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ORIGINAL PAPER

Longitudinal association between social anxiety disorder and incident alcohol use disorder: results from two national samples of US adults

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Abstract

This study assessed the association between subclinical social fears and a 12-month diagnosis of Social Anxiety Disorder (SAD) at baseline and the risk of incident Alcohol Use Disorder (AUD) at follow-up, compared to those without subclinical social fears and a 12-month diagnosis of SAD. We performed an individual participant meta-analysis based on data from two national longitudinal surveys. Wave 1 of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) was conducted in 2001–2002 in a sample of 43,093 adults and Wave 2 was conducted in 2004–2005 in 34,653 of the original respondents. Wave 1 of the National Comorbidity Survey was conducted in 1990–1992 in a sample of 8098 respondents and Wave 2 was conducted in 2001–2002 in 5001 of the original respondents. Binary logistic regression analyses were performed independently in each study and then the effect estimates were combined using random-effects meta-analysis. Neither subclinical social fears nor 12-month SAD at baseline were associated with incident AUD at follow-up. These findings conflict with reports of previous studies that a diagnosis of SAD is a risk factor for AUD in adults, and suggest that subclinical social fears are not associated with differential risk of incident AUD.

Keywords Social phobia · Alcoholism · Incidence · Prospective cohort study · Epidemiology

Introduction

In 1985, the self-medication hypothesis (SMH) was proposed to explain clinical observations of the co-occurrence between emotional and substance use disorders [1]. The SMH proposes that individuals with emotional disorders are susceptible to developing substance use disorders because the use of such substances leads to a reduction in negative affective states. The SMH further proposes that specific types of substances should correspond with specific patterns of negative affect. For example, the results of population-based studies link Social Anxiety Disorder (SAD) and Alcohol Use Disorder (AUD) on the basis of cross-sectional data [2–4], and longitudinal studies suggest that suffering from SAD is associated with increased risk of developing AUD [5–8].

A number of recent theoretical models have built on the SMH in an attempt to explain this relationship. These models regard alcohol's role in the self-medication of social anxiety as extending beyond the reduction of negative affect, and incorporate other cognitive and behavioural symptoms. For example, it has been proposed that, in addition to reducing negative affect, alcohol also plays a role in reducing attentional biases to threat-related stimuli, diminishing fear of scrutiny, and facilitating social approach behaviours [9–12]. These findings have been largely borne out in laboratory studies, where anxiety induction and the administration of alcohol occur in a controlled way. Although socially anxious individuals tend to believe that alcohol will reduce distress, its anxiolytic effects may depend on the dose and timing of administration, as well as the nature of the social situation [13].

Upon closer examination of the relevant epidemiological data underlying these models, three observations raise questions about the longitudinal association between SAD and AUD. First, previous findings indicate that SAD is associated with increased risk of alcohol dependence, but

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not abuse, based on DSM-IV criteria [6]. The relationship between SAD and risk of AUD incidence has not been directly addressed in adult samples. Second, other findings indicate that lifetime sub-threshold social fears at baseline, not SAD, put people at an increased risk of developing heavy drinking at follow-up, relative to asymptomatic controls [14]. However, this study had only 33 participants with SAD and 84 with subclinical social fears at baseline, so this issue should be addressed using larger samples. Third, the heterogeneity between the specific fears that could contribute to a diagnosis of SAD (e.g., fear of talking in front of others versus a fear of writing while someone watches) raises questions about whether the risk of incident AUD varies depending on the nature of the fear. For example, individuals with social anxiety who report having social interaction fears or unsociability may be likelier to frequently engage in alcohol use for coping reasons (e.g., to facilitate approach behaviours in social interaction settings) than are those who endorse other types of fears (e.g., performance situations) [11, 15–17].

Against this background, the present study used data from two national samples of the US population to explore the relationship between specific social fears at baseline and the frequency of incident AUD at follow-up, and to test the hypotheses that (1) a past-year diagnosis of SAD at baseline is associated with increased risk of incident AUD at follow-up relative to those without a diagnosis of past-year SAD, and (2) subclinical social fears are associated with increased risk of incident AUD at follow-up relative to those without subclinical social fears.

Methods

Sample

Data were drawn from two national longitudinal surveys: the National Comorbidity Survey (NCS) and the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) [18–20]. Wave 1 of the NESARC was conducted in a national sample of 43,093 civilian, non-institutionalized adults (18–98 years old) from all 50 US states and the District of Columbia in 2001–2002. Young adults, African-Americans and Hispanics were purposely oversampled. Face-to-face interviews were conducted by trained lay interviewers using the Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV version (AUDADIS-IV). In 2004–2005, Wave 2 of the NESARC was conducted in 34,653 of the original respondents. The overall response rates for Waves 1 and 2 were 81 and 87%, respectively, yielding a cumulative response rate of 70%.

Wave 1 of the NCS was undertaken in a national sample of 8098 civilian, non-institutionalized adults (aged 15–54 years old) who were sampled from the US in 1990–1992 [21]. Ten years later, 5001 participants from the initial sample were followed up at Wave 2. Face-to-face interviews were conducted by trained lay interviewers using the Composite International Diagnostic Interview (CIDI). Wave 1 of the NCS was based on DSM-III-R criteria, and Wave 2 was based on DSM-IV criteria. The overall response rates for Wave 1 and 2 were 82 and 88%, respectively, yielding a cumulative response rate of 72%.

Measures

In the NESARC, lifetime social fears and 12-month SAD, and AUD diagnoses were obtained using the AUDADIS-IV, which has good reliability (ICC: 0.73) for assessing lifetime social fears, fair reliability (kappa: 0.44) for assessing SAD, and good reliability (kappa: 0.74) for assessing AUD [22]. In the NCS, diagnoses were obtained using the CIDI, which has good reliability for assessing SAD (kappa: 0.64) and AUD (kappa: 0.78) [23]. Consistent with Crum and Pratt's [14] study, respondents who endorsed at least one of the core symptoms of social anxiety on the AUDADIS-IV or CIDI, but did not meet lifetime SAD criteria, were conceptualized as having subclinical social fears. Sociodemographic variables in both surveys included age, sex, education (did not complete high school; completed high school or its equivalent; some college; or a Bachelor's degree or higher), and marital status (married or cohabiting; widowed, divorced or separated; never married). In both surveys, psychiatric control variables included a lifetime diagnosis of mood (Major Depression and Dysthymia) and anxiety disorders (Panic Disorder, Generalized Anxiety Disorder, and Specific Phobias) at Wave 1. We control for these variables given that previous population-based studies have indicated that they are associated with both SAD and AUD [24–26]. Respondents who met diagnostic criteria for DSM-IV alcohol abuse, dependence, or both in the years intervening the Wave 1 and 2 interviews, but who had not met diagnostic criteria for these in the lifetime prior to the Wave 1 interview, constituted those with incident AUD. Accordingly, the incidence of AUD captures the rate at which new cases occur in a given period, or the force of morbidity of AUD.

Statistical analyses

Raw frequencies and weighted proportions were calculated to estimate the prevalence of lifetime social fears with and without a 12-month diagnosis of SAD at Wave 1. We also calculated the frequency of incident AUD at Wave 2 among those who reported having lifetime social fears with and without a 12-month SAD diagnosis at Wave 1. To test the hypotheses of the study, binary logistic regression analyses were first conducted to estimate the odds ratios (ORs)

between lifetime subclinical social fears and 12-month SAD at Wave 1 as predictors of AUD incidence at Wave 2, relative to those without subclinical social fears or 12-month SAD, respectively. The supplementary file reports the crude and adjusted (for sociodemographic and psychiatric variables) estimates for these analyses. NESARC and NCS probability weights were used to adjust for oversampling, attrition, and non-response. Next, using two random-effects meta-analysis models [27], we pooled the two adjusted ORs from the NESARC and NCS corresponding to each variable of interest: (1) 12-month SAD, and (2) subclinical social fears, as predictors of incident AUD, relative to those without 12-month SAD and subclinical social fears, respectively.

Results

Sample characteristics

Table 1 presents the sociodemographic and psychiatric characteristics of the NESARC and NCS samples. The NESARC sample consisted of 34,653 respondents, and the NCS consisted of 5001 respondents. The NESARC sample was 10 years older, and had a lower prevalence of social anxiety (one-third the prevalence of subclinical social fears, and one-half the prevalence of 12-month SAD). Both samples had similar rates of incident AUD, and sex, education, and marital status were similarly distributed.

Twenty-three percent of the NESARC respondents and 20% of the NCS respondents with one or more lifetime social fears met diagnostic criteria for SAD in the year prior to the Wave 1 interview, corresponding to weighted population prevalence estimates of 3% (SE 0.1) and 6% (SE 0.6), respectively.

Subclinical social fears and 12-month SAD as baseline predictors of incident AUD at follow-up

We tested the hypotheses that subclinical social fears and a diagnosis of 12-month SAD at baseline were associated with incident AUD at follow-up, relative to those without subclinical social fears and a diagnosis of 12-month SAD, respectively. The overall adjusted OR for incident AUD among people with 12-month SAD was 0.74 (95% CI 0.44–1.04), and the overall OR for incident AUD among people with subclinical social fears was 0.81 (95% CI 0.57–1.05). There was no evidence of an association between social anxiety at baseline and incident AUD at follow-up.

Table 1 Sociodemographic and psychiatric characteristics of NESARC and NCS samples

Raw frequencies (and weighted proportions)		
	NESARC ($n=34,653$)	NCS ($n=5001$)
Age (mean ± SD)	45 ± 17	35 ± 10
Sex		
Female	20,089 (52%)	2656 (52%)
Education		
Bachelor's degree or higher	8480 (25%)	1158 (23%)
Some college	10,474 (31%)	1268 (25%)
Completed high school or equivalent	9955 (29%)	1709 (34%)
Did not complete high school	5744 (15%)	866 (18%)
Marital status		
Married or cohabiting	18,413 (63%)	2896 (58%)
Dissolved	8564 (17%)	778 (16%)
Never married	7676 (20%)	1327 (26%)
Lifetime mood disorders		
Present	7289 (20%)	769 (16%)
Lifetime anxiety disorders		
Present	3620 (10%)	602 (12%)
Subclinical social fears		
Present	2546 (7%)	1158 (23%)
12-month SAD		
Present	1004 (3%)	315 (6%)
Incident AUD		
Present	1366 (4%)	227 (4%)

Specific lifetime social fears with and without 12-month SAD at baseline and AUD incidence at follow-up

Finally, we explored the relationship between specific social fears at baseline and the frequency of incident AUD at follow-up (see Table 2). Three percent of the NESARC respondents with lifetime social fears with or without a 12-month diagnosis of SAD at Wave 1 had developed AUD by Wave 2. Five-to-six percent of the NCS respondents with lifetime social fears (with or without SAD) at Wave 1 had developed AUD by Wave 2. In both surveys, there were no apparent differences (based on the overlap of the percentages and standard errors) (1) in the proportion of participants with lifetime social fears and 12-month SAD who subsequently developed AUD, and (2) between specific fears at baseline and frequency of incident AUD at follow-up.

Sensitivity analyses

First, we addressed whether a higher number of social fears reported by respondents is associated with increased risk of incident AUD. The adjusted OR for the association between the number of social fears at baseline and incident AUD at follow-up was 0.97 (0.93–1.01). Thus, there was no evidence of an association. Second, we assessed whether there is an interaction between age, subclinical social fears, and 12-month SAD at baseline and incident AUD at follow-up. The interaction of age and subclinical SAD at baseline indicates that there is no association with incident AUD at follow-up (OR 1.00; 95% CI 0.98–1.01). The interaction of age and 12-month SAD at baseline suggests there is a very weak association with incident AUD at follow-up (OR 0.99; 95% CI 0.97–1.00), such that the risk of incident AUD at follow-up is higher among younger individuals without a baseline diagnosis of 12-month SAD relative to those with a baseline diagnosis of 12-month SAD. Third, given that a previous study using the NCS sample reported an increased risk of dependence, but not abuse, among individuals with 12-month SAD [6], we reassessed this association in terms of the incidence of AUD using pooled longitudinal data form the NESARC and NCS. The overall OR for the association between 12-month SAD at baseline and incident alcohol abuse at follow-up is 0.63 (95% CI 0.29–0.97) and the OR for the association between 12-month SAD at baseline and incident alcohol dependence at follow-up is 0.88 (0.31–1.46). As such, there was no evidence of an association between

Table 2 Lifetime prevalence of social fears with and without 12-month SAD at Wave 1 as predictors of AUD incidence at Wave 2

Fear or avoidance of…	NESARC				NCS			
	AUD incidence given fear[a]		AUD incidence given SAD with fear[b]		AUD incidence given fear[a]		AUD incidence given SAD with fear[b]	
	%	SE	%	SE	%	SE	%	SE
Talking in front of others	3	0.3	3	0.6	6	1.6	6	1.9
Eating or drinking in public	3	0.5	3	0.9	12	5.9	5	2.5
Writing while someone else watches	3	0.5	3	0.9	6	1.6	6	2.8
Conversations with those you do not know well	3	0.1	3	1.4	–	–	–	–
Going to parties/other social gatherings	3	0.7	4	1.5	–	–	–	–
Dating	3	0.7	4	1.3	–	–	–	–
Being in small group situation	4	1.1	5	1.6	–	–	–	–
Taking part or speaking in class	3	0.4	3	0.6	–	–	–	–
Being interviewed	4	0.6	3	0.9	–	–	–	–
Taking part or speaking in a meeting	3	0.4	3	0.8	–	–	–	–
Performing in front of others	3	0.4	3	0.7	–	–	–	–
Taking an important exam	3	0.4	3	0.9	–	–	–	–
Speaking to authority figure	3	0.5	3	0.9	–	–	–	–
Public speaking	–	–	–	–	5	0.7	6	1.5
Public toilets	–	–	–	–	5	1.8	5	3.2
Speaking in small groups	–	–	–	–	4	1.1	4	1.2
Any fear	3	0.3	3	0.6	6	0.8	5	1.3

[a]AUD incidence at Wave 2 conditional on having specific fear (with or without SAD) at Wave 1

[b]AUD incidence at Wave 2 conditional on having 12-month SAD and specific fear at Wave 1

12-month SAD at baseline and incident alcohol dependence at follow-up.

Discussion

The principal findings of this study are as follows. First, 12-month SAD at baseline was not associated with higher risk of incident AUD at follow-up, relative to those without a diagnosis of 12-month SAD at baseline. Second, the presence of subclinical social fears at baseline was not associated with increased risk of incident AUD at follow-up, relative to those in whom subclinical social fears were absent. Third, neither the nature nor the frequency of social fears at baseline are associated with risk of developing AUD. In sum, neither lifetime social fears nor a diagnosis of 12-month SAD were found to predict AUD incidence.

We first compare these findings to those of a previous study indicating that SAD at baseline is associated with increased risk of DSM-IV alcohol dependence, but not abuse, at follow-up [5]. When we explored this relationship, we found that SAD at baseline is not associated with alcohol dependence, and is negatively associated with alcohol abuse at follow-up. Two differences between the methodology of Buckner et al. [6] and those of the present study are as follows: whereas Buckner et al. focused on lifetime diagnoses of SAD and AUD, we focused on 12-month SAD at baseline and incident AUD at follow-up. The reason we focused on 12-month (rather than lifetime) SAD at baseline is to reduce reliance on recall of prior SAD and to minimize the number of intervening events that could explain subsequent AUD. The incidence of AUD explains the force of morbidity of AUD over the follow-up period and excludes respondents who have previously met criteria for AUD (as opposed to lifetime estimates that focus on the number of respondents who report ever having AUD). The combination of 12-month SAD at baseline and incident AUD at follow-up in this study emphasizes the association of SAD near the time of the baseline interview with the subsequent onset of AUD. Thus, we think the present study provides a more appropriately controlled analysis than the previous study.

Second, the lack of association between lifetime subclinical social fears and incident AUD conflicts with those of a previous study: Crum and Pratt [14] reported that subthreshold social anxiety, not SAD, was associated with increased risk of AUD incidence. We believe this discrepancy can be explained largely by the difference in sample sizes. Specifically, the overall estimate of the present study, based on the combination of two larger samples, is more informative than the estimates based on 33 respondents who met SAD criteria and 86 who had subclinical social fears in Crum and Pratt's study.

Third, although the findings of the present study suggest that SAD is not associated with increased subsequent risk of AUD in a large sample of adults, we caution against generalizing these findings across the developmental spectrum. There are four reasons to address this research question more closely in adolescent samples: first, the prevalence of SAD is much higher among adolescents and young adults, and the first incidence of SAD is also estimated to occur most frequently at this time [28, 29]. Second, about half of all incident cases of substance use disorders are estimated to occur by early adulthood [25]. Third, several studies suggest that adolescents and young adults with SAD are at increased risk of developing AUD [12, 30–34], although this association has been found to vary by sex, personality, the occurrence of preceding stressful life events, and the presence of comorbid mental disorders [7, 35–38]. Finally, mechanistic accounts propose that socially anxious individuals engage in alcohol use for coping reasons, such as reducing distress and facilitating approach behaviours, that may be particularly pronounced at younger ages [10, 11]. Therefore, age may differentially influence the relationship between social anxiety and alcohol use such that the risk of incident AUD among those with social anxiety may increase into late adolescence and early adulthood, and decrease thereafter. Nonetheless, although the results of the present study indicate that younger adults have a higher risk of incident AUD, this was qualified by an interaction between age and SAD. Specifically, younger adults without SAD have a slight increase in risk of incident AUD at follow-up relative to younger adults without SAD, and there is no difference between older adults with or without SAD.

The present study is based on two nationally representative surveys that employ prospective longitudinal study designs and use standardized diagnostic instruments to ascertain SAD and AUD. A strength of the study is the combination of two estimates that are more informative than either one alone. That said, several limitations of the present study must be noted. First, the kappa for 12-month SAD in the AUDADIS-IV exhibits fair reliability [22], suggesting that classification error is not trivial. Second, due to the nature of the collection of self-report data, measurement may have been influenced by respondent biases or recall errors that led to inaccuracies in the measurement of lifetime social fears or alcohol consumption. Third, and relatedly, social fears occurring in the year prior to the interview (rather than in the lifetime) would have been a more suitable basis for the subclinical group, however, this was not possible due to design features of the NESARC and NCS that focused on lifetime occurrences. Nonetheless, this methodological characteristic is consistent with that of a previous study inferring that subclinical social fears are associated with increased risk of problem drinking [14]. Finally, there are several differences between the surveys, including that

they were initiated 10 years apart and used different diagnostic interviews. We cannot rule out that such differences contributed to discrepancies in baseline prevalence estimates of lifetime social fears and 12-month SAD between the two samples; nonetheless, the results pertaining to the hypotheses in both surveys were consistent.

Conclusions

The aim of this study was to explain the relationship between baseline social anxiety and incident AUD using two nationally representative samples of US adults. Neither lifetime social fears nor a 12-month diagnosis of SAD at baseline were associated with an increased risk of AUD incidence over 3- and 10-year follow-up periods. The nature of the social fear at baseline did not provide substantial information about the risk of AUD incidence. These findings raise questions about the solidity of the longitudinal association between social anxiety and alcohol use in adults.

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Compliance with ethical standards

Conflict of interest The authors declare no competing financial interest.

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