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The Links Between Social Anxiety Disorder, Insomnia Symptoms, and Alcohol Use Disorders: Findings From a Large Sample of Adolescents in the United States

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Social anxiety disorder (SAD) is associated with increased risk of developing an alcohol use disorder (AUD). Most of the current literature has focused on the role of acute stress responding in this relation; however, both SAD and AUDs also are linked to insomnia symptoms (i.e., difficulty falling or staying asleep). As adolescence is a sensitive period for the onset of these disorders, the present study examined if insomnia symptoms might partially account for the SAD-AUD link in a large sample of adolescents. Data from the National Comorbidity Survey–Adolescent Supplement were examined. Participants (N = 10,140) completed interviews to assess past 12-month SAD and AUD diagnostic status as well as insomnia symptoms. Analyses tested whether insomnia symptoms accounted for a significant proportion of the SAD-AUD relation. Results indicated that insomnia symptoms were positively related to both SAD and AUD status, and the relation between SAD and AUD status was significantly reduced when insomnia symptoms were included in the model. Findings remained significant after controlling for the effects of age, gender, posttraumatic stress disorder, major depressive disorder, and other drug dependence status. Experimental examination and intensive longitudinal assessment of these relationships are needed before strong conclusions can be inferred about causality and temporal relationships. The current findings do indicate insomnia may be an important indirect and stigma-free treatment target to address in prevention and treatment efforts for SAD, AUDs, and their co-occurrence.

Keywords: insomnia; social anxiety; alcohol; adolescents; youth

RESEARCH CONDUCTED WITH ADULT (e.g., Schneier et al., 2010) and adolescent (e.g., Blumenthal, Leen-Feldner, Badour, & Babson, 2011) samples consistently demonstrate a positive relation between social anxiety and alcohol use problems. Extant work indicates a high degree of co-occurrence between social anxiety disorder (SAD) and alcohol use disorders (AUDs) among adolescents (e.g., Conway, Swendsen, Husky, He, & Merikangas, 2016), despite documented "protection" from earlier initiation or regular use (Tomlinson & Brown, 2012). For example, in a large sample of adolescent twin girls, Nelson and colleagues (2000) found that SAD was associated with an approximately two-fold increase in the risk of a concurrent alcohol dependence diagnosis. Both retrospective and prospective work identifies SAD as the primary, or antecedent, disorder in the majority of cases (e.g., Zimmermann

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et al., 2003). For instance, Buckner, Schmidt, and colleagues (2008) found that adolescents diagnosed with SAD were significantly more likely to develop an AUD by age 30 years as compared to those without the diagnosis. Although compelling, the burgeoning literature examining adolescents has largely addressed the frequency of SAD-AUD co-occurrence, and work examining theoretically derived mechanisms (primarily conducted with adult samples) remains mixed.

Existing work examining this co-occurrence has largely employed Cooper's (1994) model of drinking motives, indicating that socially anxious younger and older adults typically report elevated coping motives (e.g., reducing/avoiding negative affect), and/or conformity motives (e.g., avoiding rejection) for use (e.g., Ham, Zamboanga, Bacon, & Garcia, 2009; Terlecki & Buckner, 2015; Windle & Windle, 2012). In the only studies to date to directly examine these links among adolescents, severity of social anxiety was positively related to coping motives for alcohol consumption, but not conformity, enhancement, or social motives (Blumenthal, Leen-Feldner, Frala, Badour, & Ham, 2010), and coping motives partially accounted for the association between social anxiety symptoms and current desire to drink following introduction to an interactive laboratory study (Blumenthal, Ham, Cloutier, Bacon, & Douglas, 2016). Importantly, avoidance-related alcohol use is associated with later drinking-related problems (Carpenter & Hasin, 1999; Kuntsche, Knibbe, Gmel, & Engels, 2005). In conjunction with adult work highlighting the role of managing social stress specifically in this relation (Cludius, Stevens, Bantin, Gerlach, & Hermann, 2013; Thomas, Randall, & Carrigan, 2003), the majority of clinical and empirical efforts have thus focused on acute responding to social situations and coping motives generally (e.g., Stapinski et al., 2015). Although intervention efforts targeting coping motives among anxious youth are promising (Conrod, Castellanos-Ryan, & Mackie, 2011; Conrod et al., 2013), extant work remains mixed (Anker, Kushner, Thuras, Menk, & Unruh, 2016; Lammers et al., 2015), and research indicates that broad-based negative reinforcement motives account for only a moderate amount of variance in the SAD-AUD link. Accordingly, identifying additional, specific cognitive and behavioral variables related to this link will be important in designing comprehensive intervention efforts.

A convergence of work suggests that sleep difficulties, and insomnia specifically (i.e., recurrent difficulty falling or staying asleep, or experiencing nonrestorative sleep, despite adequate opportunity), may be an important factor to consider in an effort to better understand, and ultimately treat, the SAD-AUD relation. First, research consistently indicates a positive correlation between anxiety (e.g., overall level; presence of any anxiety disorder) and sleep problems including insomnia (Johnson, Roth, & Breslau, 2006; Taylor et al., 2005; Zhang et al., 2017). For example, Hudson and colleagues (2009) found that children (ages 7–12 years) meeting criteria for an anxiety disorder also reported significantly later bedtimes, and less weekday sleep, in an online daily-diary study as compared to youth without an anxiety diagnosis. Further, in a sample of over 1,000 community-recruited adolescents (ages 13–16 years), Johnson and colleagues (2006) reported that the diagnosis of an anxiety disorder was positively related to the concurrent diagnosis of insomnia, and retrospective age of onset data indicated that anxiety was the antecedent disorder in the majority (73%) of cases. Proportional hazards models provided further support for the contention that the presence of an anxiety disorder significantly predicted subsequent onset of insomnia, although insomnia did not significantly predict the onset of a later anxiety disorder in this sample. Finally, in a sample of youth ages 7–17 years receiving cognitive behavioral treatment for anxiety, Peterman and colleagues (2016) found a significant reduction in posttreatment insomnia-relevant sleep problems (e.g., presleep arousal, parent-reported bedtime resistance), despite the fact that none of the treatment approaches explicitly addressed sleep. In terms of SAD *specifically* there remains a paucity of research; however, initial evidence suggests a positive relation consistent with work examining anxiety more generally (Buckner, Schmidt, et al., 2008; Stein, Kroft, & Walker, 1993). For instance, Buckner, Bernert, and colleagues (2008) found that social anxiety symptoms were positively correlated with those of insomnia in a sample of undergraduate students, and that participants reporting clinically significant social anxiety also reported meeting the threshold for a probable insomnia diagnosis at a much higher rate (18.2%) than an age and gender matched comparison group (4.8%).

Importantly, insomnia also is linked to the incidence and maintenance of alcohol use problems (Crum, Storr, Chan, & Ford, 2004; Roane & Taylor, 2008; Taylor, Lichstein, & Durrence, 2003; Zhang et al., 2017) as well as earlier relapse during recovery attempts (Brower, Aldrich, Robinson, Zucker, & Greden, 2001). Not only are individuals with insomnia likely to report alcohol use for sleep purposes (Brower et al., 2001), childhood sleep difficulties have been prospectively linked with problematic alcohol use (e.g., early, regular use; Wong, Brower, Fitzgerald, & Zucker, 2004). For instance, childhood overtiredness is associated with several markers of risk-related alcohol use in young adulthood, including

blackouts and binge drinking (Wong et al., 2004). These findings suggest that individuals who are overtired may eventually self-medicate with alcohol to cope with their discomfort or aid in attempted sleep initiation; however, work directly examining social anxiety, insomnia, and alcohol use problems is limited, and none has sought to directly link these related phenomenon.

Adolescence may be a period of particular interest to researchers and clinicians alike given normative changes in circadian rhythm that could translate into sleep disruptions (Crowley, Acebo, & Carskadon, 2007; Hagenauer, Perryman, Lee, & Carskadon, 2009). The neurological and somatic changes that characterize adolescence also implicate this as a developmentally sensitive period in terms of anxiety, sleep, and substance use problems (e.g., Casey & Jones, 2010; Colrain & Baker, 2011; Eiland & Romeo, 2013). Adolescence is characterized by (a) normative increases in social anxiety and experimentation with alcohol (Essau, Conradt, & Petermann, 1999; Wittchen et al., 2008), (b) the onset of dysfunctional social anxiety and problematic alcohol use (Labouvie & White, 2002; Velting & Albano, 2001), (c) normative changes in the sleep cycle priming the learning of nonnormative physiological and behavioral presleep behaviors (Crowley et al., 2007; Noland, Price, Dake, & Telljohann, 2009), and (d) a more sophisticated understanding and application of voluntary coping responses (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001), including the effects of alcohol (e.g., Jester et al., 2015; Schell, Martino, Ellickson, Collins, & McCaffrey, 2005). This suite of characteristics highlights adolescence as an important developmental epoch to study the onset of, and linkages among, problematic social anxiety, sleep, and alcohol use.

The current study was a preliminary, cross-sectional test of the relations among adolescent SAD, insomnia symptoms, and AUDs (cf. Kraemer, Yesavage, Taylor, & Kupfer, 2000). A large sample of adolescents in the United States were examined to test whether insomnia symptoms account for a significant proportion of the relation between SAD and AUD diagnostic status. The model was then further tested with pertinent, available risk markers and factors including: age (Merikangas et al., 2010), gender (Swendsen et al., 2012), posttraumatic stress disorder (PTSD; Kilpatrick et al., 2003), major depressive disorder (MDD: Kessler et al., 2003), and other drug dependence (Compton, Thomas, Stinson, & Grant, 2007) added as covariates in the final models.

Materials and Methods

The present study examined data collected in the National Comorbidity Survey–Adolescent Supplement (NCS-A). The NCS-A was designed to assess the prevalence and correlates of *DSM-IV* (American Psychiatric Association, 1994) diagnoses among United States residents age 13–18 years (Kessler et al., 2009a; Kessler et al., 2009b). Methods, weighting, and sampling procedures have been comprehensively described elsewhere (Kessler et al., 2009a; Kessler et al., 2009b; Merikangas, Avenevoli, Costello, Koretz, & Kessler, 2009).

Briefly, the survey included a dual-frame design, recruiting adolescents from households participating in the National Comorbidity Survey–Replication (NCS-R; n = 904) and a representative sample of schools in the same communities as the NCS-R households (n = 9,244). The combined response rate was 75.6% (approximately 13,400 approached; Kessler et al., 2009b). Weighting accounts for within-household probability of selection as well as residual discrepancies between sample and population sociodemographic and geographic distributions. Data were collected between February 1, 2001 and January 30, 2004, and all interviews were conducted in English. Recruitment and consent procedures were approved by the human subject committees of Harvard Medical School and the University of Michigan. Parents furnished written informed consent (provided in English), and adolescents gave written assent prior to the survey. Interested parents also were invited to complete a brief survey (63% participated); however, all data presented in the current study are from the adolescent interview. Face-to-face interviews were conducted by trained interviewers using computer-assisted personal interviews in the adolescents' homes. Interviewers asked questions, probed vague responses, and entered data into the laptop; the computer program guided interviewers through the question series. To reduce participant burden, all participants were asked diagnostic stem questions for lifetime disorders at the beginning of the interview. Responses to screener items determined which supplemental sections were administered, which also included skip logic based on participant responses, resulting in interviews lasting from 2–3 hours. Adolescents were compensated $50.

PARTICIPANTS

Participants were the 10,140 adolescents (M_{age} = 15.18; SD = 1.51; 51.1% girls) with complete predictor (e.g., SAD status) and outcome (AUD status) data. Race/ethnicity was reported as follows: 55.7% non-Hispanic White, 19.3% non-Hispanic Black, 18.9% Hispanic, and 6.1% other. Parental education (as reported by adolescent participants) included: 16.7% did not complete high school, 30.5% high school degree or equivalent diploma, 19.7% some

college, and 33.2% graduated college. Annual household income, also reported by youth, was as follows: 12.6% below $10,000, 8.6% between $10,000 and $24,999, 20.6% between $25,00 and $74,999, 12.7% between $75,000 and $199,999, 3.9% over $200,000, and 41.7% unknown or did not answer.

MEASURES

Diagnostic Assessment

The World Health Organization Composite International Diagnostic Interview Version 3.0 (CIDI), modified for the appropriate assessment of adolescents (Merikangas et al. 2009), was used to assess for past 12-month SAD, AUD, as well as diagnostic covariates (12-month PTSD, MDD, and other drug dependence). Designed to generate *DSM-IV* diagnoses, positive responses to stem questions were followed by comprehensive, fully structured interviews by trained researchers. The AUD variable in the current study included both alcohol abuse and dependence.

Insomnia Symptoms

Questions regarding insomnia symptoms were drawn from the section on health and related problems asked of all participants. Specifically, participants indicated whether they had experienced difficulty falling asleep, staying asleep, and waking too early for at least 2 weeks over the past 12 months. Each problem was asked in a yes/no format; consistent with prior work (e.g., Babson et al., 2008; Blank et al., 2015), responses were summed to generate a symptom scale of 0–3 (current study Cronbach's alpha = .67).

ANALYTIC APPROACH

All variables were re-coded such that a '0' reflected a 'no' response, no diagnosis, or male, and a '1' reflected a 'yes' response, positive diagnostic status, or female. A series of regression analyses including the weighting variable were used to test whether (1) SAD positively related to AUD status (logistic regression), (2) SAD positively related to insomnia

Table 1
Sample Descriptive Statistics by Weighting and Alcohol Use Disorder (AUD) Status

	Weighted		Unweighted	
Demographics	AUD (-)	AUD (+)	AUD (-)	AUD (+)
Age (*M, SD*)	15.14 (1.45)	16.35 (1.20)	15.11 (1.49)	16.42 (1.24)
Gender (Female)	49.1%	42.3%	51.6%	41.5%
Race/Ethnicity				
Hispanic	14.3%	16.0%	18.7%	22.6%
Black	15.6%	4.5%	20.0%	5.7%
Other	5.1%	2.3%	6.2%	5.5%
White	65.0%	77.2%	55.1%	66.3%
Household Income				
<$10k	22.2%	12.4%	24.9%	16.3$
$10k- $24,999	14.6%	14.9%	16.4%	16.3%
$25k-$74,999	34.9%	40.8%	32.0%	32.8%
$75k-$199,999	21.7%	22.6%	20.2%	23.7%
>=$200k	6.6%	9.2%	6.6%	10.9%
Poverty Income Ratio				
<= 1.5	14.9%	11.0%	17.1%	14.6%
<=3.0	19.3%	15.5%	20.2%	16.4%
<=6	31.9%	31.9%	30.6%	32.4%
>6	34.0%	41.6%	32.2%	36.6%
Insomnia Symptoms (past 2 weeks)				
Total Symptoms Endorsed (M, SD)	0.52 (0.87)	0.80 (1.00)	0.52 (0.88)	0.72 (1.02)
Trouble falling asleep (% endorsed)	22.7%	32.7%	22.3%	33.2%
Trouble staying asleep (% endorsed)	13.1%	22.1%	13.4%	21.8%
Waking too early (% endorsed)	16.6%	26.2%	17.0%	24.8%
Past Year DSM Diagnoses				
Social Anxiety Disorder	12.6%	20.7%	12.3%	19.5%
Major Depressive Disorder	7.0%	15.2%	7.2%	16.2%
PTSD	2.7%	14.9%	2.5%	9.6%
Other Drug Use Disorder	0.6%	12.7%	0.6%	12.5%

Note. Weighted = Sample weights applied; Unweighted = Sample weights not applied; AUD (-) = Past Year Alcohol Use Disorder Not Present; AUD (+) = Any Past Year Alcohol Use Disorder Present; PTSD = Posttraumatic Stress Disorder; Poverty Income Ratio = the ratio of family income to the family's poverty threshold level based on family size.

symptoms (linear regression), and (3) insomnia symptoms positively related to AUD status with SAD status in the model (logistic regression). Regression coefficients were further subjected to the Sobel test (e.g., MacKinnon & Dwyer, 1993) to ascertain whether insomnia symptoms accounted for a significant proportion of the relation between SAD and AUD. Finally, analyses were repeated to test whether these relations were robust to identified covariates.

Results

PRELIMINARY ANALYSES

In the total weighted sample, past year diagnostic rates were as follows: 4.8% past year AUD, 12.7% SAD, 7.4% MDD, 3.3% PTSD, and 1.2% other drug use disorder. The mean level of insomnia symptoms was 0.53 (SD = 0.88). Please see Table 1 for descriptive statistics on the weighted and unweighted data for adolescents with and without any past year AUD.

PRIMARY ANALYSES

Analyses of weighted data (without covariates) indicated that SAD was significantly related to AUD status (odds ratio [OR] = 1.80, 95% confidence interval [CI] = 1.79-1.81, $p < .001$), SAD was significantly related to insomnia symptoms ($\beta = .20$, $p < .001$, $sr^2 = .04$), and insomnia symptoms significantly related to AUD status (OR = 1.36, 95% CI [1.35-1.36], $p < .001$). Finally, the Sobel test indicated that the effect of insomnia symptoms on the relation between SAD and AUD status was significant ($z = 241.19$, $p < .001$), although the relation between SAD and AUD also remained significant (OR = 1.54, 95% CI [1.53, 1.55], $p < .001$). As seen in Table 2, these findings were consistent with the inclusion of age, gender, PTSD, MDD, and other drug dependence status as covariates at each stage of the model ($z = 142.84$, $p < .001$).[1]

Table 2
Analysis With Weighted Sample

	sr^2	B	OR	95% CI
DV: AUD				
Step 1				
Age		0.59	1.81	1.81-1.81
Gender		-0.44	0.63	0.63-0.64
PTSD		1.67	5.33	5.29-5.37
MDD		0.45	1.58	1.57-1.59
Drug dependence		2.82	17.93	17.76-18.10
Step 2				
Social anxiety		0.33	1.39	1.38-1.40
DV: Insomnia symptoms				
Step 1				
Age	.000	-0.00		-0.00- -0.00
Gender	.007	-0.08		-0.08- -0.08
PTSD	.023	0.76		0.76-0.77
MDD	.029	0.59		0.59-0.59
Drug dependence	.002	0.44		0.43-0.44
Step 2				
Social anxiety	.024	0.41		0.41-0.41
DV: AUD				
Step 1				
Age		0.59	1.81	1.81-1.81
Gender		-0.44	0.63	0.63-0.64
PTSD		1.67	5.33	5.29-5.37
MDD		0.45	1.58	1.57-1.59
Drug dependence		2.88	17.93	17.76-18.10
Step 2				
Insomnia		0.16	1.18	1.17-1.18
Step 3				
Social anxiety		0.29	1.34	1.33-1.35

Note. Gender coded with males = 0 (reference in logistic regression) and females = 1; PTSD = Past Year Posttraumatic Stress Disorder Diagnosis; MDD = Past Year Major Depressive Disorder Diagnosis; Drug Dependence = Any Past Year Drug Use Disorder; Insomnia = Total Insomnia Symptoms; Social Anxiety = Past Year Social Anxiety Disorder

Discussion

A substantial literature indicates that individuals with elevated social anxiety/SAD are at risk for the development of an AUD (e.g., Buckner, Schmidt, et al., 2008; Nelson et al., 2000). The majority of work addressing this relation, conducted almost exclusively with adults, has focused on the use of alcohol to manage acute social stress (Battista, Stewart, & Ham, 2010). Although promising, this literature remains mixed and effect sizes range across studies. Of note, social anxiety also has been linked to sleep difficulties, including insomnia (Johnson, Roth, & Breslau, 2006), and insomnia symptoms are related to risk-related alcohol use (Wong et al., 2004). No work has directly examined the relations among SAD, insomnia, and AUDs among youth. Given that adolescence is a sensitive period in terms of the onset of SAD (Grant et al., 2005), insomnia (Johnson, Roth, Schultz, & Breslau, 2006), and alcohol use problems (Swendsen et al., 2012), the present study examined whether insomnia symptoms partially accounted for the SAD-AUD link in a large,

[1] Given expected nonnormal distributions across each variable, primary analyses were complemented by analyses using a bootstrapping procedure via the PROCESS custom dialog for SPSS 22 (5000 bootstrapping samples; assessment of bias-corrected 95% confidence interval [CI]; Preacher & Hayes, 2008; Hayes, 2012, 2013). Analyses of unweighted data, both with and without covariates, also indicated positive relations between SAD and AUD status, SAD and insomnia symptoms, as well as insomnia symptoms and AUD status. Further, analyses indicated that the statistical indirect effect of SAD on AUD through insomnia symptoms was significant both without (corrected $\beta = .13$, $SE = .02$, 95% CI [.08, .19]; $Z = 5.39$, $p < .001$), and with (corrected $\beta = .06$, $SE = .02$, 95% CI [.02, .11]; $Z = 3.05$, $p = .002$) covariates in the model.

cross-sectional sample of U.S. adolescents. Findings indicated SAD was positively related to AUD status, and insomnia symptoms accounted for a significant proportion of the SAD-AUD relation. Further, age, gender, PTSD, MDD, and other drug dependence status did not better account for these relations.

Specifically, those with a SAD diagnosis were almost twice as likely to also be diagnosed with an AUD compared to those without a diagnosis. A diagnosis of SAD was positively associated with insomnia symptoms, and for each insomnia symptom endorsed there was a 1.36 increased odds of being diagnosed with an AUD. Importantly, markers of insomnia also partially accounted for the SAD-AUD association. These data complement ongoing work emphasizing the role of social anxiety in relation to problematic alcohol use (Buckner, Schmidt, et al., 2008; Conway et al., 2016), and call attention to difficulty sleeping, and insomnia specifically, in this relation. The findings from the current, cross-sectional analyses suggest that future work examining the indirect effect of insomnia symptoms on the link between SAD and AUD in longitudinal designs is warranted (Kraemer et al., 2000).

Despite laws limiting adolescent access to alcohol, the social settings that characterize youth drinking may serve as a precipitous learning environment for the coping-related use of alcohol (i.e., rapid pairing of distress and use-related relief). Socially anxious adolescents, who are at particular risk for the uptake of short-term (McManus, Sacadura, & Clark, 2008; Clark & Wells, 1995), ineffective (Suveg & Zeman, 2004), and escape-avoidance symptom management (Beidel & Randall, 1994; Rapee & Heimberg, 1997; Turk et al., 2001), may be especially sensitive to such learning episodes. As youth incorporate alcohol use into their budding coping repertoire, they also may learn to expand the approach to other relevant contexts including sleep-related difficulties, thus compounding risk for AUD development and maintenance. Importantly, the current data did not directly address typical motives for drinking, whether insomnia symptoms were associated with social anxiety-related rumination or worry, or the degree to which alcohol was used in response to insomnia symptoms. Although the current data provide initial support for these relations, the NCS-A did not assess these factors, so future work is needed to specify if these areas are targets for intervention.

The current study also included chronological age, reported gender, PTSD, MDD, and other drug dependence as covariates. Research designed to address the additive, interactive, and potential developmental cascade associated with these and more central factors (e.g., pubertal development, poly-substance use; Bakken, Landheim, & Vaglum, 2005; Castellanos-Ryan, Parent, Vitaro, Tremblay, & Séguin, 2013) is needed to capture the complexity of these relations. For instance, the early stages of the pubertal process are characterized by maturation of the hypothalamic-pituitary-adrenal (HPA) axis. The HPA-axis is affected by and implicated in emotion dysregulation (Condren, O'Neill, Ryan, Barrett, and Thakore, 2002; Tarullo & Gunnar, 2006), insomnia (Buckley & Schatzberg, 2005), and problematic alcohol use (Thayer, Hall, Sollers, & Fischer, 2006). Further, prior work suggests that alcohol actually disrupts sleep quality (Ebrahim, Shapiro, Williams, & Fenwick, 2013), thus exacerbating insomnia symptoms. It's possible that a vicious cycle develops, in which insomnia symptoms lead to an increase in alcohol use, and increased alcohol use leads to an increase in sleeping difficulties (Bootzin & Stevens, 2005). Experimental work addressing pre-clinical phenomena via affect induction and sleep deprivation, in conjunction with longitudinal assessments of both pre-clinical and treatment populations, will aid in specifying the mechanistic, temporal, and boundary conditions of the identified relations.

The current data suggest that alongside intervention work addressing acute stress responding (Conrod et al., 2011, 2013; Kendall, Safford, Flannery-Schroeder, & Webb, 2004), insomnia symptoms may be an important target to address in both prevention and treatment efforts for problematic alcohol use among socially anxious youth. Cognitive behavioral therapy for insomnia (CBT-I) is considered the gold standard for insomnia treatment among adults (Edinger & Means, 2005), and evidence supports the use of related treatments for adolescents with insomnia (Bootzin & Stevens, 2005; De Bruin, Bögels, Oort, & Meijer, 2015). In fact, Bootzin and Stevens found that a CBT-I related intervention for adolescents completing a substance use treatment program not only improved sleep (i.e., sleep efficiency, sleep onset latency, number of awakenings, sleep time total, and self-reported sleep quality), but also led to a decrease in substance use 12-months postintervention. This finding suggests that the treatment of sleep may have a subsequent impact on substance use behaviors. Indeed, several aspects of most CBT-I protocols target stress management (e.g., relaxation, problem solving), which might cut across the core features of related problems with both social anxiety and alcohol use. However, additional work is needed to examine the potential impact of a sleep intervention on alcohol use behaviors among socially anxious adolescents.

Finally, in addition to the use of retrospective report and cross-sectional data, the present findings should be considered in light of several limitations.

First, all study procedures were conducted in English, and the majority of participants were recruited via school-based sampling. As such, important segments of the U.S. population (e.g., non-English speaking, homeless) were not included; future efforts targeting these underrepresented groups will be key to understanding the true breadth of identified relations (Alegria et al., 2004; Merikangas et al., 2011). Second, to help alleviate the time commitment of the participants, a screener was used and those who did not endorse initial symptoms did not undergo in-depth diagnostic assessments. Unfortunately, this approach precludes comprehensive, preclinical, and more statistically powerful comparisons of continuous symptom levels. Third, consistent with prior work using these and similar data (Babson et al., 2008; Zhang et al., 2017), the assessment of sleep disruption was post-hoc and far from ideal (e.g., current study insomnia symptom $\alpha = .67$). Finally, diagnoses were based on DSM-IV criteria; replication in a more recent sample with updated diagnostic assessments (e.g., DSM-5; American Psychiatric Association, 2013) is necessary.

Together, the current findings suggest individuals with SAD may use alcohol as a means of coping with insomnia symptoms. Insomnia symptoms may be an important indirect and stigma free treatment target to address in prevention and treatment efforts for SAD, AUDs, and their co-occurrence.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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