and 216 female participants (72.5%).

Country of Birth. Although four participants did not respond to this question, the majority were born in Australia (79.2%), followed by 7.0% who were born in Europe.

Current Relationship Status. The majority of participants described themselves as “never married” (58.4%), followed by 22.1% who were in a “steady relationship (not living together)”, and 17.1% who were “married/living with partner”.

Highest Level of Education Completed. A majority of participants had completed high school (60.1%), followed by 18.8% who had a university qualification, and 15.8% who had completed a “TAFE Diploma/Certificate/Trade Qualification”. One person did not respond to this question.

Usual Occupation. This question provided for a “free text” response and 7% (21 participants) did not respond. Of the remainder the largest percentage (38.3%) described themselves as “student”, 37.2% listed occupations which were categorised as “other”, 5.7% were in “sales” and 5% were in “professional” occupations.
**Current Employment Status.** The highest percentage of participants described themselves as “employed – part-time/casual (50.0%), followed by “student” (29.9%), “employed – full-time” (9.7%) and “unemployed” (9.1%). Two participants did not respond to this item.

**Materials.** In addition to providing demographic information described above, all participants completed the following self-report measures:

**Social Anxiety Measures.** The Liebowitz Social Anxiety Scale (LSAS) (Liebowitz, 1987) is a commonly used instrument for the assessment of social anxiety disorder (SAD). It comprises 24 items which assesses levels of fear/anxiety and avoidance across a range of social interaction (e.g., “Participating in small groups”) and performance situations (e.g., “Working while being observed”). Each item is rated on a four-point Likert scale for both fear/anxiety and avoidance. For fear/anxiety, the ratings range from 0 (no fear or anxiety in this situation) to 3 (severe fear or anxiety in this situation). For avoidance, the ratings range from 0 (never avoid this situation) to 3 (usually avoid this situation). As well as separate scores for fear and avoidance of both social interaction and performance situations, the LSAS provides a total score. Because no hypotheses in this study related to the distinction between fear and avoidance, only the LSAS total score was used.

A cut-off total score of 30 has been shown to discriminate between participants with and without SAD, whilst a cut-off of 60 has been used to further classify participants with generalised SAD (Mennin et al., 2002; Rytwinski et al., 2009).

The LSAS has been found to have strong psychometric properties. Heimberg et al. (1999) evaluated 382 patients with SAD and found Cronbach’s alpha values ranging from .81 for ratings of fear in performance situations to .96 for the total score. There was also good evidence of both convergent and discriminant validity, to the extent that the LSAS tends to correlate strongly with other measures of SAD and less strongly with measures of depression. However, high correlations between the fear and avoidance subscales indicate they may not be sufficiently unique (Heimberg & Holaway, 2007; Heimberg et al., 1999). In the present sample, the Cronbach’s alpha value for the LSAS Total score was .96.

The Brief Fear of Negative Evaluation Scale (BFNE) (Leary, 1983) is a 12 item questionnaire which is commonly used to assess fear of negative evaluation, a fundamental feature of social anxiety. It is an abbreviated version of the Fear of Negative Evaluation Scale (Leary, 1995).
Scale, a 30 item scale requiring true or false as a response to each statement (Watson & Friend, 1969). Items (e.g., “I often worry that I will say or do the wrong things”) are scored on a five-point Likert scale from 0 – “Not at all characteristic of me” to 4 – “Extremely characteristic of me”. The BFNE has demonstrated good psychometric properties and increased sensitivity compared to the Fear of Negative Evaluation Scale (Weeks et al., 2005). Confirmatory factor analysis has shown that the reverse worded items in the BFNE comprise a separate factor to the straightforwardly worded items (Rodebaugh et al., 2004). This study used the revised BFNE, known as the BFNE-II, which contains 12 items with the reverse worded ones having been changed into straightforward worded items (Carleton et al., 2006). The BFNE-II has produced excellent internal consistency with a Cronbach’s alpha of .95 compared to .89 for the BFNE (Carleton et al., 2006). In the current study the Cronbach alpha coefficient for BFNE-II was .97.

**Personality Factors.** The original Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) (Torrubia et al., 2001) is a 48 item questionnaire consisting of two scales representing the motivational systems proposed by Gray’s personality model: the sensitivity to punishment scale (SP) measures individual differences on BIS functioning, and the sensitivity to reward scale (SR) measures individual differences on BAS functioning. It has shown good reliability and validity in several studies.

For the current study, a shortened version of the SPSRQ was used based on a factor solution obtained after applying confirmatory factor analysis (CFA) to the original questionnaire (O’Connor, Colder, & Hawk, 2004). More recent CFA has confirmed the same two factor model with acceptable fit in the French translation of the abbreviated SPSRQ (Lardi, Billieux, d’Acremont, & Linden, 2008). The 35 item questionnaire comprises 18 SP items and 17 SR items. SR items include topics such as money, sex partners, social events, power, and sensation-seeking, describing situations in which people could do something to obtain rewards (e.g., “Do you often do things to be praised?”). SP items measure behavioural inhibition in general situations involving the possibility of aversive consequences of novelty; and worry or cognitive processes produced by the threat of punishment or failure (e.g., “Are you easily discouraged in difficult situations?”).
Respondents are required to respond “yes” or “no” to each item. Scores are summed (“yes” responses are scored 1), yielding a total score for each scale. In the current study the Cronbach alpha coefficient for SR was .80 and for SP it was .87.

**Alcohol Expectancies.** The Alcohol Expectancies Scale (AE) (Leigh & Stacy, 1993) measures both desired and undesired expected outcomes of alcohol use. It was designed to be completed by both drinkers and non-drinkers. The latter are instructed to complete it on the basis of what they think would happen if they did drink. The AE was designed and tested with more than 1,000 college students. It consists of 34 items rated on a six-point Likert scale. Ratings range from 1 (No chance) to 6 (Certain to happen). The items are introduced by the phrase: “When I drink alcohol”. Factor analysis initially produced two constructs which represented positive and negative consequences of drinking. Further analyses resulted in each factor comprising four subscales. The positive expectancies (AEP) subscales are: social facilitation (e.g., “I am more accepted socially”), fun, sexual enhancement, and tension reduction. The negative expectancies (AEN) subscales are: social (e.g., “I become aggressive”), emotional, physical, and cognitive/performance. Good reliability was demonstrated, with Cronbach’s alphas of 0.94 and 0.88 for the positive and negative expectancy factors respectively. Cronbach’s alphas for the eight subscales ranged from .73 to .91. Test-retest reliability ($N = 120$) was 0.87, and good discriminant and convergent validity were also demonstrated (Leigh & Stacy, 1993). Recent research using the AE produced Cronbach’s alphas of .92 and .84 on the positive and negative expectancies scales respectively, whilst Cronbach’s alphas for the eight subscales varied between .58 and .91 (Monk & Heim, 2013).

In this study, the two factor structure of the AE was used: first, as the hypotheses did not distinguish between different types of positive and negative expectancies; and second, to maximise the effectiveness of the method used to deal with missing data (EM imputation method – see section on Preliminary Data Analysis in Results). In the current study the Cronbach alpha coefficient for AEP was .96 and for AEN it was .89.

**Coping.** The Brief COPE Inventory (BC) (Carver, 1997) is a 28 item questionnaire which explores the ways in which individuals cope with stressful events. It is an abbreviated version of the 60 item COPE (Carver, Scheier, & Weintraub, 1989). The BC has 14 scales representing a range of active and passive coping styles: self-distraction;
active coping; denial; substance use; using emotional support; using instrumental support; behavioural disengagement; venting; positive reframing; self-blame; planning; humour; acceptance; and religion. Each scale contains two items (e.g., “I turn to work or other activities to take my mind off things”), which are scored on a 4-point Likert scale (from 1 – “I usually don’t do this at all” to 4 – “I usually do this a lot”). The BC has been found to have good validity and reliability (Carver, 1997). Data for validity and reliability were collected from convenience samples of community residents participating in a study of recovery after Hurricane Andrew (Carver, 1997). Cronbach’s alpha for the 14 scales ranged from .50 (venting) to .90 (substance use), with all but three of the scales having Cronbach’s alphas higher than .64. The BC scales have also been grouped into two broader dimensions of adaptive (comprised of the active coping, using emotional support, using instrumental support, positive reframing, planning, humour, acceptance and religion scales), or maladaptive coping strategies (comprised of the denial, substance use, behavioural disengagement, self-distraction, venting and self-blame scales), producing Cronbach’s alphas of .83 for adaptive coping strategies and .75 for maladaptive coping strategies (Hastings & Brown, 2002). The current study also used the two dimensions of adaptive coping (BCA) and maladaptive coping (BCM). The two items comprising the substance use subscale were removed from the maladaptive coping strategies scale to eliminate potential confounding of maladaptive coping strategies with other relevant variables, that is, alcohol expectancies and risky alcohol/cannabis use. Cronbach alpha coefficients were obtained of .84 for BCA and .72 for BCM.

**Depression.** Due to copyright restrictions for the depression measure used in Study 2 (the Beck Depression Inventory), a different depression measure was used for this larger non-clinical study (Study 1). The Centre for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) is a commonly used freely available 20 item self-report scale designed to measure depressive symptoms in the general population. It asks individuals to rate feelings (e.g., “I felt depressed”) or behaviours (e.g., “I had crying spells”) during the past week on a four-point Likert scale ranging from 0 (rarely) to 3 (most of the time). The CES-D has been validated in a wide range of demographic populations, for example, older populations (Beekman et al., 1997), and primary care clinic patients (Zich, Attkisson, & Greenfield, 1990). It is highly correlated with the BDI-II (Hicks & McCord, 2012). The
CES-D shows good reliability with high alpha levels e.g., .88 to .95 (Zhang, Sun, Kong, & Wang, 2012). In the current study the Cronbach alpha coefficient for CES-D was .92.

A cut-off total score of 15 has been shown to discriminate between participants with and without depression, scores between 16 and 26 have been shown to indicate mild depression, and scores of 27 or higher have been used to indicate major depression (Geisser, Roth, & Robinson, 1997; Zich et al., 1990).

**Substance Use.** The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) (Ali et al., 2002) is a questionnaire which screens for problem or risky substance use. It was developed for the World Health Organisation (WHO) by an international group of researchers to detect substance use problems in primary care patients. The current version of the ASSIST (used in this study) was produced after testing on 1,047 participants, 350 from drug treatment facilities and 697 from Primary Health care (Humeniuk et al., 2008). Eight questions (items) are asked about ten substances: tobacco, alcohol, cannabis, cocaine, amphetamine type stimulants, inhalants, sedatives, hallucinogens, opioids and “other drugs”. The questions ask about frequency of use and associated problems for each substance. Depending on the responses to question 1 for each substance (lifetime use), and/or question 2 for each substance (use in the past 3 months), some questions are skipped. Responses to questions 2 to 5 are rated on a five-point Likert scale from “never” to “daily/almost daily”. Responses to questions 6 to 8 are rated on a three-point Likert scale from “never” to “yes in past 3 months”. Only responses to items 2 to 7 for each substance are scored.

The ASSIST shows good face, concurrent, construct and discriminative validity (Ali et al., 2002; Humeniuk et al., 2008). Cut-offs (for low, medium and high risk use) were determined using receiver operating characteristic (ROC) analysis. The Cut-offs for no/low risk are 0-10 for alcohol and 0-3 for all other substances. Cut-offs for moderate risk are 11-26 for alcohol, and 4-26 for all other substances. The high risk cut-off for all substances is 27 (and higher). The ASSIST discriminates more effectively between low and medium risk, rather than between medium and high risk (Humeniuk et al., 2008). Due to the question and stem (cumulative) structure of the ASSIST, Cronbach’s alpha coefficients were not obtained.
Procedure. After obtaining ethics approval (see Appendix A), the eight questionnaires were amalgamated and formatted for use online (see Appendix B). The order of completion of scales was as follows: demographics; LSAS; SPSRQ; AE; BC; CES-D; BFNE-II; ASSIST. The amalgamated questionnaire took approximately 20 -30 minutes to complete. The online questionnaire was distributed via a number of methods including a university website, Facebook and email contacts (snowballing). Participants self- selected by completing the online survey. First year psychology students received a partial course credit for their participation.

Consent to participate in the study was implied by completion of the questionnaires. The anonymity of the participants was maintained with no information being gathered which could identify them. Upon retrieval or downloading of the questionnaires, participants were informed in the explanatory statement of the voluntary and anonymous nature of the survey (see Appendix C). Participants could withdraw at any time prior to submission of the questionnaire by not completing the questionnaires and/or leaving the URL address. The researchers collected the data from a secure webpage.

Results

Preliminary Data Analysis

Missing data. The statistics package IBM SPSS Statistics 20 was used for all data analyses. Preliminary data analyses were conducted on all measures. SPSS Descriptives was run to ascertain what percentage of values was missing for each continuous variable (excluding “age” as it had no missing data). LSAS Anxiety, LSAS Avoidance and LSAS Total were the only variables with over 5% missing values. Cases with over 20% of missing LSAS values were deleted reducing the number of participants from 319 to 298. As the correlation between the LSAS Anxiety and LSAS Avoidance subscales was high (.887), only the LSAS Total (LSAS) variable was used.

The Expectation Maximisation (EM) imputation method available on SPSS was used to replace missing data on all relevant measures (Tabachnick & Fidell, 2007). The largest subscales were used for imputing missing values for: AE (four categorised positive subscales were combined into an AE Positive Total (AEP) subscale, and four categorised
negative subscales were combined into an AE Negative Total (AEN) subscale); and for BCOPE (eight categorised adaptive subscales were combined into a BCOPE Adaptive (BCA) subscale, and five maladaptive subscales were combined into a BCOPE Maladaptive (BCM) subscale). Little’s MCAR test was used to check if missing values were completely at random. Significant results indicating the missing values may not be random were found on four measures: LSAS ($p < .001$), ASSIST Amphetamine ($p < .001$), ASSIST Hallucinogen ($p < .001$) and ASSIST Other ($p = .003$). Data Analyses (correlations) were run on two datasets, the first without the missing values which was compared with the second containing imputed values. No significant differences in the results were found. Therefore the missing values on these four measures were also replaced with imputed values using the EM technique. Using the EM technique also ensured that all data values were within the required range.

**Descriptive statistics.** Table 1 shows means, standard deviations, score ranges, skewness and kurtosis for each of the continuous variables.
Table 1

Means, Standard Deviations, Score Ranges, Skewness and Kurtosis Values for all Continuous Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Score Range</th>
<th>Skewness (SE=0.14)</th>
<th>Kurtosis (SE = 0.28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>24.59</td>
<td>10.17</td>
<td>18-63</td>
<td>1.92</td>
<td>2.71</td>
</tr>
<tr>
<td>LSAS</td>
<td>46.84</td>
<td>26.30</td>
<td>0-135</td>
<td>0.55</td>
<td>0.29</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>24.79</td>
<td>13.26</td>
<td>0-48</td>
<td>0.05</td>
<td>-0.91</td>
</tr>
<tr>
<td>CES-D</td>
<td>20.71</td>
<td>12.04</td>
<td>0-51</td>
<td>0.50</td>
<td>-0.50</td>
</tr>
<tr>
<td>SR</td>
<td>7.53</td>
<td>3.91</td>
<td>0-17</td>
<td>0.32</td>
<td>-0.31</td>
</tr>
<tr>
<td>SP</td>
<td>10.58</td>
<td>4.83</td>
<td>0-18</td>
<td>-0.27</td>
<td>-0.87</td>
</tr>
<tr>
<td>AEP</td>
<td>77.35</td>
<td>19.17</td>
<td>19-114</td>
<td>-0.93</td>
<td>1.38</td>
</tr>
<tr>
<td>AEN</td>
<td>44.54</td>
<td>12.72</td>
<td>15-90</td>
<td>0.02</td>
<td>0.91</td>
</tr>
<tr>
<td>BCA</td>
<td>40.85</td>
<td>7.52</td>
<td>16-64</td>
<td>-0.14</td>
<td>0.97</td>
</tr>
<tr>
<td>BCM</td>
<td>22.87</td>
<td>4.67</td>
<td>10-40</td>
<td>0.41</td>
<td>0.56</td>
</tr>
<tr>
<td>ASSIST Alcohol</td>
<td>9.34</td>
<td>8.27</td>
<td>0-37</td>
<td>1.14</td>
<td>0.76</td>
</tr>
<tr>
<td>ASSIST Cannabis</td>
<td>3.05</td>
<td>7.14</td>
<td>0-39</td>
<td>3.01</td>
<td>9.15</td>
</tr>
</tbody>
</table>

Note. N = 298. Only descriptive statistics for ASSIST Alcohol and ASSIST Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; CES-D = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; ASSIST Alcohol = risky alcohol use; ASSIST Cannabis = risky cannabis use.

Normality, linearity and homoscedasticity. As seen in Table 1, the skewness figures for Age, ASSIST Alcohol (Alcohol) and ASSIST Cannabis (Cannabis) indicated that these variables were not normally distributed. Inspection of normal probability plots confirmed that these three variables had extreme positive skews.

The strong positive skew of Age indicated that most participants were young adults. A logarithm transformation (LG10) applied to this variable reduced its skewness to some extent (to 1.51). Tabachnick and Fidell (2007) indicate that in large samples, the impact of
skewness and kurtosis may not be as significant compared to smaller sample sizes. Therefore, the transformed Age variable was retained for use in data analyses.

For the proposed data analysis procedure (multiple regression), the substance use variables as DVs were to be regressed onto the IVs (predictor variables). Attempts were made to transform Alcohol and Cannabis using square root transformations; however, Cannabis remained strongly skewed. Therefore, the analysis procedure was changed to logistic regression, which allows for the prediction of discrete outcomes from continuous or discrete predictor variables.

For reasons of parsimony as well as a more balanced distribution of frequency of numbers within each group, it was decided to dichotomise each substance variable into a low risk group and a moderate to high risk group, rather than having three groups. In addition, the ASSIST discriminates more effectively between low and medium risk, rather than between medium and high risk (Humeniuk et al., 2008). Therefore, participants who scored between 0-10 on alcohol use in the ASSIST measure were classified “low risk” and all other participants (who scored between 11 and 27 on alcohol use) were classified “moderate-high risk”. The applicable ASSIST cut-offs for cannabis use were 0-4 (low risk) and 5-27 (moderate-high risk).

Scatterplots were produced to examine whether the assumptions of linearity and homoscedasticity were met. As the DVs were dichotomised there was no violation of these assumptions between DVs and the continuous predictor variables (Tabachnick & Fidell, 2007). Upon inspection of scatterplots between continuous variable pairs, these assumptions were not violated.

**Multivariate outliers and multicollinearity.** Linear regressions were run on the dichotomous DVs to test for multivariate outliers. Mahalanobis distance values were inspected and four multivariate outliers were identified as being higher than the critical value \( \chi^2 = 31.264, df=11 \), using the \( p < .001 \) criterion (Tabachnick & Fidell, 2007). Upon closer inspection of the outliers and comparing them to lower value Mahalanobis distance cases, it was ascertained that these four cases included both maximum and minimum scores for some measures and therefore were unlikely to contain reliable responses. Therefore, these outliers were removed resulting in a final sample size of \( N= 294 \).
To ensure that no predictor variables were highly correlated, Tolerance and VIF (Variation inflation factor) values were inspected after running the linear regression analysis to test for outliers. All Tolerance values exceeded .10, and no VIF values were over 10, indicating this assumption was not violated.

**Descriptive statistics for final sample.** Three of the four multivariate outlier cases removed were 19 year old males and in the low risk alcohol and cannabis groups, whilst the fourth was a 19 year old female in the medium to high risk alcohol and cannabis groups. The removal of these cases resulted in a slightly higher female to male ratio of the entire sample (73.1% female; 26.9% male), a slightly higher mean age ($M=24.67$; $SD=10.22$), and slightly higher moderate/high risk compared to low risk substance use ratios (alcohol: 33.7% moderate/high risk; 66.3% low risk; and cannabis: 20.7% moderate/high risk; 79.3% low risk). One of the outlier cases identified as Aboriginal/Torres Strait Islander, therefore there were no cases identified as such in the final sample. There were no other notable changes to demographic factors as a result of the removal of these four cases. Table 2 shows means, standard deviations, score ranges, skewness and kurtosis for the continuous measures after the removal of the four multivariate outliers.
Table 2

Descriptive Statistics for all Continuous Measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Min/Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSAS</td>
<td>47.48</td>
<td>25.90</td>
<td>0-135</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>24.88</td>
<td>13.12</td>
<td>0-48</td>
</tr>
<tr>
<td>CES-D</td>
<td>20.67</td>
<td>11.99</td>
<td>0-51</td>
</tr>
<tr>
<td>SR</td>
<td>7.48</td>
<td>3.83</td>
<td>0-17</td>
</tr>
<tr>
<td>SP</td>
<td>10.57</td>
<td>4.78</td>
<td>0-18</td>
</tr>
<tr>
<td>AEP</td>
<td>77.50</td>
<td>18.44</td>
<td>19-113</td>
</tr>
<tr>
<td>AEN</td>
<td>44.43</td>
<td>12.00</td>
<td>15-80</td>
</tr>
<tr>
<td>BCA</td>
<td>40.80</td>
<td>7.16</td>
<td>16-61</td>
</tr>
<tr>
<td>BCM</td>
<td>22.80</td>
<td>4.41</td>
<td>13-35</td>
</tr>
</tbody>
</table>

Note. N = 294. LSAS = social anxiety; BFNE-II = fear of negative evaluation; CES-D = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies.

**Age, gender and depression effects.** In order to check if age and gender needed to be controlled for in the regression analyses, differences were examined. The chi-square test for independence was conducted to determine if there were significant differences across gender in relation to risky alcohol use (Alcohol) or risky cannabis use (Cannabis). There was no significant difference in the proportion of men compared to women for Alcohol, $\chi^2(1, n = 294) = .062, \ p = .803$, or for Cannabis, $\chi^2(1, n = 294) = 1.777, \ p = .182$.

A one way ANOVA was generated to explore differences across gender for all continuous variables. Women had significantly higher mean scores compared to men on social anxiety (LSAS), $F(1, 292) = 6.600, \ p < .05$, sensitivity to punishment (SP), $F(1, 292) = 10.030, \ p < .01$, positive alcohol expectancies (AEP), $F(1, 292) = 6.837, \ p < .01$, and maladaptive coping strategies (BCM), $F(1, 292) = 9.189, \ p < .01$. Therefore, gender was subsequently controlled in the regression analyses.
As the Age variable remained skewed after transformation, both Pearson’s product-moment correlation coefficients and Spearman’s rank order correlation coefficients were obtained and checked for consistency. There were differences in terms of which of the other continuous variables were significantly associated with Age (transformed). Therefore, the effect of Age (transformed) was tested by way of Spearman’s correlation coefficients with all variables. Significant positive correlations were detected between age and risky cannabis use (Cannabis), Spearman’s \( r = .124 \), and between age and adaptive coping strategies (BCA), Spearman’s \( r = .121 \), both at the .05 level. Significant negative correlations were detected between age and sensitivity to reward (SR), Spearman’s \( r = -.242 \), at the .01 level, between age and sensitivity to punishment (SP), Spearman’s \( r = -.140 \), at the .05 level, and between age and maladaptive coping strategies (BCM), Spearman’s \( r = -.131 \), at the .05 level. These results indicated that older participants were less sensitive to reward and punishment, with less maladaptive coping strategies, with more adaptive coping strategies, but also with more risky cannabis use. These results are consistent with the literature with respect to ageing and drive sensitivity (Windsor, Pearson & Butterworth, 2012), and coping strategies (Diehl et al., 2014). The results with respect to older participants having more risky cannabis use are indirectly supported by the recent findings of Butterworth, Slade & Degenhardt (2014), who found no significant differences between age cohorts as to the time from first use of cannabis to the onset of a cannabis use disorder (given that increasing levels of potency of cannabis could be expected to lead to a faster rate of progression from first use to disorder among younger cohorts). Therefore age was subsequently controlled for in the regression analyses.

A correlational analysis was conducted to account for the effect of depression on social anxiety variables, risky alcohol use, risky cannabis use and the six individual difference variables. This demonstrated that depression (CES-D) was positively related to a number of variables: social anxiety (LSAS), fear of negative evaluation (BFNE-II), risky alcohol use (Alcohol), reward sensitivity (SR), punishment sensitivity (SP), negative alcohol expectancies (AEN) and maladaptive coping strategies (BCM). CES-D was negatively related to adaptive coping strategies (BCA). Therefore depression was subsequently controlled for in the regression analyses.
Correlational Analysis

Bivariate correlational analysis was conducted to examine the strength and direction of relationships amongst the variables. As seen in Table 3, there was a strong positive association between social anxiety (LSAS) and depression (CES-D), fear of negative evaluation (BFNE-II), and sensitivity to punishment (SP). There was a moderate positive relationship between LSAS and maladaptive coping strategies (BCM). There was a small but negative association between LSAS and adaptive coping strategies (BCA). In contrast to the hypotheses, LSAS was not related to either negative or positive alcohol expectancies.

Table 3

*Intercorrelations among Variables*

<table>
<thead>
<tr>
<th>Variable</th>
<th>LSAS</th>
<th>CES-D</th>
<th>BFNE-II</th>
<th>ALC</th>
<th>CAN</th>
<th>SR</th>
<th>SP</th>
<th>AEP</th>
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<td>-.22**</td>
<td>-.02</td>
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*Note.* LSAS = social anxiety; CES-D = depression; BFNE-II = fear of negative evaluation; ALC = risky alcohol use; CAN = risky cannabis use; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies. *p < .05, **p < .01.

There was a moderate positive association between risky alcohol use (Alcohol) and positive alcohol expectancies. Alcohol had small positive correlations with CES-D, BFNE-II, risky cannabis use (Cannabis), sensitivity to reward (SR), and negative alcohol
The hypotheses regarding associations between alcohol and other variables (except adaptive and maladaptive coping strategies) were confirmed by these results. However, there was only one (small) positive association between risky cannabis use and the hypothesised variables, namely positive alcohol expectancies (AEP). This suggests that there are similarities between alcohol and cannabis expectancies, but also that there are significant differences between alcohol and cannabis in terms of factors that are related to and/or contribute to the risky use of each of them.

Although not hypothesised, interestingly there was no positive or negative relationship between social anxiety (LSAS) and either Alcohol or Cannabis use. However, fear of negative evaluation (a core component of social anxiety) was positively related to risky alcohol use. Fear of negative evaluation was also positively related to five of the six individual differences variables; SR, SP, AEP, AEN and BCM. It was negatively related to the sixth of these variables, BCA. In comparison, LSAS was only associated with three of the six individual differences variables. Notably, fear of negative evaluation was associated with all other variables apart from cannabis, compared to social anxiety which was associated with fewer variables overall.

**Logistic Regression Analyses**

Univariate logistic regression analyses were run to examine individual effects of the variables (gender, age, depression, social anxiety, fear of negative evaluation, sensitivity to reward and punishment, positive and negative alcohol expectancies, and adaptive and maladaptive coping strategies) on risky (moderate-high) alcohol use (coded 1) relative to low risk alcohol use (coded 0). This dichotomous approach is consistent with McBride et al. (2014) and emphasises a taxonomic framework to problematic substance use. The same univariate logistic regressions were also examined in relation to cannabis use. The same codes were used for the two cannabis use groups.

All variables were then retained in a hierarchical logistic regression on alcohol and on cannabis in a multivariate model to assess changes in their relative importance when adjusting for earlier (step) factors. As age, gender and depression were correlated with several of the other variables, they were all entered at Step 1 together. Social anxiety and fear of negative evaluation were entered at Step 2; the individual difference factors (sensitivity to reward and punishment, positive and negative alcohol expectances, and
adaptive and maladaptive coping strategies) were entered at Step 3, and the interaction variables (the six individual difference factors combined with social anxiety, e.g., LSAS x SR) were entered at Step 4. Due to the size of the sample, there was insufficient power to effectively also examine three and/or four way interactions (between LSAS and up to three of the individual difference factors).

**Alcohol.** As shown in Table 4, there were five significant predictors of risky alcohol use in the univariate regressions: CES-D, BFNE-II, SR, AEP, and AEN. It is noted that SP was approaching significance in its univariate regression analysis ($p = .058$). These results partially supported the hypothesis in relation to predictors of alcohol use, as only five out of the eight factors hypothesised were significant predictors of risky alcohol use. The other three factors were social anxiety, adaptive and maladaptive coping strategies. High levels of social anxiety and maladaptive coping strategies did not predict risky alcohol use, and poor adaptive coping was not associated with risky alcohol use.

The most significant of the univariate predictors (with the highest odds ratio) was SR (reward sensitivity), that is, the odds of a person having moderate-high risk alcohol use is 1.09 times higher for someone who reports a higher level of reward sensitivity than for a person who has less reward sensitivity. In decreasing order of likely odds of predicting risky alcohol use, positive alcohol expectancies (AEP) was next, that is, the odds of a person having moderate-high risk alcohol use is 1.05 times higher for someone who reports a higher level of positive alcohol expectancies than for a person who has less positive alcohol expectancies. This factor was followed by both negative alcohol expectancies and fear of negative evaluation. Both these factors had odds ratios of 1.04. Lastly, the odds of a person having moderate-high risk alcohol use is 1.02 times higher for a person with a higher level of depression than for a person with less depression.
Table 4

*Logistic Regression Analyses for Risky Alcohol Use*

<table>
<thead>
<tr>
<th>Step</th>
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<th></th>
<th></th>
<th>Multivariate (Hierarchical)</th>
<th></th>
</tr>
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<td>p</td>
<td>Odds Ratio (95% CI)</td>
<td>p</td>
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<td>1.02 (1.00-1.05)</td>
<td>.02*</td>
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<td>.006**</td>
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<td>.000**</td>
<td>1.06 (1.04-1.08)</td>
<td>.000**</td>
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<tr>
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<td>.02*</td>
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<td>.91 (.83-99)</td>
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<td>1.00 (1.00-1.00)</td>
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<tr>
<td></td>
<td>LSAS x AEN</td>
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<td>.52</td>
<td>1.00 (1.00-1.00)</td>
<td>.52</td>
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Note. CI = confidence interval; Gender = comparing males to females; Age (LG10) = transformed variable; CES-D = depression; LSAS = social anxiety; BFNE-II = fear of negative evaluation; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies. * p < .05, ** p < .01.

In the hierarchical logistic regression, CES-D, BFNE-II, AEP, AEN and BCM were significant at the steps they were respectively entered. Of these factors, BCM was the only one that was not significant in the univariate logistic regressions, whilst one of the significant predictors from the univariate model, SR, was not significant in the multivariate model. These results partially supported the hypothesis in relation to predictors of alcohol
use, as five out of the eight factors hypothesised were significant predictors of risky alcohol use in the multivariate model. The other three factors were social anxiety, reward sensitivity and adaptive coping strategies. High levels of social anxiety and reward sensitivity did not predict risky alcohol use, and poor adaptive coping was not associated with risky alcohol use.

At step 1, depression was the only significant predictor of risky alcohol use. The odds of a person with a high level of depression having moderate-high risk alcohol use was 1.02 times higher than a person with a low depression level. At step 2, fear of negative evaluation was the only significant predictor of risky alcohol use. The odds of a person with a strong fear of negative evaluation having moderate-high risk alcohol use was 1.03 times higher than a person with a low fear of negative evaluation (after controlling for gender, age and depression).

At step 3, three of the six individual difference variables were significant predictors of risky alcohol use. The odds of a person with a high level of positive alcohol expectancies having moderate-high risk alcohol use was 1.06 times higher than a person with a low level of positive alcohol expectancies. The odds of a person with a high level of negative alcohol expectancies having moderate-high risk alcohol use was 1.03 times higher than a person with low negative expectancies. Lastly, the odds of a person with a high level of maladaptive coping strategies having moderate-high risk alcohol use was .91 times lower than a person with a low level of maladaptive coping strategies.

At step 4, none of the interaction variables reached significance. Therefore, the moderation hypotheses were not supported. It is also noted that AEP, AEN and BCM remained significant from step three (when they were first entered) to step four (the final step when the interaction variables were entered) with the same respective odds ratios. At step 4, BFNE-II was significant again (Exp (B) = 1.03, \( p = .03 \)) after losing significance at step 3. The Hosmer and Lemeshow Goodness of Fit Test supported the whole (multivariate) model \( \chi^2 (8, n = 294) = 7.083, \ p = .528 \) (step 4) or \( \chi^2 (8, n = 294) = 13.213, \ p = .105 \) (step 3).

**Cannabis.** As shown in Table 5, there was one significant predictor of risky cannabis use, positive alcohol expectancies (AEP), in the univariate logistic regressions. This result demonstrated that the odds of a person having moderate-high risk cannabis use
is 1.03 times higher for someone who reports a high level of positive alcohol expectancies than for a person who has few positive alcohol expectancies. This result only partially supported the hypotheses about predictors of risky cannabis use, as only one out of the eight factors hypothesised, significantly predicted risky cannabis use. The other seven factors were reward sensitivity, negative alcohol expectancies, adaptive and maladaptive coping strategies, depression, social anxiety and fear of negative evaluation. High levels of six of these seven factors (excluding adaptive coping strategies) did not predict risky cannabis use, while poor adaptive coping was also not associated with risky cannabis use.

In the hierarchical logistic regression, two individual difference variables were significant when first entered at step 3: punishment sensitivity (SP) and positive alcohol expectancies (AEP). The hypothesis about predictors of risky cannabis use was only partially supported in the hierarchical model, as only one of the eight variables hypothesised to be a predictor of risky cannabis use, AEP, was confirmed as such. SP was not hypothesised to be associated with risky cannabis use. The other seven factors that were so hypothesised (reward sensitivity, negative alcohol expectancies, adaptive and maladaptive coping strategies, depression, social anxiety and fear of negative evaluation), were not associated with risky cannabis use. High levels of these factors (apart from adaptive coping strategies) and poor adaptive coping, did not predict risky cannabis use.

At step 3, two of the six individual difference variables were significant predictors of risky cannabis use. The odds of a person with a high level of positive alcohol expectancies having moderate-high risk cannabis use was 1.04 times higher than a person with a low level of positive alcohol expectancies. The odds of a person with a high level of punishment sensitivity having moderate-high risk cannabis use was .90 times lower than a person with a low level of punishment sensitivity.

At step 4, none of the interaction variables reached significance. Therefore, the moderation hypotheses were not supported. It is noted that both punishment sensitivity and positive alcohol expectancies remained significant from step 3 (when they were first entered) to step 4 (the final step when the interaction variables were entered into the model), with the same respective odds ratios. Overall, it appears that the multivariate model supported the hypotheses about predictors of risky cannabis use more strongly than
the univariate models. The Hosmer and Lemeshow Goodness of Fit Test supported the whole (multivariate) model $\chi^2 (8, n = 294) = 12.351, p = .14$ (step 4) or $\chi^2 (8, n = 294) = 6.122, p = .634$ (step 3).

Table 5

Logistic Regression Analyses for Risky Cannabis Use

<table>
<thead>
<tr>
<th>Step</th>
<th>Variable</th>
<th>Univariate</th>
<th>Multivariate (Hierarchical)</th>
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<tr>
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<td>BFNE-II</td>
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<td>Step 3</td>
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<td></td>
<td>SP</td>
<td>.95 (.90-1.01)</td>
<td>.10</td>
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<tr>
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<td>BCA</td>
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<tr>
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<tr>
<td></td>
<td>LSAS x BCM</td>
<td>.43</td>
<td>1.00 (1.00-1.00)</td>
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</table>

*Note.* CI = confidence interval; Gender = comparing males to females; Age (LG10) = transformed variable; CES-D = depression; LSAS = social anxiety; BFNE-II = fear of negative evaluation; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies. * $p < .05$, ** $p < .01$.

Alcohol and cannabis. With regard to the logistic regression analyses on both substances, only one individual difference variable, positive alcohol expectancies (AEP)
was a significant predictor in univariate and multivariate models for risky alcohol and risky cannabis use. This suggests there are similarities between positive alcohol and positive cannabis expectancies. Sensitivity to reward (SR) was a significant predictor of risky alcohol use in the univariate logistic regression, whilst sensitivity to punishment (SP) was a significant (negative) predictor of risky cannabis use in the hierarchical logistic regression (after controlling for age, gender, depression and the social anxiety variables). It would appear that both these drives play an important role in problematic substance use. There were no other predictors of risky cannabis use from the individual difference variables.

There were two other predictors of risky alcohol use: negative alcohol expectancies (AEN) and maladaptive coping strategies (BCM). BCM was a negative predictor of risky alcohol use only after controlling for gender, age, depression and the social anxiety variables.

Post Hoc Mediation Analysis

As there were no significant interactions amongst any of the individual difference variables (potential moderators) with social anxiety (LSAS) in the logistic regression analyses, it was decided to conduct post hoc mediation analysis if feasible. LSAS was not significant as a predictor when entered at step two in either of the hierarchical logistic regressions with Alcohol or Cannabis. However, BFNE-II (fear of negative evaluation), a core component of social anxiety, was significant at step two, but not at step three in the regression on Alcohol. Therefore, mediation analysis was considered using BFNE-II instead of LSAS. There were three potential mediating variables: AEP (positive alcohol expectancies), AEN (negative alcohol expectancies), and BCM (maladaptive coping strategies). These three factors were significant when entered at step three of the same model. Thus, mediation was tested by examining whether BFNE-II (the predictor/independent variable), was mediated by each of these three variables, one at a time, in their relationship with Alcohol (the outcome variable).

Using the strategy proposed by Baron and Kenny (1986), additional regressions were conducted to obtain coefficients to examine mediation effects. The first step of this strategy was to obtain the unstandardized B value coefficient and the standard error value for the independent variable (BFNE-II), by regressing the first potential mediator, AEP, as the DV on BFNE-II. As AEP was reasonably normally distributed, a hierarchical linear
regression was conducted. Age (transformed), gender and depression (CES-D) were controlled for as previously; by being entered at step one. BFNE-II was entered at step two. The result showed that BFNE-II was a significant predictor of AEP, thus meeting the first requirement.

The second requirement using Baron and Kenny’s strategy to test for mediation, was to regress the DV (Alcohol) on the independent variable (BFNE-II). As the DV was dichotomous, a hierarchical logistic regression was performed, controlling for age (transformed), gender and depression at step one, and entering BFNE-II at step two. The second requirement was met with BFNE-II being a significant predictor of risky alcohol use.

The third stage of the mediation testing procedure was to obtain the unstandardized B value coefficient and the standard error value for the mediator variable (AEP), by regressing the DV (Alcohol) on both the independent variable (BFNE-II) and on the mediator variable (AEP). A hierarchical logistic regression was performed as the DV was not continuous, controlling for age (transformed), gender and depression. Both BFNE-II and AEP were entered at the second step of this regression. AEP was significant in this regression, thus fulfilling the third requirement. The two sets of coefficient and standard error figures were tested using Sobel’s test (Sobel, 1982), to obtain the indirect effect size of the independent variable ((BFNE-II) on the DV (Alcohol) via the mediator (AEP). This test confirmed that significant mediation had occurred for positive alcohol expectancies on fear of negative evaluation: $z = 2.91, p < .05$, that is, fear of negative evaluation is related to higher levels of positive alcohol expectancies which in turn are related to higher levels of risky alcohol use. The relationship between fear of negative evaluation and risky alcohol use is mediated by positive alcohol expectancies having statistically controlled for age, gender and depression.

Next, AEN (negative alcohol expectancies) was tested as a potential mediator in the relationship between BFNE-II and risky alcohol use. As AEN was reasonably normally distributed, a hierarchical linear regression was conducted. Age (transformed), gender and depression (CES-D) were controlled for as previously; by being entered at step one. BFNE-II was entered at step two. BFNE-II was not a significant predictor of AEN in the
First regression step proposed by Baron and Kenny (1986) so mediation could not be established.

Finally, BCM (maladaptive coping strategies) was tested as a potential mediator. As BCM was reasonably normally distributed, a hierarchical linear regression was conducted. Age (transformed), gender and depression (CES-D) were controlled for as previously; by being entered at step one. BFNE-II was entered at step two. The result showed that BFNE-II was a significant predictor of BCM, thus meeting the first requirement. The second requirement using Baron and Kenny’s strategy to test for mediation, was to regress the DV (Alcohol) on the independent variable (BFNE-II). As the DV was dichotomous, a hierarchical logistic regression was performed, controlling for age (transformed), gender and depression at step one, and entering BFNE-II at step two. The second requirement was met with BFNE-II being a significant predictor of risky alcohol use. The third stage of the mediation testing procedure was to regress the DV (Alcohol) on both the independent variable (BFNE-II) and on the mediator variable (BCM). A hierarchical logistic regression was performed as the DV was not continuous, controlling for age (transformed), gender and depression. Both BFNE-II and BCM were entered at the second step of this regression. However, BCM was not significant in this regression, thus the third requirement to test for mediation was not fulfilled. In summary, only positive alcohol expectancies (AEP) mediated the relationship between fear of negative evaluation and risky alcohol use.

Reverse Regression Analysis

The final hypothesis of Study 1 related to predictors of social anxiety. It was hypothesised that sensitivity to punishment, both positive and negative alcohol expectancies and maladaptive coping strategies would positively predict social anxiety, whilst adaptive coping strategies would negatively predict social anxiety. As LSAS was a continuous variable, a linear regression analysis was utilised. All independent variables, that is, gender, age (transformed), CES-D, BFNE-II, SR, SP, AEP, AEN, BCA, BCM, and the dichotomised Alcohol and Cannabis variables, were entered together, with LSAS (social anxiety) as the DV.

Assumptions were checked prior to analysis. There was no violation of assumptions. There were no signs of multicollinearity as no two variables had bivariate
correlations of .7 or higher. In addition, no Tolerance values were less than .10, nor were any VIF (Variance inflation factor) values higher than 10. The assumptions of normality, linearity, homoscedasticity and independence of residuals were checked via inspection of the residuals scatterplot and the Normal Probability Plot. There were no major deviations from normality and no apparent violations of these assumptions. The presence of outliers was checked by inspecting the Mahalanobis distances. Using an alpha level of .001, there were no values higher than the critical value ($\chi^2 = 32.909, df = 12$), using the number of independent variables as the degrees of freedom (Tabachnick & Fidell, 2007). Results for the multiple regression analysis are presented in Table 6.

Table 6

<table>
<thead>
<tr>
<th>Variable</th>
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Note. Age (Log) = transformed variable; Alcohol = risky alcohol use; Cannabis = risky cannabis use; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; CES-D = depression; BFNE-II = fear of negative evaluation.

Just over half (52.3%) of the variance in social anxiety (LSAS)) was explained by this model. In addition, the model was statistically significant ($F (12, 281) = 25.627, p < .001$). The strongest contributor was sensitivity to punishment (SP) which uniquely
explained 12% of the total variance in social anxiety. The next strongest contributor was the other personality variable, sensitivity to reward (SR) which uniquely explained 5% of the total variance in social anxiety in this model. Other significant unique contributors predicting social anxiety were depression (CES-D), fear of negative evaluation (BFNE-II0, and positive alcohol expectancies (AEP). Thus, the hypothesis was only partially supported as only two of the five hypothesised individual difference variables, SP and AEP, were significant predictors of social anxiety.

In summary, when all variables were entered together in the multiple regression with LSAS as the DV, there were several contributors to social anxiety with both sensitivity to punishment and sensitivity to reward being the strongest predictors. AEP (positive alcohol expectancies) which significantly predicted both risky alcohol and cannabis use, as well as mediating the relationship between fear of negative evaluation (a core component) of social anxiety and risky alcohol use, also made a unique contribution to social anxiety in this model. This would appear to indicate the importance of this type of cognition on the relationship between social anxiety and problematic substance use (particularly alcohol use) in both directions.

Discussion

Summary

The overall purpose of this study was to increase understanding about the nature of the relationship between social anxiety and substance use given inconsistent findings in this area of research. To the author’s knowledge, this is the first study investigating the relationship between social anxiety symptoms and risky alcohol use, as well as the relationship between social anxiety symptoms and risky cannabis use in a non-clinical sample, by way of the simultaneous exploration of the roles of three important variables: personality (drive sensitivity), alcohol expectancies and coping strategies. Findings supported the important role that sensitivity to reward plays in relation to alcohol use as well as to social anxiety, whilst punishment sensitivity was shown to play an important role in relation to cannabis use and social anxiety. Whilst relationships were demonstrated by both adaptive and maladaptive coping strategies with social anxiety, only maladaptive
coping strategies had a significant role in relation to alcohol use. Negative alcohol expectancies were highly relevant to alcohol use, whilst positive alcohol expectancies had the most important functions out of all the individual difference variables. They were significantly associated with both alcohol and cannabis use, and were also a unique predictor of social anxiety.

Research Questions

Findings indicated no significant relationship between social anxiety and alcohol use or between social anxiety and cannabis use. These results are consistent with some studies investigating social anxiety and alcohol use, for example, Eggleston et al. (2004) and Ham et al. (2007). However, they are inconsistent with others that have found a positive relationship (e.g., Lewis & O’Neill, 2000), and those studies that have found a negative relationship (e.g., Stewart et al., 2006). Research findings have more consistently demonstrated a positive relationship between social anxiety and both alcohol and cannabis use problems (Buckner et al., 2013b), as opposed to alcohol/cannabis use in terms of volume or frequency where the findings are more difficult to comprehend. The substance use scale used in this study (the ASSIST) comprised items measuring substance use as well as substance use problems. This may have accounted for the lack of association between social anxiety and substance use in Study 1.

Outcome of Hypothesis Testing

Reward and punishment sensitivity. As hypothesised, there was a positive association between punishment sensitivity and social anxiety, and punishment sensitivity predicted severity of social anxiety. In fact, punishment sensitivity was the strongest unique predictor of social anxiety. Interestingly, reward sensitivity was found to be the second strongest (negative) unique predictor of social anxiety in the “Reverse Regression Analysis”, although there was no significant association between social anxiety and reward sensitivity in the correlational analysis. The discrepancy between these two findings is matched by the inconsistent findings as to the existence of, and the direction of the relationship between social anxiety and reward sensitivity in the literature. For example, no relationship was found between these two factors in Kashdan and Roberts (2006) but a
negative association was found by Kimbrel et al. (2012). Hence, no hypothesis was put forward about the relationship between reward sensitivity and social anxiety in this study.

The hypotheses about the positive relationship between substance use and reward sensitivity as well as reward sensitivity being a predictor of substance use were partially supported. As expected, reward sensitivity was positively associated with risky alcohol use as demonstrated in numerous non-clinical studies, for example, Ivory and Kambouropoulos (2012). Also, as expected, reward sensitivity predicted risky alcohol use but only in the univariate regression. Reward sensitivity did not play a significant role in the hierarchical regression analysis when gender, age, depression and social anxiety phenomena were controlled for. This may have been due to loss of power when reward sensitivity was entered together with the other individual difference variables.

Contrary to expectations, reward sensitivity was not associated with risky cannabis use, nor did reward sensitivity predict risky cannabis use. Interestingly, the literature on the association between reward sensitivity and cannabis use is not as strong as for alcohol use. While some previous research (e.g., Simons et al., 2008), has reported a positive association between cannabis use and reward sensitivity, other research has been contradictory (e.g., Simons & Arens 2007). Notably, a minority of participants used cannabis in the previous six months in the Simons and Arens (2007) study. In the present study, a minority of cannabis users (21%) were classified as moderate-high risk users (with 79% of the cannabis users classified as “low risk”). Thus the present sample of cannabis users had a relatively similar composition to the sample studied by Simons and Arens (2007). In contrast, our sample of alcohol users had a higher percentage of moderate-high risk users (34%), with 66% of the sample classified as “low risk” for alcohol. It is likely that the modest number of participants in the moderate-high risk cannabis group in our sample affected the result with respect to the relationship between reward sensitivity and risky cannabis use.

**Alcohol expectancies.** The hypotheses that social anxiety would be positively associated with both positive and negative alcohol expectancies, and that both types of expectancies would predict severity of social anxiety were only partially upheld. Although social anxiety was not correlated with either positive or negative alcohol expectancies, positive alcohol expectancies was a unique predictor of social anxiety in the multiple
regression analysis. Furthermore, positive alcohol expectancies was the only individual difference variable that was a unique predictor of social anxiety in the multiple regression analysis.

The lack of association between positive or negative alcohol expectancies and social anxiety is inconsistent with recent evidence that has emerged in this research area including a meta-analysis conducted by Schry and White (2013). However, most of the research in this area has focused on the role of alcohol expectancies as moderators or mediators in the relationship between social anxiety and substance use (particularly alcohol use), and until, recently, many of these studies have not supported the moderating role of expectancies on social anxiety, for example, see the review by Morris et al. (2005). More recent research (e.g., Ham, 2009) supports a role for alcohol expectancies as moderators and correlates of social anxiety. However, generally this has only occurred when the alcohol expectancies have been specifically relevant to social anxiety (e.g., expectancies of social facilitation). Ham (2009) also found a strong positive association between social anxiety and negative alcohol expectancies. The positive alcohol expectancies scale used in this thesis was only partly comprised of social facilitation expectancies, with just under one-third of the items comprising this scale being social facilitation expectancies. This may account for the lack of significant correlation between social anxiety and risky substance use. However, this does not explain why positive alcohol expectancies predicted social anxiety in the “Reverse regression analysis”.

As hypothesised, both positive and negative alcohol expectancies were positively related to risky alcohol use, and predicted severity of risky alcohol use in both the univariate and multivariate logistic regression models. These results support previous findings (e.g., Hasking & Oei, 2004; Pabst et al., 2014). The hypotheses in regard to alcohol expectancies and risky cannabis use were partially supported as only positive (but not negative) alcohol expectancies were correlated with risky cannabis use, and also predicted severity of cannabis use in both the univariate and multivariate logistic regression models. These results are consistent with past findings that have identified a positive relationship between positive cannabis expectancies and frequency of cannabis use (Buckner & Schmidt, 2008). These results also align with findings of common features of cognitive processes underlying expectancies involved in both alcohol and cannabis use.
(Stacy, 1997), as well as findings associating positive and negative alcohol expectancies with positive and negative cannabis expectancies respectively (Aarons et al., 2001). However, the findings in the present study are inconsistent with those of Bucker and Schmidt (2008) where a positive association between negative cannabis expectancies and cannabis problems was found. Nevertheless, the findings from this study point to an important role played by positive alcohol expectancies in both alcohol and cannabis use.

**Coping strategies.** As hypothesised, social anxiety was positively related to maladaptive coping, and negatively related to adaptive coping strategies. These findings are consistent with those of Thomasson and Psouni (2010). However, the hypothesis about the roles of adaptive and maladaptive coping strategies as predictors of social anxiety severity was not supported, as neither adaptive or maladaptive coping strategies predicted social anxiety severity. These findings are contrary to those of Thomasson and Psouni (2010). The contrast in findings may be explained by methodological differences. Participants in the Thomasson and Psouni (2010) study were students and members of social anxiety organisations, and used different social anxiety measures. Furthermore, multiple regression analysis was conducted on the data, compared to logistic regression analysis in the present study.

The hypotheses that both risky alcohol use and risky cannabis use would be positively related to maladaptive coping strategies but negatively related to adaptive coping strategies were not supported as there were no significant correlations between these variables. These results are contrary to those in the literature particularly those pertaining to the positive association between maladaptive coping strategies and substance use. This association has been consistently found in various non-clinical populations including students (e.g., Hundt et al., 2013) and community samples (e.g., Ivory & Kambouropoulos, 2012). The hypotheses about coping strategies predicting severity of risky substance use were partially supported as maladaptive coping predicted risky alcohol use (after controlling for age, gender, depression and social anxiety phenomena) in the multivariate logistic regression, but neither adaptive or maladaptive coping strategies predicted risky cannabis use in either the univariate or multivariate logistic regression analyses. All in all, the results about the lack of significant associations between coping strategies and
substance use are somewhat surprising given that in the same sample, there were significant associations between both adaptive and maladaptive coping strategies with social anxiety.

**Moderation hypotheses.** Contrary to the hypotheses regarding moderation, there were no significant interactions between the moderator variables and social anxiety leading to risky alcohol or cannabis use. With respect to reward and punishment sensitivity, the present results are inconsistent with those of Booth and Hasking (2009), the only other researchers to have investigated the role of drive sensitivity in the relationship between social anxiety and alcohol use, using regression analyses. Notably, they did not find any two-way interactions (between social anxiety and drive sensitivity), but did find significant three-way interactions involving social anxiety, reward sensitivity (BAS) subtypes and positive expectancy subtypes. The present study can also be distinguished on the basis of sample size (this study’s participant group was smaller), differences in measures utilised (for all variables), and type of regression analysis used (this study used logistic regression).

In fact, all the hypotheses with respect to moderation in the present study were largely based on findings from previous studies where multiple regression analyses or group difference analyses were used. Due to the skewness of the substance use variables in this study, logistic regression analysis was used and participants classified into one of two groups (for each of alcohol and cannabis), that is, no-low risk or moderate-high risk alcohol or cannabis groups. With logistic regression analysis, there may not have been significant power to detect significant effects. The present results are consistent with those of a past study which also used logistic regression analysis dividing college students into non-problem or problem alcohol users (Lewis & O’Neill, 2000). Lewis and O’Neill (2000) measured five aspects of social functioning, including social anxiety and fear of negative evaluation (a core feature of social anxiety as previously discussed and also measured separately in this thesis). In addition, seven types of alcohol expectancies were measured by Lewis and O’Neill (2000). All five social functioning measures were tested for interactions with particular alcohol expectancy subscales in univariate logistic regression models. Both the social anxiety and fear of negative evaluation measures were tested for interactions with a positive alcohol expectancy subscale (relaxation and tension reduction). However, no interactions were significant in predicting problem alcohol use status (although there were significant differences between the two groups on social anxiety, fear...
of negative evaluation and all six positive expectancy types). Thus the lack of moderation with any of the three types of individual factors (drive sensitivity, alcohol expectancies, and coping strategies) in Study 1 may be partly explained by the type of regression analysis used (logistic rather than multiple regression).

**Fear of negative evaluation and mediation.** Fear of negative evaluation (measured by the BFNE-II) was positively related to risky alcohol use in the correlational analysis although social anxiety (measured by the LSAS) was not so related. This result is consistent with findings in other research (Lewis and O’Neill, 2000; Stewart et al., 2006) where both social anxiety and fear of negative evaluation were examined in relation to alcohol use. In this study, fear of negative evaluation also predicted risky alcohol use in both the univariate and multivariate logistic regression analyses. As fear of negative evaluation is a core attribute of social anxiety (Rapee & Heimberg, 1997), its role in our research was considered further.

Post hoc analysis revealed that positive alcohol expectancies mediated the relationship between fear of negative evaluation and risky alcohol use. This result is also consistent with Stewart et al. (2006) where the relationship between fear of negative evaluation and alcohol use problems was found to be mediated by the coping motive (being motivated to drink to cope with negative affect), a construct which has been found to positively correlate with positive alcohol expectancies such as tension reduction and increased confidence (Hasking et al., 2011). This finding highlights the importance of fear of negative evaluation as a key characteristic of social anxiety, particularly with regard to its association with risky alcohol use, and one which may be more closely associated with risky alcohol use compared to other (measurable) components of social anxiety. Notably, neither fear of negative evaluation or social anxiety were related to or predicted cannabis use in the present study. The low numbers of risky cannabis users in this study may have contributed to this result. In any event, the findings suggest that careful attention should be paid to fear of negative evaluation when treating people affected with social anxiety and particularly when that anxiety co-occurs with problematic alcohol use.

The importance of fear of negative evaluation in this study was also demonstrated when its relationships with the individual difference variables (drive sensitivity, alcohol expectancies and coping strategies), were examined (compared to the relationships of these
variables with the social anxiety measure). Fear of negative evaluation was significantly related to all six of these variables, compared to social anxiety which was significantly related to only the three strongest of those in their association with fear of negative evaluation, that is, in descending order of strength of association, sensitivity to punishment, maladaptive coping strategies, and adaptive coping strategies.

The findings of positive associations of both reward and punishment sensitivity with fear of negative evaluation (compared to social anxiety which was only positively related to sensitivity to punishment), is consistent with recent findings of Yen et al. (2012) who surveyed 2,348 college students about social anxiety in real-life and online interactions. These researchers used a brief measure of fear of negative evaluation to capture the cognitive symptoms of social anxiety. The Yen study was not included in the Chapter 3 Personality section as that literature review only considered literature which used more general social anxiety measures as well as the two most frequently used drive sensitivity measures (SPSRQ and BIS/BAS scales). In the Yen study, all BIS and BAS subscales (representing both punishment and reward sensitivity) were positively related to fear of negative evaluation. Thus, there appears to be a positive relationship between both drive sensitivities and the cognitive components of social anxiety at least in non-clinical populations. These findings are further supported by the findings from the multiple regression analysis on social anxiety in Study 1, which found that both reward and punishment sensitivity as well as fear of negative evaluation, were unique predictors of social anxiety. These findings suggest that treatment for individuals with symptoms of social anxiety and problematic alcohol use, ought to take into account sensitivities to both reward and punishment.

With regard to the findings of positive associations between both positive and negative alcohol expectancies with fear of negative evaluation (compared to social anxiety which was not associated with either type of expectancy), it is noted that social anxiety was found to be positively related to both expectancy types in a meta-analysis of student population studies (Schry & White, 2013). In their meta-analysis, Schry and White (2013) categorised social anxiety measures in three ways, two of which could have included a fear of negative evaluation measure. Therefore, their meta-analysis results may have captured this core feature of social anxiety to a sufficient extent for the significant positive results.
with respect to both positive and negative alcohol expectancies. However, the social anxiety measure used in this study (the LSAS), did not specifically or directly measure fear of negative evaluation. Its focus was on fear and avoidance of a range of social performance and interaction activities. Thus, the results of our study highlight the importance of this aspect of social anxiety, that is, the fear of negative evaluation in its relationship with alcohol expectancies. Overall, the importance of the fear of negative evaluation trait in the relationship between social anxiety and alcohol use was revealed especially in the exploration of the roles of drive sensitivities and alcohol expectancies in our study. The significance of fear of negative evaluation has been recognized in the latest DSM (DSM-5) with respect to the role it plays in capturing the essential features of SAD (American Psychiatric Association, 2013). Results from Study 1 emphasise this vital characteristic of social anxiety. Thus, preventative measures as well as aspects of treatment (psychoeducation and cognitive therapy) for socially anxious individuals with problematic alcohol/substance use, need to take into account the fear of negative evaluation aspect, and its relationship with both drive sensitivity, and alcohol expectancies, especially positive alcohol expectancies.

**Other predictors of social anxiety.** As discussed, three of the six individual difference variables were unique predictors of social anxiety in the “Reverse Regression Analysis”. In contrast to the logistic regression analyses which provided information about which variables increased (or reduced) the odds of having problematic (moderate to high risk) substance use, compared to not having problematic substance use (low risk), the multiple regression analysis on social anxiety provided information on a dimensional basis with respect to predictors of social anxiety severity. In order of strength starting from the strongest individual difference variable, punishment sensitivity uniquely explained 12% of the total variance in social anxiety; reward sensitivity uniquely explained 5% of the total variance in social anxiety; and positive alcohol expectancies uniquely explained 1% of the total variance in social anxiety in the same model.

The two other unique predictors of social anxiety were fear of negative evaluation and depression, each of which uniquely explained 1% of the total variance in social anxiety (when all variables were entered together in this regression model). The importance of fear of negative evaluation has been discussed in the previous section. Results from the
multiple regression analysis with respect to depression and social anxiety support past findings from population surveys as to the high comorbidity between these two disorders (e.g., Chartier et al., 2003). It is noted that in the correlational analysis in this study, depression had a large association with both social anxiety and fear of negative evaluation, with 25% of shared variance explaining the relationship between social anxiety and depression, and 28% of shared variance explaining the relationship between fear of negative evaluation and depression. Thus, depression is an important variable which is strongly associated with social anxiety phenomena, and needs to be considered in treatment interventions.

Limitations and Strengths

The use of binary logistic regression analysis was both a limitation and a strength of Study 1. Logistic regression was utilised as the proposed dependent variables, risky alcohol use and risky cannabis use, were not normally distributed, and attempted transformations did not adequately reduce the skewness, particularly of the risky cannabis use variable (Tabachnick & Fidell, 2007). Participants could have been divided into three groups with respect to level of risky alcohol and substance use (low, moderate and high). This would have provided more detail but less power as the frequency count of participants in the high risk cannabis group was very small. Therefore binary logistic regression was utilised. Even with two groups there was some question of power with respect to the low number of participants making up the moderate-high risk cannabis use group. This was a further limitation of this study in terms of lack of power for the data analyses with respect to risky cannabis use. On the other hand, a strength of having two classifications (risky versus non-risky use) of the dependent variables (risky alcohol use and risky cannabis use) was synchronistic with Study 2 group differences between the SAD (non-risky alcohol/substance use) and SAD-SUD (risky alcohol/substance use) groups. Thus using binary logistic regression was closer to a psychiatric taxonomy in terms of classifying people into those who need a treatment intervention (those with risky substance use) versus those who don’t (those with low risk substance use).

Another limitation of Study 1 was the composition of the sample itself, being comprised of both student and community respondents to the survey. Some researchers contend that the relationship between social anxiety and alcohol/cannabis use may differ
between these sample types. For example, in their meta review of social anxiety and alcohol variables, Schry and White (2013) only examined student group samples and found that whilst social anxiety was negatively correlated with alcohol use variables (frequency and quantity), social anxiety was positively associated with alcohol-related problems. In our sample, the majority of participants were first year university students. Other respondents were obtained by way of convenience sampling which is likely to have resulted in some sampling bias (another limitation of our study).

Other limitations with the sample were the predominant number of women such that gender differences could not be explored and gender had to be controlled for in the regression analyses. In addition, the mean age of 24.59 was quite young probably because the majority of participants were first year students. This also limited the generability of the results to older people, for instance, established alcohol patterns may not have been taken into account.

Another limitation of this study was its cross-sectional nature, meaning that causal inferences could not be drawn. On the other hand a strength of this study was the “reverse regression analysis, which to some extent reduces the severity of this limitation. As a result, the importance of both types of drive sensitivity and positive alcohol expectancies, was made apparent in the relationship between social anxiety and alcohol use in both directions. A further limitation was the use of self-report measures which may have contributed to recall bias.

Finally with the benefit of hindsight, including an investigation of risky cannabis use amongst a majority of participants who were first year students, resulted in few numbers with moderate-high risk cannabis and therefore only one significant finding was able to be obtained in relation to risky cannabis use. It is likely that other factors interact with social anxiety in a more significant way resulting in problematic cannabis use.

**Implications**

The findings of this study highlight the importance of sensitivity to reward and punishment, positive and negative alcohol expectancies, and adaptive and maladaptive coping strategies in the relationship between social anxiety and alcohol use in particular. The findings also emphasize the important role of positive alcohol expectancies in relation to risky cannabis use, suggesting that having positive substance use expectancies for one
type of substance can lead to risky use of another type of substance. The importance of fear of negative evaluation was also borne out by the results of our study, and these results support the recent specific inclusion of this cognition in DSM-5, as an essential characteristic of social anxiety disorder. The incorporation of these factors into education, preventative measures and treatment could potentially limit the severity of co-occurring social anxiety and problematic substance use, and therefore also increase the potential for successful treatment outcomes. Clinical implications and treatment recommendations are discussed in the final chapter of this thesis.

**Conclusion**

Much research has been conducted to clarify the nature of the relationship between two commonly occurring disorders or symptoms of social anxiety and substance use disorders. This study was the first to examine this relationship within a biopsychosocial framework. This examination confirmed the importance of biological (drive sensitivity), psychological and social (expectancies and coping strategies) factors which all play a role in the relationship between social anxiety and substance use. These findings can contribute to developing more targeted preventative, educational and treatment measures in order to prevent, contain or successfully treat co-occurring social anxiety and substance use in affected individuals. Finally, results from this study were obtained from a non-clinical sample. Much less research has been conducted with clinical samples. Study 2 looked at the roles of the same three biopsychosocial factors using the same measures, with clinical participants.
Chapter 6: Study 2 – Personality, expectancies and coping strategies in substance abusing and nonabusing socially anxious individuals.

Aims and Hypotheses

Study 2 focused on comparing clinical groups of participants diagnosed with social anxiety disorder only (SAD), or with both social anxiety disorder and substance use disorder (SAD-SUD) on the same three individual differences factors: personality (drive sensitivity), alcohol expectancies and coping strategies. Although it would have been useful to also include a clinical group with a SUD only, this would have increased the scope of the study beyond the capacity of the requirements of this thesis. Due to the smaller clinical sample size (compared to the size of the non-clinical sample in Study 1), it was not feasible to conduct regression analyses and to examine interactions (which would have allowed for direct comparisons to be made between the findings of both studies). As described earlier, very little research has been conducted with clinical samples, with respect to the SAD-SUD comorbidity, despite the high prevalence rate.

Adults diagnosed with social anxiety disorder, but no substance use disorder comprised one group (“SAD”). Adults diagnosed with both social anxiety disorder and a substance use disorder (“SAD-SUD”) were divided into two groups, those who were classified as high risk alcohol use (“SAD-SUD Alcohol”), and all other people diagnosed with both social anxiety and substance use disorders (“SAD-SUD Other”). There were two reasons for this further division within the comorbid group: firstly, to learn more about people with SAD and problematic alcohol use given that alcohol is most frequently associated with social anxiety out of all substances according to the literature; and secondly, to take full advantage of the alcohol expectancies measure. It was hoped that by splitting the SAD-SUD group in this way, more could be learned about differences within this comorbidity.

A fourth group of healthy controls with no diagnosed SAD or SUD (“CONTROL”) was also included in this study. This group was created to understand more about similarities and differences between people with diagnosed SAD or with the SAD-SUD comorbidity. Detail about the formation of these groups is contained in the Method section of this chapter.
**Aim 1.** To increase understanding of the roles of personality (reward and punishment sensitivity), alcohol expectancies (positive and negative) and coping strategies (adaptive and maladaptive) in the SAD-SUD comorbidity by examining differences between clinical groups and a healthy control group.

**Hypotheses.** Six hypotheses were put forward, one for each of the individual difference variables. It was hypothesised that:

1. The SAD group will have a lower sensitivity to reward than all three other groups (both SAD-SUD groups and the CONTROL group).
2. The SAD group will have a higher sensitivity to punishment than both SAD-SUD groups, and both SAD-SUD groups will have a higher sensitivity to punishment than the CONTROL group.
3. The SAD-SUD Alcohol group will have higher positive alcohol expectancies than all three other groups, and the SAD-SUD Other group will have higher positive alcohol expectancies than both the SAD and CONTROL groups.
4. The SAD-SUD Alcohol group will have higher negative alcohol expectancies than all three other groups, and both the SAD-SUD Other and the SAD groups will have higher negative alcohol expectancies than the CONTROL group.
5. The CONTROL group will have higher adaptive coping strategies than all three other groups (both SAD-SUD groups and the SAD group).
6. Both SAD-SUD groups will have higher maladaptive coping strategies than the two other groups, and the SAD group will have higher maladaptive coping strategies than the CONTROL group.

**Aim 2.** To increase understanding of the nature of the SAD-SUD comorbidity by examining predictors of severity of social anxiety from a clinical sample. Predictor variables were chosen (to some extent) according to the results obtained in Study 1 for this analysis.

**Hypothesis.** One hypothesis was put forward.

7. Both reward and punishment sensitivity, depression, alcohol and cannabis use will predict social anxiety severity.
Method

Participants. Clinical participants comprised 84 individuals who were recruited in one of the following ways:

(1) From another research study jointly conducted by Swinburne and Deakin Universities (Staiger et al., 2014) and funded by the Australian Research Council (“the ARC Study”). After intake assessments to enter Odyssey House, a drug and alcohol treatment residential program in Melbourne, Victoria, potential participants were invited to participate in the ARC Study. If they chose to do so, their responses at the baseline clinical interview were screened for social anxiety disorder (SAD) employing the social phobia module of the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). The MINI was also used to determine if they had an alcohol or substance use disorder, using its alcohol and substance dependence modules. All MINI modules are based on DSM-IV criteria (American Psychiatric Association, 2000) for psychological disorders. At the time of the baseline interview, participants also completed a pen and paper questionnaire containing all of the other questionnaires (described under Materials below) being employed in this research study. Consent for inclusion of responses for this study was obtained via the information statement/consent form used in the ARC study (see Appendix D).

(2) From potential participants being assessed for group treatment/therapy for social anxiety disorder at the Swinburne University Psychology Clinic (SWIN-SAG). As part of the assessment for the group treatment, a clinical interview was conducted using the MINI. Responses to the social phobia, alcohol and substance use modules were used in this study. Other components of the assessment included completion of various pen and paper questionnaires, one of which was the Brief Fear of Negative Evaluation Scale-Revised (BFNE-II). Responses to the BFNE-II completed by potential SWIN-SAG participants who also chose to complete the pen and paper questionnaire for this study, were also utilised for this study. The
information statement/consent form for this category of participants is attached at Appendix E.

(3) Through advertisements placed online by ADAVIC, a community group for anxiety disorders in Melbourne Victoria. To screen for having social anxiety disorder, potential interested participants underwent a telephone interview that employed the social phobia, alcohol dependence and substance dependence modules of the MINI. The advertisement and information statement/consent form used for this category of participants are attached at Appendix F.

(4) Through advertisements placed in the Swinburne Psychology Clinic (SWIN-CLINIC) Newsletter, an online publication emailed to registered interested persons including students, staff and former/current clients of the clinic every two months. To screen for having social anxiety disorder, potential interested participants underwent a face to face clinical interview at the Swinburne Psychology Clinic, or a telephone interview that employed the social phobia, alcohol dependence and substance dependence modules of the MINI. The same advertisement and information statement/consent form as described in the previous section were utilised.

**Inclusion and exclusion criteria for clinical participants.** Participants for the ARC Study were not required to meet criteria for SAD as defined by DSM-IV-TR (American Psychiatric Association, 2000). ARC Study participants needed to report sub-clinical levels of social anxiety as measured by the LSAS (Liebowitz, 1987). Specifically they needed to report at least one moderate social anxiety symptom as well as avoidance of at least one social situation, consistent with Merikangas et al., 1987. In fact the two participants recruited from the ARC Study who were not diagnosed with SAD according to the MINI module, scored over 60 on the LSAS which has been classified by other researchers (Mennin et al., 2002; Rytwinski et al., 2009) to exceed the minimum threshold for generalised SAD. All other clinical participants were required to have a SAD diagnosis according to the MINI.
All clinical participants (from all recruitment methods) were screened for an alcohol use disorder (AUD) and/or a substance use disorder (SUD). In order to be classified as “comorbid” (having both SAD and AUD/SUD), clinical participants were required to meet criteria for AUD or SUD as defined by DSM-IV-TR (American Psychiatric Association, 2000), and ascertained via the relevant MINI modules.

Exclusion criteria included being less than 18 years of age; evidence of florid or active psychosis; reporting of current, severe suicidality; and being unable to read English.

**Group type and source.** In total, 57 participants (67.9%) comprised the comorbid group (people diagnosed with both SAD and a SUD), and 27 participants (32.1%) comprised the group of people diagnosed with SAD but no substance use disorder (the SAD group). Most clinical participants (78 or 95%) were diagnosed with the generalised subtype of SAD (according to DSM-IV criteria), whilst 4 people (5%) were diagnosed with the “nongeneralised” subtype of SAD. Diagnosed alcohol and substance use disorder figures are set out in Table 7.

<table>
<thead>
<tr>
<th>Alcohol and Substance Use Disorder Frequencies in the Comorbid Group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number of Clinical Participants</strong></td>
</tr>
<tr>
<td>Alcohol Dependence</td>
</tr>
<tr>
<td>Alcohol Abuse</td>
</tr>
<tr>
<td>Substance Dependence</td>
</tr>
<tr>
<td>Substance Abuse</td>
</tr>
</tbody>
</table>

*Note. N = 57. Totals do not add up to 57 or 100% as some participants were diagnosed with more than one of the alcohol and/or substance use disorders.*

Slightly more than half of all clinical participants (44 or 52.4%), were recruited via the ARC Study. The next most common recruitment source of clinical participants was the SWIN-SAG participants (30 or 35.7%).
Demographic information for all clinical participants \((N = 84)\) in Study 2 is set out below.

**Age.** All participants were aged between 18 and 59 years of age \((M = 32.83; SD = 8.56)\). The commonest age of participants was 34 (9.5%) followed by ages 23, 24, 32 and 37 (all 6.0%).

**Gender.** There were 45 male (53.6%) and 39 female participants (46.4%).

**Country of Birth.** The majority of clinical participants were born in Australia (76.2%), followed by 8.3% born in New Zealand/Oceania, 7.1% born in Asia, and 6% born in Europe.

**Current Relationship Status.** The majority of participants who responded to this question described themselves as “never married” (52.4%), followed by 21.4% who were “married/living with partner”. One participant (1.2%) did not respond to this question.

**Highest Level of Education Completed.** There was a similar frequency of participants who had completed some high school ((32.1%), and who had completed a “TAFE Diploma/Certificate/Trade Qualification” (29.8%). A smaller minority of participants had a university qualification (22.6%), followed by 15.5% who had completed high school.

**Usual Occupation.** This question provided for a “free text” response and one participant (1.2%) did not respond. Of the remainder the largest percentage (40.5%) listed occupations which were categorised as “other”, followed by 15.5% who were community/personal service workers, and 9.5% in each of the categories: “professional”, “technician/trade worker” and “home duties”.

**Current Employment Status.** The majority of participants described themselves as “unemployed” (59.5%), followed by “employed – full-time” (13.1%) and “employed – part-time/casual” (11.9%). One participant (1.2%) did not respond to this item.

**Grouping of Participants for Study 2.** Two groups of clinical participants were identified initially: (a) those diagnosed solely with social anxiety disorder, that is, no diagnosis of either an alcohol use disorder or a substance use disorder (SAD, \(n = 27\)); (b) a comorbid group that was diagnosed with social anxiety disorder and either an alcohol use
disorder or a substance use disorder or with both alcohol and substance use disorders (SAD-SUD, \( n = 57 \)). The SAD-SUD group was further divided (for reasons set out in the Aims and Hypotheses section of this chapter) according to how participants scored on alcohol use in the ASSIST measure. Those that scored 27 or higher, indicating high risk of problematic alcohol use, were placed in one group (SAD-SUD Alcohol, \( n = 18 \)) and all other participants from the SAD-SUD group were renamed (SAD-SUD Other, \( n = 39 \)).

A fourth group of healthy controls (CONTROL) was created (for reasons set out in the Aims and Hypotheses section of this chapter) by selection of all participants from Study 1 who scored less than 30 on the LSAS (indicating no social anxiety), less than 11 on the alcohol scale of the ASSIST (indicating low risk of problematic alcohol use), and less than 4 on all other substance scales of the ASSIST (indicating low risk of problematic substance use). This resulted in a group size of \( n = 30 \). To maintain adequate power for the statistical analyses, it was not possible to match the controls with the clinical groups for any demographic factors. Information about demographic differences between the four groups is provided in the Results section of this chapter.

**Materials.** Materials comprised the clinical interview measure as well as self-report measures. The self-report measures included demographic questions, the responses to which have been summarized above in the “Participants” section. All of the self-report measures apart from the one used to assess depressive symptoms in the clinical sample, were the same as those used in Study 1 by the non-clinical participants. Descriptions of measures used in Study 1 are set out in the Method section of the previous chapter (Chapter 5). Reliability statistics for those previously described measures are provided in Table 8 in this section, following the descriptions of the clinical interview measure and the two depression measures. Due to the question and stem (cumulative) structure of the Alcohol, Smoking and Substance Involvement Screening Test (ASSIST), Cronbach’s alpha coefficients were not obtained for this measure.

**Clinical Interview Measure.** The Mini International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998) is a short, structured diagnostic interview. It uses decision tree logic to assess the major adult Axis 1 disorders according to DSM-IV criteria. The MINI has been shown to have high reliability and good validity when it is compared to
other diagnostic measures such as the Composite International Diagnostic Interview (CIDI) and Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders patients (SCID-P) (Lecrubier et al., 1997; Sheehan et al., 1997). In this study the MINI was used to determine clinical diagnoses of SAD, AUD and SUD. The SAD, AUD and SUD modules of the MINI take between 5 to 15 minutes to administer. The SAD, AUD and SUD modules of the MINI are attached at Appendix G.

**Depression Measures.** The Beck Depression Inventory, Second Edition (BDI-II) (Beck, Steer, & Brown, 1996) is a 21 item widely used self-report instrument that measures the presence and severity of depressive symptoms in both general and clinical populations. Participants are asked to rate each symptom over the past two weeks. Each item (e.g., sadness) is scored on a four-point Likert scale (except two items which are scored on a seven-point Likert scale), with 0 indicating a lack of the symptom and 3 indicating an extreme of the symptom. Higher total scores indicate more severe depressive symptoms. The possible range of scores is 0 - 63. The BDI-II has been shown to be a valid and reliable measure (Beck et al., 1996; Dozois & Covin, 2004). It correlates highly with the CES-D (Hicks & McCord, 2012). Scores can be classified into three broad levels of depression: between 0 and 10 indicating normal ups and downs (no depression), 11-20 indicating mild depression, and 21 and higher indicating major depression (Beck et al., 1996). In the current study, the Cronbach alpha for the clinical participants \((n = 84)\) was .91.

The Centre for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) is a commonly used freely available 20 item self-report scale designed to measure depressive symptoms in the general population. It asks individuals to rate feelings (e.g., “I felt depressed”) or behaviours (e.g., “I had crying spells”) during the past week on a four-point Likert scale ranging from 0 (rarely) to 3 (most of the time). The CES-D has been validated in a wide range of demographic populations, for example, older populations (Beekman et al., 1997), and primary care clinic patients (Zich et al., 1990). It is highly correlated with the BDI-II (Hicks & McCord, 2012). The CES-D shows good reliability with high alpha levels e.g. .88 to .95 (Zhang et al., 2012). In the current study the Cronbach alpha coefficient for CES-D for the non-clinical participants \((n = 30)\) was .88. A cut-off total score of 15 has been shown to discriminate between participants with and
without depression, scores between 16 and 26 have been shown to indicate mild depression, and scores of 27 or higher have been used to indicate major depression (Geisser et al., 1997; Zich et al., 1990).

Table 8

<table>
<thead>
<tr>
<th>Measure</th>
<th>Cronbach’s Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liebowitz Social Anxiety Scale (LSAS)</td>
<td>.98</td>
</tr>
<tr>
<td>Fear of Negative Evaluation (BFNE-II)</td>
<td>.98</td>
</tr>
<tr>
<td>Sensitivity to Reward (SR)</td>
<td>.79</td>
</tr>
<tr>
<td>Sensitivity to Punishment (SP)</td>
<td>.90</td>
</tr>
<tr>
<td>Positive Alcohol Expectancies (AEP)</td>
<td>.96</td>
</tr>
<tr>
<td>Negative Alcohol Expectancies (AEN)</td>
<td>.89</td>
</tr>
<tr>
<td>Adaptive Coping (BCA)</td>
<td>.87</td>
</tr>
<tr>
<td>Maladaptive Coping (BCM)</td>
<td>.72</td>
</tr>
</tbody>
</table>

*Note.* $N = 114$

**Procedure.** After receiving ethics approval (see Appendix A), potential clinical participants responded to one of the above methods of recruitment (see flowchart in Appendix H). They were first given/posted the (relevant) Information Statement to read to which was attached a consent form. After written consent was obtained, clinical interviews were conducted to ascertain if participants could be diagnosed with social anxiety disorder (SAD) and alcohol use/substance use disorders (AUD/SUD) using the MINI. All participants diagnosed with SAD (with or without an AUD or SUD diagnosis) then completed a pen and paper questionnaire containing the same demographics questions and other measures used in Study 1. Only the depression measure differed; the Beck Depression Inventory-II (BDI-II) was used in Study 2 compared to the CES-D which was used in Study 1.

The order of presentation of the self-report measures differed according to the recruitment method. Participants recruited via the ARC Study first attended to the baseline interview questions, commencing with the demographic questions, followed by the MINI, and finishing with the ASSIST. The baseline interview also included other questions.
relevant to the ARC Study. These participants then completed a self-report questionnaire containing the other measures for this study in the following order: SPSRQ; BFNE-II; BC; AE; LSAS; BDI-II. The self-report questionnaire also included other scales relevant to the ARC Study. The total time needed to complete both the clinical interview and self-report questionnaire (which together comprised the entire baseline interview for the ARC Study) ranged from approximately 90 minutes to three hours.

Participants recruited via SWIN-SAG were required to complete the BFNE-II as part of the assessment process for the group treatment for SAD at the Swinburne University Psychology Clinic. For these participants, the order of completion of the self-report questionnaire provided in relation to this study was as follows: demographic questions; LSAS; SPSRQ; AE; BC; BDI-II; ASSIST. All other clinical participants completed the self-report questionnaire in the same order, with the BFNE-II added as the last measure. Completion of the self-report questionnaire took between 20 to 30 minutes. The procedure for obtaining the healthy control group is set out above under “Participants”.

Results

Preliminary Data Analysis

Missing data. The statistics package IBM SPSS Statistics 20 was used for all data analyses. Preliminary data analyses were conducted on all measures. SPSS Descriptives was run to ascertain what percentage of values was missing for each continuous variable (apart from “age”). Missing percentages for some of the variables included cases where at least 80% of items on a measure, or entire measures were not completed. These were: SRSPQ (one participant did not respond to the last 15 out of the 35 items comprising this measure); AE (five participants did not respond to any items on this measure); BDI-II (one participant did not respond to any items on this measure); BCOPE (one participant did not respond to any items on this measure); and ASSIST (four participants did not respond to any items on this measure). These participants were not included in analyses involving those particular measures, but were not excluded from the sample overall. Including these missing items, the following scales and subscales had at least 5% missing values: LSAS Anxiety, LSAS Avoidance, LSAS Total, SR, AE Positive and AE Negative.
The Expectation Maximisation (EM) imputation method available on SPSS was used to replace missing data on all relevant measures (Tabachnick & Fidell, 2007). Due to the high and significant correlation between LSAS Anxiety and LSAS Avoidance subscales (.795; \( p < .001 \)), only the LSAS Total scale was used. The largest subscales were used for imputing missing values for: AE (the four categorised positive subscales were combined into an AE Positive Total subscale, as were the four categorised negative subscales); and for BCOPE (eight categorised adaptive subscales were combined into a BCOPE Adaptive subscale, and five maladaptive subscales were combined into a BCOPE Maladaptive subscale). Cases with all missing or at least 80% missing values for particular measures (as set out in the previous paragraph), were deleted for the purpose of using the EM imputation method for each of those variables. This resulted in no missing data (and therefore no need to use the EM imputation method) for the following scales or subscales: all ASSIST scales, and BCOPE Maladaptive. Little’s MCAR test was used to check if missing values were completely at random. No significant results were found for any of the measures to which the EM method was applied, indicating all missing values were random. Using the EM technique also ensured that all data values were within the required range.

**Comorbid group differences.** Due to the high percentage of ARC Study participants comprising the comorbid (SAD-SUD) group (77.2%, or 44 out of a total of 57 comorbid clinical participants), differences between ARC Study participants and all other SAD-SUD participants were explored. Independent-samples t-tests were conducted to assess differences between the two comorbid groups on all continuous variables. There were no significant differences on: Age, LSAS, BFNE-II, BDI-II, SR, SP, AEP, AEN, BCA, BCM, ASSIST Alcohol, or ASSIST Cannabis. Results are shown in Table 9. The chi-square test for independence was conducted to determine if there were significant differences across gender between the two groups, ARC Study participants and all other comorbid group participants. There was no significant difference in proportions of males to females between the two groups, \( \chi^2 (1, n = 57) = 1.309, p = .253 \). On this basis, there was no need to separate the ARC Study participants from the other clinical comorbid participants.
Table 9

Means and Standard Deviations for all Continuous Variables among ARC Study Participants and all Other Comorbid Group Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>ARC STUDY (n = 44)</th>
<th>ALL OTHER COMORBID (n=13)</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>32.93 (6.19)</td>
<td>30.54 (7.45)</td>
<td>1.170</td>
<td>55</td>
<td>.247</td>
</tr>
<tr>
<td>LSAS</td>
<td>81.26 (24.57)</td>
<td>74.08 (24.45)</td>
<td>.927</td>
<td>55</td>
<td>.358</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>33.36 (11.46)</td>
<td>40.00 (7.79)</td>
<td>-1.953</td>
<td>55</td>
<td>.056</td>
</tr>
<tr>
<td>BDI-II</td>
<td>30.73 (10.96)</td>
<td>27.08 (10.43)</td>
<td>1.066</td>
<td>55</td>
<td>.291</td>
</tr>
<tr>
<td>SR</td>
<td>7.54 (3.59)</td>
<td>8.89 (2.72)</td>
<td>-1.250</td>
<td>55</td>
<td>.217</td>
</tr>
<tr>
<td>SP</td>
<td>13.64 (3.64)</td>
<td>14.15 (3.34)</td>
<td>-.458</td>
<td>55</td>
<td>.649</td>
</tr>
<tr>
<td>AEP</td>
<td>81.19 (17.45)</td>
<td>85.31 (16.14)</td>
<td>-.754</td>
<td>55</td>
<td>.454</td>
</tr>
<tr>
<td>AEN*</td>
<td>49.29 (14.50)</td>
<td>54.95 (7.97)</td>
<td>-1.787</td>
<td>37.883</td>
<td>.082</td>
</tr>
<tr>
<td>BCA</td>
<td>37.22 (8.26)</td>
<td>39.99 (8.68)</td>
<td>-1.049</td>
<td>55</td>
<td>.299</td>
</tr>
<tr>
<td>BCM</td>
<td>25.55 (4.86)</td>
<td>24.38 (3.01)</td>
<td>.813</td>
<td>55</td>
<td>.419</td>
</tr>
<tr>
<td>ASSIST Alcohol</td>
<td>16.07 (14.57)</td>
<td>20.23 (9.98)</td>
<td>-1.178</td>
<td>28.699</td>
<td>.248</td>
</tr>
<tr>
<td>ASSIST Cannabis*</td>
<td>14.86 (13.81)</td>
<td>6.69 (12.49)</td>
<td>1.913</td>
<td>55</td>
<td>.061</td>
</tr>
</tbody>
</table>

Note. Only statistics for ASSIST Alcohol and ASSIST Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; BDI-II = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; ASSIST Alcohol = risky alcohol use; ASSIST Cannabis = risky cannabis use. *

Depression measure. As previously described, the Healthy Control Group was created by selection from Study 1 participants who completed a different depression measure (CES-D) compared to clinical participants in this study who completed the BDI-II. To utilise data about depression for all four groups being compared in this study (SAD,
SAD-SUD ALCOHOL, SAD-SUD OTHER and CONTROL), both these continuous variables were collapsed into three groups: no depression, mild depression and major depression. The respective cut off scores for each measure were obtained from relevant literature as described in the Method section of this chapter. A new categorical variable, “New Depression” was created. This allowed for comparisons to be made on a depression measure across all four groups. For analyses within each group, the original depression measure was retained.

**Descriptive statistics.** Differences between the four groups on demographic characteristics were examined. Given the theoretical importance of age, gender and depression, differences between the four groups on these variables were examined first.

**Age, gender and depression differences.** The chi-square test for independence was conducted to determine if there were significant differences across gender between the four groups, SAD-SUD Alcohol, SAD-SUD Other, SAD and CONTROL. Frequencies and percentages of males and females in the four groups are set out in Table 10.

<table>
<thead>
<tr>
<th>GENDER</th>
<th>SAD-SUD Alcohol</th>
<th>SAD-SUD Other</th>
<th>SAD</th>
<th>CONTROL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 18</td>
<td>n = 39</td>
<td>n = 27</td>
<td>n = 30</td>
</tr>
<tr>
<td>MALE</td>
<td>frequency (percent)</td>
<td>6 (33.3)</td>
<td>26 (66.7)</td>
<td>13 (48.1)</td>
</tr>
<tr>
<td>FEMALE</td>
<td>frequency (percent)</td>
<td>12 (66.7)</td>
<td>13 (33.3)</td>
<td>14 (51.9)</td>
</tr>
</tbody>
</table>

There was a significant difference in the proportions of men relative to women across the four groups, \( \chi^2 (3, n = 114) = 12.403, p = .006 \). Therefore, gender was subsequently controlled in all data analyses comparing the four groups.

The chi-square test for independence was also conducted to determine if there were significant differences across depression (the “New Depression” variable) between the four groups. However, the assumption for minimum cell count was violated in two of the cells. Therefore, a one way ANOVA was conducted to explore differences between the four
groups across depression. Levene’s test for homogeneity of variances indicated violation of this assumption, \( Levene F (3, 109) = 4.243, p < .01 \). Thus the Welch statistic was used which confirmed there was a significant difference between the four groups across depression, \( F (3, 48.741) = 76.352, p < .001 \). Next, post hoc comparisons were made using the Games-Howell test (due to the violation of the assumption of homogeneity of variances). The CONTROL group had a significantly lower mean score on depression \( (M = .20, SD = .48) \) compared to the other three groups: SAD-SUD Alcohol \( (M = 1.67, SD = .59) \), SAD-SUD Other \( (M = 1.82, SD = .39) \), and SAD \( (M = 1.58, SD = .58) \). Therefore, depression was subsequently controlled in all data analyses comparing the four groups.

A one way ANOVA was conducted to explore differences across the four groups on age. Levene’s test for homogeneity of variances indicated violation of this assumption, \( Levene F (3, 110) = 6.722, p < .001 \). Therefore, the Welch statistic was used which confirmed there was a significant difference between the four groups across age, \( F (3, 52.695) = 15.281, p < .001 \). Post hoc comparisons were made using the Games-Howell test (due to the violation of the assumption of homogeneity of variances). The CONTROL group had a significantly lower mean score on age \( (M = 22.47, SD = 6.76) \) compared to the other three groups (means and standard deviations for age are reported in Tables 11-14). Age was subsequently controlled in all data analyses comparing the four groups.

**Other demographic factors.** Group differences with respect to other demographic factors are presented in Table 11. Chi-square tests were used to determine if there were any significant differences across all other demographic variables.
Table 11

*Other Demographic Features of the Four Groups*

<table>
<thead>
<tr>
<th>Demographic Factor</th>
<th>SAD-SUD Alcohol n = 18</th>
<th>SAD-SUD Other n = 39</th>
<th>SAD n = 27</th>
<th>CONTROL n = 30</th>
<th>χ²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Country of birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>14 (77.8)</td>
<td>33 (84.6)</td>
<td>17 (63.0)</td>
<td>27 (90.0)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>4 (22.2)</td>
<td>6 (15.4)</td>
<td>10 (37.0)</td>
<td>3 (10.0)</td>
<td>7.298*</td>
</tr>
<tr>
<td>Relationship</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6 (33.3)</td>
<td>14 (35.9)</td>
<td>7 (26.9)</td>
<td>13 (43.3)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>12 (66.7)</td>
<td>25 (64.1)</td>
<td>19 (73.1)</td>
<td>17 (56.7)</td>
<td>1.681</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>7 (38.9)</td>
<td>10 (25.6)</td>
<td>15 (55.6)</td>
<td>23 (79.3)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>11 (61.1)</td>
<td>29 (74.4)</td>
<td>12 (44.4)</td>
<td>6 (20.7)</td>
<td>20.379*</td>
</tr>
<tr>
<td>Employment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3 (16.7)</td>
<td>8 (20.5)</td>
<td>22 (84.6)</td>
<td>30 (100.0)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>15 (83.3)</td>
<td>31 (79.5)</td>
<td>4 (15.4)</td>
<td>0 (0.0)</td>
<td>63.369*</td>
</tr>
</tbody>
</table>

*Note.* Total N = 114. Data were missing for 0-1 cases in some categories; *ª* = Violation of minimum expected cell count; Relationship = married or de facto; Education = completed high school or university qualification; Employment = employed, student, or other. *p < .001.

When testing for group differences with respect to “country of birth” using the chi-square test for independence, the assumption for minimum cell count was violated in one of the cells. Therefore, a one way ANOVA was conducted to explore differences across the four groups for country of birth. Levene’s test for homogeneity of variances indicated violation of this assumption, Levene F(3, 110) = 8.452, p < .001. Therefore, the Welch statistic was used, however this indicated no significant differences across the four groups on country of birth, F(3, 50.950) = 2.079, p = .115. There were also no significant differences among the groups for relationship status. However, there were significant differences across the four groups for education level and employment status. As there were already three covariates for the proposed statistical analyses (age, gender and depression), group differences on education level and employment status were not able to be taken into account in the data analyses without losing statistical power.
Continuous variables. The following four tables show means, standard deviations, score ranges, skewness and kurtosis for the continuous variables in each of the four groups. As seen in Table 12, the skewness figures (greater than 1.00) for BCM (maladaptive coping) and Cannabis, as well as the kurtosis figures (greater than -1.00 or greater than 1.00) for Age, BCA (adaptive coping), BCM and Alcohol suggest some question of normality of these variables. It is also noted that the skewness figures for SR (sensitivity to reward) and SP (sensitivity to punishment) were approaching 1.00 and -1.00 respectively; and the kurtosis figures for SR and AEP (positive alcohol expectancies) were approaching 1.00 and -1.00 respectively, indicating the possible non normality of the distributions for these variables as well.

Table 12

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Score Range</th>
<th>Skewness (SE=.54)</th>
<th>Kurtosis (SE = 1.04)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>32.17</td>
<td>6.24</td>
<td>21-40</td>
<td>-.43</td>
<td>-1.17</td>
</tr>
<tr>
<td>LSAS</td>
<td>78.39</td>
<td>23.32</td>
<td>40-132</td>
<td>.40</td>
<td>.16</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>35.28</td>
<td>12.83</td>
<td>7-48</td>
<td>-.87</td>
<td>-.37</td>
</tr>
<tr>
<td>BDI-II</td>
<td>27.83</td>
<td>11.50</td>
<td>5-46</td>
<td>-.11</td>
<td>-.66</td>
</tr>
<tr>
<td>SR</td>
<td>7.83</td>
<td>3.79</td>
<td>2-16</td>
<td>.95</td>
<td>.92</td>
</tr>
<tr>
<td>SP</td>
<td>13.11</td>
<td>4.17</td>
<td>5-18</td>
<td>-.99</td>
<td>-.07</td>
</tr>
<tr>
<td>AEP</td>
<td>88.89</td>
<td>13.30</td>
<td>66-110</td>
<td>-.25</td>
<td>-.92</td>
</tr>
<tr>
<td>AEN</td>
<td>57.49</td>
<td>12.44</td>
<td>39-85</td>
<td>.58</td>
<td>-.20</td>
</tr>
<tr>
<td>BCA</td>
<td>36.54</td>
<td>8.35</td>
<td>25-51</td>
<td>.57</td>
<td>-1.00</td>
</tr>
<tr>
<td>BCM</td>
<td>25.17</td>
<td>3.96</td>
<td>20-37</td>
<td>1.63</td>
<td>3.78</td>
</tr>
<tr>
<td>Alcohol</td>
<td>33.89</td>
<td>3.63</td>
<td>28-39</td>
<td>-.16</td>
<td>-1.21</td>
</tr>
<tr>
<td>Cannabis</td>
<td>10.94</td>
<td>14.05</td>
<td>0-39</td>
<td>1.05</td>
<td>-.38</td>
</tr>
</tbody>
</table>

Note. n = 18. Only descriptive statistics for Alcohol and Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; BDI-II = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; Alcohol = risky alcohol use; Cannabis = risky cannabis use.
As seen in Table 13, the skewness figures (greater than -1.00) for BFNE-II (fear of negative evaluation) and SP (sensitivity to punishment), as well as the kurtosis figures (greater than 1.00 or greater than -1.00) for BFNE-II, SP, AEP (positive alcohol expectancies), and Cannabis indicate some question of normality of these variables. It is also evident that the skewness figure for AEP was approaching -1.00; and the kurtosis figure for Alcohol was approaching -1.00 indicating the possible non normality of the distributions for these variables as well.

Table 13

Means, Standard Deviations, Score Ranges, Skewness and Kurtosis Values for all Continuous Variables in the Group with all other Comorbid Participants (SAD-SUD Other)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Score Range</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>STAT</td>
<td>SE</td>
<td>STAT</td>
<td>SE</td>
<td>STAT</td>
</tr>
<tr>
<td>Age</td>
<td>32.49</td>
<td>6.70</td>
<td>21- 49</td>
<td>.49</td>
<td>.38</td>
</tr>
<tr>
<td>LSAS</td>
<td>80.20</td>
<td>25.32</td>
<td>23-132</td>
<td>-.17</td>
<td>.38</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>34.69</td>
<td>10.27</td>
<td>6-48</td>
<td>-1.00</td>
<td>.38</td>
</tr>
<tr>
<td>BDI-II</td>
<td>30.85</td>
<td>10.57</td>
<td>11-54</td>
<td>-.07</td>
<td>.38</td>
</tr>
<tr>
<td>SR</td>
<td>7.85</td>
<td>3.32</td>
<td>0-16</td>
<td>.16</td>
<td>.38</td>
</tr>
<tr>
<td>SP</td>
<td>14.05</td>
<td>3.24</td>
<td>5-18</td>
<td>-1.21</td>
<td>.38</td>
</tr>
<tr>
<td>AEP (n=36)</td>
<td>78.82</td>
<td>17.93</td>
<td>19-111</td>
<td>-.98</td>
<td>.39</td>
</tr>
<tr>
<td>AEN (n=36)</td>
<td>47.23</td>
<td>12.64</td>
<td>15-72</td>
<td>-.20</td>
<td>.39</td>
</tr>
<tr>
<td>BCA</td>
<td>38.46</td>
<td>8.40</td>
<td>25-62</td>
<td>.65</td>
<td>.38</td>
</tr>
<tr>
<td>BCM</td>
<td>25.33</td>
<td>4.79</td>
<td>12-34</td>
<td>-.50</td>
<td>.38</td>
</tr>
<tr>
<td>Alcohol</td>
<td>9.23</td>
<td>8.55</td>
<td>0-26</td>
<td>.74</td>
<td>.38</td>
</tr>
<tr>
<td>Cannabis</td>
<td>13.95</td>
<td>13.84</td>
<td>0-39</td>
<td>.49</td>
<td>.38</td>
</tr>
</tbody>
</table>

Note. n = 39 unless otherwise specified. STAT = Statistic; SE = Standard Error. Only descriptive statistics for Alcohol and Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; BDI-II = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; Alcohol = risky alcohol use; Cannabis = risky cannabis use.
As seen in Table 14, the skewness figures (greater than -1.00 or 1.00) for BFNE-II (fear of negative evaluation), SP (sensitivity to punishment), AEP (positive alcohol expectancies), Alcohol and Cannabis, as well as the kurtosis figures (greater than 1.00) for BFNE-II, AEP, Alcohol and Cannabis suggested some question of normality of these variables. Moreover, the skewness figure for SR (sensitivity to reward) was approaching 1.00, as was the kurtosis figure for BCM (maladaptive coping), indicating the possible non normality of the distributions for these variables as well.

Table 14

Means, Standard Deviations, Score Ranges, Skewness and Kurtosis Values for all Continuous Variables in the Social Anxiety Group (SAD)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Score Range</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>STAT</td>
<td>SE</td>
<td>STAT</td>
<td>SE</td>
</tr>
<tr>
<td>Age</td>
<td>33.78</td>
<td>11.91</td>
<td>18-59</td>
<td>.49 .45</td>
<td>-.78 .87</td>
</tr>
<tr>
<td>LSAS</td>
<td>88.42</td>
<td>20.05</td>
<td>47-131</td>
<td>.12 .45</td>
<td>-.34 .87</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>38.46</td>
<td>7.89</td>
<td>14-48</td>
<td>-1.26 .45</td>
<td>2.37 .87</td>
</tr>
<tr>
<td>BDI-II (n=26)</td>
<td>25.48</td>
<td>12.01</td>
<td>4-50</td>
<td>.38 .46</td>
<td>-.55 .89</td>
</tr>
<tr>
<td>SR (n=26)</td>
<td>5.07</td>
<td>2.72</td>
<td>2-12</td>
<td>.95 .46</td>
<td>.42 .89</td>
</tr>
<tr>
<td>SP (n=26)</td>
<td>15.52</td>
<td>1.85</td>
<td>11-18</td>
<td>-1.00 .46</td>
<td>.41 .89</td>
</tr>
<tr>
<td>AEP (n=25)</td>
<td>74.76</td>
<td>18.30</td>
<td>19-106</td>
<td>-1.29 .46</td>
<td>2.64 .90</td>
</tr>
<tr>
<td>AEN (n=25)</td>
<td>43.91</td>
<td>12.85</td>
<td>15-67</td>
<td>-.05 .46</td>
<td>-.10 .90</td>
</tr>
<tr>
<td>BCA (n=26)</td>
<td>34.96</td>
<td>8.06</td>
<td>22-51</td>
<td>.50 .46</td>
<td>-.14 .89</td>
</tr>
<tr>
<td>BCM (n=26)</td>
<td>25.00</td>
<td>4.08</td>
<td>19-36</td>
<td>.88 .46</td>
<td>.97 .89</td>
</tr>
<tr>
<td>Alcohol (n=23)</td>
<td>5.43</td>
<td>6.15</td>
<td>0-24</td>
<td>1.86 .48</td>
<td>3.24 .94</td>
</tr>
<tr>
<td>Cannabis (n=23)</td>
<td>1.39</td>
<td>3.43</td>
<td>0-15</td>
<td>3.23 .48</td>
<td>11.57 .94</td>
</tr>
</tbody>
</table>

Note. n = 27 unless otherwise specified. STAT = Statistic; SE = Standard Error. Only descriptive statistics for Alcohol and Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; BDI-II = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; Alcohol = risky alcohol use; Cannabis = risky cannabis use.
As seen in Table 15, the skewness figures (greater than -1.00 or 1.00) for Age, CES-D (depression), SP (sensitivity to punishment), AEP (positive alcohol expectancies), BCM (maladaptive coping) and Cannabis; as well as the kurtosis figures (greater than 1.00 or -1.00) for Age, LSAS (social anxiety), CES-D (depression), AEP, BCA (adaptive coping), BCM (maladaptive coping), Alcohol and Cannabis; indicated these variables were possibly not normally distributed. The number of variables with extreme skewness or kurtosis in the CONTROL group is not surprising given that it was specifically chosen with low cut offs to indicate an absence of social anxiety and problematic alcohol or cannabis use.

Table 15

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Score Range</th>
<th>Skewness (SE=.43)</th>
<th>Kurtosis (SE = .83)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>22.47</td>
<td>6.76</td>
<td>18-44</td>
<td>1.91</td>
<td>2.99</td>
</tr>
<tr>
<td>LSAS</td>
<td>13.75</td>
<td>9.53</td>
<td>0-29</td>
<td>-.07</td>
<td>-1.27</td>
</tr>
<tr>
<td>BFNE-II</td>
<td>14.43</td>
<td>11.28</td>
<td>0-42</td>
<td>.51</td>
<td>-.56</td>
</tr>
<tr>
<td>CES-D</td>
<td>9.92</td>
<td>7.80</td>
<td>0-32</td>
<td>1.21</td>
<td>1.08</td>
</tr>
<tr>
<td>SR</td>
<td>7.41</td>
<td>4.44</td>
<td>1-17</td>
<td>.74</td>
<td>.01</td>
</tr>
<tr>
<td>SP</td>
<td>6.66</td>
<td>4.60</td>
<td>0-18</td>
<td>1.05</td>
<td>.78</td>
</tr>
<tr>
<td>AEP</td>
<td>68.40</td>
<td>20.52</td>
<td>19-104</td>
<td>-1.14</td>
<td>1.61</td>
</tr>
<tr>
<td>AEN</td>
<td>38.50</td>
<td>13.34</td>
<td>15-62</td>
<td>-.28</td>
<td>-.34</td>
</tr>
<tr>
<td>BCA</td>
<td>44.80</td>
<td>8.80</td>
<td>16-64</td>
<td>-.79</td>
<td>3.24</td>
</tr>
<tr>
<td>BCM</td>
<td>20.59</td>
<td>5.84</td>
<td>10-40</td>
<td>1.25</td>
<td>3.30</td>
</tr>
<tr>
<td>Alcohol</td>
<td>4.61</td>
<td>3.06</td>
<td>0-9</td>
<td>-.03</td>
<td>-1.38</td>
</tr>
<tr>
<td>Cannabis</td>
<td>.07</td>
<td>.37</td>
<td>0-2</td>
<td>5.48</td>
<td>30.00</td>
</tr>
</tbody>
</table>

*Note. n = 30. Only descriptive statistics for Alcohol and Cannabis are included as other substance use was not analysed according to the parameters of this study. LSAS = social anxiety; BFNE-II = fear of negative evaluation; CES-D = depression; SR = reward sensitivity; SP = punishment sensitivity; AEP = positive alcohol expectancies; AEN = negative alcohol expectancies; BCA = adaptive coping strategies; BCM = maladaptive coping strategies; Alcohol = risky alcohol use; Cannabis = risky cannabis use.*
Normality and homogeneity of variance. The skewness and kurtosis figures for the continuous variables in each of the four groups (see Tables 11 – 14 above), indicated there was some question of normality for some variables, including at least one of the proposed DVs in each of the four groups. This was confirmed by inspection of histograms and normal probability plots. The intended data analyses involved looking for significant mean differences between the four groups on six dependent variables (DVs), that is, the six individual difference variables (SR, SP, AEP, AEN, BCA and BCM) via analyses of variance or covariance. These techniques are reasonably tolerant of violations of the assumptions of normality and homogeneity of variance with large enough sample sizes and similar group sizes (Pallant, 2005; Tabachnick & Fidell, 2007). In this study the largest group (SAD-SUD Other) had a sample size of 39 compared to the smallest group (SAD-SUD Alcohol) with a sample size of 18 (less than half the size). Also, two of the DVs (SP and SR) in the smallest group (SAD-SUD Alcohol) had higher variances compared to the largest group (SAD-SUD Other). For these reasons, there was some risk of violation of the assumptions of normality and homogeneity of variance. Therefore, the use of the “bootstrap” technique when analysing the data to detect differences between the groups was contemplated.

The bootstrap technique is a resampling procedure in which random samples of the actual data are repeatedly drawn a large number of times (in this study 1000 samples were drawn each time) to empirically estimate the distribution of the statistic of interest (Kelley, 2005). The bootstrap approach does not make assumptions about the shape of the distribution of observed scores and therefore also does not assume equality of variances (based on the scores being normally distributed). The only assumption that applies to bootstrapping is that the actual sample is representative from the population from which it was taken (Neal & Simons, 2007). Therefore, it was decided to use the bootstrap procedure when testing for group differences.

Assumption testing for analyses of covariance. Due to the need to control for age, gender and depression differences amongst the four groups, further assumption testing was conducted to proceed with analyses of covariance (ANCOVA), rather than analyses of variance (ANOVA) to test for differences on the individual difference variables (SR, SP, AEP, AEN, BCA, BCM).
The ANCOVA model assumes reliability of the covariates; an absence of multicollinearity amongst the covariates; linearity between the dependent variable(s) and the covariates for each group, as well as linearity amongst the covariates; and homogeneity of regression slopes (Tabachnick & Fidell, 2007).

Two of the covariates were gender and age, and the reliability of these variables can usually be justified (Tabachnick & Fidell, 2007). The third covariate (New Depression) was a categorical variable derived from two reliable depression measures. The Cronbach alpha value for the BDI-II (completed by all clinical participants) was .91 and for the CES-D (completed by the nonclinical participants in the CONTROL group), it was .88. In addition, both the BDI-II and CES-D were highly correlated with the “New Depression” variable in all four groups. There were large positive associations between BDI-II and New Depression in the SAD-SUD Alcohol group ($r = .784, p < .01$), in the SAD-SUD Other group ($r = .723, p < .01$), and in the SAD group ($r = .792, p < .01$). There was a large positive association between CES-D and New Depression in the CONTROL group ($r = .871, p < .01$). Thus, all the covariates were considered to be reliable measures.

Both Pearson’s and Spearman’s correlation coefficients were checked amongst pairs of the three covariates (Age, Gender and New Depression) for each of the four groups. There was a significant positive association between Gender and New Depression in the SAD-SUD Alcohol group using both types of correlational analyses: ($r = .612, p < .01$ and Spearman’s $r = .623, p < .01$). As these correlation coefficients were less than .8 (Pallant, 2005), the assumption of the absence of multicollinearity amongst the covariates was met.

Linearity between pairs of covariates and between covariate and dependent variable (SR, SP, AEP, AEN, BCA, BCM) pairs were checked for each of the four groups by way of generation of scatterplots. All pairs demonstrated linearity. Therefore this ANCOVA assumption was also met.

The assumption of homogeneity of regression slopes was assessed statistically by checking to see if there was a significant interaction between each covariate and dependent variable in each of the four groups (Pallant, 2005). There were no significant interactions. Therefore this ANCOVA assumption was also upheld.
**Analysis of Covariance (ANCOVA)**

ANCOVA models were used to test for group differences amongst the four groups (SAD-SUD Alcohol, SAD-SUD Other, SAD, and CONTROL) on the six individual difference variables: sensitivity to reward (SR), sensitivity to punishment (SP), positive alcohol expectancies (AEP), negative alcohol expectancies (AEN), adaptive coping strategies (BCA), and maladaptive coping strategies (BCM). These models included age, depression (the “New Depression” variable) and gender as covariates, as these variables could theoretically impact the individual difference variables across the four groups. Due to concerns about possible violations of the assumptions of normality and homogeneity of variance, the bootstrapping procedure was used on 1000 resamples for each model, with a 95% Confidence Interval level, and a Bias-corrected and accelerated (BCa) Confidence Interval type.

**Personality factors.** Table 16 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on sensitivity to reward (SR).

Table 16

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Mean</th>
<th>Standard Error</th>
<th>BCa 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAD-SUD Alcohol</td>
<td>6.95</td>
<td>.96</td>
<td>5.30 – 8.67</td>
</tr>
<tr>
<td>n = 18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD-SUD Other</td>
<td>6.40</td>
<td>.82</td>
<td>4.86 – 7.84</td>
</tr>
<tr>
<td>n = 39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD</td>
<td>4.22</td>
<td>.69</td>
<td>2.76 – 5.39</td>
</tr>
<tr>
<td>n = 25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>8.87</td>
<td>1.14</td>
<td>6.80 – 11.31</td>
</tr>
<tr>
<td>n = 30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. a = based on 999 samples.*

This ANCOVA model revealed significant group differences on sensitivity to reward (SR): $F(3,104) = 4.290, p = .007, \eta_p^2 = .110$ (medium to large effect size); after covarying for gender, $F(1,104) = 1.891, p = .172, \eta_p^2 = .018$ (small effect size); age, $F$
(1,104) = 6.098, \( p = .015, \eta_p^2 = .055 \) (small to medium effect size); and depression, \( F(2,104) = 3.999, \ p = .021, \eta_p^2 = .071 \) (medium effect size). Bootstrapped pairwise comparisons revealed that the SAD group was significantly lower on SR compared to the other 3 groups (SAD-SUD Alcohol, SAD-SUD Other, and CONTROL). There were no significant differences on SR between the other 3 groups. This result supported the hypothesis about group differences on sensitivity to reward as the SAD group had a lower sensitivity to reward compared to all three other groups.

Table 17 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on sensitivity to punishment (SP).

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Mean</th>
<th>Standard Error</th>
<th>BCa 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAD-SUD Alcohol</td>
<td>11.93</td>
<td>1.21</td>
<td>9.34 - 14.06</td>
</tr>
<tr>
<td>( n = 18 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD-SUD Other</td>
<td>12.93</td>
<td>.78</td>
<td>11.45 - 14.27</td>
</tr>
<tr>
<td>( n = 39 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD</td>
<td>14.56</td>
<td>.65</td>
<td>13.16 - 15.67</td>
</tr>
<tr>
<td>( n = 25 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>8.30</td>
<td>1.33</td>
<td>5.93 - 10.76</td>
</tr>
<tr>
<td>( n = 30 )</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

This ANCOVA model revealed significant group differences on sensitivity to punishment (SP): \( F(3,104) = 5.875, \ p = .001, \eta_p^2 = .145 \) (large effect size); after covarying for gender, \( F(1,104) = 1.221, \ p = .272, \eta_p^2 = .012 \) (small effect size); age, \( F(1,104) = .568, \ p = .453, \eta_p^2 = .005 \) (very small effect size); and depression, \( F(2,104) = 4.335, \ p = .016, \eta_p^2 = .077 \) (medium effect size). Bootstrapped pairwise comparisons revealed that the SAD group was significantly higher on SP compared to the other 3 groups (SAD-SUD Alcohol, SAD-SUD Other, and CONTROL). In addition, the SAD-SUD Other group was significantly higher on SP than the CONTROL group. The hypothesis about group differences on sensitivity to punishment was partially supported as the SAD group had a
higher SP than all three other groups (and not just the two comorbid groups as hypothesised), and only one of the comorbid groups, SAD-SUD Other was significantly higher on SP compared to the CONTROL group (and not both comorbid groups as hypothesised).

**Alcohol expectancies.** Table 18 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on positive alcohol expectancies.

Table 18

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Mean</th>
<th>Standard Error</th>
<th>BCa 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAD-SUD Alcohol</td>
<td>90.03</td>
<td>4.02</td>
<td>82.47 – 97.98</td>
</tr>
<tr>
<td>n = 18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD-SUD Other</td>
<td>80.84</td>
<td>4.42</td>
<td>72.10 – 90.03</td>
</tr>
<tr>
<td>n = 36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD</td>
<td>75.88</td>
<td>5.11</td>
<td>65.17 – 87.61</td>
</tr>
<tr>
<td>n = 24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>64.80</td>
<td>6.90</td>
<td>49.12 – 78.02</td>
</tr>
<tr>
<td>n = 30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* a = based on 999 samples.

This ANCOVA model revealed significant group differences on positive alcohol expectancies (AEP): $F (3,100) = 3.636, p = .015, \eta_p^2 = .098$ (medium effect size); after covarying for gender, $F (1,100) = .244, p = .622, \eta_p^2 = .002$ (extremely small effect size); age, $F (1,100) = .000, p = .994, \eta_p^2 = .000$ (nil effect size); and depression, $F (2,100) = .425, p = .655, \eta_p^2 = .008$ (very small effect size). Bootstrapped pairwise comparisons revealed that the SAD-SUD Alcohol group was significantly higher on AEP than both the SAD group and the CONTROL group. There were no other significant differences between the groups. The hypothesis about group differences on positive alcohol expectancies was partially supported as the SAD-SUD Alcohol group was significantly higher on AEP than two of the three groups (SAD and CONTROL), but the SAD-SUD Alcohol group was not
significantly higher on AEP than the other comorbid group (SAD-SUD Other). Also, the SAD-SUD Other group was not significantly higher on AEP than both the SAD and CONTROL groups.

Table 19 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on negative alcohol expectancies.

Table 19

<table>
<thead>
<tr>
<th>Group Differences on Negative Alcohol Expectancies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bootstrap Statistics</td>
</tr>
<tr>
<td>Group</td>
</tr>
<tr>
<td>--------------------------------------------------</td>
</tr>
<tr>
<td>SAD-SUD Alcohol n = 18</td>
</tr>
<tr>
<td>SAD-SUD Other n = 36</td>
</tr>
<tr>
<td>SAD n = 24</td>
</tr>
<tr>
<td>CONTROL n = 30</td>
</tr>
</tbody>
</table>

This ANCOVA model revealed significant group differences on negative alcohol expectancies (AEN): $F (3,100) = 5.162, p = .002, \eta^2_p = .134$ (medium-to-large effect size); after covarying for gender, $F (1,100) = 2.568, p = .112, \eta^2_p = .025$ (small effect size); age, $F (1,100) = .297, p = .587, \eta^2_p = .003$ (negligible effect size); and depression, $F (2,100) = .013, p = .987, \eta^2_p = .000$ (nil effect size). Bootstrapped pairwise comparisons revealed that the SAD-SUD Alcohol group was significantly higher on AEN than all three other groups (SAD-SUD Other, SAD, and CONTROL). There were no other significant differences between the groups. The hypothesis about group differences on negative alcohol expectancies was partially supported as the SAD-SUD Alcohol group had significantly higher AEN than all three other groups. However, the two other clinical groups (SAD-SUD Other and SAD) did not have significantly higher AEN than the CONTROL group as hypothesised.
The ANCOVA model on AEN was the only one to reveal a significant difference between the two comorbid groups on any of the individual difference variables. This ANCOVA result may have been influenced by the small sample size of SAD-SUD Alcohol. Although the effect size was medium-to-large, power was reduced by entering the three covariates (age, gender, depression) simultaneously. None of the covariates were significant when entered together. Therefore it was decided to enter the covariates separately (using the bootstrapping procedure), in order to ascertain which of them may have affected this result. None of the covariates were significant when entered separately in the ANCOVA model on AEN (see Appendix I). However, the effect size for group differences in this ANCOVA model increased from medium/large to large in both instances when gender and age were each entered separately. Also, in both these instances (when each of age and gender were entered separately), an additional significant difference on AEN was found between the SAD-SUD Other group and the CONTROL group.

Coping strategies. Table 20 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on adaptive coping strategies (BCA).

Table 20

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Mean</th>
<th>Standard Error</th>
<th>BCa 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAD-SUD Alcohol</td>
<td>37.61</td>
<td>2.40</td>
<td>33.58 – 41.56</td>
</tr>
<tr>
<td>n = 18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD-SUD Other</td>
<td>40.75</td>
<td>2.23</td>
<td>37.03 – 44.49</td>
</tr>
<tr>
<td>n = 39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAD</td>
<td>36.18</td>
<td>2.08</td>
<td>32.64 – 39.39</td>
</tr>
<tr>
<td>n = 26</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>42.34</td>
<td>3.41</td>
<td>36.72 – 48.55</td>
</tr>
<tr>
<td>n = 30</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. a = based on 999 samples.*
This ANCOVA model revealed no significant group differences on adaptive coping strategies (BCA): $F(3, 105) = 2.018, p = .116, \eta_p^2 = .055$ (small to medium effect size); after covarying for gender, $F(1, 105) = 2.185, p = .142, \eta_p^2 = .020$ (small effect size); age, $F(1, 105) = .008, p = .927, \eta_p^2 = .000$ (nil effect size); and depression, $F(2, 105) = .907, p = .407, \eta_p^2 = .017$ (small effect size). The hypothesis about group differences on adaptive coping strategies was not supported as the CONTROL group did not have higher adaptive coping strategies than the other three groups.

Table 21 sets out the adjusted means (taking into account the effects of the covariates, age, gender and depression), the bootstrapped standard errors and the BCa 95% confidence intervals for each of the four groups on maladaptive coping strategies (BCM).

<table>
<thead>
<tr>
<th>Group</th>
<th>Adjusted Mean</th>
<th>Standard Error</th>
<th>BCa 95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAD-SUD Alcohol</td>
<td>22.31</td>
<td>1.47</td>
<td>18.76 – 24.92</td>
</tr>
<tr>
<td>SAD-SUD Other</td>
<td>22.26</td>
<td>1.39</td>
<td>19.04 – 25.14</td>
</tr>
<tr>
<td>SAD</td>
<td>22.42</td>
<td>1.19</td>
<td>19.93 – 24.61</td>
</tr>
<tr>
<td>CONTROL</td>
<td>24.27</td>
<td>2.19</td>
<td>19.97 – 28.33</td>
</tr>
</tbody>
</table>

Note. a = based on 998 samples.

This ANCOVA model revealed no significant group differences on maladaptive coping strategies (BCM): $F(3, 105) = .361, p = .781, \eta_p^2 = .010$ (small effect size); after covarying for gender, $F(1, 105) = 1.384, p = .242, \eta_p^2 = .013$ (small effect size); age, $F(1, 105) = .820, p = .367, \eta_p^2 = .008$ (very small effect size); and depression, $F(2, 105) = 11.612, p = .000, \eta_p^2 = .181$ (very large effect size). The hypothesis about group differences on maladaptive coping strategies was not supported as the two comorbid groups did not have higher maladaptive coping strategies than the two other groups (SAD and CONTROL), and the SAD group did not have higher maladaptive coping strategies than the CONTROL group.
Regression Analysis on Social Anxiety

One of the aims of this thesis was to explore predictors of social anxiety in the clinical and non-clinical samples to further understanding of the relationship between social anxiety and substance use. The clinical sample size \( (N = 84) \) was not large enough to examine the unique predictors of social anxiety severity using all the variables of interest. In the “Reverse Regression Analysis” of the non-clinical sample in Study 1, all the variables were able to be tested due to the sample size. In those analyses, five unique predictors of social anxiety were found. In order of strength, they were sensitivity to punishment, sensitivity to reward, depression, fear of negative evaluation, and positive alcohol expectancies. The three strongest independent variables in Study 1 (punishment sensitivity, reward sensitivity and depression) were included in the regression analysis in Study 2. Furthermore, given their potential importance in clinical populations, alcohol and cannabis use were added to these three variables to examine predictors of social anxiety in the clinical sample (in Study 2), thus maintaining adequate power in the multiple regression analysis (Tabachnick & Fidell, 2007).

Assumptions were checked prior to analysis. There was no violation of assumptions. There were no signs of multicollinearity as no two variables had bivariate correlations of .7 or higher. In addition, no Tolerance values were less than .10, nor were any VIF (Variance inflation factor) values higher than 10. The assumptions of normality, linearity, homoscedasticity and independence of residuals were checked via inspection of the residuals scatterplot and the Normal Probability Plot. There were no major deviations from normality and no apparent violations of these assumptions. The presence of outliers was checked by inspecting the Mahalanobis distances. Using an alpha level of .001, there were no values higher than the critical value \( (\chi^2 = 20.515, df = 5) \), using the number of independent variables as the degrees of freedom (Tabachnick & Fidell, 2007).

Results for the multiple regression analysis are presented in Table 22. Just under half (48%) of the variance in social anxiety (LSAS)) was explained by this model. In addition, the model was statistically significant \( (F (5, 73) = 13.475, p < .001) \). The strongest contributor was sensitivity to punishment (SP) which uniquely explained 29% of the total variance in social anxiety. The next strongest contributor was cannabis use (CAN), which uniquely explained 6% of the total variance in social anxiety in this model.
Two of the remaining independent variables, sensitivity to reward (SW) and depression (BDI), also made significant unique contributions to social anxiety. Alcohol use was not a unique predictor of social anxiety.

Table 22

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>sr²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>.13</td>
<td>.16</td>
<td>.07</td>
<td>.82</td>
<td>.413</td>
<td>.00</td>
</tr>
<tr>
<td>Cannabis</td>
<td>.47</td>
<td>.17</td>
<td>.26</td>
<td>2.85</td>
<td>.006</td>
<td>.06</td>
</tr>
<tr>
<td>SR</td>
<td>-1.52</td>
<td>.60</td>
<td>-.23</td>
<td>-2.52</td>
<td>.014</td>
<td>.05</td>
</tr>
<tr>
<td>SP</td>
<td>4.31</td>
<td>.67</td>
<td>.59</td>
<td>6.41</td>
<td>.000</td>
<td>.29</td>
</tr>
<tr>
<td>BDI-II</td>
<td>.45</td>
<td>.19</td>
<td>.22</td>
<td>2.39</td>
<td>.019</td>
<td>.04</td>
</tr>
</tbody>
</table>

Note. Alcohol = risky alcohol use; Cannabis = risky cannabis use; SR = reward sensitivity; SP = punishment sensitivity; BDI-II = depression.

In summary, when all variables were entered together in the multiple regression with LSAS as the DV, four of the five independent variables entered uniquely contributed to the prediction of social anxiety. Thus, the hypothesis was partly supported. The three variables chosen on the basis of their performance in the Study 1 multiple regression on social anxiety (LSAS), were again unique predictors of social anxiety in the clinical sample. Both sensitivity to punishment and depression made stronger contributions to social anxiety in the clinical sample (Study 2) compared to the non-clinical sample (Study 1). Reward sensitivity made a similar contribution to social anxiety in both studies. With respect to substance use variables, only cannabis was a unique predictor of social anxiety.

Discussion

Summary

To the author’s knowledge this is the first study using clinical samples to compare differences between individuals with social anxiety disorder (SAD) and individuals with SAD and a comorbid substance use disorder (SAD-SUD). The comorbid group was further
divided with group members who drank alcohol at a high risk level forming a separate group. A healthy control group was also utilised. Differences between these groups were examined in relation to the same three important biopsychosocial variables: personality (drive sensitivity), alcohol expectancies and coping strategies, explored with a non-clinical sample in Study 1. Findings in Study 2 highlight important differences between the social anxiety group, both comorbid groups and the healthy control group in relation to sensitivity to reward, sensitivity to punishment, positive and negative alcohol expectancies. No significant differences were evident across the groups in relation to adaptive or maladaptive coping strategies. With respect to negative alcohol expectancies, a further significant difference was found between the two comorbid groups, with the group with high risk alcohol use having a higher level of this variable. Finally in contrast to the findings from Study 1, risky cannabis use, but not risky alcohol use, was a unique predictor of social anxiety, together with punishment sensitivity, reward sensitivity and depression.

**Outcome of Hypothesis Testing**

**Reward and punishment sensitivity.** As hypothesised, the SAD (social anxiety disorder) group had lower sensitivity to reward than the other three groups. This finding is consistent with some findings from non-clinical studies (e.g. Kashdan, 2002; Kimbrel et al., 2010), where a negative relationship was found to exist between social anxiety and reward sensitivity. The finding is also arguably at least partially consistent with the finding from the one previous clinical study conducted with respect to the associations between drive sensitivity and social anxiety (Morgan et al., 2009), where, after further exploratory analysis, it was ascertained that the group of SAD patients had lower scores on one of the three (Behavioural Approach System (BAS) subscales, (BAS fun-seeking) compared to a group of controls. In addition, this finding is consistent with the numerous findings in research investigating associations between reward sensitivity and substance use. Such research has consistently found a positive association between these two variables among adults, or significant group differences on reward sensitivity. For example, Dissanbandara et al. (2014) found that heroin users were significantly higher on reward sensitivity compared to non-users; and Lyvers et al. (2014) found a substance dependent group
undergoing treatment were significantly higher on reward sensitivity compared to a community group of social drinkers.

Past research findings (in relation to social anxiety and reward sensitivity, and in relation to substance dependence and reward sensitivity), together with the present findings, indicate that socially anxious individuals without co-occurring substance use are low on reward sensitivity whilst the comorbid group is higher on reward sensitivity. This interpretation is also consistent with the recent findings of Nicholls et al. (2014) who via latent class analysis found that individuals with both subclinical and clinical levels of social anxiety as well as problematic substance use were also high on reward sensitivity. As the study conducted by Nicholls et al., (2014) involved a non-clinical sample, our findings extend this conclusion to clinical samples.

As hypothesised, the SAD group had a higher sensitivity to punishment than all three other groups. However, only one of the comorbid groups (the SAD-SUD Other group) was higher on punishment sensitivity compared to the control group. It was hypothesised that both comorbid groups would have a higher sensitivity to punishment compared to the CONTROL group. The comorbid high alcohol group’s adjusted mean was only slightly lower than the other comorbid’s adjusted group mean, so that it appears to have come quite close to being significantly higher on punishment sensitivity than the CONTROL group, as hypothesised. The present results are consistent with previous findings, particularly for the relationship between social anxiety and punishment sensitivity. In the one clinical study considering social anxiety and drive sensitivity (Morgan et al., 2009), the SAD group had significantly higher scores on punishment sensitivity, measured by the BIS (Behavioural Inhibition System) scale, compared to the control group. Non-clinical studies have consistently shown a positive relationship between social anxiety symptoms and punishment sensitivity across different populations (e.g., children, Coplan et al., 2006; and students, e.g., Kimbrel et al., 2008).

Previous findings with respect to punishment sensitivity and substance use are less clear, in both clinical and non-clinical studies. In group comparison clinical studies, no significant difference has been found on BIS between substance/alcohol dependants and controls (Franken et al., 2006), or on punishment sensitivity between substance dependent inpatients and community members with no alcohol or substance dependence (Perry et al.,
On the other hand, another group of substance dependent inpatients was recently found to have higher punishment sensitivity compared to community social drinkers (Lyvers, et al., 2014). Taken together, these two sets of research findings (the relationship between punishment sensitivity and social anxiety compared to the association between punishment sensitivity and substance use), offer general support to the present findings in that the SAD group had higher punishment sensitivity than the other groups.

**Alcohol expectancies.** The hypothesis relating to positive alcohol expectancies was partially supported as the comorbid group with high risk alcohol use (SAD-SUD Alcohol) had higher positive alcohol expectancies compared to the social anxiety group (SAD) and the healthy controls (CONTROL). These results confirm past findings in clinical samples as to the positive association between positive alcohol expectancies and risky alcohol use among alcohol and drug dependent treatment seekers at a residential rehabilitation facility (Gullo et al., 2010); as to the prediction by positive alcohol expectancies of alcohol consumption quantity and problems among alcohol and drug-dependent patients (Galen et al., 2001); and as to the stronger endorsement of positive expectancies by a clinical sample compared to a student sample (Li & Dingle, 2012).

The SAD-SUD Alcohol group was not significantly higher on positive alcohol expectancies than the other comorbid group (SAD-SUD Other) as hypothesised. This was despite findings as to the higher endorsement of both positive and negative alcohol expectancies in alcohol-dependent participants compared to community participants (Hasking & Oei, 2002a), and among alcohol-dependent patients in treatment for detoxification (Connor et al., 2007). However our two comorbid groups were not split on the basis of alcohol and/or substance dependence, but rather on high risk use of alcohol. This could to some extent explain the lack of significant difference on positive alcohol expectancies between the two comorbid groups in our study.

The present findings also support earlier findings in the clinical study which compared a social anxiety group with a comorbid social anxiety/alcohol use disorders group, and a normal control group (Tran & Haaga, 2002). Tran and Haaga (2002) looked at specific positive alcohol expectancies (social facilitation and tension-reduction) and found the comorbid group were higher on both expectancy types compared to both other groups, whilst the social anxiety group had higher social facilitation expectancies compared
to the normal controls. In the present study the expectancy scale measured total positive alcohol expectancies, and so findings are consistent with the findings of the Tran and Haaga (2002) with respect to the group differences on both types of positive expectancies.

In contrast, the current findings are inconsistent with another clinical study by Ham et al. (2002), which looked at differences on positive alcohol expectancies between a social anxiety group, a group of dysthymics and a group of normal controls. Ham and colleagues found that the social anxiety group had higher positive alcohol expectancies on three subscales (global positive expectancies, social facilitation and tension reduction) compared to the normal controls. In addition, the social anxiety group had significantly higher social assertiveness expectancies compared to the dysthymic group. Notably, individuals with alcohol/substance use disorders in the previous six months were excluded from Ham et al. (2002), although this exclusion criteria only resulted in the removal of one potential participant. Furthermore, there was no significant difference between the groups on alcohol consumption level. It may well be that this particular study did not attract socially anxiety individuals with an alcohol/substance use disorder comorbidity. Together, the results of Ham et al. (2002) and Tran and Haaga (2002) demonstrate that socially anxious individuals are likely to have higher social facilitation/assertiveness alcohol expectancies compared to normal controls. In the present study this was not able to be teased out as all positive expectancy subscales were grouped together as a higher order factor.

The hypothesis with regard to negative alcohol expectancies was partly confirmed as the comorbid high risk alcohol group (SAD-SUD Alcohol) had higher negative expectancies compared to all other three groups. The second part of the hypothesis was not supported as neither of the two other clinical groups, SAD or SAD-SUD Other, had higher negative alcohol expectancies than the CONTROL group. The results from our study confirm past findings in clinical studies as to the positive association between negative alcohol expectancies and alcohol dependence (Connor et al., 2007) or significant group differences between alcohol-dependent adults and community participants (Hasking & Oei, 2002a), or students (Li & Dingle, 2012).

Interestingly, the current results also demonstrate that alcohol and/or substance use disordered individuals who are high risk drinkers have higher negative alcohol expectancies compared to other alcohol and/or substance use disordered individuals. As pointed out
earlier, the criterion for the smaller comorbid group (SAD-SUD Alcohol) in Study 2 was high risk alcohol use. Both comorbid groups had some members diagnosed with an alcohol use disorder (AUD) only, and other members diagnosed with both an AUD and a substance use disorder (SUD). The larger comorbid group (SAD-SUD Other) also had members (the majority) who were diagnosed with a SUD only. Therefore, the significant difference between the two comorbid groups on negative alcohol expectancies appears to be based on the level of risky alcohol use (as measured by the ASSIST) and not according to diagnostic criteria for an AUD.

The significant difference between the two comorbid groups on negative alcohol expectancies was the only finding which distinguished between these two groups on an individual difference variable. Although further testing was conducted to determine if one or more of the covariates played a significant role in this finding, further analysis did not demonstrate the significance of age, depression or gender alone in influencing the result. Future research could look in more detail at why all three covariates had an effect but not individually.

Findings from Study 2 partially support earlier findings in the one other clinical study (Tran & Haaga, 2002) which compared a social anxiety group with a comorbid social anxiety/alcohol use disorders group, and a normal control group on negative alcohol expectancies. Tran and Haaga (2002) found that both clinical groups (the SAD group and the SAD-AUD group) had higher negative alcohol expectancies than the control group. This was in contrast to their hypothesis that the comorbid group would have lower negative expectancies compared to the two non-alcohol use disordered groups. The current results potentially extend the findings of Tran and Haaga (2002) by further specifying that comorbid individuals who drink alcohol at a high risk level have even higher negative alcohol expectancies than other comorbid (SAD-AUD or SAD-SUD) individuals. This finding suggests that there is a higher likelihood of the occurrence of negative consequences from high risk alcohol use, compared to the level of expectation of negative alcohol consequences from alcohol abuse or alcohol dependence.

The post hoc analysis on these findings did not reveal any significant role for age, gender or depression, although when each of age and gender was entered separately, the overall effect size increased and a further significant difference between the larger
comorbid group (SAD-SUD Other) and the control group was detected. This analysis suggests that age and gender may play important roles on their own such that individuals with co-occurring SAD-SUD (excluding those classified as high risk drinkers) who are male and older, may have more negative alcohol expectancies compared to healthy controls.

**Coping strategies.** In contrast to the hypothesis, the healthy control group did not have higher levels of adaptive coping strategies than the three clinical groups. The result differs from past clinical findings in relation to adaptive coping strategies and substance use, clinical findings in relation to adaptive coping strategies and social anxiety, and clinical findings in relation to social anxiety, substance use and coping strategies.

Although findings from non-clinical studies demonstrate a negative relationship between adaptive coping strategies and drinking behaviour (Feil & Hasking, 2008; Ivory & Kambouroupolos, 2012), as well as a moderating role of adaptive coping in the relationship between stress and drinking behaviour (Corbin et al., 2013), the evidence from clinical studies is less clear. An alcohol-dependent group has been found to have more total (both adaptive and maladaptive) coping strategies compared to community members (Hasking & Oei, 2002a), and treatment seeking alcohol-disordered participants had an increased likelihood of recovery if they relied more on adaptive (approach) coping (Moos & Moos, 2007). However, other studies have not demonstrated significant differences in the use of adaptive coping strategies where different treatment interventions were compared among treatment seekers for AUD (Litt et al., 2003) or cannabis dependence (Litt et al., 2005, 2008). Nevertheless, the two last cited studies did both demonstrate that using more (in terms of amounts and frequency) adaptive coping strategies did increase the likelihood of remaining abstinent.

With regard to studies investigating social anxiety and adaptive coping strategies, findings from Study 2 are not consistent with those of Thomasson and Psouni (2010) whose participants included members of social anxiety organisations. Thomasson and Psouni (2010) found that adaptive coping strategies were negatively related to the social anxiety measures. Their results are consistent with findings as to the negative relationship between social anxiety symptoms and adaptive coping strategies in socially anxious treatment seekers (Aldao et al., 2014). Their results suggest that in the present study the SAD group
as well as both the comorbid groups ought to have had lower adaptive coping strategies compared to the healthy controls.

Finally, the results from Study 2 are inconsistent with the findings of Tran and Haaga (2002) which remains the only previous clinical study investigating the role of adaptive coping strategies in the relationship between social anxiety disorder (SAD) and alcohol use disorder (AUD). The present results differ from those of Tran and Haaga (2002) who found both clinical groups (the social anxiety and the comorbid groups) had lower adaptive coping strategies compared to controls in situations where alcohol was not accessible, and the comorbid group used the least adaptive coping strategies when alcohol was accessible. One possible explanation for the insignificant findings in the current study may be due to age being controlled for, as with increasing age comes more life experience and the increased likelihood of using more adaptive coping strategies.

In contrast to the hypothesis, the two comorbid groups (SAD-SUD Alcohol and SAD-SUD Other) did not have higher maladaptive coping strategies than the SAD and CONTROL groups, and all three clinical groups did not have higher maladaptive coping than the CONTROL group. These results are inconsistent with findings of previous studies (both clinical and non-clinical) investigating maladaptive coping strategies and substance use, maladaptive coping strategies and social anxiety, and maladaptive coping strategies, social anxiety and substance use.

Findings from non-clinical studies consistently demonstrate a positive relationship between maladaptive coping strategies (excluding substance use as a maladaptive coping strategy) and substance use (e.g., Ivory & Kambouropoulos, 2012; Hundt et al., 2013). However, fewer clinical studies have conducted research in this area. Clinical studies investigating maladaptive coping strategies and substance use (excluding substance use as a maladaptive coping strategy), have demonstrated that cannabis dependent adolescents use significantly more avoidant (maladaptive) coping strategies than non-cannabis dependent adolescents (Cascone et al., 2011), and avoidant (maladaptive) coping strategies predict frequency of alcohol use among alcohol-dependent adults (Hasking & Oei, 2007).

The only clinical study that appears to have investigated maladaptive coping strategies (excluding drug and alcohol use as a maladaptive coping strategy) and social anxiety directly was conducted by Aldao et al. (2014). They found a strong association
between maladaptive avoidance strategies and social anxiety symptoms in socially anxious individuals undergoing CBT treatment for SAD. Thus, the present findings are inconsistent with the limited number of prior research studies in this area. The same explanation as to the lack of support for our hypotheses in relation to adaptive coping strategies is put forward in relation to the lack of support for our hypotheses about group differences on maladaptive coping strategies, that is, as age was a covariate, the model did not take into account that as one’s age increases, so does one’s experience such that there is likely to be a reduction of use of maladaptive coping strategies over time.

The role of covariates. Age, gender and depression were covariates in each of the six ancova models examining the role of the individual difference/vulnerability factors. Depression had the most impact with a very large effect size on the ancova model for maladaptive coping strategies, a medium effect size on the ancova models for reward sensitivity and punishment sensitivity, and a very small (the largest compared to age and gender) effect size on the ancova model for positive alcohol expectancies. Interestingly, despite the sizeable influence of depression on maladaptive coping strategies, there were no significant differences between the groups, considering the high comorbidity of depression with social anxiety disorder (Fehm et al., 2008), substance use disorder (Grant et al., 2004, 2006a), comorbid social anxiety and alcohol use (SAD-AUD) disorders (Schneier et al., 2010), and comorbid SAD and cannabis use disorders (SAD-CUD) (Buckner et al., 2012b).

Multiple regression on social anxiety (LSAS). Sensitivity to punishment was the strongest unique predictor of social anxiety out of the five predictor variables tested in the model. The choice of predictors was partly based on results from the multiple regression analysis on social anxiety conducted in Study 1 with the three strongest predictor variables from that analysis (sensitivity to punishment, sensitivity to reward and depression) tested in the model using the total clinical sample in Study 2. All three variables were again unique predictors of social anxiety with the clinical sample.

Given the theoretical importance of alcohol and cannabis use in comorbid social anxiety and substance use disorders, both these variables were also tested in the model relying on data from the clinical sample in Study 2. Interestingly, alcohol was not a significant predictor of social anxiety, whilst cannabis was the second strongest unique predictor of social anxiety (after punishment sensitivity) in this model. This result is
consistent with relatively recent findings as to the important unique role of cannabis as a problematic substance specifically in regard to social anxiety (Buckner et al., 2008a). Problematic cannabis use in socially anxious individuals is a far more recent field of research compared to studies which have been investigating co-occurring social anxiety and alcohol use in both clinical and non-clinical populations for several decades. Thus, the present findings extend those of Buckner et al. (2008a) in demonstrating that cannabis plays an important role in predicting social anxiety in clinical populations. Finally, the current results are consistent with the findings of Nicholls et al. (2014) which, via latent class analysis, demonstrated that socially anxious individuals with problematic substance use have high sensitivities to both punishment and reward.

**Limitations and Strengths**

A significant limitation in Study 2 was the demographic differences particularly across the three clinical groups and the healthy control group. Due to the requirements for the healthy control group (no social anxiety or risky substance use) drawn from the pool of Study 1 participants, it was not possible to match the three clinical groups for demographic factors with the control group. Thus, it was necessary to control for age and gender in the statistical analyses. Given the theoretical importance of depression to both social anxiety and substance use disorders, this was a third covariate in the models testing for group differences on the six shared vulnerability factors. Therefore, it was not feasible to control for the two other significant demographic differences between the groups, education level and employment status, as having more covariates would have compromised the power of the analysis. These demographic differences may have influenced the results, as group differences on the variables tested would have been more accurate if important demographic differences were also able to be controlled for. For future studies comparing a healthy control group with clinical groups, it is recommended that groups be able to be matched at least on age and gender and possibly other important demographic variables. Thus, due to practical considerations the importance of other demographic differences (apart from age and gender) was not taken into account in this study.

Another limitation in this study was the unequal sizes of the groups, as well as the small size of one of the groups. Thus there was some risk of violation of the assumptions of normality and homogeneity of variance. The statistical technique of bootstrapping was
therefore applied in order to obtain reliable results (Tabachnick & Fidell, 2007). On the other hand, a strength of this study was the group categories that allowed for group differences to be measured among three clinical groups including two comorbid groups and one social anxiety only group, as well as a healthy control group.

As with Study 1, two other limitations of Study 2 were its cross-sectional design, and the use of self-reports which could have contributed to recall bias. The cross-sectional nature of this study prevented any causal inferences being drawn. However, as with Study 1, the inclusion of the multiple regression analysis on social anxiety was a strength of this study as it not only confirmed the importance of both reward sensitivity and punishment sensitivity with respect to social anxiety in both clinical and non-clinical samples, it also revealed the importance of cannabis rather than alcohol in its influence on social anxiety disorder (in the clinical sample).

Finally a major strength of this study was the use of identical measures (with the exception of depression) by both a clinical sample (this study) and a non-clinical sample (Study 1). This allowed for the uniform measurement of social anxiety, alcohol and substance use, drive sensitivity, alcohol expectancies, and coping strategies, so that findings from both non-clinical and clinical samples could be compared to further our understanding about the nature of co-occurring social anxiety and substance use disorder. Future research could benefit from replication with larger sample sizes as well as healthy controls being matched for demographic factors, especially gender and age.

**Implications**

The findings of this study highlight important differences between individuals presenting with social anxiety disorder and those with co-occurring social anxiety and substance use disorders. In particular, socially anxious individuals are highly sensitive to punishment and they are the least sensitive to reward. The bigger comorbid group was also more sensitive to punishment compared to the healthy controls. Thus, there does appear to be a group of socially anxious individuals with co-occurring problematic substance use, who are highly sensitive to both reward and punishment. The tension between these two states could undermine the efforts of such affected individuals to stop the substance use.

There were also significant differences among the groups with respect to alcohol expectancies, highlighting the importance of these cognitions, both positive and negative, in
particular for individuals with comorbid social anxiety and alcohol/substance use disorders who use alcohol at a high risk level. One might predict logically that the high alcohol group would have low negative expectancies. However, it could also be predicted that there would be tension as a result of having both high positive and high negative alcohol expectancies, and this tension produces a vulnerability which motivates continued drinking.

Although the findings indicated no group differences with respect to both adaptive and maladaptive coping strategies, more research needs to be done in this area given past significant findings with respect to the associations of these factors to social anxiety and problematic substance use. As previously mentioned, not needing to control for age when comparing clinical groups with a control group could inform findings with respect to adaptive and maladaptive coping strategies. The findings of punishment and reward sensitivity as unique predictors of social anxiety reinforced their importance. In addition, cannabis but not alcohol was found to uniquely predict social anxiety in this clinical sample, suggesting its role as a co-occurring substance use disorder with social anxiety may be more important than detected so far by research in this area. The crucial roles of drive sensitivity, alcohol expectancies and cannabis use need to be taken into account in treatment interventions for sufferers of comorbid social anxiety and substance use disorders.

**Conclusion**

Study 2 explored differences between three clinical groups (social anxiety group, comorbid group with high alcohol use, all other comorbids in third group), and one healthy control group (no social anxiety and low risk alcohol/substance use), on three variables representing a biological factor (sensitivity to reward and punishment), a cognitive factor (positive and negative alcohol outcome expectancies) and a behavioural factor (adaptive and maladaptive coping strategies). The purpose behind this investigation was to inform and improve the efficacy of current treatments for individuals diagnosed with social anxiety disorder as well as a substance use disorder. In particular, the importance of punishment and reward sensitivity as well as positive and negative alcohol expectancies were borne out by the findings.

The same measures were used as in Study 1 so that comparisons could be made and conclusions drawn in order to further our understanding of and to better educate, prevent,
and treat individuals with co-occurring social anxiety and substance use disorders. Those comparisons and conclusions are discussed in the following chapter of this thesis.
Chapter 7: General Discussion and Conclusion

Summary

The first overall aim of this research was to increase understanding of the relationship between SAD and SUD. To the author’s knowledge this is the first study to do so by simultaneously investigating the roles of three factors: personality (drive sensitivity), alcohol expectancies and coping strategies, in both a clinical and a non-clinical sample. Both drive sensitivity and alcohol expectancies appear to play important roles in co-occurring social anxiety and substance use (SAD-SUD) disorders. The second overall aim of this research was to inform treatment for individuals with the SAD-SUD comorbidity, and to improve both prevention and education strategies in relation to this comorbidity. The findings from both studies can be used to fine-tune prevention and education strategies, and contribute to providing a more targeted treatment intervention for the SAD-SUD comorbidity.

Relationship between SAD and SUD

Although no relationship between social anxiety and risky alcohol or cannabis use was detected in the non-clinical sample, risky cannabis use was found to be important with respect to social anxiety in the clinical sample. This result extends recent findings in this research area and confirms the emerging importance of cannabis use and its relationship with social anxiety. As socially anxious individuals may be unlikely to present for treatment and the disorder may be unapparent to primary care providers or other health practitioners, our results suggests that signs of problematic cannabis use (or polysubstance use where one of the substances used is cannabis) may be an early warning sign of co-occurring SAD and SUD.

Sensitivity to Reward and Punishment

The findings from both studies demonstrate the important role played by both reward sensitivity and punishment sensitivity to social anxiety, substance use, and the SAD-SUD comorbidity. Results from both studies demonstrate the significant roles of these variables in predicting social anxiety. Socially anxious individuals have very low levels of reward sensitivity and very high levels of punishment sensitivity, compared to socially anxious individuals with a co-occurring substance use disorder. This suggests that
a subset of socially anxious individuals who have high levels of both punishment and reward sensitivity, are at risk of developing a substance use disorder. These results are consistent with those of Nicholls et al. (2014) who via latent class analysis reached the same conclusion. The present findings also suggest that a subset of individuals with a substance use disorder and high levels of both sensitivity drives, may also be at risk of developing social anxiety disorder. High levels of both reward sensitivity and punishment sensitivity could create tension leading to further substance use in this subtype of socially anxious individuals, resulting in a perpetual cycle of substance abuse.

Positive and Negative Alcohol Expectancies
Both studies reveal the importance of alcohol expectancies to social anxiety, substance use and the SAD-SUD comorbidity. Study 1 highlighted the important role of positive alcohol expectancies in the relationship with social anxiety and with both risky alcohol and risky cannabis use in non-clinical samples. The results indirectly confirm previous findings as to the similarities between positive alcohol and other positive substance expectancies (Aarons et al., 2001). Thus the importance of positive alcohol expectancies to risky alcohol and cannabis use can be extended to the importance of positive cannabis and other types of substance expectancies in relation to risky substance use.

The results in Study 2 highlighted the importance of both positive and negative alcohol expectancies. Positive expectancies differentiated the comorbid group with high risk alcohol use from the groups of socially anxious individuals and healthy controls, but did not differentiate the two comorbid groups. Negative alcohol expectancies were highest in the comorbid group with high risk alcohol use, and differentiated these people from the remainder of the comorbid group. This result suggests that people with more experiences of heavy or problematic alcohol use have more negative outcome expectancies. Overall these results suggest that in the early stages of alcohol or other substance use, positive expectancies are most influential, and with continued substance use (particularly alcohol use) that becomes heavy or problematic, negative alcohol expectancies also become influential. Thus socially anxious individuals with problematic and heavy alcohol or substance use are likely to have high levels of both positive and negative alcohol expectancies. This could create tension leading to further problematic substance use and thus perpetuating the comorbidity cycle.
Adaptive and Maladaptive Coping Strategies.

Despite the findings with the non-clinical sample of a positive association between social anxiety and maladaptive coping strategies and a negative relationship between social anxiety and adaptive coping strategies, these results were not borne out with the clinical sample. Past research directly examining the role of coping strategies in the relationship between social anxiety and substance use is extremely limited. Studies as to treatment outcomes for substance disordered individuals, indicate that irrespective of treatment intervention applied, participants increase their use of adaptive coping strategies and the volume and frequency of use of such strategies reduces the risk of relapse. Recent studies as to treatment outcomes for socially anxious individuals have found that optimal effects of adaptive emotion-focused coping strategies may arise when equal levels of adaptive and maladaptive strategies are being used. Our findings combined with other relevant and recent findings in this area suggest that all people use adaptive and maladaptive coping strategies, and by increasing their awareness of them, the frequency of use of adaptive coping strategies may be increased. Another important issue arising out of existing research in this area is how to ensure or increase the likelihood of continuing the use of adaptive coping strategies post treatment.

Fear of Negative Evaluation

The importance of fear of negative evaluation which has been recognized as a core feature of social anxiety disorder (SAD) in the latest edition of the DSM, DSM-5 (American Psychiatric Association, 2013), was highlighted by the non-clinical sample in the significant relationship of this cognition with all six shared vulnerability factors (sensitivity to reward and punishment, positive and negative alcohol expectancies and adaptive and maladaptive coping strategies). In addition, fear of negative evaluation specifically, rather than social anxiety generally, predicted risky alcohol use in the non-clinical sample. In both education and prevention strategies, the appearance of this trait should be emphasised as it could potentially signify the early stages of the development of social anxiety disorder. In treatment interventions, the significance of this characteristic of social anxiety needs to be clearly denounced and individualized for the patient in terms of their own thoughts and behaviour that arise as a result of or in response to fear of negative evaluation.
Clinical Implications and Recommendations for Treatment

The findings from the two studies highlight the importance of the six individual difference factors from which a targeted treatment intervention program for the SAD-SUD comorbidity is proposed. The development of an integrated treatment informed by a biopsychosocial model of the SAD-SUD comorbidity is recommended. The proposed integrated treatment could be described as an “enhanced CBT treatment for the SAD-SUD comorbidity” using a strengths-based approach and specifically designed to incorporate these three important shared vulnerabilities (drive sensitivity, alcohol expectancies and coping strategies) in the treatment of socially anxious individuals with a comorbid substance use disorder.

In addition to components of CBT used to treat both disorders (e.g., psychoeducation, cognitive therapy, behavioural therapy, relaxation strategies), the “enhanced CBT treatment for the SAD-SUD comorbidity” would include the learning of skills relevant to CBT treatments of each disorder. For example skills relevant to social anxiety disorder could include social skills training, and challenging judgmental biases characteristic of SAD, including fear of negative evaluation. The importance of the core feature of fear of negative evaluation should be highlighted in all treatment components (psychoeducation, cognitive therapy, behaviour therapy). For substance use disorder, components of CBT relevant to the affected individual’s circumstances could be incorporated, for example, motivational interviewing, harm minimization and/or relapse prevention. The enhanced-CBT treatment intervention would need to be specifically tailored to the affected individual’s needs and presentation after a detailed assessment has been conducted to obtain a history of symptoms, severity, onset or remission periods of both disorders (SAD and SUD) and any comorbid conditions. The enhanced-CBT intervention would also need to be flexible in terms of the timing of the application of its treatment components. A more detailed description of the psychoeducation, cognitive therapy, and behavioural therapy components as well as their application to drive sensitivity, alcohol expectancies and coping strategies in the proposed “enhanced CBT treatment for the SAD-SUD comorbidity” follows.

Psychoeducation. As with current treatment models of CBT, it is proposed that the enhanced CBT treatment commence with psychoeducation. This would involve explaining
the interactive nature of the SAD-SUD comorbidity relying on the shared vulnerability model within a biopsychosocial framework. The biopsychosocial framework supports the interplay of biological, psychological and social factors which perpetuate the SAD-SUD comorbidity. The shared vulnerability model allows for symptoms of either disorder to have presented before the other, or for symptoms of both disorders to have commenced around the same time. This model also allows for different courses of the comorbidity including the continuing co-occurrence of both social anxiety and substance use disorders, remission of symptoms of one of the disorders, and/or varying degrees of severity of symptoms of one or both disorders. The individual difference variables of personality (drive sensitivity), alcohol (and/or other substance) expectancies, and coping strategies could be introduced during the psychoeducation stage. In particular the coexistence of both high levels of reward and punishment sensitivity, and positive and negative expectancies could be explained and emphasised in terms of the tension these conflicts may produce, thus perpetuating the comorbidity cycle.

With respect to reward and punishment sensitivity, a strengths based approach could include harnessing the high levels of reward sensitivity and redirecting them toward other activities (instead of substance use) to enhance positive affect, given that reduced positive affect is strongly associated with social anxiety (Alden & Trew, 2013). Alden and Trew (2013) conducted a randomized control trial in which socially anxious undergraduate students were randomly assigned to one of three types of activities: an activity designed to increase positive affect (by engaging in kind acts), an activity designed to reduce negative affect (behavioural experiments) or a neutral “control” activity (activity monitoring). Only the group engaging in acts of kindness significantly increased their positive affect. Another or an alternate strategy that could be utilised to redirect the high levels of reward sensitivity could be to assist the individual to redirect their behaviour to align with important goals and values to them (a component of Acceptance and Commitment Therapy, the more recently developed form of cognitive-behaviour therapy). Thus behavioural therapy could also include some of the work involved in dealing with high levels of both reward and punishment sensitivity.

With respect to positive and negative alcohol (or other applicable substance) expectancies, a strengths-based approach could involve the affected individual identifying
both positive (e.g., social assertiveness, tension reduction) and negative (e.g., cognitive impairment, negative feelings) expectancies to increase their awareness of their thoughts about the consequences of using alcohol and/or other substances. To some extent, this is conducted via motivational interviewing, but the increased awareness would also need to be integrated with the effect of these beliefs on social anxiety. The tension produced by having conflicting cognitions in this area could be dealt with using any one or more of a number of cognitive or behavioural strategies, for example, relaxation strategies, cognitive restructuring, acceptance, mindfulness, that is, whichever of such strategies work best for the affected individual.

With respect to adaptive and maladaptive coping strategies, the affected individual would be asked to identify both adaptive (e.g., obtaining emotional support from others) and maladaptive (e.g., self-blame) coping strategies that they use to cope with stressful situations. The purpose of this activity would be to increase the affected individual’s awareness especially of adaptive coping strategies they have used in addition to maladaptive coping strategies. Their use of substances to cope with stress or their social anxiety could be acknowledged as one of many coping strategies they use, adaptive and maladaptive. Thus, dealing with coping strategies as part of the enhanced CBT treatment would entail using a strength-based approach to contain maladaptive coping strategies and focus on how to access existing adaptive coping strategies in order to overtake or supersede the maladaptive coping strategies.

In summary, the affected individual would be asked to recall their own thoughts and behaviours relating to each of these important factors (punishment and reward sensitivity, positive and negative expectancies, and adaptive and maladaptive coping strategies), as part of their overall treatment for the SAD-SUD comorbidity.

**Cognitive Therapy.** The cognitive therapy component of the enhanced CBT intervention would involve increasing the affected individual’s awareness of relevant cognitions pertaining to both social anxiety and substance use symptoms. The importance of fear of negative evaluation as a key feature of social anxiety could be introduced as part of this component, and the individual could be assisted in increasing their awareness of how they individually operationalize this attribute. The individual would also be encouraged to increase their awareness of relevant cognitions pertaining to reward and
punishment sensitivity, positive and negative alcohol (or other relevant substance) expectancies and adaptive and maladaptive coping strategies, all of which relate to the perpetuation of the SAD-SUD comorbidity cycle. The affected individual would be trained in a number of techniques designed to challenge or diffuse the intensity of relevant cognitions (to both social anxiety and substance use). Such techniques could include “cognitive restructuring” (challenging and changing unproductive thoughts) and/or “diffusion” (accepting and being ‘mindful’ of the cognitions to lessen their intensity).

**Behaviour Therapy.** The actual substance use behaviour could be targeted as part of this component of the enhanced CBT treatment intervention for the SAD-SUD comorbidity. This would depend upon the severity of the substance use disorder, discussion between the affected individual and therapist at a stage when the affected individual has become more aware of relevant cognitions to both disorders, and which cognitive therapy techniques are suitable for those cognitions. Thus at this point the affected individual could choose between various options with regard to ceasing, reducing, managing or controlling their problematic substance use. This component of the targeted intervention would also include developing a behavioural hierarchy to enable the affected individual to expose themselves to feared social situations (from less anxiety provoking to more anxiety provoking). Finally, as previously mentioned, behavioural therapy may be applied to positively reinforce pre-existing cognitions and behaviours including those pertaining to reward sensitivity and adaptive coping strategies.

**Directions for Future Research**

More family and twin studies investigating the shared vulnerability model for co-occurring social anxiety and substance use are recommended to replicate findings in this study with respect to the importance of reward and punishment sensitivity, alcohol expectancies and coping strategies, and their shared genetic and/or environmental risk factors. Longitudinal or prospective design research in the area of the SAD-SUD comorbidity would also help further clarify the nature of this comorbidity, especially post treatment effects, for example, do treated individuals continue to use adaptive coping strategies, for how long, and what are the factors that sustain use of adaptive coping strategies post treatment. Research investigating differences between risky alcohol use and risky cannabis use via specific substance-related measures, such as alcohol and cannabis
expectancies, could further increase understanding of the SAD-AUD and the SAD-CUD comorbidity. Further research looking at important factors (e.g., peer group pressure) which influence cannabis use in its relationship with social anxiety is recommended to further understand the nature of the SAD-CUD comorbidity. Finally, research that directly evaluates the proposed enhanced CBT treatment intervention that evolved from the findings of this thesis is suggested.

**Conclusion/Contributions of Current Research Project**

The purpose of this research project was to further our understanding of the nature of the SAD-SUD comorbidity in order to improve education and prevention strategies as well as to inform treatment interventions. This was conducted by way of investigating the contributions of three important vulnerability factors: drive sensitivity, alcohol expectancies and coping strategies to the relationship between social anxiety and substance use. Unlike most research in this area, our research was designed to achieve these goals by relying on a biopsychosocial framework to reflect the complexity of the SAD-SUD comorbidity, with the three factors chosen based on their roles within this framework and their demonstrated or emerging importance according to the existing literature. Furthermore, in contrast to the majority of the literature in this research area, the theoretical basis for our research project was the shared vulnerabilities model rather than the self-medication model. This excluded the necessity of demonstrating causality. Another departure from current research designs was the simultaneous investigation of alcohol and cannabis use, with findings indicating that in clinical samples, cannabis plays a very important role in relation to social anxiety. Finally by conducting this research using the same measures with both a clinical and non-clinical population, the importance of personality (drive sensitivity), alcohol expectancies and coping strategies was demonstrated in the relationship between social anxiety and substance use across the spectrum of severity of both disorders. In particular, sensitivity to both reward and punishment, as well as positive and negative alcohol expectancies are crucial to understanding and hopefully improving treatment interventions and outcomes for socially anxious individuals with a co-occurring substance use disorder.


setting: Using the Liebowitz Social Anxiety Scale. *Journal of Anxiety Disorders, 16*(6), 661-673.


APPENDIX A: Ethics Approval for Both Studies

RES
Ethics <resethics@swin.edu.au>
>

to: Michael Kyrios
    <mkyrios@swin.edu.au>

cc: FLSS Research
    <lssresearch@swin.edu.au>

date: Mon, Jan 7, 2013 at 2:13 PM

subject: SUHREC Project 2010/134
        Ethics Clearance for Project
        Extension (2)

mailed-by: swin.edu.au

To: Michael Kyrios, FLSS/ Ms Annette Raber

[BC: Ms Annette Raber]

Dear Prof Kyrios ans Ms Raber,

SUHREC Project 2010/134 Exploring the role of personality, expectancies and coping strategies in co-occurring social anxiety disorder and substance use disorder

Prof M Kyrios, FLSS/Ms Annette Raber et al

Approved Duration: 01/10/2010 To 31/12/2011, Extended To 31/12/2012, 06/07/2013


I refer to your e-mail of today’s date in which you request an extension of duration for the project to 6 July 2013.

There being no change to the approved protocol, I am authorised to issue the extension of ethics clearance in line with standard on-going ethics clearance conditions previously communicated and reprinted below.
Please contact the Research Ethics Office if you have any queries about on-going ethics clearance, citing the SUHREC project number. Copies of clearance emails should be retained as part of project record-keeping.

Best wishes for the project.

Yours sincerely

Kaye Goldenberg

for
Keith Wilkins
Secretary, SUHREC

Kaye Goldenberg
Administrative Officer (Research Ethics)
Swinburne Research (H68)
Swinburne University of Technology
P O Box 218
HAWTHORN VIC 3122
Tel +61 3 9214 8468

-----Original Message-----
From: Resethics [mailto:Resethics@groupwise.swin.edu.au]
Sent: Friday, 22 June 2012 5:03 PM
To: Michael Kyrios; annetteraber@gmail.com
Cc: Robyn Watson
Subject: SUHREC Project 2010/134 Ethics Clearance for Modification (4)

To: Prof Michael Kyrios/Ms Annette Raber, FLSS

Dear Mike and Annette,
I refer to your email of 20 June 2012 requesting clearance for a modification to the protocol to extend recruitment arrangements. The request, as emailed with attachment, was put to a SUHREC delegate for consideration.

I am pleased to advise that, as submitted to date, the further modified project/protocol may continue in line with standard ethics clearance conditions previously communicated and reprinted below.

Please contact the Research Ethics Office if you have any queries about ongoing ethics clearance, citing the SUHREC project number. Copies of clearance emails should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith

*******************************************
Keith Wilkins
Secretary, SUHREC & Research Ethics Officer Swinburne Research (H68) Swinburne University of Technology P O Box 218 HAWTHORN VIC 3122 Tel +61 3 9214 5218 Fax +61 3 9214 5267

>>> Resethics 11/05/2012 12:05 PM >>>

To: Prof Michael Kyrios FLSS-BSI; Ms Annette Raber (BC)
Dear Mike and Annette,

SUHREC Project 2010/134 Exploring the role of personality, expectancies and coping strategies in co-occurring social anxiety disorder and substance use disorder

Prof M Kyrios, FLSS; Ms Annette Raber et al

Approved Duration Extended To 31/12/2012 [Modified January 2011; July 2011; May 2012]

I refer to your email of 3 May 2012 requesting clearance for a modification to the protocol to extend the project to a non-clinical population. The request, as emailed with attachments, was put to a SUHREC delegate for consideration. I also acknowledge receipt of a progress report on the project received today.

I am pleased to advise that, as submitted to date, the further modified project/protocol may continue in line with standard ethics clearance conditions previously communicated and reprinted below.

Please contact the Research Ethics Office if you have an queries about on-going ethics clearance, citing the SUHREC project number. Copies of clearance emails should be retained as part of project record-keeping.

Best wishes for the project.

Yours sincerely

Keith

*********************************************************************************

Keith Wilkins

Secretary, SUHREC & Research Ethics Officer
Dear Mike and Annette,

SUHREC Project 2010/134 Exploring the role of personality, expectancies and coping strategies in co-occurring social anxiety disorder and substance use disorder

Prof M Kyrios, FLSS; Ms Annette Raber et al

Approved Duration 1/10/2010 To 31/12/2011 [Modified January 2011; July 2011]

I refer to your emails of 7 and 8 July 2011 requesting clearance for a modification to the protocol to extend the project to a non-clinical population. The latter email was in response to queries concerning the former, including regarding use of a non-Swinburne email address. The request was put to a SUHREC delegate for consideration.

I am pleased to advise that, as submitted to date, the further modified project/protocol may continue in line with standard ethics clearance conditions previously communicated and reprinted below.

Please contact the Research Ethics Office if you have any queries about ongoing ethics clearance, citing the SUHREC project number. Copies of clearance emails should be retained as part of project record-keeping.

Best wishes for the project.

Yours sincerely
Dear Prof Kyrios and Ms Raber,

Re: SUHREC Project 2010/134 Exploring the role of personality, expectancies and coping strategies in co-occurring social anxiety disorder and substance use disorder

Prof M Kyrios Ms Annette Raber FLSS-BSI

Approved Duration 1/10/2010 To 31/12/2011 [Modified January 2011]

I refer to your email of 11 January 2011 in which you requested a modification to the protocol by allowing for either personal or phone interviews for the clinical interview stage of the project. Your request was put to a delegate(s) of SUHREC.

I am pleased to advise that, as submitted to date, the further modified project/protocol may continue in line with standard ethics clearance conditions previously communicated and attached to this email.

Best wishes for the project.
Yours sincerely

Ann Gaeth
for Keith Wilkins
Secretary, SUHREC

>>> Ann Gaeth 22/10/2010 10:59 AM >>>

To: Prof M Kyrios FLSS-BSI; Ms Annette Raber (BC)
CC: Ms Hayley Mowat, Research Administration Assistant FLSS

Dear Prof Kyrios and Ms Raber,

Re: SUHREC Project 2010/134 Exploring the role of personality, expectancies and coping strategies in co-occurring social anxiety disorder and substance use disorder

Prof M Kyrios Ms Annette Raber FLSS-BSI

Approved Duration 1/10/2010 To 31/12/2011

I refer to the ethical review of the above project protocol undertaken by Swinburne’s Human Research Ethics Committee (SUHREC). Your response to the review, as e-mailed on 13/20 October 2010 with attachment (Deakin HREC application and approval), were put to and approved by a SUHREC delegate.

I am pleased to advise that, as submitted to date, the project has approval to proceed in line with standard ongoing ethics clearance conditions here outlined.

- All human research activity undertaken under Swinburne auspices must conform to Swinburne and external regulatory standards, including the National Statement on Ethical Conduct in Human Research and with respect to secure data use, retention and disposal.
- The named Swinburne Chief Investigator/Supervisor remains responsible for any personnel appointed to or associated with the project being made aware of ethics clearance conditions, including research and consent procedures or instruments approved. Any change in chief investigator/supervisor requires timely notification and SUHREC endorsement.

- The above project has been approved as submitted for ethical review by or on behalf of SUHREC. Amendments to approved procedures or instruments ordinarily require prior ethical appraisal/clearance. SUHREC must be notified immediately or as soon as possible thereafter of (a) any serious or unexpected adverse effects on participants and any redress measures; (b) proposed changes in protocols; and (c) unforeseen events which might affect continued ethical acceptability of the project.

- At a minimum, an annual report on the progress of the project is required as well as at the conclusion (or abandonment) of the project.

- A duly authorised external or internal audit of the project may be undertaken at any time.

Please contact me if you have any queries about the ethical review process, citing the SUHREC project number. Copies of clearance emails should be retained as part of project record-keeping.

Best wishes for the project.

Yours sincerely

Ann Gaeth
for Keith Wilkins
Secretary, SUHREC
APPENDIX B: Questionnaire

DEMOGRAPHICS

The following section asks you some general questions about yourself.

1. Age in years: □□

2. Gender: □ Male □ Female

3. Where were you born?
   □ Australia □ New Zealand/Oceania □ South America/Caribbean
   □ Africa □ Middle East □ North America
   □ Asia □ Europe □ Other (please specify) ___________________

4. Which ethnic/cultural group do you most identify with? (e.g. Australian) ________________

5. Are you Aboriginal/Torres Strait Islander? □ Yes □ No

6. What is your current relationship status?
   □ Never married □ Married/Living with Partner □ Separated/Divorced
   □ Steady relationship (not living together) □ Widowed

7. What is the highest level of education you have completed?
   □ Primary School □ TAFE Diploma/Certificate/Trade Qualification
   □ Some High School □ University qualification
   □ Completed VCE/HSC

8. What is your usual occupation? _______________________________________

9. What is your current employment status?
   □ Unemployed □ Employed – part-time/casual □ Employed – full-time
   □ Student □ Retired □ Other (please specify) __________________________

10. Do you receive any Centrelink benefits? □ Yes □ No

11. Who are your main social supports? (Tick all that apply)
    □ Parents □ Other family □ Organisational support
    □ Partner □ Friends □ Other (please specify) _________________________
12. Where do you usually live?

- Rented house
- Public housing
- Parents’ or other family members’ house
- House you own
- Homeless/No fixed address
- Other (please specify) ____________

**LSAS-SR**

For each situation listed below please rate how much you are afraid of, or would be anxious in, that situation and how much you avoid that situation, by using the two rating scales below. Make the fear or anxiety rating in the first column and the avoidance rating in the second column. Please base your ratings on the way that situations have affected you in the last week.

<table>
<thead>
<tr>
<th>Fear or Anxiety Rating</th>
<th>Avoidance Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 = No fear or anxiety in this situation</td>
<td>0 = Never avoid this situation</td>
</tr>
<tr>
<td>1 = Mild fear or anxiety in this situation</td>
<td>1 = Occasionally avoid this situation (up to 33% of the time)</td>
</tr>
<tr>
<td>2 = Moderate fear or anxiety in this situation</td>
<td>2 = Often avoid this situation (33 to 67% of the time)</td>
</tr>
<tr>
<td>3 = Severe fear or anxiety in this situation</td>
<td>3 = Usually avoid this situation (67 to 100% of the time)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Fear/Anxiety</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoidance</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

1. Telephoning in public.  
2. Participating in small groups.  
3. Eating in public places.  
4. Drinking with others in public places.  
5. Talking to people in authority.  
6. Acting, preforming or giving a talk in front of an audience.  
7. Going to a party.  
8. Working while being observed.  
9. Writing while being observed.  
10. Calling someone you don’t know very well.  
11. Talking with people you don’t know very well.  
12. Meeting strangers.  
14. Entering a room when others are already seated.  
15. Being the centre of attention.
SRSPQ

Please answer each question by ticking the “Yes” or the “No” box following the questions. Note: The following questions refer to your life generally, NOT when using alcohol or drugs.

<p>| | | | | | | | | | |</p>
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<thead>
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</thead>
<tbody>
<tr>
<td>1.</td>
<td>Does the prospect of obtaining money motivate you strongly to do some things?</td>
<td>□ Yes □ No</td>
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<td>2.</td>
<td>Do you prefer not to ask for something when you are not sure you will obtain it?</td>
<td>□ Yes □ No</td>
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<td>3.</td>
<td>Are you often afraid of new or unexpected situations?</td>
<td>□ Yes □ No</td>
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<td>4.</td>
<td>Is it difficult for you to telephone someone you do not know?</td>
<td>□ Yes □ No</td>
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<td>5.</td>
<td>Do you often do things to be praised?</td>
<td>□ Yes □ No</td>
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<td>6.</td>
<td>Do you like being the centre of attention at a party of a social meeting?</td>
<td>□ Yes □ No</td>
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<td>7.</td>
<td>In tasks that you are not prepared for, do you attach great importance to the possibility of failure?</td>
<td>□ Yes □ No</td>
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<td>8.</td>
<td>Do you spend a lot of time on obtaining a good image?</td>
<td>□ Yes □ No</td>
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<td>9.</td>
<td>Are you easily discouraged in difficult situations?</td>
<td>□ Yes □ No</td>
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<tr>
<td>10.</td>
<td>Are you a shy person?</td>
<td>□ Yes □ No</td>
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<td>11.</td>
<td>When you are in a group, do you try to make your opinions the most intelligent or funniest?</td>
<td>□ Yes □ No</td>
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<td></td>
<td>Question</td>
<td>Yes</td>
<td>No</td>
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<td>12.</td>
<td>Whenever possible, do you avoid demonstrating your skills for fear of being embarrassed?</td>
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<td>13.</td>
<td>Do you often take the opportunity to pick up people you find attractive?</td>
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<td>14.</td>
<td>When you are with a group, do you have difficulties selecting a good topic to talk about?</td>
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<td>15.</td>
<td>As a child, did you do a lot of things to get people’s approval?</td>
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<td>16.</td>
<td>Does the possibility of social advancement move you to action, even if this involves not playing fair?</td>
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<td>17.</td>
<td>Do you think a lot before complaining in a restaurant if your meal is not well prepared?</td>
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<td>18.</td>
<td>Do you generally give preference to those activities that imply an immediate gain?</td>
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<td>19.</td>
<td>Do you often have trouble resisting the temptation of doing forbidden things?</td>
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<td>20.</td>
<td>Whenever you can, do you avoid going to unknown places?</td>
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<td>21.</td>
<td>Do you like to compete and do everything you can to win?</td>
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<td>22.</td>
<td>Are you often worried by things that you said or did?</td>
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<td>23.</td>
<td>Would it be difficult for you to ask your boss for a raise (salary increase)?</td>
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<td>24.</td>
<td>Do you generally try to avoid speaking in public?</td>
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<td>25.</td>
<td>Do you, on a regular basis, think that you could do more things if it was not for your insecurity or fear?</td>
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<td>26.</td>
<td>Do you sometimes do things for quick gains?</td>
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<td>27.</td>
<td>Comparing yourself to people you know, are you afraid of many things?</td>
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<td>28.</td>
<td>Does your attention easily stray from your work in the presence of an attractive stranger?</td>
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<td>29.</td>
<td>Do you often find yourself worrying about things to the extent that performance in intellectual abilities is impaired?</td>
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<td>30.</td>
<td>Are you interested in money to the point of being able to do risky jobs?</td>
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<tr>
<td>31.</td>
<td>Do you often refrain from doing something you like in order not to be rejected or disapproved by others?</td>
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<td>32.</td>
<td>Do you like to be competitive in all of your activities?</td>
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<tr>
<td>33.</td>
<td>Would you like to be a socially powerful person?</td>
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</tbody>
</table>
34. Do you often refrain from doing something because of your fear of being embarrassed? □ Yes □ No

35. Do you like displaying your physical abilities even though this may involve danger? □ Yes □ No

AE

Here is a list of some effects or consequences that some people experience after drinking alcohol. How likely is it that these things happen to you when you drink alcohol? Please circle the number that best describes how drinking alcohol would affect you.

If you do not drink at all, you can still fill this out: just answer it according to what you think would happen to you if you did drink. Please circle the number which best fits for you.


1
No chance

2
Very unlikely

3
Unlikely

4
Likely

5
Very likely

6
Certain to happen

When I drink alcohol ____________________________________?

1. I am more accepted socially.

2. I become aggressive.

3. I am less alert.

4. I feel ashamed of myself.

5. I enjoy the buzz.

6. I become clumsy or uncoordinated.

7. I feel good.

8. I get into fights.
<table>
<thead>
<tr>
<th></th>
<th>I can’t concentrate.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>10.</td>
<td>I have a good time.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>11.</td>
<td>I have problems driving.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>12.</td>
<td>I feel guilty.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>13.</td>
<td>I get a hangover.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>14.</td>
<td>I feel happy.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>15.</td>
<td>I get a headache.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
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<tr>
<td>16.</td>
<td>I am more sexually assertive.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
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<tr>
<td>17.</td>
<td>It is fun.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>18.</td>
<td>I get mean.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
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<tr>
<td>19.</td>
<td>I have problems with memory and concentration.</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>20.</td>
<td>I am more outgoing.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>21.</td>
<td>It takes away my negative moods and feelings.</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
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<tr>
<td>22.</td>
<td>I have more desire for sex.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
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<tr>
<td>23.</td>
<td>It is easier for me to socialise.</td>
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<td>2</td>
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<td>6</td>
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</table>
This questionnaire asks you to indicate what you generally do and feel when you experience stressful events. Obviously, different events bring out somewhat different responses, but think about what you usually do when you are under a lot of stress. Please circle the most appropriate response, using the choices listed just below. Please try to respond to each item separately in your mind from each other item. Choose your answers thoughtfully, and make your answers as true for you as you can. Please answer every item. There are no "right" or "wrong" answers, so choose the most accurate answer for you - not what you think "most people" would say or do. Indicate what you usually do when you experience a stressful event.

1 = I usually don't do this at all
2 = I usually do this a little bit
3 = I usually do this a medium amount
4 = I usually do this a lot

<table>
<thead>
<tr>
<th>Item</th>
<th>Response</th>
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<th>4</th>
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</thead>
<tbody>
<tr>
<td>1.</td>
<td>I turn to work or other activities to take my mind off things.</td>
<td>1</td>
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...
2. I concentrate my efforts on doing something about the situation I'm in. | 1 | 2 | 3 | 4
3. I say to myself "this isn't real". | 1 | 2 | 3 | 4
4. I use alcohol or other drugs to make myself feel better. | 1 | 2 | 3 | 4
5. I get emotional support from others. | 1 | 2 | 3 | 4
6. I give up trying to deal with it. | 1 | 2 | 3 | 4
7. I take action to try to make the situation better. | 1 | 2 | 3 | 4
8. I refuse to believe that it has happened. | 1 | 2 | 3 | 4
9. I say things to let my unpleasant feelings escape. | 1 | 2 | 3 | 4
10. I get help and advice from other people. | 1 | 2 | 3 | 4
11. I use alcohol or other drugs to help me get through it. | 1 | 2 | 3 | 4
12. I try to see it in a different light, to make it seem more positive. | 1 | 2 | 3 | 4
13. I criticize myself. | 1 | 2 | 3 | 4
14. I try to come up with a strategy about what to do. | 1 | 2 | 3 | 4
15. I get comfort and understanding from someone. | 1 | 2 | 3 | 4
16. I give up the attempt to cope. | 1 | 2 | 3 | 4
17. I look for something good in what is happening. | 1 | 2 | 3 | 4
18. I make jokes about it. | 1 | 2 | 3 | 4
19. I do something to think about it less, such as going to movies, watching TV, reading, daydreaming, sleeping, or shopping. | 1 | 2 | 3 | 4
20. I accept the reality of the fact that it has happened. | 1 | 2 | 3 | 4
21. I express my negative feelings. | 1 | 2 | 3 | 4
22. I try to find comfort in my religion or spiritual beliefs. | 1 | 2 | 3 | 4
23. I try to get advice or help from other people about what to do. | 1 | 2 | 3 | 4
### CES-D

Click the circle for each statement which best describes how often you felt or behaved this way - DURING THE PAST WEEK.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Rarely or None of the Time (Less than 1 Day)</th>
<th>Some or Little of the Time (1-2 Days)</th>
<th>Occasionally or a Moderate Amount of the Time (3-4 Days)</th>
<th>Most or All of the Time (5-7 Days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I was bothered by things that usually don't bother me</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td>I did not feel like eating; my appetite was poor</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
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<td>I felt that I could not shake off the blues even with help from my family or friends</td>
<td>☐</td>
<td>☐</td>
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<td>I felt that I was just as good as other people</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td>I had trouble keeping my mind on what I was doing</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
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<tr>
<td>I felt depressed</td>
<td>☐</td>
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<tr>
<td>Statement</td>
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<td>I felt that everything I did was an effort</td>
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<td>I felt hopeful about the future</td>
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<td>I thought my life had been a failure</td>
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<td>I felt fearful</td>
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<td>My sleep was restless</td>
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<td>I was happy</td>
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<td>I talked less than usual</td>
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<td>I felt lonely</td>
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<tr>
<td>People were unfriendly</td>
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<td>I enjoyed life</td>
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<td>I had crying spells</td>
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<td>I felt sad</td>
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<td>I felt that people disliked me</td>
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<td>I could not get &quot;going&quot;</td>
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**BFNE**

For each question, please circle a number to indicate the degree to which you feel the statement is characteristic or true of you.
<table>
<thead>
<tr>
<th></th>
<th>Description</th>
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<th>1</th>
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<th>4</th>
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<tbody>
<tr>
<td>1</td>
<td>I worry about what other people will think of me even when I know it doesn't make a difference.</td>
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<td>1</td>
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<td>4</td>
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<tr>
<td>2</td>
<td>It bothers me when people form an unfavourable impression of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>3</td>
<td>I am frequently afraid of other people noticing my shortcomings.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>I worry about what kind of impression I make on people.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>I am afraid that others will not approve of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>I am afraid that people will find fault with me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>I am concerned about other people's opinions of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>When I am talking to someone, I worry about what they may be thinking about me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>I am usually worried about what kind of impression I make.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>If I know someone is judging me, it tends to bother me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>Sometimes I think I am too concerned with what other people think of me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
12. I often worry that I will say or do wrong things.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
</table>

### ASSIST

<table>
<thead>
<tr>
<th>Q1</th>
<th>In your life which of the following substances have you ever used?</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
<th>YES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Circle Yes or No</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
<td>NO</td>
</tr>
</tbody>
</table>

Complete Q2 to Q8 for all substances answered “Yes”

<table>
<thead>
<tr>
<th>Q2</th>
<th>In the past 3 months, how often have you used ..........?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 – Never</td>
</tr>
<tr>
<td></td>
<td>2 – Once/ twice</td>
</tr>
<tr>
<td></td>
<td>3 – Monthly</td>
</tr>
<tr>
<td></td>
<td>4 – Weekly</td>
</tr>
<tr>
<td></td>
<td>6 – Daily/ Almost Daily</td>
</tr>
</tbody>
</table>

If “Never” go to Q6 for that substance

<table>
<thead>
<tr>
<th>Q3</th>
<th>During the past 3 months, how often have you had strong desires or urges to use........?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 – Never</td>
</tr>
<tr>
<td></td>
<td>3 – Once/ twice</td>
</tr>
<tr>
<td></td>
<td>4 – Monthly</td>
</tr>
<tr>
<td></td>
<td>5 – Weekly</td>
</tr>
<tr>
<td></td>
<td>6 – Daily/ Almost Daily</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Q4</th>
<th>During the past 3 months, how often has your use of .......... led to health, social, legal or financial problems?</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 – Never</td>
</tr>
<tr>
<td></td>
<td>4 – Once/ twice</td>
</tr>
</tbody>
</table>
Q5  During the past 3 months how often have you failed to do what is normally expected of you because of your use of...... ?
0 – Never
5 – Once/ twice
6 – Monthly
7 – Weekly
8 – Daily/ Almost Daily

Q6  Has a friend, relative or anyone else ever expressed concern at your use of......?
0 - Never
6 - Yes in past 3 months
3 - Yes not in past 3 months

Q7  Have you ever tried and failed to control, cut down or stop using......?
0 - Never
6 - Yes in past 3 months
3 - Yes not in past 3 months

Q8  Have you ever used any drug by injection (non medical)?
0 - Never
2 - Yes in past 3 months
1 - Yes not in past 3 months

Scores

Study 2 Depression Measure BDI-II (Beck Depression Inventory) – not printed due to copyright restrictions
APPENDIX C: Study 1 Explanatory Statement

Exploring the role of personality, beliefs about alcohol use and coping strategies in people who feel anxious in social situations

My name is Annette Raber and I am conducting this research project as part of a Doctor of Clinical Psychology, under the supervision of Professor Michael Kyrios at Swinburne University.

Many people feel anxious in social situations. The aim of this study is to explore how personality, thoughts and beliefs about alcohol, and coping strategies affect the relationship between social anxiety and substance use. It is hoped that the findings of this research will lead to an improvement in the lives of people who feel awkward in social situations, or people who find using alcohol or other drugs helps them relax in unfamiliar social situations.

We are looking for people aged 18 years and over to participate in this research. Your involvement will require you to complete an online questionnaire which asks general questions, as well as questions about anxiety in social situations, substance use (if any), current mood, personality, beliefs about alcohol use, and coping strategies. Participation is estimated to take no more than 30 minutes.

Your responses to the questionnaire will only be accessed by the researchers involved in this project. All information relating to this project will be destroyed after the minimum period of 7 years after publication of the results. Findings from this study will be reported in a thesis, and may also be submitted for publication at a conference or in a peer-reviewed journal. The information you provide will only be used for the purposes described in this document.

Your participation in this study is voluntary and you are free to withdraw at any time. All information you provide is anonymous as no identifying information is being collected. Online submission of the survey, by clicking the “Finish” button once you have completed it, will imply your consent to participate in the study. If you do not wish to complete the questionnaire all at once, you can click the "Save" button located at the bottom of the page. This will enable you to save your responses so that you can return and complete it at a later time.

This project contains questions which may cause discomfort or distress to some participants. If you are concerned, please contact your general practitioner. Australian participants can also contact the Swinburne Psychology Clinic at Hawthorn (a low cost service), on (03) 9214 8653. Swinburne students can obtain free counselling at Swinburne Counselling Services on: (03) 9214 8025 (Hawthorn) or (03) 9215 7101 (Lilydale). Lifeline can be contacted on 131 114. International participants can contact the Lifeline International 24 hour telephone counselling
service, details regarding this service in your home country can be found by accessing their website: http://www.lifeline-international.org/looking_for_help.

If you have any enquiries about the study, please don’t hesitate to contact the researchers:

Professor Michael Kyrios  
Telephone: (03) 9214 4886  
Email: mkyrios@groupwise.swin.edu.au

Annette Raber  
Telephone: 0459 775 739  
Email: araber@swin.edu.au

Thank you for your time.
TO: Participant

Plain Language Statement

Date: 28 August 2010

Full Project Title: Improving the retention rate for residential treatment of substance abuse by sequential intervention for social anxiety

Principal Researchers: Associate Professor Petra Staiger, Professor Mike Kyrios, Dr Nicolas Kambouropoulos

Associate Researchers: Dr Stefan Gruenert, Ms Caroline Long

This Plain Language Statement and Consent Form is 8 pages long. Please make sure you have all the pages.

1. Your Consent
You are invited to take part in this research project.

This Plain Language Statement contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Plain Language Statement carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Plain Language Statement and Consent Form to keep as a record.
2. **Purpose and Background**

The purpose of this project is to examine whether addressing a person's social anxiety issues prior to entering residential drug and alcohol treatment means that it is more likely that the person will enter and remain in treatment.

Not completing residential drug and alcohol treatment is a common problem and it is possible that significant anxiety issues may contribute to this problem. We believe that addressing social anxiety symptoms before and/or at the early stages of residential treatment may help people stay in treatment, and therefore help them get the most from the residential treatment.

In order to see if helping people manage their social anxiety prior to entry into Odyssey House will help them stay in the program, we will be offering a widely used psychological treatment to randomly selected individuals and comparing their progress with people who have not had the social anxiety treatment.

A total of 90 people will participate in this project. As we want to see if including social anxiety treatment before entering the residential treatment is helpful, we need to have half the people participate in an education program on managing stress in addition to the normal Odyssey House waitlist procedure and half the people receive the social anxiety treatment in addition to the normal Odyssey House waitlist procedures. You will not be able to choose which group you are in.

Forty-five individuals will receive the two individual and two small group sessions of social anxiety treatment while on the waitlist to enter the usual Odyssey House residential treatment program and a combination of therapeutic letters and telephone booster sessions in the first two weeks of entering Odyssey House. The other participants will follow receive the stress management program and the usual waitlist procedure (i.e. you will receive standard treatment preparing you for entry into Odyssey House – this may involve you attending one or two group preparation sessions). All participants will receive standard treatment once they have entered Odyssey House.

You are invited to participate in this research project because you are a client of Odyssey House Victoria and have reported experiencing issues with social anxiety.

3. **Funding**

This research is totally funded by the Australian Research Council Linkage Project Grant.

4. **Procedures**

The first component of this project will involve completing an initial assessment. At this time you will be asked to complete a written questionnaire and take part in a clinical interview. This will take approximately 90 minutes to complete. These questions will address social anxiety issues, general anxiety and depression, drug and alcohol use and overall well-being. All participants will be asked to complete further assessment sessions approximately 6 weeks later, then 3 and 12 months after TC entry.

At the end of this first assessment appointment you will be randomly assigned to one of two groups. One group will receive two individual and two small group therapy sessions targeting social anxiety issues, whilst they are on the normal waiting period for to enter Odyssey House residential treatment. Participants in this group will be asked to attend each session, which will be held at Odyssey House, 660 Bridge Road, Richmond. The sessions will be run by experienced senior therapists and there will be about 3 or 4 participants in each group. Each session of the social anxiety intervention will be audio taped, in order to ensure quality of training and provide feedback and assistance to the therapists providing the training. The recordings will not be utilised for any data analysis and audiotapes will be destroyed at the completion of the project. Once they have entered the Therapeutic Community (TC),
members of this group will also receive a combination of therapeutic letters or telephone booster sessions from the senior therapist reminding them of the skill and information received during the social anxiety program.

The therapy follows a widely used approach to treating social anxiety symptoms, including: thought challenging, shifting from critical internal focus to the social situation and facing feared situations in a gradual way. Those randomly assigned to the other group will receive the stress management education program and then commence treatment at Odyssey House at the end of the normal waiting period. All participants will receive the usual treatment for their drug and alcohol use once they enter Odyssey House.

By consenting to take part in this project you are also giving us permission to access relevant clinical information from your first appointment (i.e. the intake interview) with Odyssey House. This information will involve any mental health information and previous treatment information. We are requesting access to this information so that we do not need to ask you again during the assessment interview conducted as part of this research.

You will be asked to complete several questionnaires at four separate points in time*:

1. Initial baseline assessment interview
2. At the end of the final group therapy session
3. Three months after entering the TC.
4. Twelve months after entering the TC

Please note that if you do not enter the TC or if you are randomly assigned to the other group, you are still eligible to be included in the study and we will follow up with you at similar time points.

The questionnaires will take approximately 40 – 90 minutes to complete on each occasion and a random sample of 20% of participants will also be asked to provide a urine sample at the third interview (3 months after entering TC). The purpose of the urine sample is to confirm any self-reported alcohol and/or drug use. You are free to refuse to provide a sample.

Examples of questions asked at each point in time are:

Your background; for example “What was your employment status prior to treatment?” and “Do you receive any Centrelink benefits?”

Your anxiety symptoms; for example “Rate your levels of Fear or Anxiety in when 1. Telephoning in public 2. Eating in public places”)

Your alcohol and drug use; for example “In your life which of the following substances have you ever used?”

5. Possible Benefits

If you are randomly assigned to receive the social anxiety intervention it is possible that you will notice improvement in your social anxiety concerns. All participants will receive the regular treatment for substance abuse that is provided by Odyssey House Victoria.
The findings from this study might assist in future modification of substance abuse programs aimed to increase the effectiveness of these programs, and in particular, for people with social anxiety issues.

We cannot guarantee or promise that you personally will receive any benefits from this project.

6. **Possible Risks**

It is possible that some people may find answering questions about any anxiety they are experiencing upsetting. If you experience any immediate distress as a result of your participation please speak with your Odyssey House Victoria clinician.

During treatment you will be asked to face potentially anxiety-provoking situations, as this has been found to be effective in overcoming anxiety. You will be able to choose these situations together with your clinician such that you experience only a manageable level of anxiety.

If you would like to speak with someone at a later stage you can contact beyondblue who will be able to assist you with any concerns and with finding appropriate support. Their phone number is 1300 22 4636.

You can suspend or end your participation in the project if distress occurs.

7. **Privacy, Confidentiality and Disclosure of Information**

All information gathered from participants will be kept confidential and secure in accordance with Deakin University guidelines, and will not be used for any other purpose than that of this research. Information will not be released to any third party without full and informed consent of the participants, except as required by law. No identifiable details will be kept with the information that is stored. All information on clients collected during this research will be destroyed after 6 years.

It is possible that the results of this study will be published in a scientific journal and/or student theses however individual responses will not be identifiable as only group data will be submitted.

In accordance with the *Freedom of Information Act* 1982 (Vic), you have the right to access and to request correction of information held about you by Deakin University.

8. **Results of Project**

At the completion of the study a summary of the project’s findings will be made available upon request. A summary of the result will be available in late 2013 by contacting Associate Professor Petra Staiger (pstaiger@deakin.edu.au or 03 9244 6876).

9. **Further Information or Any Problems**

If you require further information or if you have any problems concerning this project (for example, feelings of distress), you can contact the A/Prof Petra Staiger or Dr Stefan Gruenert.

**Associate Professor Petra Staiger**  
School of Psychology  
Faculty of Health, Medicine, Nursing and Behavioural Sciences  
Deakin University  
221 Burwood Highway  
Burwood, 3125  
03 9244 6876

**Dr Stefan Gruenert**  
CEO  
Odyssey House Victoria  
660 Bridge Road  
Richmond 3121  
03 9420 7600
10. Complaints
If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact:

The Manager, Office of Research Integrity, Deakin University, 221 Burwood Highway, Burwood Victoria 3125, Telephone: 9251 7129, Facsimile: 9244 6581; research-ethics@deakin.edu.au.

Please quote project number EC 2010-007.

11. Participation is Voluntary
Participation in any research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your routine treatment, your relationship with those treating you or your relationship with the researchers, Odyssey House Victoria, Deakin University or Swinburne University of Technology.

Before you make your decision, a member of the research team will be available so that you can ask any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from the project your participation will immediately cease and you may ask for any information obtained from you will not be used.

12. Reimbursement for your costs
You will not be paid for your participation in this trial. However, you will be reimbursed with Coles Group/MYER gift vouchers to compensate you for your time. An initial reimbursement of $25 will be paid to all participants upon completion of the first interview in person, whilst participants who complete them over the phone will receive $15. Following the same policy, subsequent interviews will be reimbursed either $20 or $10 depending on whether you travel to the interview or it is conducted over the phone. Reimbursement will be made at the end of the 3 month interview. We have not yet received funding to conduct the 12 month interview and we hope to include extra reimbursement for this component in the near future.

If you are selected to receive the social anxiety treatment, or if you complete an interview in person at Odyssey House Victoria head office, additional cost for reasonable expenses incurred to attend the training sessions will also be reimbursed upon presentation of valid receipt (eg: validated METCARD or 2 hr parking ticket).

13. Ethical Guidelines
This project will be carried out according to the National Statement on Ethical Conduct in Human Research 2007 produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.
The ethical aspects of this research project have been approved by the Human Research Ethics Committee of Deakin University and Swinburne University of Technology.

*Thank you for your time. It is greatly appreciated.*
DEAKIN UNIVERSITY
PLAIN LANGUAGE STATEMENT AND CONSENT FORM

TO: Participant

Consent Form

Date: 12 August 2010

Project Title: Improving the retention rate for residential treatment of substance abuse by sequential intervention for social anxiety

- I have read and I understand the Plain Language Statement.
- I have had an opportunity to ask questions and I am satisfied with the answers I have received.
- I freely agree to participate in this project according to the conditions in the Plain Language Statement.
- I will be given a copy of the Plain Language Statement and Consent Form to keep.
- I understand that the researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .................................................................

Signature ........................................................................ Date ____________

I agree [ ] / do not agree [ ] (please tick) that the researchers may contact the Odyssey program in order to obtain my contact details after I leave, should they change from those provided below.

I understand that a copy of the results from this study should be available after November 2013. I would [ ] / would not [ ] (please tick) like a copy to be sent to me to the address below.
APPENDIX E: SWIN-SAG Participants Explanatory Statement and Consent Form

PARTICIPANT INFORMATION SHEET AND CONSENT FORM

Exploring the role of personality, beliefs about alcohol use and coping strategies in people who feel anxious in social situations

This information sheet is for you to keep. My name is Annette Raber and I am conducting this research project as part of a Doctor of Clinical Psychology, under the supervision of Professor Michael Kyrios at Swinburne University.

Many people feel anxious in social situations. Problematic substance (alcohol/drug) use also frequently occurs in the general population and can sometimes co-occur in people who feel anxious in social situations. The aim of this study is to explore how personality, thoughts and beliefs about alcohol, and coping strategies affect the relationship between social anxiety and substance use. It is hoped that the findings of this research will enhance our understanding about the nature of these two disorders and why they may co-occur. This will hopefully lead to improved educational and preventative measures, as well as more effective treatment for people with social anxiety, and/or problematic substance use.

We are looking for people aged 18 years and over with symptoms of social anxiety to participate in this research. Your involvement will require the completion of a self-report questionnaire. You can return the questionnaire when you attend the Swinburne Psychology Clinic to assess your suitability to participate in the Social Anxiety Group (“SAG”) program.

Your participation in a SAG program in no way depends on completing this questionnaire. The enclosed self-report questionnaire asks general questions, as well as questions about social anxiety, substance use (if any), current mood, personality, beliefs about alcohol use, and coping strategies. Completing this questionnaire is estimated to take no more than 30 minutes.

Your responses to some interview questions (the MINI) as well as your responses to one of the self-report questionnaires (the BFNE-II) for the SAG program will also be used for this research project. Your responses to interview questions and questionnaires will be kept confidential on University Premises in a locked filing cabinet. They will only be accessed by the researchers involved in this project. Your data will be de-identified and stored in a password protected database for statistical analysis. All information relating to this project will be destroyed after the minimum period of 7 years after publication of the results.

Findings from this study will be reported in a thesis. Findings may also be submitted for publication at a conference or in a peer-reviewed journal. If this occurs, your anonymity is
assured and no personal information will be identifiable in the results. The information you provide in this research will only be used for the purposes described in this document.

Being in this study is voluntary and you are under no obligation to consent to participation. If you do decide to take part but later change your mind, you may withdraw from the study at any time. Your decision whether to take part, not to take part, or to take part and then withdraw, will not affect your relationship with Swinburne University or Swinburne Psychology Clinic. If you decide to participate in this research, you will need to sign the following consent form to indicate you understand the information in this document and that you give your consent to participate in the research project.

This project contains potentially sensitive questions. Some participants may find the questions and responses distressing. If you are concerned either now or later as a result of the study, please don’t hesitate to contact your doctor or mental health professional. Appointments for counselling are also available at the Swinburne Psychology Clinic for a low cost at Hawthorn campus, phone (03) 9214 8653; email psychclinic@swin.edu.au. If you are experiencing a crisis, cannot contact a counsellor and need help urgently, phone Lifeline on 131 114 or the Suicide Help Line on 1300 651 251.

If you have any further questions about the study, or if you would like to know the results, please don’t hesitate to contact the researchers:

Professor Michael Kyrios  
Telephone: (03) 9214 4886  
Email: mkyrios@groupwise.swin.edu.au

Ms Annette Raber  
Telephone: 0459 775 739  
Email: annetteraber@gmail.com

This study has been approved by the Swinburne University of Technology Human Research Ethics Committee (SUHREC) in line with the National Statement on Ethical Conduct in Human Research. If you have any concerns or complaints about the conduct of this project, please contact: Research Ethics Officer, Office of Research & Graduate Studies (H68), Swinburne University of Technology, P O Box 218, HAWTHORN VIC 3122. Tel (03) 9214 5218.

Thank you for your time.
CONSENT FORM

Exploring the role of personality, beliefs about alcohol use and coping strategies in people who feel anxious in social situations

Annette Raber (Doctorate Student)
Professor Michael Kyrios (Principal Research Supervisor)

1. I have read and understood the information provided in the Participant Information Sheet for this research project.
2. I consent to participate in the project named above.
3. I agree to the use of my responses to interview questions from the MINI, as well as my responses to the BFNE-II questionnaire, all required for the SAG program, for the purpose of this research project.
4. I agree to complete questionnaires as described in the Participant Information Sheet.
5. I acknowledge that:
   a. my participation is voluntary and that I am free to withdraw from the project at any time without explanation;
   b. the project is for the purpose of research and not for profit;
   c. any personal or health information about me which is gathered in the course of and as the result of my participating in this project will be (i) collected and retained for the purpose of this project and (ii) accessed and analysed by the researcher(s) for the purpose of conducting this project;
   d. my anonymity is preserved and I will not be identified in publications.
6. By signing this document I agree to participate in this project.

Name of Participant: ………………………………………………………………………………………………………..

Signature & Date: ………………………………………………………………………………………………………
APPENDIX F: ADAVIC and SWIN-CLINIC Advertisement and Participants
Explanatory Statement/Consent Form

ADVERTISEMENT

Social Anxiety and Substance Use

Swinburne University is seeking individuals who experience anxiety in social situations for a study which is investigating the relationship between social anxiety, personality, coping strategies, beliefs about the use of alcohol, and use of substances. We are interested in exploring why some people with social anxiety use alcohol or other substances whilst others don’t.

We are interested in people who experience anxiety in social situations who are 18 years old or older.

As a participant you will be required to take part in a clinical interview (either in person or by phone) and to complete a questionnaire pack. Answering questions during the interview and completing the self-report questionnaire should take no more than 45 minutes in total.

If you are interested in being a part of this study, please contact Annette Raber (Associate Investigator) via email: annetteraber@gmail.com or by phone: 0459 775 739.

If you have any questions or comments about the study, please forward them on to Annette Raber or Professor Michael Kyrios (Principal Investigator) via email: mkyrios@swin.edu.au.

PARTICIPANT INFORMATION SHEET AND CONSENT FORM

Exploring the role of personality, beliefs about alcohol use and coping strategies in people who feel anxious in social situations

This information sheet is for you to keep. My name is Annette Raber and I am conducting this research project as part of a Doctor of Clinical Psychology, under the supervision of Professor Michael Kyrios at Swinburne University.

Many people feel anxious in social situations. Problematic substance (alcohol/drug) use also frequently occurs in the general population and can sometimes co-occur in people who feel anxious in social situations. The aim of this study is to explore how personality, thoughts and beliefs about alcohol, and coping strategies affect the relationship between social anxiety and substance use. It is hoped that the findings of this research will enhance our understanding about
the nature of these two disorders and why they may co-occur. This will hopefully lead to improved educational and preventative measures, as well as more effective treatment for people with social anxiety, and/or problematic substance use.

We are looking for people aged 18 years and over with symptoms of social anxiety to participate in this research. Your involvement will require you to take part in a clinical interview (either in person at the Swinburne Psychology Clinic or by phone) to assess your suitability to participate in this research. You will be asked to answer some questions about your symptoms of social anxiety and any symptoms you may have of substance abuse. You will also be asked to complete a self-report questionnaire which asks general questions, as well as questions about social anxiety, substance use (if any), current mood, personality, beliefs about alcohol use, and coping strategies. Participation in this study (answering questions at interview and completion of questionnaires) is estimated to take no more than 45 minutes.

Your responses to interview questions and questionnaires collected for this research project will be kept confidential on University Premises in a locked filing cabinet. They will only be accessed by the researchers involved in this project (listed above). Your data will be de-identified and stored in a password protected database for statistical analysis. All information relating to this project will be destroyed after the minimum period of 7 years after publication of the results.

Findings from this study will be reported in a thesis. Findings may also be submitted for publication at a conference or in a peer-reviewed journal. If this occurs, your anonymity is assured and no personal information will be identifiable in the results. The information you provide in this research will only be used for the purposes described in this document.

Being in this study is voluntary and you are under no obligation to consent to participation. If you do decide to take part but later change your mind, you may withdraw from the study at any time. Your decision whether to take part, not to take part, or to take part and then withdraw, will not affect your relationship with Swinburne University or Swinburne Psychology Clinic. If you decide to participate in this research, you will need to sign the following consent form to indicate you understand the information in this document and that you give your consent to participate in the research project.

This project contains potentially sensitive questions. Some participants may find the questions and responses distressing. If you are concerned either now or later as a result of the study, please don’t hesitate to contact your doctor or mental health professional. Appointments for counselling are also available at the Swinburne Psychology Clinic for a low cost at Hawthorn campus, phone (03) 9214 8653; email psychclinic@swin.edu.au. If you are experiencing a crisis, cannot contact a counsellor and need help urgently, phone Lifeline on 131 114 or the Suicide Help Line on 1300 651 251.
If you have any further questions about the study, or if you would like to know the results, please don’t hesitate to contact the researchers:

Professor Michael Kyrios  
Telephone: (03) 9214 4886  
Email: mkyrios@groupwise.swin.edu.au

Ms Annette Raber  
Telephone: 0459 775 739  
Email: annetteraber@gmail.com

This study has been approved by the Swinburne University of Technology Human Research Ethics Committee (SUHREC) in line with the National Statement on Ethical Conduct in Human Research. If you have any concerns or complaints about the conduct of this project, please contact: Research Ethics Officer, Office of Research & Graduate Studies (H68), Swinburne University of Technology, P O Box 218, HAWTHORN VIC 3122. Tel (03) 9214 5218.

Thank you for your time.
CONSENT FORM

Exploring the role of personality, beliefs about alcohol use and coping strategies in people who feel anxious in social situations

Annette Raber (Doctorate Student)
Professor Michael Kyrios (Principal Research Supervisor)

1. I have read and understood the information provided in the Participant Information Sheet for this research project.
2. I consent to participate in the project named above.
3. I agree to be interviewed by the researcher at the Swinburne Psychology Clinic to assess suitability to participate in this research.
4. I agree to complete questionnaires as described in the Participant Information Sheet.
5. I acknowledge that:
   a. my participation is voluntary and that I am free to withdraw from the project at any time without explanation;
   b. the project is for the purpose of research and not for profit;
   c. any personal or health information about me which is gathered in the course of and as the result of my participating in this project will be (i) collected and retained for the purpose of this project and (ii) accessed and analysed by the researcher(s) for the purpose of conducting this project;
   d. my anonymity is preserved and I will not be identified in publications.
6. By signing this document I agree to participate in this project.

Name of Participant: ……………………………………………………………………………

Signature & Date: ……………………………………………………………………………
APPENDIX G: The SAD, AUD and SUD Modules of the MINI

SOCIAL PHOBIA (Social Anxiety Disorder)

(☐ MEANS: GO TO THE DIAGNOSTIC BOX, CIRCLE NO AND MOVE TO THE NEXT MODULE)

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>F1</strong></td>
<td>In the past month, did you have persistent fear and significant anxiety at being watched, being the focus of attention, or of being humiliated or embarrassed? This includes things like speaking in public, eating in public or with others, writing while someone watches, or being in social situations.</td>
</tr>
<tr>
<td></td>
<td>NO</td>
</tr>
</tbody>
</table>

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>F2</strong></td>
<td>Is this social fear excessive or unreasonable and does it almost always make you anxious?</td>
</tr>
<tr>
<td></td>
<td>NO</td>
</tr>
</tbody>
</table>

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>F3</strong></td>
<td>Do you fear these social situations so much that you avoid them or suffer through them most of the time?</td>
</tr>
<tr>
<td></td>
<td>NO</td>
</tr>
</tbody>
</table>
F4 Do these social fears disrupt your normal work, school or social functioning or cause you significant distress?

SUBTYPES

Do you fear and avoid 4 or more social situations?

If YES Generalized social phobia (social anxiety disorder)

If NO Non-generalized social phobia (social anxiety disorder)

EXAMPLES OF SUCH SOCIAL SITUATIONS TYPICALLY INCLUDE

- INITIATING OR MAINTAINING A CONVERSATION,
- PARTICIPATING IN SMALL GROUPS,
- DATING,
- SPEAKING TO AUTHORITY FIGURES,
- ATTENDING PARTIES,
- PUBLIC SPEAKING,
- EATING IN FRONT OF OTHERS,
- URINATING IN A PUBLIC WASHROOM, ETC.

NOTE TO INTERVIEWER: PLEASE ASSESS WHETHER THE SUBJECT’S FEARS ARE RESTRICTED TO NON-GENERALIZED (“ONLY 1 OR SEVERAL”) SOCIAL SITUATIONS OR EXTEND TO GENERALIZED (“MOST”) SOCIAL SITUATIONS. “MOST” SOCIAL SITUATIONS IS USUALLY OPERATIONALIZED TO MEAN 4 OR MORE SOCIAL SITUATIONS, ALTHOUGH THE DSM-IV DOES NOT EXPLICITLY STATE THIS.
### ALCOHOL DEPENDENCE / ABUSE

(☐ MEANS: GO TO DIAGNOSTIC BOXES, CIRCLE NO IN BOTH AND MOVE TO THE NEXT MODULE)

<table>
<thead>
<tr>
<th>Question</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I1</strong> In the past 12 months, have you had 3 or more alcoholic drinks, - within a 3 hour period, - on 3 or more occasions?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Question</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>I2 In the past 12 months:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a Did you need to drink a lot more in order to get the same effect that you got when you first started drinking or did you get much less effect with continued use of the same amount?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b When you cut down on drinking did your hands shake, did you sweat or feel agitated? Did you drink to avoid these symptoms (for example, &quot;the shakes&quot;, sweating or agitation) or to avoid being hungover?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c During the times when you drank alcohol, did you end up drinking more than you planned when you started?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d Have you tried to reduce or stop drinking alcohol but failed?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>e On the days that you drank, did you spend substantial time obtaining alcohol, drinking, or recovering from the effects of alcohol?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
f Did you spend less time working, enjoying hobbies, or being with others because of your drinking? NO YES

g If your drinking caused you health or mental problems, did you still keep on drinking? NO YES

ARE 3 OR MORE I2 ANSWERS CODED YES?

* IF YES, SKIP I3 QUESTIONS AND GO TO NEXT MODULE. “DEPENDENCE PREEMPTS ABUSE” IN DSM IV TR.

I3 In the past 12 months:

a Have you been intoxicated, high, or hungover more than once when you had other responsibilities at school, at work, or at home? Did this cause any problems? NO YES (CODE YES ONLY IF THIS CAUSED PROBLEMS.)

b Were you intoxicated more than once in any situation where you were physically at risk, for example, driving a car, riding a motorbike, using machinery, boating, etc.? NO YES

c Did you have legal problems more than once because of your drinking, for example, an arrest or disorderly conduct? NO YES
d If your drinking caused problems with your family or other people, did you still keep on drinking?

**ARE 1 OR MORE 13 ANSWERS CODED YES?**

---

**SUBSTANCE DEPENDENCE / ABUSE (NON-ALCOHOL)**

(☐ MEANS: GO TO THE DIAGNOSTIC BOXES, CIRCLE NO IN ALL DIAGNOSTIC BOXES, AND MOVE TO THE NEXT MODULE)

---

**Now I am going to show you / read to you a list of street drugs or medicines.**

J1 a In the past 12 months, did you take any of these drugs more than once, to get high, to feel elated, to get “a buzz” or to change your mood?

| Cocaine: snorting, IV, freebase, crack, "speedball". |
| Narcotics: heroin, morphine, Dilaudid, opium, Demerol, methadone, Darvon, codeine, Percodan, Vicodin, OxyContin. |
Hallucinogens: LSD ("acid"), mescaline, peyote, psilocybin, STP, "mushrooms", "ecstasy", MDA, MDMA.

Phencyclidine: PCP ("Angel Dust", "Peace Pill", "Tranq", "Hog"), or ketamine ("Special K").

Inhalants: "glue", ethyl chloride, "rush", nitrous oxide ("laughing gas"), amyl or butyl nitrate ("poppers").

Cannabis: marijuana, hashish ("hash"), THC, "pot", "grass", "weed", "reefer".

Tranquilizers: Quaalude, Seconal ("reds"), Valium, Xanax, Librium, Ativan, Dalmane, Halcion, barbiturates, Miltown, GHB, Roofinol, "Roofies".

Miscellaneous: steroids, nonprescription sleep or diet pills. Cough Medicine? Any others?

SPECIFY THE MOST USED DRUG(s):

WHICH DRUG(s) CAUSE THE BIGGEST PROBLEMS?:

FIRST EXPLORE THE DRUG CAUSING THE BIGGEST PROBLEMS AND MOST LIKELY TO MEET DEPENDENCE / ABUSE CRITERIA.

IF MEETS CRITERIA FOR ABUSE OR DEPENDENCE, SKIP TO THE NEXT MODULE. OTHERWISE, EXPLORE THE NEXT MOST PROBLEMATIC DRUG.

Considering your use of (NAME THE DRUG / DRUG CLASS SELECTED), in the past 12 months:

a Have you found that you needed to use much more (NAME OF DRUG / DRUG CLASS SELECTED) NO YES
to get the same effect that you did when you first started taking it?

b When you reduced or stopped using (NAME OF DRUG / DRUG CLASS SELECTED), did you have NO YES
withdrawal symptoms (aches, shaking, fever, weakness, diarrhea, nausea, sweating, heart pounding, difficulty sleeping, or feeling agitated, anxious, irritable, or depressed)?
Did you use any drug(s) to keep yourself from getting sick (withdrawal symptoms) or so that you would feel better?

IF YES TO EITHER, CODE YES.
c  Have you often found that when you used \textit{(NAME OF DRUG / DRUG CLASS SELECTED)}, you ended up taking more than you thought you would? NO  YES

d  Have you tried to reduce or stop taking \textit{(NAME OF DRUG / DRUG CLASS SELECTED)} but failed? NO  YES

e  On the days that you used \textit{(NAME OF DRUG / DRUG CLASS SELECTED)}, did you spend substantial time (>2 hours), obtaining, using or recovering from the drug, or thinking about the drug? NO  YES

f  Did you spend less time working, enjoying hobbies, or being with family or friends because of your drug use? NO  YES

g  If \textit{(NAME OF DRUG / DRUG CLASS SELECTED)} caused you health or mental problems, did you still keep on using it? NO  YES

\textbf{ARE 3 OR MORE J2 ANSWERS CODED YES?}

\textbf{SPECIFY DRUG(S): ____________________________}

\textbf{* IF YES, SKIP J3 QUESTIONS, MOVE TO NEXT DISORDER.}

\textit{“DEPENDENCE PREEMPTS ABUSE” IN DSM IV TR.}

\textbf{Considering your use of \textit{(NAME THE DRUG CLASS SELECTED)}, in the past 12 months:}

J3  a  Have you been intoxicated, high, or hungover from \textit{(NAME OF DRUG / DRUG CLASS SELECTED)} more than once, when you had other responsibilities at school, at work, or at home? NO  YES
Did this cause any problem?

(CODE YES ONLY IF THIS CAUSED PROBLEMS.)

b Have you been high or intoxicated from (NAME OF DRUG / DRUG CLASS SELECTED) more than once in any situation where you were physically at risk (for example, driving a car, riding a motorbike, using machinery, boating, etc.)?

  NO  YES

c Did you have legal problems more than once because of your drug use, for example, an arrest or disorderly conduct?

  NO  YES

d If (NAME OF DRUG / DRUG CLASS SELECTED) caused problems

  YES

  with your family or other people, did you still keep on using it?

  NO

ARE 1 OR MORE J3 ANSWERS CODED YES?

SPECIFY DRUG(s): ________________________________
APPENDIX H: Flowchart of Procedure for Clinical Participants by Recruitment Method

ARC STUDY  
\(n = 44\)  
Clinical Interview  
In person/by phone  
Self-report questionnaire  
In person/by post

SWIN-SAG  
\(n = 30\)  
Clinical Interview  
In person  
Self-report questionnaire  
In person/by post

ADOVIC  
\(n = 4\)  
Clinical Interview  
By phone  
Self-report questionnaire  
By post

SWIN-PSYC CLINIC  
\(n = 6\)  
Clinical Interview  
In person  
Self-report questionnaire  
In person/by post
**APPENDIX I: Entering Covariates Separately in AEN (negative alcohol expectancies)**

**ANCOVA**

*AEN ANCOVA – Covariates entered separately*

<table>
<thead>
<tr>
<th>Covariates</th>
<th>F value</th>
<th>Significance</th>
<th>Partial Eta Squared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>2.820</td>
<td>.096</td>
<td>.026</td>
</tr>
<tr>
<td>All groups</td>
<td>9.102</td>
<td>.000</td>
<td>.208</td>
</tr>
<tr>
<td>Age</td>
<td>.342</td>
<td>.560</td>
<td>.003</td>
</tr>
<tr>
<td>All groups</td>
<td>8.166</td>
<td>.000</td>
<td>.191</td>
</tr>
<tr>
<td>Depression</td>
<td>.028</td>
<td>.973</td>
<td>.001</td>
</tr>
<tr>
<td>All groups</td>
<td>5.106</td>
<td>.002</td>
<td>.131</td>
</tr>
</tbody>
</table>

*Note. AEN = negative alcohol expectancies.*