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INSOMNIA, SELF-MEDICATION, AND RELAPSE TO ALCOHOLISM

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Abstract

Objective—This study investigated the frequencies of insomnia and its self-medication with alcohol in a sample of alcoholic patients, as well as the relationship of these variables to alcoholic relapse.

Method—Subjects were 172 men and women receiving treatment for alcohol dependence. They completed a sleep questionnaire, measures of alcohol problem severity and depression severity, and polysomnography after at least two weeks of abstinence.

Results—Using eight items from the Sleep Disorders Questionnaire, >60% of subjects were classified as having symptomatic insomnia during the six months prior to treatment entry. Compared to patients without insomnia, patients with insomnia were more likely to report frequent alcohol use for sleep (55% vs. 28%; $\chi^2=12.03$, $df=1$, $p=0.001$), had significantly worse polysomnographic measures of sleep continuity, and had greater severity scores for both alcohol dependence and depression. Among 74 alcoholics who were followed a mean of five months after treatment, 60% with baseline insomnia vs. 30% without baseline insomnia relapsed to any use of alcohol ($\chi^2=6.16$, $df=1$, $p=0.02$). Insomnia remained a robust predictor of relapse after applying logistic regression analysis to control for other variables. A history of self-medicating insomnia with alcohol did not significantly predict subsequent relapse.

Conclusions—The majority of alcoholic patients entering treatment reported insomnia symptoms. Given the potential link between insomnia and relapse, routine questions about sleep in clinical and research settings are warranted.

INTRODUCTION

Although it is commonly accepted that individuals with alcohol dependence sleep poorly (1–3), few studies have investigated the rates of insomnia in alcoholic patients. Baekeland et al. (4) reported that 36% of 294 alcoholic outpatients had high scores on sleep disturbance as measured by a clinic physician. Similarly, Feuerlein (5) reported that 37% of 184 inpatients and outpatients with alcoholism had “sleep disturbance” as determined by a semi-standardized interview. Caetano et al. (6) investigated insomnia as a withdrawal symptom and found a 42% rate among 445 people referred to educational programs for driving under the influence, and a 67% rate among 748 men admitted to detoxification and residential treatment centers. These insomnia rates (36% to 67%) are higher than those found in the general population (17% to 30%) (7). Nevertheless, comparisons between studies are

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difficult, because different insomnia measures and time frames were employed, and because samples may have differed in demographics, drinking severity, presence of alcohol withdrawal, and diagnostic comorbidity.

The occurrence of insomnia and its self-medication with alcohol has not been investigated simultaneously in alcoholic samples (4–6). Nevertheless, other studies provide useful frequency estimates of self-medication. Skoloda et al. (8) reported that 62% of treated alcoholics believed that alcohol helped them sleep. Similarly, Mamdani et al. (9) found that 60% of 92 inpatient males with alcoholism reported hypnotic use of alcohol. These self-medication rates are greater than rates reported in the general population (6% to 13%) and in people with initial insomnia (15% to 28%) (10–12). Perhaps the highest rate of self-medication was reported in a sample of 155 older women (aged ≥ 85 yr) with symptomatic insomnia, in which 70% used alcohol for sleep (13).

Given that insomnia during early recovery has been linked to relapse (8,14–16), the frequency and correlates of insomnia in alcoholics are important areas of investigation. The present study of patients with alcoholism investigated (a) the frequency and clinical correlates of insomnia, (b) the frequency of drinking to self-medicate insomnia, and (c) the relationships between insomnia, self-medication and subsequent relapse. We reasoned that patients with alcoholism who reported insomnia would be more likely to self-medicate insomnia with alcohol, and therefore, more likely to relapse than patients without insomnia. We also hypothesized that self-reported insomnia would be associated with female gender, older age, drinking severity, depression severity, and decrements in sleep continuity as measured by polysomnography (PSG).

METHODS

Subjects

Subjects with DSM-III-R diagnoses of alcohol dependence were recruited to participate in a study examining the effects of aging and alcoholism on sleep (17). Because six patients had missing data on critical sleep items as described below, the sample size for data analyses was reduced from 178 to 172. The recruitment sites and a description of these subjects have been reported previously (14). Briefly, about 50% of subjects were recruited from the inpatient alcohol unit at the Ann Arbor Veterans Affairs Medical Center and 50% were recruited from inpatient and outpatient alcohol treatment programs of the University of Michigan Medical Center. The use of multiple treatment programs for recruitment insured a demographically heterogeneous sample (14).

Subjects were screened for medical and psychiatric conditions that might influence sleep. We excluded individuals with current major depression, a history of psychosis, bipolar disorder, borderline personality disorder, heart disease, severe liver disease, seizure disorder (except seizures related to alcohol withdrawal), degenerative central nervous system disease, cerebrovascular disease, or recent loss of consciousness due to head trauma. Psychiatric diagnoses were determined using a revised edition of the Diagnostic Interview Schedule (DIS-III-R) (18). A past history of major depression, current dysthymic disorder, or anxiety disorders were not cause for exclusion. Patients with antisocial personality disorder were not excluded unless the investigators judged the disorder would interfere with study procedures. Other causes for exclusion included medications that affect sleep: psychotropic medications, centrally-acting antihypertensives, oral corticosteroids, and theophylline. Patients who met DSM-III-R criteria for abuse or dependence on substances other than alcohol were not excluded, as long as they were substance-free (except for nicotine) for two weeks prior to their sleep study. Subjects were excluded if they worked night shifts or intentionally stayed awake during usual bedtime hours.

The protocol for this study was approved by the University of Michigan Medical Center Institutional Review Board. After complete description of the study to the subjects, written informed consent was obtained.

Procedures

The baseline evaluation consisted of a complete medical history, physical examination, psychiatric interview, substance use history, psychosocial history, and a sleep evaluation. Seventy-four (43%) of the original 172 subjects completed follow-up interviews either by telephone or in person, a mean of five months after baseline. Research assistants, who were blind to the results of subjects' sleep evaluations, conducted the follow-up interviews to assess whether subjects reported any alcohol use during the interim period, defined as a relapse for the purpose of this study.

At baseline, subjects underwent one or more nights of nocturnal PSG at the University of Michigan Sleep Laboratory a minimum of two weeks after admission to a treatment center and their last drink. In some cases, subjects were studied from their hospital rooms, which were connected by telemetry to the Sleep Laboratory. The mean time period between PSG and the patient's last drink was 31.5 (S.D. 15.5) days. Only data from the first night of sleep monitoring were used for this report, because not all subjects completed more than one night of study.

Sleep was monitored using standard polysomnographic techniques, including an electroencephalogram (EEG: C3/A2) and electrooculogram. Data were digitized using a bedside portable computer and then transmitted to sleep analyzing computers in the control room. The EEG channel was digitally filtered to yield a nominal band pass of 0.1 to 30 Hz. Data were displayed at a rate of 10mm/sec as a virtual polygraph page on a high-resolution monitor and stored to a hard disk at 250 samples/sec.

Records were masked to remove identifying information so that scorers were blind to diagnostic and treatment status, then manually scored using 1-min epochs (19). Scoring for sleep latency was defined in this study as the time from start of recording to onset of stage 2, 3, 4 or rapid eye movement (REM) sleep with a duration of at least 10 min and with no more than 2 min of Stage 1 or one min of stage 1 plus 1 min of wakefulness. Slow wave sleep (SWS) was calculated by adding the times spent in stage 3 and stage 4 sleep. Corrected REM sleep latency was defined as the time between sleep onset and the first REM period (at least 3 min of REM sleep within 30 min of each other) minus intermittent wakefulness during that interval.

Instruments and Measures

The *Sleep Disorders Questionnaire* (SDQ) is a 175-item, self-administered instrument that was given at baseline to measure subjective sleep quality and complaints related to nocturnal breathing disturbance, leg movements, daytime sleepiness, and psychiatric distress (20). Subjects responded to individual questions about the past 6 months (such as "I have trouble getting to sleep at night") by choosing among five Likert-scaled response categories: 1 - Never or Strongly Disagree, 2 - Rarely or Disagree, 3 - Sometimes or Not Sure, 4- Usually or Agree, and 5 - Always or Agree Strongly. We classified subjects at baseline with insomnia if they scored a 4 or 5 on any one of eight items from the SDQ that we judged *a priori* to reflect complaints of insomnia (Table 1). Another SDQ question, "I often use alcohol in order to get to sleep" (item 105), was used as the measure of self-medication. If a subject scored 4 or 5 on this question, then that subject met the threshold for self-medication.

The DIS-III-R (18) yielded a lifetime symptom count for alcohol dependence (maximum = 9), which was used as a measure of diagnostic severity. The *Michigan Alcoholism Screening Test* provided another index of lifetime drinking problem severity (21). The *Carroll Depression Scale* (22) assessed self-reported depressive symptoms at baseline. Higher scores indicate greater severity of depression. At follow-up, drinking was determined by self-report, which is generally considered valid when conditions of trust and lack of negative consequences for relapse are established (23).

Data Analyses

The rates of insomnia and drinking to self-medicate insomnia were expressed as percentages of the total sample (N=172). The sample was then divided into two groups of patients according to the presence or absence of insomnia, and differences between groups were tested by chi square (χ^2) for dichotomous variables and by t tests for continuous variables. Continuous variables are expressed as means $\pm$ S.D. Non-normally distributed continuous variables were transformed to approximate a normal distribution prior to testing. Many variables can be generated and analyzed from PSG, which can lead to type I errors when comparing groups on all such measures. To minimize the risk of type I error, the number of tests run were restricted to variables for which there were *a priori* hypotheses. Three measures of sleep continuity (sleep latency, wake time after sleep onset [WASO], and sleep efficiency) were hypothesized to be worse for patients *with* than *without* self-reported insomnia. Percentage of total sleep time spent in slow wave sleep (SWS%), percentage of time spent in rapid eye movement (REM%) sleep, and corrected REM sleep latency were compared between patients with and without insomnia, because these variables have been associated with alcoholic relapse in other studies (14,24–26). We hypothesized that patients with insomnia would have decreased SWS%, increased REM%, and decreased corrected REM sleep latency. All tests were two-tailed, and alpha was set at 0.05. For analyses of the follow-up group (N=74), we used univariate analyses to compare the followed and not followed groups on baseline variables. Any differences between the two groups were entered as covariates, using logistic regression analyses to predict relapse in the follow-up group. Collinearity was evaluated using the tolerance statistic that ranges from 0 to 1, with lower values indicating more collinearity (27). For example, a tolerance value of 0.25 indicates that 75% of a variable's variance can be explained by other entered variables. Tolerance values > 0.6 indicated acceptable levels of collinearity.

RESULTS

The percentage of subjects scoring positive on each of the eight insomnia-related SDQ questions appear in Table 1. Using the combination of eight items from the SDQ, 104 (60.5%) of 172 alcoholic patients were classified as having insomnia. Patients classified with insomnia had significantly longer sleep latency times and lower sleep efficiency values on PSG than patients without insomnia (Table 2). Although not significantly different, the insomnia group had higher WASO values than the group without insomnia.

Patients with insomnia scored higher on both measures of alcohol severity (DSM-III-R symptom count and MAST score) and depression severity than did patients without insomnia (Table 2). No significant differences were found for demographic variables. Patients with insomnia were more likely to report using alcohol often for sleep than patients without insomnia (54.8% vs. 27.9%; $\chi^2 = 12.03$, $df = 1$, $p = 0.001$). Altogether, 76 (44.2%) of 172 patients responded that they often used alcohol to get to sleep. We found no significant differences between groups in REM%, SWS%, or corrected REM sleep latency values, although patients with insomnia tended to have shorter corrected REM sleep latency values than patients without insomnia (61.2 ± 47.5 vs. 75.3 ± 47.0 min, $t=1.91$, $df=170$, $p=0.058$).

A total of 36 (48.6%) of the 74 followed patients relapsed. Because the follow-up rate was only 43%, we compared followed (N=74) and non-followed (N=98) patients on baseline characteristics. We found no significant differences between followed and non-followed patients in terms of insomnia rates, age, gender, race, marital status, education, number of DSM-III-R alcohol dependence symptoms, MAST scores, sleep efficiency, sleep latency, SWS%, REM%, corrected REM sleep latency, or recruitment site. Followed patients had higher WASO values than non-followed patients (38.1 ± 34.7 vs. 28.5 ± 25.0 min, $t=-2.01$, $df=126.7$, $p=0.046$) and also had higher depression scores (9.0 ± 7.8 vs. 6.9 ± 5.5 , $t=-1.96$, $df=125.8$, $p=0.052$). Therefore, these two variables were included in the regression analyses below.

Followed patients with baseline insomnia were more likely to relapse (59.6%) than followed patients without baseline insomnia (29.6%) ($\chi^2 = 6.16$, $df = 1$, $p = 0.02$). Patients with and without insomnia had nearly identical follow-up durations (142.9 ± 54.6 vs. 142.4 ± 57.1 d; $t=-0.04$, $df=72$, $p=0.97$), so they had similar risk periods for relapse. To determine the effect of insomnia on relapse after controlling for severity of alcohol dependence (MAST score and DSM-III-R alcohol dependence symptom count), depression severity (Carroll score), and WASO, we employed a logistic regression analysis in which all variables were forced to stay in the model. The sample size for this analysis was N=70, because of missing variables for four patients. We found that baseline insomnia was the only significant predictor of relapse (Wald=4.90, $df=1$, $p=0.027$). Collinearity was acceptable with tolerance values ranging from 0.64 to 0.92.

Followed patients who used alcohol to self-medicate insomnia had a higher relapse rate (59.5%) than patients who did not self-medicate (37.8%), which approached significance ($\chi^2 = 3.46$, $df=1$, $p=0.063$). After controlling for drinking severity, depression severity, and WASO using another forced-entry logistic regression analysis, the self-medication variable remained non-significant (Wald = 2.70, $df=1$, $p=0.10$). Again, collinearity statistics were acceptable with tolerance values ranging from 0.74 to 0.94.

DISCUSSION

To our knowledge, the occurrence of symptomatic insomnia and drinking to self-medicate insomnia have not been simultaneously studied in alcoholic patients. Using a measure derived from the SDQ, over 60% of our sample reported high levels of insomnia symptoms during the 6 months prior to treatment. Also, nearly 45% of alcoholic patients reported using alcohol *often* in order to get to sleep during the 6 months prior to treatment. Patients with insomnia were about twice as likely to report using alcohol to sleep as patients without insomnia were (55% vs. 28%). Similar rates of insomnia (4–6) and its self-medication with alcohol (8,9) have been reported in other studies of alcoholic patients in treatment, even though frequency rates across studies will vary according to sample characteristics, time frame, and measures of insomnia. A similar rate of self-medicating insomnia with alcohol among patients with schizophrenia (56%) (28) suggests that some of our findings may generalize to other patient populations as well.

That alcohol-dependent patients use alcohol to self-medicate insomnia is both understandable and maladaptive. On the one hand, if insomnia is a withdrawal symptom, either acute or protracted (6,29), then relief drinking is a likely strategy, especially given that alcohol has a reinforcing effect in individuals with insomnia (30). On the other hand, there is general scientific consensus that both acute and chronic alcohol use disrupt sleep patterns (1–3,31,32). Therefore, self-medication of insomnia with alcohol, even if reinforcing, may paradoxically worsen insomnia. Consequently, a vicious cycle may develop in which alcohol initially makes it easier to fall asleep; but as tolerance to this sedative effect

develops, the sleep-disruptive effects of alcohol become more apparent if not more severe (31). Some patients may persist with self-medication despite worsening insomnia, because their drinking behavior is ingrained and reinforcing, and they feel desperate for sleep.

Severity of both alcohol dependence and depression was significantly associated with insomnia. Because we excluded patients with current major depression, depressive symptoms in this study were most likely related to alcohol, dysthymia, or social stressors. We did not find higher rates of insomnia in women than in men, or as a function of increasing age, contrary to some studies of the general population (33,34) and primary care practices (35). Age and gender differences for insomnia may disappear among patients seeking treatment for alcohol dependence. Although alcohol severity and depression severity were significantly associated with insomnia, we did not measure anxiety symptoms despite recent studies that support a significant relationship between anxiety and sleep disturbance in alcoholic patients (36,37). Therefore, further studies are needed before making more than tentative conclusions about the correlates of insomnia among alcoholic patients.

Consistent with other studies (8,14–16), baseline insomnia predicted relapse to alcohol. Baseline insomnia remained a predictor of relapse even after controlling for severity of alcohol dependence and depressive symptoms. Contrary to our hypothesis, patients with a history of self-medicating insomnia with alcohol were not significantly more likely to relapse than non-medicating patients.

Of the eight insomnia items, “I feel that I have insomnia” and “I have been unable to sleep at all for several days” were least sensitive, with only 6% and 4% of the sample endorsing these respective items (Table 1). Other insomnia items were endorsed by 18% to 32% of the sample. This suggests that patients who report symptoms of insomnia do not necessarily think of themselves as having insomnia. Asking patients if they have insomnia may be analogous to asking them if they are alcoholic; patients may avoid these labels because of denial, stigma, their own definitions of these terms which exclude themselves, and/or fear of treatment implications. Not being able to sleep at all for several days is uncharacteristic of alcohol dependence except for extreme cases of alcohol withdrawal, such as delirium tremens that occur in only about 5% of alcohol-dependent patients (38). This may explain the insensitivity of that question. Future studies should address the optimal screening questions for insomnia among alcoholic patients.

Several methodological issues may limit the interpretation of results. First, we studied alcoholic patients who underwent PSG for research purposes. It is possible that alcoholics in treatment who have sleep complaints are very likely to volunteer for sleep studies, thus skewing our frequency rates for insomnia and its self-medication with alcohol. However, our rates of insomnia were comparable in range to other studies (4–6), and provide further evidence of the frequency of these phenomena. Nevertheless, many patients may have entered the study because of their sleep problems, which could limit the generalizability of these results.

Second, our eight-item measure of insomnia was derived from a standardized and validated sleep questionnaire (20), but full psychometric testing of the abbreviated questionnaire was beyond the scope of this article. For example, we did not calculate the sensitivity and specificity of the eight-item insomnia measure, because we did not conduct standardized clinical interviews for insomnia, and PSG by itself is not a gold standard for the evaluation of insomnia (39,40). Nevertheless, in addition to face validity, the insomnia measure was associated with drinking and depression severity, with polysomnographic measures of sleep fragmentation, and with relapse, providing evidence of concurrent, external, and predictive validity, respectively.

Third, only 43% of patients were followed over time. The low follow-up rate reflects the fact that longitudinal outcomes were added as a secondary area of interest after starting our primary investigations on the effects of alcoholism and aging on sleep abnormalities (17). It is true that followed and not followed patients did not differ on any baseline variables except depression and WASO; and when these two variables were entered into the logistic regression analysis, insomnia remained a robust predictor of relapse. Nevertheless, the low follow-up rate remains a limitation, because patients lost to follow-up have potentially higher relapse rates than followed patients. Fourth, only one night of sleep was recorded, which did not allow subjects to adjust to the “first night effect” of sleeping under novel conditions. Fifth, relapse status was determined solely by self-report without biochemical or other corroboration, such as by a friend or family member. Still, whatever self-report bias occurred was expected to be similar across comparison groups.

In summary, symptoms of chronic insomnia and the use of alcohol to aid sleep were prevalent in alcoholic patients. Insomnia was significantly associated with severity of alcohol dependence, depressive symptoms, and polysomnographic measures of poor sleep continuity; and it was predictive of drinking during the follow-up period. These results suggest that alcoholic patients at risk for relapse are easily identifiable by routine questions about sleep. The potential for improved drinking outcomes by treating insomnia in alcoholic patients is now being investigated (41–43).

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References

1. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and psychiatric disorders: a meta-analysis. *Arch Gen Psychiatry* 1992;49:651–668. [PubMed: 1386215]
2. National Institute on Alcohol Abuse and Alcoholism: Alcohol and sleep. *Alcohol Alert* 1998;41:1–4.
3. Vitiello MV. Sleep, alcohol, and alcohol abuse. *Addiction Biology* 1997;2:151–158.
4. Baekeland F, Lundwall L, Shanahan TJ, Kissin B. Clinical correlates of reported sleep disturbance in alcoholics. *Q J Stud Alcohol* 1974;35:1230–1241. [PubMed: 4445463]
5. Feuerlein W. The acute alcohol withdrawal syndrome: findings and problems. *Br J Addict* 1974;69:141–148.
6. Caetano R, Clark CL, Greenfield TK. Prevalence, trends, and incidence of alcohol withdrawal symptoms: analysis of general population and clinical samples. *Alcohol Health Res World* 1998;22(1):73–79. [PubMed: 15706737]
7. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment: prevalence and correlates. *Arch Gen Psychiatry* 1985;42:225–232. [PubMed: 2858188]
8. Skoloda, TE.; Alterman, AI.; Gottheil, E. Sleep quality reported by drinking and non-drinking alcoholics. In: Gottheil, EL., editor. *Addiction Research and Treatment: Converging Trends*. Elmsford, NY: Pergamon; 1979.
9. Mamdani MB, Hollyfield RL, Ravi SD, Dorus W, Borge GF. Sleep complaints and recidivism in alcoholics reporting use of alcohol as a hypnotic (abstract). *Sleep Research* 1988;17:293.
10. Johnson EO, Roehrs T, Roth T, Breslau N. Epidemiology of alcohol and medication as aids to sleep in early adulthood. *Sleep* 1998;21:178–186. [PubMed: 9542801]
11. Ancoli-Israel S, Roth T. Characteristics of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. *I Sleep* 1999;22 (suppl 2):S347–S353.

12. Walsh J, Ustun TB. Prevalence and health consequences of insomnia. *Sleep* 1999;22(suppl 3):S427–S436. S446–S450.
13. Johnson JE. Insomnia, alcohol, and over-the-counter drug use in old-old urban women. *J Community Health Nurs* 1997;14:181–188. [PubMed: 9282538]
14. Brower KJ, Aldrich MS, Hall JM. Polysomnographic and subjective sleep predictors of alcoholic relapse. *Alcohol Clin Exp Res* 1998;22:1864–1871. [PubMed: 9835309]
15. Drummond SPA, Gillin JC, Smith TL, DeModena A. The sleep of abstinent pure primary alcoholic patients: natural course and relationship to relapse. *Alcohol Clin Exp Res* 1998;22:1796–1802. [PubMed: 9835298]
16. Foster JH, Peters TJ. Impaired sleep in alcohol misusers and dependent alcoholics and the impact upon outcome. *Alcohol Clin Exp Res* 1999;23:1044–1051. [PubMed: 10397289]
17. Aldrich MS, Brower KJ, Hall JM. Sleep-disordered breathing in alcoholics. *Alcohol Clin Exp Res* 1999;23:134–140. [PubMed: 10029214]
18. Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of Mental Health Diagnostic Interview Schedule: its history, characteristics, and validity. *Arch Gen Psychiatry* 1981;38:381–389. [PubMed: 6260053]
19. Rechtschaffen, A.; Kales, AA. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Washington, DC: Government Printing Office; 1968. NIH Publication No. 204
20. Douglass AB, Bornstein R, Nino-Murcia G, Keenan S, Miles L, Zarcone V Jr, Guilleminault C, Dement WC. The Sleep Disorders Questionnaire. I: creation and multivariate structure of SDQ. *Sleep* 1994;17(2):160–167. [PubMed: 8036370]
21. Selzer ML. The Michigan Alcoholism Screening Test (MAST): the quest for a new diagnostic instrument. *Am J Psychiatry* 1971;127:1653–1658. [PubMed: 5565851]
22. Carroll BJ, Feinberg M, Smouse PE, Rawson SG, Greden JF. The Carroll rating scale for depression. I. Development, reliability and validation. *Br J Psychiatry* 1981;138:194–200. [PubMed: 7272609]
23. Babor TF, Stephens RS, Marlatt GA. Verbal report methods in clinical research on alcoholism: response bias and its minimization. *J Stud Alcohol* 1987;48:410–424. [PubMed: 3312821]
24. Gillin JC, Smith TL, Irwin M, Butters N, Demodena A, Schuckit M. Increased pressure for rapid eye movement sleep at time of hospital admission predicts relapse in nondepressed patients with primary alcoholism at 3-month follow-up. *Arch Gen Psychiatry* 1994;51:189–197. [PubMed: 8122956]
25. Allen RP, Wagman AMI, Funderburk FR. Slow wave sleep changes: alcohol tolerance and treatment implications. *Adv Exp Med Bio* 1977;85A:629–640. [PubMed: 920496]
26. Clark CP, Gillin JC, Golshan S, Demodena A, Smith TL, Danowski S, Irwin M, Schuckit M. Increased REM sleep density at admission predicts relapse by three months in primary alcoholics with a lifetime diagnosis of secondary depression. *Biol Psychiatry* 1998;43:601–607. [PubMed: 9564445]
27. SPSS Inc. SPSS Base 10.0 Applications Guide. Chicago, IL: Author; 1999. p. 221
28. Noordsy DL, Drake RE, Teague GB, Osher FC, Hurlbut SC, Beaudett MS, Paskus TS. Subjective experiences related to alcohol use among schizophrenics. *J Nerv Ment Dis* 1991;179:410–414. [PubMed: 1869869]
29. Satel SL, Kosten TR, Schuckit MA, Fischman MW. Should protracted withdrawal from drugs be included in DSM-IV? *Am J Psychiatry* 1993;150:695–704. [PubMed: 8097618]
30. Roehrs T, Papineau K, Rosenthal L, Roth T. Ethanol as a hypnotic in insomniacs: self administration and effects on sleep and mood. *Neuropsychopharmacology* 1999;20:279–286. [PubMed: 10063488]
31. Aldrich, M. Effects of alcohol on sleep. In: Gomberg, ESL.; Hegedus, AM.; Zucker, RA.; Bethesda, MD., editors. *Alcohol Problems and Aging* 33. Vol. 33. 1998. National Institute on Alcohol Abuse and Alcoholism Research Monograph Series(NIH Publication No. 98–4163)
32. Castaneda R, Sussman N, Levy R, O'Malley M, Westreich L. A review of the effects of moderate alcohol intake on psychiatric and sleep disorders. *Rec Dev Alcohol* 1998;14:197–226.

33. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbance and psychiatric disorders. JAMA 1989;262:1479–1484. [PubMed: 2769898]
34. Costa e Silva JA, Chase M, Sartorius N, Roth T. An overview of insomnias and related disorders--recognition, epidemiology, and rational management. Sleep 1996;19:412–416. [PubMed: 8843532]
35. Hohagen F, Rink K, Kappler C, Schramm E, Riemann D, Weyerer S, Berger M. Prevalence and treatment of insomnia in general practice. Eur Arch Psychiatry Clin Neurosci 1993;242:329–336. [PubMed: 8323982]
36. Mackenzie A, Funderburk FR, Allen RP. Sleep, anxiety, and depression in abstinent and drinking alcoholics. Substance Use & Misuse 1999;34:347–361. [PubMed: 10082061]
37. Robinson EAR, Cardinez MMV, Brower KJ. Anxiety symptoms predict initial insomnia among alcoholic outpatients (abstract). Alcohol Clin Exp Res 2000;24(suppl 5):144A.
38. Hersh D, Kranzler HR, Meyer RE. Persistent delirium following cessation of heavy alcohol consumption: diagnostic and treatment implications. Am J Psychiatry 1997;154:846–851. [PubMed: 9167514]
39. Reite M, Buysse D, Reynolds C, Mendelson W. The use of polysomnography in the evaluation of insomnia. Sleep 1995;18:58–70. [PubMed: 7761745]
40. Chervin, RD. Use of clinical tools and tests in sleep medicine. In: Kryger, MH.; Roth, T.; Dement, WC., editors. Principles and Practice of Sleep Medicine. 3. Philadelphia: Saunders; 2000.
41. Karam-Hage M, Brower KJ. Gabapentin treatment for insomnia associated with alcohol dependence (letter). Am J Psychiatry 2000;157:151. [PubMed: 10618048]
42. Longo LP, Johnson B. Treatment of insomnia in substance abusing patients. Psychiatric Annals 1998;28:154–159.
43. Greeff AP, Conradie WS. Use of progressive relaxation training for chronic alcoholics with insomnia. Psychol Rep 1998;82:407–412. [PubMed: 9621711]

Table 1Percentage of Sample Endorsing Individual Insomnia Items ($N = 172$)

Sleep Disorders Questionnaire (20) Item ^a	Percent
I get too little sleep at night (item 1)	30.2
I often have a poor night's sleep (item 2)	25.6
I have trouble getting to sleep at night (item 3)	30.2
I wake up often during the night (item 4)	31.4
I have been unable to sleep <u>at all</u> for several days (item 43)	3.5
I feel that my sleep is abnormal (item 44)	18.0
I feel that I have insomnia (item 45)	6.4
I have a problem with my sleep (item 87)	23.3
Any one of the above	60.5

^aPatients who answered the questionnaire item with a Likert response category of 4 or 5 were scored as positive for that item (see text).

Table 2Characteristics of Patients with Alcoholism at Baseline^a

Variable	With Insomnia	Without Insomnia	Comparison ^c	
	(N = 104) ^b	(N = 68) ^b	χ^2 or t	df
Age (yr)	41.5 ± 11.78	39.1 ± 9.7	-1.45	170
Male (%)	86.5	82.4	<i>0.56</i>	1
Caucasian (%)	81.0	88.2	<i>1.61</i>	1
Married (%)	27.6	32.8	<i>0.51</i>	1
Education (yr)	12.8 ± 1.8	13.3 ± 1.6	1.93	157
DSM-III-R Alcohol Symptoms (#)	7.8 ± 1.6	7.2 ± 1.5	-2.16*	159
MAST score	42.8 ± 10.6	39.4 ± 10.5	-2.10*	168
Depression Score	10.3 ± 7.1	4.1 ± 3.6	-7.53***	158.9
Often Uses Alcohol to Sleep (%)	54.8	27.9	<i>12.03***</i>	1
Sleep Latency (min) ^d	41.8 ± 37.1	29.8 ± 28.5	-2.18*	170
Mean Sleep Efficiency (%)	77.6 ± 12.4	83.1 ± 10.2	3.13**	160.9
WASO (min)	34.7 ± 31.9	29.5 ± 26.5	-1.10	170
Mean Slow Wave Sleep (%)	8.1 ± 9.2	9.0 ± 9.3	0.66	170
Mean REM Sleep (%)	20.3 ± 7.1	20.5 ± 6.3	0.16	170
Corrected REM sleep latency (min)	61.2 ± 47.5	75.3 ± 47.0	1.91	170

^aMeans ± standard deviations are shown for continuous variables. Percentages are shown for dichotomous variables.^bSample sizes may differ for some variables due to missing data.^cDichotomous and continuous variables were analyzed by two-tailed chi square (χ^2) tests (values italicized) and t tests, respectively. Degrees of freedom (df) are in decimal form when t tests with unequal variances were used.^dFor sleep latency, testing was performed on log transformed values not shown in the table.

* p < 0.05

** p < 0.01

*** p ≤ 0.001