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IMPLICATIONS OF COMORBID ALCOHOL DEPENDENCE AMONG INDIVIDUALS WITH SOCIAL ANXIETY DISORDER

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Abstract

Background—Social anxiety disorder (SAD) is highly comorbid with alcohol use disorders (AUD) yet the nature of this comorbidity remains unclear. To better understand these associations, we first examined whether SAD was related to AUD above and beyond relevant covariates. Second, we examined the psychosocial impairment associated with the comorbidity of SAD and AUD versus SAD without AUD. Third, the temporal sequencing of SAD and AUD among comorbid individuals was examined.

Methods—Participants included 5,877 (50% females) adults from the National Comorbidity Survey.

Results—As predicted, SAD was related to alcohol dependence (not abuse) after controlling for relevant conditions, indicating that SAD is linked to more severe alcohol impairment and that this link is not better accounted for by other pathology. Results also supported the hypothesis that the addition of alcohol dependence to SAD resulted in greater impairment across a variety of domains relative to SAD without alcohol dependence (e.g., greater rates of health care utilization, other psychiatric diagnoses, health problems, and greater interpersonal stress). Additionally, for the majority of comorbid individuals, SAD onset predated alcohol dependence onset, suggesting SAD increases vulnerability for misusing alcohol.

Conclusions—Together, these data lend support for the contention that SAD may serve as a risk for alcohol dependence and indicate that the co-occurrence of these two conditions may result in greater personal and public health care costs.

Keywords

social phobia; social anxiety; alcohol; comorbidity; impairment

INTRODUCTION

Social anxiety disorder (SAD) is associated with high rates of alcohol use disorders.[1] Almost half of individuals with DSM-IV lifetime SAD meet criteria for lifetime prevalence of an AUD [2] compared to 18.6%[3] in the general population. Furthermore, the 12-month prevalence of AUD among SAD individuals is 13.1%[2] versus 8.5% among the general population.[4]

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Among patients seeking treatment for alcohol-related problems, 23–39% meet diagnostic criteria for SAD.[5–8] SAD appears to be associated with increased risk of alcohol *dependence* more so than abuse[2,9,10] suggesting that SAD is linked to a risk of greater alcohol-related impairment. Among the anxiety disorders, SAD shows a particularly problematic risk profile for comorbid AUD, as SAD is associated with higher rates of AUD relative to most other anxiety disorders[10] and adolescent SAD (but not other anxiety or mood disorders) appears to serve as a risk for adult alcohol dependence.[9]

Despite the high comorbidity rates between SAD and AUD, the extant literature linking AUD to SAD is limited in a number of ways. First, the impact of AUD on SAD is understudied as little empirical attention has focused on how individuals with comorbid SAD–AUD differ from unimorbid cases. Second, earlier work examining differences between unimorbid and comorbid individuals has been limited to treatment settings and information about the impact of comorbidity among non-treatment samples is limited. A third limitation in the extant literature is that the temporal relationship between SAD and AUD has not been fully elucidated so it is currently unclear whether SAD predicts later AUD or vice versa. Fourth, the majority of studies in this area tend to combine alcohol abuse and alcohol dependence diagnoses, making it difficult, if not impossible, to demarcate whether individuals with SAD are at increased risk for alcohol abuse or dependence. A fifth limitation to the extant literature is that little attention has been paid to the role of other types of psychopathology in the relation between SAD and AUD.[10]

We know of no systematic examination of the impact of AUD on individuals with SAD. The few studies examining this question indicate that the high rates of comorbidity between SAD and AUD are associated with greater impairment than either disorder without the other. For instance, among treatment-seeking participants in Project MATCH,[11] patients with current AUD and a lifetime diagnosis of SAD were more likely to have major depressive episodes, experience more severe alcohol dependence and exhibit lower occupational status and less perceived peer social support than patients with AUD without SAD.[8,12] Similarly, when compared to patients with SAD and no history of AUD, patients with current SAD and a lifetime history of AUD have been found to exhibit more severe SAD symptoms and are less likely to be married, indicating relative impairments in interpersonal functioning.[6] However, several critical domains have not been explored. Regardless of comorbidity, SAD in and of itself is associated with increased personal and societal costs. To illustrate, SAD is associated with impairment in mental and physical health as evidenced by high rates of comorbidity with other anxiety and mood disorders[13] and reports of poor perceived physical health.[14] Approximately 22% of individuals with SAD receive public assistance.[13] Relative to unimorbid cases, individuals with SAD and other comorbid conditions have reported greater impairment in mental and physical health and to be more likely to inform a health care provider of their condition.[15] If, compared to unimorbid cases, individuals with both SAD and AUD also experience greater rates of other psychopathology and physical health problems as well as engage in increased health care utilization, public health costs could be even greater for this group.

The work that has examined the impact of comorbidity on those with unimorbid SAD or AUD has examined these relations in treatment settings.[6,8,12] We could find no reports regarding the impact on functioning of AUD comorbidity among individuals in the general population who may not be in treatment. Given that epidemiological data indicate that 80–95% of people with SAD report receiving *no* treatment,[2,13] patterns observed in treatment-seeking populations may or may not reflect the experiences of the majority of people with SAD. Clarification of the impact of comorbidity could contribute substantially to our understanding of the nature of this comorbidity and may inform prevention and treatment efforts. To illustrate, social support is associated with better response to AUD treatment.[16] If individuals with

comorbid SAD and AUD do in fact demonstrate poorer social support as suggested by earlier work,[6,12] treatment of comorbid individuals may need to incorporate specific strategies to improve social functioning.

The temporal relations between AUD and SAD among comorbid individuals remain unclear. Evaluation of typical age of onset of SAD and AUD suggests that SAD serves as a risk factor for subsequent AUD (i.e., it precedes the future development of alcohol use problems). Mean age of onset is usually earlier for SAD than AUD among individuals with co-occurring SAD and AUD.[6,10,17–19] Additionally, adolescent SAD has been found to significantly predict adult alcohol dependence.[9] Yet, these studies typically examine mean or typical age of onset, limiting the ability to examine whether for some individuals there is a shared diathesis or even whether for some, AUD occurs before SAD. Given evidence suggesting that anxiety disorders can serve as risk for the development of AUD and that alcohol dependence appears to serve as a risk for the development of at least some anxiety disorders,[20] the reliance on mean age of onset date may obfuscate the possibility that alcohol misuse may serve as a risk for subsequent SAD in at least some comorbid individuals. Differential temporal relations may be associated with different types of impairment. Individuals who develop SAD after AUD as a consequence of embarrassment regarding alcohol-related activities may present somewhat differently than individuals with SAD who develop AUD as a result of using alcohol to manage their social anxiety. However, to date, the relevance of differential temporal relationships remain empirically unclear and unstudied.

Similarly, it is currently unclear whether individuals with SAD are at increased risk for alcohol abuse or dependence as the few studies in this area usually combine alcohol abuse and alcohol dependence diagnoses.[6,8,12,21] This distinction is important because these are unique types of substance use problems.[22,23] For instance, abuse is not merely a prodromal phase of alcohol dependence but a unique condition[23] with dependence characterized by compulsive alcohol use behaviors and/or physiological tolerance or withdrawal.[24] Dependence seems to be a more chronic and impairing condition relative to alcohol abuse.[23] Given evidence suggesting that individuals with SAD may be particularly vulnerable to alcohol dependence, [2,9,10] future work is needed to more directly and definitively examine the relations between SAD and alcohol abuse and dependence.

It is also important to examine the role of other types of psychopathology in the relation between SAD and AUD as it is unknown whether SAD demonstrates a specific relationship to AUD after accounting for other types of psychopathology, including internalizing and externalizing disorders, related to alcohol problems. It may be that the high rates of AUD among individuals with SAD are simply attributable to co-occurring axis I pathology. Indeed, SAD often co-occurs with other anxiety disorders as well as depression[14] and AUD are often prevalent in individuals with other anxiety conditions[25] and depression.[26–27] Similarly, both social anxiety and alcohol problems are related to other substances such as marijuana[29–31] Externalizing disorders such as conduct disorder demonstrate high comorbidity with anxiety disorders[32] and predict later AUD.[33] These high rates of comorbidity suggest that the link between SAD and AUD could be attributable to other types of related psychopathology. Only one known study has evaluated whether SAD is linked to AUD above and beyond relevant axis I psychopathology.[9] Although this study found SAD onset to be prospectively linked to subsequent AUD onset after accounting for other psychopathology, the small number of individuals in some diagnostic categories indicates further work with large representative samples is needed to examine the unique explanatory power of SAD in relation alcohol-related problems.

The present investigation contributes to the elucidation of the relationship between SAD and AUD. The first goal of this study was to replicate and extend earlier work in this area[9,10] by

examining the comorbidity of SAD and its specificity with AUD (i.e., abuse and dependence) after controlling for theoretically relevant variables (e.g., gender, depression, conduct disorder, other anxiety disorders) using a large epidemiological database. Consistent with earlier work, [9] it was hypothesized that after controlling for relevant variables, SAD would be significantly related to alcohol dependence but not abuse. The second goal was to replicate and extend earlier findings[6] that comorbid SAD and AUD is related to greater impairment relative to SAD alone. Specifically, it was predicted that, relative to individuals with SAD without alcohol dependence, people with comorbid SAD and alcohol dependence would demonstrate greater impairment across several domains including social support, mental health, physical health, and health care utilization.

The third goal of this study was to build upon past work regarding temporal patterns of AUD and SAD in several ways. Earlier work has demonstrated that mean age of onset of SAD precedes that of AUD.[10] We strove to extend this finding by predicting that although the majority of people with comorbid SAD and AUD would demonstrate earlier age of SAD onset relative to AUD onset, some individuals would demonstrate AUD onset before SAD onset. This prediction is consistent with past findings that examine differential temporal relations with AUD in samples that combine individuals with various anxiety disorder diagnoses.[5] In addition, we investigated whether unimorbid individuals' age of SAD and AUD onset differed from those with comorbid SAD and AUD. It was hypothesized that comorbid cases would exhibit earlier age of onset relative to unimorbid SAD but, consistent with earlier work,[8] not unimorbid AUD cases. Finally, we examined whether individuals with SAD before AUD differed from individuals with AUD before SAD in terms of demographics and level of impairment (in areas of social support, mental health, physical health, and health care utilization). On the basis of earlier work with panic-prone substance users,[34] we hypothesized these two groups to differ. Specifically, consistent with the notion that people with SAD before AUD would demonstrate earlier age of onset and a more chronic course of psychopathology, it was hypothesized that this group would demonstrate greater impairment.

METHODS

This study draws on the National Comorbidity Survey (NCS), a nationwide epidemiological study designed to assess the prevalence and psychosocial correlates of DSM-III-R psychiatric disorders.[35] The NCS was administered to a national US household sample of over 8,000 individuals. A sub-sample of respondents ($N = 5,877$) completed a more extensive second survey that extended the information assessed in the initial phase and incorporated questions related to the diagnostic criteria for psychiatric disorders. This study will therefore focus on this smaller sub-sample. The NCS was administered in the early 1990s by extensively trained individuals who were supervised by the Survey Research Center at the University of Michigan. In total there were 158 interviewers who had, on average, 5 years of experience interviewing with the Survey Research Center. For more information on the surveyor agreement/consistency, quality assurance, and data quality checks please refer to.[35]

SAMPLING, WEIGHTING, AND PARTICIPANTS

The participants were selected through a multistage area, probability sample (based on household) within a stratified sample of counties within the United States. Response rate was 82.6%. A series of weights were used to take into account national population distributions, non-response, and variations in probability of selection. Participants were interviewed in their home. Informed consent was obtained from each participant as well as from parents for those under the age of 17 years. There was an equal distribution of men and women (50% female), and the average age was 33.2 years ($SD = 10.70$; range from 15 to 54 years). Ethnicity was as follows: 75.6% Caucasian, 11.6% African American, 9.4% Hispanic, and 3.4% categorized as

“Other”. Further details on the sampling, weighting, and participants can be found elsewhere. [36,37]

MEASURES

Participant’s psychiatric diagnoses—A modified version of the Composite International Diagnostic Interview (CIDI) 1.0,[38] a comprehensive semi-structured interview, was used to assess the participant’s lifetime DSM-III-R psychiatric diagnoses.[39] The reliability and validity of the CIDI have been established. To date, a number of studies have demonstrated good to excellent κ coefficients for the majority of diagnostic sections, including the ones relevant to this study.[40,41] Moreover, the CIDI has been found to have good agreement coefficients with routine clinical diagnoses, checklist diagnoses, and other scales.[42] Disorders assessed by the CIDI included illicit substance abuse and/or dependence (SUD), alcohol abuse and/or dependence, major depressive disorder (MDD), bipolar, dysthymia, panic disorder, agoraphobia with or without panic disorder, social phobia, simple phobia, generalized anxiety disorder, posttraumatic stress disorder (PTSD), antisocial personality disorder (ASPD), and conduct disorder (all lifetime history). Age of onset was determined for all endorsed diagnoses by retrospective recall. It is of note that DSM-III-R criteria are comparable to DSM-IV criteria for the majority of the disorders of interest for the present investigation. For instance, although there are nine possible symptoms of substance dependence in DSM-III-R, the DSM-IV combined some of these symptoms to simplify the criteria and reduce redundancy. Dependence criteria for both DSM versions are quite similar.[43] Regarding SAD, there are very slight differences between DSM-III-R and DSM-IV criteria (e.g., the inclusion of minors in DSM-IV). Further, there is evidence of excellent concordance between Axis I diagnoses derived from the Structured Clinical Interview for DSM Disorders (SCID) for DSM-III-R and the SCID for DSM-IV e.g.[44]

Social anxiety-related impairment—The extent to which social anxiety resulted in self-reported life interference was assessed using two questions. First, participants rated the extent to which their social anxiety fear(s) ever interfered with their life or activities. Second, participants rated the degree to which avoidance of social anxiety-provoking situations interfered with their life or activities. Interference caused by social anxiety and avoidance was each assessed using a 4-point Likert scale (ranging from *a lot to not at all*). Given data suggesting that social anxiety-related fears and avoidance are not distinct aspects of social anxiety,[45] these two items were summed to create a total social anxiety-related impairment. Reliability analyses indicated strong internal consistency among these two items ($\alpha = 0.80$).

Perceived physical and mental health problems—We utilized three items to characterize a participant’s self-reported physical and mental health. Participants were asked to rate their overall mental health and subsequently rate their overall physical health using a 5-point Likert scale (ranging from *excellent to poor*). Also, participants were presented with a list of serious health problems and asked to indicate which they had experienced during the last year. Health problems included: arthritis, rheumatism, asthma, blindness or deafness, bronchitis, tuberculosis, diabetes, hypertension, heart problems, hernia, kidney, liver disease, lupus, thyroid, autoimmune disorders, neurological problems, stroke, stomach or gallbladder disease, ulcers, cancer, and any other serious health problem. Responses were coded 0 (absent) or 1 (present) and items were summed to obtain a cumulative health problem variable. Single-item self-report measures of health are associated with objective measures of health.[46]

Perceived social support—The NCS included several items on perceived social support.[47] These measures have been found to have adequate reliability.[48] Similar to earlier NCS investigations examining these social support items,[48,49] three separate measures of perceived social support were obtained. The first addressed support from participants’ spouses

or partners for those who were married or living in a marriage-like relationship. The second and third measures addressed support from non-spouse relatives and friends, respectively. Participants rated only applicable relationships. That is, if they did not have a partner, for example, they would indicate that item was not applicable and not rate the quality of support for that domain. For each of these three relationship categories, perceived support was assessed with a six-item questionnaire on which participants were asked to rate the extent to which the person(s) cared about, understood, and appreciated the participant, as well as how much the participant believed he or she could rely on this person(s) for help when in need, how much the participant could open up to this person(s) about personal worries, and how much the participant could relax and feel at ease around this person(s). Participants rated their answers on a 4-point Likert-type scale ranging from (1) *Not at all* to (4) *A lot*. Possible scores accordingly ranged from a minimum of 6 (reflecting minimal perceived support) to a maximum of 24 (reflecting high perceived support). Reliability analyses indicated strong internal consistency for all three support scales ($\alpha = 0.83, 0.87, \text{ and } 0.88$, respectively, for partner, relative, and friend support). Examination of the inter-item correlation matrices revealed moderately strong inter-item correlations between all items on each of the three scales (r between 0.36 and 0.54, 0.43–0.62, and 0.46–0.68, respectively, for partners, relatives, and friends), suggesting that each of the three separate support scales was unidimensional, and that items were not so strongly intercorrelated as to be redundant.

Perceived social stress—Participants' perceived interpersonal stress and/or conflict with partners/spouses, relatives, and friends were also assessed independently with six-item questionnaires.[49] For each of the three relationship categories, each participant was asked to rate how often the person/group in question made too many demands, made him/her feel tense, argued, criticized, let him/her down, and got on his/her nerves. Participants responded using a 4-point Likert-type scale ranging from (1) *Not at all* to (4) *A lot*. Possible scores on each scale accordingly ranged from a low of 6 (reflecting minimal conflict and stress) to a high of 24 (reflecting high stress and conflict). Reliability analyses indicated strong internal consistency for all three stress scales ($\alpha = 0.81, 0.83, \text{ and } 0.82$, respectively, for partner, relative, and friend stress/conflict). Examination of the inter-item correlation matrices revealed moderately strong, positive inter-item correlations between all items on each of the three scales (r between 0.32 and 0.55, 0.35–0.58, and 0.34–0.57, respectively, for partners, relatives, and friends), suggesting that the interpersonal stress scales were unidimensional and that items were not so strongly intercorrelated as to be redundant.

Health care utilization—Included in the diagnostic section for each of the specific types of disorders were a series of questions that assessed health care utilization in relation to each disorder. First, participants were asked whether or not they had ever told a health care provider about their symptoms. This question was asked with regard to general practitioners, mental health specialists, and other professionals (e.g., nurses, pastors). In regard to SUD, individuals were also questioned with regard to the utilization of substance-specific self-help groups and substance-specific treatment programs. Finally, for each disorder, participants were asked about their utilization of medication to relieve symptoms. For the purposes of this report, we created a dichotomous *health care utilization* variable for each axis I disorder (0 = no utilization; 1 = utilization), which captured whether or not an individual had ever approached each of the above-listed entities-seeking treatment for their symptoms. These measures of health care utilization for each disorder are similar to those used in earlier NCS investigations. [50,51]

STATISTICAL ANALYSES

First, bivariate zero-order correlations were conducted to examine the relations between SAD, gender, and those diagnoses found in earlier work to be related to SAD and/or AUD (i.e.,

lifetime history of generalized anxiety disorder, simple phobia, panic disorder, agoraphobia with or without panic disorder, PTSD, MDD, dysthymia, bipolar I disorder, SUD, ASPD, or conduct disorder). The relations among alcohol abuse and dependence and these diagnoses can be found elsewhere.[10] Next, bivariate logistic regression analyses were performed with SAD and each of the criterion variables (i.e., alcohol abuse and dependence). Third, a multivariate logistic regression analysis was conducted to assess the unique contribution of SAD to relevant criterion variables, above and beyond the shared variance explained by relevant risk factors. Gender was entered in step 1 of the model, followed by other psychiatric diagnoses in step 2. In step 3 of the model, lifetime history of SAD was entered. This model ensures that observed effects for predictor variable at level 3 cannot be attributed to the variance with the variables at levels 1 or 2.[52]

To better understand the implications for impairment of alcohol dependence among those with SAD, χ^2 analyses, one-way analyses of variance (ANOVAs), and logistic regressions were conducted to examine whether individuals with both SAD and alcohol dependence differed from those with SAD without alcohol dependence in terms of demographics (gender, race, years of education, family income, marital status) and the experience of other psychiatric diagnoses. Given the large number of analyses conducted, Bonferroni corrections were utilized to control for type I error ($P = .05/16 = .003$). Next, we examined whether individuals with SAD with and without a history of alcohol dependence differed on psychosocial, physical, and psychiatric functioning. Specifically, t tests and χ^2 analyses were conducted to test for group differences in interpersonal functioning (e.g., perceived social support, perceived social stress), perceived physical and mental health, and health care utilization (related to social fears). Again, Bonferroni corrections were applied to control for type I error ($P = .05/14 = .004$).

To examine temporal relations of SAD and alcohol dependence among comorbid individuals, we examined the number of individuals in the following groups: (1) age of onset of SAD reported to precede that of alcohol dependence (referred to as SAD–AUD), (2) age of onset of SAD reported to follow that of alcohol dependence (referred to as AUD–SAD), and (3) age of onset of SAD reported to occur within the same year as that of alcohol dependence. Next, one-way ANOVAs were conducted to investigate differences in ages of onset of SAD and alcohol dependence between unimorbid and comorbid participants as well as between the SAD–AUD and AUD–SAD groups.

To better understand the implications of differential temporal relations of SAD and AUD among comorbid individuals, χ^2 analyses, t tests, and logistic regression analyses were conducted to examine whether the SAD–AUD and AUD–SAD groups differed in terms of demographics and other psychiatric disorders. We also tested whether these groups differed in interpersonal functioning (e.g., perceived social support, perceived social stress), perceived physical and mental health, and health care utilization (related to social fears and alcohol use problems). Bonferroni corrections were also applied to these analyses.

RESULTS

RELATIONS AMONG PREDICTOR AND CRITERION VARIABLES

Lifetime history of SAD was significantly and positively correlated with lifetime history of all other disorders and female gender ($r = 0.09$ – 0.32 , $P < .001$). Consistent with prediction, bivariate logistic regression analyses indicated that a history of SAD did not increase risk for alcohol abuse (OR = 1.23, 95% CI = .97–1.57, $P = .09$), but did significantly increase the odds of alcohol dependence (OR = 2.26, 95% CI = 1.88–2.70, $P < .001$).

UNIQUE RELATIONSHIP BETWEEN SOCIAL ANXIETY DISORDER AND ALCOHOL DEPENDENCE

Given that SAD was only associated with alcohol dependence (not abuse), a multivariate logistic regression analysis was conducted to test the hypothesis that SAD would be related to alcohol dependence above and beyond the shared variance explained by relevant risk factors. As predicted, even after controlling for statistically significant covariates (11 comorbid psychiatric disorders and gender), lifetime history of SAD remained associated with a significantly increased odds of alcohol dependence (OR = 1.49, 95% CI = 1.19–1.88, $P < .001$).

IMPACT OF COMORBIDITY

Next, the impact of comorbidity was tested. First, we tested the hypothesis that comorbid cases would differ from people with SAD without alcohol dependence (i.e., unimorbid cases) on demographic and other psychiatric diagnoses (Table 1). The two groups differed on sex, marital status, and race. Specifically, the comorbid cases were more likely to be men and Caucasian, whereas the unimorbid group was more likely to have never been married. The two groups did not differ on any other demographic variable. Concerning other psychiatric diagnoses, comorbidity was associated with greater rates of the following diagnoses: PTSD, MDD, other SUD, conduct disorder, and ASPD.

Next, we tested the impact of comorbidity on perceived psychosocial, physical and psychiatric functioning (Table 2). In regard to psychosocial functioning, comorbidity was associated with significantly less social support from friends but was unrelated to social support from partners and non-spousal relatives. Comorbidity was also associated with greater relationship stress concerning relatives and friends (but not partners). Interestingly, the unimorbid individuals reported greater social anxiety-related impairment. The two groups did not significantly differ on perceived mental and physical health status, although there was a trend for comorbidity to be associated with greater physical health problems. Regarding health care utilization, comorbidity was associated with greater likelihood to have informed mental health specialists and other professionals about their social fears as well as greater use of prescription medication to manage social fears.

TEMPORAL RELATIONS BETWEEN SOCIAL ANXIETY DISORDER AND ALCOHOL DEPENDENCE

Given data establishing a unique link between SAD and alcohol dependence, we pursued analyses designed to test our hypothesis that the majority of comorbid cases would demonstrate earlier age of SAD onset relative to AUD onset (i.e., SAD–AUD group), but that some comorbid individuals would exhibit AUD onset before SAD onset (i.e., AUD–SAD group). As expected, the majority of participants ($n = 156$, 80.1%) fell into the SAD–AUD group. However, the AUD–SAD group constituted a substantial number of individuals within the overall comorbid group ($n = 31$, 15.7%). A small minority ($n = 8$, 4.2%) reported that the age of onset of SAD occurred in the same year as onset of alcohol dependence (M age of onset = 16.9 years, $SD = 4.23$). Given their small number, this latter group was excluded from further analyses.

Next, we examined the hypothesis that comorbid cases would exhibit earlier age of onset relative to unimorbid SAD, but not unimorbid AUD, cases. To test whether comorbidity was associated with earlier age of SAD relative to unimorbid SAD, one-way ANOVA indicated these three groups (unimorbid SAD, SAD–AUD, and AUD–SAD) significantly differed on age of onset of SAD, $F(2, 783) = 83.55$, $P < .001$. Post hoc comparisons were conducted using Tukey's HSD procedure. As expected, the significant mean difference (3.71, $P < .001$) between unimorbid SAD and SAD–AUD groups indicates that the age of onset of SAD among unimorbid SAD individuals ($M = 15.06$, $SD = 7.66$) was significantly later than SAD–AUD

individuals ($M = 11.35$, $SD = 4.43$). The significant mean difference (-14.67 , $P < .001$) between unimorbid SAD and AUD–SAD groups indicates that the age of onset of SAD among unimorbid SAD people was earlier than AUD–SAD people ($M = 29.72$, $SD = 9.55$). The significant mean difference (-18.37 , $P < .001$) between SAD–AUD and AUD–SAD groups indicates that the age of onset of SAD among SAD–AUD individuals was earlier than AUD–SAD individuals. To test whether comorbidity was associated with earlier age of alcohol dependence relative to unimorbid alcohol dependence, a second one-way ANOVA was performed comparing age of onset for unimorbid alcohol dependence, SAD–AUD, and AUD–SAD groups. This model was not significant, $F(2, 783) = .38$, $P = .68$, indicating that the age of alcohol dependence onset among those with unimorbid alcohol dependence ($M = 21.66$, $SD = 6.34$), SAD–AUD ($M = 22.17$, $SD = 7.43$), and AUD–SAD ($M = 21.69$, $SD = 6.74$) did not significantly differ between groups.

Last, we tested whether the SAD–AUD group exhibited greater impairment than the AUD–SAD group. In addition to the variables examined in Tables 1 and 2, we also tested whether the groups differed on rate of health care utilization associated with their AUD. First, we tested whether the two groups differed on demographic variables and other psychiatric diagnoses and found no significant differences ($P > .003$). Next, we examined whether the SAD–AUD and AUD–SAD groups differed in terms of perceived psychosocial, physical, and psychiatric functioning. The two AUD–SAD were significantly more likely to report using prescription medications to manage social fears ($\chi^2(1, 185) = 9.93$, $P = .002$). There were no other significant differences ($P > .004$).

DISCUSSION

This study serves as the first known systematic, epidemiological investigation of the impact of lifetime AUD on individuals with SAD. Findings from this study contribute to our understanding of the relations between SAD and AUD in several ways. First, as predicted, SAD was significantly associated with alcohol dependence, but not abuse. Importantly, this relationship emerged even after controlling for relevant variables, including gender and other psychiatric pathology. These data, in conjunction with past research,[2,9,10] support the contention that SAD is indeed related to alcohol dependence and that this effect is not attributable to other factors related to SAD and AUD.

Second, this study determined that the co-occurrence of alcohol dependence among individuals with SAD was related to impairment across a wide range of domains, including less peer social support, greater stress in peer and relative relations, and greater health care utilization, as well as a tendency toward greater self-reported physical health problems. These data support past work suggesting that individuals with both SAD and alcohol dependence are particularly vulnerable to problems in interpersonal functioning.[6,12] We extended this work by determining that the combination of SAD and alcohol dependence was associated with greater impairment in mental health (as evidenced by greater vulnerability to a large number of other psychiatric disorders). Further, the comorbid group demonstrated higher rates of health care utilization which is important to consider in light of the tremendous societal cost associated with the nonpsychiatric medical treatment for individuals with anxiety disorders.[53] Thus, greater health care utilization associated with their SAD combined with higher rates of nonpsychiatric medical treatment among people with anxiety disorders suggests that the mental and physical health-related impairment associated with SAD–AUD comorbidity may result in greater personal and societal costs.

Prominent theoretical models of the SAD–AUD comorbidity posit that SAD serves as a risk factor for the development of AUD, as it is believed that individuals with social anxiety use alcohol to manage distress associated with their concerns regarding negative evaluation and

scrutiny by others, increasing their AUD vulnerability,[1,9,54,55] Cross-sectional work provides partial support for this contention, as socially anxious individuals are more likely to report using alcohol in situations involving negative affect and perceived peer pressure[54] and to avoid social censure.[56] Our examination of the temporal relations between SAD and alcohol dependence among comorbid individuals suggests there may be multiple pathways by which SAD–AUD comorbidity can develop. Specifically, our findings indicate that, for the vast majority of individuals with co-occurring SAD and alcohol dependence (80%), onset of SAD preceded that of alcohol dependence. This evidence of temporal antecedence supports patterns observed prospectively.[9] When considered in light of these prospective findings, our data support the notion that SAD may serve as a risk factor for alcohol dependence.[57] Combined with our finding that this relation remains after controlling for relevant covariates, our data are consistent with the idea that SAD may serve as a *unique* risk factor for the development of alcohol dependence.[9]

However, our data also indicate that in some instances of comorbidity, individuals experienced SAD onset *after* the onset of alcohol dependence. This novel finding suggests that there may be at least *two* developmental pathways for comorbid SAD and alcohol dependence (SAD precedes alcohol dependence for the majority of comorbid individuals although for some, alcohol dependence precedes SAD) and is consistent with earlier work examining other anxiety conditions.[5,34] It may be that some individuals with alcohol dependence demonstrate a predisposition toward social anxiety (i.e., shyness). These people also may tend to experience alcohol-related impairment in social domains, increasing their anxiety regarding negative evaluation in subsequent social interactions. However, future prospective work is necessary to determine whether alcohol dependence does, in fact, serve as a risk for the development of SAD and to then investigate the causal mechanisms underlying this newly identified group of alcohol dependent individuals who suffer from later onset SAD, as this population may respond differently to treatment and prevention efforts.

The identification of different temporal relations of SAD and AUD among comorbid individuals does not preclude the possibility that SAD and alcohol dependence share a reciprocal relationship. Socially anxious individuals report using alcohol in situations involving negative affect.[54] However, the present data combined with prospective findings [9,58] suggest that when SAD individuals use alcohol, they tend to experience alcohol-related impairment. This impairment may make SAD people more socially anxious when social situations are encountered as they may fear negative evaluation by others based on past alcohol-related behaviors. These individuals may then experience an increased urge to use alcohol to manage their increased negative affect associated with possible negative evaluation, thereby creating a vicious cycle. Future work examining a reciprocal model of social anxiety and alcohol dependence will provide valuable information regarding the mechanisms underlying the development of alcohol dependence among individuals with SAD.

It is noteworthy that the differential temporal relations of SAD and alcohol dependence among comorbid individuals were associated with almost no significant differences in impairment. This finding suggests that the comorbidity of SAD and alcohol dependence (regardless of the temporal relationship of onset) is associated with greater impairment relative to individuals with SAD without alcohol dependence.

This study has some limitations that suggest important directions for additional work in this area. First, the cross-sectional nature of the data hinders the ability to tease apart causal relations and prospective work is necessary to delineate the causal relations among SAD, AUD, and associated impairment. Second, the present investigation relies on self-report data of psychiatric symptomatology, age of onset, etc. Future work using a variety of research methodologies (e.g., cross-informant assessment, laboratory-based manipulation of social

anxiety) is therefore necessary. Third, single-item, self-report assessments of health may have psychometric limitations. However, research has shown strong convergent validity between single-item measures and full-item versions of scales.[59] With regard to single-item self-report measures specifically of health, a number of validation studies have been conducted with longer and more objective health rating.[60,61] Indeed, objective measures of health are the strongest predictor of single-item self-report measures of health.[46] Furthermore, perceived health has been shown to provide an accurate gauge of physical health outcomes, [62,63] to possess good reliability,[64] have good predictive validity,[65] and good agreement with physician diagnosis.[66] Fourth, items assessing social support were unidimensional in nature. Although our findings serve as an important initial step in understanding the complex relations among social support, SAD, and AUD, future work may benefit from the examination of multiple facets of social support that may be particularly relevant to people with SAD and/or AUD. Fifth, diagnostic decisions were made using DSM-III-R criteria. Although excellent concordance has been found between axis I diagnoses made with DSM-III-R and DSM-IV criteria,[43,44,67] future work is necessary to determine whether the observed relationships generalize to samples diagnosed using more contemporary conceptualizations of pathological symptomatology.

In sum, the present data are consistent with the notion that SAD serves as a significant and unique risk factor for the development of subsequent alcohol dependence. In contrast, it appears for at least some individuals, the problematic use of alcohol may contribute to increase in social anxiety. Our findings indicate that regardless of the developmental pathways by which the comorbidity develops, individuals with lifetime histories of both SAD and alcohol dependence are at particular risk for impairment across a variety of domains that may result in greater personal and societal costs. Given the impairment experienced by this group, further work is clearly warranted to uncover the mechanisms underlying the associations between SAD and alcohol dependence to inform prevention and treatment programs for high-risk individuals.

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TABLE 1

Impact of comorbidity on demographics and other psychiatric disorders

Criterion variable	Unimorbidity (<i>n</i> = 599)	Comorbidity (<i>n</i> = 195)	Statistic	<i>P</i>
Gender (% male)	35.9	60.0	$\chi^2(1, 793) = 35.14$	$3.07e^{-009}$
Race (% Caucasian)	77.1	84.1	$\chi^2(1, 793) = 4.29$	.038
Years of education (<i>M, SD</i>)	12.27 (2.10)	12.09 (2.11)	$F(1, 792) = .98$	.322
Family income <\$20,000 (%)	30.0	32.8	$\chi^2(1, 794) = .55$	.458
Never married (%)	36.9	27.7	$\chi^2(1, 793) = 5.50$	.019
Generalized anxiety disorder (%)	10.5	21.0	OR = 2.26, CI = 1.47–3.48	.0002
Specific phobia (%)	35.1	41.0	OR = 1.29, CI = .93–1.80	.128
Panic disorder (%)	9.7	11.7	OR = 1.22, CI = .73–2.04	.450
Agoraphobia (%)	20.0	27.2	OR = 1.48, CI = 1.02–2.16	.039
Posttraumatic stress disorder (%)	12.5	25.0	OR = 2.31, CI = 1.54–3.46	.00005
Major depressive disorder (%)	33.6	46.2	OR = 1.71, CI = 1.23–2.38	.001
Dysthymia (%)	13.2	20.0	OR = 1.65, CI = .92–10.57	.067
Bipolar I disorder (%)	.8	2.6	OR = 3.12, CI = 1.09–2.52	.019
Illicit substance use disorders (%)	9.8	49.0	OR = 8.75, CI = 5.94–12.90	$6.21e^{-028}$
Conduct disorder (%)	16.0	38.5	OR = 3.30, CI = 2.30–4.74	$9.34e^{-011}$
Anti-social personality disorder (%)	2.8	22.1	OR = 9.57, CI = 5.32–17.19	$4.46e^{-011}$

Unimorbidity, individuals with SAD without alcohol dependence; comorbidity, individuals with both SAD and alcohol dependence. To control for type I error, Bonferroni corrections were utilized ($P = .05/16 = .003$). Bolded values are significantly different at the .003 level. CI = 95% confidence interval.

SAD, social anxiety disorder.

TABLE 2

Impact of comorbidity on perceived psychosocial, physical and psychiatric functioning

Criterion variable	Unimorbidity (<i>n</i> = 599)	Comorbidity (<i>n</i> = 195)	Statistic	<i>P</i>
Social support (partner) (<i>M</i> , <i>SD</i>)	21.75 (2.81)	22.07 (2.44)	$t^2(1, 462) = -1.08$	.281
Social support (relatives) (<i>M</i> , <i>SD</i>)	20.13 (3.92)	19.27 (4.23)	$t^2(1, 784) = 2.58$	.010
Social support (friends) (<i>M</i> , <i>SD</i>)	20.09 (3.60)	18.82 (5.05)	$t^2(1, 258.22) = 3.24$	.001
Relationship stress (partner) (<i>M</i> , <i>SD</i>)	14.46 (3.78)	14.52 (3.72)	$t^2(1, 461) = -.14$	.888
Relationship stress (relatives) (<i>M</i> , <i>SD</i>)	13.48 (4.18)	14.65 (4.09)	$t^2(1, 785) = -3.40$	.001
Relationship stress (friends) (<i>M</i> , <i>SD</i>)	12.24 (3.62)	13.58 (3.61)	$t^2(1, 790) = -4.44$	1.04e ⁻⁰⁰⁵
Social anxiety-related impairment	4.61 (1.76)	3.99 (1.57)	$t^2(1, 364.15) = 4.63$	5.06e ⁻⁰⁰⁶
Perceived physical health (<i>M</i> , <i>SD</i>)	.86 (.35)	.83 (.38)	$t^2(1, 792) = .98$	.329
Perceived mental health (<i>M</i> , <i>SD</i>)	.87 (.34)	.84 (.36)	$t^2(1, 793) = 1.00$	.318
Physical health problems (#)	.62 (1.05)	.91 (1.39)	$t^2(1, 270.67) = -2.70$	.007
Told general practitioner about social fears (%)	4.7	8.8	$\chi^2(1, 792) = 4.58$	.032
Told mental health specialist about social fears (%)	8.0	22.2	$\chi^2(1, 790) = 28.69$	8.48e ⁻⁰⁰⁸
Told other professionals about social fears (%)	8.4	20.0	$\chi^2(1, 792) = 19.99$	7.78e ⁻⁰⁰⁶
Used prescription medication for social fears (%)	4.5	12.9	$\chi^2(1, 791) = 16.73$	4.30e ⁻⁰⁰⁵

Unimorbidity, individuals with SAD without alcohol dependence; comorbidity, individuals with both SAD and alcohol dependence. To control for type I error, Bonferroni corrections were utilized ($P = .05/14 = .004$). Bolded values are significantly different at the .004 level.

SAD, social anxiety disorder.