Does Impulsivity Help in Understanding the Relationship Between Social Anxiety and Alcohol Use Disorders?

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Thesis Summary

Social anxiety disorder (SAD) and alcohol use disorders (AUDs) are highly prevalent diagnoses which co-occur at a high rate within clinical and epidemiological populations. Studies concur that the high rates of comorbidity between social anxiety and alcohol use disorders are related to increased chronicity and severity, poorer quality of life, and less effective treatment outcomes. Although the relationship between social anxiety and alcohol use disorders has been the subject of extensive exploration, previous studies have failed to draw consistent conclusions about the direction and nature of this relationship. Understanding and delineating factors that underlie this relationship are important precursors to effective diagnosis and treatment, and individual variables such as impulsivity have recently been considered in the literature as a factor that may impact this relationship. Thus, the aim of this thesis was to empirically evaluate impulsivity as a contributor to the comorbidity between social anxiety and alcohol use disorders; to better understand the risk and maintenance factors involved in this relationship. The relationship between social anxiety and alcohol use disorders was investigated empirically using both clinical, non-clinical, treatment-seeking and non treatment-seeking samples, and employing optimal experimental methodologies.

Four separate studies were conducted: The first examined the rates of alcohol use disorders and the impact of alcohol use and drinking behaviours on outcomes for treatment targeting social anxiety disorder, and showed how planned alcohol consumption prior to social situations and drinking during social situations are associated with more severe social anxiety symptoms. The second investigated the role of a two-factor model of impulsivity using both trait and behavioural measures—to differentiate between dependent and non-dependent drinkers with, and without, comorbid social anxiety—and found that both components are useful in differentiating between those with, and without, this comorbidity. The third examined the physiological and subjective responses to alcohol ingestion and perceived intoxication, and their association with impulsivity and alcohol expectancies, in a treatment-

seeking sample of individuals with comorbid social anxiety disorder and alcohol problems, and revealed how impulsivity and social anxiety may maintain problematic drinking via two pathways involving the subjective and physiological effects of alcohol. The role of alcohol expectancies was observed to be less influential in regulating responses to alcohol cues. The final study explored whether impulsivity influenced social anxiety and alcohol treatment outcomes in individuals with comorbid social anxiety and alcohol use disorders. This study found that higher impulsivity was related to more severe social anxiety and alcohol dependence initially, but also a greater reduction in these symptoms following treatment. These findings highlight that understanding the impact of impulsivity on treatment targeting these problems may have prognostic utility and suggests that these individuals may benefit from a well structured therapeutic approach.

Taken together, these studies advance our understanding of the relationships between these highly comorbid disorders that can have a severe impact on quality of life. Understanding these relationships is essential because in order to decrease comorbidity and the problems associated with it, we must first understand the risk and maintenance factors involved in the relationship. This body of work contributes to the literature by using a contemporary two-factor model of impulsivity to extend our knowledge about the co-occurrence between social anxiety and alcohol use problems. Specifically, how two components of impulsivity, reward drive and rash impulsivity, relate to who develops SAD-AUD comorbidity, how these problems are maintained, and how individuals with these problems respond to treatment interventions.

Statement of Candidate

I certify that the work in this thesis entitled "Understanding the Relationship between Social Anxiety and Alcohol Use Disorders" has not previously been submitted for a degree nor has it been submitted as part of requirements for a degree to any other institution other than Macquarie University. I also certify that the thesis is my own work and it has been written by me. In addition, I certify to the best of my knowledge and belief, it does not contain material from published sources without proper acknowledgement.

Each of the empirical chapters lists multiple authors. I, Mirjana Subotic-Kerry, am the primary author of each chapter. The contributions of each author to the empirical chapters are as follows:

Chapter Two: Data for this study were obtained from an existing data set from an earlier clinical trial, which was designed and run by Prof. Rapee, Associate Professor Baillie, Mr. Gaston, and A/Prof. Abbott (all listed as co-authors). Ms. Wagner created the questionnaire designed for the study (listed as a co-author). I was responsible for the data analysis and preparation of the manuscript for publication. A/Prof. Baillie and Dr. Stapinski provided guidance throughout these processes and provided feedback on the manuscript.

Chapter Three: Ms. Tulloch and I worked collaboratively to design the general features of the study and jointly prepared the Ethics Committee application. Data were collected by Ms. Tulloch and me. I was responsible for the management of the data analysed for Chapter Three, specifically. I was also responsible for the for the conception of the research question, the planning and execution of data analyses, interpretation of results, and preparation of the manuscript for publication. A/Prof. Baillie and Dr. Stapinski provided guidance throughout these processes and provided feedback on the manuscript.

Chapter Four: General features of the overall design of this study were planned in discussion between co-authors Dr. Stapinski, Ms. Tulloch and me, with advice from A/Prof. Baillie. Data were collected and managed by Dr. Stapinski, Ms. Tulloch, Mr. Bakovic and me.

I was responsible for the conception of the research question, the planning and execution of data analyses, interpretation of results, and preparation of the manuscript for publication. A/Prof. Baillie provided guidance throughout these processes, and both A/Prof Baillie and Dr. Stapinski provided feedback on the manuscript.

Chapter Five: Data were collected as part of a larger clinical trial, which was led by A/Prof. Baillie, Prof. Teesson, Dr. Sannibale, Prof. Haber, and Prof. Rapee (all listed as co-authors). I worked with A/Prof. Baillie and Dr. Stapinski to design the study that formed the basis of Chapter Five. I was responsible for the collection and management of the data analysed for this chapter. Dr. Stapinski assisted with the imputation analyses; I conducted the primary data analyses under the supervision of Dr. Stapinski and A/Prof. Baillie. I was responsible interpretation of results, and preparation of the manuscript for publication, with feedback from A/Prof. Baillie and Dr. Stapinski.

A/Prof. Baillie and I spent a considerable amount of time discussing the ideas in Chapters One and Six, and both A/Prof. Baillie and Dr. Stapinski provided conceptual and editorial feedback on these chapters. Susan Taylor proofread all six chapters. Mark Bakovic proofread Chapters Two, Three, Four and Five, and Kristen Tulloch proofread Chapters One, Three and Four.

The research in this thesis was approved by the Human Research Ethics Committees at Macquarie University, reference number: 5201001453 on 20/12/2010, reference number: HE26JUN2009-R00026 on 18/03/2010, and reference number HE28MAR2008-R05758 on 02/05/2008. Approval letters can be found in Appendix A.

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They say it takes a village. In my case, with two young children, I feel like it took a small country.

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Lastly, my sincerest thanks to all the participants who gave their time and effort to make this research possible.

Chapter One:

General Introduction

CHAPTER 1 – GENERAL INTRODUCTION

General Introduction

This thesis examines impulsivity as a contributor to the comorbidity between social anxiety and alcohol use disorders. A robust body of research has found that heightened impulsivity acts as a risk factor for excessive alcohol use and alcohol-related problems (e.g., Dawes, Tarter, & Kirisci, 1997; Verdejo-Garcia, Lawrence, & Clark, 2008). The relationship between social anxiety and impulsivity, however, is less straightforward. While there is a high comorbidity between social anxiety disorder and disorders typically characterised by impulsive symptoms (e.g., alcohol use disorders; Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997; Merikangas, Stevens, et al., 1998), several studies have also reported little or no direct association between anxiety and impulsivity (e.g., Askenazy, Caci, Myquel, Darcourt, & Lecrubier, 2000; Eisenberg et al., 2009; Hussong, Curran, & Chassin, 1998; Krueger, Caspi, Moffitt, White, & Stouthamer-Loeber, 1996; O'Brien & Frick, 1996). Recent cross-sectional survey data suggests high levels of impulsivity may strengthen the relationship between social anxiety and alcohol problems (e.g., Booth & Hasking, 2009), and that high impulsivity also characterises a more "impulsive" socially anxious subgroup of individuals (e.g., Nicholls, Staiger, Williams, Richardson, & Kambouropoulos, 2014). Combined with the prominence of impulsivity in alcohol use, and the link between social anxiety and alcohol problems, the recent evidence for the role of impulsivity in this relationship highlights the importance of examining the *nature* of this role as a potential functional link between these co-occurring disorders. By implementing a range of cross-sectional, clinical trial and laboratory data, and applying a novel neurobiologically informed model of impulsivity incorporating two facets, reward drive and rash impulsivity, this thesis asks: "How does impulsivity inform our understanding of the relationship between social anxiety and alcohol use disorders?"

Social anxiety disorder commonly co-occurs with alcohol use disorders within clinical and epidemiological populations, with each condition doubling to tripling the risk of the other

(Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997; Merikangas et al., 1998). In Australia, the 12-month prevalence rates of alcohol abuse and/or dependence in individuals diagnosed with social anxiety disorder was approximately 16% among the 10,641 Australian adults who completed the National Survey of Mental Health and Wellbeing (Burns & Teesson, 2002). Conversely, 3.7% of individuals with alcohol use disorders had a diagnosis of social anxiety disorder (Burns & Teesson, 2002), and were twice as likely as the general population to develop social anxiety disorder (Teesson et al., 2010). Individually, these disorders are associated with marked distress and decreased quality of life. The experience of these disorders combined can have an even greater negative impact on an already compromised quality of life (e.g., Wolitzky-Taylor, Operskalski, Ries, Craske, & Roy-Byrne, 2011).

Cumulative evidence over the past few decades from epidemiological and clinical studies examining the unique connection between social anxiety and alcohol use disorders has shown the interactions between these disorders to be complex and multifaceted, emphasising the importance of delineating maintenance factors that contribute to this relationship. Social anxiety disorder may increase the risk of the development of alcohol use disorders, and may alter the presentation and the course of illness for them. Similarly, alcohol use disorders may alter the presentation and the course of illness for social anxiety disorders. Specifically, co-occurring social anxiety and alcohol use disorders are associated with greater symptom severity, higher levels of psychiatric comorbidity, increased levels of disability, and poorer treatment response relative to either disorder alone (Bakken, Landheim, & Vaglum, 2005; Brady & Lydiard, 1993; Burns & Teesson, 2002; Burns, Teesson, & O'Neill, 2005; Randall, Thomas, & Thevos, 2001; Schneier, Martin, Liebowitz, Gorman, & Fyer, 1989; Teesson, Slade, & Mills, 2009; Thomas, 1999; Wolitzky-Taylor, Operskalski, Ries, Craske, & Roy-Byrne, 2011). However, despite evidence from clinical and epidemiological studies demonstrating a strong relationship between social anxiety and alcohol use disorders, the

direction of the relationship between social anxiety and alcohol-related variables has been mixed in non-clinical samples. Here, social anxiety has been related to both increased (Kidorf & Lang, 1999) and decreased alcohol use (Bruch et al., 1992; Eggleston, Woolaway-Bickel, & Schmidt, 2004), and in some instances, no relationship has been found (Ham & Hope, 2005; Tran, Haaga, & Chambless, 1997). Thus, the relationship between social anxiety and alcohol use disorders appears to be affected by other variables such as contextual and individual factors (Morris, Stewart, & Ham, 2005).

The common comorbidity of social anxiety and alcohol use disorders has significant public health implications as well as consequences for the individual and society. Despite the substantial public health impact and personal cost of this co-occurrence, there exists a poor understanding of the mechanisms underlying this association and the optimal way to treat this population. There are a number of promising theories to account for the relationship between social anxiety and alcohol use disorders. However, these theories struggle to account for the inconsistencies in the literature. The relationship between social anxiety and alcohol appear to be affected by contextual and individual factors, suggesting there is scope for an alternative approach in examining this relationship.

Comorbidity has emerged as an important clinical and research concern. Literature to date has been dominated by single disorder models, and there is a need for a more integrated understanding of these problems. This thesis aims to advance our understanding of the nature of the comorbid relationship between social anxiety and alcohol use disorders. It consists of six chapters, four of which are self-contained empirical papers. This first chapter provides an overview and context for the four empirical papers that make up chapters two to five. The sixth and final chapter places the four empirical papers in a broader theoretical context. In the remainder of this chapter, the themes of this thesis are introduced, a review of the pre-existing findings on the relationship between social anxiety and alcohol use disorders will be

presented, and an outline of the aims and structure of the program of research will be provided.

For the purpose of this thesis, the definition of alcohol use disorders will be that provided in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Association, 2000) which refers to two disorders, alcohol abuse and alcohol dependence, with specific criteria for each. In the most recent revision of the manual, DSM-5 (American Psychiatric Association, 2013), alcohol abuse and alcohol dependence have been combined into a single diagnosis called "alcohol use disorder" which is measured on a continuum from mild to severe. Because these criteria have been only recently published, the studies presented in this thesis had not yet adopted this new classification system.

Similarly, the definition of social anxiety disorder will be those provided in DSM-IV. The DSM-5 has made very minor changes in the definition of social anxiety disorder. Social anxiety disorder, also known as social phobia in DSM-IV, is only referred to as social anxiety disorder in DSM-5. Additionally, in DSM-5, the minimum symptom period of six months or longer has been expanded to include adults in addition to children. The term social anxiety disorder is used throughout this thesis for consistency.

Comorbidity Among Mental Disorders

In the field of mental health, comorbidity refers to the co-occurrence of two or more mental disorders (as defined in the *Diagnostic and Statistical Manual of Mental Disorders* or the WHO's *International Classification of Diseases*) in an individual either at the same time or in some causal sequence (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Ollendick & King, 1994). Common mental disorders are often correlated, and have consistently been found to co-occur more frequently than would be expected by chance (Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Kessler et al., 1994). Indeed, comorbidity seems to be the rule rather than the exception with mental disorders as is clear from studies in several countries

including Australia (Andrews, Slade, & Issakidis, 2002; Hall, Degenhardt, & Teesson, 2009; Merikangas, Stevens, et al., 1998).

Comorbidity could arise for a number of reasons. First, comorbidity may occur due to inadequate assessment and conceptualisation of mental disorders. Extending the work of Klein and Riso (1993), Neale and Kendler (1995) developed several theoretical models of comorbidity to explain the variation of two disorders when they occur simultaneously. For instance, comorbidity may result from one disorder generating symptoms of another disorder, and vice versa, or appear when the risk factors for two disorders are correlated. Alternatively, one disorder may serve as a risk factor for the other disorder, subsequently influencing the development of the other. Thus, two co-occurring disorders may have indirect effects on each other, as well as sharing some causal variables, and impacting contexts that act to either exacerbate or weaken the other disorder.

Comorbidity may also be artefactual due to study methodology (Caron & Rutter, 1991). For example, estimates of comorbidity could be due to chance, or may be susceptible to potential confounding by sampling biases or population stratification. Berkson's bias (Berkson, 1946) refers to the observation that disorders that frequently co-occur are more common in clinical, or treatment-seeking samples, than in the general population, even when the likelihood of seeking treatment is independent from disorder severity. Thus, research in clinical samples may overstate the strength of comorbidity.

The causes or causal chains of disorders are complex, and may involve genetic, biological, environmental and social risk factors (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Risk factors play a significant role in prediction and prevention and can be defined as a type of correlate that is linked to an increased probability of an outcome (Kraemer et al., 1997). Risk factors differ from other correlates that are positively or negatively associated with an outcome in that they precede the outcome and can be applied to separate a population into two subgroups (high and low risk; Kraemer et al., 1997). Kraemer et al. (1997) describe three

types of risk factors: fixed markers, variable markers and causal risk factors. A fixed and nonvarying marker is a risk factor that cannot change, whereas a variable marker does not alter the risk of an outcome when manipulated (Kraemer et al., 1997). Finally, a causal risk factor can be modified, and when altered, can change the outcome (Kraemer et al., 1997). Any disorder may have numerous risk factors, some of which will not be causal and will play only a minor role in the development or course of a disorder (Kraemer et al., 1997). In the case of comorbidity, the presence of one disorder may act as a risk factor; making the other more likely. Alternatively, comorbidity may indicate that the pathway leading to the development of one disorder is the same or similar as the pathway by which they develop another disorder as a result of shared risk factors for the two disorders.

These terms risk, marker, and cause are often used in different ways in the literature. The same term is frequently used to describe different concepts, and the same concepts are frequently described by different terms (Kraemer et al., 1997). For example, a fixed marker may be referred to as a vulnerability or susceptibility factor or a causal risk factor (Kraemer et al., 1997). It is essential to distinguish among different types of risk factors for prevention and intervention. For instance, risk factors for a specific outcome may vary in type and strength in different populations (Offord & Kraemer, 2000). In addition, risk factors may vary for the onset of a disorder compared to those for remission or relapse, and relate to prevention, treatment and maintenance respectively (Offord & Kraemer, 2000). Further, risk factors can only be measured in populations with variation in the frequency of the risk factor and outcome (Kraemer et al., 1997).

Models of Comorbidity Between Social Anxiety and Alcohol Use Disorders.

The comorbidity among social anxiety and alcohol use disorders is well documented. A number of processes have been proposed to underlie the comorbidity between these disorders including: (1) common risk factors may account for two independent disorders that co-occur (the common factor model), (2) anxiety symptoms are caused by the effects of alcohol

intoxication and/or withdrawal (the substance-induced model), and (3) one disorder directly or indirectly causing the other (self-medication model).

There is some evidence that the relationship between anxiety and alcohol is the result of common underlying variables such as biological (e.g., genetic; Merikangas, Stevens, et al., 1998; Tambs, Harris, & Magnus, 1997) or personality traits (e.g., anxiety sensitivity; DeHaas, Calamari, Bair, & Martin, 2001; DeMartini & Carey, 2011). Others argue that alcohol problems are primary, and symptoms of anxiety are a consequence of chronic alcohol use and repeated alcohol withdrawal. For example, there is evidence that certain individuals develop social anxiety that is secondary to alcohol dependence (Schuckit & Hesselbrock, 1994). Additionally, the arousing and depressant effects of alcohol use either during intoxication or withdrawal can be seen to complicate diagnosis of social anxiety, by masking or exacerbating anxiety symptoms (McHugh, 2015). However, empirical evaluation of the common factor and substance-induced models has been sparse and limited.

The most prominent perspective for social anxiety and alcohol use disorder comorbidity in the clinical and research literature is the self-medication hypothesis (Carrigan & Randall, 2003; Kushner, Abrams, & Borchardt, 2000). In line with earlier explanatory models for social anxiety and alcohol use comorbidity, including the tension reduction hypothesis (Cappell & Greeley, 1987; Conger, 1956) and the stress-response dampening hypothesis (Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sher & Levenson, 1982), the self-medication hypothesis proposes that alcohol is used as a means to alleviate symptoms of anxiety and distress (Khantizian, 1985), and through a process of reinforcement, repeated use of alcohol in this context may lead to dependence (Kushner, Abrams, & Borchardt, 2000). The pharmacological and/or alcohol expectancy effects (i.e., the cognitive, affective and behavioural beliefs an individual holds about drinking; Brown, Goldman, Inn, & Anderson, 1980) are two proposed mechanisms through which alcohol reduces aversive mood states. Indeed, empirical evidence shows that adults who expect positive outcomes drink more

frequently (Fromme, Stroot, & Kaplan, 1993), consume greater amounts of alcohol (Christiansen, Goldman, & Brown, 1985), and show more signs of problem drinking (Werner, Walker, & Greene, 1993). However, expectancies may also operate without reference to whether or not drinking reduces aversive mood states. For example, individuals who believe alcohol is tension reducing may still choose to drink even if objective evidence suggests that drinking worsens their negative affect.

The self-medication hypothesis is consistent with studies showing an age onset of social anxiety disorder that predates that of alcohol problems (Bakken, Landheim, & Vaglum, 2003; Bakken et al., 2005; Cox, Norton, Swinson, & Endler, 1990; Crum & Pratt, 2001; Merikangas, Stevens, et al., 1998; Schuckit et al., 1997), and studies indicating that social anxiety disorder tends to be primary and substance-independent when the two disorders co-occur (Kessler et al., 1997; Kushner, Sher, & Beitman, 1990). Further support for the self-medication hypothesis comes from studies showing that a large proportion of individuals with social anxiety frequently report using alcohol to cope with their social anxiety (e.g., Buckner et al., 2012; Carrigan & Randall, 2003), and that self-medication with alcohol predicts the onset of alcohol dependence (Kushner, Sher, & Erickson, 1999).

Whilst this hypothesis is consistent with the acute anxiolytic effects of alcohol, the self-medication hypothesis is less consistent with the longer-term effects of chronic alcohol use and withdrawal which results in increased anxiety symptoms (Kushner, Abrams, & Borchardt, 2000; Morris, Stewart, & Ham, 2005). Specifically, ingestion of large doses of alcohol in situations individuals find fearful may inhibit anxiety-inducing thoughts and reduce attention to anxiety inducing stimuli; factors believed to underlie anxiety disorders (Baillie et al., 2013). Thus, alcohol may worsen anxiety symptoms and may maintain anxiety disorders (Carrigan & Randall, 2003). More recent explanations see interactive processes occurring between the two disorders, where anxiety and alcohol disorders influence and maintain each other (Kushner, Abrams, & Borchardt, 2000). Kushner, Abrams, and Borchardt (2000) argue

that the mechanisms involved in the initiation of comorbidity may be different to those involved in maintaining it. For example, anxiety disorders may be responsible for the development of and relapse to chronic alcohol use, whereas other mechanisms may function to maintain ongoing alcohol use (Kushner, Abrams, & Borchardt, 2000). Increasingly, research has moved towards identifying variables that either moderate or mediate the relationship between social anxiety and alcohol use disorders (Morris, Stewart, & Ham, 2005).

Whilst the common factor, substance-induced and self-medication models offer some explanation of the comorbid relationship between social anxiety and alcohol use disorders, there is scope for the examination of other variables that may contribute to the mechanisms that underlie this relationship. For instance, although social anxiety and alcohol use disorders frequently co-occur, a substantial proportion of individuals with social anxiety disorder do not develop alcohol use disorders (Buckner et al., 2012). Thus, heterogeneity exists between individuals with social anxiety disorder as only some appear to be at greater risk of developing an alcohol use disorder. This indicates that there may be contextual and/or individual factors that moderate the relationship between social anxiety and alcohol use disorders, and these would be useful to identify.

Understanding Comorbidity - Towards a New Approach

Comorbidity among mental disorders presents a significant conceptual issue for psychopathology research and clinic work (Krueger & Markon, 2006). For instance, in research, these issues relate to selecting cases to investigate when the typical case does not fit precisely into a specific diagnostic category (Krueger & Markon, 2006). Similarly, in the clinic, these issues relate to conceptualising cases that seem to consist of two distinct disorders occurring simultaneously (Krueger & Markon, 2006). These problems stem in part from the system of classifying mental disorders since the publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III). Since this publication,

mental disorders are conceptualised as being present or absent on the basis of a threshold established by polythetic diagnostic criteria (i.e., based on a broad set of criteria; only some of which are essential; Widiger & Lynam, 1998). Consequently, diverse diagnostic and prognostic profiles are observed in individuals who meet criteria for a specific mental disorder (Krueger, Watson, & Barlow, 2005). Whilst membership based on differences between observable symptoms, signs or behaviours to classify disorders maximises clinical utility and reliability (Widiger & Sankis, 2000), the underlying processes may not map coherently to those markers to reveal processes that underlie each disorder, thus limiting validity (Goldberg, 2015).

Research indicates that though structural models treat psychopathological disorders as monothetic entities (i.e., membership based on the presence of a defined set of attributes), far more within-category heterogeneity exists among individuals with mental disorders such as social anxiety disorder (e.g., Hofmann, Heinrichs, & Moscovitch, 2004). Similarly, the failure to identify a single mechanism of social anxiety and alcohol use disorder comorbidity may stem from the myriad subtypes of comorbid syndromes, as well as to the heterogeneity of substance use disorders (Merikangas, Mehta, et al., 1998). For example, drinking alcohol induces a variety of effects and phenomena that result from the interaction of pharmacological, psychological and contextual influences that may differ from the risk factors and individual characteristics associated with alcohol use disorders (Merikangas, Mehta, et al., 1998).

Structural models of psychopathological diagnostic data have identified patterns of correlations among common DSM-defined mental disorders and found two distinct but related dimensions—internalising and externalising—to account for the widespread comorbidity among common mental disorders that is present in individuals in the community (Krueger, 1999). Internalising psychopathology is characterised by negative affect and includes anxiety and mood disorders whereas externalising psychopathology encompasses

substance use disorders and antisocial personality disorder (Krueger, Caspi, Moffitt, & Silva, 1998). Both dimensions have been consistently linked to personality traits in the broad domain of negative emotionality and neuroticism, however disorders in the externalising dimension have also been linked to disinhibition and impulsivity (Krueger, Markon, Patrick, & Iacono, 2005). Evidence from factor analytic, behaviour-genetic and longitudinal studies also indicate significant overlap between internalising and externalising disorders (Kim-Cohen et al., 2003; Krueger, 1999; Slade & Watson, 2006; Watson, 2005).

The Role of Impulsivity in Understanding the Social Anxiety-Alcohol Relationship

Impulsivity and Alcohol.

Several maladaptive personality traits have been found to be more prevalent among individuals with alcohol problems compared to the general population. That is, those individuals who begin consuming alcohol may be more likely to have pre-existing traits (e.g., impulsivity) that increase their risk of developing alcohol problems and inhibit their capacity to quit (de Wit, 2009; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; Verdejo-Garcia et al., 2008). There is evidence from cross-sectional studies that elevated levels of impulsivity, as assessed with either self-report or behavioural measures, are associated with problematic alcohol use and alcohol use disorders (see Verdejo-Garcia et al., 2008 for a review). Findings from prospective studies also reveal increased impulsivity as a risk factor for problematic alcohol use and dependence in adulthood (e.g., Caspi, 2000; Fergusson, Boden, & Horwood, 2008). This relationship has been replicated in both clinical and non-clinical populations and adolescents (Dom, De Wilde, Hulstijn, Van Den Brink, & Sabbe, 2006; Franken & Muris, 2006; Ibáñez et al., 2010).

Despite the role impulsivity plays in the initiation and maintenance of problematic alcohol use, very few studies have assessed the impact of impulsivity on alcohol treatment outcomes (Loree, Lundahl, & Ledgerwood, 2015). Those studies that have examined impulsivity and alcohol outcomes have indicated that impulsivity may be an important variable in predicting

relapse risk and treatment drop out among alcohol abusing individuals (e.g., Charney, Zikos, & Gill, 2010; Evren, Durkaya, Evren, Dalbudak, & Cetin, 2012; Kravitz, Fawcett, McGuire, Kravitz, & Whitney, 1999; Muller, Weijers, Boning, & Wiesbeck, 2008; Stevens et al., 2014). Further, a recent review by Loree et al. (2015) found that higher pre-treatment impulsivity is generally associated with poorer treatment outcomes; specifically, increased alcohol consumption and shorter time to relapse.

Two-Factor Conceptualisation of Impulsivity in Alcohol Misuse.

Traditionally, definitions of impulsivity include factors such as inadequate planning, decreased response inhibition, and the preference for short-term over long-term gains despite negative consequences (Dawe & Loxton, 2004; Evenden, 1999; Moeller et al., 2001). However, a more recent conceptualisation of impulsivity combining evidence from factor analytic studies and research on the neuroscience of substance use, focuses on two separate but interconnected systems which facilitate the development and maintenance of drug use (Dawe, Gullo, & Loxton, 2004; Dawe & Loxton, 2004). This 2-Component Approach to Reinforcing Substances (2-CARS; Gullo & Dawe, 2008) model describes two factors, rash impulsivity and reward drive, that are believed to have separate influences on the development and course of alcohol use problems.

The first component, reward drive, relates to individual differences in sensitivity to rewarding stimuli in the environment (Dawe & Loxton, 2004). For example, in the context of alcohol use, reward sensitive individuals may be more likely to engage in alcohol use for the pleasurable effects expected, and will experience greater positive responses to reward cues (Gullo & Dawe, 2008). Positive relationships have been found between reward drive and levels of hazardous drinking in community samples of adults, high school and university students (Franken & Muris, 2006; Knyazev, 2004; Loxton & Dawe, 2001; O'Connor & Colder, 2005). In addition, several laboratory studies show that reward dive is associated with a bias to detect and react to drug-related stimuli (Franken, 2002; Kambouropoulos & Staiger,

2001, 2004b). Theoretically, the underlying motivational system reflects Gray's (1987).

Behavioural Approach System (BAS) which is thought to be related to the mesolimbic

dopamine system (Dawe et al., 2004). This component is also similar to the trait "novelty

seeking" as described in Cloninger's (1987) psychobiological model of personality. Both

theories propose that the underlying neural system of this personality dimension is a

behavioural approach/activation system and relate to differences in sensitivity to reward

stimuli.

The second component of Dawe et al.'s (2004) two-factor model, rash impulsivity, is

associated with the tendency to act rashly and without consideration of consequences. This

component is believed to be associated with the functioning of the prefrontal "executive"

system: the orbitofrontal cortex and the ventromedial prefrontal cortex (Dawe & Loxton,

2004). Specifically, individuals high on rash impulsivity may have difficulty changing or

stopping their behaviour despite negative consequences. In the context of alcohol use, once

drinking behaviour has been initiated, individuals high on this trait are thought to have a

decreased ability to cease alcohol use. Evidence indicates that a lack of control over

behavioural responses may play a role in the continuation of substance use (Jentsch & Taylor,

1999; Lubman, Yucel, & Pantelis, 2004). High levels of rash-impulsivity in childhood have

also been found to predict later drug use in a number of longitudinal studies (McGue, Iacono,

Legrand, Malone, & Elkins, 2001; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004).

Dawe et al. (2004) propose that individuals differ in the degree to which they express these

two traits of impulsivity.

In their 2-Component Approach to Reinforcing Substances (2-CARS) model describing

how impulsivity conveys risk for substance abuse, Dawe et al. (2004) labelled the first

component *Reward Drive*. In an earlier publication, this trait is referred to as *Reward*

Sensitivity (Dawe & Loxton, 2004), and has also been referred to as *Sensitivity to Reward*.

The second component, *Rash Impulsivity*, has been referred to as *Disinhibition.* These labels

are used interchangeably in the literature. For consistency, the terms reward drive and rash impulsivity are used throughout this thesis.

Impulsivity, Alcohol and Social Anxiety.

To date, very few studies have examined the effect of impulsivity on the social anxiety and alcohol relationship. Booth and Hasking (2009) were the first to examine the role of one facet of impulsivity, reward drive, in the relationship between social anxiety and drinking behaviour. Specifically, they assessed the moderating role of reward drive and alcohol expectancies, in the relationship between symptoms of social anxiety and alcohol use. In a community sample of young adults, three-way relationships between reward drive, alcohol expectancies and social anxiety were observed to predict alcohol consumption. That is, they found that for individuals with elevated reward drive who held strong tension reduction expectancies, a positive relationship between the anxiety subscale in the measurement of social anxiety and drinking was present (Booth & Hasking, 2009). This relationship was not observed for individuals scoring higher on the avoidance social anxiety subscale. They argue that elevated reward drive in individuals with lower levels of avoidance who approach social situations with fear, a belief that alcohol will reduce this anxiety or arousal is associated with increased alcohol use, thus providing negative reinforcement of the drinking response (Booth & Hasking, 2009). Therefore reward drive may moderate the relationship between social anxiety and alcohol use.

A limitation of this study however, is that it only focussed on one component of impulsivity, reward drive and did not measure rash impulsivity. Given the well-established link between rash impulsivity and alcohol use (e.g., George, Connor, Gullo, & Young, 2010; Verdejo-Garcia et al., 2008), examination of this component is important. Further, the study used a non-clinical sample. Thus, the role of reward drive in this relationship may differ for those with more severe levels of social anxiety and alcohol problems.

More recent work by Nicholls et al. (2014) identified two separate social anxiety subgroups within a community sample via an online questionnaire. The first group consisted of individuals with characteristics typical of those widely used to describe individuals with social anxiety, such as inhibited and risk averse. The second group, however, was characterised by higher levels of trait rash impulsivity and reward drive, risk-taking and comorbid substance use. This "impulsive" or "approach-motivated" subgroup is implicated in the relationship between social anxiety and substance use (Nicholls et al., 2014). Their work extends previous research that also supports the incidence of a subgroup of individuals with social anxiety that is characterised by elevated levels of risk taking and frequent alcohol use (Kashdan, Elhai, & Breen, 2008; Kashdan & Hofmann, 2008; Kashdan, McKnight, Richey, & Hofmann, 2009; Tillfors, Van Zalk, & Kerr, 2013). However, these earlier studies treated impulsivity as a unidimensional construct, focussing on rash impulsivity only. Thus, Nicholls et al. argue that it is likely that variables such as impulsivity affect which type of individuals with social anxiety disorder are more sensitive to the influence of other properties of substances such as alcohol. This in turn may result in these individuals learning to drink to cope with their anxiety, and puts them at greater risk of developing a substance dependence disorder (Nicholls et al., 2014).

Understanding the Comorbid Social Anxiety-Alcohol Relationship

Given that the co-occurrence of social anxiety disorder and alcohol use disorders compromises treatment efficacy for either disorder, and is associated with increased symptom severity and poorer clinical outcomes, more work is needed to understand the nature of this relationship. Identifying the variables that may underlie and/or maintain these commonly co-occurring problems is essential to inform the development of preventative and treatment interventions that target the mechanisms involved to reduce the considerable burden associated with comorbid social anxiety and alcohol use disorders.

Whilst the current theoretical models provide some explanation of the relationship between social anxiety and alcohol use disorders, there is scope for an alternative approach. Examining impulsivity in this relationship may provide a better understanding of underlying mechanisms given its prominence in alcohol use disorders and the well-established link between social anxiety and alcohol use. There may be individual factors that affect whether a person with social anxiety also develops an alcohol use disorder, and impulsivity is a promising candidate. In order to investigate this question, this thesis will examine a contemporary model of impulsivity derived from factor analytic structure and theory (Dawe et al., 2004), that may moderate the relationship between co-occurring social anxiety and alcohol use disorders.

In summary, examining other variables such as impulsivity that may impact the complex interaction between social anxiety and alcohol use disorders is an important step to better understand the risk and maintenance factors involved in this relationship, and also for the development of more effective treatment interventions. In this context, this thesis addresses some fundamentally important research questions: Does impulsivity play a role in the relationship between social anxiety and alcohol use disorders? What impact do alcohol use and drinking behaviours have on social anxiety symptoms? Do individuals with social anxiety disorder, or at least a subgroup of them, have elevated levels of impulsivity? What impact does impulsivity have on social anxiety and alcohol treatment outcomes? Currently, very few published studies have examined these questions in relation to this specific and common profile of disorder comorbidity. Thus, the aim of this program of research was to evaluate empirically, the impact of impulsivity on the relationship between social anxiety and alcohol use disorders. Specifically, how reward drive and rash impulsivity may relate to who develops SAD-AUD comorbidity, how these problems are maintained, and how these individuals respond to treatment interventions.

Methodological Considerations

Design.

A large proportion of studies investigating the comorbid relationship between social anxiety and alcohol use have used epidemiological surveys and self-report questionnaires providing cross-sectional data. Whilst this contribution is significant and informative, such designs are restricted in that they may be unsuccessful in isolating or identifying potential causal mechanisms that underlie this relationship. Additionally, the use of self-report methodology is limited by the extent to which individuals can honestly and accurately report their behaviour and internal processes. Consequently, the use of laboratory-based, experimental methodology is essential to increasing our understanding of how these variables interact. For example, alcohol use disorders result from chronic exposure to a psychoactive substance, alcohol, within a complex individual and social context. Alcohol consumption has both positive, pleasurable properties which maintain alcohol use via positive reinforcement, and a capacity to reduce negative affect states which sustains alcohol use via negative reinforcement (Drummond, Tiffany, Glautier, & Remington, 1995). However, alcohol use via positive and negative reinforcement cannot explain all drinking behaviour.

Alcohol consumption effects take place in a context that will become increasingly conditioned (Drummond, Tiffany, Glautier, & Remington, 1995). Cue reactivity refers to the induction of a range of different responses to alcohol-related cues (Drummond et al., 1995). These cue-elicited responses can be psychological (e.g., subjective report of craving, changes in positive and negative affect), physiological (e.g., changes in skin conductance, heart rate) and behavioural (e.g., increased number of drinks consumed) (Carter & Tiffany, 1999; Drummond, 2000). An example of the impact of these cues is provided from studies demonstrating that alcohol-dependent individuals relapse more often in the presence of alcohol-related cues (Carter & Tiffany, 1999; Drummond, 2000). Thus, it is not only the direct pharmacological effects of alcohol on the individual, but also cues associated with

alcohol that affect drinking behaviour and relapse. Therefore, the role of cues in alcohol use is worth investigating as it may play a significant role in initiating and maintaining alcohol use (Drummond, 2000; Drummond et al., 1995).

Physiological reactivity to alcohol cues has been found to predict relapse risk in alcohol-dependent individuals following treatment (Garland, Franken, & Howard, 2012). Proponents of several models of addiction argue that at the neurobiological level, chronic exposure to a substance such as alcohol renders the neural system hypersensitive to the substance use incentives (e.g., Koob & Le Moal, 2001, 2008; Robinson & Berridge, 1993). The strong association between a stimuli and a reward in the brain has been termed "incentive salience" (Robinson & Berridge, 1993). It is argued that chronic exposure to alcohol conveys heightened incentive salience via mesocorticolimbic sensitisation to conditioned stimuli associated with alcohol (Robinson & Berridge, 2001). Exposure to alcohol and alcohol-related cues can result in a conditioned appetitive response that may lead to obsessive and compulsive alcohol-seeking behaviours (Robinson & Berridge, 2008). This subsequently serves to motivate alcohol use in alcohol-dependent individuals; even after lengthy periods of abstinence (Robinson & Berridge, 2008).

However, a lack of consistency between physiological responses to alcohol-related stimuli and the self-report measure of craving has been demonstrated previously, with low correlations between physiological and self-report measures of craving reported (Carter & Tiffany, 1999; Tiffany, 1990). In an effort to circumvent issues related to sole use of survey data, laboratory-based methodologies including the cue reactivity paradigm have been used in Chapters 3 and 4. Specifically, these chapters incorporate physiological indices of reactivity, which might prove a powerful tool in identifying and assessing reinforcing properties driving alcohol use. Heart rate reactivity to alcohol has been conceptualised as an physiological index of alcohol-induced reward (Ray, McGeary, Marshall, & Hutchison, 2006). In conjunction

with variables such as impulsivity, this allows for the examination of potential mechanisms underlying the relationship between social anxiety and alcohol.

The Samples.

Most studies examining the relationship between social anxiety and alcohol use disorders tend to use a base population of non-clinical participants generally from colleges and universities (see Battista, Stewart, & Ham, 2010 for a review) resulting in a paucity of studies in clinical samples of treatment-seeking individuals. Moreover, clinical trials of treatments for alcohol use disorders often exclude or neglect assessment of the presence of anxiety disorders, and randomised controlled trials focused on anxiety disorders typically exclude those with comorbid substance use disorders (McHugh, 2015). Thus, studies including individuals with comorbid social anxiety and alcohol use disorders are less common and are potentially limited by sample selection measures that may yield comorbid disorders of lower severity (McHugh, 2015). For instance, it is not uncommon for clinical trials involving alcohol use disorders to exclude concurrent antidepressant use, thus likely excluding many individuals with anxiety disorders (McHugh, 2015). In addition, many laboratory studies examining the impact of alcohol on social anxiety have included individuals with a broad range of social anxiety scores; ranging from individuals scoring below or at a predetermined cut-off score on trait social anxiety, to those meeting diagnostic criteria for social anxiety disorder (see Battista et al., 2010 for a review). It is likely that individuals with lower levels of social anxiety respond to alcohol differently than individuals with more severe levels. Indeed, research indicates that individuals with high levels of social anxiety react differently to alcohol compared to individuals with lower levels (e.g., Holroyd, 1978). Ideally, a full range of the phenomena under study is required to ensure that the effects observed are not due to a threshold.

Similarly, prior research involving alcohol administration has typically excluded individuals with alcohol use disorders due to concerns that consumption would adversely affect their condition or motivation for treatment. These concerns do not seem to be valid as a

comprehensive review concluded that there is no compelling evidence that participation in this type of research has adverse effects on individuals with alcohol use disorders (Dolinsky & Babor, 1997). Exclusion of individuals with more severe alcohol problems has subsequently resulted in the inclusion of "social drinkers": that is, individuals who consume as little as one drink per month in most alcohol administration studies (Battista et al., 2010). There is research showing that heavy and/or hazardous drinkers use and react to alcohol differently than light drinkers (Eddy, 1979; Pohorecky, 1991; Zarantonello, 1986). These exclusions result in an unrepresentative sample and therefore influence the quality of the research. Thus, the generalisability and relevance of the findings from such studies are limited, as participants do not represent the entire range of individuals in the population who suffer from social anxiety and alcohol use disorders and related symptoms.

The examination of impulsivity, social anxiety and alcohol use disorders in a sample of both clinical and non-clinical participants, both treatment-seeking and non-treatment seeking, across a variety of symptom levels will thus provide important insights into the relationships between these disorders, and is likely to be relevant to both epidemiological and clinical research.

The Samples Used in this Thesis.

The studies presented in this thesis recruited three samples with the aim of representing a wide variety of symptom levels and included more severe cases that have often been excluded in previous research.

The first study, (described in Chapter 2) was a secondary analysis from a clinical trial of self-reported alcohol use and drinking behaviours obtained from a treatment-seeking group of participants with social anxiety.

The second study sample (described in Chapter 3) had greater symptom variation in social anxiety and alcohol use, and provided responses for an experimental study that included four

groups of individuals that were diagnosed as having social anxiety disorder, an alcohol use disorder, both social anxiety and alcohol use disorders or no disorder at all.

The final sample was a clinical sample seeking treatment for symptoms of social anxiety and alcohol use disorders, which allowed for the examination of impulsivity as a mechanism underlying this comorbid presentation (described in Chapter 4), and the impact of impulsivity on treatment outcomes (described in Chapter 5).

Thesis Structure

This thesis is in the form of six chapters, four of which are self-contained empirical papers. This first chapter has provided an introduction to the main issues examined in the following chapters. Chapter 2 examines the rates of comorbid social anxiety and alcohol use disorders in a clinical sample diagnosed with social anxiety disorder seeking treatment for their social anxiety symptoms. This study is a secondary analysis from a clinical trial and also examines whether overall pre-treatment alcohol use negatively impacts social anxiety symptoms. More specifically, it includes an examination of alcohol use before (i.e., planned goal-directed behaviour akin to reward drive), during and after social situations on social anxiety outcomes. This chapter provides evidence for investigating whether components of impulsivity such as planned, goal-directed behaviours are protective in individuals with comorbid social anxiety and alcohol use disorders. Results from this chapter also provide rationale for the inclusion of alcohol expectancies in later chapters to investigate the role they may play in this comorbid relationship.

As a consequence of some interesting results emerging for the impact of drinking behaviour on social anxiety symptoms after treatment in Chapter 2, the study in Chapter 3 introduces a two-factor model of impulsivity (2-CARS; Gullo & Dawe, 2008) to investigate whether specific aspects of the impulsivity construct (e.g., reward drive and rash impulsivity) would differentiate between individuals with co-occurring social anxiety and alcohol use disorders from individuals with social anxiety alone. This study used an experimental

approach and also examined the effects of alcohol-related stimuli on physiological and psychological variables between the four groups of clinical and non-clinical individuals.

Chapter 4 extends from the previous chapter by exploring the pharmacologic and expectancy responses to alcohol cues and alcohol ingestion, and their association with both factors of Dawe et al.'s (2004) model of impulsivity. Additionally, this study measured and controlled for alcohol expectancies. This experimental study specifically used a treatment-seeking clinical sample of participants with comorbid social anxiety and alcohol use disorders and included both contextual cues and alcohol ingestion to examine impulsivity and cue reactivity as a potential underlying mechanism.

Chapter 5 examines the impact that impulsivity variables may have on treatment outcomes. Specifically, it examines the influence of reward drive and rash impulsivity on treatment outcomes for social anxiety and alcohol variables in treatment-seeking individuals with comorbid social anxiety and alcohol use disorders.

Chapter 6 presents a general discussion that critically examines these four studies' findings together in the context of the literature, discusses the limitations of the body of research as a whole, and explores the broad implications and possible avenues for future research.

All of the chapters have been edited to form a consistent body. The reference list for each paper has been combined to form a single integrated reference list and moved to the end of the thesis; each chapter is presented in APA style format; terms that refer to the same concepts or ideas have been made consistent between chapters; and Australian spelling has been used throughout. Chapters 2 through 5 are in the form of self-contained empirical papers that have been prepared for publication. Each chapter introduces new ideas with some overlap as they stand alone as manuscripts for publication.

In summary, the co-occurrence of social anxiety and alcohol use disorders is associated with marked distress, less effective treatment outcomes and decreased quality of life. This thesis extends the existent literature by enhancing our understanding of social anxiety and

alcohol use disorders through four empirical studies that employ diverse research methodologies to examine the hypothesised role of impulsivity in the SAD-AUD relationship. Implementing a range of clinical trial and laboratory methods, the current thesis explores whether trait and behavioural components of reward drive and rash impulsivity play a role in who develops SAD-AUD (Chapter 3), how these problems are maintained (Chapters 2, 3 and 4), and how individuals with this comorbidity respond to treatment interventions (Chapters 2 and 5). The findings in this body of work highlight a role for impulsivity in individuals with these commonly co-occurring problems.

Chapter Two

Does Alcohol Consumption at Pre-treatment Impact CBT Treatment for Social Anxiety Disorder?

Mirjana Subotic-Kerry, Andrew J. Baillie, Lexine A. Stapinski, Joanne Wagner, Maree J. Abbott, Jonathan E. Gaston, & Ronald M. Rapee

Abstract

The prevalence of comorbid social anxiety and alcohol use disorders in the community, and the complex interactions that occur between these two sets of disorders has emerged as a significant clinical, public health and research issue over the past decade. However, very few studies have examined the impact of alcohol use on outcomes for treatment targeting anxiety disorders. The purpose of this present study was twofold. In an analysis of data from an existing randomised control study on the treatment of social anxiety disorder (Rapee, Abbott, Baillie, & Gaston, 2007), we examined: 1) the rates of comorbid social anxiety disorder (SAD) and alcohol use disorder (AUD), and 2) the effect of alcohol use on outcomes targeting social anxiety disorders. Specifically, we examined the effect of pre-treatment alcohol consumption and alcohol use before, during and after social situations on a composite measure of social anxiety. This was explored in 172 adults presenting with generalised-type social anxiety disorder. Diagnostic severity ratings at one month post-treatment and three-month follow-up were examined, as were several self-report measures assessing severity of social anxiety. The present study found a low incidence of AUD in this sample of individuals with SAD. Results also indicated that pre-treatment alcohol consumption did not lead to poorer social anxiety treatment outcomes; however alcohol use before and during social situations was associated with more severe social anxiety symptoms. These findings suggest that pre-treatment alcohol consumption does not necessarily limit treatment gains, however, consuming alcohol prior to and during social situations may indicate the use of alcohol as a safety behaviour to cope with anxiety related to these interactions.

Does Alcohol Consumption at Pre-treatment Impact CBT Treatment for Social Anxiety Disorder?

Social phobia, or social anxiety disorder, frequently occurs in conjunction with alcohol use problems and disorders within clinical and epidemiological populations (Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997). Individuals with both clinical and subclinical social anxiety have a higher risk of abusing alcohol or having an alcohol use disorder compared to the general population (e.g., Buckner & Schmidt, 2009; Buckner et al., 2008; Crum & Pratt, 2001). Specifically, the risk of developing an alcohol use disorder doubles or triples among those with a social anxiety disorder when compared to those without this disorder (Kessler et al., 1997). Individuals with this comorbidity often present with a more complex and severe clinical profile, less able social functioning, and greater reliance on services (Randall et al., 2001; Schneier et al., 1989; Sullivan, Fiellin, & O'Connor, 2005). Comorbid anxiety disorders generally have a negative impact on alcohol treatment outcomes, with a greater relapse rate and heavier drinking in comorbid individuals compared to those without a comorbid anxiety disorder (e.g., Burns & Teesson, 2002; Wolitzky-Taylor et al., 2011). In contrast, less is known about how alcohol use problems affect anxiety disorder outcomes. Given the frequency of co-occurring social anxiety and alcohol use disorders, and the physical and psychological difficulties that individuals with these problems face, clarifying which factors may impact treatment outcomes will have considerable implications for implementing better and more lasting treatments, and may extend the current understanding of the different factors that impact the development and maintenance of this relationship.

Several comorbidity models have been proposed to explain why these two disorders might co-occur. One hypothesis is that there is a direct causal relationship between the two problems, suggesting that the presence of one disorder makes development of the other more likely (Kushner et al., 2000). Indeed, current evidence indicates that social anxiety typically

precedes the initiation of alcohol consumption and alcohol use disorders (Bakken, Landheim, & Vaglum, 2003; Bakken et al., 2005; Cox, Norton, Swinson, & Endler, 1990; Crum & Pratt, 2001; Merikangas, Stevens, et al., 1998; Schuckit et al., 1997), suggesting that the presence of social anxiety may lead to alcohol use problems. Here, social anxiety may act as a risk factor for problematic alcohol use as socially anxious individuals may use alcohol to "self-medicate"; that is, to control anxiety and/or negative affect related to their concerns about negative evaluation and how others perceive them (Carrigan & Randall, 2003). The self-medication hypothesis proposes that the pharmacological and/or psychological (or expectancy) effects of alcohol lead to a reduction in aversive mood and physiological states, thereby reinforcing the use of alcohol (Cappell & Greeley, 1987; Kushner et al., 2000). Consistent with this possibility, many individuals report self-medicating with alcohol in an effort to manage anxiety (e.g., Bolton, Cox, Clara, & Sareen, 2006; Crum et al., 2013; Lazareck et al., 2012). In addition, Thomas, Randall, and Carrigan (2003) found that individuals with co-occurring social anxiety and alcohol use disorders reported drinking in anticipation of a social event and during social situations in an attempt to alleviate anxiety and feel more comfortable. A large proportion of these individuals also reported that they would avoid events if alcohol was unavailable.

More recent models have been proposed to explain how alcohol use may impact social anxiety indirectly through its effects on cognitive processing. Prominent cognitive models of social anxiety (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997) propose that social anxiety disorder is maintained by defective processing of internal and external information regarding an individual's social performance, and overestimating the probability and costs of receiving negative evaluation from others. Safety behaviours are various strategies used by socially anxious individuals before or during anxiety-inducing situations in an effort to prevent feared outcomes, and have been identified as one of the key processes that maintain social anxiety (Rapee & Heimberg, 1997; Wells et al., 1995). Alcohol use may maintain

social anxiety by acting as an avoidant or "safety behaviour" in response to threatening stimuli, thus preventing normal desensitisation processes (Baillie & Sannibale, 2007; Tran & Haaga, 2002). For example, an individual with social anxiety who regularly drinks before social situations because they believe they cannot manage without it, may never have the opportunity to question this belief. In addition to disrupting proper evaluation of core beliefs related to anxiety, an individual may also attribute any positive experiences or social success to the alcohol rather than their own skills or personal characteristics. Further, the apparent need to drink may be viewed as additional evidence of incompetency, regardless of actual performance in the social situation. In this way, alcohol use may promote negative self-evaluations and maintain poor coping self-efficacy. Thus a vicious cycle ensues, whereby socially anxious individuals rely on alcohol, which acts to maintain unrealistic beliefs about their inability to function without it.

These explanatory models propose several reasons why alcohol may be consumed by individuals with social anxiety, and how alcohol may temporarily reduce anxiety and become negatively reinforcing. Consequently, alcohol use may interfere with treatment for social anxiety disorder. Specifically, if alcohol consumption is being used as a safety behaviour or as a coping mechanism to deal with anxiety, content addressing social interactions in treatment (such as exposure tasks) may lead to increasing alcohol consumption, and in turn, affect the consolidation of learning (Evert & Oscar-Berman, 1995). Further, drinking may interfere with treatment gains for social anxiety by dampening fear activation during exposure, thus preventing the full benefits of exposure (Foa & Kozak, 1986). This could subsequently lead to worsening alcohol abuse or dependence and fewer improvements on social anxiety outcomes. Moreover, even mild to moderate alcohol consumption has been found to adversely affect cognitive functioning, and cognitive deficits may either directly influence treatment outcome or impact other factors that contribute to treatment success such as retention in treatment programs and comprehension of information imparted during therapy (Bates, Bowden, &

Barry, 2002). Additionally, comorbid anxiety and alcohol use disorders also appear to be associated with greater severity and/or chronicity compared to the presence of either disorder alone (Chambless, Cherney, Caputo, & Rheinstein, 1987; Cox, Norton, Dorward, & Fergusson, 1989), typically leading to more chaotic living environments and higher lifetime service utilisation (Perkonigg et al., 2006), and this may also negatively impact treatment.

The impact that alcohol use has on treatment for social anxiety, however, is an area that has received minimal research attention. Only a few studies to date have examined the effects of a co-occurring alcohol use disorder on treatment for social anxiety, with mixed results. For example, a recent study found that individuals with mild to moderate alcohol problems in primary care can be effectively treated for anxiety disorders, with the majority of their results showing no predictive effects of alcohol use severity on treatment outcomes (Wolitzky-Taylor et al., 2015). Similarly, Bruce et al. (2005) reported that having a comorbid substance use disorder, including alcohol use disorders, did not significantly impact recovery or recurrence for social anxiety disorder compared to individuals without this comorbidity. In contrast, McEvoy & Shand (2008) showed that alcohol use prior to treatment predicted change in social anxiety, but only for anxiety related to interaction rather than performance. Apart from these three studies, research in this area predominantly looks at how alcohol consumption impacts levels of social anxiety (e.g., Abrams, Kushner, Medina, & Voight, 2001) and only includes individuals with non-clinical levels of social anxiety. The examination of rates of alcohol use disorders in individuals seeking treatment for anxiety disorders is also an area that has received very minimal attention primarily because alcohol use disorders are typically an exclusion criterion for entry into anxiety treatment programs (McHugh, 2015).

The purpose of the current paper is to address the current limited research investigating the effect of alcohol use on treatment. This paper examines the role of alcohol consumption on treatment for social anxiety in a clinical sample of treatment-seeking individuals with social anxiety disorder. The current study extends previous research by assessing the impact of

overall pre-treatment alcohol consumption on social anxiety symptoms following treatment, and specifically examines the impact of alcohol use prior to, during and after social situations on social anxiety severity. This study addresses three main questions regarding the relationship between social anxiety and alcohol use. Firstly, we examine the rates of AUDs in treatment-seeking adults with social anxiety disorder referred to a university anxiety disorders clinic. Secondly, we examine whether pre-treatment (baseline) levels of alcohol consumption influence the effectiveness of treatment for social anxiety in this sample. Finally, we examine whether a planned drinking behaviour such as consuming alcohol before, in addition to alcohol use during, and after social situations, influences the effectiveness of treatment for social anxiety in this sample. Consistent with cognitive models suggesting alcohol consumption is a safety behaviour employed to dampen anxiety, it was hypothesised that greater alcohol consumption pre-treatment would be associated with less improvement (i.e., fewer reductions in social anxiety symptom severity) following treatment for anxiety. In addition, it was hypothesised that drinking alcohol prior to and during social situations would result in fewer treatment gains as measured by the social anxiety symptom score, as alcohol use before and during exposure to social situations may disrupt the beneficial effects of exposure, dampen anxious arousal during social interactions, and prevent disconfirmation of threat cognitions.

Materials and Methods

Participants

Data was obtained from a clinical group of participants recruited to a randomised controlled trial for Cognitive Behaviour Therapy (CBT) treatment of social anxiety disorder at the Centre for Emotional Health at Macquarie University in Sydney, Australia. Study design, procedures and outcome results from this trial are detailed in Rapee et al. (2007). Participants were recruited via referral from general practitioners, mental health professionals, and through occasional media coverage and word of mouth. All individuals were screened via telephone

and those reporting social anxiety related issues were invited to attend a structured interview. Participants were included if they were over 18 years old and met diagnostic criteria for social anxiety disorder as their primary disorder as determined using the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; Di Nardo, Brown, & Barlow, 1994). The only planned exclusions were problems requiring immediate attention such as current suicidal intent, severe substance abuse or dependence, or active psychosis, assessed during the structured interviews. Diagnostic interviews were conducted by graduate students who had been trained by experienced clinical psychologists. A sample of recorded pre-treatment interviews were selected for reliability coding by a second rater. A primary diagnosis of social anxiety disorder was rated with high reliability ($\kappa = .86$; $p < .001$). Participants receiving concurrent pharmacotherapy were accepted provided that the dose had been consistent for three months and there were no plans to change.

All participants in the current study had received an ADIS-IV clinical severity rating for social anxiety disorder of at least 4 or above, where a higher score reflects increased severity. Within the final sample of 172 participants 91.3% met criteria for the generalised subtype of social anxiety disorder, and the mean clinical severity rating for social phobia was 6.23 ($SD =$ .98). Comorbidity with other Axis I disorders was high, as would be expected in a severely affected sample, with 45.3% meeting criteria for an additional anxiety disorder, 34.3% meeting criteria for an additional mood disorder, and 4.1% having an alcohol use disorder. The mean age of the sample was 35.33 years ($SD = 10.81$), age range 19 to 76 years, 52.3% participants were female, and 54.7% of the sample had never married. Just under half (45.9%) of the participants were university educated with a Bachelor's degree or higher.

Measures

Participants were assessed with the following measures at a pre-treatment interview. All questionnaires except the MAUQ were then completed at 12 weeks and 24 weeks from the time of the initial assessment interview.

Alcohol use.

Participants completed a questionnaire designed for this study to assess quantity and frequency of alcohol consumption and situations in which alcohol is consumed. The Macquarie Alcohol Use Questionnaire (MAUQ) consists of 28 items (see Appendix B). Participants indicated how much alcohol they consume before, during and after typical social situations (e.g., going to a party with strangers, eating at a restaurant), and three comparison non-social situations. For the current study, only the 25 items relating to social situations were considered. A three factor model (-2LL = -2668.69; AIC = 5541.38) of *before* (10 items), *during* (6 items) and *after* (9 items) was found to provide the most optimal fit to the data than a single factor model (-2LL = -2832.92; AIC = 5863.84) (χ^2 = 164.23, df = 3, p < .001). Internal consistencies were calculated for the three subscales, and all Cronbach's alphas indicated good internal consistency (*before* subscale: α = .89; *during* subscale: α = .84; *after* subscale: α = .86).

The MAUQ also includes two questions regarding how often alcohol is consumed in a 30 day period, and how many standard drinks are consumed on average in a typical drinking session. From these items, we calculated a composite measure of quantity-frequency as an indicator of overall alcohol consumption.

Social anxiety.

Participants completed several self-report measures which assessed symptoms and severity of social anxiety.

The Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) consists of 20 items which assess cognitive, affective and behavioural responses to a variety of situations requiring interaction with others (e.g., attending a party, initiating and maintaining conversations). Participants rate how representative the statements are of their reactions on a 5-point scale ranging from "Not at all like me" (0) to "Extremely characteristic or true of me" (4). The total score ranges from 0 to 80, where higher scores indicate greater anxiety in social interactions.

The Social Phobia Scale (SPS; Mattick & Clarke, 1998) is a companion scale to the SIAS and consists of 20 items that pertain to situations which involve being observed or scrutinised by others (e.g., public speaking, eating in front of others). Items are rated on the same 5-point scale described above and scores also range from 0 to 80, with higher scores representing greater anxiety about being observed by others. Both the SIAS and the SPS have been found to have excellent psychometric properties (Mattick & Clarke, 1998; Peters, 2000), with each showing good (SIAS: α = .82) and excellent (SPS: α = .91) internal consistency in the current study.

The Brief Fear of Negative Evaluation Scale (BFNE; Leary, 1983) assesses the cognitive aspects relevant to social anxiety, specifically fear and distress over negative evaluation. Respondents rate the degree to which each of the 12 statements applies to them on a 5-point Likert scale ranging from "Not characteristic of me at all" (1) to "Extremely characteristic of me" (5). Scores range from 12 to 60, with higher scores reflecting greater apprehension about the possibility of being negatively evaluated by others. The BFNE scale has good psychometric properties (Rodebaugh et al., 2004), and Cronbach's alpha indicated acceptable levels of internal consistency in this sample (α = .70).

The Albany Panic and Phobia Questionnaire – Social Phobia Subscale (APPQ-S; Rapee, Craske, & Barlow, 1994) has 27 items measuring introceptive (8 items), agoraphobic (9 items), and social situational fear (10 items). Only the items relating to social fears were used in the current study. Participants rate their degree of fear in a variety of situations on a Likert scale ranging from "No fear" (0) to "Extreme fear" (8). The APPQ has sound reliability and validity with a consistent factor structure (Brown, White, & Barlow, 2005). The AAPQ showed excellent internal consistency in this study (α = .91).

The Self Consciousness Scale – Social Anxiety Subscale (SCS-A; Fenigstein, Scheier, & Buss, 1975) was designed to measure both public and private self-consciousness and social anxiety. Participants indicate their self-consciousness and anxiety on a 5-point Likert scale

ranging from "Extremely uncharacteristic" (0) to "Extremely characteristic" (4). Only the social anxiety subscale (6 items) was used in the present study. The SCS has shown excellent psychometric properties in several translations and has been used extensively in the social anxiety literature (Rapee et al., 2007), with an acceptable Cronbach alpha of .77 in the current study.

Procedure

Following their diagnostic assessment, eligible participants were randomly allocated to one of four treatment conditions: standard therapist-led CBT group treatment, "pure" self-help through written CBT materials, self-help augmented with five group sessions with a therapist, and waiting list. The standard group treatment consisted of ten two-hour manualised group sessions (groups comprised approximately six individuals) with two graduate psychology student therapists. Participants in the "pure" self-help condition were instructed to read and work through the book *Overcoming Shyness and Social Phobia: A Step by Step Guide* (Rapee, 1998). Participants in the augmented self-help condition were instructed to read and work though the same book as those in the pure self-help group, and also attended five two-hour group sessions (approximately five to seven individuals) with a graduate psychology student therapist. Participants allocated to the wait-list condition received no treatment for 12 weeks and at the end of this period were offered the best available treatment. All participants took part in their allocated treatment program across 12 weeks and at the conclusion of their allocated treatment program, the questionnaire measures were collected a second time. Three months following the completion of their treatment program participants completed a diagnostic interview and the self-report symptom measures for a third time. The Macquarie University Ethics Committee approved the study procedures and all participants gave informed consent prior to participating in the treatment program.

Data Scoring and Analyses

The primary objective of the current study was to evaluate the impact of baseline alcohol consumption on symptoms of social anxiety as assessed by a composite of social anxiety symptom measures and baseline alcohol use before, during and after social situations on post treatment symptoms of social anxiety. As per previous research (Clark et al., 1994) to reduce the number of statistical tests performed, and therefore Type I error rate, several related social anxiety questionnaires were combined into a standardised composite symptom score. This score, reflecting total social anxiety symptom severity, consisted of the average of standardised scores on the clinician-rated severity of social anxiety disorder derived from the ADIS-IV, the SPS and SIAS, the BFNE and the social anxiety subscales of the AAPQ and SCS.

Prior to analyses, data for all dependent variables were screened for outliers and normality of distribution. The normality of the residuals was adequate for all dependent variables except for overall baseline alcohol consumption. The composite measure of quantity-frequency was used an indicator of overall alcohol consumption, and the resulting score was log transformed to reduce skewness.

Linear mixed models containing random intercept and slope parameters were used to evaluate change on social anxiety symptom composite scores from baseline to post-treatment and three-month follow-up following the procedure recommended by Gibbons et al. (1993). As fixed effects we entered time, baseline log alcohol consumption with an interaction term into the model. Subjects were entered as a random effect, time point was entered as a within-subjects factor and treatment type was covaried. Thus, change across time as a function of overall alcohol consumption was evaluated. A fixed-effects model that predicted social anxiety symptom severity using time, baseline alcohol consumption, and alcohol use before, during and after social situations as continuous predictors was also evaluated to determine the

impact of these variables in response to treatment. The model fit was compared using

likelihood-ratio tests. All analyses were conducted using SPSS version 20.0 for Windows.

Results

Rates of comorbid social anxiety and alcohol use disorder diagnoses and treatment

response.

In this sample, 4.1% (*n* = 7) met DSM-IV criteria for comorbid social anxiety disorder and

alcohol dependence. The mean clinical severity rating for alcohol use disorders was 5.29, *SD*

= 1.11. This represents fewer cases than expected given data reported by several recent

epidemiological surveys (e.g., Burns & Teesson, 2002; Grant et al., 2005; Hasin, Stinson,

Ogburn, & Grant, 2007; Teesson et al., 2010). However, 22% of the sample reported that they

drink alcohol daily, and 21.7% reported that they regularly binge drink; consuming five or

more drinks in a typical drinking session (see Table 1). This exceeds the Australian National

Health and Medical Research Council (2009) guideline levels for short-term harm.

Table 1

Baseline alcohol consumption of treatment-seeking individuals with social anxiety disorder

MAUQ	*n*	%
Frequency of use[a]		
Never	20	11.7
Once or twice a month	48	28.1
Once or twice a week	63	36.8
Once or twice a day	27	15.8
More than twice a day	13	7.6
Quantity of use[b]		
1 or fewer	37	22
2 to 4	92	54.8
5 or more	39	23.3

[a] *n* = 171; [b] *n* = 168

Figure 1 shows the social anxiety symptom score at the three time points: baseline, post-

treatment and three-month follow-up for the seven comorbid individuals and the mean social

anxiety symptom score over time for individuals with SAD and no AUD. At baseline, four of

the seven comorbid individuals have a social anxiety symptom score above the mean score for

individuals with SAD and no AUD; with three comorbid individuals scoring below the mean.

Thus, it appears that these seven comorbid individuals have only slightly more severe social anxiety at baseline than those without this comorbidity. Examination of the scores at post and three-month follow-up show that most of the comorbid cases score above the average trajectory of individuals without comorbid AUD, indicating that they do worse on average as treatment progresses.
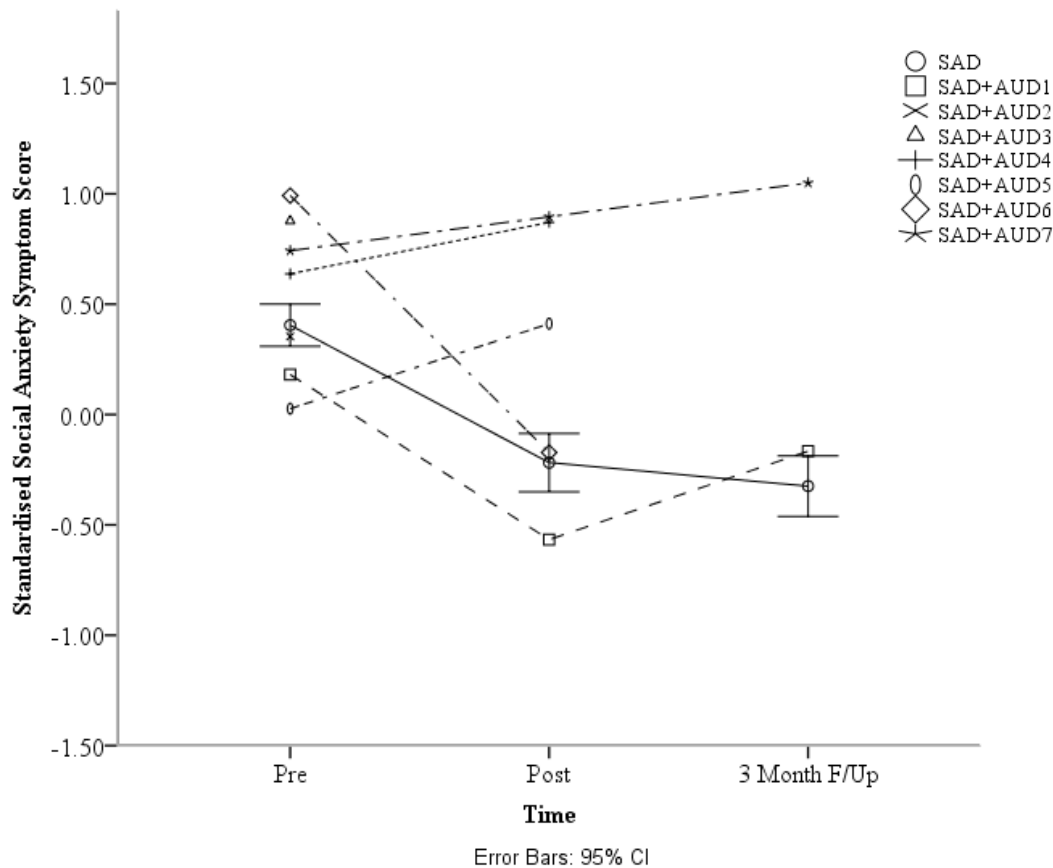
Figure 1. Composite social anxiety symptom scores over time for comorbid SAD-AUD individual cases (*n* = 7), and mean composite social anxiety symptom scores over time for individuals with SAD and no AUD (*n* = 172).

It was not possible to examine whether presence of a comorbid alcohol use disorder moderated treatment gains as size of the comorbid group was too small for linear mixed model analyses to be performed. However, a repeated measures ANOVA, with diagnosis (SAD-AUD vs. SAD) as a between-subjects factor, and time (pre, post, three-month follow-up) as a within-subjects factor showed weak evidence for poorer social anxiety treatment outcomes over time for the comorbid individuals, with the interaction between diagnosis and time approaching significance, $F(2,169) = 2.65$, $p = .073$. However, this effect was small, η_p^2

= .03, and the observed power was low at .52. Given the increased power required to detect a

hypothesised interaction versus a main effect, this marginal interaction was broken down in

simple effects analyses. There was no simple main effect between comorbid individuals with

SAD-AUD and individuals with SAD at pre-treatment ($p = .56$), and weak evidence at post-

treatment ($p = .07$). However, there was a significant diagnostic group effect at three-month

follow-up ($p = .02$). Inspection of the means in Table 2 shows that the comorbid SAD-AUD

individuals reported more severe social anxiety symptoms at three-month follow-up compared

to individuals with SAD and no AUD. A significant main effect of time was also observed,

$F(2,169) = 4.95$, $p = .008$. There was no simple main effect of time for the comorbid SAD-

AUD individuals (all p's > .52), but a significant simple main effect of time was observed in

the SAD and no AUD group (all p's < .01), suggesting that the treatment was effective in

reducing symptoms of social anxiety in individuals without comorbid SAD-AUD.

Table 2

Mean composite social anxiety symptom scores over time by diagnosis

Diagnosis	n	Pre-treatment M (SD)	Post-treatment M (SD)	Three-month follow-up M (SD)
SAD	165	.41 (.62)	.22 (.86)	-.32 (.89)
SAD-AUD	7	.54 (.36)	.38 (.57)	.46 (.50)

Relationship between pre-treatment alcohol consumption and change in social

anxiety symptoms.

Table 1 provides pre-treatment (baseline) frequency and quantity of alcohol use for

individuals with social anxiety disorder. There was a significant main effect of time on the

social anxiety composite score, $F(2,317.97) = 21.98$, $p < .0001$, indicating that treatment

reduced social anxiety symptoms in general across the sample. However, there was no

significant main effect for baseline alcohol consumption on the social anxiety composite

symptom score, $F(1,169.57) = 2.23$, $p = .14$, or interaction between time and baseline alcohol

consumption, $F(2,317.97) = .16$, $p = .85$, suggesting that baseline alcohol use did not impact

treatment gains as assessed by social anxiety symptom severity .

Relationship between pre-treatment alcohol consumption, alcohol use in social situations and change in social anxiety symptoms.

The mean number of drinks (quantity of alcohol use) before, during and after various social situations for the sample are shown in Table 3.

Table 3

Overall number of standard drinks consumed before, during and after twenty-five different social situations (N = 171)

	Before	During	After
No. of standard drinks			
3+	41	247	41
2	82	219	42
1	162	112	98
None	1425	448	1358

Note: Ten, six and nine social situations were described for before, during and after respectively.

Initially, models were estimated to assess the interaction between overall alcohol consumption, alcohol use before, during and after on change with treatment. There was no evidence of a significant interaction for any of these variables (all F values < 1.35, all p values > .26). The best fitting model containing random slope and treatment effects based on log likelihood included main effects of before, during and after and no interaction terms. Results from this final model indicated a significant main effect of time, $F(2,315.55) = 103.84$, $p < .0001$, indicating treatment reduced social anxiety symptoms in general across the sample. A significant main effect of alcohol consumption before social situations, $F(1,163) = 10.41$, $p = . 002$, and during social situations was also observed $F(1,163) = 4.26$, $p = .041$, indicating that the tendency to drink alcohol prior to and during social situations is related to more severe social anxiety symptoms across all time points. There was no significant effect of baseline alcohol consumption, $F(1,163) = .54$, $p = .46$, or alcohol consumption after social situations, $F(1,163) = .99$, $p = .32$.

Discussion

The first aim of this study was to identify the rates of alcohol use disorders (AUDs) in patients receiving treatment for social anxiety at a university anxiety disorders clinic. Although the initial sample size was quite large, the current study yielded a lower incidence of AUD than expected given recent research on social anxiety disorders. For instance, the 12-month prevalence of alcohol use disorders ranges between 13% and 17% in samples of individuals diagnosed with social anxiety disorder in comparison to a rate of 4% to 9% in the general population (Burns & Teesson, 2002; Grant et al., 2005; Hasin et al., 2007; Teesson et al., 2010). Specifically, in Australia, there was a higher prevalence of alcohol use disorders in a recent survey, the National Survey of Mental Health and Well Being (NSMH&W), with 16.7% of individuals with social anxiety disorder meeting criteria for an alcohol use disorder. In the United States, results from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) showed that among respondents with 12-month social anxiety disorder who sought treatment, 11.3% had comorbid alcohol abuse or dependence. Only 4% of the patients with social anxiety disorder in the current study met criteria for an alcohol use disorder which is less than half the proportion found in recent surveys. However, despite the low incidence of AUDs in the current sample, approximately one-fifth of the sample, reported engaging in risky drinking behaviours such as consuming alcohol daily or more than one drinking session daily, and heavy "binge" drinking (>5 drinks during a typical drinking session).

The lower rates of comorbid AUDs found in this study are likely due to the fact that it was a treatment-seeking sample. Treatment-seeking among individuals with comorbid alcohol and other substance-use disorders is low (Teesson & Gallagher, 1999), and individuals in the current study were seeking treatment for issues relating to their social anxiety. Previous research has reported that most patients with comorbid anxiety and alcohol use disorders are more likely to receive treatment for their alcohol problems in a substance abuse specialty

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clinic than for their anxiety in another setting (Havassy, Alvidrez, & Mericle, 2009). Further, individuals with problems requiring immediate attention, such as severe substance abuse or dependence, were excluded from the current study. Thus, some individuals with AUDs may have been excluded due to the presence of an additional substance use disorder as comorbidity between AUDs and substance use is common (Burns & Teesson, 2002). In addition, denial is a central component of alcohol use disorders (Brady, Tolliver, & Verduin, 2007), so individuals with this problem may have limited understanding about the negative impact their drinking has on their health and quality of life. There may also be a degree of shame surrounding an individual's use of alcohol which could also pose an obstacle to full disclosure about alcohol use and its impact. Similarly, many individuals may not disclose the full extent of their alcohol use and/or problems with alcohol due to concerns about being judged negatively for their use and/or due to concerns regarding whether they may have to address any issues with alcohol before being treated for social anxiety (Morris, Stewart, & Ham, 2005).

The second aim of the study was to examine the impact of overall pre-treatment alcohol consumption on social anxiety symptoms following treatment. Overall, participants showed improvement on the social anxiety symptom measures, and alcohol consumption did not significantly affect treatment gains. This finding suggests that individuals with social anxiety disorders benefitted substantially from social anxiety treatment regardless of level of alcohol consumption. These results are consistent with a recent study by Wolitzky-Taylor et al. (2015) that reported no predictive effects of alcohol use severity on outcome and Bruce et al.'s (2005) finding that a comorbid substance use disorder did not affect outcomes for social anxiety disorder. However, the impact of alcohol use on social anxiety outcomes may be different for individuals with more severe alcohol problems, with weak evidence suggesting that the treatment was less effective in reducing symptoms of social anxiety in individuals with comorbid SAD-AUD.

Finally, the impact of alcohol use was explored further by examining specific drinking behaviours such as alcohol consumption before, during and after social situations on treatment outcomes as measured by the social anxiety composite. Interestingly, higher alcohol use before and during social situations was associated with significantly more severe social anxiety symptoms at all time points. This suggests that the function of alcohol use may be more important than overall level of alcohol use; that is, overall alcohol consumption may be less important than the use of alcohol as a safety behaviour (e.g., prior to anxiety-inducing social situations). However, although consumption of alcohol before social situations was associated with more severe social anxiety symptoms, there was no impact on treatment gains, indicating that these individuals still benefitted from treatment.

The greater level of severity of social anxiety symptoms for those who drank alcohol prior to and during social situations may be due to individuals with more severe social anxiety symptoms being more likely to use alcohol to self-medicate; that is, to cope with social situations. For example, several studies with adults indicate that higher levels of social anxiety are associated with the deliberate consumption of alcohol as a strategy to minimise anxiety and depressed mood (McIntosh & Ritson, 2001; Schneier et al., 1989; Thomas et al., 2003). Planned consumption of alcohol use to cope with aversive emotional states may prevent successful coping strategies which may lead to further adverse consequences. If social situations and anxious feelings repeatedly precede alcohol use and the rewarding effects of anxiety reduction, these situations and outcomes may become conditioned stimuli to signal the drinking response before encountering a social situation or when feelings of anxiety arise (Morris, Stewart, & Ham, 2005). While alcohol may have short-term anxiolytic effects, prolonged exposure to alcohol may lead to withdrawal, increased anxiety symptoms and other emotional states (Kushner et al., 2000). Subsequently, individuals may use greater quantities or consume alcohol more frequently in situations that cause anxiety. Drinking before and during social situations can also perpetuate anxiety by inhibiting disconfirmation of threat-

related beliefs regarding perceived or actual danger present in various social situations (Clark & Wells, 1995; Rapee & Heimberg, 1997). Here, alcohol use may interfere with the beneficial effects of successful social interactions in several ways.

Firstly, consuming alcohol prior to and/or during social situations may prevent socially anxious individuals from processing perceived evaluations from other individuals when present in the situation due to the interfering effects of alcohol on memory. Indeed, studies of state-dependent or context-specific learning indicate that what an individual learns while intoxicated may be retained less well in a drug-free state (e.g., Morissette, Spiegel, & Barlow, 2008; Weingartner, Adefris, Eich, & Murphy, 1976). Thus, high doses of alcohol can disrupt the mechanisms involved in memory and what an individual learns compared to the alcohol-free state. Disruption of these cognitive processes may lead to an initial reduction in anxiety which then acts as a mechanism by which alcohol use becomes reinforcing. In the longer term, anxiety levels may actually be maintained because any beneficial feedback related to the social interaction where alcohol was consumed (either prior to or during) may not generalise to drug-free contexts, thereby maintaining social anxiety.

Secondly, consuming alcohol prior to and/or during social situations may also interfere with the beneficial effects of successful positive interpersonal interactions because those with social anxiety may be more likely to attribute the absence of a feared outcome to the use of alcohol, and as a consequence, expect the feared outcome when alcohol is not being used or is unavailable. For instance, if a socially anxious individual uses alcohol prior to or during a feared social situation, they may be unsuccessful in adapting underlying maladaptive beliefs they have about their social performance; attributing their ability to cope with the social situation to alcohol. Thus, alcohol intake may function to reinforce underlying beliefs that maintain SAD, despite allowing the individual to participate in events that might otherwise be avoided. It has been suggested that individuals who credit their improvement to themselves maintain those improvements better than individuals who attribute treatment gains to external

sources such as effects of drugs or clinician's ability (Brewin & Antaki, 1982). For example, individuals with panic disorder and agoraphobia who attributed improvements to the prescribed medication had more severe fear and avoidance of social situations and relapsed more often at follow-ups compared to individuals without this external attribution (Basoglu, Marks, Kilic, Brewin, & Swinson, 1994). In the current study, the association between drinking prior to and during social situations and more severe anxiety symptoms may have been due to a reduction in individual self-effort attributions that could have led to feelings of inadequacy, reductions in self-esteem and additional negative self-appraisals. Furthermore, elevated symptoms of social anxiety may also be maintained if an individual's performance during a social interaction is negatively affected or poorly received because of alcohol, thus reinforcing an individual's negative self-representation.

Limitations and Future Directions

There are several limitations to our findings. Firstly, the low number of cases of comobid individuals with social anxiety disorder and alcohol use disorder precluded analysing the treatment outcomes between these two groups using linear mixed models. The analysis that was conducted was severely underpowered and thus, these results should be interpreted with caution. Future research should therefore examine whether comorbid AUDs moderate or mediate the effects of treatment for social anxiety. Secondly, specific information about daily patterns of alcohol use was not obtained. Instead, participants were only required to provide self-report information of alcohol use. Although quantity-frequency assessments of alcohol use can provide reliable information about number of alcohol drinking days and overall average consumption, they generally do not take into account infrequent episodes of heavy and light drinking (Sobell, Sobell, Klajner, Pavan, & Basian, 1986). Thus, alcohol use is frequently underestimated. Furthermore, heavier consumption days, which are related to alcohol-related problems, are usually not reported in quantity and frequency methods (Sobell et al., 1986). More sophisticated methods of evaluating alcohol use, for example, exact

number and type of beverage, may be useful in future research. Nevertheless, this study is one of a few to examine the rates of alcohol use disorders and the impact of alcohol use in a treatment-seeking sample of individuals with social anxiety disorder, and a strength is that specific contexts of alcohol use were evaluated, and a combination of measures assessing symptoms and severity of social anxiety were used.

Conclusion

The present study is one of very few to have examined the impact of alcohol use on treatment outcomes for social anxiety disorder. Firstly, we found that the prevalence of AUDs was not common in the current treatment-seeking sample of individuals with SAD; however, there was weak evidence to suggest that treatment was less effective in reducing symptoms of social anxiety in those few individuals with comorbid social anxiety and alcohol use disorders. Further research with a larger sample size is needed to confirm this. Secondly, individuals improved on a composite measure of social anxiety symptoms following treatment, and pre-treatment alcohol use did not significantly affect treatment gains. However, findings from the current study suggest that a planned drinking behaviour, specifically, drinking alcohol prior to social situations and drinking during social situations are associated with more severe social anxiety symptoms. Consuming alcohol prior to and during social situations may worsen anxiety by directly causing anxious symptoms, negatively impacting social performance, maintaining threat appraisals and/or dampening anxious arousal during exposure, thus disrupting the benefits of exposure. This finding has important implications regarding the treatment and prevention of these highly comorbid disorders as early treatment focussed on attenuating drinking could serve to prevent the development of alcohol abuse or dependence among high-risk individuals.

Chapter 3

Social Anxiety and Alcohol: The Role of Reward Drive, Rash Impulsivity and Cue Reactivity

Mirjana Subotic-Kerry, Andrew J. Baillie, Lexine A. Stapinski & Kristen J. Tulloch

Abstract

Background: The comorbidity between social anxiety disorder (SAD) and alcohol use disorders (AUD) is prevalent and linked to negative health consequences. The underlying mechanisms of this relationship are poorly understood and have therefore been identified as an important area of research. The present study investigated the role of impulsivity, specifically reward drive and rash impulsivity, in this relationship in an effort to better understand this co-occurrence.

Method: Participants included alcohol-dependent drinkers ($n = 26$), alcohol-dependent drinkers with social anxiety disorder ($n = 11$), non-dependent drinkers with social anxiety disorder ($n = 18$) and non-dependent drinkers ($n = 33$). Trait measures of reward drive and rash impulsivity and a behavioural measure of rash impulsivity, the Iowa Gambling task (IGT) were administered. A cue reactivity paradigm was employed to examine the impact of alcohol and water cues on self-reported craving, mood and physiological responding (heart rate, skin conductance).

Results: Alcohol-dependent drinkers with and without comorbid SAD scored higher on trait measures of impulsivity and showed greater increases in self-reported craving, skin conductance and heart rate when exposed to alcohol cues than non-dependent drinkers with and without comorbid SAD. Individuals with comorbid SAD+AUD reported significant increases in negative affect when exposed to the alcohol cue. Alcohol-dependent drinkers without comorbid social anxiety performed worse on the IGT compared to those with comorbid SAD+AUD.

Conclusions: Dependent drinkers are characterised by trait and behavioural measures of impulsivity which may perpetuate alcohol consumption. Socially anxious individuals high on impulsivity may be susceptible to development of alcohol dependence via a mechanism involving negative affect regulation.

Social Anxiety and Alcohol: The Role of Reward Drive, Rash Impulsivity and Cue Reactivity

Co-occurrence of social anxiety disorder (SAD), and alcohol use disorders (AUDs) is common in both clinical and community samples (Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997; Merikangas, Stevens, et al., 1998), yet the mechanisms underlying this relationship are not fully understood. Explanations for the SAD-AUD link suggest that alcohol is used as a coping mechanism to self-medicate or dampen social anxiety symptoms (Khantizian, 1985; Sher & Levenson, 1982), as social anxiety typically precedes alcohol problems, and tends to be primary (Bakken et al., 2003, 2005; Cox et al., 1990; Merikangas, Stevens, et al., 1998; Schuckit et al., 1997). While it is well established that individuals with social anxiety disorder have greater risk for an alcohol use disorder than individuals without a diagnosis of social anxiety disorder (Burns & Teesson, 2002; Grant et al., 2004; Himle & Hill, 1991; Kessler et al., 1997), the association between social anxiety symptoms and alcohol consumption is not straightforward. Results have varied from study to study, with social anxiety related to increased and decreased alcohol use, and in some instances, no relationship has been found (see Morris et al., 2005 for a review). These mixed findings suggest that the presence of social anxiety is not uniformly associated with problematic alcohol use. Rather, it seems that some socially anxious individuals are at greater risk for problematic drinking while others may be protected from it, suggesting that other factors may moderate this relationship. Given that the co-occurrence of SAD and AUD increases impairment, distress and complicates treatment, investigating processes involved in this relationship is of great importance.

A two-factor impulsivity-related model (2-CARS; Dawe et al., 2004) derived from biologically-based processes has been applied to understanding the vulnerability to addiction, including alcohol. Central to this framework are two separate but interconnected systems; observable at the neurophysiological, trait and behavioural levels. The first, reward drive,

relates to an individual's sensitivity to rewarding stimuli in the environment which motivates drug use, and the second, rash impulsivity, is associated with impaired decision-making, and a reduced capacity to control one's behaviour (Dawe et al., 2004). For example, it is argued that heightened sensitivity to the rewarding properties of alcohol, and/or reduced inhibitory control may lead to problematic alcohol use, dependence and relapse. Individual differences in these impulsivity factors may help to explain why some individuals with SAD are at increased risk for alcohol use disorders, while others are not.

Evidence that reward drive and rash impulsivity play a role in the development and continuation of alcohol use comes from multiple sources. For instance, elevated trait impulsivity has been found in heavier drinkers compared to light drinkers (Kambouropoulos & Staiger, 2001, 2004a). Alcohol use is also associated with rash impulsivity indicated by impaired decision-making, as measured by the Iowa Gambling Task (IGT) which assesses capacity to prioritise longer-term outcomes over immediate rewards (Dom et al., 2006; Fein, Klein, & Finn, 2004). Here, impaired decision-making has been found to predict relapse in alcohol-dependent patients (Bowden-Jones, McPhillips, Rogers, Hutton, & Joyce, 2005). Positive relationships have also been found between reward drive and levels of hazardous drinking in adults, high school and university students (Franken & Muris, 2006; Knyazev, 2004; Loxton & Dawe, 2001; O'Connor & Colder, 2005). In addition, several laboratory studies show that increased reward drive is associated with a bias to detect and react to drug cues (Franken, 2002; Kambouropoulos & Staiger, 2001, 2004a), suggesting that reward drive, at the behavioural level, is driven by automatic, cue-elicited processes that may arise after repeated exposure to drug-related cues. Studies using cue reactivity paradigms indicate that individuals with AUDs show increased craving and physiological reactivity (increased salivation, changes in heart rate) to alcohol-related stimuli compared to individuals without this disorder (Coffey, Saladin, Libet, Drobes, & Dansky, 1999; McCusker & Brown, 1991; Rubonis et al., 1994). Thus, cue reactivity can be an important indicator of alcohol problems

in that such reactions are stronger in individuals with alcohol problems than those without. Heightened reward drive and reactivity to alcohol-related cues is also associated with intense psychological and physical cravings for alcohol (Franken, 2002; Kambouropoulos & Staiger, 2001). In combination, this profile of elevated reactivity and craving for alcohol places individuals at greater risk of chronic relapsing alcohol use problems.

While there is strong support for these factors in the maintenance of alcohol use problems, little research has examined how rash impulsivity and reward drive influence the SAD-AUD connection. Studies finding no SAD-AUD relationship may have selected for low levels of impulsivity, or perhaps the relationship between alcohol, impulsivity and social anxiety changes over adolescence and early adulthood. Colder, Chassin, Lee, and Villalta (2010) and Hussong, Jones, Stein, Baucom, and Boeding (2011) have proposed an internalising pathway into substance use that may occur during adolescence that is thought to develop due to self-medication with alcohol to cope with negative affect. It has also been suggested that individuals with anxiety disorders are more susceptible and condition more readily to alcohol cues due to overlapping processes with the brain's reward and stress/anti-reward systems (Kushner, Maurer, Menary, & Thuras, 2011). Kushner et al. (2011) found that compared to individuals without an anxiety disorder, individuals with anxiety disorders progressed or "telescoped" faster from regular alcohol use to alcohol dependence. They concluded that because the stress/anti-reward system that relieves negative states is up-regulated in anxious individuals, less exposure may be required for the development of alcohol dependence.

Reward drive has also been found to contribute to the relationship between social anxiety and alcohol use in a large sample of adults (Booth & Hasking, 2009). Specifically, adults with high trait reward sensitivity showed a positive relationship between social anxiety and drinking when they had strong tension reduction expectancies. More recently, Nicholls, Staiger, Williams, Richardson, and Kambouropoulos (2014) identified two separate social anxiety subgroups; specifically, an "impulsive" subgroup characterised by high levels of trait

reward drive, rash impulsivity and substance use. However, as these studies employed only self-report questionnaires, it is not known whether exposure to rewarding environments and/or alcohol stimuli in individuals with SAD-AUD translates into increased craving and cue reactivity. Understanding alcohol craving, cue reactivity and rash impulsivity in individuals with these problems may help explain why some individuals with social anxiety go on to develop problems with alcohol, while others do not. That is, it may be that socially anxious individuals with high reward drive and rash impulsivity are susceptible to AUDs, but those with social anxiety alone are not.

In an effort to better understand the SAD-AUD co-occurrence, we evaluated two impulsivity factors, reward drive (RD) and rash impulsivity (RI) that may characterise alcohol use within a student and community sample. The present study employed trait self-report measures of RD and RI, a behavioural measure of RI: the Iowa Gambling Task (IGT), and a behavioural measure of RD: craving. We used a cue reactivity experimental design to assess whether these measures would differentiate firstly; between alcohol-dependent and non-dependent drinkers and secondly, between socially anxious individuals with, versus without, comorbid alcohol dependence, and thirdly, between alcohol-dependent drinkers, with and without, comorbid SAD. Based on the extant literature we hypothesised that (a) dependent drinkers would exhibit significantly higher levels of trait RD and RI when compared to non-dependent drinkers; (b) dependent drinkers would perform worse on the behavioural task measuring risky decision-making (RI) when compared to the non-dependent drinkers; and (c) dependent drinkers would report a significant increase in RD-related appetitive responses (e.g. self-reported craving and positive affect) and autonomic arousal (heart rate and skin conductance) from the water to alcohol cue when compared to the non-dependent drinkers. Additionally, it was hypothesised that (d) comorbid SAD+AUD individuals would be higher in trait and behavioural measures of RD and RI, with increases in craving and autonomic arousal from the water to alcohol cue when compared to the non-dependent drinkers with

SAD. Finally, based on Kushner et al. (2011) "telescoping" theory that individuals with anxiety disorders may have a lower threshold for developing alcohol dependence and progress from regular drinking to alcohol dependence faster than individuals without anxiety disorders, it was hypothesised that (e) comorbid SAD+AUD individuals would report greater increases in craving and autonomic arousal to the alcohol cue compared to the dependent drinkers without SAD.

Method

Participants

A sample of adults meeting diagnostic criteria for social anxiety disorder and/or alcohol use disorder, and adults with no diagnosis of either ($N = 78$, female = 51.3%, age range 18 to 55, $M = 22.85$, $SD = 6.23$) were recruited from educational institutions and the community via posters and online media advertising requesting individuals who drank at least monthly or more. Following a telephone screening, individuals who were potentially eligible for the study were scheduled for an in-person interview, at which time informed consent was obtained. Of the 200 individuals screened, 81 were invited for the interview. Individuals were not eligible if they were younger than 18 years, screening positive for psychosis, alcohol withdrawal, suicidal ideation or any disorders other than SAD, AUD and/or specific phobias or reported substance use issues for drugs other than alcohol, tobacco or caffeine. All participants reported a beverage preference for either beer or wine. A clinical interview was conducted using the Anxiety Disorders Interview Schedule (ADIS; Brown, DiNardo, & Barlow, 1994) to determine diagnoses and rule out exclusionary Axis I disorders using DSM-IV-TR criteria (American Psychiatric Association, 2000). Diagnostic interviews were conducted by graduate students who had been trained by experienced clinical psychologists. The Timeline Follow-back (Sobell & Sobell, 1992) and Alcohol Use Disorders Test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) were also included to assess quantity and frequency of

drinking in the recent past. Three participants met criteria for an Axis I diagnosis other than SAD or AUD and were excluded from the study.

Participants were characterised into four groups according to diagnostic criteria. Criteria for inclusion in the alcohol-dependent group was current diagnosis of clinical or subclinical alcohol abuse or dependence via the ADIS-IV. Twenty-six participants (96%) met DSM-IV criteria with one participant reporting sub-threshold symptoms. The mean clinical severity rating for alcohol abuse or dependence was 4.59 (SD = 1.01), on an 8-point scale where a higher score indicates greater severity. Non-dependent drinkers had no current or past alcohol abuse or dependence diagnosis, no prior treatment for an alcohol-use disorder, but reported consuming alcohol at least monthly or more. Participants were classified as having social anxiety if they met some or all criteria for SAD as a current disorder. For those with social anxiety, the mean clinical severity rating for the disorder was 4.07 (SD = 1.77), with 18 participants (62%) meeting all criteria and 11 individuals reporting sub-threshold symptoms of social anxiety. The final sample included 16 dependent drinkers (20.5%), 11 dependent drinkers with comorbid social anxiety (14.1%), 33 non-dependent drinkers (42.3%) and 18 non-dependent drinkers with social anxiety (23.1%). University students and the community sample did not significantly differ in gender, diagnosis of social anxiety or alcohol use disorders, $\chi^2(1, N = 78) = .44$, $p = .51$, $\chi^2(1, N = 78) = .09$, $p = .76$, and $\chi^2(1, N = 78) = 2.81$, $p = .09$ respectively, and were therefore combined for the analysis.

Measures

Trait measure of reward drive.

The Behavioural Inhibition/Behavioural Activation Scale (BIS/BAS; Carver & White, 1994) contains 20-items, and yields scores on three BAS related subscales (*drive, reward responsiveness, fun-seeking*) and one BIS subscale. In order to reduce the number of variables, only the BAS subscales (*drive*; four items and *reward responsiveness*; five items) were selected to represent reward sensitivity used in the analyses. Further, the BAS fun-

seeking subscale does not seem to represent this construct in a straightforward manner and results from previous research have led to the suggestion of excluding it (Boog, Goudriaan, van de Wetering, Deuss, & Franken, 2013). The BIS/BAS scales have good internal consistency (Campbell-Sills, Liverant, & Brown, 2004). Similarly, the BAS scales correlate well with alternative measures of such constructs, in particular extraversion, novelty-seeking and reward dependence (e.g., Jorm et al., 1998). In this study, the BIS/BAS scales displayed acceptable internal consistency with Cronbach's alphas, ranging from .71 (Reward responsiveness) to .73 (Drive) in this study.

Behavioural measures of reward drive.

Craving. The Alcohol Urge Questionnaire (AUQ; Bohn, Krahn, & Staehler, 1995) is an eight-item measure providing an index of acute craving. The AUQ has excellent internal consistency (Cronbach's α = .91; Bohn et al., 1995), and this was replicated in the current sample: α = .91 (baseline), α = .93 (water cue), and α = .92 (alcohol cue).

Mood. Participants completed the Positive Affect Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) to assess state mood. The PANAS consists of two 10-item mood scales, one measuring positive affect (PA), the other measuring negative affect (NA). Participants were asked to indicate "To what extent you feel this way right now, that is, at the present moment" from "Very slightly, or not at all" (1) to "Extremely" (5) for items corresponding to positive and negative feelings and emotions, such as "Excited" and "Guilty". Internal consistencies for the PANAS are very high, with reported alphas of .93 for positive affect and .89 for negative affect (Gomez, Cooper, & Gomez, 2000). Internal consistencies for the PANAS were good to excellent, with alphas ranging from .83 for baseline positive affect to .94 for negative affect (alcohol cue) in the current study.

Physiological measures. Heart rate and skin conductance were recorded as a psychophysiological index of alcohol-induced reward as heart rate has been associated with appetitive motivational states (e.g., Ray, McGeary, Marshal, & Hutchison, 2006). Participants

were fitted with electrocardiogram (ECG) for heart rate and skin conductance level (SCL, sometimes termed galvanic skin response: GSR) recording equipment, and responses were monitored using LabChart 7 software linked to a PowerLab Data Acquisition System (ML116) and amplifiers. The signals were sampled at 1000 Hz in a sound-proofed room with a constant temperature. Skin conductance was recorded using electrodes in a bipolar placement on the index and middle fingers of the non-dominant hand, and mean skin conductance level (SCL) (in microsiemens) at each time point was calculated using LabChart 7 software. The ECG was recorded using disposable Ag/AgCL electrodes placed in Lead I position on the limbs, and mean heart rate in beats per minute (BPM) was calculated at each time point.

Trait measure of rash impulsivity.

Rash impulsiveness was measured with the Impulsiveness subscale of Eysenck's 54-item Impulsiveness (I_7-IMP), Venturesomeness, and Empathy Scale (I_7; Eysenck, Pearson, Easting, & Allsopp, 1985). The I_7 comprises of 19 items such as "Do you usually make up your mind quickly?" and respondents are required to answer with either a "Yes" or "No" response. Reliabilities for the I_7-IMP are good with studies reporting Cronbach alphas ranging from .83 to .84 (Eysenck et al., 1985), and this was replicated in the current sample, $\alpha = .83$ (I_7-IMP subscale).

Behavioural measures of rash impulsivity.

A computerised version of the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994) using Inquisit (Version 3) was used to assess an individual's decision-making skills. Participants are required to choose a card from one of four decks (A, B, C, and D) during 100 successive trials. Each deck of cards is associated with differing levels of risk and reward. Two decks (A and B) are weighted to give out larger rewards, but are also associated with much larger costs. Decks labelled C and D are advantageous with moderate and more consistent gains and smaller losses. Participants were instructed to try to gain as

much money as possible by the end of the game. We used the mean IGT net-score (advantageous decks-disadvantageous decks) as a dependent variable. A higher score means that a participant is more often choosing advantageous decks. This task has good ecological validity with strong associations found between deficits in behavioural measures of executive function and real-world behavioural problems (Li, Lu, D'Argembeau, Ng, & Bechara, 2010).

Materials

Neutral and alcohol vignettes.

Two short descriptive audio scripts (vignettes) were recorded detailing a neutral and alcohol-related scene. These vignettes were employed for two reasons. Firstly, research suggests that sensory imagery is a powerful tool for inducing craving because it is a key component of the cognitive system that underpins human motivation (Litt & Cooney, 1999). Secondly, these stimuli have good ecological validity, with both vignettes constructed to cover a sequence of common events, such as waiting for a friend at the beach (water) and at a bistro (alcohol). The neutral stimulus has also been employed in previous research on tobacco craving (e.g., Taylor, Harris, Singleton, Moolchan, & Heishman, 2000). The vignettes were constructed to be as similar in structure, content and language as possible.

Procedure

Following their diagnostic assessment, eligible participants attended a laboratory session at the university. Participants were instructed to abstain from alcohol 24 hours prior to the laboratory session, to eat a light meal approximately three hours before, and to avoid caffeinated beverages from then until the experiment to minimise factors contributing to inter-individual psychophysiological response. All appointments were conducted in the afternoon (2.30pm onwards), to mimic as much as possible typical drinking conditions (Staiger, Greeley, & Wallace, 1999).

To create a naturalistic setting, the experiment was conducted in a simulated bar, designed to maximize contextual cues associated with a drinking environment (e.g. alcohol serving bar,

cocktail glasses, bar stools, armchairs, dim lighting). On arrival, electrodes for physiological measurement were fitted, and participants were instructed to complete a battery of questionnaires (AUQ, BIS/BAS, I_7, PANAS), and a computer task (IGT). Participants sat quietly and watched a neutral DVD while baseline physiological measurements were recorded over a five-minute period. Individuals then participated in a cue reactivity procedure following guidelines provided by Monti et al. (1987). Each cue consisted of a pre-recorded vignette and in vivo handling (sight, smell) of beverages for a three-minute duration. A five-minute washout DVD consisting of neutral scenes was administered between each cue presentation. The alcohol cue consisted of the participants preferred type of alcoholic beverage (either 250mL of full-strength beer or 100mL of wine; both containing 10g. alcohol) presented in the original packaging and poured into appropriate glassware. The water cue was a commercially labelled bottle of spring water. Self-reported urge to drink, mood and physiological measurement were taken after each cue presentation. The alcohol stimulus was presented after the water cue to all participants to avoid carryover effects (Rohsenow & Niaura, 1999). Participants were each paid $AUD40 for their participation.

Data Scoring and Analyses

Descriptive statistics obtained using SPSS version 21.0 software were used to characterise the sample and check for univariate and multivariate non-normality. Principal analyses using multivariate analysis of variance (MANOVA) compared the effects of drinker severity (dependent vs. non-dependent), and social anxiety diagnosis (social anxiety vs. no social anxiety) on four measures of impulsivity: BAS-Drive, BAS-Reward, I_7-IMP and net score on the IGT. A $2 \times 2 \times 2$ mixed multivariate analysis of covariance (MANCOVA) was used with drinker severity (dependent vs. non-dependent) and social anxiety diagnosis (social anxiety vs. no social anxiety) as between-subjects factors and cue condition (water vs. alcohol) as the within-subjects factor to evaluate differences in self-reported craving ratings, positive and negative affect and physiological measures. Previous studies suggest a disconnect between

self-report and physiological measures of cue reactivity (Drobes & Thomas, 1999; Rohsenow et al., 1992), thus self-report measures of craving and affect were examined separately to the physiological measurements (skin conductance and heart rate). Baseline scores were included as covariates to take into account individual differences in baseline craving ratings, positive and negative affect, and physiological responding.

Results

In this sample, 10.3% of participants reported that consumed alcohol monthly or less. A total of 70 (89.7%) reported drinking at least twice or more within the past month. These values mirror drinking patterns in a similar sample of Australian students (Mean age = 23.5 years; $SD = 7.6$), where a total of 1701 (87.6%) respondents stated that they currently drink alcohol (Utpala-Kumar & Deane, 2010). This study also reported comparable quantity and frequency of alcohol use to our results presented in Table 4.

Differences between diagnostic groups on demographic and alcohol-related measures.

Table 4 provides a description of the demographic characteristics and alcohol-related variables for the four diagnostic groups. The groups were similar with regard to gender and age. A MANOVA was performed to examine differences between alcohol drinker severity (dependent vs. non-dependent) and social anxiety diagnosis (social anxiety vs. no social anxiety) on several alcohol-related variables (see Table 4 for multivariate results).

Table 4

Demographic and alcohol-related variables for dependent and non-dependent drinkers with, and without, social anxiety

	AUD (*n* = 16)	SAD+AUD (*n* = 11)	ND (*n* = 33)	ND+SAD (*n* = 18)
Demographics				
Age in years, mean (SD)	22.63 (5.90)	23.36 (4.34)	21.91 (4.63)	24.44 (9.43)
Female, %	31.3	54.5	60.6	50.0
Alcohol-related variables mean (95% CI)				
Drinks per drinking day[a]	10.14	6.65	4.37	3.90
	(8.38 to 11.91)	(4.52 to 8.78)	(3.13 to 5.62)	(2.24 to 5.57)
Percent days abstinent[a]	47.29	45.45	77.40	78.89
	(36.16 to 58.42)	(32.03 to 58.88)	(69.53 to 85.27)	(68.40 to 89.38)
Age of first drink in years	12.91	14.64	14.88	16.50
	(11.30 to 14.51)	(12.70 - 16.57)	(13.74 - 16.01)	(14.99 - 18.01)
AUDIT consumption	9.38	8.55	4.75	4.78
	(8.18 to 10.57)	(7.11 to 9.99)	(3.91 to 6.00)	(3.65 to 5.90)
AUDIT dependence	3.13	3.82	.59	.39
	(2.61 to 3.64)	(3.20 to 4.44)	(.23 to .96)	(-.10 to .87)
AUDIT consequences	7.19	7.55	2.50	2.83
	(5.68 to 8.70)	(5.72 to 9.32)	(1.43 to 3.70)	(1.41 to 4.26)
Multivariate Effects				
Variable	η_p^2		*F*	*p*
AUD diag	.71		$F(6,68) = 27.31$	< .001***
SAD diag	.19		$F(6,68) = 2.62$	.03*
AUD diag*SAD diag	.11		$F(6,68) = 1.32$	.26

Note. AUD = Alcohol-dependent drinker without social anxiety disorder; SAD+AUD = Alcohol-dependent drinker with social anxiety disorder; ND = Non-dependent drinker without social anxiety disorder; ND+SAD = Non-dependent drinker with social anxiety disorder.

[a] Past 30 days.

* *p* < .05; *** *p* < .001

Alcohol-dependent drinkers with and without comorbid social anxiety reported a greater number of drinking days, fewer days abstinent from drinking and higher overall scores on the three subscales of the AUDIT compared to their non-dependent counterparts (all *p* values < .001). Dependent drinkers overall reported taking their first alcoholic drink at an earlier age than non-dependent drinkers with and without social anxiety (*p* = .02). A main effect for social anxiety diagnosis was also observed, with individuals with a diagnosis of SAD reporting fewer drinks per drinking day compared to those without a diagnosis of SAD (*p* = .03). Age of first drink was also significantly higher for those with social anxiety disorder compared to those without (*p* = .04).

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Differences between diagnostic groups on measures of reward drive and rash impulsivity.

A MANOVA was used to examine whether dependent drinkers had higher levels of reward drive (BAS-Drive and BAS-Reward) and rash impulsivity (I_7-IMP and IGT) compared to non-dependent drinkers, and whether dependent drinkers with comorbid SAD displayed higher levels of trait and behavioural measures of impulsivity compared to non-dependent drinkers with SAD (see Table 5). There was a significant main effect of drinker severity on the combined dependent variables ($p < .001$), but not of social anxiety diagnosis ($p = .38$). The overall multivariate effect of drinker severity by social anxiety diagnosis interaction on these measures approached significance ($p = .06$). To further investigate these effects, separate univariate ANOVAs on the outcome variables were examined.

There was a significant drinker severity by social anxiety diagnosis interaction on the net-score on the IGT (behavioural measure of rash impulsivity), $F(1,74) = 6.79$, $p = .01$; $\eta_p^2 = .08$. In direct opposition to hypotheses, there was a significant simple main effect of SAD diagnosis within the AUD group ($p = .01$), with comorbid SAD+AUD individuals performing better than individuals with alcohol dependence only. As hypothesised, a significant difference was observed between the alcohol-dependent group and the non-dependent group ($p = .04$), with alcohol-dependent individuals performing worse on the task than non-dependent individuals.

As hypothesised, significant and large drinker severity effects on the trait measure of rash impulsivity I_7-IMP, $F(1,74) = 26.58$, $p < .001$; $\eta_p^2 = .26$, and the trait measure of reward drive (BAS-Drive), $F(1,74) = 7.27$, $p < .01$; $\eta_p^2 = .09$ were also found, indicating that dependent drinkers reported higher levels of trait impulsivity and BAS-Drive than non-dependent drinkers. No effects were found for BAS-Reward ($p > .05$).

Table 5

Mean impulsivity scores for dependent and non-dependent drinkers with, and without, social anxiety

	AUD (*n* = 16)	SAD+AUD (*n* = 11)	ND (*n* = 33)	ND+SAD (*n* = 18)
Reward drive measures				
BAS-Drive				
M	12.69	12.09	11.48	10.56
SD	1.96	1.97	2.39	1.54
BAS-Reward				
M	16.81	17.09	17.55	16.78
SD	2.01	1.87	2.15	2.73
Rash impulsivity measures				
I_7(IMP)				
M	11.88	13.00	7.97	6.61
SD	4.29	3.74	4.13	3.99
IGT				
M	-9.63	8.73	1.70	-3.56
SD	21.88	20.83	15.82	18.58
Multivariate Effects				
Variable	η_p^2	*F*		*p*
AUD diag	.28	$F(4,71) = 6.82$		< .001***
SAD diag	.06	$F(4,71) = 1.06$		.38
AUD diag × SAD diag	.12	$F(4,71) = 2.31$		.06†

Note. AUD = Alcohol-dependent drinker without social anxiety disorder; SAD+AUD = Alcohol-dependent drinker with social anxiety disorder; ND = Non-dependent drinker without social anxiety disorder; ND+SAD = Non-dependent drinker with social anxiety disorder; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale; I_7(IMP) = Impulsiveness subscale ; IGT = Iowa Gambling Task.

† *p* < .10; *** *p* < .001

Effects of cue induction on alcohol craving, and positive and negative mood states between diagnostic groups.

A mixed MANCOVA was conducted to test whether dependent drinkers reported an increase in self-reported craving and positive affect during the alcohol exposure compared to non-dependent drinkers, and whether increases in craving were higher during alcohol cue exposure for dependent drinkers with comorbid SAD compared to those with alcohol dependence alone and non-dependent drinkers with SAD. Mean craving ratings, state positive and negative mood scores by cue induction phase across the experiment and overall MANCOVA results are shown in Table 6. Analyses revealed a significant difference across the two cue conditions (*p* = .04). Inspection of the univariate effects indicated that the alcohol cue induced greater craving than the water cue, $F(1,72) = 7.07$, *p* = .01, $\eta_p^2 = .09$ (manipulation

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check). Multivariate tests showed significant interactive effects between drinker severity and cue condition ($p = .03$) and also between cue condition, drinker severity and social anxiety diagnosis ($p = .001$). However, the interaction between social anxiety diagnosis and cue condition was not significant.

Follow-up univariate analyses revealed a moderate and significant interaction effect between cue condition and drinker severity on craving for alcohol, $F(1,71) = 5.88$, $p < .01$, $\eta_p^2 = .08$. As hypothesised, there was a significant simple main effect of drinker severity in response to the water and alcohol cues, with the alcohol-dependent individuals with and without comorbid SAD, reporting higher self-reported craving to the sight and smell of water and alcohol compared to the non-dependent drinkers ($p < .001$). Similarly, there was a simple main effect of condition in individuals with alcohol dependence, with alcohol-dependent drinkers with and without comorbid SAD, showing greater self-reported craving reactions to the sight and smell of alcohol compared to the water stimulus ($p < .001$).

There was a significant interaction between cue condition, drinker severity and social anxiety diagnosis, $F(1,71) = 17.48$, $p < .001$, and this effect was large, $\eta_p^2 = .20$. A simple main effect of time was found depending on diagnostic group, with the comorbid SAD+AUD group reporting an increase in negative affect during the alcohol cue exposure in comparison to the water stimulus ($p < .001$). Individuals with comorbid SAD+AUD also showed higher negative affect in response to the alcohol cue compared to individuals with SAD only ($p = .01$).

Contrary to our hypotheses, no univariate tests were significant for cue condition, drinker severity and social anxiety diagnosis in relation to positive affect (all F values < 2.32, all p values $> .13$), indicating that in this sample the diagnostic groups did not differ in positive affect, and that the cue manipulation had no significant effect on positive mood states.

Table 6

Adjusted mean craving and mood ratings by cue condition for dependent and non-dependent drinkers with and without social anxiety

	AUD		SAD+AUD		ND		ND+SAD	
	Water	Alcohol	Water	Alcohol	Water	Alcohol	Water	Alcohol
Urge to drink								
M	25.57	38.97	23.86	35.92	23.15	32.65	21.57	28.72
SE	1.95	1.89	2.28	2.21	1.34	1.30	1.82	1.76
Positive Affect								
M	24.70	26.86	24.03	25.71	25.21	24.88	25.48	25.98
SE	1.32	1.93	1.54	2.26	.91	1.33	1.23	1.80
Negative Affect								
M	12.59	11.92	12.17	15.21	12.36	13.12	11.18	11.22
SE	.46	.79	.54	.92	.32	.54	.43	.74

Multivariate Effects

Variable	η_p^2	F	p
Cue	.11	$F_{(3,69)} = 2.89$	.04*
Cue × AUD Diag	.12	$F_{(3,69)} = 3.13$	.03*
Cue × SAD Diag	.06	$F_{(3,69)} = 1.43$	.24
Cue × AUD Diag × SAD Diag	.21	$F_{(3,69)} = 6.13$	.001**

Note. AUD = Alcohol-dependent drinker without social anxiety disorder; SAD+AUD = Alcohol-dependent drinker with social anxiety disorder; ND = Non-dependent drinker without social anxiety disorder; ND+SAD = Non-dependent drinker with social anxiety disorder.

* $p < .05$; ** $p < .01$

Effects of cue induction on skin conductance and heart rate between diagnostic groups.

Mean skin conductance level (SCL) and mean heartbeats per minute (BPM) were examined separately for imaginal (audio recording of the vignette) and in vivo (visual and olfactory exposure to beverage) cues. Differences were observed in reactivity during the audio compared to in vivo exposure; therefore the data were kept as separate dependent variables for the analysis. Mean SCL and mean BPM across the experiment are shown in Table 7.

To assess whether dependent drinkers would experience greater increases in skin conductance and heart rate between the water and alcohol cue exposure, and if comorbid SAD+AUD individuals would also exhibit increases reactivity compared to non-dependent drinkers with SAD and dependent drinkers without SAD, a mixed MANCOVA was performed (overall F tests for the MANCOVA are shown in Table 7).

In opposition to hypotheses, the analysis revealed no significant main effect of cue type ($p = .86$) or interaction between cue condition, drinker severity, and diagnosis of social anxiety ($p = .96$). There was, however, a significant interaction effect for cue condition and drinker severity ($p < .02$). Follow-up univariate tests showed skin conductance during the in vivo exposure changed differentially across cues (water – alcohol) depending on drinker severity, $F(1,72) = 4.23$, $p = .02$, and this effect was moderate, $\eta_p^2 = .06$. As hypothesised, simple effects analyses revealed that skin conductance increased after the presentation of alcohol compared to the water stimulus for the alcohol-dependent drinkers ($p < .001$) and those with comorbid SAD+AUD ($p = .008$). Similarly, greater heart rate increases as a reaction to alcohol versus water during in vivo exposure were observed for alcohol-dependent drinkers compared to those without this diagnosis, $F(1,72) = 6.70$, $p = .01$, and this effect was large, $\eta_p^2 = .09$. Physiological reactivity to the imaginal cue exposure did not differ by drinker severity. No other main effects or interactions were significant (all F values $< .34$, all p values $> .45$).

Table 7

Adjusted mean skin conductance level (µS) and heart rate (BPM) by cue condition for dependent and non-dependent drinkers, with and without social anxiety

	AUD		SAD+AUD		ND		ND+SAD	
	Water	Alcohol	Water	Alcohol	Water	Alcohol	Water	Alcohol
SCL Audio								
M	24.88	25.06	25.70	24.78	23.74	23.65	26.57	26.31
SE	1.02	1.34	1.24	1.62	.71	.93	.97	1.27
SCL in vivo								
M	23.75	27.18	23.79	26.85	23.13	24.32	24.95	26.31
SE	1.01	1.51	1.22	1.83	.70	1.05	.96	1.44
BPM Audio								
M	67.01	67.04	66.96	67.22	66.36	66.39	66.67	66.08
SE	.95	.95	1.15	1.15	.66	.66	.90	.90
BPM in vivo								
M	68.67	69.84	69.27	70.14	68.23	67.81	69.14	68.20
SE	.93	.97	1.12	1.17	.64	.67	.88	.92

Multivariate Effects

Variable	η_p^2	F	p
Cue	.02	$F_{(4, 69)} = .33$	.86
Cue*AUD diag	.16	$F_{(4, 69)} = 3.25$	.02*
Cue*SAD diag	.02	$F_{(4, 69)} = .34$	.85
Cue*AUD diag*SAD diag	.01	$F_{(4, 69)} = .15$	.96

Note. AUD = Alcohol-dependent drinker without social anxiety disorder; SAD+AUD = Alcohol-dependent drinker with social anxiety disorder; ND = Non-dependent drinker without social anxiety disorder; ND+SAD = Non-dependent drinker with social anxiety disorder. SCL = Skin conductance level (µS); BPM = Beats per minute.

* $p < .05$

Discussion

Using a two-component model of impulsivity, we examined whether reward drive and rash impulsivity could be used to explain differences between alcohol-dependent individuals with and without comorbid social anxiety disorder, and non-dependent individuals with and without social anxiety disorder. A comprehensive assessment of these factors was conducted and revealed that diagnostic groups could be differentiated on some, but not all components of impulsivity.

Differences between groups on trait measures of impulsivity

Similar to previous findings (e.g., Kambouropoulos & Staiger, 2004b, 2007; Knyazev, 2004) dependent drinkers overall were found to score higher on one measure of reward drive (BAS-Drive) and rash impulsivity (I_7-IMP) than non-dependent drinkers overall. This finding is also consistent with recent evidence of an "impulsive" social anxiety subtype characterised by heightened reward drive, rash impulsiveness, risky behaviour and more severe alcohol and substance use (Nicholls et al., 2014). These dimensions of impulsivity are associated with motivation to pursue goals (i.e., potentially rewarding experiences), and unplanned impulsive behaviour (Dawe & Loxton, 2004). The elevated reward drive in dependent drinkers with, and without, comorbid SAD suggests these individuals may be highly sensitive to rewarding cues and be inclined to experience strong behavioural approach when rewarding cues, such as alcohol, are present. This hypersensitivity and persistence in pursuing incentive cues may be implicated in motivation to drink alcohol. Similarly, higher scores on rash impulsivity suggests dependent individuals may be characterised by behavioural disinhibition and a tendency to act spontaneously, without consideration of consequences. In addition, individuals high on this trait may find it harder to refrain from drinking in environments where alcohol is available.

Although these impulsivity traits are believed to possess temporal stability, the possibility that elevated BAS-Drive and I_7-IMP are a consequence of problematic alcohol use cannot be

ruled out. Furthermore, no differences were observed for the BAS-Reward scale, also believed to measure reward drive. This may be due to individuals in the current study actually displaying similar levels of this trait or could be due to the reward subscale measuring a different aspect of reward sensitivity compared to the drive subscale.

Differences between groups on behavioural measures of impulsivity

Contrary to hypotheses, while individuals with alcohol dependence showed poorer performance on the Iowa Gambling Task compared to non-dependent drinkers, this result was not significant. Interestingly, in direct opposition to hypotheses, the comorbid SAD+AUD group showed decreased rash impulsivity (i.e., performing better) compared to the alcohol-dependent group. Less risky decision-making on this task requires that an individual sacrifice an immediate reward for smaller longer-term rewards to prevent larger losses. The two-factor model recognises that executive control processes are vital for effectively avoiding the immediate approach towards rewarding stimuli and for developing optimal patterns of behaviour. The reduced capacity of alcohol-dependent individuals to balance short-term and long-term consequences of a decision may be the result of an overactive automatic attention and memory system for indicating the occurrence of reward cues, and/or a diminished ability to control one's behaviour. These deficits may contribute to problematic drinking directly, or reflect pre-existing vulnerabilities. While participants with AUDs alone exhibited heightened rash impulsivity as measured by this behavioural task, those with comorbid SAD+AUD did not. This could be because individuals with SAD+AUD may be more fearful about greater losses (punishment) resulting from higher risk-taking, or they could be more receptive to positive reinforcement (reward), as decision-making in this task is thought to result from learning of reward and punishment. The reverse could also be true; that is, individuals with alcohol dependence may lack anticipatory anxiety before making a bad (i.e., "risky") decision (Kambouropoulos & Staiger, 2001).

According to Dawe et al.'s (2004) two-factor model of impulsivity, craving and positive affect are thought to represent behavioural levels of reward drive. Consistent with our hypotheses and replicating previously reported findings (e.g., Carter & Tiffany, 1999), alcohol-dependent individuals with, and without, comorbid SAD exhibited greater increases in self-reported craving than non-dependent individuals when exposed to an alcohol beverage. Differences in physiological responding was also observed as a function of the mode of cue induction. In vivo exposure of the alcohol cue elicited an elevated physiological response in both heart rate and skin conductance for dependent drinkers with, and without, comorbid SAD, but not in non-dependent drinkers with, and without, SAD. The finding of increased subjective craving and physiological reactivity to alcohol cues in dependent drinkers in this study supports a general appetitive motivational model of alcohol use. That is, reactivity to cues may represent an underlying motivational process that contributes to problematic alcohol use. Specifically, Robinson and Berridge (1993) propose that over time the neural system in susceptible individuals becomes sensitised and changes due to the addictive substance and this serves to act as a maintaining factor.

Interestingly, increased reactivity to alcohol was not observed during imaginal cue exposure of the alcohol cue. These findings indicate that the in vivo cue involving sight and smell of the alcohol beverage was more salient than the imaginal cue, which involved thinking about drinking. Imaginal cue exposure is commonly used as part of alcohol treatment and this indicates that incorporating in vivo exposure may be important to adequately elicit a state of craving or to extinguish cue-specific craving in therapeutic programs.

Contrary to hypotheses, we did not observe a difference in subjective craving and physiological reactivity between individuals with alcohol dependence and those with additional social anxiety. Kushner et al.'s (2011) model suggests a hypersensitive stress/anti-reward brain system in anxious individuals that may lower the threshold for alcohol dependence, however this was not borne out in our results which may suggest the level of

alcohol-related craving and autonomic arousal is similar for anxious and non-anxious drinkers once dependence has developed. This study included individuals with both clinical and subclinical levels of social anxiety and therefore the hypersensitive stress/anti-reward brain system in anxious individuals described by Kushner et al. may not have been as pronounced as would be if a more severe sample were utilised. This result may also be due to methodological differences in how a lower threshold for dependence was measured. In this study, we took reactivity to alcohol cues to reflect a lower threshold for dependence whereas in Kushner et al.'s study, a lowered threshold for dependence was reflected by a shorter time from commencing alcohol use to developing dependence symptoms.

While dependent drinkers with and without comorbid SAD showed increases in subjective and objective measures of alcohol craving compared to the non-dependent drinkers, exposure to the alcohol cue did not lead to significant increases in positive affect for any participants. This aspect of reward drive may therefore be a less important element of cue reactivity. Other affective processes may be more important; for instance, it has been suggested that reinforcement associated with alleviating negative affect is vital to understanding addiction (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Thus, individuals may consume alcohol in an attempt to regulate negative affect, rather than to attain a positive affective state. Indeed, prominent theoretical models of the SAD-AUD link suggest that individuals with social anxiety use alcohol to manage distress associated with their concerns regarding negative evaluation and scrutiny by others (Khantizian, 1985). Results of the current study support this model, with higher negative affect observed during exposure to alcohol, compared to responses to the water cue, for individuals with comorbid SAD+AUD. The higher level of negative affect reported by the comorbid individuals is of interest as it suggests a role for the aversive motivation in alcohol consumption. Exposure to alcohol and alcohol cues in the absence of alcohol ingestion may prompt problematic alcohol use due to a need to alleviate negative affect and/or an expectation that alcohol will relieve distress (e.g., expected negative

reinforcing properties; Cooney et al., 1997), and repeated use of alcohol in this context could lead to dependence. However, individuals with SAD are prone to experience negative affect and this pathway to alcohol use can be seen as typical for these individuals as they have more opportunities to associate negative mood with drinking. In addition, negative mood states may promote impulsive action by undermining rational, advantageous decision-making and/or sensitising reward pathways and active avoidance (Gullo, Loxton, & Dawe, 2014).

Limitations

There were a number of limitations to this study that warrant consideration. Firstly, it is likely that the small group sizes limited to the power to detect some smaller effects, thus replication using larger samples is required. The sample also included non treatment-seekers and not all participants met full criteria for social anxiety and alcohol use disorders. Thus, while the sample shows a broader range of symptom levels with the inclusion of subclinical individuals, the range of alcohol problems and social anxiety may not be representative of treatment-seeking individuals and those on the higher spectrum of clinical severity of these disorders. Additionally, it is unclear whether the vignettes did add to craving induction as although participants were encouraged to adopt an imaginal mode of thinking about the water and alcohol cues during the audio recording of the vignette, no differences were observed in autonomic arousal for the different cues during this period. A final caveat relates specifically to the experimental cue reactivity paradigm. Following standard cue reactivity principles, cue presentation order was held constant, therefore the way in which exposure to an alcohol cue influences subsequent reactions to the water stimulus is unknown.

Conclusion

These findings offer additional evidence of heightened cue reactivity in dependent drinkers, and uniquely extend results to dependent drinkers with comorbid social anxiety disorder. Results suggest that in individuals with alcohol dependence, with and without comorbid social anxiety disorder, alcohol misuse may be associated with differences in trait

and behavioural reward drive and rash impulsivity, including craving and autonomic arousal in the presence of alcohol cues. Reward drive and rash impulsivity may play a key role in influencing why some individuals with social anxiety go on to develop problems with alcohol while others do not; perhaps acting as a vulnerability, moderating factor, or maintaining alcohol use. Individuals with comorbid SAD-AUD may be vulnerable to alcohol misuse via an additional mechanism involving negative affect regulation which may act to motivate substance use, provide reinforcement or prompt impulsive action. However, individuals with comorbid social anxiety and alcohol use disorders appear less susceptible to the decision-making impairments observed for dependent drinkers. Evidence from the present study indicates that the consideration of specific facets of impulsivity may be one avenue to examine mechanisms underlying the relationship between social anxiety and alcohol use. Given the high rates of comorbidity between SAD and AUDs, the generation of empirical data on these aspects of impulsivity—including cue reactivity—may be beneficial for incorporation in several strategies of intervention for individuals with these problems, such as craving management, coping skills and contextual cue avoidance and extinction. Future studies may benefit from the use of treatment-seeking samples and examination of how ingestion of alcohol impacts upon craving and reactivity to alcohol cues. Hence, this may provide additional insights into the influence of reward drive and rash impulsivity and whether these relationships generalise to more severe populations.

Chapter Four

Influence of Alcohol on Social Anxiety and Alcohol Comorbidity: An Investigation of Impulsivity, Pharmacologic and Expectancy Effects

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Abstract

Although the relationship between social anxiety and alcohol problems has been the subject of extensive exploration, the underlying mechanisms are still not clearly understood. It is well established that both the pharmacologic and expectancy effects of alcohol and alcohol-related stimuli impact alcohol drinking behaviour and relapse. In individuals with comorbid social anxiety disorder and alcohol problems, the effects of alcohol may be influenced by contextual and individual factors that deserve to be more thoroughly investigated. This study examined physiological and subjective responses to alcohol's pharmacological and expectancy effects, and their association with impulsivity and alcohol expectancy in treatment-seeking individuals with comorbid social anxiety and alcohol problems. Thirty-nine participants were randomly assigned to one of three experimental groups: alcohol, placebo alcohol or soft drink. Subjective ratings for desire to drink alcohol, subjective stimulant and sedative effects, and physiological recordings of skin conductance and heart rate were obtained. Higher levels of impulsivity was associated with greater subjective stimulating effects as well as increased heart rate. Higher levels of social anxiety was associated with greater subjective sedative effects. Pharmacologic and expectancy effects were evident in individuals who received alcohol and placebo alcohol, with greater increases in heart rate, and those receiving alcohol also showed increases in skin conductance. These findings emphasise the importance of individual differences in alcohol responses, and support a relationship between subjective and physiological effects which may serve to maintain hazardous alcohol use.

Influence of Alcohol on Social Anxiety and Alcohol Comorbidity: An Investigation of

Impulsivity, Pharmacologic and Expectancy Effects

Social anxiety disorder (SAD) and alcohol use disorders (AUD) co-occur at a high rate in epidemiological and clinical samples (Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997; Merikangas, Stevens, et al., 1998). Approximately 50% of individuals with a lifetime diagnosis of SAD suffer from lifetime prevalence of an AUD (Grant et al., 2005) versus 18.6% among the general population (Kessler, Chiu, Demler, Jin, Merikangas, & Walters, 2005). Furthermore, the 12-month prevalence of AUD among individuals with SAD is 13.1% (Grant et al., 2005) compared to 8.5% in the general population (Grant et al., 2004). Social anxiety tends to precede the onset of alcohol abuse or dependence (Bakken et al., 2003, 2005; Cox et al., 1990; Merikangas, Stevens, et al., 1998; Schuckit et al., 1997), leading many to believe that individuals with social anxiety use alcohol as a coping mechanism to alleviate distress and symptoms associated with anxiety (Carrigan & Randall, 2003; Khantizian, 1985; Sher & Levenson, 1982). However, using alcohol as a coping strategy may be maladaptive as alcohol consumption and subsequent intoxication can lead to depressive symptoms and cognitions (Raimo & Schuckit, 1998), and longer term use can cause withdrawal symptoms resulting in increasing levels of anxiety (Kushner et al., 2000; Morris et al., 2005). Indeed, the subset of individuals with comorbid SAD-AUD experience greater symptom severity and less effective treatment outcomes than individuals with either disorder alone (Brady & Lydiard, 1993; Burns & Teesson, 2002; Burns et al., 2005). Understanding why individuals with these problems continue to consume alcohol when it may increase distress and exacerbate symptoms in both the short and long-term is one of the key challenges in disentangling this complex relationship. A greater understanding of the factors that contribute to the escalation and maintenance of problematic drinking is essential to guide prevention and early intervention strategies for individuals with these disorders.

One possible source of vulnerability to developing alcohol problems is the degree of subjective arousal (i.e., self-reported effects) to alcohol (Fischman & Foltin, 1991). Both individuals' experiences of self-reported craving and subjective reactions to alcohol have been linked to alcohol-related outcomes (Courtney et al., 2013; King, de Wit, McNamara, & Cao, 2011). For instance, higher levels of craving and greater sensitivity to the stimulating and rewarding effects of alcohol as measured by the Biphasic Alcohol Effects Scale have been associated with increased alcohol consumption and an elevated risk of developing an alcohol use disorder (e.g., King et al., 2011; Ray, Mackillop, & Monti, 2010; Ray, McGeary, Marshall, & Hutchison, 2006). Specifically, craving for alcohol has been linked to reduced control over alcohol consumption (Bohn et al., 1995; Kozlowski, Mann, Wilkinson, & Poulos, 1989) and relapse (Ludwig & Wikler, 1974), while the subjective stimulating effects of alcohol have been proposed as key contributors to the development and maintenance of craving (Baker, Morse, & Sherman, 1986). Furthermore, extensive research employing cue reactivity paradigms or controlled laboratory exposure to environmental cues has shown that these subjective responses to alcohol are significantly affected by alcohol-related stimuli and contextual factors (Monti et al., 1987; Rohsenow et al., 1992).

There is also substantial variability in the degree to which individuals react to alcohol-related stimuli, with some individuals showing little to no reactivity when exposed to alcohol cues (Litt, Cooney, & Morse, 2000), and others demonstrating considerable responses to alcohol cues (Carter & Tiffany, 1999). This pattern of reactivity has been demonstrated in a recent meta-analysis of functional neuroimaging studies of alcohol cue reactivity which reports greater cue-elicited activation of certain brain areas in individuals with AUD compared to controls (Schacht, Anton, & Myrick, 2012). This heterogeneity in magnitude and direction of effects across and within studies may be a factor in why some individuals go on to develop problems with alcohol while others do not, and may also provide some insight into how alcohol problems are maintained once they develop in those with alcohol use disorders.

One explanation for this heterogeneity of reaction to alcohol and related stimuli is that responses to alcohol and contextual cues may also be impacted by personality (e.g., impulsivity) and cognitive processes (e.g., alcohol expectancies). For instance, individual differences in cue reactivity could be related to variation in two components of impulsivity, reward drive and rash impulsivity (2-CARS model; Dawe et al., 2004). Specifically, these traits are associated with sensitivity to stimuli that predict the occurrence of a reward (i.e., sensitivity to reward or "reward drive") and to an inability to exercise sufficient self-control in the presence of tempting environmental stimuli (i.e., rash impulsivity). Heightened sensitivity to rewarding stimuli has been found to be associated with greater physiological responses to alcohol (e.g., increased heart rate; Brunelle et al., 2004), and stronger conditioning to alcohol cues (e.g., changes in skin conductance; Glautier, Bankart, & Williams, 2000). Additionally, higher scores on measures of reward drive have been associated with increased craving and approach behaviour (Franken, 2002; Kambouropoulos & Staiger, 2001), while impaired response inhibition; that is, high rash impulsivity, could lead to greater cue-elicited craving via a difficulty in controlling an appetitive response to highly rewarding stimuli such as alcohol or alcohol cues (Dawe & Loxton, 2004). Although few empirical studies have been conducted in this area, Papachristou, Nederkoorn, Havermans, van der Horst, and Jansen (2012) found that compared to light drinkers, deficits in response inhibition in heavy drinkers led to greater craving in response to alcohol cues, showing how this component of impulsivity may be a risk factor for excessive drinking.

Heterogeneity in drinking behaviours may be further explained by the system of assumptions and beliefs an individual has about alcohol's effects; that is, alcohol expectancies (Brown, Goldman, Inn, & Anderson, 1980). An individual's expectations about alcohol can motivate drinking, independent of alcohol's pharmacological properties and effects (e.g., Abrams & Kushner, 2004). Indeed, many studies have demonstrated that alcohol expectancies are important in regulating drinking behaviour. Coping expectancies, for example, are

associated with drinking larger amounts, more often, and alcohol-related problems (Grant & Stewart, 2007). Furthermore, an increased expectation that alcohol reduces tension and anxiety has been reported in individuals with comorbid social anxiety compared to those without this comorbidity (Tran & Haaga, 2002), and is associated with both drinking to cope and problematic levels of alcohol use (Brown, 1985; Cooper, Frone, Russell, & Mudar, 1995). Similarly, social facilitation expectancies; that is, beliefs that drinking will produce positive social effects, have been strongly associated with alcohol consumption in socially anxious college students (Burke & Stephens, 1999).

More recently, alcohol expectancies have been proposed to be a mechanism through which trait impulsivity conveys risk (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010). In their proposed 2-CARS model, Gullo et al. (2010) found that positive alcohol expectancies mediated the relationship between reward drive and problematic alcohol use in both young adult and treatment-seeking individuals. Similarly, Booth and Hasking (2009) found that reward drive and alcohol expectancies influenced the relationship between social anxiety and alcohol use. Specifically, adults with high trait reward drive showed a positive relationship between social anxiety and drinking when they reported strong tension reduction expectancies. The authors suggest that heightened reward drive may be involved in motivating individuals to place themselves in rewarding environments where alcohol and its effects can be experienced both directly and indirectly, via contextual cues and observing others drink. It is thought that these experiences subsequently lead to the development of more positive alcohol expectancies (Booth & Hasking, 2009). However, this study was limited by a reliance on retrospective assessment of these relationships using only self-report questionnaires.

The current study is the first to investigate the subjective and physiological effects of drinking cues and alcohol ingestion on cue reactivity in individuals with social anxiety and alcohol use disorders. In the current study, expectancy was both controlled for in design; by

including a placebo alcohol condition, and measured as a trait. Further, in addition to reward drive, the moderating role of rash impulsivity was examined using both trait and behavioural measures. These personality and cognitive factors have been identified as risk factors for both alcohol use disorders and social anxiety, but examination of these constructs to date has been conducted in disparate areas of research. The current study will be the first to examine how specific personality traits and cognitive factors relate to acute alcohol intoxication and perceived intoxication.

Based on the argument by Dawe and Loxton (2004) and Gullo and Dawe (2008) that reward drive is associated with reward-related learning and positive alcohol expectancies, it was hypothesised that reward drive and performance enhancement alcohol expectancies would lead to subjective reports of greater craving and stimulant effects and increases in physiological arousal (e.g., skin conductance, heart rate). Similarly, we expected that higher levels of trait and behavioural rash impulsivity would be associated with higher levels of craving and physiological arousal. Furthermore, based on research showing strong links between social anxiety, tension reduction and social facilitation alcohol expectancies, it was hypothesised that social anxiety and alcohol expectancies of tension reduction and social lubrication would be associated with greater feelings of sedation and craving, and decreases in physiological arousal.

Method

Design

To test the hypotheses, socially anxious problem drinkers were randomly allocated to receive either a drink containing alcohol, a drink they believed contained alcohol (placebo) or a soft drink. Self-report and psychophysiological measures were taken on three occasions (pre-drinking, during-drinking, post-drinking). In this paper the main focus is the relationship between self-report measures of impulsivity, alcohol expectancies and subjective and physiological responses to the three beverage conditions.

Participants

A sample of adults seeking treatment for comorbid social anxiety disorder and hazardous or harmful alcohol use ($N = 39$, female = 48.7%, age range 20 to 59, $M = 35.77$, $SD = 10.75$) were recruited from a larger randomised controlled trial for the treatment of comorbid social anxiety and alcohol dependence (for details see Baillie et al., 2013). Individuals were invited to participate in this study prior to receiving 10 sessions of CBT treatment. A total of 75 participants were screened. Fifteen declined to participate, ten individuals were pursuing a goal of abstinence, three were excluded due to risk of severe alcohol withdrawal and three were excluded based on current medication or medical conditions. A further two participants withdrew from treatment and three did not attend the scheduled experimental session. All participants met Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association [APA], 2000) criteria for social anxiety disorder as assessed using the Anxiety Disorders Interview Schedule (ADIS; Di Nardo et al., 1994) at the entry point of the treatment study, and a score of eight or above on the Alcohol Use Disorders Identification Test[1] (Saunders, Aasland, Babor, De La Fuente, & Grant, 1993). Individuals were only eligible for inclusion in the study if they were of legal age to drink (over 18 years), and had consumed an alcohol dose equivalent to the dose used in this study in the previous two months. Participants with additional diagnoses were also included, with the exception of those with psychosis and/or abuse/dependence of substances other than alcohol, caffeine or nicotine. Additional exclusion criteria for participation in the current study are listed in the section titled "*Ethical Considerations*". Fifty-nine percent of participants received a primary diagnosis of Alcohol Use Disorder, while 41% had Social Anxiety Disorder as their primary disorder. Seventy-four percent had an additional anxiety disorder, 51.3% had additional depression or dysthymia and 7.7% had a condition not included in the ADIS, for example,

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[1]All participants except one met full DSM-IV-TR (APA, 2000) diagnostic criteria for an alcohol use disorder as assessed using the Anxiety Disorders Interview Schedule.

eating disorders, which was listed as "other"[2]. The majority of participants in the current

sample were born in Australia (61.5%), never married (53.8%) and in full-time employment

(56.4%). Less than half (35.9%) of participants were university educated with a Bachelor's

degree or higher.

Ethical Considerations

Individuals with alcohol use disorders have typically been excluded from research

involving alcohol administration due to concerns that alcohol consumption would negatively

affect their current condition or motivation for treatment. This has resulted in unrepresentative

samples and subsequently influenced the quality of the research. A comprehensive review of

the ethical, clinical and scientific issues in empirical research involving alcohol administration

showed that there was little evidence that participating in this type of research would result in

adverse effects on individuals with alcohol dependence (Dolinsky & Babor, 1997). In

collaboration with the Human Research Ethics Committee at Macquarie University, the

procedures used in this study included the following exclusion criteria to minimise the

potential for physical or psychological harm: i) current suicidal ideation, ii) any medical

conditions, current medications or doctor's advice that would preclude consumption of

alcohol, iii) current pregnancy or lactation, iv) at risk of severe alcohol withdrawal as

measured by a score of $\geq$20 on the revised Clinical Institute Withdrawal Assessment for

Alcohol Scale (CIWA-Ar; Sullivan, Sykora, Schneiderman, Naranjo, & Sellers, 1989), v)

criteria met for substance abuse other than alcohol (as assessed by ADIS-IV), vi) commitment

to pursuing a goal of abstinence from alcohol at the time of recruitment. Furthermore, all

participants were asked to attend the laboratory session via public transport or to arrange

transport with a friend/family member in the event they were allocated to receive alcohol.

Those who received alcohol were asked to remain in the laboratory until their blood alcohol

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[2] Diagnoses listed as "other" were conditions suspected by the interviewers based on
participants disclosure, however these conditions were not directly assessed as they are not
included in Anxiety Disorders Interview Schedule.

concentration had decreased to .02% or lower, and were provided with a taxi to return home to minimise the risk of physical harm or accident occurring while under the influence of alcohol. Full details regarding the ethical considerations and procedures of this study are available upon request.

Materials

Diagnostic assessment. The Anxiety Disorders Interview Schedule (ADIS; Di Nardo et al., 1994) is a structured interview designed to assess and diagnose anxiety, mood, somatoform and substance use disorders according to DSM-IV-TR criteria. Each diagnosis receives a clinical severity rating (CSR) from 0 to 8, indicating the clinical and functional severity of the disorder. Ratings ≥ 4 indicate that full diagnostic criteria were met. Participants had a mean baseline severity score of 6.19 (*SD* = .74) for their primary diagnosis and 5.33 (*SD* = .98) for their secondary diagnosis. Participants were assessed by clinical psychologists and inter-rater reliability for the clinical severity score was high for both SAD (κ = .94; $p < .001$) and AUD (κ = .90; $p < .001$).

Symptom measures of social anxiety and alcohol dependence.

Social Anxiety. The Social Interaction Anxiety Scale (SIAS) and the Social Phobia Scale (SPS) (Mattick & Clarke, 1998) were used to assess social anxiety. Each scale contains 20 items on which respondents rate their experiences in social situations associated with social anxiety *DSM-IV* criteria (e.g., SIAS), and describe situations in which the person is the focus of attention and observed by others (e.g., SPS; public speaking). Items are rated on a 5-point scale ranging from "Not at all like me" (0) to "Extremely characteristic or true of me" (4). The total score ranges from 0 to 80 for each scale, where higher scores indicate greater anxiety in social interactions and increased tension about being observed by others respectively. These components also load onto a single higher order factor of social anxiety. Both the SIAS and the SPS have been found to have excellent psychometric properties (Mattick & Clarke, 1998; Peters, 2000), and showed good internal consistency in the current study (α = .88).

Alcohol dependence. The Severity of Alcohol Dependence Questionnaire (SADQ; Stockwell, Sitharthan, McGrath, & Lang, 1994) is a 20-item questionnaire designed to measure the presence and severity of alcohol dependence. Each item is scored on a 4-point scale, ranging from "Almost never" to "Nearly always" giving a possible range of 0 to 60. A score of over 30 indicates severe alcohol dependence. Research has demonstrated evidence of good concurrent validity in a clinical sample ($r = .71$ with the Alcohol Problems Questionnaire and $r = .81$ with the Short Alcohol Dependence Data questionnaire; Heather, Booth & Luce, 1998). Cronbach's alpha indicated excellent internal consistency in this sample for the SADQ items ($\alpha = .91$).

Measures of alcohol use and subjective stimulant and sedative effects.

Alcohol use. The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) is a 10-item self-report measure of drinking behaviour. The AUDIT has three subscales assessing alcohol use quantity and frequency (three items; e.g., "How often do you have a drink containing alcohol?"); alcohol dependence (three items; e.g., "How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?"); and alcohol-related problems (four items; e.g., "Have you or someone else been injured as a result of your drinking?"). Scores above eight indicate risky or hazardous alcohol use, with scores above 20 indicating high risk and almost certain dependence. In the current study, 30 participants (76.9%) scored 20 or above. The psychometric properties of the AUDIT have been well established for use in various populations (e.g., university students; Daeppen, Yersin, Landry, Pécoud, & Decrey, 2000), and acceptable levels were replicated in the current sample ($\alpha = .72$).

Recent alcohol use was measured via the timeline follow-back method (Sobell & Sobell, 1992) to provide a comprehensive account of participants' frequency and quantity of alcohol consumption over the previous month. Participants were asked to estimate the amount of alcohol consumed on each drinking occasion during the specified time period. The number of

standard drinks consumed was then divided by the number of drinking days to yield an index of average number of drinks per drinking day. The timeline follow-back method has high test-retest reliability (.79 to .96) over 30- to 90-day periods, and strong concordance between self-reported information and collateral reports regarding consumption levels (Sobell & Sobell, 1992).

Craving. The Alcohol Urge Questionnaire (AUQ; Bohn et al., 1995) was used to measure acute craving with eight statements, rated on a 7-point scale for agreement. The total score ranges from 8 to 56, where higher scores indicate greater craving for alcohol. The AUQ has excellent internal consistency (α = .91; Bohn et al., 1995), and this was replicated in the current study (α = .91).

Subjective stimulant and sedative effects. The Brief Biphasic Alcohol Effects Scale (B-BAES; Rueger, 2009) is a brief six-item adjective-rating scale that is sensitive to ethanol-induced stimulant- and sedative-like effects. The participant indicates the extent to which they are feeling each effect on an 11-point scale from "Not at all" (0) to "Extremely" (10). The Stimulation scale is measured by summing the scores of the use of the following adjectives: *energized, excited,* and *up.* The Sedation score is measured by summing the use of the items: *sedated, slow thoughts,* and *sluggish*. These subscale scores show excellent internal consistency and very strong correlations with the full BAES scores (Rueger, 2013), and showed acceptable internal consistency in the current study (α = .73).

Alcohol expectancies. The Alcohol Outcome Expectancies measure (Kushner, Sher, Wood, & Wood, 1994) assesses the beliefs individuals hold about the consequences of drinking and consists of four subscales. Only the performance enhancement (nine items; e.g., "Drinking helps me perform certain tasks better"), tension reduction (nine items; e.g., "Drinking makes me feel less tense or nervous") and social lubrication (eight items; e.g., "Drinking makes me feel less shy") subscales were utilised in the current study. Response scales for each item ranged from "Not at all" (0) to "A lot" (4). Previous research with this

scale has demonstrated the validity of the measure in prospective research (Sher, Wood, Wood, & Raskin, 1996), and each subscale showed good internal consistency in this study with Cronbach alphas ranging from .79 (tension reduction) to .88 (performance enhancement).

Perceived intoxication. Participants rated their intoxication level by using a Likert-type scale ranging from "Not at all intoxicated" (0) to "Extremely intoxicated" (10). Participants estimated the amount of alcohol they received by using a second scale ranging from 0.00 to 0.10 or more (blood alcohol level).

Blood alcohol concentration (BAC). Blood alcohol concentration (BAC) was measured with an ACS Alert J5 Breathalyser (Alcohol Countermeasure Systems, Canada). The measuring range for this device is 0.000 to 0.450% BAC which was calibrated to display blood alcohol concentration as a percentage (% BAC). This gave a reading consistent with Australian drink-driving legislation and thus is in a familiar format to participants. As % BAC (equivalent to g/100L) is also equivalent to "blood alcohol level" used in other measures in this study and other international reporting standards (e.g., permille), we used it throughout for consistency.

Physiological recording. Heart rate and skin conductance level (SCL) were recorded because they have previously been used in psychopathology and addiction research as indices for the emotional and arousing nature of alcohol-related stimuli (e.g., Cooney et al., 1997; Kaplan et al., 1985). For all participants, three electrocardiogram (ECG) leads were placed in Lead I position on the limbs to monitor heart rate, and a galvanic skin response device was attached to the middle phalanges (segment) of the first and second fingers of the non-dominant hand to monitor SCL. LabChart 7 software linked to a PowerLab Data Acquisition System (ML116) and amplifiers were used to monitor and record mean heart rate in beats per minute (BPM) and mean skin conductance level (in microsiemens) at each time point. The signals were sampled at 1000 Hz in a sound-proofed room with a constant temperature.

Measures of impulsivity.

Reward drive. The Behavioural Inhibition/Behavioural Activation System scales (Carver & White, 1994), are 20 self-report items that assess reward drive. Participants were administered the complete questionnaire, however only the BAS subscales *drive* (four items; e.g., "I go out of my way to get things I want") and *reward responsiveness* (five items; e.g., "It would excite me to win a contest"), were used in the analysis as measures of reward drive in the current study. Inconsistent findings have been reported for the fun-seeking subscale as it correlates with measures that load on both facets of impulsivity (Caseras, Avilia, & Torrubia, 2003). Participants respond on a Likert scale ranging from "Not very true for me" (1) to "Very true for me" (4). Higher scores indicate greater levels of BAS sensitivity. The BIS/BAS scales have been found to be valid measures, with internal consistencies ranging from .66 (Fun Seeking) to .84 (Drive; e.g., Carver & White, 1994; Leone, 2001; Meyer, Johnson, & Winters, 2001), and this was replicated in the current sample (Drive: $\alpha = .85$; Reward responsiveness: $\alpha = .69$).

Rash impulsivity. Self-reported rash impulsivity was assessed using the Impulsivity scale from the 54-item Eysenck Impulsiveness Questionnaire (I_7) (Eysenck et al., 1985). This scale, the I_7-IMP, comprises 19 dichotomously scored (yes/no) items and assesses a tendency to act on impulse without sufficient forethought (e.g., "Do you usually make up your mind quickly?"). This subscale has been found to have good reliability ($r = 0.87$; Whiteside & Lynam, 2003) and showed acceptable internal consistency in the current study ($\alpha = .74$).

The Iowa Gambling Task (IGT, Bechara et al., 1994) was used as a behavioural measure of rash impulsivity using Inquisit (Version 3). Participants are required to choose a card from one of four decks (A, B, C, and D) during 100 successive trials. Each deck of cards is associated with differing levels of risk and reward. Two of the decks (A and B) are weighted to give out larger rewards, but are also associated with much larger costs. The decks labelled C and D are advantageous with moderate and more consistent gains and smaller losses.

Participants were instructed to try to gain as much money as possible by the end of the game. We used the net-score (advantageous decks-disadvantageous decks) across all trials as a dependent variable.

Procedure

Prior to attending the laboratory session and as a requirement of the screening process for the clinical trial, participants were asked to complete the following questionnaires: SIAS, SPS, SADQ, I_7, BIS/BAS, and the alcohol outcome expectancy measure. Eligible participants then attended a laboratory session which commenced between 3.00pm and 4.30pm as time of day can moderate the effect of alcohol consumption (Rohsenow & Marlatt, 1981). Participants were instructed to abstain from drinking heavily the night before, to avoid alcohol entirely on the day, to eat a medium sized meal three hours prior to the session, and to refrain from drinking caffeinated beverages on the day to avoid confounding due to variable metabolic rates and rates of gastric emptying (Abrams et al., 2001; Holt, 1981; Kushner et al., 1996; Rohsenow & Marlatt, 1981).

Participants were taken into in a dimmed laboratory room containing a wooden bar, cocktail and wine glasses, two armchairs opposite a 66 cm wall-mounted television and alcohol-related paraphernalia (posters and advertisements of alcohol, gambling and sports). Upon arrival, informed written consent was obtained and a baseline breath test was conducted to verify blood alcohol concentration of 0.000%. Participants were randomly allocated to one of three beverage conditions: i) given alcohol/expecting alcohol (alcohol group), ii) given placebo/expecting alcohol (placebo group), iii) given no alcohol/expecting no alcohol (control/soft drink). Conditions were double-blind so that neither the participant nor the experimenter knew the beverage contents in the alcohol or placebo group until the conclusion of the experimental session. Beverages were allocated and prepared by a drinks attendant in a separate room, who also conducted breathalyser tests. All other procedures were carried out by the experimenter.

Baseline or "pre-drinking" physiological recordings were taken for five minutes while participants watched a video containing neutral content. Participants were then instructed to complete several questionnaires including, the AUQ, B-BAES, a computerised task; the IGT, and a subjective intoxication form. After completing the questionnaires and the computerised task, participants were given two beverages; in the control group they were informed these were non-alcoholic, and in the alcohol and placebo groups, participants were informed the beverages were alcoholic and contained approximately two to four standard drinks. The time to consume the beverages was standardised across participants who were instructed to drink steadily over a 10-minute period (Rohsenow & Marlatt, 1981). This was followed by a 25-minute absorption period during which participants spent 15 minutes watching a neutral video and 10 minutes completing questionnaires, including the AUQ. Physiological measurements were recorded during the 15 minutes of watching the video. BAC was measured and all participants were asked to view and sign the breathalyser printout. To aid deception, the printout gave a fixed reading of "BAC = 0.053%" to participants allocated to the alcohol and placebo conditions. Actual readouts were recorded separately. Participants were again asked to complete the AUQ, B-BAES and the subjective intoxication questionnaire, rating any perceived effects of alcohol. Other cognitive and performance testings were obtained as part of another investigation. At the conclusion of the final task, all participants were debriefed and, in view of the deception involved in the procedure, individuals were asked to re-consent to their data being used. Participants allocated to receive alcohol were asked to stay an additional one to two hours, until their BAC returned to 0.02%. They were provided with food, DVDs and reading materials during this time and transported home by a car service to ensure safety. All participants were paid $AUD50 for their time.

Beverage Manipulation. In the alcohol condition the volume of alcohol administered was adjusted for gender and body weight (Curtin & Fairchild, 2003) with consideration of differences in total body water level as examined by (Watson, 1988), in order to target a BAC

of 0.05%. Drink volumes for the placebo and control groups were prepared to produce a total quantity of beverage comparable to the alcohol condition. Drinks consisted of a 3:1 mixture of lemonade and lime cordial to vodka (alcohol group); lemonade and lime cordial to flat tonic water alcohol substitute (placebo group); and lemonade and lime cordial to mineral water (control group). Mineral water was used in the control group to ensure beverage volume equivalence without varying the amount of flavoured mixer. All drinks were served with one ice cube.

To enhance credibility of the placebo administration and aid deception, the following alcohol/placebo procedures were adapted from Rohsenow and Marlatt (1981) and Martin and Sayette (1993): i) preparation of the beverage occurred in full view of the participant, with real or placebo alcohol poured from a commercially labelled vodka bottle and the beverage mixed in a cocktail shaker; ii) a 5 ml alcohol floater was added to the surface of the beverage to provide alcohol-related sensory cues (Kushner et al., 1996); iii) during beverage consumption and absorption, all participants watched a neutral wildlife video in order to minimise attention to interoceptive cues associated with the consumption of alcohol; iv) 95% ethanol solution was sprayed twice on an insulated glass holder before beverage preparation as per similar procedures by Sayette, Breslin, Wilson, and Rosenblum (1994). The soft drink control beverage was poured, pre-mixed, from a clearly marked lemonade bottle, with none of the accoutrements of alcoholic beverage presentation.

Manipulation checks.

Perceived intoxication. The subjective intoxication form was administered both at the beginning of the experimental session and after consumption of the beverage. Following beverage consumption, approximately 85% of individuals in the placebo group reported feeling at least "slightly intoxicated" (compared to 100% of individuals in the alcohol group). Mean perceived intoxication scores in the placebo and alcohol groups were significantly different at both times $t(23) = 4.60$, $p < .001$. However, mean subjective feeling of

intoxication was significantly greater than zero post beverage $t(12) = 4.91$, $p < .001$ for

participants in the placebo group. That is, while not feeling as intoxicated as the alcohol group

after beverage consumption, participants in the placebo group generally felt at least somewhat

intoxicated. All individuals in the control group receiving soft drink rated their intoxication

level as "not at all intoxicated". Additionally, 100% of individuals in the placebo group

estimated their BAC levels to be 0.02% or higher (compared to 100% of individuals in the

alcohol group who estimated 0.05% or higher), indicating that the deception worked for

individuals receiving placebo alcohol. Finally, as noted, the target BAC for individuals in the

alcohol group was 0.05%. Following consumption of the beverages and a 25-minute

absorption period, we administered a breathalyser test to each participant, with all control and

placebo group participants recording 0.000% BAC. Results indicate that the mean BAC

recorded for participants in the alcohol group was close to this target ($M = 0.059$, $SD = 0.009$

g/dl).

Data Scoring and Analyses

To examine the effects of the experimental manipulation, analyses were conducted to

compare the three groups: alcohol ($n = 12$), placebo ($n = 13$), and control ($n = 14$) on five

measures of alcohol cue reactivity: subjective craving ratings, differential stimulant and

sedative effects, skin conductance and heart rate. Self-report and physiological measurements

were analysed separately in light of past research suggesting a disconnect between

pharmacological and expectancy measures of responses to alcohol (Drobes & Thomas, 1999;

Rohsenow et al., 1992). A series of planned contrasts were conducted to (1) compare the

experimental group to the placebo group, and (2) compare the control group to the

experimental and placebo groups.

A mixed ANCOVA was conducted to examine the impact of reward drive (BAS-Drive and

BAS-Reward) and performance enhancement on B-BAES Stimulation. As reward drive and

performance enhancement expectancies were anticipated to impact this relationship, they

were measured and included in the analysis as covariates. Additional covariates entered into the analysis were sex, SIAS-SPS total score and SADQ to control for initial levels of social anxiety and alcohol dependence and gender effects. This analysis was repeated with SIAS-SPS total score, tension reduction and social lubrication as covariates. In addition, SADQ and gender were also controlled for.

<div align="center">

Results

</div>

Descriptive statistics for symptom measures and measures of impulsivity.

Descriptive statistics obtained using SPSS version 22.0 software were used to characterise the sample (see Table 8) and check for univariate and multivariate non-normality. The groups did not significantly differ in mean age, number of comorbid DSM-IV diagnoses, latency from their last meal, number of alcohol drinking days in the past month, or the total number of standard drinks consumed in this time period (all p's > .10). A one-way between-groups ANOVA to assess initial differences of baseline alcohol urge to drink (AUQ) revealed no significant differences between the groups, $F(2,36) = .04$, $p = .96$, indicating that participants in the three conditions were not initially different in terms self-reported craving. These results indicate that random assignment was effective in producing similar groups.

Table 8

Descriptive statistics for social anxiety and alcohol symptom measures and measures of impulsivity

Measure	Total (N = 39)	
	M (SD)	Range
Social anxiety		
ADIS Social Anxiety CSR	5.78 (.92)	4 - 7
SIAS	49.79 (10.88)	21 - 73
SPS	33.67 (14.58)	10 - 69
Alcohol		
ADIS Alcohol CSR	5.66 (1.07)	3 - 8
AUDIT	24.41 (5.81)	8 - 33
SADQ	15.66 (11.46)	0 - 57
Impulsivity		
BAS-Drive[a]	8.91 (2.47)	4 - 14
BAS-Reward[a]	15.57 (2.32)	11 - 20
I_7-IMP[a]	9.43 (3.97)	4 - 20
IGT[b]	-1.75 (17.60)	-36 - 40

Note. ADIS = Anxiety Disorders Interview Schedule; CSR = Clinician Severity Rating; AUDIT = Alcohol Use Disorders Identification Test; SADQ = Severity of Alcohol Dependence Questionnaire; SIAS = Social Anxiety Interaction Scale; SPS = Social Phobia Scale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale; I_7-IMP = Impulsiveness subscale; IGT = Iowa Gambling Task.

[a] *n* = 35; [b] *n* = 36

Effect of beverage manipulation on self-reported craving, stimulant and sedative effects and physiological arousal.

A mixed model ANOVA with beverage condition (alcohol, placebo, control) as a between-subjects factor and time (pre-drinking, during-drinking, post-drinking) as within-subjects factors was used to examine ratings on the AUQ (see Table 9). The was a significant main effect of time, $F(2,34) = 7.72$, $p = .002$, and this effect was large ($\eta_p^2 = .31$). There was no significant interaction between time and beverage condition, $F(4,68) = 1.82$, $p = .14$; $\eta_p^2 = .10$ indicating that individuals in the three beverage groups did not differ in self-reported craving.

Post hoc tests revealed that self-reported craving post-drinking ($M = 30.81$, $SD = 12.05$) was significantly higher than craving during beverage consumption ($M = 25.97$, $SD = 10.22$) across the three beverage groups. Inspection of the bivariate relationships did not show any significant relationships between craving and any impulsivity or expectancy measures (see Table 10). Due to the lack of association with these variables on experimental condition, no further analyses were conducted on the AUQ.

Table 9

Measures of craving, stimulant and sedative effects and physiological variables by beverage group

	Alcohol (n = 12)			Placebo (n = 13)			Control (n = 14)		
	Pre-drinking	During-drinking	Post-drinking	Pre-drinking	During-drinking	Post-drinking	Pre-drinking	During-drinking	Post-drinking
AUQ Mean, (SD)	26.50 (12.28)	24.83 (11.54)	27.17 (13.00)	26.54 (11.59)	30.77 (10.70)	34.00 (11.63)	25.57 (7.05)	24.43 (7.93)	30.98 (11.53)
B-BAES STIM Mean, (SD)	14.00 (4.92)	-	13.50 (6.78)	11.54 (4.96)	-	9.77 (4.57)	9.38 (5.11)	-	7.46 (5.21)
B-BAES SED Mean, (SD)	6.75 (6.17)	-	11.92 (5.14)	8.54 (5.62)	-	14.00 (6.00)	8.46 (4.52)	-	11.77 (6.11)
SCL (µS) Mean, (SD)	26.40 (10.98)	28.27 (12.87)	29.87 (13.69)	17.60 (7.48)	16.45 (7.16)	17.46 (7.97)	27.09 (10.85)	24.95 (9.88)	24.50 (9.87)
HR (BPM) Mean, (SD)	71.41 (14.06)	75.81 (12.71)	77.21 (14.40)	73.01 (11.61)	74.30 (11.94)	75.84 (11.60)	69.34 (13.89)	68.36 (13.31)	69.64 (13.54)

Univariate & Multivariate Effects

Variable	η_p^2	F	p
AUQ			
Time	.31	$F_{(2,34)} = 7.72$	.002**
Time × Beverage Condition	.10	$F_{(4,70)} = 1.86$	.13
B-BAES STIM & B-BAES SED			
Time	.36	$F_{(2,34)} = 9.34$	.001**
Time × Beverage Condition	.03	$F_{(4,68)} = .57$	.68
SCL & HR			
Time	.31	$F_{(4,33)} = 3.69$	.01*
Time × Beverage Condition	.23	$F_{(8,66)} = 2.45$	.03*

Note. AUQ = Alcohol Urge Questionnaire; B-BAES STIM = Brief Biphasic Alcohol Effects Scale – Stimulation subscale; B-BAES SED = Brief Biphasic Alcohol Effects Scale – Sedation subscale; SCL = Skin conductance level (µS); HR = Heart rate; BPM = Beats per minute.

* $p < .05$; ** $p < .01$

A mixed MANOVA was used to examine ratings on the B-BAES Stimulation and B-BAES Sedation subscales measuring the subjective stimulant and sedative effects at baseline and at post-drinking (see Table 9 for means). There was a significant main effect of time $F(2,34) = 9.34$, $p = .001$, and this effect was large ($\eta_p^2 = .36$). However, there was no significant interaction between time and beverage condition, $F(4,68) = .57$, $p = .68$; $\eta_p^2 = .03$. To further investigate the significant main effect of time, separate univariate ANOVAs on the outcome variables were examined and post hoc tests conducted. A significant effect of time on the Sedation scores was found, $F(1,35) = 19.01$, $p < .001$; $\eta_p^2 = .35$, indicating that individuals reported higher levels of feelings of sedation post-drinking ($M = 12.58$, $SD = 5.72$) compared to the pre-drinking time point ($M = 7.95$, $SD = 5.37$). For Stimulation scores, a significant effect of time was also observed, $F(1,35) = 4.44$, $p < .05$; $\eta_p^2 = .11$, with slightly higher levels of stimulation reported at pre-drinking ($M = 11.58$, $SD = 5.22$) compared to post-drinking ($M = 10.16$, $SD = 5.95$).

A mixed MANOVA was also performed to assess changes in skin conductance and heart rate. Mean skin conductance level (SCL) and mean heart rate (BPM) across the experiment are shown in Table 9. The analysis revealed a significant main effect of time, $F(4,33) = 3.69$, $p = .01$; $\eta_p^2 = .31$, and a significant interaction between time and beverage condition, $F(8,66) = 2.45$, $p = .03$; $\eta_p^2 = .23$. Univariate tests showed significant and large interactions between time and beverage conditions for both skin conductance, $F(4,72) = 4.96$, $p = .001$; $\eta_p^2 = .22$ and heart rate, $F(4,72) = 3.30$, $p = .015$; $\eta_p^2 = .16$.

To further examine these interaction effects, a simple effects analysis was performed separately for skin conductance and heart rate to examine the effect of beverage condition on these physiological variables between the three time periods: pre-drinking, during-drinking and post-drinking. Simple effects analyses revealed that skin conductance levels increased from pre-drinking to during-drinking ($p = .05$), and from during-drinking to post-drinking ($p = .03$) for individuals in the alcohol group. Skin conductance decreased from pre-drinking to

during-drinking (p = .018), and from pre-drinking to post-drinking (p = .02) for those in the soft drink group. Examination of the means (Table 9) shows a decrease in SCL across the time periods for those receiving placebo alcohol, however, results from post hoc tests were non-significant (all p's > .16). There was also a significant difference observed between the alcohol and placebo groups at baseline (p = .03), and the placebo and soft drink groups at pre-drinking (p = .018), with individuals in the placebo group showing lower skin conductance than those receiving alcohol and soft drink. A similar pattern of results was also observed during-drinking, with the placebo group significantly different from the alcohol (p = .006) and soft drink groups (p = .036), and at post-drinking, with those receiving placebo showing lower levels of skin conductance compared to those receiving alcohol (p = .01).

Simple effects analyses revealed that heart rate increased from pre-drinking to during-drinking (p = .001), and from pre-drinking to post-drinking (p = .001) for individuals in the alcohol group. An increase in heart rate from the pre-drinking time point to the post-drinking time point (p = .06), and from during-drinking to post-drinking (p = .07) approached significance in those receiving placebo alcohol. Individuals receiving soft drink did not show any significant differences between any time points (all p's > .14). There was no significant difference in heart rate between the beverage conditions at any time point (all p's > .15)

Table 10

Summary of correlations for social anxiety, alcohol, impulsivity and outcome variables

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Social Anxiety																						
1. SIAS-SPS score	-	.15	.15	.28	.21	.28	.29	.24	-.08	.03	.07	.01	-.01	.20	-.14	.19	-.04	-.17	-.17	.08	-.03	-.06
Alcohol																						
2. TLFB drinks/day		-	.40*	.24	.12	.10	.31	.37*	.05	.29	.47**	.32	.26	.35*	-.01	.11	.07	.05	.04	.10	.12	.08
3. PE-AE			-	.26	.46**	.27	.31	.10	.05	.19	.35*	.29	.34*	.25	.08	.22	-.12	-.21	-.27	.05	.01	.01
4. TR-AE				-	.42**	-.19	-.17	-.05	-.15	.27	.02	-.10	-.20	-.39*	.02	-.02	-.12	-.18	-.26	.09	.10	.12
5. SL-AE					-	-.25	-.01	.01	-.24	.24	.34*	.32	.21	.00	.08	.27	.03	-.05	-.05	-.04	-.05	-.10
Impulsivity																						
6. BAS-Drive						-	.31	.57**	.24	-.05	.03	-.04	.24	.42**	-.41*	-.28	-.07	-.04	-.02	-.08	-.05	-.04
7. BAS-Reward							-	.26	.08	.05	.14	.16	.49**	.37**	-.18	.17	-.06	-.16	-.10	-.05	.05	.08
8. I7-IMP								-	.03	.17	.29	.16	.14	.28	.08	-.08	-.26	-.12	-.04	.35*	.40*	.36*
9. IGT Net score									-	-.09	-.08	-.05	-.17	-.02	-.01	-.11	-.10	-.26	-.24	-.18	-.17	.15
Outcome variables																						
10. Pre-drink AUQ										-	.58**	.43**	.02	-.02	.17	.33*	-.21	-.25	-.28	.14	.13	.11
11. During-drink AUQ											-	.83**	.05	.14	.32	.39**	-.06	-.07	-.03	.13	.05	.003
12. Post-drink AUQ												-	-.04	.12	.35*	.27	-.07	-.07	-.05	.16	.09	.04
13. Pre-drink STIM													-	.75**	-.34*	.12	.06	.11	.15	.03	.11	.10
14. Post-drink STIM														-	-.15	-.03	.13	.26	.29	.03	.11	.09
15. Pre-drink SED															-	.32*	-.14	-.07	-.06	.16	.15	.15
16. Post-drink SED																-	-.11	-.07	-.06	.16	.11	.07
17. Pre-drink SCL																	-	.85**	.76**	-.19	-.16	-.13
18. During-drink SCL																		-	.95**	-.04	.03	.03
19. Post-drink SCL																			-	-.12	-.04	-.03
20. Pre-drink HR																				-	.93**	.90*
21. During-drink HR																					-	.97*
22. Post-drink HR																						-

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; TLFB: Time Line Follow-Back; PE-AE = Performance Enhancement Alcohol Expectancy; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social Lubrication Alcohol Expectancy; BAS-Drive = Behavioural Activation System – Drive subscale; BAS-Reward = Behavioural Activation System – Reward Responsiveness subscale; I7-IMP = Impulsiveness subscale; IGT = Iowa Gambling Task; AUQ = Alcohol Urge Questionnaire; STIM = Brief Biphasic Alcohol Effects Scale – Stimulation subscale; SED = Brief Biphasic Alcohol Effects Scale –Sedation subscale; SCL = Skin conductance level; HR = Heart rate.

* $p < .05$; ** $p < .001$

Relationship between impulsivity, alcohol expectancies, stimulant effects and physiological arousal.

Bivariate correlations among covariates and outcome variables at the pre-drinking, during-drinking and post-drinking time points are shown in Table 10. Both facets of reward drive were positively associated with subjective stimulant effects while the trait measure of rash impulsivity, the I_7-IMP, was associated with higher levels of heart rate at all time points. We did not include the IGT in any analyses as no significant associations were observed for this variable.

An ANCOVA was performed to test the hypothesis that reward drive and performance enhancement expectancies would be associated with greater subjective stimulant effects. Results revealed that BAS-Drive ($p = .04$), but not BAS-Reward was significantly related to stimulation effects (see Table 11). To illustrate these effects, the BAS-Drive variable was split into a 2-categorical variable (median split; see Figure 2). Examination of this figure shows that individuals high in reward drive (i.e., BAS-Drive) reported greater subjective stimulant effects at the two time points compared to individuals low in BAS-Drive.

Table 11

Analysis of covariance results examining the impact of impulsivity and alcohol expectancies on stimulant effects and physiological arousal

	F(df)	p	η_p^2
Effects on Stimulation			
Primary variables			
Time	$F(1,25) = .5.05$	.34	.04
Time × BAS Drive	$F(1,25) = 4.50$	.04*	.16
Time × BAS RR	$F(1,25) = .98$	.33	.04
Time × PE-AE	$F(1,25) = 2.20$	.15	.08
Additional covariates			
Time × Sex	$F(1,25) = 2.90$	.10	.10
Time × SIAS-SPS total score	$F(1,25) = 2.79$	.11	.10
Time × SADQ	$F(1,25) = 1.27$	.27	.05
Time × Condition	$F(2,25) = .13$	.97	.01
Effects on HR			
Primary variables			
Time	$F(2,24) = .58$	.57	.05
Time × PE-AE	$F(2,24) = 3.07$	.07	.20
Time × BAS-Drive	$F(2,24) = .22$	.81	.02
Time × BAS-Reward	$F(2,24) = 5.03$	.02*	.30
Time × I$_7$-IMP	$F(2,24) = .44$	.65	.04
Time × Condition	$F(4,48) = 1.51$	.22	.11
Additional covariates			
Time × Sex	$F(2,24) = .12$	.89	.01
Time × SIAS-SPS total score	$F(2,24) = 3.95$	.03*	.25
Time × SADQ	$F(2,24) = .55$	.58	.04
Effects on GSR			
Primary variables			
Time	$F(2,24) = .07$	.94	.01
Time × PE-AE	$F(2,24) = 11.37$	.27	.10
Time × BAS-Drive	$F(2,24) = .19$	.83	.02
Time × BA-Reward	$F(2,24) = 83$	.45	.06
Time × I$_7$-IMP	$F(2,24) = .66$	.53	.05
Time × Condition	$F(4,48) = 1.55$	.21	.11
Additional covariates			
Time × Sex	$F(2,24) = .43$	.65	.04
Time × SIAS-SPS total score	$F(2,24) = .16$	.85	.01
Time × SADQ	$F(2,24) = .11$	.90	.01

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; PE-AE = Performance Enhancement Alcohol Expectancy; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale; I$_7$-IMP = Impulsiveness subscale; SCL = Skin conductance level; HR = Heart rate.

* $p < .05$
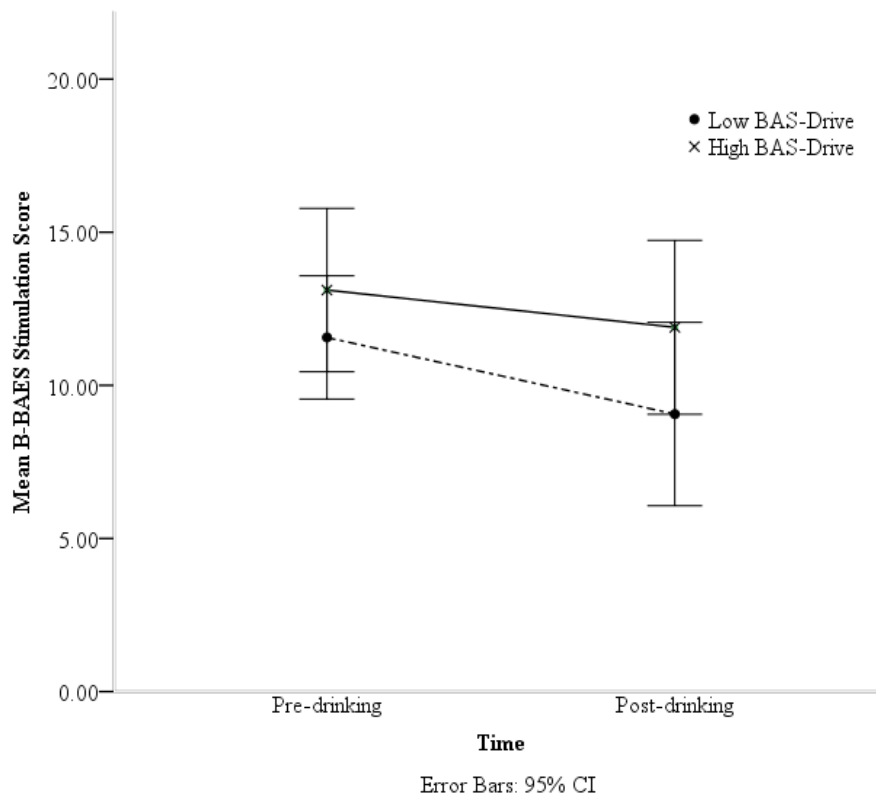
Figure 2. Mean subjective stimulation scores over time for comorbid SAD-AUD individuals with high and low levels of reward drive (BAS-Drive) ($N = 35$).

Similarly, to examine the impact of impulsivity on physiological arousal, separate ANCOVAs were conducted for heart rate and skin conductance level at the pre-drinking and post-drinking time points and the same covariates used previously with the addition of I_7-IMP. Only two covariates, social anxiety (SIAS-SPS) and BAS-Reward (both $p < .05$) were significantly associated with heart rate. Figures 3 and 4 depict mean heart rate over time by BAS-Reward and SIAS-SPS respectively. BAS-Reward and SIAS-SPS were split into a 2-categorical variable so the pattern of findings for these continuous variables can be interpreted.

Examination of Figure 3 illustrates that individuals with higher reward drive as measured by BAS-Reward (median split) exhibited higher heart rate during-drinking and post-drinking compared to individuals low in BAS-Reward. Examination of Figure 4 shows that individuals with higher social anxiety (median split) showed lower mean heart rate during-drinking and post-drinking compared to individuals with low social anxiety. No significant covariates were found for skin conductance level.

Figure 3. Mean heart rate over time for comorbid SAD-AUD individuals with high and low levels of reward drive (BAS-Reward) (*N* = 35).

Figure 4. Mean heart rate over time for comorbid SAD-AUD individuals with high and low levels of social anxiety (SIAS-SPS total score) (*N* = 39).

Relationship between social anxiety, alcohol expectancies, sedative effects and physiological arousal.

To test the hypothesis that social anxiety, tension reduction and social lubrication expectancies would be associated with greater feelings of sedation, an ANCOVA was performed. The results for this analysis are shown in Table 12. There was a significant interaction between the measure of social anxiety and time ($p = .03$). No other covariates contributed significantly to the model. To illustrate these effects, the SIAS-SPS total score variable was split into a 2-categorical variable (median split; see Figure 5). Examination of this figure shows that individuals with high social anxiety reported greater subjective sedation effects at the post-drinking time point compared to those with low social anxiety.

Separate ANCOVAs were conducted to examine the relationship with the above covariates and physiological arousal. Results in Table 12 show no significant relationship for any covariates for heart rate or skin conductance level (all p's > .11).

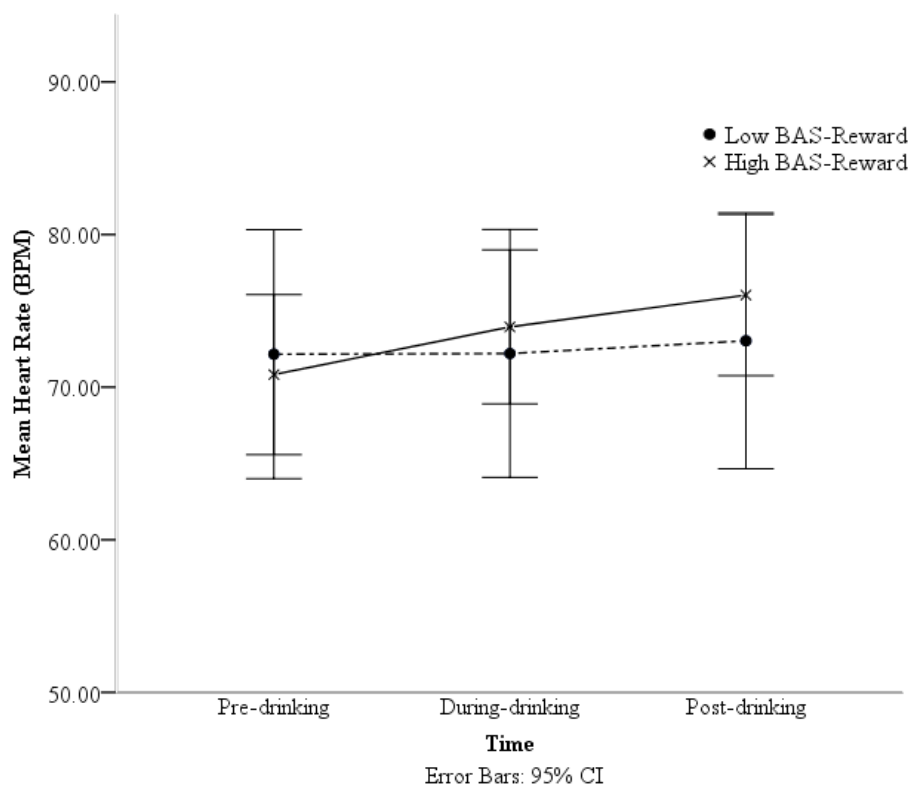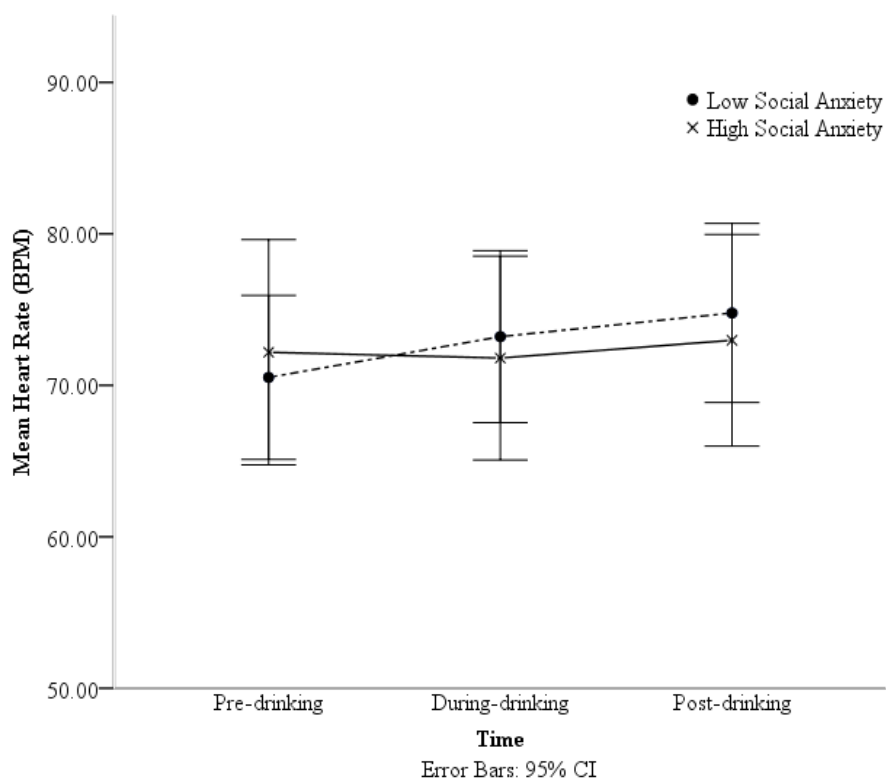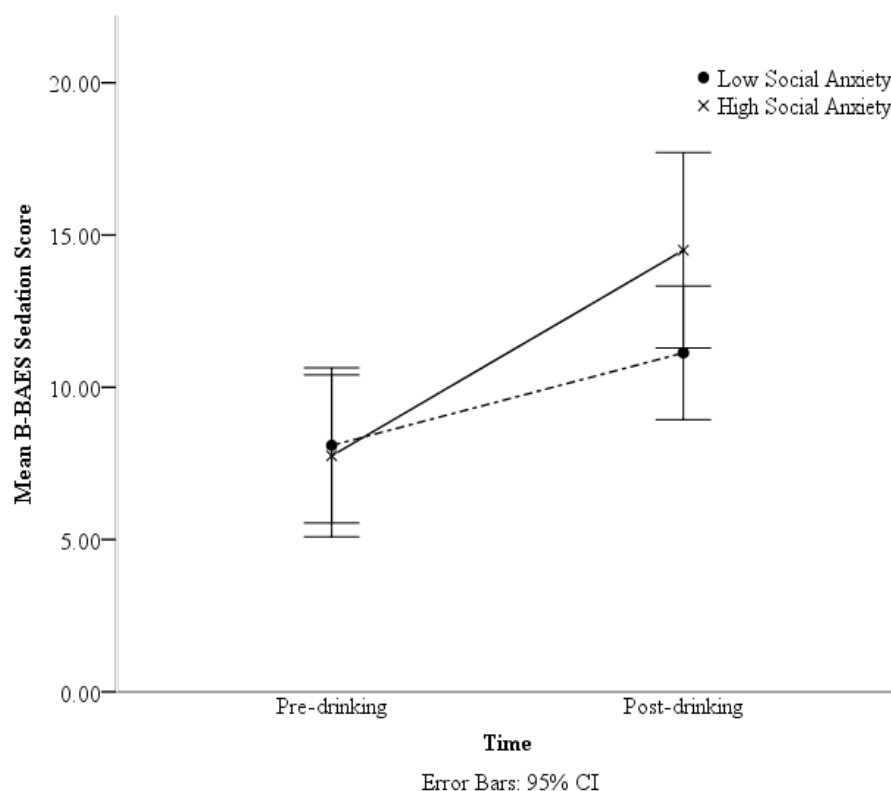*Figure 5.* Mean subjective sedation scores over time for comorbid SAD-AUD individuals with high and low levels of anxiety (SIAS-SPS total score) ($N = 39$).

Table 12

Analysis of covariance results examining the impact of social anxiety, alcohol expectancies on sedation effects and physiological arousal

	F(df)	p	η_p^2
Effects on Sedation			
Primary variables			
Time	$F(1,29) = .01$	.94	.00
Time × SIAS-SPS total score	$F(1,29) = 5.06$	.03*	.15
Time × TR-AE	$F(1,29) = 2.75$	.11	.09
Time × SL-AE	$F(1,29) = .27$	.61	.01
Time × Condition	$F(2,29) = .63$	.54	.04
Additional covariates			
Time × Sex	$F(1,29) = 1.51$	.23	.05
Time × SADQ	$F(1,29) = .27$	.61	.01
Effects on HR			
Primary variables			
Time	$F(2,29) = .43$	.65	.03
Time × SIAS-SPS total score	$F(2,29) = 1.24$	.31	.08
Time × TR-AE	$F(2,29) = .87$	.43	.06
Time × SL-AE	$F(2,29) = 2.08$	.14	.13
Time × Condition	$F(4,58) = 1.75$	.15	.11
Additional covariates			
Time × Sex	$F(2,29) = .49$	.62	.03
Time × SADQ	$F(2,29) = .01$	.99	.001
Effects on GSR			
Primary variables			
Time	$F(2,29) = .03$	.97	.002
Time × SIAS-SPS total score	$F(2,29) = .34$	.72	.02
Time × TR-AE	$F(2,29) = 2.16$	.13	.13
Time × SL-AE	$F(2,29) = .24$	.79	.02
Time × Condition	$F(4,58) = 1.98$	.11	.12
Additional covariates			
Time × Sex	$F(2,29) = .27$	.77	.02
Time × SADQ	$F(2,29) = .12$	.89	.01

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; TR = Tension Reduction Alcohol Expectancy; SL = Social Lubrication Alcohol Expectancy; SCL = Skin conductance level; HR = Heart rate.

* $p < .05$

Discussion

This study examined physiological and subjective responses to alcohol's pharmacological and expectancy effects, and their association with impulsivity and alcohol expectancy in a sample of treatment-seeking individuals with co-occurring social anxiety and alcohol problems. Interesting effects were found between impulsivity; specifically, reward drive, subjective stimulant effects and physiological reactivity. A strong association was also observed between social anxiety and the sedative effects of alcohol.

Relationship between impulsivity, alcohol expectancies, stimulant effects and physiological arousal

Our analysis of the relationship between impulsivity, performance enhancement expectancies and subjective stimulant and sedative effects suggests that individuals with higher levels of reward drive and reward responsiveness (both components of reward sensitivity or "reward drive") report greater stimulating effects from exposure to alcohol and alcohol-related cues. Specifically, higher levels of reward drive was associated with greater stimulant effects at the pre-drinking time point and following consumption of a beverage. Reward drive also correlated negatively with levels of sedation. These findings are somewhat consistent with those reported by Leeman et al. (2014), who found that impulsive individuals described increased stimulant and lower sedative effects to alcohol.

Heart rate reactivity was also found to be positively related to self-reported impulsivity. Specifically, the trait measure of rash impulsivity, I_7-IMP, showed a positive association with increased heart rate prior to drinking, during drinking and following consumption of beverages. Similarly, higher reward drive (as measured by reward responsiveness) was also significantly related to increases in heart rate which is in line with previous studies that found an association between novelty seeking and heart rate reactivity (e.g., Brunelle et al., 2004). Increased heart rate reactivity has been shown to be directly proportional to the reinforcing properties of a stimulus (Fowles, 1983), and is believed to act as an indicator of alcohol-elicited reward (Brunelle, Barrett, & Pihl, 2006).

These results suggest that impulsive individuals may experience enhanced reinforcing stimulant effects and increased heart rate from receiving alcohol, placebo alcohol or interestingly, even soft-drink, in a typical drinking environment. It has been proposed that trait impulsivity affects learning by producing a bias in which the positive outcomes (e.g., feeling energised, excited) of a prior drinking occasion (actual or observed) are more likely to be retained in memory, subsequently influencing the likelihood of future alcohol consumption

(Gullo et al., 2010). Increased sensitivity to stimulating effects of a beverage in a bar environment may therefore provide a strong motivation to drink, and could lead to or maintain problematic alcohol use. Thus, a relationship between impulsivity and stimulant effects has implications for our understanding of factors that may place individuals at continued risk for heavy drinking and negative consequences.

In contrast to the other measures of impulsivity, the Iowa Gambling Task, a behavioural measure of rash impulsivity, did not show relationships with any variables included in the study. It may be that behavioural and self-report measures assess somewhat disparate aspects of rash impulsivity and/or were not sensitive or appropriate to detect any relationship with subjective and objective responses to alcohol. For instance, the IGT may reflect state-like differences in impulsivity as opposed to longer-term behavioural tendencies and this may engage a different set of cognitive processes and executive functions (Verdejo-Garcia et al., 2008). Furthermore, rash impulsivity is proposed to reflect individual differences in decision-making and impulse control, thus it may play less of a role in subjective and physiological response to alcohol, placebo alcohol and alcohol-related stimuli; instead, influencing actual drinking behaviour (Dawe & Loxton, 2004).

Relationship between social anxiety, alcohol expectancies, sedative effects and physiological arousal

In the current study, self-reported feelings of sedation increased as a function of time, with higher levels of sedation reported after beverage consumption compared to baseline. As hypothesised, the social anxiety symptom score, but not tension reduction or social lubrication expectancies, was significantly related to subjective sedative effects. Social anxiety was also a negative predictor of increases in stimulant effects as assessed by heart rate. Higher social anxiety was associated with lower heart rate during and following consumption of a beverage. This relationship could represent a different pathway to alcohol use compared to that of impulsivity. For example, the consumption of the beverages and exposure to alcohol-related

cues may have induced feelings of sedation that parallel those of anxiety reduction. Specifically, subjective feelings of sedation and slowed thinking processes may be associated with feelings of dampened anxiety, aspects of which may be perceived positively. These feelings are in direct contrast to typical symptoms of anxiety such as feelings of panic, fear, racing thoughts and heart palpitations, and may thus act as negative reinforcers. This may explain why social anxiety is commonly linked to the risk of developing alcohol problems; as heightened anxiety may be associated with greater sensitivity to reinforcing sedation effects.

Alcohol expectancies, pharmacological and expectancy effects

Contrary to our expectations, limited relationships were observed between alcohol expectancies and any measure of impulsivity, social anxiety or subjective stimulant and sedative effects. There are some notable differences between earlier studies that have found these relationships (e.g., Booth & Hasking, 2009; Gullo et al., 2010) and the current study. Firstly, differences in the measures used to assess alcohol expectancies and impulsivity and the different samples utilised may explain these inconsistent results. Specifically, the scales used by Gullo et al. (2010) to measure positive alcohol expectancy included several scales assessing tension reduction, social facilitation, sex and fun. Further, Gullo et al. also utilised a second expectancy questionnaire comprising six scales (68 items), suggesting that other factors in addition to performance enhancement may influence the relationship between reward drive and hazardous alcohol use (Gullo et al., 2010). Secondly, Gullo et al. examined the relationships between these variables in association with hazardous alcohol use as the outcome variable, while we were interested in the relationship between these variables and responses to alcohol consumption or perceived consumption. Moreover, while it is widely accepted that alcohol expectancies develop well before tangible encounters with drinking, it is also true that they change over time as a result of drinking experiences (e.g., Young, Connor, & Feeney, 2011). The current study included a treatment-seeking clinical sample, and the high level of alcohol dependence and its interaction with social anxiety could indicate that

alcohol expectancies may regulate drinking behaviour via a different mechanism. Here, the crucial factor may not be the presence of strong expectations, but whether other situational variables match or exceed those expectations (Morris et al., 2005).

In addition to personality and cognitive factors, the experimental design used in this study allowed for the separation of contextual factors from expectancy and pharmacological effects which may have influenced reactivity to alcohol and alcohol-related stimuli. The effects of actually ingesting alcohol were most prominent physiologically, and lead to increased arousal. Here, differences in autonomic reactions were observed as a function of type of beverage consumed. Skin conductance increased significantly during beverage consumption compared to the pre-drinking time point for individuals receiving alcohol and not in those receiving placebo alcohol and soft drink, suggesting that this increase in skin conductance level is due to the pharmacologic properties of alcohol. In addition to changes in skin conductance levels, alcohol consumption has also been found to increase heart rate (Brunelle, Barrett, & Pihl, 2007). In the current study, heart rate increases following alcohol consumption were observed in line with previous studies (e.g., Brunelle et al., 2007). However, there was weak evidence showing an increase in heart rate from pre-drinking to during-drinking and from consumption of the beverage to post consumption in individuals consuming placebo alcohol. Together, these results suggest that both ingestion of alcohol and the belief that one was receiving alcohol increased heart rate. These findings highlight the potential usefulness of including both of these indices to disentangle expectancy versus actual ingestion effects.

In contrast to physiological responses, craving and subjective stimulation/sedation effects appear to be unrelated to actual or perceived alcohol consumption and may instead be induced by contextual cues. The lack of time by beverage group effects in self-reported craving was unexpected and may have been due to the contextual drinking cues present in the laboratory environment. The current study attempted to make the laboratory "bar" environment as naturalistic as possible and may have stimulated subjective craving in all participants, even

those who received no alcohol. Proponents of classical conditioning argue that the repetitive associations between stimuli and the reinforcing effects of alcohol attain incentive value and subsequently trigger appetitive reactions, even in the absence of alcohol consumption (Drummond, 2000; Robinson & Berridge, 1993). Consistent with this, Field and Cox (2008) argue that drug-related stimuli initially elicit anticipation for the substance, and this expectation leads to craving.

Surprisingly, participants receiving alcohol did not report an increase in stimulant effects from pre-drinking to post-drinking. The questionnaires following consumption of the beverage were timed to correspond with the ascending limb of the BAC curve. However, it is possible that not enough time was allowed during the absorption phase as the drinks were consumed within a relatively short interval (15 minutes). It is also possible that the alcohol dose was not high enough. For instance, the inclusion of individuals with social anxiety and high levels of alcohol use may have caused restriction in range problems as all participants were drinkers with quite severe alcohol problems. Specifically, the high levels of alcohol problems (including hazardous consumption) reported by individuals in the current study may have led to the development of tolerance. That is, exposure to the dose of alcohol in the current study may not have been sufficient in eliciting heightened stimulant effects for those in the alcohol condition. Alternatively, the absence of craving and stimulant effects experienced between individuals receiving alcohol, placebo or soft drink could be related to a deficient self-regulatory system (Schuckit, 1994). For instance, individuals may have failed to receive sufficient feedback regarding their level of intoxication due to the severe nature of alcohol problems experienced by the current sample; subsequently resulting in fewer craving and stimulant effects being reported

Strengths and Limitations

The present study has a number of strengths and limitations. To our knowledge, this study is the first to consider the combination of self-report and objective reactivity to alcohol in

conjunction with personality variables such as impulsivity, and cognitive processes such as alcohol expectancy. In addition, the inclusion of a placebo condition allowed differentiation of the pharmacologic and expectancy effects of alcohol. The sampling design utilised in the present study represents both a strength and a limitation. Prior research involving alcohol administration has typically excluded individuals with alcohol use disorders due to concerns that alcohol consumption would have adverse consequences on their condition or motivation for treatment (Wolitzky-Taylor et al., 2011). These exclusions have therefore resulted in an unrepresentative sample, usually a non-clinical student population, and thus limits generalising them to the clinical context. It also underscores the importance of conducting research with clinical samples, in which these personality and cognitive factors may play a more prominent role in alcohol use. However, due to the difficulties inherent in recruiting clinical samples, our small sample size, although representative of alcohol administration studies, may have been insufficient to detect differences between the conditions. One way to address this in future studies would be through the use of a within-subjects design. However, issues regarding response fatigue, carryover effects and administering placebo and alcohol to the same individual warrant further consideration.

In addition, the values for skin conductance for individuals randomly allocated to the placebo group were as much as a standard deviation lower at pre-drinking which could reflect a failure of the randomisation due to low sample size. Therefore, these results should be interpreted with caution. Replication in larger samples may provide more stable estimates of associations between cue responses (subjective and objective measures) and indices of appetitive motivation (or impulsivity and alcohol expectancy). Finally, associations between alcohol expectancies, social anxiety and impulsivity may present differently in other studies due to the inclusion of positive reinforcement expectancy measures, and a distinction should be made between negative reinforcement expectancies and positive reinforcement expectancies in future studies.

Conclusion

Despite these limitations, our results replicate and expand the current understanding of how cues associated with alcohol and actually receiving or expecting alcohol contribute to maintaining dependence via two separate pathways. The first pathway suggests that traits related to reward drive and rash impulsivity may convey risk for maintaining hazardous drinking via increased sensitivity to subjective stimulating effects and physiological responding. Our findings also suggest a different pathway may exist that contributes to continued alcohol use via increased sensitivity to subjective sedative effects as severity of social anxiety increases. This may explain the social anxiety and alcohol use disorders association, as greater social anxiety is linked to greater sensitivity to reinforcing sedation effects. Finally, our results suggest that it is not only the pharmacological effects and subjective stimulant and sedative effects that may elicit craving, but also the expectation to receive alcohol and being present in a typical drinking environment surrounded by alcohol cues. Thus, the assessment of these variables as risk markers may be a valuable tool which can inform prevention efforts though improved identification of individuals at risk, or assist in informing treatment such as cue exposure therapies. The long-term implications of impulsivity and alcohol expectancies and their prediction in relapse and treatment outcomes should be addressed in future work.

Chapter Five

Impulsivity as a Predictor of Treatment Outcome in Individuals with Comorbid Social

Anxiety and Alcohol Use Disorders

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Abstract

Social anxiety is frequently comorbid with alcohol use disorders and their co-occurrence often results in less effective treatment outcomes. The purpose of this study was to examine different components of impulsivity for their utility in predicting outcome of Motivational Interviewing and Cognitive Behavioural Therapy (MI/CBT) among individuals with comorbid social anxiety disorder and alcohol use disorders. A subset of participants ($N = 60$) part of a larger clinical trial assessing the effectiveness of an integrated MI/CBT for alcohol problems and social anxiety or MI/CBT for alcohol problems only were included. Random effects regression was used to examine the association between impulsivity and the rate of improvement in four treatment outcomes: social anxiety symptoms, severity of alcohol dependence, number of alcohol drinks consumed per day as well as number of drinking days. Higher trait rash impulsivity was associated with better social anxiety outcomes controlling for other variables. The impact of impulsivity on alcohol-related outcomes was mixed, with weak evidence suggesting that individuals high in rash impulsivity at baseline improved to a greater extent over time in severity of alcohol dependence. Additionally, baseline impulsivity was predictive of better treatment outcomes in quantity, but not frequency, of alcohol use. In contrast, alcohol expectancies did not impact the degree of change over time for any social anxiety or alcohol outcome measure. Understanding the impact of impulsivity on treatment outcomes may have prognostic utility and our results suggest that individuals with co-occurring social anxiety and alcohol use disorders, high on impulsivity, may particularly benefit from the skills imparted through CBT.

Impulsivity as a Predictor of Treatment Outcome in Individuals with Comorbid Social

Anxiety and Alcohol Use Disorders

There is a well-documented association between social anxiety disorder (SAD) and alcohol use disorders (AUDs). Clinical and epidemiological studies consistently show an increased prevalence of SAD among individuals with AUDs compared with those with no AUDs, and an increased likelihood of AUDs among those with a primary diagnosis of SAD compared to those without SAD (Burns & Teesson, 2002; Grant et al., 2004; Kessler et al., 1997; Merikangas, Stevens, et al., 1998). The presence of anxiety disorders is associated with poorer alcohol use disorder outcomes, such as greater relapse, increased drop-out rates and greater long-term alcohol consumption compared to individuals with an AUD and no comorbid anxiety diagnosis (Morley, Baillie, Sannibale, Teesson, & Haber, 2013; Wolitzky-Taylor et al., 2011). In addition, individuals reporting this comorbidity are significantly more disabled and utilise health services more often than those with an AUD and no SAD (Teesson et al., 2009). Although there are numerous efficacious interventions for both anxiety disorders and alcohol use problems separately, relatively few interventions have been conducted and evaluated for individuals with comorbid anxiety and alcohol use problems. To date, only two randomised controlled trials (RCTs) have explored the efficacy of treating co-occurring social anxiety and alcohol use disorders (e.g., Randall et al., 2001; Schadé et al., 2005), and evaluation of these treatments showed no clear advantage for treatment focussed on the comorbidity over treatment for alcohol alone. Moreover, although these treatments lead to significant improvement for a large portion of individuals, a significant number of individuals remain symptomatic following treatment. The identification of factors influencing treatment outcomes offers substantial benefits for improving the delivery of treatment, and has the potential to greatly reduce the burden of these disorders and alleviate suffering at an individual level. Thus, research has increasingly attempted to detect early markers or risk factors for treatment outcomes.

The identification of predictors of alcohol and social anxiety treatment outcome serves not only to recognize underlying factors that perpetuate this relationship, but also to delineate subgroups for which specific treatments may need to be developed. To date, a wide range of baseline client characteristics have been examined and identified as potentially relevant to alcohol use treatment outcomes, including socio-demographic (e.g., gender), drug-related (e.g., alcohol dependence severity), cognitive (e.g., alcohol expectancies) and psychosocial (e.g., personality) factors (Adamson, Sellman, & Frampton, 2009; King & Canada, 2004; Loree et al., 2015). Recently, there has been increasing recognition of the importance of individual factors such as impulsivity in initiating and maintaining substance use, and predicting treatment outcomes. For example, several studies have indicated that impulsivity may be an important variable in predicting treatment outcomes and relapse risk among alcohol abusing individuals (e.g., Charney et al., 2010; Stevens et al., 2014). Further, a recent review by Loree et al. (2015) found that higher pre-treatment impulsivity is generally associated with poorer treatment outcomes; specifically, increased alcohol consumption and shorter time to relapse. Thus, impulsivity appears to be a key predictor of alcohol use treatment outcomes and warrants more attention in the improvement of treatment response.

Impulsivity may play a role in the SAD-AUD relationship and in the treatment response of these individuals. The term impulsivity has recently been defined by two factors, reward drive and rash impulsivity which may influence alcohol use through different pathways (2-CARS model; Dawe et al., 2004). Specifically, these traits are associated with heightened sensitivity to rewarding stimuli (i.e., reward drive) and to behavioural disinhibition, or lack of control over behavioural responses (i.e., rash impulsivity; Dawe et al., 2004). Results from laboratory studies have shown an association between reward drive and a bias to attend and respond to drug cues and drug-related stimuli (e.g., Franken, 2002; Kambouropoulos & Staiger, 2001, 2004a). Dependent drinkers with and without comorbid social anxiety disorder scored higher on trait measures of impulsivity and showed greater increases in reactivity to alcohol stimuli

compared to non-dependent drinkers with and without this comorbidity (Study 2, Chapter 3).

Higher levels of craving assessed in cue reactivity have been associated with worse outcomes

in individuals with alcohol use problems such as higher relapse and dropout rates (e.g.,

Cooney et al., 1997; Miller, Westerberg, Harris, & Tonigan, 1996). In addition, longitudinal

studies have found that disinhibition/rash impulsivity in childhood predict increased risk of

problematic substance use in later adolescence and early adulthood (McGue et al., 2001;

Tarter et al., 2004), and predict alcohol dependence severity into adulthood (Tracy, 1994).

Addiction severity, in turn, has been consistently linked to worse treatment outcomes

(Adamson et al., 2009; Charney et al., 2010). Indeed, impulsivity and drug severity at

admission predicted poorer treatment outcome three months following completion of

treatment in 144 adult substance users (Staiger, Dawe, Richardson, Hall, & Kambouropoulos,

2014).

More recently, reward drive has been linked to problematic alcohol use via alcohol

expectancies (Gullo et al., 2010). Alcohol expectancies are both positive and negative beliefs

about the consequences of drinking (Brown et al., 1980), and have been associated with levels

of alcohol drinking (Sher et al., 1996), and poorer treatment outcomes (Jones, Corbin, &

Fromme, 2001). Individuals with high reward drive are believed to experience greater

rewarding effects of alcohol while intoxicated (Brunelle et al., 2004; Dawe et al., 2004), thus

strengthening positive expectancies and resulting in increased motivation to drink.

Alcohol expectancies and reward sensitivity also appear to play a role in the relationship

between social anxiety and alcohol use, with evidence to suggest that these factors moderate

the relationship (Booth & Hasking, 2009). Specifically, a positive relationship between one

component of social anxiety (e.g., fear) and alcohol use was observed for individuals with

high reward drive who reported strong tension reduction expectancies. The authors argue that

individuals predisposed to reward, who approach social situations with fear and have

expectations that drinking will help them cope, may subsequently consume more alcohol.

However, our own research found limited relationships between alcohol expectancies and any measure of impulsivity (i.e., reward drive and rash impulsivity), in a sample of treatment-seeking individuals with comorbid SAD-AUD (Study 3, Chapter 4). Rather, a relationship was observed between impulsivity and subjective effects of consuming alcohol, with comorbid individuals who reported greater impulsivity reporting more stimulating effects as well as increased heart rate (Study 3, Chapter 4). Thus, higher levels of impulsivity may help account for the poorer outcomes in individuals with comorbid SAD-AUD via increased subjective and physiological responses to alcohol.

Despite evidence of the role that impulsivity plays in problematic alcohol use and, more recently, social anxiety and alcohol use comorbidity, no study has examined the impact of impulsivity on the treatment outcomes for individuals with these comorbid disorders. Given the key role of impulsivity in the pathophysiology of alcohol use disorders, and the possible interaction with alcohol expectancies and subjective responses following alcohol consumption in individuals with co-occurring social anxiety, this paper examines the relationship between aspects of impulsivity and treatment outcomes in individuals with comorbid social anxiety and alcohol use disorders. Understanding the relationship between patient characteristics, intervention and outcome in this population is crucial for identifying the key elements of useful treatments and developing more effective treatment strategies.

In this study, we used data collected from a randomised controlled treatment trial to examine the association between impulsivity; specifically, trait reward drive and rash impulsivity, and social anxiety and alcohol outcomes following treatment. In line with previous studies, we examined the predictive value of gender and baseline social anxiety severity, alcohol dependence severity, and number of alcohol drinks per drinking day, on treatment outcome. In addition, we explored additional variables associated with alcohol use, social anxiety and impulsivity, such as alcohol expectancies. Based on previous research and our own research showing increased reactivity in comorbid individuals, it was predicted that

higher impulsivity would be associated with poorer alcohol and social anxiety outcomes during the first six months following the end of the treatment program. Specifically, higher trait rash impulsivity and reward drive were hypothesised to be associated with fewer reductions in alcohol dependence severity, number of drinks per day, number of drinking days and social anxiety symptoms across all three time points following the completion of a treatment program. Further, alcohol expectancies were included to investigate whether there were independent effects of impulsivity on treatment outcome that are not driven by alcohol expectancies.

Method

Participants

This study included a subset of participants ($N = 60$) part of a larger randomised controlled trial assessing the effectiveness of an integrated Motivational Interviewing (MI) and Cognitive Behavioural Therapy (CBT) intervention for comorbid social anxiety and alcohol use disorders compared with MI and CBT for the alcohol use disorder alone (see Baillie et al., 2013 for more details). Participants ranged in age from 20 to 65 years ($M = 37.12$, $SD = 11.35$), and 38.3% were female. For inclusion in the trial, participants met Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) criteria for social anxiety disorder and alcohol abuse or dependence as determined by the Anxiety Disorders Interview Schedule – IV (ADIS-IV; Di Nardo et al., 1994). Adequate cognition and English-language skills were also required to provide valid consent and complete research interviews and tasks. Participants were excluded from the trial if there was evidence of active suicidal intent, comorbid psychosis, severe cluster A or B personality disorders, dependence on benzodiazepines or substances other than alcohol and tobacco, current injecting drug use or need for intensive detoxification (a score >20 on CIWA-AR; Sullivan et al., 1989). Participants were randomly allocated to one of two treatment conditions: $n = 32$ received the integrated treatment for both alcohol use and social anxiety

disorder, while $n = 28$ received treatment for alcohol only although they also had a diagnosis of social anxiety disorder. Treatment was delivered at two treatment sites, either the Macquarie University Centre for Emotional Health or the Royal Prince Alfred Hospital Drug Health Services.

The majority of participants in the current study were born in Australia (85%), never married (51.6%) and in full-time employment (58.3%). Less than half were university educated with a Bachelor's degree or higher (38.3%). There were no baseline differences between the two treatment groups except for a significant difference in gender, (Alcohol Only: 53.6% female, Integrated: 25% female), $\chi^2(1, N = 60) = 5.16$, $p = .034$; thus gender was included as a covariate in analyses.

Materials/Measures

Clinician rated diagnostic severity. Participants were interviewed using the ADIS-IV (DiNardo et al., 1994). Diagnoses (used to determine inclusion/exclusion in the clinical trial) and clinician severity ratings were assigned by graduate clinical psychology students or registered clinical psychologists. Clinician severity ratings ranged from 0 to 8, where a score of 4 or more indicates clinically significant interference, and were used to assess diagnostic severity of social anxiety and alcohol abuse/dependence. Data from our laboratory using this interview has indicated a very high inter-rater reliability for the clinical severity score for both SAD ($\kappa = .94$) and AUD ($\kappa = .90$).

Primary outcome measures. Primary outcome measures for alcohol were the Severity of Alcohol Dependence Questionnaire (SADQ; Stockwell et al., 1994), and number of standard alcoholic drinks per drinking day, and number of drinking days as assessed using the timeline follow-back (TLFB) method (Sobell & Sobell, 1992). The primary outcome measure for social anxiety disorder was a composite score derived from the Social Phobia Scale and Social Interaction Anxiety scales (Mattick & Clarke, 1998).

Symptom measures of social anxiety and alcohol dependence.

Severity of social anxiety. The Social Interaction Anxiety Scale and the Social Phobia Scale (SIAS, SPS; Mattick & Clarke, 1998) were used to assess social anxiety. The SIAS contains 20 items on which respondents rate their experiences in social situations associated with social anxiety and social phobia DSM-IV criteria. The SPS consists of 20 items that describe anxiety in situations in which the person is the focus of attention and observed by others (e.g., public speaking, eating, writing). Items are rated on a 5-point scale ranging from "Not at all like me" (0) to "Extremely characteristic or true of me" (4). The total score ranges from 0 to 80 for each scale, where higher scores indicate greater anxiety in social interactions and greater anxiety about being observed by others respectively. These components also load onto a single higher order factor of social anxiety. Both the SIAS and the SPS have been found to have excellent psychometric properties, and to discriminate between socially phobic, normal, and other phobic populations (Mattick & Clarke, 1998; Peters, 2000). Internal consistency was good ($\alpha = .87$; SIAS) and excellent ($\alpha = .93$; SPS) in the current sample.

Alcohol dependence. The Severity of Alcohol Dependence Questionnaire (SADQ; Stockwell et al., 1994) is a self-administered, 20-item questionnaire designed to measure the presence and severity of alcohol dependence. There are five subscales with four items in each: physical withdrawal symptoms, affective withdrawal symptoms, craving and relief drinking, typical alcohol consumption, and reinstatement of dependence after a period of abstinence. Each item is scored on a 4-point scale, ranging from "Almost never" to "Nearly always" giving a possible range of 0 to 60. A score of over 30 indicates severe alcohol dependence. The SADQ has demonstrated evidence of good concurrent validity in a clinical sample ($r = .71$ with the Alcohol Problems Questionnaire, and $r = .81$ with the Short Alcohol Dependence Data questionnaire (Heather, Booth & Luce, 1998). The SADQ has good internal consistency in the current sample ($\alpha = .88$).

Alcohol expectancies. The Alcohol Outcome Expectancies measure (Kushner et al., 1994) assesses the beliefs individuals hold about the consequences of drinking and consists of four subscales. Only two subscales were utilised in the current study: tension reduction (nine items; e.g., "Drinking makes me feel less tense or nervous") and social lubrication (eight items; e.g., "Drinking makes me feel less shy"). Response scales for each item ranged from "Not at all" (0) to "A lot" (4). This measure has demonstrated validity in prospective research (Sher, Wood, Wood, & Raskin, 1996), and each subscale showed good internal consistency in this study with Cronbach alphas ranging from .81 (social lubrication) to .88 (tension reduction).

The 10-item Alcohol Expectancies for Social Evaluative Situations Scale (AESES; Bruch et al., 1992) was used as a measure of positive alcohol expectancies and specifically examines the degree to which individuals endorse positive alcohol outcome expectancies for social facilitation. Each item is scored on a 5-point scale, with items rated on how "true" the effect is. Scores on this measure correspond with other alcohol expectancy measures and correlate to alcohol consumption measures at levels comparable to other alcohol expectancy measures (Bruch et al., 1992; Tran et al., 1997). In the present study, the AESES showed excellent internal consistency ($\alpha = .94$).

Measures of alcohol use and effects of alcohol.

Alcohol use. Recent alcohol use was measured via the timeline follow-back (TLFB) method (Sobell & Sobell, 1992) to assess the quantity and frequency of drinking in the 30 days before enrolment in the study and at each follow-up time point. The TLFB has shown good reliability in a range of administration methods with clinical samples (Sobell, Brown, Leo, & Sobell, 1996). Additionally, the TLFB has shown concurrent validity, with intraclass correlations above .90 between the TLFB and self monitoring methods in an Australian sample for variables such as total number of drinks, number of drinks per day, and number of abstinent days (Sobell et al., 2001).

Measures of impulsivity.

Reward drive. The Behavioural Inhibition/Behavioural Activation System scales (BIS/BAS; Carver & White, 1994), contain 20-items that assess traits including reward drive. The BIS scale is measured on one subscale (seven items), and the BAS scale includes three subscales: reward responsiveness (five items), drive (four items), and fun-seeking (four items). Only the BAS subscales *drive* (e.g., " If I see a chance to get something I want I move on it right away") and *reward responsiveness* (e.g., "When I get something I want, I feel excited and energized"), were used in the analyses as previous studies using factor analysis reveal that both BAS-Drive and BAS-Reward responsiveness of the BIS/BAS scales load on one factor that can be defined as reward drive (Boog, Goudriaan, van de Wetering, Deuss, & Franken, 2013). BAS-Drive measures persistence in achieving goals. BAS-Reward responsiveness measures a person's anticipation of and response to rewarded behaviour. Participants respond on a Likert scale ranging from "Not very true for me" (1) to "Very true for me" (4). Higher scores indicate greater levels of BAS sensitivity. The BIS/BAS scales have been found to be valid measures, with internal consistencies ranging from acceptable (.78; Reward Responsiveness) to good (.85; Drive) in the current study.

Rash impulsivity. In this study, self-reported rash impulsivity was assessed using the Impulsivness scale (I_7-IMP) from the 54-item Eysenck Impulsiveness Questionnaire (I_7; Eysenck et al., 1985). This scale comprises 19 dichotomously scored (yes/no) items and assesses a tendency to act on impulse without sufficient forethought (e.g., "Do you often long for excitement?"). This subscale has been found to have good reliability ($r = 0.87$; Whiteside & Lynam, 2003) and has acceptable reliability in the current sample ($\alpha = .78$).

Procedure

Macquarie University Human Research Ethics Committee (HE28MAR2008-R05758) and the Sydney South West Area Health Service Ethics Review Committee approved the study procedures. The wider trial is registered with the Australian New Zealand Clinical Trials

Registry (ACTRN12608000228381). Potential participants made contact via a range of referral sources, including general practitioners, specialist drug and alcohol and anxiety clinics, newspaper advertisements, online advertising, and occasional media coverage. At first contact, participants received a brief screening assessment over the telephone. A subsequent structured clinical interview was used to confirm diagnostic status, diagnostic severity of social anxiety and alcohol problems, and eligibility for the trial. Eligible participants were randomised to the integrated treatment or alcohol alone intervention. All participants received 10 individually delivered CBT sessions incorporating CBT and motivational interviewing components. All treatment sessions were conducted by clinical psychologists. See Baillie et al. (2013) for randomisation details as well as further details about the treatment conditions. Differences between the two treatment conditions are not the focus of this paper, however all analyses were adjusted for treatment allocation.

Participants in the two conditions received equivalent doses of face-to-face CBT and MI over a total of approximately three to five months. Participants were considered non-completers (i.e., discontinued) if they missed more than two treatment sessions, or if they failed to complete the course of treatment within five months from assessment. Forty-four (73.3%) participants completed treatment (i.e., attended greater than 80% of CBT sessions) in the current sample.

Prior to treatment, participants completed self-report measures of pre-treatment alcohol dependence, severity of social anxiety symptoms, alcohol expectancies, and impulsivity. Approximately one-month, three-months and six-months from the end of all treatment sessions, measures of alcohol dependence, social anxiety symptoms and alcohol use were again completed. Participants who had discontinued treatment were also contacted for post and follow-up assessments which were calculated as if treatment was completed three months from initial date of assessment. All follow-up assessments consisted of the TLFB interview (covering the past 30 days). At the three-month follow-up the ADIS was readministered and

all pre-treatment diagnoses were reassessed. Participants were paid a total of $AUD50 for completing the follow-up assessments.

Data Scoring and Analyses

All analyses were conducted using STATA 12.0. Random effects regression models, or linear mixed models (LMM) were used to test for the effect of impulsivity on social anxiety and alcohol use outcomes within an intention-to-treat framework. All analyses included gender, baseline variables for social anxiety (SPS-SIAS), alcohol dependence (SADQ), quantity of alcohol use (TLFB drinks per drink day) and treatment allocation (alcohol only or integrated) as covariates in order to examine treatment outcome adjusted for initial symptom levels and controlling for treatment differences. Random effect regression models or mixed models have been used in clinical trials and are suited to repeated measures, missing observations, and longitudinal data (Gibbons et al., 1993).

All models included a random intercept and preliminary models were estimated to determine the most appropriate covariance structure for each regression model (Rabe-Hesketh & Skrondal, 2008). An unstructured covariance structure was used for all analyses as it provided the best model fit according to the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) (Singer & Willett, 2003; Twisk, 2013). We tested for linear effects across the time points, but a categorical approach was a better fit.

For each of the four outcome measures, an initial model (Model 1) of change over time was established with only treatment allocation and time included. In Model 2, variables representing treatment allocation and all covariates including gender, baseline SADQ, baseline SPS-SIAS, baseline TLFB drinks per day were included to take into account variance attributable to established predictors. Covariate by time interaction terms, representing the effect of each covariate on change over time, were tested, with only significant interaction effects retained in the model. Once this base model had been established, measures of impulsivity and alcohol expectancies were entered sequentially to determine the improvement

in prediction with the inclusion of these measures. In Model 3, the impulsivity variables, I_7-IMP, BAS-Drive and BAS-Reward were added to the model, and again, interaction with change over time was tested. Finally, in Model 4, the expectancy variables and expectancy by time interaction terms were entered. Thus, Model 4 included baseline covariates, impulsivity variables and expectancy variables, and any significant covariate by time interaction terms. Optimal model specification was determined by comparing model fit using likelihood-ratio tests and results assessing incremental fit for each model are reported with the likelihood-ratio test statistic and associated *p*-value.

Missing data. Analyses based on complete-case data can be biased if data is not missing completely at random. The degree of missing data in this study was: SIAS-SPS total score and SADQ (90% completed at least one post treatment assessment; 73% completed two or more); TLFB drinks per drink day and TLFB per cent number of drink days (93% completed at least one post treatment assessment; 75% completed two or more). Missing data were handled using maximum likelihood (ML) estimation and based on the intention-to-treat principle which includes all participants in the sample ($N = 60$). ML is a highly efficient way of using all available information to estimate parameters rather than deleting cases with missing data (Rabe-Hesketh & Skrondal, 2008; Schafer & Graham, 2002). This approach is widely accepted and has been employed in numerous studies applying multilevel models to substance use outcomes (Rabe-Hesketh & Skrondal, 2008). To further examine the potential impact of missing data, sensitivity analyses were conducted with 100 datasets, each imputed using multiple imputation by chained equations (Royston, 2009). This procedure assumes data are missing at random (MAR) conditional on the variables in the imputation model. To ensure plausibility of the MAR assumption, our imputation model included a number of auxiliary socio-demographic, mental health and drinking variables predictive of incomplete variables and/or missingness (full list available on request). Estimates were combined according to Rubin's rules using the Stata *mim* command (Royston, Carlin, & White, 2009).

Results

Table 13 shows unadjusted mean scores for the social anxiety composite, the measure of alcohol dependence, measures of impulsivity, alcohol expectancies, drinks per day and number of drink days over the four assessment points. Unadjusted mean scores for all predictor and outcome variables separated by treatment allocation are presented in Supplementary Table 1; Appendix C. Bivariate correlations for all variables used in analyses are presented in Supplementary Table 2; Appendix C.

Symptoms of social anxiety: SPS-SIAS total score.

An initial model (Model 1) examining change over time was an appropriate fit to the data (LL = -784.95, $\chi^2(7, N = 60) = 159.51$, $p = 0.000$). The model incorporating all established predictors (Model 2) showed no evidence for an effect of gender, alcohol dependence or baseline drinks per day on change over time, but significantly improved prediction of treatment outcome compared to (Model 1) (likelihood-ratio test, $\chi^2(3, N = 60) = 12.08$, $p = 0.01$). A model with the addition of impulsivity variables (Model 3) significantly improved model fit (likelihood-ratio test, $\chi^2(6, N = 60) = 12.77$, $p = 0.046$). In Model 4, there was no evidence of any interactions with change over time for any expectancy variables, and no significant increase in fit with the addition of expectancy variables (likelihood-ratio test, $\chi^2(3, N = 60) = 3.94$, $p = 0.268$). The partial regression coefficients for the best fitting model for the SPS-SIAS symptom composite are shown in Table 14.

Table 13

Unadjusted means and standard deviations of predictor and outcome variables in the total sample

Variable	Pre Mean (SD)	Post Mean (SD)	3-month Mean (SD)	6-month Mean (SD)
I7-IMP	9.58 (3.92)	-	-	-
BAS-Drive	9.80 (2.61)	-	-	-
BAS-Reward	16.33 (2.27)	-	-	-
AESES	41.25 (7.74)	-	-	-
TR-AE	27.18 (5.81)	-	-	-
SL-AE	19.50 (4.83)	-	-	-
SIAS-SPS total score [a,b,d,f]	81.57 (22.82)	51.40 (23.21)	49.00 (24.05)	44.57 (22.42)
SADQ [a,b,d,f]	16.17 (10.78)	10.19 (9.78)	8.06 (8.17)	6.03 (5.61)
Total no. drinks, past 30 days [a,c,e,f]	187.47 (150.48)	55.10 (62.37)	105.63 (169.02)	49.13 (57.08)
Number of drink days, past 30 days (%) [a,c,e,f]	18.07 (8.22)	9.42 (8.32)	13.41 (10.10)	9.53 (8.78)

Note. I7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation System – Drive subscale; BAS-Reward = Behavioural Activation System – Reward Responsiveness subscale; AESES = Alcohol Expectancies for Social Evaluative Situations Scale; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social Lubrication Alcohol Expectancy; SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; TLFB: Time Line Follow-Back.

[a] n = 60 at Pre; [b] n = 48 at Post; [c] n = 45 at Post; [d] n = 36 at 3-month; [e] n = 46 at 3-month; [f] n = 34 at 6-month; [g] n = 40 at 6-month

Table 14

Final random regression model predicting SIAS-SPS symptom change with treatment

Variable	Coefficient	SE	Z	p	95% CI
Gender	-1.09	5.14	-.21	0.831	-11.17 to 8.98
Allocation	1.21	5.36	.23	0.822	-9.30 to 11.71
Allocation × Time (1MFU)	-16.88	4.93	-3.43	0.001**	-26.54 to -7.22
Allocation × Time (3MFU)	-13.85	7.09	-1.95	0.051[†]	-27.74 to .04
Allocation × Time (6MFU)	-13.51	8.05	-1.68	0.093	-29.29 to 2.27
Baseline SADQ	1.00	.28	3.63	0.000***	.46 to 1.54
Baseline drinks per day	-1.10	.46	-2.38	0.017*	-2.01 to -.19
I_7-IMP	1.51	.74	2.05	0.040*	.08 to 2.96
I_7-IMP × Time (1MFU)	-2.22	.64	-3.48	0.000***	-3.47 to -.97
I_7-IMP × Time (3MFU)	-1.92	.93	-2.06	0.039*	-3.74 to -.10
I_7-IMP × Time (6MFU)	-2.10	1.00	-2.09	0.037*	-4.08 to -.13
BAS-Drive	-.67	1.05	-.64	0.525	-2.73 to 1.39
BAS-Reward	.52	1.08	.48	0.632	-1.60 to 2.63

Note. SADQ = Severity of Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale.

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Within the final fully adjusted model, there was a large main effect of alcohol dependence (SADQ) on social anxiety symptoms collapsed across time, with higher SADQ predicting higher SPS-SIAS severity. In contrast, baseline alcoholic drinks per day (TLFB) was associated with a decrease in overall severity of social anxiety, but not with degree of change over time. However, rash impulsivity as assessed by the I_7-IMP variable, was associated with degree of change in social anxiety symptoms over time as is shown by its significant interaction with time (see Table 14). To illustrate these effects, the I_7-IMP variable was split into a 3-categorial variable (tertile split; see Figure 6) so the pattern of findings on this continuous variable can be visualised to clarify the relationship. Examination of Figure 6 shows that higher baseline rash impulsivity was associated with higher social anxiety initially, but this higher impulsivity group also improved to a greater extent over time. The impulsivity variables BAS-Drive and BAS-Reward responsiveness did not impact social anxiety outcomes at any time point.

Predictive Margins with 95% CIs
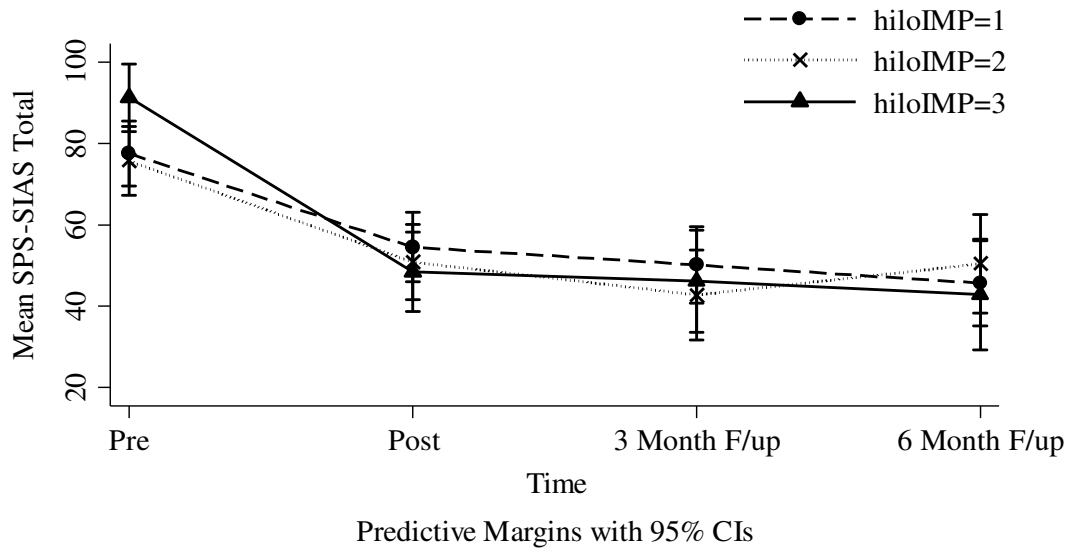
Figure 6. SPS-SIAS symptom scores over time for comorbid SAD-AUD individuals with low, medium and high levels of rash impulsivity (I_7-IMP) ($N = 60$).

Severity of alcohol dependence: SADQ.

Four random regression models were fitted to SADQ scores. The categorical model (Model 1) established change over time (LL = -610.44, $\chi^2(7, N = 60) = 75.99$, $p = 0.000$). A random slope model containing allocation, gender, baseline social anxiety, baseline alcohol dependence, baseline alcoholic drinks per day and any significant interactions between these variables and time (Model 2) significantly improved prediction of treatment outcome compared to Model 1 (likelihood-ratio test, $\chi^2(6, N = 60) = 58.49$, $p = 0.000$). There was weak evidence that Model 3 containing all of the impulsivity variables and two-way interactions between all predictors and time was a better fit than the previous model, (likelihood-ratio test, $\chi^2(12, N = 60) = 19.73$, $p = 0.072$), indicating that impulsivity explained additional variance for this outcome. Model 4 containing the expectancy variables was not a better fit than the more parsimonious models, (likelihood-ratio test, $\chi^2(9, N = 60) = 14.16$, $p = 0.117$). Estimates of specific effects for the final fully adjusted model are shown in Table 15.

Table 15

Final random regression model predicting SADQ symptom change with treatment

Variable	Coefficient	SE	Z	p	95% CI
Gender	2.10	1.51	1.39	0.163	-.85 to 5.06
Allocation	2.85	1.43	2.00	0.05†	.05 to 5.65
Baseline SPS-SIAS	.16	.04	3.55	0.000***	.07 to .24
SPS-SIAS × Time (1MFU)	-.15	.04	-3.53	0.000***	-.24 to -.07
SPS-SIAS × Time (3MFU)	-.18	.05	-3.34	0.001**	-.27 to -.08
SPS-SIAS × Time (6MFU)	-.16	.05	-3.00	0.003**	-.26 to -.07
Baseline drinks per day	1.06	.16	6.79	0.000***	.75 to 1.36
Baseline drinks per day × Time (1MFU)	-.17	.14	-1.21	0.226	-.43 to .10
Baseline drinks per day × Time (3MFU)	-.36	.17	-2.16	0.031*	-.68 to -.03
Baseline drinks per day × Time (6MFU)	-.82	.17	-4.92	0.000***	-1.15 to -.50
I_7-IMP	-.03	.30	-.11	0.915	-.61 to .55
I_7-IMP × Time (1MFU)	-.38	.29	-1.33	0.185	-.94 to .18
I_7-IMP × Time (3MFU)	-.63	.33	-1.93	0.054†	-1.28 to .01
I_7-IMP × Time (6MFU)	.51	.32	1.60	0.110	-.11 to 1.13
BAS-Drive	-.22	.46	-.48	0.629	-1.13 to .68
BAS-Drive × Time (1MFU)	.98	.43	2.28	0.023*	.14 to 1.82
BAS-Drive × Time (3MFU)	.67	.48	1.39	0.164	-.27 to 1.62
BAS-Drive × Time (6MFU)	-.23	.50	-.45	0.650	-1.20 to .75
BAS-Reward	-.29	.48	-.60	0.548	-1.23 to .65
BAS-Reward × Time (1MFU)	.34	.44	.77	0.441	-.53 to 1.21
BAS-Reward × Time (3MFU)	-.21	.50	-.42	0.676	-1.19 to .77
BAS-Reward × Time (6MFU)	.24	.51	.48	0.631	-.75 to 1.24

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale;

† *p* < .10; * *p* < .05; ** *p* < .01; *** *p* < .001

In addition to baseline social anxiety, and baseline alcoholic drinks per day[3], there was marginal evidence for an association between impulsivity as assessed by the I_7-IMP and BAS-Drive variables with degree of change over time for alcohol dependence. However, there was weaker evidence at the one month time point for BAS-Drive after taking into account missing data in the sensitivity analyses (see Supplementary Table 4; Appendix C).

Figure 7 depicts alcohol dependence (SADQ) over time by rash impulsivity (I_7-IMP) split into a 3-categorical variable so the pattern of findings on this continuous variable can be

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[3] The treatment allocation effect for alcohol dependence symptoms is not described despite being marginally significant. Treatment allocation and treatment allocation by time interactions were included in analyses to provide a more conservative test of the value of impulsivity, and as these findings are part of the larger clinical trial and are not the focus of this paper, they will not be discussed further.

interpreted. Higher impulsivity was associated with higher alcohol dependence initially, and this higher impulsivity group appears to improve to a greater extent at the three-month follow-up time point relative to baseline.
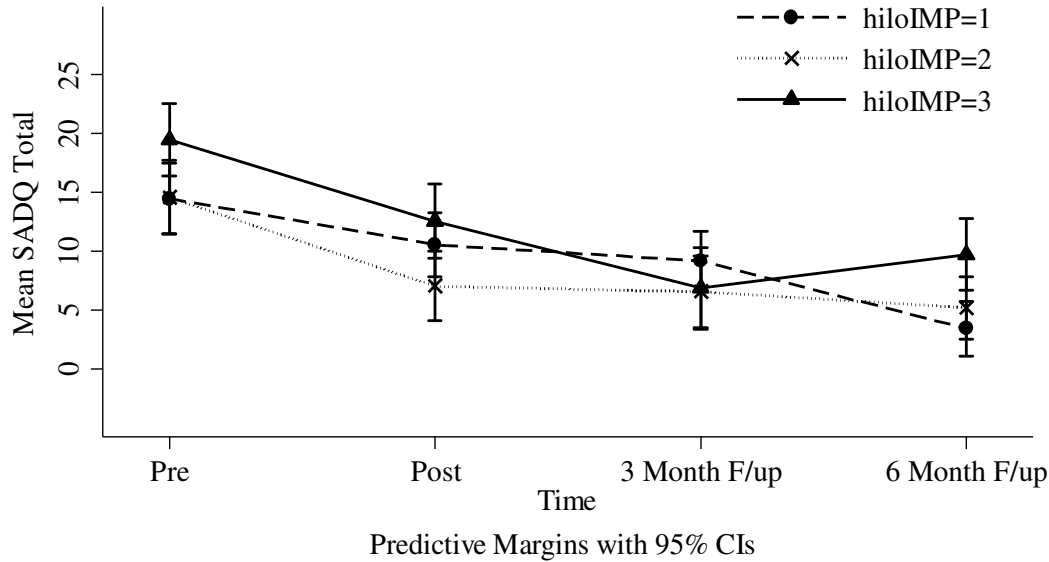
Figure 7. Severity of alcohol dependence scores over time for comorbid SAD-AUD individuals with low, medium and high levels of rash impulsivity (I_7-IMP) ($N = 60$).

Drinks per drinking day.

Four random regression models were fitted to TLFB drinks per day. An initial model (Model 1) with only allocation and time was an appropriate fit to the data (LL = -520.04, $\chi^2(7, N = 60) = 57.44$, $p = 0.000$). A random slope model with baseline covariates and significant interactions between alcohol dependence with change over time (Model 2) significantly improved fit compared to Model 1, (likelihood ratio test, $\chi^2(6, N = 60) = 51.48$, $p = 0.000$). Model 3 included three impulsivity variables and all interactions between the impulsivity variables and time, and contributed significantly to prediction of change over time, (likelihood ratio test, $\chi^2(12, N = 60) = 21.73$, $p = 0.041$). Model 4 included alcohol expectancy variables and a significant interaction between tension reduction and change over time, but was not a better fit than Model 3 (likelihood ratio test, $\chi^2(6, N = 60) = 11.38$, $p = 0.09$). Thus, there was no evidence of any impact on the rate of change in drinks per day for any of the expectancy variables employed (see final model in Table 16).

Table 16

*Final random regression model predicting TLFB drinks per day change with treatment*

Variable	Coefficient	SE	Z	p	95% CI
Gender	-2.62	1.33	-1.96	0.050[†]	-5.23 to -.003
Allocation	-.85	1.232	-.64	0.520	-3.45 to 1.74
Allocation × Time (1MFU)	1.56	1.21	1.29	0.200	-.81 to 3.92
Allocation × Time (3MFU)	-.11	1.35	.08	0.933	-2.54 to 2.77
Allocation × Time (6MFU)	1.07	1.62	.66	0.509	-2.10 to 4.24
Baseline SPS-SIAS	-.04	.03	-1.46	0.143	-.10 to .01
Baseline SADQ	.41	.06	6.39	0.000***	.28 to .53
SADQ × Time (1MFU)	-.27	.06	-4.93	0.000***	-.38 to -.16
SADQ × Time (3MFU)	-.18	.06	-2.96	0.003**	-.30 to -.06
SADQ × Time (6MFU)	-.26	.07	-3.51	0.000***	-.41 to -.12
I_7-IMP	.16	.19	.85	0.400	-.21 to .54
I_7-IMP × Time (1MFU)	-.73	.18	-4.07	0.000***	-1.08 to -.38
I_7-IMP × Time (3MFU)	-.41	.19	-2.17	0.030*	-.77 to -.04
I_7-IMP × Time (6MFU)	-.56	.24	-2.31	0.021*	-1.04 to -.09
BAS-Drive	.18	.30	.58	0.561	-.42 to .77
BAS- Drive × Time (1MFU)	.63	.29	2.18	0.029*	.06 to 1.20
BAS- Drive × Time (3MFU)	.22	.30	.72	0.473	-.38 to .81
BAS- Drive × Time (6MFU)	.39	.36	1.10	0.272	-.31 to 1.10
BAS-Reward	.36	.31	1.14	0.253	-.25 to .97
BAS-Reward × Time (1MFU)	-.62	.28	-2.21	0.027*	-1.16 to -.07
BAS-Reward × Time (3MFU)	-.27	.29	-.94	0.347	-.83 to .29
BAS-Reward × Time (6MFU)	-.41	.34	-1.23	0.218	-1.07 to .24

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale.

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Within the final fully adjusted model, baseline alcohol dependence was associated with degree of change over time for alcohol use as measured by TLFB drinks per day. More severe alcohol dependence at baseline was associated with greater change in alcohol quantity over time. Rash impulsivity (I_7- IMP) was also associated with degree of change over time. Examination of Figure 8 illustrates that higher baseline rash impulsivity (tertile split) was associated with higher baseline alcoholic drinks per day, and this higher impulsivity group also improved to a greater extent over time with treatment; reporting a greater decrease in drinks per day. There was weaker evidence for a significant association for reward drive (BAS-Drive and BAS-Reward Responsiveness) at the one-month time point after taking into

account missing data in the sensitivity analyses (see Supplementary Table 5; Appendix C),

and these effects on change in drinking quantity over time were opposite in direction.

Specifically, more severe BAS-Drive at baseline was associated with less change in drinking

quantity, whereas higher BAS-Reward Responsiveness at baseline was associated with greater

change in total drinks consumed per drink day.
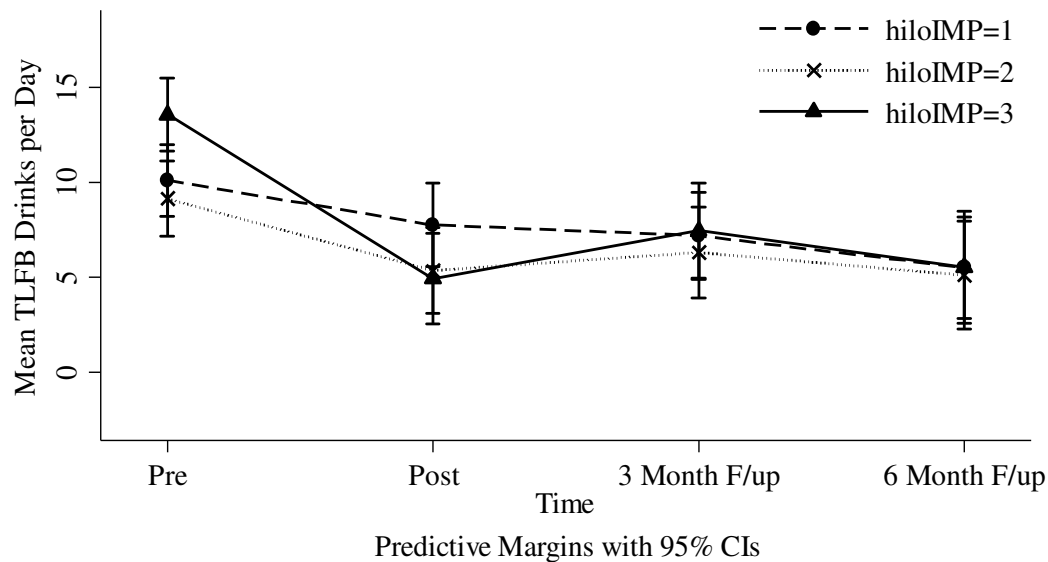
Figure 8. Timeline follow-back mean drinks per day over time for comorbid SAD-AUD individuals with low, medium and high levels of rash impulsivity (I_7-IMP) ($N = 60$).

Number of drinking days.

Model 1 with treatment allocation and time established change over time (LL = -658.71,

$\chi^2(7, N = 60) = 62.33$, $p = 0.000$). A random intercept model containing the baseline

covariates gender, social anxiety, alcohol dependence, and drinks per day (Model 2), was not

a significantly better fit to the pattern of drinks per day over time than the previous model

with only treatment group and time, (likelihood-ratio test, $\chi^2(10, N = 60) = 15.71$, $p = 0.11$).

Model 3 containing all of the impulsivity variables and significant interactions between all

predictors and time was not a better fit than the earlier models, (likelihood-ratio test, $\chi^2(6, N = 60) = 9.54$, $p = 0.15$), indicating that the impulsivity variables were not adding to the model.

The inclusion of expectancy variables in Model 4 was a significantly better fit than Model 3

(likelihood-ratio test, $\chi^2(3, N = 60) = 11.67$, $p = 0.009$) due to the large main effect of tension

reduction expectancies on number of drinking days collapsed across time, with higher tension

reduction expectancies predicting a greater number of drinking days.

Table 17

Final random regression model predicting TLFB number of drinking days change with treatment

Variable	Coefficient	SE	Z	p	95% CI
Gender	.84	1.84	.46	0.647	-2.76 to 4.45
Allocation	-2.18	2.03	-1.08	0.282	-6.15 to 1.79
Allocation × Time (1MFU)	3.24	2.43	1.33	0.183	-1.52 to 8.00
Allocation × Time (3MFU)	5.94	2.45	2.42	0.016*	1.13 to 10.74
Allocation × Time (6MFU)	1.84	2.56	.72	0.472	-3.17 to 6.85
Baseline SPS-SIAS	-.13	.04	-3.12	0.002**	-.21 to -.05
Baseline SADQ	-.07	.14	-.55	0.584	-.34 to .19
SADQ × Time (1MFU)	-.22	.15	-1.47	0.142	-.52 to .07
SADQ × Time (3MFU)	-.07	.15	-.49	0.621	-.36 to .21
SADQ × Time (6MFU)	-.25	.15	-1.64	0.101	-.55 to .05
Baseline drinks per day	-.29	.22	-1.35	0.178	-.72 to .13
Baseline drinks per day × Time (1MFU)	.26	.24	1.08	0.280	-.21 to .72
Baseline drinks per day × Time (3MFU)	.27	.24	1.11	0.268	-.20 to .74
Baseline drinks per day × Time (6MFU)	.07	.25	.27	0.787	-.42 to .56
I_7-IMP	.17	.24	.71	0.478	-.30 to .64
BAS-Drive	.16	.37	.43	0.668	-.56 to .88
BAS-Reward	.73	.48	1.53	0.127	-.21 to 1.67
BAS-Reward × Time (1MFU)	-.06	.54	-.12	0.905	-1.13 to 1.00
BAS-Reward × Time (3MFU)	-1.20	.53	-2.27	0.023*	-2.24 to -.17
BAS-Reward × Time (6MFU)	.27	.57	.47	0.640	-.85 to 1.39
Baseline TR-AE	.63	.18	3.48	0.000***	.28 to .99
Baseline SL-AE	-.12	.22	-.56	0.577	-.55 to 31
Baseline AESES	-.13	.13	-1.02	0.309	-.38 to .12

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of
Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural
Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward
Responsiveness subscale; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social
Lubrication Alcohol Expectancy; AESES = Alcohol Expectancies for Social Evaluative Situations
* $p < .05$; ** $p < .01$; *** $p < .001$

A significant interaction was revealed between BAS-Reward and change over time. Higher

BAS-Reward was associated with higher number of drink days initially, and this higher

impulsivity group appeared to improve to a greater extent at the three-month follow-up time

point relative to baseline. However, this effect was only marginal ($p = .10$) after imputing

missing data in the sensitivity analysis (see Supplementary Table 6; Appendix C). There was

no evidence of an effect on change in drinking quantity over time for any other impulsivity or

expectancy variables (see Table 17).

Sensitivity Analysis Using Imputation.

Sensitivity analyses with multiply imputed data confirmed the primary analyses. The resulting fraction of missing information (FMI) estimates were no larger than .77 and the Monte Carlo standard errors were acceptably small, indicating that 100 imputed datasets was more than sufficient. There was no great difference in the pattern of results derived from analyses with imputed data. However, there was weaker evidence at some time points after imputation of missing data (see Supporting Information Tables S3-S6).

Discussion

In a sample of individuals with comorbid social anxiety and alcohol use disorders receiving CBT targeted at either alcohol alone or both problems combined, baseline impulsivity was predictive of greater reduction in social anxiety symptoms. To our knowledge, this is the first paper to examine the effect of impulsivity on social anxiety outcomes. There was marginal evidence to suggest that high impulsivity also predicted greater reduction with treatment in symptoms of alcohol dependence. A similar pattern was observed for quantity but not frequency, of alcohol use, consistent with the expected role of rash impulsivity.

Specifically, individuals with higher trait rash impulsivity improved to a greater extent over time on the social anxiety symptom measure. There was also weak evidence suggesting that while high rash impulsivity at baseline was associated with higher alcohol dependence severity initially, these highly impulsive individuals improved to a greater extent at three months following treatment. This finding of greater improvement relating to higher impulsivity was unexpected, and is in contrast to findings from previous studies showing that impulsivity prior to beginning treatment is associated with poorer alcohol outcomes such as shorter time to relapse (Loree et al., 2015; Stevens et al., 2014).

Similarly, analyses of outcomes also indicated that all measures of impulsivity had some effects on quantity of alcohol use, (i.e., drinks per day), with individuals high in trait rash

impulsivity at pre-treatment showing improvements in quantity of alcohol consumption over time. Contrary to expectation, the impulsivity variables did not explain additional variance for frequency of alcohol use, and there was no significant effect of these predictors on number of drinking days. This may be due to frequency of use and quantity consumed measuring different aspects of drinking behaviour (Vogel-Sprott, 1983), to which impulsivity might contribute differently. This result may also be a reflection of this measure being a poor index of consumption. For example, frequency of alcohol consumption as commonly measured by percentage days abstinent or mean number of drinking days does not take into account infrequent episodes of heavy "binge" and light drinking.

The association between higher impulsivity and greater reduction in social anxiety and alcohol outcomes may indicate that impulsivity is a marker of overall severity, and higher severity is often associated with greater improvement (e.g., Greenfield et al., 2008). A possible cause of the observed changes may be due to regression artefacts (i.e., regression towards the mean) that occur when repeated measurements are made on the same subject (Stigler, 1997). Alternatively, individuals with high trait impulsivity may benefit most from the therapy delivered in this clinical trial, and the development of skills to manage their impulsivity. Indeed, Litt, Babor, DelBoca, Kadden and Cooney (1992) found that a more severe subgroup of alcohol-dependent individuals had better outcomes with a more structured treatment intervention focussed on coping skills. The treatment programs used in this study were based on a combination of cognitive behaviour therapies with a motivational interviewing style that was designed to help the patient identify high-risk situations for relapse, learn and rehearse strategies for coping with these situations, develop problem solving skills, and recognise and cope with craving and urges (Baillie et al., 2013; Stapinski et al., 2014). All participants also attended an initial session dedicated to motivational enhancement which focussed on abstinence or reduced drinking.

The techniques used in the treatment may be particularly beneficial for highly impulsive patients in a number of ways. Firstly, it may improve their capacity to identify and modify problematic behavioural patterns. Specifically, the type of treatment employed may assist in enhancing the capacity for self-regulation of mood, arousal and behaviour; even in those high on impulsivity. Secondly, it is possible that the treatment changed levels of impulsivity and this change in impulsiveness is predictive of improvement in treatment outcome. While evidence suggests that personality traits are generally stable and enduring, changes have been reported in some individuals (Asendorpf, 2008). However, as the measures of impulsivity were only administered at baseline, it was not possible to measure change in this variable. Further, the therapeutic approach employed in treatment may assist inhibitory control by creating a conflict with an individual's treatment goals (e.g., abstinence) when provided with the opportunity to continue drinking, or increase the salience of longer-term goals, thus strengthening their reward value (Gullo & Dawe, 2008). These potential mechanisms warrant further exploration. Additionally, these results may also be explained by another variable that mediates the effect of reward drive and/or rash impulsivity on social anxiety and alcohol outcomes.

Notably, rash impulsivity rather than reward drive was a consistent predictor of treatment outcome. This could indicate that components of impulsivity such as reward drive and rash impulsivity may be distinct constructs in this clinical sample and rash impulsivity may be more relevant to alcohol use. Further, rash impulsivity might be a better predictor of treatment outcome once alcohol problems are more established, as they are in this treatment-seeking sample of individuals with comorbid SAD-AUD.

Another factor that was found to impact treatment outcome was social anxiety symptom severity. Results revealed that symptoms of social anxiety was predictive of greater reduction in alcohol dependence severity. In particular, individuals with higher symptoms of social anxiety at pre-treatment improved to a greater extent over time on severity of alcohol

dependence. The improvement in this alcohol outcome may be attributable to the skills acquired in treatment. The CBT employed in treatment incorporated cue exposure that may have assisted these individuals with their craving when exposed to affectively challenging conditions. Furthermore, the treatment also focussed on the development of strategies to manage stressful situations and/or cognitions related to alcohol as a tension reducer that may be triggers for craving/alcohol use. Thus, these skills may assist the individual to choose an alternate behaviour to prevent or alleviate craving, thereby reducing the need to self-medicate with alcohol for mood problems. This subsequently influences dependence on alcohol as a coping strategy.

Interestingly, impulsivity was found to be a far stronger predictor of both social anxiety and alcohol outcomes than were alcohol expectancies or evaluations. These results are in line with some previous studies that have not found a relationship between alcohol expectancies and response to alcohol treatment outcomes (Jones et al., 2001). This may be due, in part, to substantial overlap between these constructs, such that when they are placed in one model, only one will account for a significant portion of the variance in these outcome measures. Moreover, while it is widely accepted that alcohol expectancies are associated with the initiation of alcohol use (e.g., Smith, Goldman, Greenbaum, & Christiansen, 1995), the high level of alcohol dependence in the current sample and its interaction with social anxiety may indicate that once dependence has been established, other mechanisms play a more influential role in maintaining the problem and influencing treatment outcomes. In addition, negative expectancies; that is, the belief that alcohol use will result in undesirable outcomes, has been found to be associated with better treatment outcomes and cessation of drinking (Jones et al., 2001). The expectancy measures included in this study were related to tension reduction, social facilitation and perceptions about the effects of alcohol in social situations; that is, items relating more to beliefs that drinking alcohol will result in positive outcomes. Further, the most important contribution of alcohol expectancies may be their ability to predict other

aspects of drinking behaviour such as the onset of drinking problems (e.g., Jones et al., 2001) or other outcomes that were not included in the current study.

This study has several limitations. Firstly, our analysis is based on a relatively small sample. A second limitation of the current study was the use of two self-report measures of trait impulsivity. It has been suggested that behavioural and self-report measures of impulsivity represent different constructs (Boog et al., 2013). Behavioural measures or neurocognitive tasks of these constructs which are believed to be more proximal measures of the neurobiological processes underlying impulsive behaviour, can provide a complementary method for measuring impulsivity that does not rely solely on recall or interpretation of past behaviour (Gottesman & Gould, 2003). A promising direction for future research would be a multi-method examination of the components of impulsivity. Additionally, besides the variables included in the current study, there are several other known predictors of alcohol outcomes such as the number of previous treatments, duration of alcohol use disorders and age of onset (Adamson et al., 2009), and social anxiety outcomes such as comorbidity with other anxiety disorders and therapist expectations (Mululo, de Menezes, Vigne, & Fontenelle, 2012). However, the purpose of the current study was not to account for all the possible variance in outcome, but rather to examine the effects of impulsivity on social anxiety and alcohol outcomes.

Despite these limitations, this study is the first to examine how impulsivity may impact treatment from CBT provided to individuals presenting with comorbid social anxiety and alcohol use problems. The findings of the reported study extend those of previous research by offering some indication of how impulsivity; in particular, higher rash impulsivity may be associated with a greater reduction in alcoholic drinks per day and alcohol dependence symptoms following treatment, and offer novel evidence indicating that the presence of more severe baseline rash impulsivity is associated with greater reduction in social anxiety symptoms following treatment. These findings suggest that impulsivity may be an important

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variable to predict treatment outcomes, and could offer important prognostic information to clinicians. High impulsivity might be valuable when making individual treatment plans by serving as a marker to indicate that a person may be likely to benefit significantly from a skills based approach to develop strategies to manage craving, impulsivity and behaviours associated with impulsivity. Interventions that improve inhibitory control, impulsive decision-making or craving may represent valuable therapeutic strategies in the short-term in individuals with co-occurring social anxiety and alcohol use disorders with high trait impulsivity. Future studies need to consider indirect pathways by which impulsivity exerts its influence on treatment outcomes.

Chapter Six

General Discussion

General Discussion

This thesis demonstrates how reward drive and rash impulsivity impact the relationship between social anxiety and alcohol use disorders. The findings from the current studies largely support a role of impulsivity in the relationship between social anxiety and alcohol use disorders (Chapters 3, 4 and 5). Implementing a range of cross-sectional, clinical trial and laboratory methods, the current thesis explored whether impulsivity (1) distinguishes between individuals with social anxiety disorder, with and without comorbid alcohol use disorders, (2) may be a mechanism that contributes to maintaining problematic alcohol use in individuals with established comorbid social anxiety and alcohol use disorders; and (3) influences treatment outcomes for individuals with these co-occurring disorders undergoing intervention for these issues. The implications and limitations of each empirical study have been considered in each chapter throughout this thesis. This final chapter critically analyses the program of research as a whole, starting with an overview of the research findings. The overall pattern of findings of the studies are interpreted and then discussed within the context of a contemporary conceptualisation of impulsivity: the 2-Component Approach to Reinforcing Substances model (2-CARS; Gullo & Dawe, 2008); and models of addiction, development and comorbidity. Finally, clinical implications, methodological considerations, limitations and directions for future research are discussed.

Overview of the Four Empirical Studies

Minimal investigation has been conducted into the prevalence and impact of co-occurring alcohol use disorders among individuals seeking treatment for anxiety disorders. Chapter 2 aimed to address this gap in the literature by examining the rates of comorbid social anxiety and alcohol use disorders in a sample of individuals seeking treatment for social anxiety. A secondary aim of this study was to examine the impact of co-occurring alcohol use disorders and alcohol use on social anxiety outcomes following treatment targeting social anxiety. Two alcohol-related behaviours, drinking prior to and during social situations, were identified as

important in influencing social anxiety. Specifically, while treatment gains were unaffected by level of pre-treatment alcohol use, drinking prior to social situations—a behaviour thought to be goal-directed and planned—was associated with more severe social anxiety symptoms. Drinking alcohol during social situations was also associated with greater social anxiety severity. This suggests that these drinking behaviours may serve as problematic safety behaviours (e.g., using alcohol to manage anticipatory anxiety before or during a social situation) that exacerbate symptoms of social anxiety. Further, the presence of comorbid AUD was relatively rare in the sample, but when it occurred it appeared to interfere with treatment outcome.

Given the impact of drinking prior to social situations on severity of social anxiety, it was important to identify individuals who may be most susceptible to developing alcohol use problems. Chapter 3 sought to examine whether impulsivity was characteristic of alcohol dependent individuals in general, and specifically, whether heightened impulsivity distinguished alcohol dependent individuals with comorbid social anxiety disorder from those individuals with social anxiety disorder alone. This chapter introduced a two-component framework of impulsivity in an undergraduate and community sample. Overall, results suggested a pattern of elevated trait and behavioural reward drive and trait rash impulsivity reported by dependent drinkers with and without co-occurring social anxiety disorder compared to non-dependent drinkers, including those with social anxiety.

In Chapter 4, this two-component framework of impulsivity was applied to examine physiological and subjective responses to alcohol ingestion, and their association with impulsivity and alcohol expectancy in a treatment-seeking sample of individuals with comorbid social anxiety and alcohol use disorders. Higher levels of impulsivity was associated with greater perceived stimulating effects and greater physiological arousal. This could be considered as a mechanism by which impulsivity contributes to continued drinking in individuals with social anxiety and alcohol use disorders.

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Having established a relationship between self-reported impulsivity, social anxiety and alcohol use, Chapter 5 explored whether impulsivity influenced social anxiety and alcohol treatment outcomes in individuals with comorbid social anxiety and alcohol use disorders receiving treatment for both of these problems together or alcohol issues alone. Few interventions have been developed for individuals with these co-occurring problems, and little is known about factors that affect the course of treatment. This study aimed to address this gap in the literature by examining factors that may impact treatment outcomes for these co-occurring problems. It is also the first study to examine the impact of impulsivity on social anxiety outcomes. Higher impulsivity was related to more severe social anxiety and alcohol dependence initially, but also a greater reduction in these symptoms following treatment. These findings highlight that understanding the impact of impulsivity on treatment targeting social anxiety and alcohol use may have prognostic utility, and suggests the skills imparted through cognitive behaviour therapy could represent a valuable therapeutic strategy in individuals with elevated impulsivity and co-occurring social anxiety and alcohol use disorders.

Implications of this Research

A Role for Impulsivity in the SAD-AUD Relationship.

Goal-directed planning or approach behaviour towards a rewarding substance is considered to underlie one component of impulsivity, reward drive, as described in the two-component reinforcement of substances model (2-CARS; Dawe et al., 2004). Specifically, reward drive as a trait reflects persistence or drive in acquiring desired goals (Dawe & Loxton, 2004). The procurement of and subsequent use of substances, such as alcohol requires a significant amount of goal-directed planning (Dawe & Loxton, 2004). Thus, consuming alcohol prior to encountering a social stressor or situation (Chapter 2) can be understood as an example of this behaviour. This behaviour requires both motivation and preparation to acquire the substance, in this instance alcohol, for subsequent consumption.

On a behavioural level, reward drive has been linked to reward conditioning; that is, the learning process that occurs when two stimuli are repeatedly paired (Tiffany, 1990), craving and attention to rewarding stimuli (Dawe et al., 2004; Gullo & Dawe, 2008). Results from Chapter 2 indicate how reward conditioning between alcohol and drinking prior to social situations could potentially develop. Reward drive is believed to increase susceptibility to noticing and remembering the positive effects or outcomes of alcohol consumption (Gullo et al., 2010; Harnett, Lynch, Gullo, Dawe, & Loxton, 2013). Due to the anxiolytic properties of alcohol, proponents of the self-medication hypothesis argue that individuals with social anxiety typically drink to reduce their anxiety (Khantizian, 1985). This in turn could encourage learning via attending to, and retaining positive substance outcomes, which are created via negative reinforcement (i.e., a behaviour is strengthened by the removal of aversive stimuli) (Kushner et al., 2000). For instance, if an individual consistently drinks alcohol prior to social situations to ease anxiety and the alcohol provides relief, this response may occur each time alcohol or related stimuli is encountered. Consequently, this may lead to the formation of stronger conditioned associations to alcohol and related stimuli. However, trait and behavioural measures of reward drive were not administered in this study, so we were unable to directly examine whether elevated reward drive was associated with increased alcohol consumption or with specific drinking behaviors.

There was evidence to suggest a role for reward drive in mechanisms that may function to maintain problematic drinking, such as through cue-elicited self-reported craving for alcohol (Chapter 3), increased physiological response to alcohol and alcohol-related stimuli (Chapters 3 and 4), and via differences in the subjective experience of stimulating and sedative effects (Chapter 4). At the behavioural level, the risk expressed by reward drive was observed by increased reward conditioning which was operationalised as stronger cue reactivity (i.e., self-reported craving, positive affect and physiological arousal) as cue reactivity is believed to be an indicator of approach behaviour towards alcohol (Carter & Tiffany, 1999). Both dependent

drinkers and those individuals with additional co-occurring social anxiety found alcohol-related cues more arousing than cues connected with water as reflected in skin conductance, heart rate reactivity, and self-reported craving compared to individuals with social anxiety only and neither disorder (Chapter 3). Similarly, higher reward drive was associated with higher physiological responding to alcohol and related cues, and compared to soft drink, ingestion of alcohol or placebo alcohol resulted in an increase in skin conductance and heart rate for individuals with social anxiety and alcohol use disorders (Chapter 4). Higher reward drive was also associated with increases in the subjective experience of stimulant effects (Chapter 4).

Memories associated with the stimulant qualities of alcohol as well as learned associations between internal states and alcohol and related stimuli or environmental context can impact both the initiation of use and the regulation of consumption (e.g., Carter & Tiffany, 1999; Fox, Bergquist, Hong, & Sinha, 2007; Sinha et al., 2009; Vengeliene, Bilbao, Molander, & Spanagel, 2008; Weiss, 2005). According to Tiffany's (1990) cognitive processing model (CPM), repeated drug use over time becomes an over-learned, automatic behaviour. Likewise, Baker et al. (2004) suggest that the motivation for drug use may not reflect conscious cognitive control. Therefore, for individuals with alcohol use disorders it appears that processing of alcohol-related stimuli may be more automatic; eliciting stronger responses, and this was observed in individuals with alcohol dependence and co-occurring social anxiety disorder (Chapters 3 and 4). Thus, high levels of impulsivity may maintain problematic alcohol use in individuals with co-occurring social anxiety and alcohol use disorders via increased sensitivity to the positively reinforcing and heightened subjective stimulant and sedative effects.

Results from Chapter 3 suggest that social anxiety may operate differently depending on co-occurring levels of externalising problems such as alcohol dependence. Specifically, a portion of individuals with social anxiety might be more vulnerable to problem drinking, and

impulsivity may help to differentiate these individuals. Internalising symptoms (e.g., social anxiety) have been identified as both risk (e.g., King, Iacono, & McGue, 2004) and protective factors (e.g., Fleming, Mason, Mazza, Abbott, & Catalano, 2008) in developmental models of substance use. Our results suggest that social anxiety may not function as a protective factor when co-occurring with symptoms of externalising disorders such as impulsivity. Colder et al. (2013) argue that whether elevated internalising problems function as a risk or protective factor may be determined on their co-occurrence with symptoms of externalising disorders. Indeed, they found that co-occurring externalising an internalising problems were associated with higher levels of substance (e.g., alcohol) use in the future (Colder et al., 2013).

Similarly, Nicholls et al. (2014) reported support for two distinct social anxiety subgroups; the first with typical characteristics of social anxiety such as low levels of risk-taking, minimal levels of impulsivity and substance use (avoidant-motivated). The second subgroup presented with high levels of reward drive and rash impulsivity, substance use and risk-taking (approach-motivated) (Nicholls et al., 2014). Individuals with co-occurring social anxiety and alcohol use disorders had higher levels of trait impulsivity, and experienced greater arousal to alcohol cues than individuals with social anxiety disorder alone (Chapter 3). However, whether elevated levels of impulsivity reported by individuals with co-occurring social anxiety and alcohol use disorder were present before symptoms of social anxiety, or are the result of problematic alcohol use, can only be determined by prospective studies.

Classification systems proposed to categorise subtypes of alcohol dependence have often placed impulsivity in a more severe subgroup characterised by early onset of alcohol use, and symptoms relating to externalising problems (Babor et al., 1992; Cloninger, 1987, 1995). However, in individuals with social anxiety, Colder et al. (2010), Hussong et al. (2011) and Cloninger (1987) propose that a self-medication pathway to alcohol problems may not surface until late adolescence or early adulthood, suggesting that internalising problems function as a protective factor for substance use in early adolescence. According to these models, self-

medication with substances to alleviate emotional distress is thought to be the most common mechanism if internalising symptoms are acting as a marker of risk (Hussong et al., 2011). Problems may begin to emerge as a result of neurocognitive changes during a crucial developmental period; the transition from early adolescence into adulthood (Colder et al., 2010). This period is represented by impulsive behaviour and risky decision-making thought to be mediated by interactions between two brain systems which are believed to be in competition with each other (Spear, 2000).

The first system is associated with increases in sensitivity to reward and an increase in motivation for positive arousal (Chambers, Taylor, & Potenza, 2003; Steinberg, 2007, 2008). The second system, described as an underdeveloped cognitive regulation system, leads to poor self-control of emotions and behaviours that are not fully mature until early adulthood (i.e., early 20s; Chambers et al., 2003; Steinberg, 2007, 2008). These brain systems are proposed to reflect reward drive and rash impulsiveness traits respectively (Gullo & Dawe, 2008). These cognitive changes occur during a period of heightened vulnerability; that is, when autonomy increases, parental interactions decrease and peer relationships become influential (Colder et al., 2010). Subsequently, individuals may begin using substances to cope with negative emotions or heighten positive emotions.

Similar to more recent models of comorbidity that describe interactive processes between symptoms of social anxiety and alcohol use (Kushner et al., 2000), the relationship between impulsivity and alcohol may not be simple cause and effect, but rather bidirectional, complex and incremental over time. For instance, impulsivity may act as a predisposing factor to drinking initiation, or may be a consequence of the neuroadaptive changes that result from chronic alcohol use (e.g., severity of dependence, years of drinking) (Dick et al., 2010; Jentsch & Taylor, 1999; Verdejo-Garcia et al., 2008). It is likely that an interaction between all of these factors exists (Kambouropoulos & Staiger, 2004), where prolonged alcohol

exposure results in additional impaired impulse control, and a cycle of increased impulsivity and alcohol consumption over time develops (de Wit, 2009; Verdejo-Garcia et al., 2008).

Relationship between Impulsivity and Alcohol Expectancies in Individuals with SAD-AUD Comorbidity.

Previous research has found that alcohol expectancy mediates the relationship between impulsivity and alcohol use with elevated levels of reward drive thought to predict a stronger expectation of positive outcomes, resulting in harmful alcohol consumption (Gullo et al., 2010). Similarly, research suggests that alcohol expectancies and reward drive moderate the relationship between alcohol use and social anxiety (Booth & Hasking, 2009). We observed limited relationships between alcohol expectancies and both subjective and autonomic cue reactions to alcohol stimuli and ingestion of alcohol (Chapter 4). Furthermore, both trait reward drive and rash impulsivity were found to be a stronger predictor of social anxiety and alcohol outcomes following treatment (Chapter 5). This unexpected pattern of findings may be due to lack of statistical power. An alternate explanation relates to the presentation of individuals in clinical samples. The high level of both alcohol dependence and social anxiety in the treatment-seeking sample (Chapters 4 and 5) suggests that once alcohol use has reached a certain threshold where the diagnosis meets criteria for alcohol dependence, relationships between these variables may not be found. Other variables such as comorbidity with other disorders, severity of symptoms and whether other situational variables match expectations may play a more prominent role (Morris et al., 2005). Furthermore, alcohol expectancies could interact with reward drive in the early stages and initiation of alcohol use, and play less of a role once problems are more established. The current findings point to this area as a useful direction for future research to investigate.

Implications for Treatment.

Several implications for treatment are indicated by our results. Firstly, results from Chapter 2 showed that patients who use alcohol as a safety behaviour report more severe symptoms of

social anxiety, but still benefit from treatment. A recent meta-analysis conducted by Schry and White (2013) found that social anxiety was negatively correlated with alcohol use variables (e.g., quantity and frequency of use), but was positively correlated with alcohol problems and symptoms of alcohol dependence. Thus, whether or not alcohol use negatively impacts symptoms of social anxiety may be related to how an individual perceives their alcohol use and problems caused by alcohol consumption, rather than their actual drinking levels. Our results also distinguish between drinking levels and drinking problems in relation to social anxiety. Specifically, drinking levels did not impact recovery from comorbid SAD-AUD (Chapter 2), but AUD diagnosis did negatively impact recovery from this comorbid condition. Furthermore, strategies that enhance regulation of aversive emotions, as well as identification and management of barriers to adaptive coping methods may assist in disrupting the processes that maintain social anxiety. As a consequence, this could potentially help in reducing the use on alcohol as a strategy to cope.

Secondly, patients, when provided cognitive behavioural therapy (CBT) to target alcohol use problems, individuals with high impulsivity actually benefit more (Chapter 5). This might be because learning effective coping skills to help with emotion regulation and problem resolution results in more adaptive coping efforts and fewer adverse alcohol-related consequences. Along these lines, a study by Litt, Babor, DelBoca, Kadden, and Cooney (1992) found grouping clients into distinct subgroups using Babor et al.'s (1992) type A/type B classification system was prognostic of outcome, and may be useful in matching clients to treatment. The more severe group (type B) fared best in the coping skills treatment, whereas those with less severe problems (type A) had better outcomes overall, and responded better to interactional therapy. The authors conclude that the more severe clients may be better suited to the more structured cognitive behavioural therapy (CBT), and benefit from clear goals and procedures (Litt et al., 1992).

Similarly, more recent research evaluating a school-based alcohol prevention program "Preventure" targeted at specific personality profiles including impulsivity and sensation-seeking, was effective in preventing the escalation in drinking and binge drinking in high-risk adolescents at six and twelve months following treatment (Conrod, Castellanos, & Mackie, 2008). Similar to the treatment provided in the sample from Chapter 5, the Preventure intervention incorporates motivational interviewing components, CBT and psychoeducation (Conrod, Stewart, Comeau, & Maclean, 2006). Thus, structured interventions that improve cognitive inhibitory control, and reduce impulsive decision-making may represent a beneficial treatment approach for individuals with co-occurring social anxiety and alcohol use disorders, who report high levels of rash impulsiveness (Chapter 5). Future investigations are needed to replicate the influence of impulsivity on social anxiety and alcohol outcomes in larger samples.

Summary.

Taken together, these studies provide answers to the research questions raised in Chapter 1. Yes, impulsivity plays a role in the relationship between social anxiety and alcohol use. This body of research indicates that the co-occurrence of social anxiety and alcohol use disorders might reflect different patterns of symptoms that result from shared risk factors such as reward drive and rash impulsivity. Specifically, trait and behavioural components of reward drive and trait rash impulsivity appear to play a role in who develops SAD-AUD comorbidity, how these problems are maintained, and how these individuals respond to treatment interventions. Individuals with comorbid social anxiety and alcohol use disorders and elevated impulsivity may benefit from a well structured therapeutic approach targeting coping skills to reduce their social anxiety and alcohol problems.

The Measurement and Conceptualisation of Impulsivity and Mental Disorders

Conceptualisation and Measurement of Impulsivity.

A number of mental disorders specify impulsivity as a core symptom in both the fourth edition of the *Diagnostic and Statistical Manual for Mental Disorders* (DSM–IV; American Psychiatric Association, 2000) and the *International Statistical Classification of Diseases and Related Health Problems* (ICD-10; Organisation, 2008). Given the prominence of impulsivity in the identification of numerous psychiatric disorders, it is somewhat surprising that the way in which impulsivity is measured and conceptualised, differs to a great degree in the literature (Whiteside & Lynam, 2001). Research has endeavoured to understand impulsivity from a range of theoretical perspectives, such as behavioural and personality approaches. Here impulsivity is defined as an observable behaviour (i.e., Barratt, 1993) or an individual trait that influences behaviour (i.e., Eysenck & Eysenck, 1977) respectively. Within these frameworks, several measures exist to assess similar constructs within the domain of impulsivity, resulting in considerable overlap between different models (Depue & Collins, 1999; Gullo et al., 2014). Thus, research between impulsivity and alcohol use has been studied using an assortment of different measures, complicating research in this area and making comparisons across studies challenging. In this thesis, impulsivity was assessed based a contemporary model derived from factor analytic studies and research on the neuroscience of substance use (Dawe et al., 2004), and both self-report and behavioural measures of reward drive and rash impulsivity were used that have been previously implicated in substance use research (e.g., George et al., 2010; Kabbani & Kambouropoulos, 2013; Kambouropoulos & Staiger, 2004b; Verdejo-Garcia et al., 2008).

While there is general consensus that impulsivity is multi-dimensional, disagreement still exists over the number and type of factors that are most important to understanding vulnerability to addiction (Gullo et al., 2014). Gullo et al. (2014) provide compelling evidence for the use of a two-factor model over models with additional components. Specifically, they

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argue that a two-factor framework incorporates evidence from neuroscience relating to specific neurocognitive pathways, thus providing the most parsimonious account with adequate explanatory power. In contrast, in other models with additional scales such as the UPPS-P (Cyders et al., 2007), considerable overlap exists between the five scales and research indicates that only one of these scales (Negative Urgency), provides unique contribution to substance use (Gullo et al., 2014). Gullo et al. raise the important question of whether the tendency to partake in rash action when experiencing high negative emotion (i.e., negative urgency) is fundamentally different from a general tendency to engage in rash action. That is, does negative urgency as an additional factor offer unique contribution more than trait rash impulsivity described in more parsimonious models including the two-component framework?

Negative urgency could provide an alternate explanation for the results obtained in Chapter 3 which found that individuals with comorbid social anxiety and alcohol use reported higher negative affect during exposure to the alcohol cue compared to the water cue. The increase in negative affect reported by individuals with this comorbidity may cue desire for alcohol as a result of conditioning that occurs if an individual has repeatedly consumed alcohol to alleviate undesirable emotions. For individuals with heightened negative urgency, strong negative affect may prompt involvement in harmful drinking behaviour in order to alleviate or distract from adverse emotions, despite the long-term harmful consequences of these actions (Whiteside & Lynam, 2001). Rash actions triggered by negative emotion provide immediate reinforcement and subsequently, an individual may perform the risky behaviour more often (Cyders et al., 2007). However, negative urgency was not directly measured in this study and thus deserves further exploration in future studies to examine whether it offers unique contribution above and beyond alternate models of impulsivity. Additionally, future research comparing theoretical models with additional factors to examine whether these other

components add unique variance to predictions relating to substance use above the two-factor model will assist in working towards a more uniform conceptualisation of impulsivity.

Conceptualisation and Measurement of Mental Disorders.

This thesis assessed the comorbidity between social anxiety and alcohol use disorders using a semi-structured interview, the Anxiety Disorder Interview Schedule for DSM-IV (ADIS; Brown et al., 1994). The use of a semi-structured interview focuses on the presence and severity of symptoms and permitted differential diagnosis among the anxiety disorders and substance use disorders. Disorders were thus measured, analysed and conceptualised as dichotomous constructs (i.e., presence/absence) according to DSM-IV criteria based on the presence of a set number of symptoms and indicated by a diagnostic threshold (Krueger et al., 1998). Given that when these two disorders are present in an individual, treatment outcomes are less effective and problems are often more debilitating, it was important to assess these problems in individuals with higher levels of severity.

A further motivation for including individuals with more severe symptoms was previous research showing that individuals with severe/clinical levels of social anxiety disorder and/or alcohol use disorders respond differently to alcohol and alcohol-related cues than those individuals with subclinical or lower levels (e.g., Fox et al., 2007; Sinha et al., 2009). Indeed, this was illustrated by the results in Chapter 3. Furthermore, there was weak evidence in Chapter 2 to suggest that there may be diagnostic threshold. The sample of individuals included in this study were adults with social anxiety disorder seeking treatment for their anxiety. There were some individuals in this sample who reported no, mild or moderate alcohol use, in addition to those that reported heavy or harmful use. Only a very small number ($n = 7$) were reported to have a comorbid alcohol use disorder. The findings suggest that individuals with social anxiety disorder are able to be successfully treated for their anxiety, even when consuming alcohol at hazardous levels, as nearly a quarter of the sample reported (e.g., in both frequency and quantity of alcohol use). However, there was weak evidence

suggesting that individuals with comorbid alcohol use disorders tend to do worse on average as treatment progressed, with fewer reductions in anxiety symptoms compared to those without this comorbidity. These findings point to future directions that would be useful to investigate, specifically, replication with a larger number of individuals with comorbid alcohol use disorders.

It is important to note, however, that harms and consequences are not just explained by the presence of a disorder. For example, in DSM-III, substance dependence was limited to the occurrence of physiological tolerance or withdrawal symptomatology. In a revised version (DSM-III-R Carroll, Rounsaville, & Bryant, 1994), the diagnostic manual was expanded to include the importance of the substance to an individual, in addition to continued use and subsequent concerns for restricting use (Schuckit et al., 1999). Current research suggests that any disproportionate craving, urge, motivation, attention for a substance is effectively a neurophysiological pathology (Goldstein & Volkow, 2011; Gould, 2010; Grant, Brewer, & Potenza, 2006). Drug craving has only recently been added as a criterion for an alcohol use disorder in DSM-5 as it was not included in DSM-IV (American Psychiatric Association, 2013). Subsequently, Widiger and Sankis (2000) argue that DSM may overlook less obvious physiological withdrawal that might account for future behavioural relapses that occur outside of the DSM defined boundaries of withdrawal. Furthermore, when disorders are conceptualised as present/absent on the basis of a threshold, the association between two disorders can differ with variation in presenting symptoms, thus changing the threshold of one or both disorders (Widiger & Trull, 2007).

Whilst the use of DSM diagnoses provides agreement on observable symptoms, signs or behaviours and thus has high reliability, these symptoms may reflect multiple mechanisms and therefore lack validity (Widiger & Lynam, 1998). A more thorough understanding of the risk factors of a disorder and its progress may require a recognition of the underlying factors, such as genetic or environmental factors, and the interaction between them (Widiger &

Lynam, 1998). This has lead to the establishment of the Research Domain Criteria (RDoC): a framework for research on mental disorders that aims to integrate findings from genetic, molecular and cellular and current behaviour-brain systems neuroscience research that is associated with mental disorders (Insel et al., 2010). Using the RDoC as a complementary framework alongside the current classification systems for characterising disorders will likely reveal additional processes that underlie complex behaviours such as those that arise from comorbidity. Specifically, by better defining the phenotype, the causes and mechanisms may be easier to identify.

Methodological Considerations

Self-report measures are generally used to measure longer-term, relatively stable trait characteristics. The self-report scales assessing trait impulsivity measure a range of specific daily behaviours and arise from interactions between biological, cognitive and psychosocial processes; providing them with good ecological validity (Moeller et al., 2001). However, they are reliant on the assumption that individuals have accurate insight into their behaviour and symptoms when completing it (Moeller et al., 2001). This may be particularly problematic among those individuals with heightened trait impulsivity, who may be limited by the extent to which they are able to accurately report upon internal processes. Similarly, due to the effects of chronic alcohol use, individuals with alcohol problems may also lack insight or cognitive capacity to accurately report their own behaviour.

Given these issues, behavioural measures have been introduced as an objective way to examine impulsivity which are less affected by demand effects and lack of insight (Moeller et al., 2001). Behavioural measures of impulsivity are more sensitive to temporal fluctuations, and thus reflect state-like variations in dimensions of impulsivity (Verdejo-Garcia et al., 2008). However, generalisability to daily behaviour is somewhat limited, and performance on these tasks may not accurately reflect rash impulsive action that takes place in real-life situations (Evenden, 1999; Moeller et al., 2001). Moreover, these behavioural tasks may

assess different aspects of the construct, with some studies showing inconsistent and weak associations between behavioural and self-report measures of impulsivity (e.g., Dougherty, Mathias, Marsh, & Jagar, 2005; Lane, Cherek, Rhoades, Pietras, & Tcheremissine, 2003; Reynolds, Ortengren, Richards, & de Wit, 2006). Furthermore, behavioural measures of impulsivity may be assessing several different cognitive processes and executive functions, and deficits in performance could signal other cognitive shortfalls in addition to, or instead of impulsivity, that are essential for decision-making (e.g., attention and memory) (Dick et al., 2010; Noel et al., 2011).

A strength of this thesis was the multi-method approach using a combination of self-report and behavioural assessments of impulsivity. By including both objective and self-report measures, we have been able to identify firstly, that impulsivity is likely to be a multi-dimensional construct as was reflected in our results from Chapters 3, and 4 that found differences between trait and behavioural measures of impulsivity. However, it is a limitation of this thesis that the IGT was not included in all studies (only in Chapters 3 and 4). Secondly, we have identified the components most relevant to understanding the relationship between social anxiety and alcohol use disorders. Specifically, trait and behavioural components of reward drive and trait rash impulsivity appear to be factors that play a role in this relationship (Chapters 3 and 4).

Similarly, objective assessment of psychophysiological arousal can yield different results to self-report measures. As indicated by the findings in Chapter 4, physiological responses to alcohol, placebo alcohol, and alcohol-related stimuli differed from self-reported craving and subjective stimulant and sedative effects. This may have been due to the choice of baseline in the current studies. Baseline measurements in Chapters 3 and 4 were taken in the simulated laboratory bar set up rather than a neutral environment (i.e., without alcohol cues present) which may have contributed to elevated levels of self-reported craving prior to actually receiving and ingesting alcohol. Our findings demonstrate how alcohol use may be

maintained in clinical individuals via differences in physiological and subjective experiences to alcohol and alcohol stimuli (Chapter 4). Thus, as shown by these results, it is crucial to go beyond self-report methodologies and incorporate diverse evaluation methods in order to understand the complex and multifaceted effects of alcohol ingestion. Furthermore, it is important to consider the most appropriate type of pre-manipulation baseline relevant to research questions.

Finally, the use of a naturalistic bar environment allowed examination of the important role of non-pharmacologic factors (e.g., contextual stimuli) in both clinical (Chapter 4) and non-clinical samples (Chapter 3). Results from both of these studies demonstrate the important role of contextual cues that may become conditioned stimuli from their association with the effects of alcohol through drinking. However, while the use of a simulated bar environment allows researchers to understand the effects of alcohol as they occur in real life, they are also limited by alcohol administration procedures such as the number, and dispensation, of drinks. For instance, a strength of the study in Chapter 4 was the administration of vodka in a cocktail mix; a common alcoholic drink consumed at bars and parties. This particular mode of administration has likely been paired with previous drinking experiences, and thus may be receptive to the influence of alcohol expectancies. Subsequently, this may have greater ecological validity and better reflect "real-life" drinking episodes. However, the dosing paradigm has limited ability to reflect maintenance of a drinking episode. Specifically, once an individual decides to consume five or more drinks, for example, this becomes difficult to model in a laboratory setting. Moreover, the pharmacological and expectancy effects of having five or more drinks may differ to the administration of two to four standard drinks. Few studies have been conducted to examine excessive levels of drinking in individuals with alcohol use disorders due to concerns that alcohol consumption would be detrimental to their condition (Wolitzky-Taylor et al., 2011) despite no compelling evidence that partaking in this type of research *per se* has adverse effects on alcohol dependent individuals (Dolinsky &

Babor, 1997). Thus, excluding individuals with more acute problems severely impacts upon the quality of the research. Future research using a naturalistic bar environment with careful alcohol administration, involving clinical participants with care (i.e., using our exclusion criteria; Chapter 4), is a promising way to conduct more ecologically valid research within these important ethical restrictions.

Limitations and Future Directions

This thesis has methodological strengths in its well-characterised sample of alcohol-dependent individuals with social anxiety disorder, combination of cross-sectional, clinical trial and laboratory data, variety of methods utilising self-report, psychophysiological and behavioural measures, and application of a contemporary framework of impulsivity giving rise to innovative questions. However, there are some limitations that were evident across the empirical studies will be discussed in greater detail here.

The main limitation of the studies included in this thesis were relatively small sample sizes. Practical considerations including the costs of funding larger samples, availability of suitable participants, duration of study, and resources were factors that contributed to the sample sizes in the studies included in this thesis. However, whilst the number of participants studied were comparable to the research literature in this area, the small sample sizes limit generalisability to the general population. It will be important for the findings to be replicated in larger representative samples.

Another limitation that deserves further mention relates to the relationship between cue-elicited responses such as craving and autonomic arousal and drug-seeking behaviours. While it is widely accepted that exposure to alcohol and stimuli that signal drinking in individuals with alcohol dependence may contribute to alcohol use and relapse, the studies in this thesis did not directly measure behavioural outputs such as actual consumption (in the laboratory) or relapse to drinking. The preceding chapters did, however, consider craving from a number of different angles. Despite the advantages of defining and evaluating craving in terms of actual

behaviour, individuals do not always describe experiencing craving prior to every drinking occurrence, nor does an awareness of craving translate into actual drug-seeking or consumption behaviour (Tiffany, 1990; Tracy, 1994). Recreational and problematic alcohol use do not exist in a vacuum, but are often part of a wider set of social, mood and contextual factors in addition to craving, such as affect, abstinence goals, type and feature of stimuli, availability of the drug and expectations of consumption (Cox & Klinger, 2004). Thus, the mechanisms inferred from elevated cue reactivity on their own may not equate to problematic alcohol use, rather they may increase the risk for it.

Finally, it is also a potential limitation that some studies included in this thesis (Chapters 3 and 4) allowed for inclusion of individuals with sub-threshold symptoms of social anxiety and alcohol use disorders whereas others (Chapters 2 and 5) required full diagnostic threshold to be met for inclusion. This impacts direct comparability of the studies to one another.

The findings presented in this thesis point towards some interesting future directions beyond those already discussed. Future research, with larger samples, should continue to assess the direct and indirect effects of impulsivity on drinking behaviour in individuals with social anxiety and alcohol use disorders. Specifically, a better understanding of the cognitive processes and mechanisms underlying cue reactivity could inform models of comorbidity, and investigating whether elevated cue reactivity leads to problematic use or less successful treatment outcomes may assist in developing predictions about early markers and individual factors to target in treatment. Equally important, an understanding of how impulsivity, social anxiety and alcohol problems emerge from a developmental perspective is a critical theoretical and empirical direction. Examination of these factors in young adults is required as adolescence is a time during which individuals are at risk for substance experimentation and harmful use, and may reveal important mechanisms involved in how these problems develop. Specifically, clarity regarding for whom and when social anxiety operates as a risk/protective factor is an important avenue for prevention.

Conclusion

Social anxiety and alcohol use disorders are common and prevalent problems, and together these disorders have been associated with marked distress, less successful treatment outcomes and decreased quality of life. The studies included in this thesis are some of the first to investigate the role of impulsivity in this comorbid relationship. These studies have combined different methods and utilised a contemporary model of impulsivity drawing from addiction research, in clinical and non-clinical samples, with varying levels of social anxiety and alcohol problems. This thesis confirm the role of impulsivity in alcohol risk, and uniquely extend results to individuals with additional social anxiety. Specifically, in individuals with comorbid SAD-AUD, reward drive and rash impulsivity may predispose an individual to alcohol problems and/or maintain established alcohol dependence through mechanisms such as heightened vulnerability to stimulant effects of alcohol, increased cue-elicited responses in the presence of alcohol-related cues and ingestion of alcohol, and a propensity to approach rewarding stimuli and engage in risky behaviour. This thesis also demonstrates how the 2-CARS framework may be one way to provide new insights into the SAD-AUD relationship, and advance our understanding of the relationship between these commonly co-occurring problems.

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Appendix A

Ethics Approval

MACQUARIE
University

Mirjana Subotic <mirjana.subotic@mq.edu.au>

Ethics application reference-5201001453- Final approval
3 messages

Ethics Secretariat <ethics.secretariat@mq.edu.au>   Mon, Dec 20, 2010 at 12:41 PM
To: Dr Andrew James Baillie <andrew.baillie@mq.edu.au>
Cc: Ms Kristen Tulloch <kristen.tulloch@mq.edu.au>, Miss Mirjana Subotic <mirjana.subotic@mq.edu.au>

Dear Dr Baillie

Re: "Alcohol cravings, impulsivity and cue reactivity: How do socially
phobi and non-socially phobic individuals differ?" (Ethics Ref: 5201001453)

Thank you for your recent correspondence. Your response has addressed the
issues raised by the Human Research Ethics Committee and you may now
commence your research.

The following personnel are authorised to conduct this research:

Dr Andrew James Baillie- Chief Investigator/Supervisor
Miss Mirjana Subotic & Ms Kristen Tulloch- Co-Investigators

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL
EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

1.   The approval of this project is conditional upon your continuing
compliance with the National Statement on Ethical Conduct in Human Research
(2007).

2.   Approval will be for a period of five (5) years subject to the provision
of annual reports. Your first progress report is due on 20 December 2011.

If you complete the work earlier than you had planned you must submit a
Final Report as soon as the work is completed. If the project has been
discontinued or not commenced for any reason, you are also required to
submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/
human_research_ethics/forms

3.   If the project has run for more than five (5) years you cannot renew
approval for the project. You will need to complete and submit a Final
Report and submit a new application for the project. (The five year limit
on renewal of approvals allows the Committee to fully re-review research in
an environment where legislation, guidelines and requirements are
continually changing, for example, new child protection and privacy laws).

4.   All amendments to the project must be reviewed and approved by the
Committee before implementation. Please complete and submit a Request for
Amendment Form available at the following website:

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/
human_research_ethics/forms

5.   Please notify the Committee immediately in the event of any adverse

effects on participants or of any unforeseen events that affect the
continued ethical acceptability of the project.

6.   At all times you are responsible for the ethical conduct of your
research in accordance with the guidelines established by the University.
This information is available at the following websites:

http://www.mq.edu.au/policy/

http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/
human_research_ethics/policy

If you will be applying for or have applied for internal or external
funding for the above project it is your responsibility to provide the
Macquarie University's Research Grants Management Assistant with a copy of
this email as soon as possible. Internal and External funding agencies will
not be informed that you have final approval for your project and funds
will not be released until the Research Grants Management Assistant has
received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external
organisation as evidence that you have Final Approval, please do not
hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of
final ethics approval.

Yours sincerely
Dr Karolyn White
Director of Research Ethics
Chair, Human Research Ethics Committee

MACQUARIE UNIVERSITY

Lexine Stapinski <lexine.stapinski@mq.edu.au>

FW: (HE28MAR2008-R05758). Randomized controlled trial of treatment for alcohol use disorders and social phobia

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Ethics Secretariat <ethics.secretariat@mq.edu.au>   **Wed, May 19, 2010 at 1:26 PM**
To: Lexine Stapinski <lexine.stapinski@mq.edu.au>
Cc: Andrew Baillie <andrew.baillie@mq.edu.au>

Dear Lexine and Andrew

RE: Randomized controlled trial for alcohol use disorders and social phobia (REF: HE28MAR2008-R05758)

Thank you for resending your responses to the sub-Committee's queries regarding your amendment request (dated 26 November 2008). Your responses are fine and the following amendments to the above study have been approved:

1. The addition of Ms Lexine Stapinski, Ms Kristen Tulloch, Ms Sonia Haggman and Ms Mirjana Subotic as personnel to the study.

2. The addition of the following measures to the assessment battery:

(i) the Glasgow Sleep Effort Scale (Broomfield & Espie, 2008)
(ii) The Family Tree Questionnaire (Mann, Sobell, Sobell & Pavan 1985)
(iii) Insomnia Severity Index (Morin, 1993)
(iv) Alcohol Abstinence Self-efficacy Scale (DiClemente, Carbonari, Montgomery & Hughes 1994)
(v) Sheehan Disability Scale (Sheehan, Harnett-Sheehan & Raj, 1996)
(vi) Credibility Expectancy Questionnaire (Devilly & Borkovec 2000)
(vii) Drinker Inventory of Consequences (Forcehimes, Tonigan, Miller, Kenna & Baer 2007)
(viii) The Obsessive Compulsive Drinking Scale (Anton, Moak & Latham 1995)
(ix) Alcohol Expectancies for Social Evaluative Situations Scale (Bruch, Rivet, Heimberg & Levin 1997)

3. The removal of the Newcastle Alcohol Problems Scale from the assessment battery.

4. To collect the questionnaires online.

5. A change in the recruitment strategy. As well as online recruitment you will also offer the participants the opportunity to complete an online screening for the project. This will involve the completion of the Alcohol Use Disorders Identification Test (AUDIT) and the Mini Social Phobia Inventory (MiniSPIN). A flyer will also be used to recruit participants.

6. Participants will receive individual treatment rather than group treatment.

7. Blood will be collected by Douglass Hanly Moir. Costs will be covered by the NHMRC grant for this project.

8. Consent forms have been updated.

Please accept this email as formal notification that the above amendments have been approved. Please be advised that this approval does not extend to your amendment request to include stories of recovery via the website or media interview (submitted April 23 2010).

If you have any questions or concerns please do not hesitate to contact the Ethics Secretariat on the contact details shown below.

Nicola Myton

[Quoted text hidden]

--
Office of the Deputy Vice Chancellor (Research)

Ethics Secretariat

Research Office
Level 3, Research HUB, Building C5C
Macquarie University
NSW 2109

Ph: +61 2 9850 6848
Fax: +61 2 9850 4465

Email:

For Enquiries: ethics.secretariat@mq.edu.au

Appendix B

MAUQ Questionnaire

MAUQ

Instructions: Please **colour in the circle** next to your answer to each of the questions. **DO NOT tick, cross or circle your answer. Please use a blue or black pen.** DO NOT use a pencil.

1. How often do you have a standard alcoholic drink (ie, one glass of wine or beer, one nip of spirits)?

   ○   Never
   ○   Once or twice a month
   ○   Once or twice a week
   ○   Once or twice a day
   ○   More than twice a day

2. In a typical drinking session, how many standard drinks do you consume?   _____

3. In the <u>past month</u>, what is the largest number of standard drinks you have consumed in one session?

                          _____

4. When are you <u>most</u> likely to drink:

   ○   When you feel down or blue
   ○   When you feel anxious or uptight
   ○   When you are having a good time
   ○   Other _____

5. Typically, how many alcoholic drinks would you have in the following situations?

	None	One	Two	Three or more
a. Going to a party with strangers				
Before going	○	○	○	○
During the party	○	○	○	○
After the party	○	○	○	○
b. Watching TV at home				
While watching	○	○	○	○
c. Going for a job interview				
Before going	○	○	○	○
After the interview	○	○	○	○
d. Going on a date				
Before going	○	○	○	○
During the date	○	○	○	○
After the date...................................	○	○	○	○

Typically, how many alcoholic drinks would you have in the following situations?

	None	One	Two	Three or more
e. Eating at a restaurant with close friends/family				
Before going to the restaurant.....................	○	○	○	○
At the restaurant	○	○	○	○
After returning home	○	○	○	○
f. Eating at a restaurant with acquaintances				
Before going to the restaurant	○	○	○	○
At the restaurant	○	○	○	○
After returning home	○	○	○	○
g. An important meeting, (eg. at work, parent/teacher night				
Before the meeting	○	○	○	○
h. Giving a speech or presentation in front of others				
Before the presentation	○	○	○	○
After the presentation	○	○	○	○
i. During dinner at home	○	○	○	○
j. Meeting a group of new people				
Before meeting them	○	○	○	○
After meeting them	○	○	○	○
k. Phoning a friend to ask them to dinner or a movie				
Before calling	○	○	○	○
While in the phone	○	○	○	○
After calling	○	○	○	○
l. When having difficulty sleeping	○	○	○	○
m. Calling a company to complain about poor service				
Before calling	○	○	○	○
While on the phone.....................................	○	○	○	○
After calling...	○	○	○	○

Appendix C

Supplementary Tables

Supplementary Table 1. Unadjusted means and standard deviations of predictor and outcome variables separated by treatment allocation

Variable	Alcohol Only (*n* = 28)	Integrated (*n* = 32)
Gender, % Female	53.6	25
ADIS Alcohol CSR, mean (SD)	5.89 (1.20)	5.84 (1.11)
ADIS Social Anxiety CSR, mean (SD)	5.43 (.96)	5.42 (.91)
Baseline, mean (SD)		
SIAS-SPS total score	79.54 (23.35)	83.34 (22.57)
SADQ	14.75 (9.24)	17.41 (11.97)
Total no. drinks, past 30 days	185.74 (135.69)	188.98 (164.49)
Number of drink days, past 30 days (%)	19.43 (8.13)	16.88 (8.24)
I_7-IMP	9.39 (3.84)	9.75 (4.04)
BAS-Drive	9.79 (2.23)	9.81 (2.93)
BAS-Reward	16.00 (2.21)	16.63 (2.32)
AESES	42.00 (8.40)	40.59 (7.18)
TR-AE	27.00 (5.80)	27.34 (5.90)
SL-AE	19.32 (5.77)	19.66 (3.91)
1 month follow-up, mean (SD)		
SIAS-SPS total score[a]	57.48 (28.42)	46.67 (17.32)
SADQ[a]	9.10 (8.22)	11.04 (10.92)
Total no. std. drinks, past 30 days[b]	50.77 (49.31)	58.56 (71.96)
Number of drink days, past 30 days (%)[b]	9.25 (8.10)	9.56 (8.66)
3 month follow-up, mean (SD)		
SIAS-SPS total score[c]	57.50 (30.54)	43.59 (17.54)
SADQ[c]	7.21 (7.84)	8.59 (8.52)
Total no. drinks, past 30 days[d]	69.75 (86.08)	133.24 (209.69)
Number of drink days, past 30 days (%)[d]	11.70 (11.37)	14.73 (9.01)
6 month follow-up, mean (SD)		
SIAS-SPS total score[e]	49.13 (28.90)	41.10 (15.77)
SADQ[e]	4.31 (3.45)	7.10 (6.46)
Total no. drinks[g], past 30 days[f]	52.42 (59.54)	46.70 (56.41)
Number of drink days, past 30 days (%)[f]	10.00 (10.11)	9.17 (7.88)

Note. ADIS = Anxiety Disorders Interview Schedule; CSR = Clinician Severity Rating; SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; TLFB: Time Line Follow-Back; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social Lubrication Alcohol Expectancy; BAS-Drive = Behavioural Activation System-Drive subscale; BAS-Reward = Behavioural Activation System-Reward Responsiveness subscale; I_7-IMP = Impulsiveness subscale; AESES = Alcohol Expectancies for Social Evaluative Situations Scale.

[a] *n* = 28 for alcohol only and *n* = 32 for integrated; [b] *n* = 45 for total sample, *n* = 20 for alcohol only and *n* = 25 for integrated; [c] *n* = 14 for alcohol only and *n* = 22 for integrated; [d] *n* = 46 for total sample, *n* = 20 for alcohol only and *n* = 26 for integrated; [e] *n* = 13 for alcohol only and *n* = 21 for integrated; [f] *n* = 40 for total sample *n* = 17 for alcohol only and *n* = 23 for integrated.

Supplementary Table 2. Summary of correlations for impulsivity, alcohol and social anxiety variables

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. I₇-IMP	-	.46**	.17	.09	-.01	.07	.24	-.12	-.03	-.11	.21	.17	.03	.50**	.10	.06	-.06	-.18	-.14	.01	-.09	-.28
2. BAS-Drive		-	.42**	.04	-.03	-.08	.02	-.05	-.02	-.25	.06	.30*	.21	.15	.25	.07	.20	-.13	.09	.07	.04	-.10
3. BAS-Reward			-	.01	-.10	-.05	.12	-.01	-.01	-.20	.12	.27	.08	.13	.19	.01	.07	.18	.05	.04	-.17	.14
4. AESES				-	.32*	.56*	.14	.09	-.07	-.02	.05	.15	-.003	-.08	.12	.07	.07	-.12	-.07	-.003	-.18	-.21
5. TR-AE					-	.54**	.33*	.14	.17	.15	.48**	.27	.34*	.36*	.25	.10	.25	.03	.09	-.05	.12	-.08
6. SL-AE						-	.30*	.12	.06	.20	.32*	.15	.03	.03	.04	-.01	-.04	-.03	-.10	-.17	-.13	-.23
7. Pre SPS-SIAS							-	.39**	.24	.18	.39**	.07	-.07	.14	-.27*	-.06	-.08	-.17	-.38**	-.22	-.28	-.30
8. Post SPS-SIAS								-	.82**	.80**	.15	.13	.22	-.03	.01	.04	.04	-.12	-.05	-.11	-.19	-.26
9. 3m SPS-SIAS									-	.82**	.16	.15	.46**	.13	.05	-.04	.12	-.15	.02	-.13	-.06	-.17
10. 6m SPS-SIAS										-	.02	.04	.25	.02	-.12	-.04	-.07	.07	-.05	-.08	-.08	-.06
11. Pre SADQ											-	.73**	.53**	.69**	.27*	-.11	.18	-.26	-.18	-.32*	-.09	-.43**
12. Post SADQ												-	.75**	.60**	.56**	.08	.44**	-.27	.03	-.29	.02	-.34**
13. 3m SADQ													-	.41*	.67**	-.05	.50**	-.34	.06	-.27	-.03	-.35*
14. 6m SADQ														-	.37*	.21	.24	-.04	.15	.12	.25	-.11
15. Pre TLFB TD															-	.31*	.76**	.13	.63**	.16	.47**	.06
16. Post TLFB TD																-	.41**	.38*	.25	.82**	.54**	.37*
17. 3m TLFB TD																	-	.06	.39**	.14	.62**	-.04
18. 6m TLFB TD																		-	.46**	.48**	.29	.81**
19. Pre TLFB DD																			-	.43**	.60**	.54**
20. Post TLFB DD																				-	.50**	.65**
21. 3m TLFB DD																					-	.33*
22. 6m TLFB DD																						-

Note. I₇-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation System – Drive subscale; BAS-Reward = Behavioural Activation System – Reward Responsiveness subscale; AESES = Alcohol Expectancies for Social Evaluative Situations Scale; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social Lubrication Alcohol Expectancy; SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; TLFB TD = Time Line Follow–Back Total Drinks; TLFB DD = Time Line Follow–Back Drink Days.

* $p < .05$; ** $p < .001$

Supplementary Table 3. Final random regression model predicting SIAS-SPS symptom change with treatment after multiple imputation of missing data

Variable	Coefficient	SE	t	p	95% CI
Gender	-0.29	5.43	-0.05	0.96	-10.95 to 10.36
Allocation	1.51	5.61	0.27	0.79	-9.49 to 12.51
Allocation × Time (1MFU)	-15.33	6.76	-2.27	0.02*	-28.60 to -2.07
Allocation × Time (3MFU)	-17.02	9.05	-1.88	0.06†	-34.79 to 0.75
Allocation × Time (6MFU)	-13.91	9.22	-1.51	0.13	-32.01 to 4.18
Baseline SADQ	0.89	0.29	3.10	0.002**	0.33 to 1.46
Baseline drinks per day	-0.99	0.48	-2.07	0.04*	-1.93 to -0.05
I_7-IMP	1.52	.77	1.98	0.048*	.01 to 3.03
I_7-IMP × Time (1MFU)	-1.94	.81	-2.39	0.02*	-3.54 to -.35
I_7-IMP × Time (3MFU)	-1.41	1.15	-1.22	0.03*	-3.67 to .86
I_7-IMP × Time (6MFU)	-1.57	1.14	-1.37	0.02*	-3.82 to .68
BAS-Drive	-.64	1.09	-0.58	0.56	-2.78 to 1.50
BAS-Reward	.67	1.10	0.60	0.55	-1.49 to 2.82

Note. SADQ = Severity of Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale

† $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Supplementary Table 4. Final random regression model predicting SADQ symptom change with treatment after multiple imputation of missing data

Variable	Coefficient	SE	*t*	*p*	95% CI
Gender	1.82	1.54	1.18	0.24	-1.20 to 4.83
Allocation	1.22	1.49	0.82	0.41	-1.70 to 4.14
Baseline SPS-SIAS	.16	.04	3.95	0.000***	.08 to .24
SPS-SIAS × Time (1MFU)	-.16	.05	-3.03	0.002**	-.26 to -.06
SPS-SIAS × Time (3MFU)	-.17	.06	-3.00	0.003**	-.28 to -.06
SPS-SIAS × Time (6MFU)	-.16	.05	-3.14	0.002**	-.26 to -.06
Baseline drinks per day	1.06	.14	7.47	0.000***	.78 to 1.34
Baseline drinks per day × Time (1MFU)	-.21	.16	-1.30	0.192	-.53 to .11
Baseline drinks per day × Time (3MFU)	-.30	.24	-1.25	0.212	-.78 to .17
Baseline drinks per day × Time (6MFU)	-.86	.17	-4.94	0.000***	-1.21 to -.52
I_7-IMP	-.03	.27	-0.11	0.910	-.56 to .50
I_7-IMP × Time (1MFU)	-.22	.33	-0.67	0.51	-.87 to .43
I_7-IMP × Time (3MFU)	.70	.36	1.96	0.05[†]	-.001 to 1.40
I_7-IMP × Time (6MFU)	-.40	.42	-0.95	0.340	-1.22 to .42
BAS-Drive	-.26	.42	-0.61	.54	-1.08 to .57
BAS-Drive × Time (1MFU)	.89	.50	1.76	0.078[†]	-.10 to 1.87
BAS-Drive × Time (3MFU)	.66	.64	1.03	0.30	-.59 to 1.90
BAS-Drive × Time (6MFU)	-.34	.52	-0.65	0.52	-1.36 to .68
BAS-Reward	-.24	.44	-0.54	0.59	-1.10 to .62
BAS-Reward × Time (1MFU)	.39	.52	0.75	0.45	-.63 to 1.42
BAS-Reward × Time (3MFU)	-.19	.62	-0.30	0.77	-1.40 to 1.03
BAS-Reward × Time (6MFU)	.25	.54	0.46	0.64	-.80 to 1.30

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale;

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Supplementary Table 5. Final random regression model predicting TLFB drinks per day change with treatment after multiple imputation of missing data

Variable	Coefficient	SE	*t*	*p*	95% CI
Gender	-1.91	1.45	-1.31	0.19	-4.76 to .94
Allocation	-.68	1.54	-0.44	0.66	-3.69 to 2.33
Allocation × Time (1MFU)	1.21	2.19	0.55	0.58	-3.09 to 5.51
Allocation × Time (3MFU)	-.46	2.10	-0.22	0.83	-4.59 to 3.68
Allocation × Time (6MFU)	.23	1.87	0.12	0.90	-3.43 to 3.90
Baseline SPS-SIAS	-.02	.03	-0.48	0.63	-.09 to .05
Baseline SADQ	.39	.07	5.21	0.000***	.24 to .54
SADQ × Time (1MFU)	-.23	.09	-2.57	0.01*	-.40 to -.05
SADQ × Time (3MFU)	-.17	.09	-1.89	0.06†	-.34 to .01
SADQ × Time (6MFU)	-.30	.10	-3.18	0.002*	-.49 to -.12
I_7-IMP	.14	.22	0.65	0.52	-.29 to .58
I_7-IMP × Time (1MFU)	-.72	.30	-2.40	0.02*	-1.31 to -.13
I_7-IMP × Time (3MFU)	-.33	.28	-1.17	0.24	-.88 to .22
I_7-IMP × Time (6MFU)	-.33	.29	-1.13	0.26	-.90 to .24
BAS-Drive	.23	.35	0.66	0.51	-.46 to .92
BAS- Drive × Time (1MFU)	.67	.47	1.41	0.16	-.26 to 1.60
BAS- Drive × Time (3MFU)	.30	.44	0.67	0.50	-.57 to 1.16
BAS- Drive × Time (6MFU)	.02	.46	0.04	0.96	-.88 to .92
BAS-Reward	.34	.36	0.93	0.35	-.37 to 1.05
BAS-Reward × Time (1MFU)	-.69	.44	-1.57	0.12	-1.55 to .17
BAS-Reward × Time (3MFU)	-.39	.42	-0.93	0.35	-1.22 to .44
BAS-Reward × Time (6MFU)	-.33	.39	-0.83	0.41	-1.10 to .45

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale.

† *p* < .10; * *p* < .05; ** *p* < .01; *** *p* < .001

Supplementary Table 6. Final random regression model predicting TLFB number of drinking days change with treatment after multiple imputation of missing data

Variable	Coefficient	SE	*t*	*p*	95% CI
Gender	1.36	2.18	0.63	0.53	-2.91 to 5.64
Allocation	-2.13	2.41	-0.89	0.38	-6.86 to 2.59
Allocation × Time (1MFU)	4.20	3.37	1.24	0.21	-2.42 to 10.82
Allocation × Time (3MFU)	4.45	3.46	1.29	0.20	-2.34 to 11.23
Allocation × Time (6MFU)	-.18	3.47	-0.05	0.96	-7.00 to 6.63
Baseline SPS-SIAS	-.13	.05	-2.57	0.01*	-.23 to -.03
Baseline SADQ	-.11	.16	-0.71	0.48	-.43 to .20
SADQ × Time (1MFU)	-.24	.21	-1.13	0.26	-.65 to .18
SADQ × Time (3MFU)	-.01	.20	-0.03	0.98	-.401 to .391
SADQ × Time (6MFU)	-.29	.20	-1.49	0.14	-.68 to .09
Baseline drinks per day	-.26	.26	-1.02	0.310	-.77 to .24
Baseline drinks per day × Time (1MFU)	.23	.31	0.73	0.47	-.38 to .84
Baseline drinks per day × Time (3MFU)	.17	.31	0.56	0.58	-.44 to .79
Baseline drinks per day × Time (6MFU)	.18	.33	0.54	0.59	-.46 to .81
I_7-IMP	.26	.29	0.89	0.37	-.31 to .84
BAS-Drive	.10	.43	0.24	0.81	-.74 to .94
BAS-Reward	.77	.57	1.36	0.17	-.34 to 1.89
BAS-Reward × Time (1MFU)	-.075	.70	-0.11	0.92	-1.45 to 1.30
BAS-Reward × Time (3MFU)	-1.15	.69	-1.66	0.10[†]	-2.50 to .21
BAS-Reward × Time (6MFU)	-.02	.75	-0.02	0.98	-1.48 to 1.45
Baseline TR-AE	.72	.23	3.17	0.002*	.28 to 1.17
Baseline SL-AE	-.12	.26	-0.46	0.65	-.62 to .39
Baseline AESES	-.19	.15	-1.27	0.21	-.49 to .10

Note. SIAS-SPS = Social Interaction Anxiety Scale – Social Phobia Scale; SADQ = Severity of Alcohol Dependence Questionnaire; I_7-IMP = Impulsiveness subscale; BAS-Drive = Behavioural Activation Scale – Drive subscale; BAS-Reward = Behavioural Activation Scale – Reward Responsiveness subscale; TR-AE = Tension Reduction Alcohol Expectancy; SL-AE = Social Lubrication Alcohol Expectancy; AESES = Alcohol Expectancies for Social Evaluative Situations

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$