Chapter 4
Co-Morbidity of Social Phobia and Alcohol Use Disorders: A Review of Psychopathology Research Findings

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Various empirical studies have demonstrated a relationship between Social Phobia (SP) and Alcohol Use Disorders (AUD), with the presence of one disorder increasing the risk of having the other by two to three times in epidemiological samples (Kushner, Krueger, Frye, & Peterson, this volume). Epidemiological findings also show SP to be co-morbid with a variety of other Substance Use Disorders (SUD) (see chapter 1), but the literature on psychopathology and causal explanations of SP's association with specific illicit drugs and nicotine is just emerging (e.g., Baker, 2001; Sontag, Wittchen, Hofler, Kessler, & Stein, 2000). Thus, this chapter review focuses on psychopathology research findings for the co-morbidity of SP and AUD, with the goal of using the existing literature to build a conceptual framework to delineate the SP-AUD specific association that may also be useful in guiding the next generation of research on SP and other SUD.

In this chapter we review the literature on a spectrum of current biological and psychosocial explanations for the co-occurrence between SP and AUD. Our literature review places primacy on investigations conducted with clinical/diagnosed and subclinical samples of SP and AUD to reduce redundancy with a recent comprehensive review of this topic (Morris, Stewart, & Ham, 2005) and to avoid possible distraction by results that may not be specific to our population of interest. To provide a context for our review, we begin with an overview of the literature on SP-AUD co-morbidity rates, highlighting the effects of symptom severity on the relationship between SP and AUD. We review empirical studies testing directly and indirectly the following hypotheses on the mechanisms linking SP to AUD: (1) Individuals with SP and AUD are genetically predisposed to both disorders, (2) Neurochemical disturbances are related to the occurrence of co-morbid SP and AUD, (3) Individuals with SP use alcohol because it has anxiolytic properties, and (4) Individuals with SP

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consume alcohol because they expect that alcohol use will reduce social anxiety and/or lack confidence in their ability to abstain or moderate their drinking in stressful social situations. The initial results in these research areas suggest that both biological and cognitive factors can account for the co-morbid relationship of SP and AUD. Our chapter concludes with a preliminary model of how genetics, neurobiology, cognitive, and social learning experiences might be considered jointly to provide an integrative and more complete understanding of SP-AUD causal mechanisms than can be achieved by focusing on any single causal pathway alone.

Prevalence of Co-morbid Social Phobia and Alcohol Use Disorders

AUD and SP are the second and third most prevalent psychiatric disorders in the United States, occurring at the rates of 14.1% and 13.3%, respectively (Kessler et al., 1994). In the National Comorbidity Survey conducted with epidemiological samples, the lifetime prevalence of alcohol abuse or dependence was about twice as high (24% vs. 14%) among people with SP as among those without this disorder (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996; see also Kushner et al., this volume). Furthermore, there is an emerging body of data showing that the strength of relationship between SP and AUD is moderated by the severity of the other condition. The following section focuses on data that suggest moderating effects of symptom severity, which have not been previously reviewed.

Severity of Alcohol Dependence

Two investigations with clinical and community samples showed that individuals with SP have higher rates and severity of alcohol dependence than comparator groups, despite little, if any, difference in quantity and frequency of alcohol consumption. Thomas, Thevos, and Randall (1999) found that compared to alcoholics without SP ($n = 397$), individuals with co-morbid SP and alcohol dependence ($n = 397$) endorsed more severe alcohol dependence despite no differences in quantity and frequency of alcohol consumption. Also, in a study comparing treatment-seeking individuals with SP ($n = 54$) or dysthymia ($n = 23$) with normal controls ($n = 27$), typical drinking levels did not differ across groups (Ham, Hope, White, & Rivers, 2002). This finding is notable, considering the research suggesting that SP participants are significantly more likely to be dependent on alcohol compared to dysthymic individuals (e.g., Kessler et al., 1997).

Researchers have suggested several interpretations for these contrasting results for alcohol consumption and severity of alcohol dependence. Thomas et al. (1999) noted that their results may have reflected an artificially inflated severity of alcohol dependence among alcoholics with SP compared to

alcoholics without SP because their alcohol dependence measures (Alcohol Dependence Scale and Alcohol Dependence module of the Structured Clinical Interview for DSM-III-R) were also sensitive to overlapping anxiety symptoms. There was substantial overlap between these measures' alcohol withdrawal symptoms (e.g., anxious feelings, shakes, racing heart, sweating, etc.) and anxiety symptoms that SP individuals experience. In addition, the instruments used to assess alcohol consumption in the above studies may not specifically target the drinking patterns most relevant to socially anxious individuals. Their alcohol use measures assessed overall or typical consumption frequency and quantity rather than frequency of heavy drinking episodes, which is more characteristic of socially anxious individuals' drinking patterns when they do drink.

Investigators have also suggested that the rates and severity of alcohol dependence are not an artifact of the measurement methods, but instead indicate that individuals with co-morbid SP and alcohol dependence are more psychologically dependent on alcohol than alcoholics without SP, in spite of no differences in overall quantity or frequency of use. This position is supported by findings showing that compared to alcoholics without co-morbid SP, those with SP purposively drink alcohol to enhance their social functioning (e.g., Thomas et al., 1999) and reported significantly greater difficulties in controlling alcohol use during their worst lifetime period (Lepine & Pelissolo, 1998). Also supporting the psychological dependence hypothesis is Stewart, Morris, Mellings, and Komar's (2006) finding that coping motives for drinking explained the positive relationship between social anxiety and alcohol problems, despite a lack of an association between social anxiety and drinking levels. Further research is necessary to determine whether method factors or psychological dependence better account for these findings.

Severity of Social Phobia

Two investigations with community samples revealed another counterintuitive finding on the moderating effect of social anxiety severity—that individuals with sublinical social anxiety showed higher risk for alcohol dependence than those who met diagnostic criteria for SP. In their prospective study, Crum and Pratt (2001) found that individuals with subclinical SP ($n = 84$) showed a significantly higher risk for developing an AUD than those with clinical SP ($n = 33$) or no psychiatric disorder ($n = 1044$). Similar findings emerged in the Zurich Cohort Study of Young Adults (Merikangas, Avenevoli, Acharyya, Zhang, & Angst, 2002), which found that the adjusted odds ratio for a co-morbid AUD among individuals with subclinical SP ($n = 70$) was almost double the ratio found among those with clinical SP ($n = 36$). Crum and Pratt (2001) provided two hypotheses to account for these findings: (1) those with clinical SP may be more likely to avoid anxiety-producing

situations that may elicit alcohol use, and (2) individuals with clinical SP may seek treatment more frequently, which could result in early identification and intervention for coincident problem drinking. Future prospective studies are needed to examine these hypotheses and understand why individuals with subclinical SP are more likely than individuals with clinical SP to develop a co-morbid AUD.

Summary and Conclusions

Overall, the prevalence of co-morbid SP and AUD indicates that these disorders frequently co-occur. Interesting findings on the severity of AUD and SP are also noteworthy. First, despite similar levels of alcohol consumption, individuals with SP have elevated rates and severity of alcohol dependence compared to alcoholics without SP. Secondly, individuals with subclinical levels of SP are more likely to have a co-morbid AUD than individuals with clinical SP. These counterintuitive findings are worthy of future research efforts to increase our growing understanding of the complex nature of SP-AUD co-morbidity.

Genetics of Co-morbid Social Phobia and Alcohol Use Disorders

Research highlighting the role of genetics in the etiology of co-morbid SP and AUD can provide a rich empirical resource for delineating the linkage between these disorders. Both twin and family studies have been conducted to investigate the disorders' genetic relationships.

Twin Studies

The twin study method is based on the knowledge that monozygotic twins share all of their genes and dizygotic twins share about 50% of them. Any excess degree of similarity between the monozygotic twins when compared to the dizygotic twins should be due to the influence of genes because all twin pairs are assumed to share the same respective family environments. To date there have been two published twin studies that examined co-morbidity of AUD and SP disorder/symptoms.

In a study with 2,431 pairs of female adolescent twins, Nelson et al. (2000) found that SP, AUD, and major depressive disorder (MDD) shared to varying degree a common additive genetic risk factor. However, AUD also had a disorder-specific additive genetic component, indicating that AUD had

a unique genetic risk factor not shared by SP or MDD. The three disorders did not share any common environmental risk factors. Knopik and colleagues (2004) conducted a study with 2,723 pairs of monzygotic and dizygotic twins to determine the genetic effects on alcohol dependence risk after controlling for other psychiatric factors, including social anxiety problems as determined by a non-diagnostic measure. Based on the residual heritability after accounting for genetic and environmental risk factors, the authors concluded that social anxiety, along with other psychiatric problems, played only a minor role in mediating genetic risk of alcohol dependence. The limited data from these two twin studies suggest that genetics may play a role in the co-morbidity of SP and AUD, though the extent and nature of their genetic link remains to be tested with investigations focusing specifically on this co-morbid relationship.

Family Studies

Family studies were created to explore genetic contributions by examining patterns of familial aggregation of a disorder among relatives of affected probands (individuals with the index disorder). To investigate the genetic influences involved with co-morbid disorders, family studies are designed to inspect patterns of co-aggregation of both disorders. The constellation of familial aggregation provides evidence for or against specific mechanisms of co-morbidity (Merikangas, 1990). Results from family studies can suggest one of two potential mechanisms for the development of a co-morbid condition: (1) a cross-transmission mechanism with the occurrence of each disorder representing an alternative manifestation of shared (common) risk factors, or (2) a causal mechanism with one of the co-morbid disorders predisposing an individual to develop the counterpart disorder.

To demonstrate that co-morbid SP and AUD occur based on cross-transmission, probands with pure (non-co-morbid) forms of either disorder must have relatives at risk for (1) the proband's disorder alone, (2) the co-morbid disorder that the proband does not have, and (3) co-morbid SP and AUD. Alternatively, to show that the co-morbid disorders are causally linked and not connected through cross-transmission, probands with either SP or an AUD must (1) have relatives at risk for the proband's disorder alone and (2) have relatives at risk for the proband's disorder plus the co-morbid condition, but (3) *not* have relatives at risk for the counterpart disorder alone (i.e., the disorder that the proband does not have). For example, if probands with SP have relatives at risk for SP and co-morbid SP and AUD, but do not have relatives at risk for an AUD alone, then the results would indicate SP is causally related to AUD as the alcohol-related disorders do not occur outside the presence of SP.

The Yale Family Study on the familial aggregation of anxiety and AUD was designed to evaluate whether a cross-transmission or causal mechanism better accounts for the relatively frequent co-morbidity of these disorders (Merikangas et al., 1998). Results of this study showed a significant risk for SP among relatives of probands with SP, along with a significant 2.4 odds ratio for alcoholism among socially phobic relatives of probands with SP. Notably, the association was stronger for women and alcohol dependence than men and alcohol abuse, respectively. However, SP in the probands did not increase the risk for a pure AUD among their relatives. Taken together, these results suggest that the two disorders do not share common genetic risk factors by the cross-transmission mechanism, and that SP can cause or contribute to the development of co-occurring AUD.

Summary and Conclusions

These twin and family studies provide an intriguing etiological perspective on SP-AUD co-morbidity. Notably, Merikangas et al. (1998) finding that SP was causally linked to the development of AUD is consistent with clinical studies indicating that SP typically predates AUD in this co-morbid condition (see chapter 1). Overall, these results suggest that both SP and AUD are genetically influenced, but the transmission of this co-morbid condition occurs in a sequential SP to AUD pattern. However, further study is necessary to replicate these findings and determine how these disorders are coupled and transmitted in affected individuals and their relatives.

Neurobiology of Co-morbid Social Phobia and Alcohol Use Disorders

Although few studies have been designed to illuminate the neurobiological factors associated with the co-occurrence of SP and AUD, a review of research examining each independent condition provides useful insight into the neurobiological factors common to both. Data from these two bodies of literature implicate serotonin, gamma amino butyric acid (GABA), and dopamine as potential contributors to co-morbid SP and AUD.

Serotonin (5-hydroxytryptamine, 5-HT)

Pharmacological challenge studies, wherein dynamic measurements after administration of a substance are used to probe endocrine or neurotransmitter

functioning, have provided a useful window into the potential role of serotonin in SP. Both m-chlorophenylpiperazine (m-CPP), a partial serotonin agonist, and fenfluramine, a serotonin releasing agent, have been used in challenge studies to evaluate the role of serotonin in SP (e.g., Hollander et al., 1998, $N = 81$). The results of these studies demonstrated an increase in anxiety after administration of the challenge agents, implicating serotonin dysfunction in SP. The potential role of serotonin in AUD has also been illuminated in empirical studies. For example, Schuckit et al. (1999) followed a group of 41 men ages 21 to 35 who showed low alcohol response, a risk factor for alcoholism because it is a precursor to high tolerance, in a 15-year pilot prospective study and found that low alcohol response was also related to serotonin dysfunction.

The role of serotonin in SP and AUD is also shown in several studies wherein selective serotonin reuptake inhibitors (SSRIs), medications designed to increase serotonin levels by blocking serotonin reuptake, alleviated symptoms of SP (e.g., Stein et al., 1998, $N = 187$) and AUD (e.g., Malcolm, Anton, Randall, & Johnston, 1992, $N = 67$). Further, in a small clinical trial ($N = 15$) testing the efficacy of paroxetine in treating co-morbid SP and AUD, Randall, Johnson, et al. (2001) found that paroxetine yielded improvement for each co-morbid disorder and that reductions in alcohol-related symptoms typically lagged behind SP symptom relief. Notably, the differential treatment response suggests that AUD symptom reduction may have been related to relief from SP symptoms. Another interesting finding is that buspirone, a serotonin partial agonist, has been found effective for alcoholism only when the AUD is accompanied by an anxiety disorder such as SP (see Johnson, 2004). Taken together, results of the reviewed studies suggest that serotonin dysfunction is involved in both SP and AUD. Extrapolating from these findings, it can be suggested that serotonin may be functionally involved in the development of co-morbid SP-AUD, as temporary reductions in social anxiety may result from the increased serotonin activity achieved through alcohol consumption.

Gamma Amino Butyric Acid (GABA)

The role of GABA dysfunction in SP is suggested by findings that benzodiazepines such as alprazolam and clonazepam, which are believed to act on the $GABA_A$ receptor, are effective at providing short-term relief of SP symptoms. For example, Davidson et al. (1993) found in a placebo-controlled clinical trial that a large difference emerged in response rates between individuals with SP receiving clonazepam (80%) and those receiving placebo (20–25%). Research has also shown GABA functioning to be related to alcoholism (Nutt & Malizia, 2001). Lingford-Hughes and colleagues (2005) found a

reduced sensitivity of GABA-BZD receptors for an agonist medication, midalozam, among alcoholics ($n = 11$) compared to a control group ($n = 10$), which implicates GABA dysfunction in AUD. Thus, GABA dysfunction is implicated in both SP and AUD. Furthermore, research on GABA has also revealed a potential direct connection between anxiety and alcoholism, as Nutt (1999) recognized that alcohol's capacity for GABA augmentation is related to its anxiolytic effects. Essentially, as alcohol consumption enhances GABA activity, the increase in GABA results in decreased anxiety levels. GABA may be involved in the development of co-morbid SP and AUD through this receptor mechanism.

Dopamine

A large body of research has indicated that dopamine is involved in the reinforcing properties of alcohol consumption. The relationship between dopamine and alcohol consumption has been shown across animal and human experimental studies, as well as clinical studies examining treatment of AUD with dopamine agonists (Tupala & Tiihonen, 2004). Dopamine involvement in SP is suggested by neuroimaging studies (e.g., Schneier et al., 2000, $N = 20$), along with findings that social anxiety is induced by drugs that block dopamine transmission (Mikkelsen, Detlor, & Cohen, 1981, $N = 15$). Research has also shown that dopamine-enhancing monoamine oxidase inhibitors (MAOIs) are efficacious in reducing the symptoms of SP (Liebowitz, Campeas, & Hollander, 1987), though this medication class is not commonly used to treat SP. Across studies, it appears that dopamine dysfunction is related to both SP and AUD. Furthermore, dopamine may be functionally involved in the development of co-morbid SP and AUD, as alcohol consumption increases dopamine activity, which decreases social anxiety.

Summary and Conclusions

A variety of investigations have suggested that the serotonin, GABA, and dopamine neurotransmitter systems may be involved in both SP and AUD. However, future research is necessary to further evaluate whether and how these neurotransmitters are involved in the co-occurrence of SP and AUD. Also, considering that neurotransmitters do not function in isolation but instead are interconnected, additional efforts that examine the interplay of these neurotransmitters may afford a more comprehensive view of their roles in the co-morbidity between SP and AUD.

Effects of Alcohol on Social Anxiety

Research on the co-morbidity between SP and AUD indicates that SP symptoms often predate alcoholism by several years (Thomas et al., 1999; Tran & Haaga, 2002), and that SP is typically primary when the two disorders co-occur (Kessler et al., 1997; Kushner, Sher, & Beitman, 1990). Based upon this typical order of onset, the most popular explanatory models involve some form of a self-medication hypothesis in which individuals with SP use alcohol to reduce their social fears. In particular, alcohol consumption has been proposed as negatively reinforcing to those experiencing social anxiety due to its ability to reduce tension (Conger, 1956) and dampen one's stress response (Sher, 1987). Along these lines, it has been suggested that alcohol exerts its effects by inhibiting an individual's neurological stress response or by disrupting the self-appraisal process (Sayette, 1993). A number of clinical reports and investigations lend credibility to the self-medication hypothesis, as individuals with SP frequently report using alcohol to cope with their anxiety symptoms (Carrigan & Randall, 2003). Also, Kushner, Abrams, and Borchardt (2000) concluded in a recent review that anxiety disorders such as SP can initiate and maintain alcohol use. Yet, despite the converging evidence for the self-medication hypothesis, research evaluating whether alcohol actually reduces social anxiety has produced overall mixed results.

Empirical Studies

To date only three published studies have directly examined the acute effects of alcohol on social anxiety in individuals with diagnosed SP, each using an alcohol versus placebo design and a speech challenge as an analogue social anxiety situation. It is noteworthy that across time, each successive study has improved upon the design of the study preceding it.

Naftolowitz, Vaughn, Ranc, and Tancer, (1994) initiated this line of research in a study with individuals diagnosed with DSM-III (American Psychiatric Association, 1980) SP ($n = 9$) and age- and sex-matched controls ($n = 9$). This study used a within-subject design in which all participants received a low dose of alcohol (approximately .03% Blood Alcohol Concentration [BAC]) prior to a 10-minute speech on the first study day and placebo prior to another speech on the second study day. The results indicated that alcohol did not reduce subjective anxiety ratings, or alter hormone levels, blood pressure or pulse in the expected directions. However, as noted by the authors, several study limitations may have hindered clear conclusions to be drawn from their findings—a lack of counterbalancing of order of within-subject study conditions, insufficient power due to very small sample size, alcohol dosage being too low to elicit anxiolytic effects, limited credibility of the placebo drink, and low external validity of the social anxiety stressor.

The next study, which included 40 individuals diagnosed with DSM-III-R (American Psychiatric Association, 1987) SP, produced similar results (Himle et al., 1999). All participants in this study engaged in two impromptu speeches across a single experimental session. Each participant received placebo prior to the first speech challenge, but prior to the second speech half of the participants received alcohol while the other half again received placebo. The hypothesis that alcohol reduces social anxiety was again not supported, as no evidence emerged in subjective anxiety ratings, heart rate, or negative versus positive cognitions between the alcohol and placebo conditions. This study improved upon the Naftolowitz et al. (1994) study by enhancing power through inclusion of more participants and using a between-subject design. Despite these improvements, some remaining limitations may have reduced the finding of significant group differences; these included use of alcohol dosage (.03% BAC) too low to produce anxiolytic effects, a sample size being too small to detect medium effect sizes, a speech challenge not producing sufficiently high anxiety, and 25% of the participants being on anxiolytics medications that may have reduced alcohol's effects for these individuals.

Abrams, Kushner, Medina, and Voight (2001) conducted the most recent study of alcohol's direct effects on social anxiety. In contrast to previous research, these investigators employed a three- group design with an alcohol group that expected and received alcohol ($n = 20$), a placebo group that expected but did not receive alcohol ($n = 21$), and a control group that expected and received a non-alcoholic beverage ($n = 20$). The use of this design allowed an evaluation of not only the pharmacological effects of alcohol (alcohol group vs. placebo group), but also the expectancy effects of alcohol (placebo group vs. control group). Of the 61 participants in this study, 90% met DSM-IV (American Psychiatric Association, 1994) criteria for SP, and 10% met all criteria except for the criterion that social anxiety significantly interferes with functioning. All participants engaged in three consecutive 45-minute phases, including a pre-beverage speech phase, a beverage phase, and a post-beverage speech phase. The results of this study showed that both the pharmacological and expectancy effects of alcohol additively contributed to a reduction in social anxiety.

The design used by Abrams and colleagues provided the best investigation to date on the acute effects of alcohol on social anxiety. Noteworthy is this study's targeted .05% BAC in the received/expected alcohol condition, a level greater than the average .03% BAC in the two prior investigations; the .05% BAC is an important methodological change because this BAC level typically produces desired alcohol effects of relaxation and tension reduction in moderate drinkers. Furthermore, the speech challenge produced a level of anxiety higher than that found with the study conducted by Himle and colleagues (1999), and the placebo manipulation check indicated that participants reported subjective feelings of intoxication. Finally, the inclusion of a control beverage condition allowed for an evaluation of the contribution of alcohol expectancies (expected

alcohol effects based on the belief that one consumed alcohol) to the effects of alcohol on social anxiety.

Summary and Conclusions

It is difficult to draw any firm conclusions regarding the effects, if any, of alcohol on social anxiety. It is notable that with each successive study the methodology has improved, and that the most recent and most methodologically sound study (Abrams et al., 2001) did reveal a pharmacological effect of alcohol on social anxiety. Further research is needed in this area with additional improvements in research design to investigate more fully the acute effects of alcohol on SP. Following Abrams et al.'s lead, the effects of expectancy on subjective alcohol effects should be considered in future alcohol administration investigations. The next section on the impact of cognitive variables, especially alcohol expectancies, on alcohol consumption in socially anxious individuals further demonstrates the importance of cognitive variables in the co-morbidity of SP and AUD.

Cognitive Variables Linking Social Phobia to Alcohol Use Disorders

Studies testing the hypotheses that cognitive variables moderate and mediate the relationship between social anxiety and alcohol use are recently growing in number (see recent review by Morris et al., 2005), but very few have been conducted with diagnosed/clinical samples of SP and/or AUD participants. Most of these studies were conducted with undergraduate non-clinical samples consisting of participants who varied widely in their social anxiety or alcohol use problems, with the majority having no or few symptoms associated with these problems. These studies examined the role of social-facilitating alcohol outcome expectancies as a moderator that determines the strength and/or the direction of the relationship between non-clinical social anxiety and alcohol use or drinking related problems. In addition to reviewing research on alcohol outcome expectancies, this chapter also considers a broader range of cognitive variables specific to social situations, coping drinking motives, problem-focused coping, and drink refusal self-efficacy where empirical data suggest that these variables might contribute substantively to understanding the roles of cognitive factors linking SP and AUD. In contrast to alcohol expectancies, drinking motives are reasons why people drink (e.g., because it makes social gathering more sociable). Problem-focused coping is the use of cognitive and behavioral strategies in stressful situations to solve a current problem (e.g., coming up with a couple of different solutions to the problem), and

drink refusal self-efficacy addresses one's confidence in his or her ability to resist alcohol consumption in high-risk situations.

Social learning models provide a useful framework for considering the explanatory roles of alcohol cognitive variables in the co-morbidity of SP and AUD. Consistent with more general principles of social learning theory (Abrams & Niaura, 1987), Burke and Stephens (1999) proposed a social cognitive model to explain the relationship of social anxiety to heavy drinking in college students that is also applicable to conceptualizing the relationship between SP and AUD in general. Most relevant to this discussion is the model's proposal that socially anxious individuals are likely to drink heavily when both of the following conditions exist: (1) they believe that alcohol facilitates social interactions, and (2) they lack other strategies to cope with social anxiety, skills to moderate heavy drinking, and/or drink refusal self-efficacy (low confidence in their ability to resist heavy drinking) in social situations. Specifically, this model indicates that the relationship between social anxiety and heavy drinking is moderated or determined by both alcohol expectancies for social facilitation and coping skills/drink refusal self-efficacy specific to social drinking situations.

Non-clinical Studies Testing Alcohol Expectancies' Moderator Effects

Studies with undergraduate non-clinical samples often did not meet criteria required to test a mediator hypothesis because a direct, positive relationship between social anxiety and alcohol use was not found (Baron & Kenny, 1986). Thus, a moderator hypothesis was consequently tested in these investigations. Two separate studies ($N = 229$ and $N = 521$) conducted by Tran and colleagues at a Northeastern university and a Midwestern university in the United States found very similar results indicating a moderating effect of social-facilitating alcohol expectancies on the relationship between social anxiety and alcohol use/problems, but not in the exact direction initially predicted by Tran, Haaga, and Chambless (1997). Specifically, among participants who did not expect alcohol to reduce their anxiety in social situations, high-social-anxiety participants reported *lower* alcohol consumption; high- and low-social anxiety participants who expected alcohol to reduce their social anxiety *did not differ* in their alcohol consumption (Tran et al., 1997; Tran, Smith, Rofey, & Corcoran, 2002). Only part of these results was replicated in studies with undergraduates conducted by three other research groups (Bruch, Heimberg, Harvey, & McCann, 1992; Bruch, Rivet, Heimberg, & Levin, 1997; Eggleston, Woolaway-Bickel, & Schmidt, 2004; Ham & Hope, 2005). Similar to our result for the low expectancy group, other groups found that social anxiety/shyness was associated with less alcohol consumption/problems, when variance accounted for by alcohol expectancies was considered; however, this effect was a main effect of social anxiety/shyness on alcohol consumption/problems in these studies (Ns = 187–543),

rather than as an interaction effect of social anxiety moderated by low level of alcohol expectancies specific to social situations.

This protective function of social anxiety against high alcohol consumption found in non-clinical undergraduate samples contradicts the consistent epidemiological findings that AUD are more prevalent among individuals with clinical and subclinical SP than found in the general population (e.g., Magee et al., 1996). Along with the fact that moderator studies have been extensively reviewed recently, these conflicting findings reinforce our decision to provide a more detailed coverage of investigations that examined cognitive variables in individuals with clinically and subclinically severe SP and AUD symptoms. Including studies conducted with subclinical social anxiety participants in this review is important in light of the two previously-discussed investigations showing higher prevalence of AUD in individuals with subclinical social anxiety compared to those with SP (Crum & Pratt, 2001; Merikangas et al., 2002).

Studies with Socially Anxious Individuals

Ham and colleagues conducted two studies that found support for situation-specific alcohol expectancies, both of which excluded individuals with co-morbid AUD. In the first study, Ham et al. (2002) compared positive alcohol expectancies in adults with SP ($n = 54$) to those with dysthymia ($n = 23$) and normal controls without any psychiatric disorders ($n = 27$). Their results showed that SP individuals reported stronger expectancies of alcohol's social assertion/facilitation effect than dysthymic individuals; however, no group difference was observed on expectancies of general tension reduction. This situational effect found within the domain of tension/anxiety reduction expectancies was also repeatedly found in investigations conducted by Tran and colleagues (Tran et al., 1997; Tran et al., 2002; Tran, Anthenelli, Smith, Corcoran, & Rofey, 2004). In addition, SP participants were also found to differ from normal controls on expectancies of social assertion, general tension reduction, and global positive changes. Ham, Carrigan, Moak, and Randall (2005) replicated the group difference on expectancies of social assertion when they compared undiagnosed socially anxious ($n = 17$) and non-socially anxious ($n = 45$) individuals from the community. Furthermore, partial correlations with social anxiety as covariates showed that higher social assertion expectancies were related to greater alcohol consumption. It should be noted that the socially anxious participants in this study and the next two studies were selected with stringent criteria such that most would likely meet criteria for SP (c.f. Thomas, Randall, & Carrigan, 2003).

Including research participants that partially overlapped with Ham et al.'s (2005) study sample, the research group at the Medical University of South Carolina published two studies on coping motives for drinking using both explicit and implicit cognitive measures. Results from Thomas et al.'s (2003)

study showed that socially anxious community volunteers ($n = 23$) were more likely than non-anxious controls ($n = 23$) to report using alcohol to feel more comfortable, avoiding social situations if alcohol was not available, and experiencing greater anxiety relief from alcohol use. Carrigan, Drobes, and Randall (2004) found that explicit cognitive measures of drinking to cope also predicted response latency to alcohol-related and social-threat words in the Stroop test, an implicit cognitive measure, among community volunteers ($N = 87$) with a wide range of social anxiety and alcohol use patterns. It is evident from the recent publications that leading alcohol and co-morbidity investigators view coping motives as a promising cognitive mediator to account the co-morbidity of SP and AUD.

Studies with Socially Anxious and Problem-Drinking Individuals

To date four studies have been conducted with participants who reported both social anxiety and alcohol use problems, three of which included participants with diagnosed SP and AUD based on structured diagnostic interviews. Thomas et al. (1999) found that treatment-seeking socially phobic alcoholics ($n = 397$) from Project MATCH reported more symptoms of alcohol dependence and more frequent drinking to improve sociability than alcoholics without SP ($n = 1329$). Abrams and Kushner (2004) further demonstrated in a well-controlled experimental study that general tension-reduction alcohol expectancies marginally moderated the association between consumption of placebo beverage and anxiety responding in individuals with SP. This result showing that alcohol expectancies tended to produce an apparent dose-response psychological effect in the absence of a physical alcohol effect confirms the power of alcohol expectancies in motivating drinking behavior. Furthermore, prior studies demonstrating situation-specificity of general versus social tension-reduction expectancies suggest that this relationship between alcohol expectancies and placebo consumption would likely have been stronger if expectancies of social facilitation, rather than expectancies of general tension reduction had been used in the Abrams and Kushner study.

Drawing from cognitive theory of SP (Beck & Emery, 1985) and a social-learning model of AUD (Abrams & Niaura, 1987), Tran and Haaga (2002) compared three groups of community volunteers on coping responses and alcohol outcome expectancies to determine what distinguishes SP individuals with AUD from those without AUD. The comparison groups included SP participants with current alcohol abuse or dependence ($n = 19$), SP individuals without lifetime AUD ($n = 19$), and normal controls without any current psychiatric disorder ($n = 21$). All three groups differed from each other on the strength of their social facilitating alcohol expectancies and the frequency of which they used problem-focused coping in an alcohol-accessible stressful social situations, which is intended to reflect their general coping

style with social fears when alcohol is present. Specifically, SP-AUD individuals reported the strongest social-facilitating alcohol expectancies and the least use of problem-focused coping, while SP-only individuals scored between normal controls' and SP-AUD individuals' scores on the expectancy and coping measures. Together with Ham et al. (2002, 2005) results, Tran and Haaga's findings suggest that having elevated social anxiety puts one at risk for developing an AUD through decreased ability to use problem-solving strategies in social situations and greater expectations for alcohol's social-facilitating and social-anxiety-reduction effects. Additional support for the social learning model of AUD came from Tran et al. (2004) investigation showing that compared to hazardous drinkers with low social anxiety ($n = 76$), those with high social anxiety ($n = 76$, 51% with subclinical and 49% with clinical symptoms) reported stronger beliefs that alcohol facilitates their social interactions and less confidence in their ability to resist alcohol use in situations when others are drinking. Again demonstrating the situation-specificity predicted by the social learning model, the two groups also did not differ on alcohol expectancies and drink-refusal self-efficacy related to general stress reduction.

Summary and Conclusions

Most of the available literature on cognitive variables linking SP to AUD was based on studies investigating alcohol outcome expectancies. The most apparent result that emerged from this body of investigations is that SP individuals hold specific social-facilitating alcohol expectancies that distinguish them from normal controls and individuals having disorders with overlapping symptoms, including major depression and panic disorder. Also, situation-specificity of alcohol expectancies was demonstrated by consistently different patterns of results for expectancies of social facilitation (social anxiety reduction) and general tension reduction. In addition, recent studies also highlight coping drinking motives, self-efficacy, and problem-focused coping as potentially important cognitive variables to consider in accounting for the relationship between SP and AUD. Given the infancy of research on the latter cognitive variables in the SP-AUD co-morbidity literature, further investigations to refine their constructs and measurements with respect to drinking in social situations and in response to social fears would likely facilitate future research on their roles as moderators and mediators of the association between SP and AUD. The finding that the mean scores of SP individuals who have not developed AUD were between the scores of SP-AUD participants and normal controls suggests that these cognitive vulnerabilities may exist on a continuum, putting individuals with SP at increased risk for developing AUD compared to those without SP.

While the above conclusions based on cross-sectional findings are important for developing a working model for how cognitive variables influence the

development of co-morbid SP and AUD, they remain working hypotheses without prospective and further experimental studies that can empirically test causal relationships among social anxiety, potential cognitive mediators, and alcohol use problems in subclinical and clinical populations. Considering prior research indicating a negative or non-significant association between social anxiety and alcohol use in non-clinical undergraduate samples (e.g., Tran et al., 1997; Eggleston et al., 2004), it would be more conceptually sound and statistically appropriate to test for cognitive mediator effects in research samples consisting of individuals with both social anxiety and alcohol use problems.

General Conclusions and Future Directions

Review of the literature shows initial support for the hypotheses that genetics, neurobiology, and cognitive factors are involved in the co-morbidity of SP and AUD. As with any new areas of research, causal interpretations of the empirical data are limited by the small number of investigations in each research domain and methodological limitations, especially the use of cross-sectional methods. Given the correlational and cross-sectional designs that have been largely used to date, these findings remain working hypotheses to be tested in prospective and experimental investigations.

It is clear from the literature reviewed that much remains to be done in explicating the causal mechanisms for SP-AUD co-morbidity. Both model development and empirical studies are needed to facilitate this effort. Traditionally, biological and cognitive models to explain causal mechanisms are developed and tested independently. Such independent testing does not provide an integrative and complete picture of factors that may jointly influence development of AUD in SP individuals. On a practical level, recruitment of co-morbid research participants whose prevalence rates are lower than the singly diagnosed can be challenging and would benefit from collaborative efforts of investigators studying different causal contributors to the SP-AUD relationship. Based on the current data, we propose a preliminary model of how these variables might be considered jointly. Figure 4.1 provides a schematic representation of this model. In this model, genetics, neurobiology, and cognitive factors are hypothesized to contribute directly to the development of SP-AUD co-morbidity. Genetic factors are expected to influence neurobiological and possibly cognitive factors that further increase the vulnerability of SP individuals to problematic drinking. Consistent with the limited body of genetic findings, a causal mechanism from SP to AUD is proposed to explain their co-morbidity. Furthermore, any genetic influences on cognitive factors are expected to be moderated by social learning experiences. In particular, socially anxious individuals who have developed alcohol expectancies for social facilitation through vicarious learning or direct experience with alcohol's reinforcing effects may drink to cope with their social anxiety and have a low

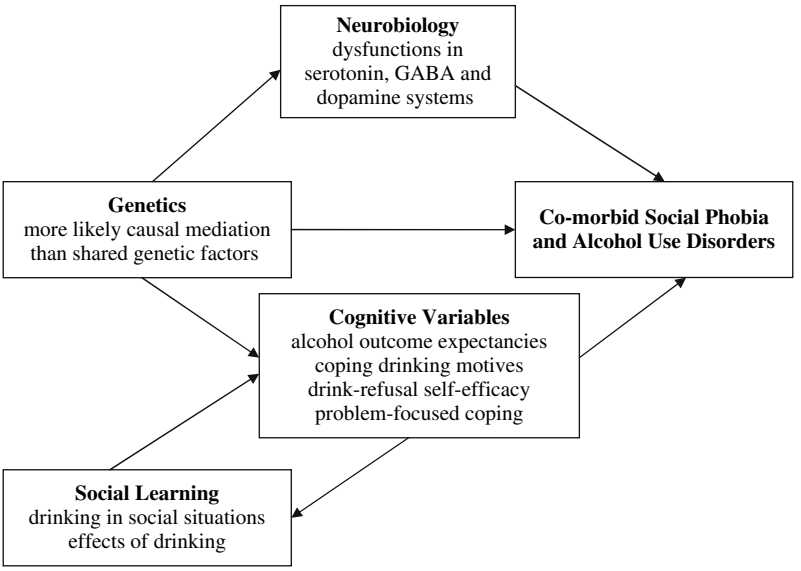

Fig. 4.1 Biopsychosocial model for co-morbidity of social phobia and alcohol use disorders

confidence in their ability to resist using alcohol in social situations. Within the context of this general model, well-developed specific models on how SP is linked to AUD can be developed and empirically tested. Furthermore, our model may provide a framework for building systematic research on the causal mechanisms linking SP to other SUDs. Specific biopsychosocial models of psychopathology may also inform development of comprehensive and ideally integrative treatment for individuals with or at risk for developing co-morbid SP and AUD (for review of current treatments, see chapter 8). Examining co-morbid causal relationships in the context of an integrated biopsychosocial context is consistent with the current trend for interdisciplinary research promoted by the National Institutes of Health, the primary funding source for innovative and scientifically important research on psychopathology and treatment of psychiatric and substance use disorders in the United States.

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