

Published in final edited form as:

J Psychiatr Res. 2009 January ; 43(4): 477–483. doi:10.1016/j.jpsychires.2008.04.012.

Understanding social anxiety as a risk for alcohol use disorders: Fear of scrutiny, not social interaction fears, prospectively predicts alcohol use disorders

Julia D. Buckner^{a,*} and Norman B. Schmidt^b

^a Department of Psychiatry, Yale University School of Medicine, 389 Whitney Ave., New Haven, CT 06511, USA

^b Department of Psychology, Florida State University, Tallahassee, FL 32306-1270, USA

Abstract

Increasing evidence indicates that social anxiety may be a premorbid risk factor for alcohol use disorders (AUD). The aim of this study was to replicate and extend previous work examining whether social anxiety is a risk factor for AUD by evaluating both the temporal antecedence and non-spuriousness of this relationship. We also examined whether social anxiety first-order factors (social interaction anxiety, observation anxieties) served as specific predictors of AUD. A non-referred sample of 404 psychologically healthy young adults (i.e. free from current or past Axis I psychopathology) was prospectively followed over approximately two years. Social anxiety (but not depression or trait anxiety) at baseline significantly predicted subsequent AUD onset. The relationship between social anxiety and AUD remained even after controlling for relevant variables (gender, depression, trait anxiety). Further, social anxiety first-order factors differentially predicted AUD onset, such that observation anxieties (but not social interaction anxiety) were prospectively linked to AUD onset. This study provides further support that social anxiety (and fear of scrutiny specifically) appears to serve as an important and potentially specific AUD-related variable that deserves serious attention as a potential vulnerability factor.

Keywords

Alcohol use disorders; Social anxiety; Social phobia; Prospective studies; Comorbidity; Risk factors

1. Introduction

Alcohol use disorders (AUD) and social anxiety disorder (SAD) are highly comorbid (e.g. Kessler et al., 1997). For instance, 48% of individuals with a lifetime diagnosis of SAD also meet criteria for a lifetime diagnosis of AUD (Grant et al., 2005). These high rates are cause for concern because patients with both AUD and SAD demonstrate greater impairment and more severe symptoms than patients with only one of these disorders (Buckner et al., in press; Schneier et al., 1989; Thomas et al., 1999). Theories of the SAD–AUD link posit that

*Corresponding author. Tel.: +1 850 644 1070; fax: +1 850 644 7739. buckner@psy.fsu.edu (J.D. Buckner).

Contributors

Dr. Schmidt designed the study and wrote the protocol. The first author generated the present study's hypotheses, performed data analyses, and conducted the literature review. All authors contributed to and have approved the final manuscript.

Conflict of interest

No author has any conflicts of interest to report regarding this manuscript.

individuals with social anxiety use alcohol to manage anxiety and that something about their alcohol use increases AUD risk (e.g. Buckner et al., in press, 2006, 2008b). In other words, theoretical models of social anxiety posit that social anxiety acts as a risk factor for the development of AUD.

Garber and Hollon (1991) suggest three criteria for establishing whether a construct acts as a risk factor in psychopathology research. First, the vulnerability factor must be correlated with the outcome. Second, the vulnerability factor must demonstrate temporal precedence. Third, the relation between the vulnerability and outcome variables must be non-spurious (i.e. not due to a third variable). Regarding the first criterion, social anxiety consistently covaries with alcohol problems and AUD (see Morris et al., 2005). Concerning the second criterion, social anxiety appears to temporally precede AUD. Examination of mean age of onset suggests SAD onset typically predates AUD onset in comorbid patients (Buckner et al., in press, 2008a; Randall et al., 2001; Terra et al., 2006). Prospective studies generally indicate a link between social anxiety and alcohol problems, though an association between the formal diagnoses is less consistent. One study found adolescent SAD predicted alcohol dependence by age 30 (Buckner et al., 2008a). Yet, among German adolescents, SAD predicted greater rates of hazardous drinking (but not AUD) (Zimmermann et al., 2003) and in adults, sub-clinical (but not clinical) SAD was linked to AUD 13 years later (Crum and Pratt, 2001). Finally, less work has examined the non-spurious criterion regarding the social anxiety –AUD link, though initial findings suggest that SAD is related to AUD even after controlling for gender and a wide range of relevant Axis I and Axis II disorders (Buckner et al., in press, 2008a).

Thus, the evidence indicates that social anxiety appears to be a risk for at least some types of problematic drinking. Initial findings also indicate that this relation may be specific to social anxiety and not the result of relevant cooccurring variables (e.g. depression, other anxiety). It therefore follows that there may be something about the nature of SAD that increases AUD risk. Three components of social anxiety have been identified (Safren et al., 1998): interaction anxiety (fear of social interactions), observation anxiety (fear of being observed by others), and fear others will notice symptoms of anxiety. Each component may be associated with different mechanisms related to AUD risk. For example, individuals with interaction anxiety may use alcohol for social facilitation. This hypothesis is supported by the finding that social anxiety is related to the expectation that alcohol will increase social assertiveness (Ham et al., 2002) although social anxiety does not appear related to social facilitation motives for alcohol use (Buckner et al., 2006; Ham et al., 2007). Alternatively, it may be that individuals with observation anxieties use alcohol to attenuate observable anxiety reactions. However, we know of no studies that have examined this hypothesis.

The first aim of this study was to further establish whether social anxiety is a risk factor for AUD by evaluating both the temporal antecedence and non-spuriousness of this relationship. These relations were examined among psychologically healthy young adults (i.e. free from current or past Axis I psychopathology). This approach was adopted in an effort to limit the extent to which findings could be confounded with pre-existing psychopathology. If other types of psychopathology common to AUD were present, non-spuriousness could be more difficult to examine as relations could be due to other psychopathology relevant to both social anxiety and AUD. Additionally, the use of a young, healthy sample allows for the examination of the relations between social anxiety and AUD prior to the point at which social anxiety and alcohol use problems could interact to create a vicious cycle of social anxiety leading to alcohol use which in turn produces greater social anxiety and greater reliance on alcohol to mediate social anxiety reactions (see Buckner et al., in press, 2008b).

It was expected that baseline social anxiety would predict new AUD at follow-up and that this relation would remain after controlling for variables that co-occur with both social anxiety and

AUD (gender, depression, trait anxiety). Our second aim was to refine knowledge about potential mechanisms that may account for the social anxiety –AUD link by examining more specific components of social anxiety in the prediction of AUD.

2. Materials and methods

2.1. Sampling and recruitment

The present study was a secondary analysis of data collected in the context of an intervention study that targeted individuals with high anxiety sensitivity (AS), or fear of anxiety-related sensations (Schmidt et al., 2007b). The original study utilized a multi-stage recruitment process to target high-risk (based on self-reported anxiety sensitivity) individuals from the Columbus, OH metropolitan area middle and high schools ($n = 46$), the Ohio State University ($n = 263$), and the Columbus, OH community ($n = 96$). Recruitment strategies differed across target. Print media were used to recruit from the community. OSU undergraduates were recruited from introductory psychology classes and received course credit for their initial assessment. School-based recruitment methods were considerably more involved and directed through administrative offices at OSU to school districts. School districts and school principals received descriptions of the project. Upon their consent, investigators arranged with specific schools to administer the study description to individual classrooms. Investigators conducted brief presentations regarding the nature of the study in classrooms for over 2700 school children. Students were also provided with a written description, a copy of the *anxiety sensitivity index (ASI)* (Reiss et al., 1986) used for screening, and a consent form. Students were instructed to take the forms home for their parents to read and complete. As an incentive, students received \$5 for completed consent forms regardless of whether they and their parents consented to participate in the study. To examine whether there were differences between those recruited from middle/high schools, university, or community, baseline demographic (age, race, gender) and psychiatric variables (anxiety treatment history, social anxiety measures (total scores and subscales), depression, trait anxiety) were analyzed using multivariate analysis of variance (MANOVA) models for continuous variables and χ^2 tests for nominal/categorical variables. Although there were no differences between these groups on any demographic or psychiatric variable (p 's $>.05$), recruitment group status was included in regression analyses to ensure observed effects were not better accounted for by this variable.

All interested participants were screened to determine potential eligibility and were invited to participate based on their scores on the ASI, the most common measure of AS, and questions about prior psychiatric treatment. Interested individuals with no current or recent psychiatric history (no diagnoses in the past 12 months and no current Axis I diagnosis) and scoring >1.5 standard deviations above the mean for a non-clinical community sample (Schmidt and Joiner, 2002) were considered as possible participants. Although the intervention in the original study resulted in reductions in AS, this reduction appears to have been specific to AS and no reductions in other types of anxiety, depression, or other psychopathology were observed (Schmidt et al., 2007b). In the present report, we statistically control for experimental condition in all regression analyses to ensure observed effects were not better accounted for by this variable.

The demographic makeup of the sample at baseline was as follows: by design the sample was relatively young, with approximately 95% under the age of 25 (see Table 1). The sample was primarily Caucasian (74%) with 10% African-American, 9% Asian-American, 2% Hispanic, and 3% Other. The average and modal grade level was one year of college. Completion of college was the most frequently reported level of parental education with 30% of mothers and 29% of fathers finishing college (for more information see Schmidt et al., 2007a,b,2006).

2.2. Measures

The *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-Patient Edition (SCID)* (First et al., 1994) was utilized during the clinical interview to determine Axis I diagnoses. Interviews were conducted by advanced graduate students in clinical psychology who had received extensive training in SCID administration and scoring. Training included reviewing SCID training tapes, observing taped SCID administration, observing live SCID administration, and conducting SCID interviews with a trained interviewer. Interviewers received feedback throughout this process until they demonstrated high reliability. These same training procedures have been used in other projects in our laboratory that have generated high inter-rater reliability for all Axis I diagnoses (Schmidt et al., 1997). A consensus method of diagnosis was used at weekly staff meetings where positive diagnostic findings were reviewed. For some follow-up evaluations, the SCID was conducted over the telephone. In the case of co-occurring diagnoses, the designation of primary diagnosis was based on the condition that created the most distress and impairment for the individual. Raters at follow-up were blind to baseline assessment data. Inter-rater reliability projects conducted in the laboratory over the project's course showed high levels of reliability among these raters (Schmidt et al., 2005).

The *Social Phobia Scale (SPS)* and the *Social Interaction Anxiety Scale (SIAS)* (Mattick and Clarke, 1998) were used to assess social anxiety. When administered together, items from these two measures tap the three components of social anxiety outlined above and comprise three subscales (Safren et al., 1998). Although these three components load onto a single, higher order factor of social anxiety, they represent differentiated first-order factors. The social interaction subscale comprised 17 items, the observation anxiety scale consisted of 11 items, and the fear others will notice scale comprised 12 items (see Table 1 for ranges, *M*'s, and *SD*'s). All subscales demonstrate high internal consistency (α 's = .90 interaction anxiety; .85 observation anxiety; .89 fear others will notice anxiety). In the present study, scores ranged from 0–56 (SIAS: *M* = 20.34, *SD* = 13.28; SPS: *M* = 12.74, *SD* = 11.34).

The *Spielberger Trait Anxiety Inventory (STAI)* (Spielberger et al., 1983) was used to measure trait anxiety. The trait anxiety measure is a 20-item self-report measure in which participants rate from 1–4 the degree to which each item (e.g. "I feel frightened") reflects how they generally feel (Spielberger et al., 1983). Excellent reliability and convergent validity have been reported and individuals with anxiety disorders score significantly higher than non-clinical participants (Spielberger et al., 1983).

The *Beck Depression Inventory (BDI)* (Beck and Steer, 1987) was used to measure depression over the past 2 weeks. Each question is scored on a 0–3 scale. Total scores on the BDI can range from 0 to 63, with higher scores reflecting greater levels of depressive symptoms. The BDI has yielded adequate reliability estimates and has been well validated as a measure of depressive symptomatology (Beck et al., 1988; Beck and Steer, 1987).

2.3. Procedure

Following written, informed consent, participants completed the clinical interview and, if eligible, a battery of self-report measures. Eligible participants were then randomly assigned to one of two conditions (treatment or control). Those assigned to the treatment condition received an intervention designed to reduce levels of AS whereas control participants received a control intervention that focused on health and nutrition but did not directly address AS (see Schmidt et al., 2007 for details). Both conditions involved information delivered via an audio–visual computer presentation lasting approximately 30 min followed by 10 min spent with an experimenter. Community and high school participants received \$25 as compensation for the baseline assessment whereas college students received course credit.

The study design included both a 12- and a 24-month follow-up evaluation though there was attrition resulting in missing data at both follow-up evaluations. However, the percentage of participants completing either a 12- or 24-month follow-up did not differ across experimental condition. Participants who dropped out of the study did not differ from study completers on baseline demographics, clinical variables, cognitive vulnerability, or condition (p 's $>.10$). Participants received \$25 for completion of a follow-up evaluation.

The present study utilized the 24-month assessment data to examine onset of new AUD diagnoses at this later time point. For follow-up assessments, participants were given the option of returning to the clinic to complete the evaluation or having it conducted over the telephone since studies comparing phone versus in-person interviews have shown little difference (Fenig et al., 1993; Sobin et al., 1993). During the follow-up SCID evaluations, only current (versus lifetime) diagnoses were evaluated to determine onset of new diagnoses. Self-report measures were mailed to participants within a week of the scheduled live or telephonic interview. Those undergoing a telephone interview were instructed to mail completed measures back to the project. During the follow-up period, there was a total incidence of approximately 4% ($n = 11$) new AUD diagnoses (in all cases AUD was the primary diagnosis).

2.4. Statistical analyses

First, bivariate correlations were performed to examine the relations between baseline psychiatric measures and demographic variables. Partial correlations were conducted to control for condition.¹ Next, logistic regression analyses were performed to examine whether baseline demographic variables and non-social anxiety psychiatric measures (trait anxiety, depression) predicted follow-up AUD onset.

Third, to test whether various components of social anxiety predicted follow-up AUD, a second series of logistic regression analyses was conducted. For each regression model, follow-up AUD diagnosis (0 = absent, 1 = present) served as the dependent variable. SIAS–SPS total score and subscales served as independent variables and separate regressions were performed for each. Finally, to examine non-spuriousness of observed relations, additional hierarchical logistic regression analyses were conducted to examine whether significant SIAS–SPS measures were prospectively linked to AUD onset after controlling for variables significantly related to baseline SIAS–SPS scores or follow-up AUD. At level 1, condition², gender, depression, and trait anxiety were entered. At level 2, the independent variable was entered. Thus, any observed effects of variables at level 2 are unique and cannot be attributed to variance with factors at level 1 (Cohen and Cohen, 1983).

3. Results

3.1. Descriptive information

Table 2 displays correlations between baseline age, gender, social anxiety and other psychiatric measures. SIAS–SPS total score and subscales were significantly associated with depression and trait anxiety. AUD onset was related to male gender (OR = .21, 95% CI = .06–.83, $p = .03$) but was not significantly associated with any other baseline covariate: trait anxiety (OR = 1.03, 95% CI = .90–1.17, $p = .67$), depression (OR = 1.08, 95% CI = .98–1.19, $p = .14$), and age (OR = .99, 95% CI = .85–1.16, $p = .99$).

¹These data are derived from a primary prevention study that included both an experimental and a prospective design. The present report is specifically focused on the longitudinal (versus the experimental) component of the design. At follow-up, 2.2% ($n = 3$) of patients in the treatment condition exhibited an AUD compared to 5.1% ($n = 8$) in the control condition. This difference was not statistically significant, $\chi^2 = 1.69$, $p = .19$. Nevertheless, in the present report, we statistically controlled for experimental condition in all analyses.

²To further rule out the possibility that experimental condition could have impacted the findings, all relevant analyses were run using only participants in the control condition. These analyses yielded the same pattern of findings as the overall sample.

3.2. Prediction of alcohol use disorders using various components of social anxiety

Consistent with expectation, SIAS–SPS total predicted AUD onset ($OR = 1.03$, 95% $CI = 1.01–1.06$, $p < .05$). Observation anxiety ($OR = 1.13$, 95% $CI = 1.03–1.23$, $p < .01$) and fear that others will notice anxiety symptoms ($OR = 1.09$, 95% $CI = 1.02–1.17$, $p = .01$) also predicted AUD. AUD was not significantly related to interaction anxiety ($OR = 1.04$, 95% $CI = .99–1.10$, $p = .13$). After controlling for gender, depression, and trait anxiety, SIAS–SPS total score, observation anxiety, and fear others will notice anxiety symptoms remained significant predictors of AUD onset (Table 3).

4. Discussion

The present findings provide support for the contention that social anxiety is implicated in the psychopathogenicity of AUD, corroborating prior work that found SAD appeared to serve as a risk for AUD (e.g. Buckner et al., in press, 2008a; Crum and Pratt, 2001). Specifically, social anxiety was correlated with AUD and temporally preceded AUD onset. Importantly, observed relations occurred even after controlling for a variety of relevant variables (e.g. trait anxiety, depression, gender). Further, neither trait anxiety nor depression at baseline predicted subsequent AUD onset. These data, in conjunction with prior evidence that SAD appears to serve as a risk for AUD after controlling for other internalizing and externalizing disorders (Buckner et al., 2008a), suggest the relation of social anxiety and SAD to subsequent AUD may be non-spurious.

Notably, this investigation serves as the first known prospective examination of components of social anxiety that may be particularly relevant to AUD development. It appears that fear of scrutiny (i.e. observation anxieties) may be particularly relevant in AUD development. Thus, individuals that use alcohol to manage observable anxiety reactions appear to be at greater risk for alcohol-related problems. Further work aimed at identifying the ways in which particular components of social anxiety lead to AUD could provide invaluable information that could inform prevention and treatment efforts.

Our finding that observation fears appear to serve as a risk for AUD does not preclude the possibility that, for some individuals, problematic alcohol use may result in increased social anxiety. In fact, data from the National Comorbidity Survey suggest that although for most people with comorbid AUD and SAD, SAD onset temporally precedes that of AUD onset, for some, AUD onset may have occurred prior to SAD (Buckner et al., in press). It follows that for some people, the experience of alcohol-related impairment in social domains may increase observational fears regarding negative evaluation in subsequent social interactions. In other words, social anxiety (specifically observational fears) and alcohol-related problems may work in concert to produce a vicious cycle of alcohol use to manage social anxiety followed by increased social anxiety in subsequent social interactions resulting in greater alcohol use, etc (Buckner et al., 2008b). Thus, further prospective work is necessary to fully delineate the complex relations between social anxiety and AUD.

This study should be considered in light of limitations that suggest the need for additional empirical work in this area. First, although the initial sample size was quite large, the study yielded a relatively low incidence of psychopathology (particularly AUD onset) during the follow-up interval and replication with larger samples is necessary to strengthen confidence in the present findings. Second, the length of the follow-up interval was relatively brief in the context of the development of psychopathology (approximately two years) and future work should examine social anxiety and observation anxieties over longer intervals. Third, this study utilized individuals participating in a primary prevention study concerning AS. Although we statistically controlled for experimental condition, replication using a naturalistic design could help assuage concerns regarding study design. We attempted to strengthen confidence that the

experimental component of the study design did not interfere with the reported effects by replicating our results using only the control condition participants.²

In sum, exaggerated anxiety regarding being observed by others may be particularly important in the development of AUD. This finding has important implications regarding the treatment and prevention of these highly comorbid disorders, as early treatment focused on attenuating observation-related fears could serve to prevent AUD development among high-risk individuals.

Acknowledgments

Funding Source

This research was supported in part by a National Research Service Award from the National Institute of Drug Abuse (F31 DA12457-01) awarded to Julia D. Buckner. NIDA had no further role in the study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

We have no acknowledgements to report.

References

- Beck, AT.; Steer, RA. Manual for the revised beck depression inventory. San Antonio, TX: Psychological Corporation; 1987.
- Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol* 1988;56:893–7. [PubMed: 3204199]
- Buckner JD, Eggleston AM, Schmidt NB. Social anxiety and problematic alcohol consumption: The mediating role of drinking motives and situations. *Behav Ther* 2006;37:381–91. [PubMed: 17071215]
- Buckner JD, Schmidt NB, Lang AR, Small J, Schlauch RC, Lewinsohn PM. Specificity of social anxiety disorder as a risk factor for alcohol and cannabis dependence. *J Psychiat Res* 2008a;42:230–9. [PubMed: 17320907]
- Buckner JD, Timpano KR, Zvolensky MJ, Sachs-Ericsson N, Schmidt NB. Implications of comorbid alcohol dependence among individuals with social anxiety disorder. *Depress Anxiety*. in press
- Buckner JD, Ledley DR, Heimberg RG, Schmidt NB. Treating comorbid social anxiety and alcohol use disorders: Combining motivation enhancement therapy with cognitive-behavioral therapy. *Clin Case Stud* 2008b;7:208–23.
- Cohen, J.; Cohen, P. Applied multiple regression/correlation analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Erlbaum Associates; 1983.
- Crum RM, Pratt LA. Risk of heavy drinking and alcohol use disorders in social phobia: a prospective analysis. *Am J Psychiat* 2001;158:1693–700. [PubMed: 11579004]
- Fenig S, Levav I, Kohn R, Yelin N. Telephone vs face-to-face interviewing in a community psychiatric survey. *Am J Public Health* 1993;83:896–8. [PubMed: 8498632]
- First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JB. Structured clinical interview for Axis I DSM-IV disorders – Patient Edition (SCID-I/P, Version 2.0). New York: Biometrics Research Department, New York State Psychiatric Institute; 1994.
- Garber J, Hollon SD. What can specificity designs say about causality in psychopathology research? *Psychol Bull* 1991;110:129–36. [PubMed: 1891514]
- Grant BF, Hasin DS, Blanco C, Stinson FS, Chou SP, Goldstein RB, et al. The epidemiology of social anxiety disorder in the United States: results from the national epidemiologic survey on alcohol and related conditions. *J Clin Psychiat* 2005;66:1351–61.
- Ham LS, Hope DA, White CS, Rivers PC. Alcohol expectancies and drinking behavior in adults with social anxiety disorder and dysthymia. *Cognitive Ther Res* 2002;26:275–88.
- Ham LS, Bonin M, Hope DA. The role of drinking motives in social anxiety and alcohol use. *J Anxiety Disord* 2007;21:991–1003. [PubMed: 17275253]

- Kessler RC, Crum RM, Warner LA, Nelson CB, Schulenberg J, Anthony JC. Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Arch Gen Psychiat* 1997;54:313–21. [PubMed: 9107147]
- Mattick RP, Clarke JC. Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behav Res Ther* 1998;36:455–70. [PubMed: 9670605]
- Morris EP, Stewart SH, Ham LS. The relationship between social anxiety disorder and alcohol use disorders: a critical review. *Clin Psychol Rev* 2005;25:734–60. [PubMed: 16042994]
- Randall CL, Thomas S, Thevos AK. Concurrent alcoholism and social anxiety disorder: a first step toward developing effective treatments. *Alcohol: Clin Exp Res* 2001;25:210–20. [PubMed: 11236835]
- Reiss S, Peterson RA, Gursky DM, McNally RJ. Anxiety sensitivity, anxiety frequency and the predictions of fearfulness. *Behav Res Ther* 1986;24:1–8. [PubMed: 3947307]
- Safren SA, Turk CL, Heimberg RG. Factor structure of the Social Interaction Anxiety Scale and the Social Phobia Scale. *Behav Res Ther* 1998;36:443–53. [PubMed: 9670604]
- Schmidt NB, Joiner TE. Structure of the Anxiety Sensitivity Index psychometrics and factor structure in a community sample. *J Anxiety Disord* 2002;16:33–49. [PubMed: 12171212]
- Schmidt NB, Trakowski JH, Staab JP. Extinction of panicogenic effects of a 35% CO-sub-2 challenge in patients with panic disorder. *J Abnorm Psychol* 1997;106:630–8. [PubMed: 9358693]
- Schmidt NB, Eggleston AM, Trakowski JH, Smith JD. Does coping predict CO-sub-2-induced panic in patients with panic disorder? *Behav Res Ther* 2005;43:1311–9. [PubMed: 16086982]
- Schmidt NB, Zvolensky MJ, Maner JK. Anxiety sensitivity: prospective prediction of panic attacks and Axis I pathology. *J Psychiat Res* 2006;40:691–9. [PubMed: 16956622]
- Schmidt NB, Buckner JD, Keough ME. Anxiety sensitivity as a prospective predictor of alcohol use disorders. *Behav Modific* 2007a;31:202–19.
- Schmidt NB, Eggleston AM, Woolaway-Bickel K, Fitzpatrick KK, Vasey MW, Richey JA. Anxiety Sensitivity Amelioration Training (ASAT): a longitudinal primary prevention program targeting cognitive vulnerability. *J Anxiety Disord* 2007b;21:302–19. [PubMed: 16889931]
- Schneier FR, Martin LY, Liebowitz MR, Gorman JM, Fyer AJ. Alcohol abuse in social phobia. *J Anxiety Disord* 1989;3:15–23.
- Sobin C, Weissman MM, Goldstein RB, Adams P, Wickramaratne P, Warner V, et al. Diagnostic interviewing for family studies: comparing telephone and face-to-face methods for the diagnosis of lifetime psychiatric disorders. *Psychiatr Genet* 1993;3:227–33.
- Spielberger, CD.; Gorsuch, RL.; Lushene, RE.; Vagg, PR.; Jacobs, GA. Manual for the state-trait anxiety inventory (form Y). Palo Alto, CA: Mind Garden; 1983.
- Terra MB, Barros HMT, Stein AT, Figueira I, Jorge MR, Palermo LH, et al. Social anxiety disorder in 300 patients hospitalized for alcoholism in Brazil: high prevalence and undertreatment. *Compr Psychiat* 2006;47:463–7. [PubMed: 17067869]
- Thomas SE, Thevos AK, Randall CL. Alcoholics with and without social phobia: a comparison of substance use and psychiatric variables. *J Stud Alcohol* 1999;60:472–9. [PubMed: 10463803]
- Zimmermann P, Wittchen H-U, Höfler M, Pfister H, Kessler RC, Lieb R. Primary anxiety disorders and the development of subsequent alcohol use disorders: a 4-year community study of adolescents and young adults. *Psychol Med* 2003;33:1211–22. [PubMed: 14580076]

Table 1

Means (SD) or percentages of baseline demographic and psychiatric variables for entire sample and by follow-up AUD status

Dependent variable	Entire sample <i>N</i> = 405	AUD3- <i>n</i> = 284	AUD+ <i>n</i> = 11
Age	19.34 (3.85) Range = 12.2–51.0	19.31 (4.20) Range = 12.2–51.0	19.20 (1.69) Range = 16.8–23.2
Gender	61.0% female	63.3% female	27.3% female
SIAS-SPS total	32.89 (23.00) Range = 0–110	33.53 (23.37) Range = 0–110	61.45 (28.33) Range = 17–110
SIAS-SPS: interaction fears	17.40 (11.53) Range = 0–54	17.63 (11.82) Range = 0–53	27.82 (12.04) Range = 12–50
SIAS-SPS: observation fears	5.62 (5.72) Range = 0–31	5.77 (5.87) Range = 0–31	13.45 (7.99) Range = 0–24
SIAS-SPS: fear others will notice	10.12 (7.94) Range = 0–36	10.36 (8.01) Range = 0–36	20.18 (10.41) Range = 1–36
anxiety	5.47 (5.19) Range = 0–24	5.75 (5.20) Range = 0–24	8.41 (6.69) Range = 0–18
Depression	44.61 (4.46) Range = 33–59	44.60 (4.55) Range = 33–59	45.27 (4.25) Range = 39–54

Note. AUD- = no AUD onset at follow-up assessment. AUD+ = AUD onset at follow-up assessment. SIAS-SPS = The *Social Interaction Anxiety Scale* and the *Social Phobia Scale* (Mattick and Clarke, 1998). Depression was measured using the *Beck Depression Inventory* (Beck and Steer, 1987) and trait anxiety with the *Spielberger Trait Anxiety Inventory* (Spielberger et al., 1983). Gender was coded male = 1, female = 2.

Table 2

Correlations between types of social anxiety, demographic variables, depression, and trait anxiety

Baseline variable	1	2	3	4	5	6	7	8
1. SIAS-SPS total	—							
2. SIAS-SPS: interactions fears	.89***	—						
3. SIAS-SPS: observation fears	.88***	.64***	—					
4. SIAS-SPS: fear others will notice anxiety	.95***	.77***	.83***	—				
5. Depression	.56***	.52***	.50***	.52***	—			
6. Trait anxiety	.36***	.42***	.24**	.33***	.34***	—		
7. Gender	.05	.03	.04	.06	.16	.04	—	
8. Age	.05	.01	.10	.05	-.11	-.07	-.09	—

Note. Partial correlations controlling for condition. SIAS-SPS = The Social Interaction Anxiety Scale and the Social Phobia Scale (Mattick and Clarke, 1998). Depression was measured using the Beck Depression Inventory (Beck and Steer, 1987) and trait anxiety with the Spielberger Trait Anxiety Inventory (Spielberger et al., 1983).

* $p < .05$,
 ** $p < .01$,
 *** $p < .001$.

Table 3

Hierarchical logistic regression models predicting AUD onset

Predictor	OR	95.0% CI		<i>p</i>
		Lower	Upper	
<i>SIAS-SPS total</i>				
Step 1				
Experimental group	0.50	0.11	2.15	ns
Recruitment group	1.00	1.00	1.00	ns
Depression	0.97	0.84	1.11	ns
Trait anxiety	0.98	0.84	1.14	ns
Gender	0.21	0.05	0.88	<.05
Step 2				
SIAS-SPS total	1.05	1.01	1.08	<.01
<i>Observation fears</i>				
Step 1				
Experimental group	0.44	0.10	1.90	ns
Recruitment group	1.00	1.00	1.00	ns
Depression	1.00	0.88	1.13	ns
Trait anxiety	0.99	0.85	1.16	ns
Gender	0.19	0.05	0.80	<.05
Step 2				
Observation fears	1.16	1.05	1.27	<.01
<i>Fear others will notice anxiety</i>				
Step 1				
Experimental group	0.50	0.12	2.13	ns
Recruitment group	1.00	1.00	1.00	ns
Depression	0.98	0.86	1.12	ns
Trait anxiety	0.99	0.85	1.15	ns
Gender	0.18	0.04	0.76	<.05
Step 2				
Fear others will notice anxiety	1.13	1.04	1.23	<.01

Note. SIAS-SPS = The Social Interaction Anxiety Scale and the Social Phobia Scale (Mattick and Clarke, 1998). Depression was measured using the Beck Depression Inventory (Beck and Steer, 1987) and trait anxiety with the Spielberger Trait Anxiety Inventory (Spielberger et al., 1983).