The relationship between social anxiety disorder and alcohol use disorders: A critical review

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Abstract

Epidemiological studies have demonstrated a significant co-morbidity between social anxiety disorder (SAD) and alcohol use disorders (AUDs). Despite the fact that many studies have demonstrated strong relationships between SAD and AUD diagnoses, there has been much inconsistency in demonstrating causality or even directionality of the relationship between social anxiety and alcohol-related variables. For example, some studies have showed a positive relationship between social anxiety and alcohol-related variables, while others have shown a negative relationship or no relationship whatsoever. In an attempt to better understand the relationship between social anxiety and alcohol, some researchers have explored potential moderating variables such as gender or alcohol expectancies. The present review reports on what has been found with regard to explaining the high co-morbidity between social anxiety and alcohol problems, in both clinical and non-clinical socially anxious individuals. With a better understanding of this complex relationship, treatment programs will be able to better target specific individuals for treatment and potentially improve the efficacy of the treatments currently available for individuals with co-morbid SAD and AUD.
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The current review seeks to provide a comprehensive examination of the relationship between social anxiety disorder (SAD) and alcohol use disorders (AUDs), commonly co-occurring conditions that result in a significant impact on society. Previous reviews on the topic have either focused on anxiety disorders

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more broadly (e.g., Kushner, Abrams, & Borchardt, 2000) or have neither fully explored the inconsistencies in the current explanatory models nor provided directions for treatment (e.g., Carrigan & Randall, 2003). There continue to be many important gaps in our understanding of the relationship between SAD and AUDs. Thus, the current paper will review the status of research on the co-morbidity of the disorders, the popular explanatory models of the relationship, limitations of the current models, and proposed mechanisms to consider in a new model. We will conclude with a consideration of the implications of the results of our review for treatment for the co-morbid disorders.

1. Social anxiety disorder and alcohol use disorders

SAD, referred to as social phobia in the Diagnostic and Statistical Manual of Mental Disorders-4th Edition-Text Revision (DSM-IV-TR; American Psychiatric Association [APA], 2000), is characterized by an intense and importunate fear of being regarded and subsequently judged negatively by others. The individual believes that he/she will act inappropriately or that his/her physiological symptoms of anxiety, such as sweating or heart palpitations, will be obvious to those around him/her and thus lead to further embarrassment and critical appraisal (APA, 2000). Those with SAD will invariably attempt to avoid those situations which lead to distress such as attending an office party or will endure those experiences with great duress. SAD occurs quite frequently in the general population, with lifetime prevalence for males at approximately 11% and approximately 15% for females (Kessler et al., 1994). SAD is the third most common psychological disorder, surpassed only by depression and AUDs (Kessler et al., 1994; Stein, Torgrud, & Walker, 2000). The typical age of onset of SAD is approximately 13–15 years of age (Ballenger et al., 1998; Chartier, Walker, & Stein, 2003) but it has been diagnosed in children as young as 8 years of age (Beidel & Turner, 1998). If untreated, SAD has a chronic pattern that continues into adulthood.

Another class of common and debilitating disorders is that of the AUDs. In the DSM-IV-TR (APA, 2000), AUDs include alcohol dependence and alcohol abuse—the former referring to a physiological and/or psychological dependence on alcohol where the individual continues to consume alcohol despite negative psychological or physical consequences. Alcohol abuse refers to a generally less severe symptom presentation than alcohol dependence. In alcohol abuse, the individual may use alcohol in hazardous situations, may continue using alcohol despite problems in social or interpersonal domains, or may experience problems arising in occupational or familial settings relating to their alcohol use (DSM-IV-TR; APA, 2000). Lifetime prevalence rates for alcohol abuse are 12.5% in men and 6.4% in women; for alcohol dependence, the rates increase to 20.1% for men and 8.2% for women (Kessler et al., 1997).

2. Prevalence of co-occurrence

Despite the fact that the construct of SAD was only introduced in the 1970s (Marks, 1970), there has been an increasing degree of evidence demonstrating a strong relationship between SAD and AUD (Kessler et al., 1994; Ross, Glaser, & Germanson, 1988; Schneier, Johnson, Hornig, & Liebowitz, 1992; Schneier, Martin, Liebowitz, Gorman, & Fyer, 1989). A variety of research methods have been employed to examine the relationship between SAD and AUDs, including epidemiological studies of co-morbidity rates in clinical and general population samples, investigation of co-morbidity within

subcategories of the SAD construct, and examination of the relative order of onset of SAD and AUD to establish temporality of the relationship. We review each of these types of studies below.

High rates of co-occurring SAD and AUD can be found in both clinical samples and in the general population. For example, according to the National Co-morbidity Study, lifetime prevalence rates of alcohol dependence among those with SAD were 24% (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996), compared to lifetime prevalence rates of alcohol dependence without SAD of 14.1% (Kessler et al., 1997). In a multi-site study by Thomas, Thevos, and Randall (1999) examining individuals seeking treatment for an AUD, it was shown that 23% met diagnostic criteria for SAD—an elevated rate compared to the base-rate of about 7% for SAD in the general population. In the Epidemiological Catchment Area (ECA) survey, it was demonstrated that the lifetime prevalence of alcoholism was more than twice as great among those with SAD (17%), compared to those without SAD (8%) (Davidson, Hughes, George, & Blazer, 1993). Similarly, in a college sample of 89 students, Kushner and Sher (1993) demonstrated that having an SAD diagnosis (established via structured interview) greatly increased the participants' chances of having an AUD. Thus, regardless of whether one examines rates of AUD in those seeking treatment for SAD or rates of SAD in those seeking treatment for an AUD, and regardless of whether one looks within clinical or non-clinical general population samples, we consistently see a strong co-morbidity between SAD and AUD diagnoses. Those with SAD appear to be between 2 and 3 times more likely to develop an AUD compared to those without SAD (Kushner, Sher, & Beitman, 1990), and those with an AUD seem to be up to 10 times more likely to display SAD than those without an AUD (APA, 2000; Kessler et al., 1997).

Despite consistent evidence across studies that there is substantial co-morbidity between SAD and AUD, the precise rates of co-occurrence vary widely between studies. This variability is likely due to a number of factors, including a lack of agreement on what is meant by the term co-morbidity (Kushner et al., 2000; Schuckit & Hesselbrock, 1994), along with methodological, population, and theoretical orientation differences between studies. Furthermore, it is likely that the co-morbidity rates are in reality higher than have been reported in many studies, for several reasons, including the likely possibility that many patients suffering from an AUD may not recognize that their social anxiety symptoms are clinically significant, and thus, downplay the importance of their symptoms during an assessment (Marshall, 1994). Similarly, many patients presenting for treatment for social anxiety may not readily admit to problems they might be experiencing with alcohol due to concerns about being evaluated negatively for their excessive consumption and/or due to concerns that they might be mandated to treatment for the alcohol problem before they have their social anxiety addressed. Moreover, alcohol interventions often utilize a group treatment modality, which poses another barrier to those with SAD honestly disclosing alcohol problems as the socially anxious individual may wish to avoid such treatment due to fears of the group situation. For such reasons, co-morbidity rates reported in the literature may be underestimates of the actual rates of co-occurrence of these two disorders.

In attempts to better understand the nature of the relationship between SAD and AUD, some co-morbidity studies have examined whether or not the strong co-morbidity with AUDs is true of all subtypes of SAD. For example, Kessler, Stein, and Berglund (1998) used the data from the NCS to further subdivide SAD into two subcategories, social anxiety characterized solely by public speaking fears and social anxiety characterized by at least one non-public speaking fear (i.e., closer to the 'generalized' subtype of social phobia listed in the DSM-IV-TR; APA, 2000). Kessler et al. (1998) demonstrated that co-morbid AUD was higher in those individuals with SAD who demonstrated both public speaking and non-public speaking fears. Similarly, Thomas, Randall, and Carrigan (2003)

distinguished between two aspects of social anxiety: fear of social interaction and concerns about performance in social situations. They found that individuals with SAD were more likely to drink to cope with social anxiety in social interaction than in performance situations (Thomas et al., 2003). Together, these data suggest that considering possible subtypes of SAD may be important in understanding the co-morbidity of SAD with AUDs. They also appear to suggest that those with a more generalized form of SAD, or those with predominantly social interaction types of concerns (as opposed to performance fears), may be those most likely to display co-morbid AUDs. These possibilities are deserving of further study.

In addition to the information provided by considering SAD subtypes in explaining SAD–AUD co-morbidity, it may also be useful to consider the SAD construct as symptoms lying on a continuum of severity. Some epidemiological work has considered whether AUDs co-occur with sub-clinical levels of social anxiety. For example, in a long-term follow-up of participants in the ECA study, those with sub-clinical social anxiety (i.e., showing some symptoms but not enough to meet formal diagnostic criteria) at the baseline interview were more likely to develop alcohol abuse at the 13-year follow-up, compared to those without social anxiety at the baseline interview (Crum & Pratt, 2001). Thus, it seems that a relationship may exist between social anxiety and alcohol problems even at sub-clinical levels of social anxiety. As noted by Chilcoat and Breslau (1998), if there is a causal relationship between SAD and AUD, then a "dose–response" relationship should be evident with increasing levels of symptoms of one disorder corresponding to increases in levels of symptoms of the other disorder. The Crum and Pratt (2001) epidemiological findings begin to establish this dose–response relationship. We return to this issue of relations between levels of symptoms of the two disorders in subsequent sections of this paper where we review the findings of studies employing alternative research methods.

Some of the co-morbidity studies have gathered data on the relative order of onset of the two disorders in co-morbid cases, to determine whether there is any consistent pattern in relative onset. Unlike some other anxiety disorders that are highly co-morbid with AUD (e.g., panic disorder), SAD typically presents prior to the development of the AUD (Heckelman & Schneier, 1995; Kushner et al., 1990; Merikangas & Angst, 1995; Schneier et al., 1989) making SAD most often the 'primary diagnosis' in co-morbid individuals. In their review, Kushner et al. (1990) demonstrated that among those with co-morbid SAD–AUD, the median age of SAD onset preceded the development of the AUD by 2 years. If SAD and AUD are causally related, it could be that the SAD causes the AUD or vice versa (Stewart, 1996). Due to the fact that SAD most often precedes AUD, it has been argued that SAD is a risk factor for (rather than a consequence of) AUD (e.g., Ham, Hope, White, & Rivers, 2002). Establishing temporality is a necessary prerequisite for determining causality (i.e., if SAD causes AUD, then SAD must precede AUD development), but temporality alone cannot establish causation (Chilcoat & Breslau, 1998).

In summary, a variety of epidemiological research methods have been employed over the past couple of decades to establish a relationship between the diagnoses of SAD and AUD. This type of research strongly demonstrates that those with SAD have a greater chance of displaying AUD and similarly, those with an AUD are more likely to display SAD, compared to those without these disorders. It has also shown that those with certain SAD subtypes (i.e., generalized form of SAD; those with interaction vs. performance fears) appear at greatest increased risk for co-morbid AUD, that those with subclinical SAD might also be at increased risk for AUDs, and that SAD tends to most often precede AUD development in co-morbid cases. There have been a number of theories offered to explain this robust relationship between the two disorders. It is to these theories that we now turn our attention.

3. Theoretical models to explain the co-morbidity

There have been a number of cognitive, behavioral, and psychosocial models that have been applied to explaining the relationship between SAD and AUD. Given the data showing SAD to be temporally antecedent to AUD development in co-morbid cases (Kushner et al., 1990), most of these models have worked on the assumption that SAD causes the AUD. Given alcohol's known anxiolytic properties, it is typically argued that socially anxious individuals drink to alleviate their physiological and psychological symptoms of anxiety, brought on by their fear of social situations.

One such model that has been applied to the understanding of the SAD–AUD link is the tension reduction theory (TRT). Early studies by Conger (1951) and others focused on alcohol's pharmacological effects using animals and stress-inducing tasks. The results of these studies essentially demonstrated that when presented with a stressful situation, the consumption of alcohol significantly reduced the release of stress-related hormones (Pohorecky & Brick, 1987) and increased the speed at which animals approached feared stimuli (Conger, 1951). Based on the results of these studies, Conger (1956) suggested that alcohol was reinforcing because of its tension reducing properties, with "tension" being very broadly defined. These observations led to the development of the TRT. According to Kushner et al. (1990), the TRT provides a means of explaining the relationship between SAD and AUDs, as the theory would predict that individuals with SAD: (a) will learn to consume alcohol to relieve tension associated with social situations, and (b) will develop a reliance on alcohol to relieve these unpleasant symptoms in the future. Thus, according to applications of the TRT, those with SAD would consume alcohol to combat tension associated with interpersonal interactions or performance situations. In addition, since Conger (1956) had argued that alcohol's reinforcing properties were based on avoidance reduction in approach-avoidance situations, application of the TRT to the understanding of SAD–AUD co-morbidity might also lead to the prediction that individuals with SAD would use alcohol to reduce their avoidance of feared social situations—an effect presumably mediated via alcohol-induced tension reduction. Similarly, more recent cognitive interpretations of the traditional TRT (e.g., Goldman, Del Boca, & Darkes, 1999) would predict that socially anxious individuals would possess greater expectancies with regard to alcohol's tension-reducing effects and thus would be more likely to consume alcohol when experiencing tension, compared to non-socially anxious individuals.

However, the TRT has been widely criticized for a variety of reasons including its overly-broad definition of 'tension' (not only anxiety but other negative affective states), its lack of consideration of the specific situations that provoke tension, and its failure to consider important individual difference variables (e.g., that some people might be more sensitive to alcohol's tension-reducing effects than others; Sher & Levenson, 1982). Given these criticisms, a new model was developed, which became known as the Stress Response Dampening model (SRD; Sher & Levenson, 1982). An extension of the traditional TRT, the SRD argues that individuals consume alcohol to reduce their reactivity to stressful situations and thus, consume alcohol when anticipating or experiencing anxiety provoking or stressful situations. Thus, according to this model, alcohol is most effective as an anxiolytic when it is consumed during or before a stress-inducing event or situation (Sher & Levenson, 1982). This explicit consideration of specific stress-inducing situations is one advantage of the SRD relative to the original TRT. In the case of those with SAD, the stress-inducing events or situations would be those involving social interactions or social performance. Another advantage of the SRD theory is that it explicitly recognizes the role of individual difference variables (Greeley & Oei, 1999), positing that some are more sensitive to alcohol's SRD effects than others with those most sensitive being at most risk for developing

alcohol problems (Finn & Pihl, 1988). When applied to explaining the SAD–AUD link, the SRD would predict that individuals with social anxiety are more sensitive than others to the SRD effects of alcohol when in a socially stressful situation. As a consequence, the SRD model also predicts that those with SAD will consume more alcohol, more frequently, when socially stressed, compared to someone low in social anxiety.

Another model used to explain the relationship between social anxiety and alcohol abuse is the Self-Medication Hypothesis (SMH) (see review by Carrigan & Randall, 2003). This model, like the SRD model, has its roots in the TRT. The SMH was first described by Khantzian (1985) and essentially argues that the psychotropic effects that drugs (in this case alcohol) have on an individual's psychological state lead some individuals to develop a dependence on the drug. According to Chutuape and de Wit (1995), there are three main assumptions of the SMH: (a) that the psychological variables (e.g., social anxiety) develop prior to the drug (alcohol) use problems; (b) that the drug (e.g., alcohol) provides respite from the symptoms; and (c) respite from the negative symptoms develops into continued and problematic usage. When applied to the case of SAD–AUD co-morbidity, the SMH predicts that alcohol will reduce anxiety in socially anxious individuals, and this reduction in anxiety will lead to an increased desire in socially anxious individuals to consume alcohol when in social (i.e., anxiety provoking) situations (Chutuape & de Wit, 1995; Carrigan & Randall, 2003). The chief advantage of this theory over the previous two models is its broad applicability (i.e., it serves not only as an explanation of the SAD–AUD link, but also as a useful theory for understanding other common forms of co-morbidity such as between depression and substance use disorders).

When the three aforementioned models are applied separately, they fail to explain a large number of research findings, but when the key elements from the TRT, SRD, and SMH are combined, they offer a more cohesive model to explain the mechanisms involved in the SAD–AUD relationship. Specifically, these theories argue that alcohol reduces physiological and cognitive anxiety or arousal, providing negative reinforcement of the drinking response. Since social situations and internal anxiety symptoms consistently precede the drinking response and the rewarding consequences of anxiety relief, these situations and symptoms become conditioned cues to signal the onset of alcohol consumption whenever anxiety is experienced or social interactions or performance are anticipated.

We will now review studies that have investigated predictions of these theories. As we will see, the evidence for these theories has not always been consistent. We will attempt to examine these inconsistencies to determine if any patterns emerge that might help refine the models to better explain the relationship of social anxiety and alcohol.

4. Testing the models

Despite all of the evidence demonstrating a strong relationship between the diagnoses of SAD and AUD, attempts to understand why this relationship exists have been less fruitful. Surprisingly, despite the growing amount of research in the area, much about the relationship between SAD and AUD is still unclear; even the direction of the relationship between social anxiety and alcohol-related variables has been inconsistently demonstrated in research examining the overlap of specific symptoms of the two disorders. For example, some studies have demonstrated a positive relationship (Abrams, Kushner, Medina, & Voight, 2001; Kidorff & Lang, 1999; Lewis & O'Neill, 2000; Morris, Stewart, Theakston, & Mellings, 2004), while others have found a negative relationship (Holle, Heimberg, Sweet, & Holt,

1995; Pihl & Yankofsky, 1979) or even no relationship (Eggleston, Woolaway-Bickel, & Schmidt, 2004; Ham & Hope, 2005). We have organized our review of these studies around the direction of the relationship observed, in an attempt to make sense of the findings and determine if any consistent patterns emerge that might shed light on the reasons for the discrepancies across studies. It should be noted that given that drinking patterns of undergraduates are quite different than older adults (Leigh, 1989); we will therefore differentiate between studies investigating clinically-diagnosed individuals with SAD and those investigating socially anxious undergraduates. Studies examining both types of populations add further evidence and different perspectives to the larger question of how social anxiety is related to problem alcohol usage, and thus both are vital to improving our understanding of this multifaceted relationship.

4.1. Positive relationship between social anxiety and alcohol-related variables

Several studies have provided evidence consistent with the predictions of the TRT, SRD, and SMH models as applied to understanding the relationships of social anxiety and alcohol. These studies fall into several categories including those testing whether alcohol has anxiolytic effects for SAD individuals in socially stressful situations, and those testing whether socially anxious individuals drink more than others when confronted with social stress, and whether socially anxious individuals experience more alcohol-related problems.

4.1.1. General population

One study by Abrams et al. (2001) used the alcohol challenge method to determine if alcohol has anxiolytic effects for those with SAD when they are experiencing social stress. These authors employed a speech-challenge paradigm, and randomly assigned 61 participants (55 who satisfied the DSM-IV-TR [APA, 2000] criteria for social phobia and 6 who met all criteria except for criterion E, which stipulates that the social anxiety must significantly interfere with functioning) to one of three conditions: an alcohol, placebo, or no alcohol control group. The experiment consisted of a pre-alcohol speech challenge, an alcohol consumption phase, followed by a post-drinking speech. The results demonstrated that drinking was indeed anxiolytic for SAD individuals undergoing social stress. Both the alcohol and placebo groups demonstrated a greater reduction in performance anxiety from the first to the second speech than did the control group. The fact that the anxiolytic effects were seen in both the alcohol and placebo groups highlights the importance and strength of anxiolytic expectancies about alcohol in those with SAD.

In another study examining the speech-challenge paradigm in 44 individuals diagnosed with SAD, Abrams, Kushner, Medina, and Voight (2002) found that participants drank more alcohol following the public speaking challenge than a control task (i.e., silently reading a magazine). Although this finding did not hold for alcohol consumption prior to the speech challenge vs. the control task, it provides evidence that increased social anxiety may lead to increased drinking among SAD individuals.

4.1.2. Undergraduates

Using self-report questionnaire methods, Lewis and O'Neill (2000) were interested in examining the relationship of social anxiety-related variables (as assessed by the Social Avoidance and Distress and the Fear of Negative Evaluation Scales, Watson & Friend, 1969; and Cheek and Buss Shyness Scale; Cheek, 1983) to alcohol problems (as assessed by the Rutgers Collegiate Substance Abuse Screening Test;

Bennett et al., 1993) in 116 undergraduates. The results demonstrated that that those classified as problem drinkers reported greater social anxiety and shyness, compared to those characterized as non-problem drinkers, confirming the significant positive relationship between social anxiety and problem drinking predicted by the TRT, SRD, and SMH models.

Similar results were obtained in a study conducted by our group (Morris et al., 2004). We found that there was a significant positive relationship between social anxiety and problem alcohol usage (measured with the Rutgers Problem Alcohol Index (RAPI; White & Labouvie, 1989)) when social anxiety was measured with the Brief Fear of Negative Evaluation scale (Leary, 1983) for 161 undergraduate drinkers (those who reported consuming at least 1 alcoholic beverage in the past year). Thus, the results of these two studies (Lewis & O'Neill, 2000; Morris et al., 2004) converge in documenting significant positive relationships between social anxiety levels and measures of alcohol use problems that are consistent with the substantial epidemiological evidence of a link between SAD and AUDs. These findings also document the expected dose–response relationship between symptoms of each disorder that one would expect if the two disorders were causally related (Chilcoat & Breslau, 1998).

Our study also investigated potential mediator variables that might help explain the observed relationship between social anxiety (in this case fear of negative evaluation) and alcohol problems. To this end, we also administered a measure of drinking motives—Cooper's (1994) four-factor Drinking Motives Questionnaire Revised. This measure asks individuals to rate how often they drink for a variety of reasons, and provides scores on four distinct drinking motives scales: Coping (to reduce or avoid negative affective states such as anxiety), Conformity (to reduce or avoid social censure), Social (for affiliative reasons), and Enhancement (to increase positive affective states). We found that fear of negative evaluation scores were positively correlated with all four drinking motives scores, particularly Coping and Conformity motives. We also found that Coping motives mediated the relationship between fear of negative evaluation and drinking problems. This finding means that those students who fear negative evaluation experience more alcohol problems than others at least partly because when they drink, they drink to cope with negative emotions (Morris et al., 2004). These findings point to the importance of considering drinking motives when considering the SAD–AUD relationship in future research.

Although producing results consistent with the predictions of the TRT, SRD, and SMH, the previous two studies suffer from the limitations of retrospective self-report methods. Laboratory-based methodologies, including the stress-induced drinking paradigm, have also been used to examine the relationship of social anxiety to alcohol-related variables in undergraduates. In one such study, Kidorf and Lang (1999) provided access to alcohol to 84 undergraduate participants who were high or low in trait social anxiety following a baseline (i.e., non-stressful) or stress-induction (i.e., video recorded speech on participants' least desirable traits) phase, which took place on separate testing days. The results demonstrated that drinking for all participants increased in the stress-induction phase relative to the baseline phase, providing evidence for the TRT prediction that anxiety leads to increased alcohol consumption. Moreover, those with high trait social anxiety drank more than the other participants in the stress-induction phase, consistent with predictions of the TRT, SRD, and SMH models. Although one could criticize the lab-based stress-induced drinking methodology used in this study for being somewhat artificial, the results are nonetheless important as they suggest that socially anxious participants do drink more alcohol than others when confronted with a socially stressful event. Moreover, the results of the study by Kidorf and Lang (1999) provide behavioral validation of previous self-report findings linking social anxiety levels with problematic drinking levels (Lewis & O'Neill, 2000; Morris et al., 2004).

4.1.3. Clinical sample

Similar results to those reported by Abrams et al. (2001) were obtained by Himle et al. (1999), who demonstrated that treatment-seeking individuals with SAD receiving either alcohol or a placebo prior to a speech task did not differ in subjective, physiological, or cognitive anxiety reduction. They concluded that while alcohol does not directly reduce social anxiety in SAD individuals, the belief that one has consumed alcohol may do so. However, since only SAD individuals were examined in both of these studies (Abrams et al., 2001; Himle et al., 1999), it is not possible to conclude whether SAD individuals are more sensitive to these effects of drinking than others. Moreover, given the Thomas et al. (1999) findings that SAD individuals are more likely to drink to cope with social interaction than with social performance fears, it would be interesting to repeat this type of study in future using a social interaction type stressor in place of the speech stressor used by Abrams et al. (2001) and Himle et al. (1999).

4.2. Negative relationship between social anxiety and alcohol-related variables

Despite the high SAD–AUD co-morbidity rates found in epidemiological studies, both in treatment and general population samples, and the studies reported in the previous section documenting the expected positive relationship between social anxiety levels and drinking-related variables, there are a growing number of studies, albeit largely with undergraduate samples, that have demonstrated a negative direction relationship between social anxiety levels and alcohol-related variables.

4.2.1. Undergraduates

One such study that demonstrated an inverse relationship between social anxiety and alcohol usage was conducted by Eggleston et al. (2004). In this study, 284 undergraduate drinkers (defined as consuming at least one alcoholic beverage in the last 6 months) were administered a range of questionnaires, including the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) and measures of alcohol use levels. The results demonstrated that, contrary to predictions of the TRT, SRD, and SMH models, mean days drinking alcohol a week and mean number of binges per week were both significantly negatively correlated with social anxiety levels. Eggleston et al. (2004) suggested that perhaps socially anxious individuals found that the most effective way to lessen their social anxiety was not, in fact, to consume alcohol but rather to avoid the situation where others socialize, resulting in less alcohol being consumed by socially anxious students.

In another self-report questionnaire study, Ham and Hope (2005) examined the relationship between alcohol consumption (assessed by the Alcohol Uses Questionnaire; AUQ; Addictive Behaviors Research Center, 1997) and social anxiety (Interaction Anxiousness Scale; IAS; Leary, 1983) in a sample of 316 university students. Contrary to their original hypotheses, social anxiety was found to have a negative relationship with alcohol usage. In attempting to account for the unexpected negative relationship between social anxiety and drinking levels, the authors suggested that perhaps socially anxious students avoid social situations (e.g., parties) in which most college drinking would take place (Ham & Hope, 2005; cf. Eggleston et al., 2004).

Finally, in the previously-mentioned study conducted by our group (Morris et al., 2004), we found that there was a significant negative relationship between social anxiety and self-reported number of average drinks-per-week when social anxiety was measured with the Social Avoidance and Distress Scale (Watson & Friend, 1969). This finding is particularly interesting because it occurred within the

same study where a significant positive relationship was observed between social anxiety and problem alcohol usage when social anxiety was conceptualized as fear of negative evaluation. This pattern of findings may help to give us some clues about why inconsistencies have been observed across studies. This pattern suggests that while a negative relationship may be observed between social anxiety indices and alcohol use levels, a positive relationship may still exist with alcohol use problems. In fact, both self-report studies supporting a positive relationship between social anxiety and alcohol-related variables have used alcohol problems as the dependent measure (Lewis & O'Neill, 2000; Morris et al., 2004) while all results supporting a negative relationship between social anxiety and alcohol-related variables have examined alcohol use levels (Eggleston et al., 2004; Ham & Hope, 2005; Holle et al., 1995; Morris et al., 2004). Although drinking levels and alcohol problems are inter-correlated constructs, they are conceptually and empirically distinct with stress being more predictive of alcohol problems than of drinking levels (McCreary & Sadava, 1998). In fact, the DSM-IV-TR (APA, 2000) definitions of the AUDs recognize that one can develop problems with alcohol even at relatively lower drinking levels (i.e., minimum drinking levels are not specified in the diagnostic criteria). It may be that socially anxious individuals do not drink particularly often or heavily, but when they do drink, they are drinking for problematic reasons related to social anxiety management, thereby putting them at risk of developing a dependence on alcohol to cope with these kinds of situations.

The pattern of findings from the Morris et al. (2004) study also points to the importance of considering variability due to the ways in which social anxiety has been conceptualized across studies. Some subtypes or aspects of social anxiety may be more strongly linked to alcohol abuse than others (cf. Thomas et al., 1999). The Morris et al. (2004) findings suggest that while socially avoidant people may drink less frequently and heavily than others, those who fear negative evaluation may be particularly prone to experiencing problems related to their drinking.

4.2.2. Clinical samples

Holle et al. (1995) were interested in examining possible differences in frequency of alcohol consumption between 71 individuals with SAD and a sample of 39 community controls. Holle et al. (1995) administered a self-report questionnaire which measured the participants' frequency of consumption of a variety of beverages, including caffeinated and alcoholic drinks. Contrary to predictions of the TRT, SRD, and SMH models, results showed that individuals with SAD consumed wine and spirits significantly less often, compared to the non-socially anxious control group. However, it is difficult to make strong conclusions from the Holle et al. (1995) because they excluded those participants with co-morbid alcohol problems from the study, which arguably removed the most pertinent subpopulation of socially anxious individuals.

4.3. No relationship between social anxiety and alcohol-related variables

There have been several studies which have failed to show clear directionality in the relationship between social anxiety and alcohol-related variables, further adding to the difficulty in delineating this complex relationship. Findings in this category will be described next.

4.3.1. Undergraduate samples

In the previously-described study by Ham and Hope (2005) with undergraduates, they additionally used the AUQ to measure alcohol related problems as well as the measure of social anxiety (IAS)

described previously. Contrary to their original hypotheses (and to their findings of a significant negative relationship between social anxiety and alcohol use levels), social anxiety did not have a significant relationship with alcohol-related problems. This result thus stands in opposition to the findings of Lewis and O'Neill (2000) and Morris et al. (2004) of significant positive relationships between social anxiety and alcohol problems among undergraduates. However, Ham and Hope's (2005) results were in the context of a model that controlled for other important variables such as alcohol outcome expectancies. We will discuss the potentially important role of expectancies in greater detail in a subsequent section.

In the previously-described study by Eggleston et al. (2004) with undergraduate drinkers, they additionally administered the RAPI. The results demonstrated that, consistent with the findings of Ham and Hope (2005), drinking problems were not significantly related to social anxiety scores. The Social Interaction Anxiety Scale (SIAS) was used as opposed to the fear of negative evaluation measures used in the studies supporting a positive relation between social anxiety and alcohol problems by Lewis and O'Neill (2000) and Morris et al. (2004).

Bruch et al. (1992) examined the relationship between shyness and alcohol use by administering a number of questionnaires, including the Cheek and Buss Shyness Scale, and a brief questionnaire about frequency and quantity of alcohol usage, to 543 undergraduates. In bivariate correlations, the shyness measure was not significantly related to alcohol use levels—a result that was inconsistent with Lewis and O'Neill's (2000) finding of a significant positive relationship between scores on this same measure and alcohol problems. However, Bruch et al. (1992) examined alcohol use levels whereas Lewis and O'Neill (2000) examined alcohol problems which could account for the inconsistency. Nonetheless, it appears that shyness is not consistently linked with alcohol-related variables. Despite the fact that measures of social anxiety and shyness are inter-correlated (Jones, Briggs, & Smith, 1986), Leary (1986) has argued that social anxiety and shyness are in fact distinct constructs, and thus, caution is necessary in assuming that results from studies focusing on shyness vs. social anxiety are comparable or interchangeable.

4.3.2. Clinical samples

Ham et al. (2002) also failed to find a relationship between social anxiety and alcohol consumption levels. However, an important difference between the Ham et al. (2002) and the other studies mentioned earlier in this section of the paper is that the participants in the Ham et al. (2002) study were diagnosed with SAD (as assessed by the Anxiety Disorders Interview Schedule-Revised; DiNardo & Barlow, 1988) whereas the other studies focused on socially anxious undergraduates, thereby improving the generalizability of the findings to the larger SAD–AUD co-morbidity literature. Ham et al. (2002) administered a number of questionnaires to participants and found that in contrast to their initial prediction, there were no significant differences in alcohol consumption levels between the 3 groups of participants (including 54 with SAD, 23 with dysthymia, and 27 normal controls), thereby extending findings of no relationship between social anxiety and alcohol-related variables to a clinical sample. There were also no group differences in the proportion of individuals who did not consume any alcohol. However, again, these findings focused on alcohol use levels as opposed to alcohol use problems—the latter being the variable most consistently linked to social anxiety. Further, those with AUDs were excluded from participation in this study making this study susceptible to the same criticisms noted earlier with respect to the Holle et al. (1995) study.

4.4. Summary

Although there is a good degree of evidence from epidemiological studies (e.g., Kessler et al., 1997) of a high rate of co-morbidity between the diagnostic categories of SAD and AUD, in studies that have attempted to examine relations between levels of symptoms of each disorder, there is still much inconsistency in delineating the nature and direction of the relationship. Some studies have found a significant positive relationship between problem social anxiety and alcohol-related variables consistent with predictions of the TRT, SRD, and SMH models. However, other studies have found a negative relationship between these variables and some no relationship whatsoever. Certainly, methodological differences could account for much of the variability in the results, in that each study seems to use different measures of not only SAD symptoms (e.g., social avoidance vs. fear of negative evaluation) but also different alcohol-related measures (e.g., drinking levels vs. alcohol problems). Further, the studies vary in terms of the populations they examine: some used clinical individuals with SAD, while others used undergraduates high and low in social anxiety, or "shy" individuals. Moreover, some have excluded those with AUDs from study.

Although these differences make it difficult to draw any firm conclusions, some preliminary conclusions can be suggested on the basis of the pattern of the findings. It appears that it is measures of drinking problems as opposed to drinking levels that are most consistently positively linked to social anxiety levels. It also appears that certain aspects of social anxiety (e.g., those involving social avoidance) may be negatively related to drinking levels, either because these individuals avoid the types of social situations where alcohol is typically consumed, and/or because they fear the adverse social consequences of displaying drunken behavior. Also, it appears that certain aspects of social anxiety, in particular fears of negative evaluation, are more likely than other aspects of social anxiety to show a positive relationship with alcohol problems. Finally, it is important to highlight that nearly all of the studies which failed to show a positive relationship between SAD and AUD used undergraduate or non-clinical samples. Further research is necessary to further clarify why findings of relations between social anxiety and drinking-related variables do not always extend to samples of undergraduates. It is possible, for example, that the types of situations where socially anxious individuals are theoretically most likely to drink heavily (e.g., at parties) are normatively heavy drinking situations among undergraduates making it more difficult to distinguish the drinking behavior of socially anxious undergraduates from that of their non-socially anxious peers.

It is also likely, as suggested by the SRD model, that there are other moderating variables which are affecting which types of socially anxious individuals are most sensitive to the anxiolytic properties of alcohol, and thus learn to drink to alleviate anxiety in social situations. We turn our attention next to a review of such potential moderating variables.

5. Potential moderating variables

Recently, there has been a trend towards exploring possible moderator variables to help explain why some socially phobic individuals abuse alcohol, while others may avoid excessive consumption of alcohol (Cooper, Russell, Skinner, Frone, & Mudar, 1992). Moderator variables are those that influence the strength and/or the direction of the relationship between the independent and dependent variables (Baron & Kenny, 1986). These can be differentiated from mediator variables, which

attempt to explain why and how a relationship exists (e.g., Morris et al.'s (2004) finding that coping drinking motives mediates the relation between fear of negative evaluation and alcohol problems). In contrast, moderator variables try to explain when a relationship will exist (Baron & Kenny, 1986). One potential moderating variable in the SAD–AUD relationship are alcohol outcome expectancies.

5.1. Alcohol expectancies

The literature exploring alcohol expectancies has been growing at a prodigious rate over the last decade, particularly as related to social anxiety (e.g., Bruch et al., 1992; Ham et al., 2002; Kidorff & Lang, 1999; Kushner, Sher, Wood, & Wood, 1994; Lewis & O'Neill, 2000; Tran & Haaga, 2002; Wilson, Brick, Adler, Cocco, & Breslin, 1989). Alcohol expectancies refer to an underlying belief about the effects of alcohol on cognition, emotions, physiology, and behavior (Brown, Goldman, Inn, & Anderson, 1980; Burke & Stephens, 1999). They are said to have developed from prior experiential and observational learning (Del Boca, Darkes, Goldman, & Smith, 2002) and have been shown to influence drinking patterns and consequences (Goldman et al., 1999). Beliefs about the effects of alcohol may be important in explaining the link between SAD and AUD as has been demonstrated in the placebo-controlled alcohol challenge studies with individuals with diagnosed SAD discussed previously (i.e., Abrams et al., 2001; Himle et al., 1999). These findings suggest their role as a possible mediating variable. We will now turn our attention to research using self-report methods that has evaluated alcohol expectancies' role as possible moderating variables that might help explain inconsistencies in the observed relations between social anxiety levels and alcohol-related variables.

Some researchers have argued for a model where alcohol expectancies act as a moderating variable in the relationship between social anxiety and problem alcohol usage (Bruch, Rivet, Heimberg, & Levin, 1997; Tran, Haaga, & Chambless, 1997). According to this proposed model, socially anxious individuals will drink alcohol when they believe it will lead to positive outcomes, such as being more sociable at a party, but will not drink when they believed that it will produce unpleasant consequences, such as cognitive or behavioral impairment prior to giving a speech (Eggleston et al., 2004). Despite the fact that some researchers have suggested that alcohol expectancies are one of the best predictors of drinking behavior (Christiansen & Goldman, 1983; Goldman et al., 1999), the results from a number of studies which have attempted to apply alcohol expectancies to explaining the SAD–AUD relationship have produced inconsistent findings (Eggleston et al., 2004; Kushner et al., 1994; Tran et al., 1997).

Tran et al. (1997) administered several questionnaires to 229 undergraduates, including the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998), the Alcohol Expectancies for Social Evaluation Situations Scale (Bruch et al., 1992), along with a measure of frequency and quantity of alcohol consumption. Interestingly, similar to some prior findings (e.g., Morris et al., 2004), social anxiety was negatively related to alcohol consumption levels overall. The results also showed situational specificity of alcohol expectancies as moderator variables. Expecting specifically that alcohol would reduce anxiety in social situations moderated the relation between social anxiety and alcohol consumption; no such moderating effect was observed for general tension reduction expectancies. Among participants who did not expect alcohol to reduce their anxiety in social situations, high social anxiety individuals reported lower alcohol consumption levels than did low social anxiety individuals. High and low social anxiety individuals who expected alcohol to reduce their social anxiety did not

differ in their alcohol consumption. The results from Tran et al. (1997) thus demonstrated that alcohol expectancies did indeed moderate the relationship between social anxiety and alcohol consumption, although not exactly in the manner predicted.

In the previously-reported questionnaire study by Lewis and O'Neill (2000) with undergraduates, the authors also administered an alcohol expectancy measure (Alcohol Expectancy Questionnaire-Adolescent Form; Christiansen, Goldman, & Inn, 1982), in addition to the social anxiety, shyness, and alcohol problem measures mentioned earlier. Although the results demonstrated that relative to non-problem drinkers, the problem drinkers had more positive expectancies of alcohol, including increased arousal, sexual enhancement, social skills enhancement, increased relaxation, and global positive change expectancies (cf. Zarantonello, 1986), the authors did not find that the social anxiety measures interacted with positive alcohol outcome expectancies in predicting drinking problems. These findings stand in contrast to the results of Tran et al. (1997) who did find a social anxiety by expectancy interaction in the prediction of a drinking measure. However, Tran et al. (1997) examined alcohol use levels as the outcome measure whereas Lewis and O'Neill (2000) examined alcohol problems.

The next two studies did not explicitly examine the moderating influences of alcohol outcome expectancies in accounting for the inconsistent link between social anxiety and alcohol-related variables, but rather considered alcohol expectancies as the dependent measure to determine if there are consistent relationships between social anxiety and particular types of alcohol outcome expectancies. For example, Tran and Haaga (2002) compared individuals with SAD (classified using the Social Interaction Anxiety Scale), individuals with co-morbid SAD and lifetime AUD (classified using the Michigan Alcoholism Screening Test; Selzer, 1971), and normal controls, on their alcohol outcome expectancies. The co-morbid individuals held more positive expectancies about alcohol than those in the other two groups, reporting greater expectations that alcohol would lessen their social anxiety and reduce their tension. This finding is consistent with a moderator perspective in suggesting the possibility that only those SAD individuals with social anxiety reduction and tension reduction expectancies develop co-morbid AUD, but the results are limited by the cross-sectional (as opposed to longitudinal) design of the study. It is interesting to note that with regard to negative expectancies of alcohol usage, which some have argued as may be a protective factor against alcohol abuse (Leigh, 1987a, 1987b), Tran and Haaga (2002) found no significant differences between the SAD individuals with and without co-morbid AUD; in fact, the two SAD groups held significantly more negative expectancies than the normal control group. According to Tran and Haaga (2002), these results suggest that negative expectancies have little protective impact on the drinking behavior of individuals with co-morbid SAD–AUD. Rather, negative expectancies may be more a consequence (than a cause) of the problematic drinking behavior of those with SAD (see Jones, Corbin, & Fromme, 2001, for a review of this alternative perspective on negative alcohol expectancies). However, it is important to note that the "negative" expectancies examined in Tran and Haaga's (2002) study were not specific to social effects of drinking, thus it is not known whether this particular type of negative alcohol expectancy would play a protective role. Also, little is known about how alcohol expectancies differ across those with SAD alone and comorbid SAD–AUD when examined from a social contextual framework. In other words, it is unclear whether these two groups differ in their alcohol expectancies with regard to social situations vs. performance or public speaking engagements. Further research is necessary to understand how social context and expectancies influence which individuals with SAD develop AUDs and which do not.

Another study to employ expectancy measures in the investigation of the SAD–AUD link was the previously-mentioned study by Ham et al. (2002). These authors used questionnaires to examine alcohol expectancies in those diagnosed with SAD, in those diagnosed with dysthymia, and in normal controls. The results demonstrated that those with SAD had more positive expectancies regarding alcohol's tension reducing properties, and more global positive change expectancies, compared to normal controls, but there were no differences in these two expectancy domains when compared to those with dysthymia. Ham et al. (2002) also found that social assertiveness expectancies were greater for the SAD group than for both the dysthymics and the normal control groups. Finally, these authors found that higher levels of social assertiveness and tension reduction and lower levels of global positive change expectancies predicted increased alcohol consumption levels for the SAD participants (Ham et al., 2002). Unfortunately, due to a small sample size in both the dysthymics ($N=23$) and normal controls ($N=27$), similar regression analyses could not be performed in these groups. Future research is necessary to determine whether the above results would also extend to these two groups or whether they would prove unique to those with SAD.

In summary, the results from studies examining expectancies suggest that those with SAD, along with those with AUD, and those with co-morbid SAD and AUD tend to hold more positive alcohol outcome expectancies, compared to those low in social anxiety and those without an AUD. Those positive expectancies which are elevated in socially anxious individuals include general tension reduction expectancies, social assertiveness expectancies, and specific expectancies for alcohol to reduce anxiety in social situations. These very expectancies do appear to be related to drinking levels among SAD individuals (Ham et al., 2002). Moreover, there is some limited evidence that alcohol expectancies may moderate the relationship between social anxiety and alcohol-related variables. There does appear to be some situational specificity in this moderator relationship in that expectancies for alcohol to reduce anxiety in social situations, and not general tension reduction expectancies, have been shown to moderate the relationship between social anxiety and alcohol use levels (Tran et al., 2002). The evidence for alcohol expectancies to moderate the relationship between social anxiety and alcohol problems is inconsistent, however, with one study supporting (Tran & Haaga, 2002) and another not supporting (Lewis & O'Neill, 2000) this moderator role. With regard to negative expectancies, the results from studies such as Tran et al. (2002) show that negative expectancies about alcohol do not seem to affect the socially anxious individual's decision to drink, suggesting that treatment programs should target positive expectancies when treating these individuals. Finally, it is possible that some inconsistencies in this literature may be due to alcohol expectancies interacting with other variables. Examining these variables simultaneously with expectancies may provide a clearer picture of the relationship between social anxiety and problem alcohol usage.

6. Other variables interacting with expectancy

In an attempt to further understand how alcohol expectancies affect the development of co-morbid SAD–AUD, some researchers have begun examining how so-called third variables (e.g., situational factors, gender) might interact with alcohol expectancies. The ultimate goal of such studies has been to further clarify why those with social anxiety are more likely to develop AUD, compared to those without social anxiety, and to better specify which individuals with SAD are most likely to develop co-morbid AUDs.

6.1. Situational factors

Ham, Carrigan, Moak, and Randall (in press) were interested in examining whether socially anxious individuals held different expectancies about alcohol based on potential outcomes in different situations, or whether these individuals had more global expectations. Ham et al. (in press) administered several questionnaires to 62 participants (17 clinically socially anxious (as assessed by the SIAS and the Social Phobia Scale (SPS; Mattick & Clarke, 1998)) and 45 non-socially anxious), including two self-report measures, the Alcohol Expectancies in Social Evaluative Situations (Bruch et al., 1992) and the Drinking Expectancy Questionnaire (Young & Knight, 1989). The results demonstrated that those with social anxiety possessed more positive alcohol expectancies for specific social situations compared to those low in social anxiety. Also, in line with expectancy theory, social situation-specific alcohol expectancies were associated with increased drinking and alcohol dependence levels. In fact, the Ham et al. (in press) results are consistent with prior research demonstrating that socially anxious individuals appear to consider the situation, and likely, what will be required of them before consuming alcohol. For example, Turner, Beidel, Dancu, and Keys (1986) found that a significant number of socially anxious individuals consume alcohol prior to attending social events while Abrams et al. (2002) found their socially anxious participants, when given a choice, consumed very little alcohol before a speech task, compared to a control silent-reading task. These findings are both consistent with the results of Thomas et al. (2003) who, as mentioned previously, found that drinking for SAD individuals was more commonly used to cope with social interaction than performance situations.

In summary, studies on the role of situational factors involved in the alcohol expectancies of socially anxious individuals suggest that socially anxious individuals possess dynamic, ever-changing expectancies, which depend on the situational demands regarding what is required of them. If the situational demands require them to be sociable and interact with others, they will likely consume alcohol, but if the situational demands require them to perform in some manner, such as giving a speech, they tend to abstain from alcohol perhaps for fear of embarrassing themselves. An obvious extension from research on expectancies is research examining gender differences in alcohol expectancies. It seems likely that women would expect different outcomes when they drink alcohol, compared to men, and that gender differences in expectancies might help explain some of the inconsistencies to date with respect to the role of expectancies in the SAD–AUD relationship.

6.2. Gender

Prior research has shown that female drinkers respond differently to alcohol than males. For example, women develop higher plasma concentrations when consuming equal amounts of alcohol compared to men (Zilberman, Tavares, Blume, & el-Guebaly, 2003) and the time frame between the first use of alcohol to seeking treatment for an AUD is much shorter in women, compared to men (Zilberman et al., 2003). In terms of gender differences in the SAD–AUD relationship, it has been shown that females with co-morbid SAD–AUD demonstrate stronger avoidance of their feared situations/stimuli, compared to males with co-morbid SAD–AUD (Randall, Thomas, & Thevos, 2000), showing that gender may indeed be an important factor to consider when attempting to understand this form of co-morbidity.

Abrams and Wilson (1979) showed that women who believed they had consumed alcohol demonstrated increased social anxiety, less self-disclosure, and no increase in sexual feelings, in

comparison to men who demonstrated the exact opposite reactions—specifically, decreased social anxiety, increased self-disclosure, and increased sexual arousal. Thus, with respect to the relationship between SAD and AUD, these findings would suggest that socially anxious men would be more likely than socially anxious women to abuse alcohol to manage their social fears given socially anxious men's apparently greater expectancies of social anxiety reduction, social assertion, and sexual enhancement relative to those of women. As alcohol use among women has been changing to be more similar to that of men's (e.g., Maney, 1990), it is important for researchers to attempt to replicate these findings today given that the Abrams and Wilson study was conducted over 25 years ago.

Kushner et al. (1994) examined whether alcohol expectancies moderated the relationship between anxiety and alcohol usage. They found that a composite anxiety measure (including a social anxiety measure) was predictive of alcohol consumption among males with stronger tension reduction expectancies but that this relationship was not evident among women with strong tension reduction expectancies (Kushner et al., 1994). More recently, Abrams and Kushner (2004) found that a placebo alcohol manipulation resulted in a greater reduction in symptoms of anxiety while giving a speech for men with SAD having high tension reduction expectancies than SAD men with low tension reduction expectancies. This pattern was not found for SAD women or individuals in the control beverage group; however, the sample size was relatively small ($N=41$). Thus, these findings are consistent with propositions presented earlier that alcohol expectancies moderate the relationship between social anxiety and alcohol-related variables (e.g., Tran et al., 1997), but they further demonstrate that gender further moderates this relationship with the positive relationship between social anxiety and alcohol use being observed only for men with strong tension reduction expectancies.

Using behavioral observation rather than self-report measures, the study by Kidorf and Lang (1999), discussed in an earlier section of this paper, also examined whether participant gender interacted with alcohol expectancies in predicting alcohol consumption levels in response to a public speaking stressor. The results demonstrated that all participants consumed more alcohol when anticipating giving the speech relative to the baseline (non-stressful) phase, but that this was particularly true of males who expected alcohol to improve their assertiveness levels. Thus, social assertiveness expectancies interacted with gender in predicting drinking levels when participants were anticipating having to give a speech. Although, as reported previously, social anxiety levels also predicted greater alcohol consumption in anticipation of the social stressor, the authors did not examine whether expectancies and gender together interacted with social anxiety in predicting alcohol consumption levels in response to social stress.

In summary, models that attempt to explain or account for inconsistencies in the relationship between social anxiety and alcohol-related variables must consider the findings that women seem to possess different alcohol expectancies than men in domains relevant to social functioning (e.g., social anxiety reduction expectancies, social assertiveness expectancies, and sexual enhancement expectancies). The models must also account for findings that alcohol expectancies appear to moderate the social anxiety-drinking relationship to a greater extent in men than women. Unfortunately, current explanatory models (e.g., TRT, SRD, and SMH) do not explicitly take gender into consideration, thus, greatly reducing their utility and explanatory capability. Given the findings reviewed in this section, it would appear useful for future epidemiological studies to address whether men are more likely than women to display increased rates of co-morbid SAD–AUD (relative to gender-specific base-rates).

7. Limitations of the TRT, SRD, and SMH Models

Although we have discussed the strengths of the TRT, SRD, and SMH models (and adaptations of these including expectancy-based models) in accounting for the high co-morbidity between SAD and AUD, these models do not consider several facts that are important in considering the complex relationship between SAD and AUD. First, all three models have the same treatment implication-namely, treat the SAD (since it is causally related to the AUD) and the AUD should also resolve since there would be no more social anxiety motivating problem drinking. However, this view fails to consider that once it develops, the AUD may take on a life of its own such that the AUD might persist even with adequate treatment of the SUD. Second, the TRT, SRD, and SMH all fail to consider that once co-morbidity is established, the AUD might actually maintain or worsen the SAD for several reasons. For example, if a co-morbid individual is consistently using alcohol to allow for entry into feared social situations, their fear of these situations would fail to habituate since alcohol would block the experience of a sufficient fear response to promote habituation (Foa & Kozak, 1986). Moreover, the individual who uses alcohol in this manner may fail to modify underlying maladaptive beliefs about their social adequacy, believing that the only reason they coped effectively with a given social event was because they drank—thus, alcohol abuse might serve to maintain underlying beliefs that keep the SAD alive despite permitting the individual to attend social functions they might otherwise avoid. Additionally, the socially anxious person who abuses alcohol may experience increased anxiety following drinking bouts either as a function of physiological withdrawal and/or worries about their social performance when under the influence of alcohol, which might in turn worsen their social anxieties. If the SAD is indeed causally antecedent to the initial development of the AUD in co-morbid individuals, this does not preclude these other mechanisms by which the alcohol abuse might maintain or worsen the SAD in the longer-term. Future research should consider the possibility of a vicious cycle existing between SAD and AUD by which both disorders interact with one another in a mutually-maintaining manner (see Stewart, 1996).

The TRT, SRD, and SMH models also fail to fully consider the impact of contextual factors in the relationships between SAD, AUD, and alcohol outcome expectancies. A more adequate model would have to consider expectancies about the type of social situation (e.g., social interaction vs. performance), accessibility of alcohol within the situation, perceived appropriateness of alcohol use within the context (including gender norms), and amount of distress, fear, and avoidance one has regarding the situation. Evidence suggests that individuals with SAD may seek out alcohol to relieve social anxiety more so in social interaction situations than in performance situations, and that individuals have varying alcohol outcome expectancies across contexts more generally (e.g., MacLatchy-Gaudet & Stewart, 2001; Wall, McKee, & Hinson, 2000). Thus, it stands to reason that consideration of social context in conjunction with alcohol outcome expectancies would provide a more comprehensive approach to examining the SAD–AUD relationship that could account for the inconsistencies in the literature. Such a model could explain why some individuals with SAD drink very little while others are at a high risk for AUDs.

8. Treatment of co-morbid social anxiety and alcohol use disorders

Despite the high co-morbidity rates between SAD and AUD, little research has examined the most effective treatment approach for these individuals be it a psychosocial approach, a pharmacological

approach, or some combination of these treatment approaches (Herbert, 1995; Thevos, Roberts, Thomas, & Randall, 2000). Further, treatment for AUD and SAD, as well as other co-morbid non-AUD diagnoses in general, tend to be completed separately (i.e., by different treatment providers at different times), with a lack of coordination of care. In fact, as is the case for other types of co-morbid anxiety–substance use disorders, many clinics and research trials exclude treatment-seeking individuals with SAD who have a co-morbid AUD, at the very least requiring the completion of alcohol treatment prior to SAD treatment (see Stewart, 1996). It is currently unclear whether the current strategy of alcohol treatment prior to SAD treatment for co-morbid SAD and AUD is more effective than other strategies, such as matching of specific alcohol treatments based on individual differences or simultaneous/combined treatments of both SAD and AUD.

8.1. Psychosocial treatments

The most ambitious attempt at investigating psychosocial treatment outcomes for co-morbid individuals was done in Project MATCH (Project MATCH Research Group, 1993). Specifically, Project MATCH involved randomly assigning participants to Twelve-Step Facilitation (TSF), Cognitive-Behavioral Coping Skills (CBT), or Motivational Enhancement Therapy (MET), and examining outcomes and symptoms patterns across treatments to help determine if particular treatments are most suited for particular clients. It should be noted, however, that the treatments in Project MATCH were not designed specifically to treat SAD but rather, were designed to examine patient–treatment matching for those with AUD as a function of matching variables such as co-morbid disorders.

In one of the few studies to examine treatment outcomes among SAD individuals with AUD, Thevos et al. (2000) used the Project MATCH database to retrospectively examine whether co-morbid SAD–AUD clients assigned to CBT would have better treatment outcome compared to co-morbid SAD–AUD clients assigned to TSF. The results demonstrated that outpatient females with co-morbid SAD–AUD experienced better treatment outcomes when they were treated with CBT, compared to TSF (based on slower relapse rates). Interestingly, outpatient males with co-morbid SAD showed a marginally better treatment outcome when treated with TSF, compared to CBT, though this difference was not statistically significant. The Thevos et al. (2000) results are important because they do suggest that some clients (in this case, female clients with co-morbid SAD–AUD) benefited more from being matched to specific AUD treatments (in this case CBT as opposed to TSF).

In one of the few studies of its kind, Randall, Thomas, and Thevos (2001) examined whether treatment of those with co-morbid SAD–AUD was more successful in either an alcohol-only treatment or a combined alcohol and social anxiety treatment (both CBT in orientation). Both treatments were equivalent in duration (12-weeks) and both were manualized. The results of the study found that there was no advantage to simultaneously treating both the social anxiety and the alcohol problems. Interestingly, despite the fact that by post-treatment both groups showed markedly less drinking relative to baseline, the group that received the combined treatment exhibited poorer improvement, compared to the group that received alcohol-only treatment. Specifically, at the end of treatment, the combined social anxiety-alcohol treatment group was drinking more alcohol, more often, compared to the alcohol-only treatment group. The results were inconsistent with the authors' original hypothesis which had predicted that the dual-treatment approach would lead to less overall drinking, compared to the alcohol-only treatment, presumably because if the participants were feeling less socially anxious, they would drink less (as predicted by the TRT, SRD, and SMH models). However, engaging in exposure to feared social

situations without the aid of alcohol as required in the combined treatment approach could have lead to greater anxiety levels and thus lower treatment adherence. Further research is warranted to examine this assertion and to determine if there needs to be a focus on developing treatments that lead to greater treatment adherence. Moreover, the demands required of the combined treatment approach may have been too great for many co-morbid individuals, leading to greater drop-out in the combined treatment. In fact, the analyses were intent-to-treat analyses which would penalize the combined treatment for greater drop-out. Methods should be considered to make combined treatments less demanding of clients while still including all necessary components, in an attempt to retain as many individuals as possible, thereby maximizing treatment outcomes (see Conrod & Stewart, in press).

8.2. Pharmacological treatment

Along with cognitive-behavioral approaches to treating SAD, certain drug therapies have also shown to be effective, including selective serotonin reuptake inhibitors (SSRIs) (Davidson, 1998). Randall, Thomas, Thevos, Sonne et al. (2001) examined the effectiveness of one particular SSRI-paroxetine—for the treatment of co-morbid SAD–AUD. The results showed that those randomly assigned to the paroxetine group demonstrated a more significant improvement in their SAD symptoms, compared to the placebo group. These results were important because, despite the fact that paroxetine is well-established in the treatment of SAD, these findings were the first to show the efficacy of paroxetine in clients with SAD that is co-morbid with AUD. With regard to whether reducing social anxiety led to reduced alcohol consumption (as would be suggested by the TRT, SRD, and SMH; Carrigan & Randall, 2003), the results showed that there were no significant differences overall between the paroxetine and placebo groups with regard to frequency and quantity of alcohol consumption. However, by the seventh session, the paroxetine group was showing lower consumption rates. Randall, Thomas, Thevos, Sonne et al. (2001) speculated that with a longer course of treatment than that offered in their study (e.g., 12–21 sessions), perhaps the differences between paroxetine and placebo on alcohol consumption outcomes would become statistically significant.

8.3. Combined treatment

Further research is also necessary to determine whether a bimodal approach combining drug treatment and psychotherapeutic techniques can be efficacious. Bimodal treatment has been shown to be effective in at least one preliminary study. Liappas, Paparrigopoulos, Tzavellas, and Christodoulou (2003) examined social anxiety symptoms only during a detoxification period (4–5 weeks in length) in two groups of individuals with co-morbid SAD–AUD. One group of 21 patients received short-term psychotherapy alone and another group of 33 received short-term psychotherapy combined with mirtazapine (serotonin and norepinephrine reuptake inhibitor). The results demonstrated a greater reduction in social anxiety symptoms in the combined treatment compared to the psychotherapy alone condition. Unfortunately, the study did not examine changes in AUD symptomology or severity from pre- to post-treatment.

In summary, there has been very little research into treatment of co-morbid social anxiety and alcohol use disorders (Schade et al., 2003). A thorough review of the literature found only 12 studies examining treatment outcomes, with several of the studies suffering from major methodological flaws (Schade et al., 2003). Considering the high co-occurrence of these two disorders, it is disconcerting that so little

research has been done with regard to exploring which types of treatments or components within a treatment are most effective in reducing both social anxiety symptoms and alcohol dependence and abuse. Ideally, more research on a large scale such as Project MATCH is required whereby the studies are longitudinal, and include multiple sites across North America. Further, the treatment matches in these studies would be theory based, with clear outcome measures and treatment goals.

Finally, one important methodological change which needs to be made to future research in the SAD area is to not exclude those with co-morbid AUDs from treatment outcome trials. Too often this important subpopulation is excluded to ensure that the sample is "purer" but this limits generalizability and is effectively ignoring an important sub-group of SAD individuals that may be most in need of effective treatments. Whether or not those SAD clients with AUD fare more poorly than those SAD clients without AUD in traditional SAD treatments remains untested to date.

We believe to be most effective, interventions for co-morbid SAD–AUD must start early, preferably when the socially anxious individual is an adolescent. Early interventions with social anxiety may thwart the development of maladaptive patterns of coping-related alcohol use from developing in the first place, and effectively prevent the development of co-morbid SAD–AUDs. In fact, recent research by Kendall, Safford, Flannery-Schroeder, and Webb (2004) has demonstrated that early intervention with anxiety disorders (including SAD) can later impact positively on substance abuse levels. These researchers contacted youths (aged 9–13 years at time of treatment) who had been diagnosed with a primary anxiety disorder (i.e., SAD or generalized anxiety disorder) and had received a 16-week session of CBT for their anxiety disorder, approximately 7.4 years after they had received their treatment. Two of the primary goals of this long-term follow-up study were: (a) to examine the degree to which treatment gains on anxiety symptoms had been maintained; and (b) to examine substance use levels among treated individuals, in relation to a normative sample. The results demonstrated that not only did the majority of the youths maintain their anxiety disorder treatment gains, but importantly, successful treatment of the anxiety disorder was related to less substance abuse more than 7 years post-treatment. These results highlight the importance of treating anxiety disorders before they become lifelong conditions and before maladaptive coping strategies such as alcohol abuse become established/ingrained. It would be interesting for future work to focus specifically on the early treatment of SAD and its impact specifically on alcohol use and problems at longer-term follow-up since the Kendall et al. (2004) study examined early intervention with anxiety disorders (SAD and GAD alike) on substance use more broadly.

Presuming that one is treating an individual who has already developed a co-morbid SAD–AUD problem, we recommend that an ideal combined treatment would involve the following components. First, the individual should be provided with a model that helps them understand how their two problems are inter-related (i.e., they should be acquainted with the notion of the vicious cycle) (see Conrod, Stewart, Comeau, & Maclean, submitted for publication). They should be acquainted with various coping strategies for dealing with social anxiety, both positive and negative (including alcohol misuse), and should be trained in means to avoid the negative ones (see Conrod et al., 2004). In conjunction with cognitive restructuring techniques to address cognitive patterns maintaining social anxiety, part of the treatment should also consist of examining and challenging these clients' current alcohol outcome expectancies and attitudes towards alcohol. In particular, the thoughts which prompt the desire to drink alcohol in specific social situations could be identified and replaced through cognitive restructuring techniques. It would also be important to include steps involving exposure to various feared social situations without the aid of alcohol when working through the SAD client's fear hierarchy. In particular, literature on dealing with "safety signals" in the cognitive-behavioral treatment of anxiety disorders (e.g.,

Ranchman, 1984) could be incorporated into the treatment of such co-morbid individuals, particularly with respect to phasing out their use of alcohol to reduce behavioral avoidance of feared social situations. It should be cautioned that these clinical suggestions are untested speculations that emerge from the findings of our review. Future research in the form of a randomized, controlled trial would be necessary to determine if this type of integrated treatment is more effective than individual treatment of either disorder alone, or of sequential treatment of the two disorders in treating either the SAD and/or AUD symptoms. One would need to pay particular attention to not overwhelming the patient with this type of combined treatment, given the disappointing early results of Randall, Thomas and Thevos (2001) discussed above (see also discussion by Conrod & Stewart, in press).

9. Conclusions and future directions

It has only been in the last decade or so that researchers have begun to seriously explore not only which variables contribute to the high co-morbidity between SAD and AUD, but also to examine which treatments are most effective in treating these complicated patients. The current models for explaining the relationship, including the TRT, SRD, and SMH, although quite promising explanations when considered together, also have several serious shortcomings, including failing to explicitly take individual differences such as gender and factors like social context into consideration. Perhaps other models are necessary to further our understanding, as they would provide an alternative and unique angle by which to understand this complex relationship. We recommend taking an approach that considers social contextual variables (i.e., type of situation [social interaction vs. performance], accessibility and appropriateness of alcohol use, and avoidance, fear, and distress regarding the situation) along with alcohol outcome expectancies and gender to explain the SAD–AUD relationship. To our knowledge, a social contextual framework has not yet systematically been applied to the SAD–AUD relationship and we believe that much could be gleaned from increased use of this perspective in this area of co-morbidity research.

With regard to the current models (TRT, SRD, SMH), they have received some support in the empirical literature, though the findings have not always been consistent. Part of the problem with studies testing these models to date relates to numerous inconsistencies across studies, including the fact that myriad methods for measuring social anxiety and alcohol-related variables have been used across studies. Further, many studies employ very different participants, from those who are shy, to university students scoring high on one or more of a variety of social anxiety measures, to those who are seeking treatment for SAD or an AUD. There will have to be some agreement on which measures are most accurate in diagnosing and classifying individuals, and in providing continuous indices of important features of these two disorders, with the goal of conducting studies that can be reasonably compared. Our review of the literature suggests the tentative conclusion that of the continuous social anxiety measures used to date, those measuring fear of negative evaluation appear most consistently linked to alcohol-related variables. And of the alcohol-related measures used across studies, those measuring alcohol problems (as opposed to alcohol use levels) appear most consistently linked in a positive direction to social anxiety variables.

Recently, research has turned towards exploring which potential variables moderate the relationship between social anxiety disorder and alcohol problems. Several of those variables, including gender, situational context, alcohol expectancies, drinking motives (and their inter-relationships), have produced

interesting results, but further research is necessary. Our review suggests the tentative conclusion that specific alcohol outcome expectancies regarding expectancies for social anxiety reduction are important in explaining the drinking behavior of co-morbid individuals, and that these may be particularly important for men. Similarly, coping drinking motives appear particularly important in contributing to the co-occurrence of social anxiety and drinking problems. Further research is necessary to explore how motivation and expectancy interact in explaining the co-morbidity between SAD and AUD.

With regard to treatments for those suffering from SAD and AUD, little is known about the most effective treatment, though large, multi-site research projects such as Project MATCH are an excellent first step. Additional research is needed to further explore the unexpected finding that a CBT approach designed to simultaneous treat social anxiety and alcohol problems in co-morbid individuals led to worse drinking outcomes than a CBT alcohol-only treatment (Randall, Thomas, & Thevos, 2001). The currently accepted treatment approach for treating those with co-morbid SAD–AUD, is to first treat the alcohol problem, often in a 12-step program, and then to treat the SAD, typically using some form of CBT. This is problematic because the AUD treatment facilitators are generally not trained in SAD treatment, and similarly, the SAD treatment facilitators are often not trained in AUD treatment, and thus the sequential treatment fails to adequately address the inter-relations between the two disorders. In order to better treat those co-morbid with SAD–AUD, more needs to be done to educate and integrate these two treatment protocols, since it makes little sense to treat these disorders as if they were completely separate entities, when we know from research and clinical experience that they are intertwined. As previously mentioned, beginning with alcohol, treatment can be very problematic for an individual with SAD, as the group settings (e.g., 12-step program) likely cause significant social anxiety that could lead to avoidance of the setting or difficulty actively participating and attending to the group content. Without facilitators trained in dealing with SAD, the prognosis would seem to be poor for socially anxious individuals.

More research is also necessary in determining whether bimodal approaches are more effective than single-mode treatments. Further understanding is also needed in determining whether individual or group CBT is best for co-morbid individuals, as some may find the group atmosphere highly intimidating (possibly contributing to drop out) while others may find that group treatment really gives them the support they need to conquer their alcohol addiction as well as an ideal setting for practicing social exposure.

In summary, it is imperative that more research is done to identify which variables contribute to the relationship between SAD–AUD. By identifying the key variables in this relationship, more effective treatments can be developed, and thereby lower the concerning co-occurrence rates of these two disorders. We hope that this review has provided some insight into which variables play a role in the relationship between social anxiety and problem alcohol usage that might prove beneficial in improving treatments in future for those with this common form of psychiatric co-morbidity.

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