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The Association of Alcohol Severity and Sleep Quality in Problem Drinkers

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Abstract

Aims: The association between alcohol use and sleep problems is well established and clinically meaningful, particularly as predictors of relapse. This study aims to elucidate the relationship between sleep disturbances and alcohol problems in a non-treatment-seeking community sample using an alcoholism problem severity factor.

Methods: Participants were problem drinkers ($N = 295$) from the Los Angeles community who had a breath alcohol content (BrAC) of 0.00 g/dl when they completed an in-person assessment battery comprised of measures of sleep quality, anxiety and depression, cigarette smoking, as well as multiple assessments of alcohol use and alcohol use problems.

Results: A series of hierarchical regressions showed that alcohol problem severity explained a significant amount of variance in sleep disturbance beyond demographic, mood and smoking variables. Alcohol problem severity was predictive of the PSQI global score ($B = 1.11$, $P < 0.001$), perceived sleep quality factor ($B = 0.18$, $P < 0.001$) and daily disturbance factor ($B = 0.28$, $P < 0.001$). However, contrary to study hypothesis, alcohol problem severity was predictive of improved sleep efficiency ($B = -0.14$, $P < 0.05$).

Conclusions: In sum, alcohol problem severity may be predictive of sleep disturbances. Given the complex nature of these relationships, further work is needed to develop adequate treatment for sleep disturbance during alcohol recovery. Nonetheless, this study suggests that as alcohol problem severity increases so do sleep problems. Thus, attending to sleep problems at early stages of alcohol problems may be warranted.

INTRODUCTION

The relationship between sleep disturbances and alcohol use is well documented (Stein and Friedmann, 2006). In the general population, 29.9% report some symptoms of insomnia and the prevalence of clinical insomnia is ~9.5% based on the combination of DSM-IV and ICD diagnostic criteria (Morin *et al.*, 2006). However, in alcohol-dependent samples, the prevalence of sleep problems has been reported to be as high as 72% (Foster *et al.*, 1998). Despite the fact that alcohol use can worsen sleep quality, particularly when consumed in relatively large quantities or over long durations (Colrain *et al.*, 2014), the intention to use alcohol to mitigate sleep difficulties is commonly cited as a reason for individuals initiate, and continue, regular alcohol use (Johnson *et al.*, 1998). Patients entering treatment for alcohol have been shown to use alcohol significantly more frequently as a sleep aid if they have comorbid insomnia compared with patients who do not (Brower *et al.*, 2001). Furthermore, a large portion of the cost burden associated with insomnia is accounted for the use of alcohol as a sleep aid (Daley *et al.*, 2009).

Evidence suggests that sleep is dysregulated during all stages of an alcohol use disorder (AUD), including active heavy use, early abstinence and prolonged abstinence (Foster and Peters, 1999). Symptoms of insomnia have been observed in alcohol-dependent patients at rates as high as 91% (Zhabenko *et al.*, 2012). In the National Epidemiological Survey on Alcohol and Related Conditions (NESARC), those with a lifetime diagnosis of alcohol dependence reported a prevalence of alcohol withdrawal-related insomnia of 31.7% (Brower and Perron, 2010). During recovery, sleep problems have been shown to predict relapse (Brower *et al.*, 1998), even when patients' quality of life and other psychiatric symptoms are improving (Cohn *et al.*, 2003). Nevertheless, effective approaches to the treatment of sleep disturbances, especially during alcohol recovery, are lacking (Arnedt *et al.*, 2007). There is some indication that cognitive behavioral therapy (Arnedt *et al.*, 2011) and certain pharmacotherapies (e.g. acamprosate, gabapentin; Kolla *et al.*, 2011; Perney *et al.*, 2012) may be particularly promising at targeting sleep disturbance in alcohol-dependent patients.

Severity of alcohol dependence may be associated with the prevalence and severity of sleep disturbances. Extant literature suggests that patients entering treatment for alcohol with comorbid insomnia have greater scores on the Michigan Alcoholism Screening Test and greater depression severity than patients without clinical insomnia (Brower *et al.*, 2001). In a large, Polish sample of inpatient and outpatient alcohol-dependent individuals ($N = 302$), insomnia was associated with greater severity of alcohol dependence, earlier age of onset of alcohol use and higher drinking frequency and quantity (Zhabenko *et al.*, 2012). Conversely, Foster and Peters (1999) reported that Pittsburgh Sleep Quality Index (PSQI) scores were not correlated with the severity of alcohol dependence, measured by the Severity of Alcohol Dependence Questionnaire, in a sample of alcohol-dependent outpatients. Methodological issues such as differences in sample characteristics and assessments, for sleep in particular, may account for some of the inconsistencies in the literature. The majority of studies examine treatment-seeking populations; therefore, less is known about the role of sleep impairment in non-treatment-seeking individuals who are currently drinking and experiencing alcohol-related problems.

Given the high comorbidity of sleep problems and AUDs, the primary aim of this study is to test whether alcohol problem severity, using a multifactorial alcoholism severity score, serves as a predictor of sleep disturbance in a large community sample of non-treatment-seeking problem drinkers. We hypothesize that a greater alcohol problem severity score will be associated with poorer sleep quality as indicated by PSQI scores. As it has been observed that individuals suffering from sleep problems have greater risk of relapse to alcohol (Brower *et al.*, 2001), studies that can advance our understanding of the relationship between alcohol use and sleep disturbance have the potential to inform diagnosis and intervention.

METHODS

Participants and procedures

A number of non-treatment-seeking problem drinkers ($N = 295$) were recruited from the greater Los Angeles area for a study investigating the subjective effects of alcohol (Ray *et al.*, 2013). Based on eligibility from a telephone interview, participants were invited to UCLA for an in-person assessment where they provided written informed consent and completed a battery of individual differences measures. Inclusion criteria were: (a) age 21–65; (b) self-reported problems with alcohol; and (c) endorse consuming ≥48 drinks per month. The purpose of the 48 drink cut-off was to identify heavy drinkers who would likely meet for an AUD during baseline visit, a requirement of entry into the study (72% of the sample met criteria for alcohol dependence). Exclusion criteria were: (a) treatment-seeking or history of treatment for alcohol problems within the past month; (b) the self-reported current use of illicit substances (other than marijuana) and (c) self-reported lifetime diagnosis of schizophrenia, bipolar disorder or psychotic disorders.

Measures

Participants completed a demographic questionnaire which included age, education, marital status, ethnicity, family history of alcohol problems and smoking status. The Pittsburgh Sleep Quality Index (PSQI; Buysse *et al.*, 1989), a well-validated, 19-item measure, was used to assess sleep quality and quantity over the past month. Seven components of sleep difficulties are computed, including sleep quality, latency, duration, efficiency, disturbances, the use of medication and daytime dysfunction. A global sleep quality score is calculated by adding the seven component scores as described by Buysse *et al.* (1989). A global score <5 is indicative of clinically relevant insomnia. A three-factor scoring model has been validated in various samples (Cole *et al.*, 2006; Casement *et al.*, 2012) whereby the factors capture: (a) sleep efficiency (e.g. 'During the past month, what time have you usually gone to bed at night?' 'How long has it usually taken you to fall asleep each night?'), (b) perceived sleep quality (e.g. 'How would you rate your sleep quality overall?' 'How often have you taken medication (prescribed or over the counter) to help you sleep?') and (c) daily disturbances (e.g. 'During the past month, how often have you had trouble staying awake while driving, eating meals or engaging in social activity?'). The three-factor scoring model was shown to better capture the multidimensional nature of sleep disturbances (Cole *et al.*, 2006), hence the three-factor approach to the PSQI was used in this study. Observed Cronbach alpha for the PSQI was 0.79 for the current sample.

The Timeline Follow-back (TLFB) was used to assess alcohol use frequency and quantity over the past 30-days (Sobell *et al.*, 1986). Specifically, drinks per drinking day and number of drinking days were computed.

The Beck Depression Inventory-II (BDI-II; Beck *et al.*, 1996), a 21-item measure, assessed severity of depressive symptomatology over the preceding 2 weeks. Total scores range from 0 to 63, where mild, moderate and severe depression are indicated by scores of 14–20, 21–30 and ≥31, respectively. Observed Cronbach alpha for the BDI was 0.95 demonstrating excellent reliability in the current sample.

The Beck Anxiety Inventory (BAI; Beck and Steer, 1996), a 21-item measure, assessed severity of anxious symptomatology over the preceding 2 weeks. Total scores range from 0 to 63, where mild, moderate and severe anxiety is indicated by scores of 8–15, 16–25 and ≥26, respectively. Observed Cronbach alpha for the BAI was 0.96, showing excellent reliability.

The Fagerstrom Test for Nicotine Dependence (FTND; Heatherton *et al.*, 1991) is a 6-item measure which assessed the level of nicotine dependence. Total scores range from 0 to 10, where scores greater than 5 indicate moderate to severe nicotine dependence.

Alcoholism severity

The Structured Clinical Interview for DSM-IV (SCID) was used to assess the presence of alcohol abuse and/or dependence at baseline assessment and absence of exclusionary disorders (First *et al.*, 1995) by master's level clinicians who were supervised by a licensed psychologist. The symptoms of alcohol dependence and abuse from the SCID were totaled for a sum of 11 possible symptoms. To aide interpretation of these findings in the context of DSM-5 AUD (American

Psychiatric Association, 2013), we examined symptom count across abuse and dependence which are collapsed into AUD in DSM-5 (excluding craving which was not assessed). In this sample, 12 participants (4%) did not report any symptoms of alcohol abuse or dependence and 16 participants (5%) reported only one symptom. Thus, it appears this sample would be representative of a sample recruited based on DSM-5 AUD.

Other measures of alcohol use included the CIWA-Ar (Sullivan *et al.*, 1989), the Alcohol Dependence Scale (ADS; Skinner and Horn, 1984), the Drinkers Inventory of Consequences (DrInC-2R; Miller *et al.*, 1995) and the Penn Alcohol Craving Scale (PACS; Flannery *et al.*, 1999). Given the shared variance amongst these measures, a principle components analysis was conducted in the full sample for these five measures to derive a single alcohol problem severity factor (Ray *et al.*, 2013). Factor scores were computed for each subject and used as an index of alcoholism severity in this study.

Data analysis plan

Means, standard deviations and percentiles were calculated for all demographic variables for the total sample and separately for those who did or did not meet the PSQI global cut-off score for clinically relevant insomnia (see Table 1). T-tests and χ^2 tests were run to assess for differences between groups. A series of hierarchical regressions were run in SAS Statistical Software (SAS 9.3; Cary, NC) to test the primary hypotheses. The primary dependent measures were PSQI global score and the three factors scores derived from the PSQI components. First, demographic variables, including sex, age, education, employment status, ethnicity and family history of AUDs, were entered into block 1 of each model. Second, BAI and BDI were entered to account for psychiatric symptomatology. Third, the FTND total score was entered. The final block comprised of the alcohol problem severity score. R^2 change was calculated and tested at each step. While this approach is quite conservative, requiring alcohol severity to explain variance over and above a host of other factors, it can also result in anomalous outcomes in some rare situations (Simmons *et al.*, 2011). Therefore, in keeping with recommendations from Simmons *et al.* we also examined the association between alcohol problem severity and sleep outcomes in the absence of covariates and the results were identical in their direction and statistical significance. Additionally, analyses were run including only those participants who would meet for an AUD according to DSM-5 (i.e. endorsed two or more DSM-IV symptoms, therefore craving is not included). Results were identical in their direction and statistical significance except for PSQI factor 1 which was non-significant.

Table 1. Means and standard deviations of participants' demographics, psychiatric variables and total PSQI score are presented for the full sample, those without clinically meaningful insomnia, and those with insomnia

	Full sample	No insomnia ($n = 61$)	Insomnia ($n = 239$)	Statistical test
Age, M (SD)	31.0 (10.5)	28.6 (9.1)	32.3 (11)	$t = -1.93$; $P = 0.05$
Male, % (*N*)	73.6 (217)	80.4 (45)	72.0 (172)	$\chi^2 = 1.64$; $P = 0.2$
White, % (*N*)	55.4 (163)	60.0 (33)	54.4 (130)	$\chi^2 = 0.57$; $P = 0.45$
African American, % (*N*)	24.5 (72)	12.7 (7)	27.2 (65)	$\chi^2 = 5.06$; $P = 0.02$
Asian, % (*N*)	8.2 (27)	12.7 (7)	8.4 (20)	$\chi^2 = 1.01$; $P = 0.31$
Latino, % (*N*)	22.5 (66)	27.3 (15)	21.3 (51)	$\chi^2 = 0.90$; $P = 0.34$
Native American, % (*N*)	6.5 (19)	3.6 (2)	7.1 (17)	$\chi^2 = 0.89$; $P = 0.34$
Employed, % (*N*)	66 (194)	34.6 (19)	27.6 (81)	$\chi^2 = 2.43$; $P = 0.29$
DPDD, M (SD)	7.1 (4.6)	6.8 (3.9)	7.4 (5.1)	$t = -0.74$; $P = 0.46$
Drinking days, M (SD)	18.1 (7.2)	16.6 (7.1)	18.4 (7.2)	$t = -1.88$; $P = 0.06$
Binge drinking days, M (SD)	12.23 (8.1)	11.2 (7.8)	12.5 (8.2)	$t = -1.05$; $P = 0.29$
Dependence, % (*N*)	72 (213)	62.3 (38)	74.5 (175)	$\chi^2 = 3.56$; $P = 0.06$
DSM-IV symptom count, M (SD)	5.3 (2.8)	4.5 (2.5)	5.5 (2.9)	$t = -2.5$; $P = 0.01$
BDI-II score (SD)	21.6 (12.7)	15.3 (10.4)	23.4 (13.3)	$t = -4.13$; $P < 0.001$
BAI score (SD)	18.9 (12.9)	14.2 (11.8)	20.2 (13.2)	$t = -3.08$; $P = 0.002$
ADS score (SD)	15.4 (7.4)	12.2 (5.8)	16.3 (7.7)	$t = -3.69$; $P < 0.001$
DRINC score (SD)	41.3 (22.5)	30.5 (13.4)	43.8 (23.5)	$t = -4.05$; $P < 0.001$
DRINC physical consequences (SD)	15.2 (3.3)	14.1 (2.3)	15.4 (3.5)	$t = -2.74$; $P = 0.007$
DRINC intrapersonal consequences (SD)	16.6 (4.0)	14.7 (2.9)	17.0 (4.0)	$t = -3.92$; $P < 0.001$
DRINC social consequences (SD)	14.4 (3.3)	12.7 (2.5)	14.7 (3.4)	$t = -4.19$; $P < 0.001$
DRINC interpersonal consequences (SD)	18.6 (5.3)	16.2 (3.4)	19.1 (5.6)	$t = -3.79$; $P < 0.001$
DRINC impulse consequences score (SD)	20.5 (5.7)	18.0 (3.3)	21.0 (6.0)	$t = -3.64$; $P < 0.001$
CIWA score (SD)	5.7 (6.9)	3.9 (4.3)	6.1 (7.6)	$t = -2.26$; $P = 0.02$
FTND score (SD)	11.3 (1.8)	11.7 (1.7)	11.2 (1.9)	$t = 1.43$; $P = 0.16$
PSQI global score (SD)	7.4 (3.3)	3.2 (1.1)	8.4 (2.8)	$t = -14.19$; $P < 0.001$
PSQI Factor 1 Sleep efficiency	1.3 (1.1)	0.7 (1.1)	1.5 (1.0)	$t = -5.1$; $P < 0.001$
PSQI Factor 2 Sleep quality	1.1 (0.6)	0.5 (0.3)	1.2 (0.6)	$t = -9.6$; $P < 0.001$
PSQI Factor 3 Daily disturbances	1.1 (0.6)	0.6 (0.4)	1.3 (0.5)	$t = -10.4$; $P < 0.001$
Sleep duration	6.5 (2.0)	7.6 (1.8)	6.2 (1.9)	$t = 4.8$; $P < 0.001$

RESULTS

Sample characteristics

As presented in Table 1, the sample averaged 31 years in age, primarily consisted of Caucasians (55%) and the majority were employed (66%). Nearly 23% reported being current, daily cigarette smokers. In the past 30 days, participants reported an average of 18.1 drinking days and 12.2 binge drinking days. The BDI and BAI means were 21.6 and 18.9, respectively, indicating the sample experienced moderate symptoms of depression and anxiety in the 2 weeks prior to the assessment visit.

Additionally, Table 1 shows that significant group differences were observed for multiple drinking measures such that those endorsing clinically meaningful insomnia reported a greater number of drinking days ($P = 0.06$), higher ADS score ($P < 0.001$), higher DRINC score ($P < 0.001$) and higher levels of withdrawal ($P < 0.02$). The insomnia group also exhibited a trend towards higher rates of dependence than the non-sleep impaired group ($P = 0.06$) and endorsed a greater number of total DSM-IV alcohol abuse and dependence symptoms ($P = 0.01$). Those who experienced insomnia were older than those who did not ($P = 0.05$) but did not differ in sex, ethnicity or employment status. The group experiencing insomnia also reported worsened depression ($P < 0.001$) and anxiety ($P = 0.002$) symptoms compared with the group who did not report insomnia.

Sleep quality and alcohol severity

Overall, the sample reported low sleep efficiency, poor sleep quality and significant daily disturbances with respect to available PSQI norms (Buysse *et al.*, 1989). In total, 76% of the sample ($n = 239$) reported clinically relevant insomnia (i.e. PSQI global score >5) and the mean PSQI global score of the entire sample was 7.4 (Table 1). Results from the hierarchical model predicting global PSQI score are presented in Table 2. In this model, demographic variables entered in step 1 accounted for 5.76% of the variance ($P < 0.01$). The addition of mood variables in step 2 accounted for an additional 6.56% ($P < 0.05$) and the inclusion of FTND score did not improve the model (R^2 change: 1.21%, $P > 0.10$). Finally, alcohol problem severity was included at step 4 which significantly explained an additional 9% ($B = 1.11$, $P < 0.001$) of the variance in global sleep quality. A positive relationship appeared such that greater alcohol problem severity predicted worsened global PSQI score.

In terms of sleep efficiency (PSQI factor 1), demographic variables accounted for 5.81% of the variance ($P < 0.02$). Inclusion of mood variables then accounted for an additional 8.24% ($P < 0.001$) and nicotine dependence accounted for a further 1% ($P > 0.10$). Over and above these other factors, severity of alcohol problems explained an additional 1.3% ($P < 0.05$) of the variance in sleep efficiency. A negative relationship between alcohol problem severity and sleep efficiency emerged, such that greater alcohol problem severity predicted better sleep efficiency ($B = -0.14$, $P < 0.05$).

For sleep quality, demographic variables accounted for 3.51% of the variance ($P < 0.15$). The addition of mood variables accounted for an additional 4.27% ($P < 0.01$) and smoking severity accounted for an additional 1% ($P < 0.06$). Over and above these other factors, alcohol problem severity accounted for a further 7% ($B = 0.18$, $P < 0.001$), indicative of a positive relationship such that increased alcohol problem severity predicted worsened sleep quality.

Demographic variables accounted for 2.55% of the variance in daily disturbance (P = NS), mood variables accounted for an additional 5% ($P < 0.001$) and nicotine accounted for a further 5.29% ($P < 0.001$). In the final step, alcohol problem severity accounted for an additional 22% ($B = 0.28$, $P < 0.001$), indicating a positive relationship such that increased alcohol problem severity predicted worsened daily disturbances in sleep.

Table 2. Results of hierarchical regression analysis predicting PSQI global score and three PSQI factors and R^2 change for each block (unstandardized regression coefficients are presented)

	PSQI global			Sleep efficiency			Sleep quality			Daily disturbances		
	R^2 change	*B*	*P*	R^2 change	*B*	*P*	R^2 change	*B*	*P*	R^2 change	*B*	*P*
Block 1												
Sex	0.0576**	*−0.864*	*0.06*	0.0581*	−0.007	0.962	0.0351	**−0.199**	**0.01**	0.0255	**−0.156**	**0.011**
Age		**0.062**	**0.002**		**0.02**	**0.002**		0.0004	0.911		0.001	0.675
Education		−0.006	0.736		0.0007	0.901		−0.002	0.548		0.0009	0.676
Employed		0.343	0.424		0.083	0.537		0.043	0.557		−0.07	0.23
Ethnicity		−0.061	0.787		0.07	0.331		−0.049	0.207		−0.042	0.175
Family history		−0.256	0.534		−0.107	0.41		−0.067	0.344		−0.072	0.207
Block 2												
Depression (BDI-II)	0.0656***	**0.096**	**0.001**	0.0824***	**0.033**	**0.0001**	0.0427**	**0.01**	**0.047**	.0529***	**0.008**	**0.037**
Anxiety (BAI)		−0.04	0.125		−0.012	0.177		−0.003	0.475		−0.002	0.645
Block 3												
Nicotine dependence (FTND)	0.0121*	*−0.002*	*0.056*	0.0089	0.0003	0.285	0.0117[a]	−0.0001	0.572	.0472***	*−0.0002*	*0.089*
Block 4												
Alcohol problem severity	0.0912***	**1.11**	**<.001**	0.0132*	**−0.138**	**0.043**	0.0745***	**0.18**	**<.001**	.2179***	**0.277**	**<.001**

*$P < .05$.
**$P < .01$.
***$P < .001$.
[a]Trend.
Bold items are significant, italic items are trend.

DISCUSSION

The link between alcohol use and sleep problems is well established and clinically meaningful, particularly, with respect to sleep disturbances as predictors of relapse (Brower *et al.*, 1998, 2001). The purpose of this study was to elucidate the relationship between sleep disturbances and alcohol problems in a non-treatment-seeking community sample using an innovative alcoholism problem severity factor. Alcohol problem severity explained a significant amount of variance in each model, over and above demographic variables, mood variable (e.g. BDI, BAI) and nicotine dependence severity. Furthermore, the alcohol problem severity factor predicted greater impairment in global sleep quality, perceived sleep quality and daily disturbance scores, as measured by the PSQI global and factor scores. Contrary to hypotheses, the opposite relationship was observed for sleep efficiency such that increased alcohol problem severity predicted better sleep efficiency. While this finding is contrary to the study's hypothesis, it raises the possibility that more severe drinkers used alcohol so heavily that use led to the reported increase in sleep efficiency, defined as the percent of time in bed that is actually spent sleeping. This non-treatment-seeking, active alcohol using sample may rely on alcohol to improve sleep in this manner whereas other studies in this area analyze treatment-seeking samples who desire to reduce alcohol use. Furthermore, impaired recall may also be a component of this finding at which point objective measures of sleep (e.g. polysomnography) would be informative. Nevertheless, consistent with the current literature, this study replicates and extends findings that active alcohol users experience significant sleep problems (Brower and Perron, 2010) by demonstrating a relationship between alcohol problem severity and sleep disturbances, particularly in regard to perceived sleep quality and daily disturbances. Other research has shown similar associations (e.g. Brower *et al.*, 2001; Zhabenko *et al.*, 2012), though often relying on single measures (e.g. MAST; Brower *et al.*, 2001) to analyze comparable data. This study used a novel factor score to characterize alcohol problem severity, an index that combined several reliable and clinically relevant measures that may more accurately capture the multifactorial elements of alcohol use and problems. The factor taps several clinically relevant domains of alcohol use and related problems, including symptoms of alcohol dependence, heaviness of use, withdrawal and psychosocial consequences.

The study should be considered in light of its strengths and limitations. This study's strengths include a diverse, community sample of men and women, the majority of whom met criteria for an AUD, the use of well-validated and widely employed measures of alcohol-related problems and sleep disturbances and the use of a robust alcohol problem severity factor which captures various domains of alcohol use and associated consequences. In addition, unlike the majority of studies on the alcohol-sleep association among individuals with AUDs, which focus on treatment-seeking or recently abstinent individuals, this study characterizes sleep problems experienced by individuals who are actively experiencing alcohol problems for which they are not receiving treatment.

Several limitations of the present study warrant comment. First, only subjective measures were employed in this secondary analysis. To further investigate the role of alcohol severity and sleep, future studies should consider the use of objective measures, such as polysomnography or actigraphy, to fully capture sleep disturbances in active alcohol users. Furthermore, the current study did not assess for a history of sleep problems diagnosed via objective measures (e.g. sleep apnea). Second, the lack of a control group comprising social drinkers or non-drinkers, may limit the interpretation of results. Longitudinal studies examining the impact of alcohol on sleep, and vice versa, are needed to demonstrate the reciprocal relationship therein. Third, the findings from this study do not shed light on the cause of the observed sleep disturbances, which may, in part, be accounted for psychiatric and medical comorbidities that further complicate the alcohol-sleep relationship. Such comorbidities may also impact sleep functioning, although the results reported here were robust after controlling for measures of depression and anxiety, suggesting a direct association between the degree of alcohol problem severity and sleep disturbance in this sample.

Improved treatments for sleep disturbance in early recovery are greatly needed (Arnedt *et al.*, 2007; Kaplan *et al.*, 2014). Studies regarding psychosocial and pharmacological interventions have yielded promising results, albeit in primarily smaller samples with short follow-up periods. Promising therapies appear to be acamprosate, gabapentin and CBT which have been shown to improve treatment outcomes for individuals in alcohol recovery suffering from disturbed sleep. Trazadone, another pharmacotherapy used in alcohol treatment-seeking populations who experience sleep difficulty, has shown promise at treating sleep difficulties in this population; however iatrogenic effects on drinking-related outcomes have been observed (Friedmann *et al.*, 2003, 2008). Moreover, one component of CBT for insomnia is 'sleep education,' in which patients learn about many of the domains of sleep, some of which may be directly affected by alcohol use, such as sleep quality and efficiency (Edinger and Carney, 2008). Through this treatment, patients learn to self-monitor their sleep quality and learn strategies to improve it, as well as sleep hygiene. Research that advances our understanding of the impact of alcohol on these variables could have great utility as part of the sleep education process. Another potential avenue for investigation is the combination of these interventions, such as CBT and pharmacotherapy, which may lead to innovative treatment approaches for this unique subpopulation of alcohol users at high risk of relapse.

In conclusion, this study advances the field by demonstrating a robust association between alcoholism severity and sleep disturbance across a range of sleep domains and in a large community sample of non-treatment-seeking problem drinkers. These results are robust even after controlling for several demographic and clinical variables of interest. In sum, these results argue for increased attention to sleep disturbance of the course of alcoholism severity and even within non-treatment-seeking samples of problem drinkers.

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CONFLICT OF INTEREST STATEMENT

None declared.

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