Published in final edited form as:
Subst Abus. 2005 March ; 26(1): 1–13.

Disturbed Sleep and Its Relationship to Alcohol Use

Michael D. Stein, MD and **Peter D. Friedmann, MD, MPH**
Brown Medical School, Division of General Internal Medicine, Rhode Island Hospital, 593 Eddy Street, Providence, RI 02903

Abstract

Study Objectives—To review evidence of an association between disturbed sleep and alcohol use.

Design—We searched MEDLINE, PSYCHINFO, ETOH, BIBLIOSLEEP and the Rutgers Alcohol Studies databases between January 1966 and August 2002. Search terms included *alcohol-related disorders or alcoholism* in combination with *sleep, sleep initiation and maintenance disorders, or sleep apnea syndromes.* The search produced over 440 citations. We reviewed 107 relevant articles, of which 60 included quantitative measures of both alcohol use and sleep.

Measurements and Results—Behavioral studies suggest that up to 2 to 3 standard drinks before bedtime initially promotes sleep, but these effects diminish in as few as 3 days of continued use. Clinical investigations support a relationship between sleep disturbance and alcohol use, but variability in the definition and measurement of these domains and a preponderance of cross-sectional studies make uncertain the strength and direction of the association.

Conclusions—The association of insomnia with alcohol use disorders suggests that the clinical evaluation of patients with sleep problems should include a careful assessment of alcohol use. Future studies of this relationship should employ prospective designs with standardized, validated measures of both sleep and alcohol use. Rigorous treatment studies for chronic insomnia in alcohol dependent patients are also needed.

Keywords

Disturbed sleep; alcohol use; insomnia

INTRODUCTION

Disturbed sleep is a common complaint in medical settings. For example, in the Medical Outcomes Study, 16% of persons reported severe insomnia and 34% reported mild insomnia over the prior 4 weeks, and a substantial proportion of the remaining 50% of the sample reported symptoms of sleep disturbance (1). At two-year follow-up, 59% of persons with mild insomnia and 83% of patients with severe insomnia still had sleep problems. Insomnia has been associated with subsequent morbidity and mortality, in addition to causing or worsening somatic symptoms that contribute to quality-of-life decline (1–3).

Address correspondence to: Peter D. Friedmann, MD, MPH, Division of General Internal Medicine, Rhode Island Hospital, 593 Eddy Street, Providence, RI 02903 (pfriedmann@lifespan.org).

Chronic insomnia is generally defined as difficulty initiating or maintaining sleep for a period longer than three weeks. Briefer periods of sleep disturbance are most often stress-related or due to acute illness (4). Insomnia frequently has a mix of contributing causes, and clinicians need to assess psychological (e.g., depression and anxiety), medical (e.g., pain and obstructive sleep apnea), and lifestyle-related (e.g., caffeine consumption) risk factors (5,6). Substance use problems underlie approximately 10–15% of chronic insomnia (7). Of adult Americans, as many as 70% drink alcohol, and half of these experience an alcohol-related problem at some point in their lives. These problems are likely to be more prevalent among the 10% of Americans who drink alcohol daily (8,9).

Historically, alcohol has been used as a sedative (7). However, recognition of the complexities of the relationship between alcohol and insomnia is important for several reasons. Sleep disturbance may be a sign of alcohol abuse or dependence. As such, clinical alertness to insomnia as a symptom of alcohol problems might facilitate timely intervention. Sleep disturbance is common among patients in remission from alcohol use disorders, and understanding this relationship may help clinicians assist patients in recovery. Recognition of alcohol problems among insomniacs might also lead clinicians to alter their treatment of sleep complaints, limiting, for example, their use of sedative-hypnotic agents.

An estimated 10 million people consult health care practitioners for sleep disorders. Our goal is to assist providers in recognizing the clinical significance of alcohol use in persons who also have sleep disturbance. Therefore, this review outlines: (1) alcohol's effect on normal sleep, and the response to its use by persons with sleep disturbance; (2) the epidemiological evidence linking alcohol use and sleep disorders; (3) the mechanisms whereby alcohol disturbs sleep; and (4) the association of alcohol use, psychiatric disorders, and sleep.

METHODS

Selection of Studies

Searches of the following databases identified studies that examined the relationship between alcohol consumption and sleep disturbance: MEDLINE (through PUBMED January 1966 to August 2002); PSYCHINFO (1967 through 2002); National Institute on Alcohol Abuse and Alcoholism ETOH database (1981–2002); BIBLIOSLEEP (1990–2001); and the Rutgers Alcohol Studies Database (1989–2001). The PUBMED search used the keywords *alcohol-related disorders, alcohol intoxication, or alcoholism* in combination with *sleep, sleep initiation and maintenance disorders, sleep apnea syndromes, sleep stages, or polysomnography*, limited to the following study types: *randomized controlled trials, meta-analysis, or epidemiologic studies*; this strategy yielded 54 articles. The PSYCHINFO search used the keywords alcohol abuse, alcoholism, alcohol intoxication (acute or chronic), alcohol withdrawal, alcohol drinking attitudes, alcoholic beverages, alcoholic psychosis, sobriety or alcohol rehabilitation, in combination with *sleep, sleep disorders, insomnia, sleep apnea, sleep onset, REM sleep, NREM sleep, sleep wake cycle, sleep treatment or hypnotic drugs*, limited to the following study types: *randomized clinical trials, meta-analysis, cohort studies, case-control studies, review or guideline*, this strategy yielded 41 articles. The ETOH search used the keywords *sleep or sleep disorders*, limited to the following study types: *randomized controlled trials, case-control studies, meta-analysis, prospective studies or literature reviews and guidelines*; this strategy yielded 89 articles. The BIBLIOSLEEP search used the keywords *alcohol, alcoholic, or alcoholism, limited* to the following study types: *randomized, case-control, cohort or prospective studies*; this strategy yielded 79 articles. The Rutgers Alcohol Studies Database search used the keywords *sleep, insomnia, dyssomnia, polysomnograph, dream, circadian, or somnolence*, limited to this strategy yielded 177 articles. All searches were limited to human subjects and English language. We searched the reference lists of published trials, overviews and book chapters for additional studies. After elimination of

duplicates, we reviewed 107 relevant articles. We then excluded 11 articles focused on treatment. Of the remaining articles, 60 were clinical investigations with quantitative measures of both alcohol use and sleep. One author read each article and constructed tables. A second author checked the tables for accuracy. Consensus resolved discrepancies. This article presents a qualitative review because extreme heterogeneity in study design, methods of measurement and quality precluded the performance of meta-analysis.

RESULTS

Alcohol's Effect on Normal Sleep

Sleep consists of two states: rapid eye movement (REM) and non-REM sleep (NREM) (10). Non-REM sleep is divided into four stages. Stage 1 is the lightest stage, from which it is easiest to arouse the sleeper and stage 2 is intermediate. Stages 3 and 4 are referred to as slow wave sleep or delta sleep per electroencephalogram (EEG). Sleep typically begins with stage 1 sleep, and progresses to deeper stages of NREM sleep when entry into the first period of REM sleep occurs. REM and NREM sleep then alternate in approximately 90-minute cycles. The states of sleep are homeostatically regulated such that deprivation of REM sleep leads to pressure to enter REM and is accompanied by a rebound increase in REM once begun. Similar effects are observed after slow wave sleep deprivation.

Alcohol is commonly used as a sleep aid–that is, an agent for initiating sleep. Alcohol consumed in the evening has generally predictable effects on REM sleep, slow wave sleep, and sleep time and continuity, but effects on sleep latency (time to fall asleep) are more variable (Table 1). Sleep researchers have performed experiments with healthy non-alcoholic subjects using alcohol doses ranging from 0.16 to 1.0 g/kg (one to three ounces), yielding breath alcohol concentrations as high as 105 mg percent (11). Alcohol's inhibition of REM sleep has been observed since the 1960s (12) in normal subjects who ingested high doses (1g/kg) within an hour of bedtime, although these REM inhibition effects are less consistent with lower alcohol doses. REM reduction becomes less pronounced with continued alcohol use, but a REM rebound often appears with alcohol cessation. Slow wave sleep increases after moderate to high bedtime alcohol use, but the effect of lower doses on slow wave sleep are again less certain. Slow wave sleep effects diminish with repeated nights of alcohol consumption (13). The effects of alcohol on sleep continuity and total sleep are quite variable but appear to be dose related. Lower doses may increase total sleep time, whereas higher doses may lead to short-term withdrawal, increasing sympathetic activity and sleep disruption especially during the second half of the night.

Alcohol can have either a stimulating effect that increases sleep latency (time to fall asleep) or a sedating effect that induces sleep, depending on dose and the interval from drinking to bedtime. Stimulating effects are noted at low doses and as blood alcohol levels rise, usually in the first hour after use. In contrast, sedating effects occur at high doses and as blood levels fall (14–17). Interestingly, few studies have focused on the stimulant properties of alcohol, which may not only relate to insomnia but to the vulnerability to alcohol use problems over time. Late afternoon ("happy hour") drinking, as much as six hours before bedtime, also disrupts sleep, even though alcohol is no longer in the brain at bedtime (18). This phenomenon suggests a relatively long-lasting change in sleep regulation.

Alcohol's sedating effects are dose dependent for moderate consumption (0.4–0.8 g/kg; 2–3 drinks; a standard drink is considered 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits, each contains approximately 0.5 ounces of alcohol) and last for several hours (16,19). With increasing amounts, up to six drinks, sleep latency generally decreases. As with other short-acting sedatives, rebound occurs and arousal is heightened 2–3 hours after blood alcohol concentrations fall close to zero. Clinically, arousals increase in association with

increased catecholamine concentration later in the night. Metabolized at a rate of approximately one glass of wine per hour, after 5 drinks at 10 p.m., the alcohol level will be near zero at 3 a.m., with an increase in arousal from this time onwards. REM rebound occurs in the second half of the night associated with intensive dreaming or nightmares. These effects contribute to sleep fragmentation. Tolerance to alcohol's effect on sleep architecture develops, with a normalization of polysomnographic changes after 3–9 nights of use in normal subjects (13, 20). Tolerance to the sedative effects occurs after 3 to 7 days of exposure (7). However, little is known about the long-term effects of moderate alcohol consumption on sleep in persons without alcohol dependence.

Alcohol's Effect on Insomnia

Alcohol is perceived to be a convenient, cost-effective and low risk hypnotic, and two studies have addressed such self-treatment in general population samples (21,22). In these studies 15–28% of subjects used alcohol to help them sleep. Two-thirds of those who use alcohol for sleep do so for less than a week at a time, but 15% used alcohol for more than 4 weeks (22). Males are 1.37 times as likely as females to use alcohol as sleep aid. Each higher quartile in level of difficulty falling asleep was increasingly associated with alcohol use for sleep (22). In another study, 67% of persons who complained of insomnia and reported using alcohol to help them sleep felt it was effective (23). Those who used alcohol as a sleep aid had a higher mean daytime sleepiness after adjusting for level of insomnia, total sleep time and sociodemographic factors. This finding is consistent with laboratory studies that report alcohol quickly loses its effectiveness as a hypnotic, while retaining its sleep disturbing properties.

While experts suggest that hypnotic effects of bedtime alcohol decrease with chronic use and may lead to greater late night sleep disturbance, only one study has characterized the effects of alcohol on the sleep of persons with insomnia (11). This report included twenty subjects, 11 with disturbed sleep for at least a year, and 9 controls, who were moderate social drinkers (< 14 drinks/week) and reported no other drug use. Subjects completed assessments of sleep and mood after controlled drinking over multiple nights. Persons who reported previous use of alcohol as a hypnotic had higher nightly laboratory self-administration. Moderate alcohol doses (0.45 g/kg) suppressed REM in both controls and insomniacs. No consistent changes in sleep latency or sleep time were detected, although some improvements in sleep architecture (increased stages 3 and 4, and decreased stage 1) were noted among insomniacs. There was no second half of the night disruption of sleep in normals as had been reported with higher doses of alcohol in other studies, nor were there disturbances in insomniacs' sleep. Insomniacs did report that consuming alcohol before sleep improved their mood; this affective response may reinforce alcohol's use. Nonetheless, the effects of repeated nightly alcohol use both on sleep and on continued drinking among insomniacs remains of concern given past data on the development of tolerance within a week of repeated ingestion.

In general, persons who are partially sleep-deprived (e.g., insomniacs) show greater sedation soon after alcohol consumption than those without sleep deficits. However even among the well rested, reduction in alertness enough to impair performance occurs in the morning after evening drinking (24). Impairment in reaction time and performance persist for several hours after blood levels drop to zero (24). People who use alcohol as a sleep aid are more tired and show lower daytime alertness than people who abstain from alcohol at night (19). Alcohol intake prior to sleep is associated with a greater risk of severe daytime sleepiness (OR 2.1; 95% CI 1.4–3.0) (25). Thus sleep deprivation with even low-dose alcohol can place an individual at high risk for accidents on the road, at work and at home (19,24,26). As little as one ounce of alcohol in sleep-deprived persons can increase the risk of accidents (16). Most worrisome is moderate alcohol use among chronically sleep-deprived populations such as shift workers and young adults who are at high risk for falling asleep while driving.

The intensity of insomnia will dictate whether an individual seeks treatment with either prescription or over-the-counter medications, or self-treatment. The majority of persons with sleep difficulty do not consult their physician with insomnia, which raises the question of how frequently alternative substances are used for sleep (6). Both insomnia and sleeping pill use increase with age, and alcohol is often used in conjunction with over-the-counter sleep medications (27,28).

Sleep Disorders and Alcohol Use Disorders in Epidemiological and Clinical Studies

Most epidemiological and clinical studies suggest a relationship between alcohol use disorders and insomnia (Table 2). In the Epidemiological Catchment Area study (N = 7954), the prevalence of chronic insomnia (defined as two or more weeks with trouble falling or staying asleep or waking too early in the prior six months, not always associated with alcohol use) was 10.2% (6). Alcohol abuse disorders (DSM-III diagnosed) were significantly increased among those with insomnia (OR 2.4; 95% CI 1.0–6.1), although these figures were likely underestimates because the definition of insomnia required that it not be solely related to alcohol use. In the six other community insomnia surveys (sample sizes 200–3445) that included alcohol use as a covariate in their analyses (Table 2), the prevalence of insomnia was 13–53%. Several studies did not detect a significant relationship between alcohol use and insomnia (1,2,29).

Lack of standard definitions and measurements of both insomnia and alcohol use render uncertain the magnitude of any association. For insomnia, questions and their time frame (e.g., past month vs. last 12 months) vary. Some studies have defined “sleep problems” through self-report (again, over varying periods of time), while other studies have defined “sleep problems” in terms of polysomnographic measures (e.g., latency, percentage of REM sleep). Some self-report measures (e.g., amount) tend to underestimate sleep disturbance while other measures (e.g., time to get to sleep) tend to over-estimate insomnia in comparison to polysomnography (30,31). Many persons with self-reported insomnia do not demonstrate objective sleep abnormalities on polysomnography (10). These definitional and methodological issues make interpretation of the literature difficult.

Measures of alcohol use also vary greatly across studies (Table 2). In an analysis of the Medical Outcomes Study data, the measure of alcohol use was limited to a dichotomous categorical definition–no history of use, past or current use (1). Ford and Kameron used abuse/dependence diagnoses from the DSM to characterize alcohol use; Tachibana et al. (33) reported on days of alcohol use per week; Harma et al. (34) reported on annual consumption; Fabsitz et al. (35) reported the number of drinks consumed in two weeks; and Janson et al. (32) used the CAGE alcohol screening questionnaire. In addition, the preponderance of cross-sectional studies limits conclusions regarding causal direction. Use of standardized definitions and measures of insomnia and alcohol consumption, and prospective designs would improve future investigations.

Among patients with diagnosed alcohol dependence, the rate of sleep disturbance is higher than the general population. The six studies of patients in alcohol treatment reported insomnia rates of 25–72% (36–41) (Table 3). Again, differing definitions and measurement of insomnia and alcohol dependence, as well as varied case mix among the samples, make comparison of these studies difficult.

Among alcohol dependent persons, acute intoxication induces sleep onset, albeit with disruptions in the latter half of the night. Sleep is more severely disturbed during withdrawal and recovery, with longer sleep latency, more arousals, poor sleep efficiency, reduced slow wave sleep and REM rebound (42–45). Cross-sectional studies suggest that for nearly half of alcohol dependent patients sleep disturbance persists for months after last use (46,47), and can

last for 2 years or longer (48,49). Most reports suggest that their sleep improves slowly over time (50,51).

Alcohol-dependent patients with insomnia are more likely to report using alcohol to improve sleep than those with out insomnia(41). If insomnia is a withdrawal symptom, then relief drinking seems a reasonable and reinforcing strategy, though counter productive. A substantial proportion of abstinent alcohol dependent persons describe having been aware that alcohol disturbed their sleep, but that they needed to drink to get to sleep (52). As these individuals become desperate for sleep, alcohol initially makes it easier to fall asleep until sleep disruption develops. Alcohol use perpetuates sleep disturbance, which in turn provokes greater alcohol use. Thus, sleep disturbance during early recovery has been linked to relapse (6), even after controlling for severity of alcohol dependence and depressive symptoms (41). Polysomnographic studies also correlate abnormalities in sleep architecture during abstinence with worse prognosis after alcohol treatment (42,50,53–56).

Alcohol-dependent patients are commonly told to focus on abstinence and sleep improvement will follow. This approach will be unacceptable to many because sleep disturbances can persist despite prolonged abstinence. Therefore, sleep disturbance during early recovery should be monitored closely with careful consideration of both behavioral and pharmacological treatment. Brower et al. reported that patients who report symptoms of insomnia do not necessarily think of themselves as having insomnia, just as alcoholics do not accept labeling (41). Alcoholics at risk for relapse are easily identifiable by routine questions about sleep (4). Waking up often, getting too little sleep, and having trouble getting to sleep are often endorsed. The potential for improving drinking outcomes by treating sleep disturbance is being investigated.

Alcohol's Effect on Sleep Through Other Mechanisms

Sleep-disordered breathing may be an additional contributor to sleep complaints and sleep disruption in heavy drinkers. Even after a single drink normal sleepers can develop snoring and even obstructive sleep apnea (OSA) resulting in oxygen desaturations (7,57). Alcohol relaxes upper airway dilator muscles (decreasing airway patency) increasing nasal and pharyngeal resistance (58,59), and it prolongs the time required to arouse or awaken after an apnea occurs (60). Alcohol also selectively depresses hypoglossal nerve activity and alters carotid body chemoreceptor function.

Alcohol exacerbates sleep-related breathing disorders, and the two to four percent of Americans with OSA are particularly susceptible. Heavy drinkers appear to be at increased risk for OSA, especially if they snore, though even modest amounts of alcohol greatly increase the frequency and severity of apneas among persons with OSA, especially in the first hours of sleep when blood alcohol levels are highest (61–63). The combination of OSA and alcohol increases a person's risk of heart attack, stroke and sudden death (64). Alcohol's worsening of apneic events, increasing sleep disruption and daytime fatigue, can also impair driving and increase rates of motor vehicle accidents. Among OSA subjects who consumed 14 or more drinks per week, self-reports of sleep-related accidents are fivefold higher compared to those who drink lesser amounts (65).

Alcohol impairs sleep other ways as well. Alcohol may increase movement disorders that disturb sleep. Men and women who consume two or more drinks per day had two-to threefold increase in periodic leg movements that fragment sleep (66). Alcohol may also provoke sleepwalking, especially when taken in combination with methylphenidate, diphenhydramine or amitryptiline (67). Alcohol consumption can induce gastritis, esophageal reflux and polyuria that can disrupt sleep. Finally, more frequent awakenings during the second half of the night

(often from thirst and polyuria) may lead to un-steadiness and falls during nighttime trips to the bathroom, particularly among the elderly.

DISCUSSION

Overlap of Alcohol-Related Insomnia with Other Psychiatric Disorders

Ford and Kameron, in an epidemiological study of a large community sample, reported that 7% of persons with insomnia had alcohol abuse compared to 3.8% of persons with no sleep complaint; 40.4% of persons with any psychiatric disorder had insomnia compared to 16.4% of persons with no sleep complaint (6). Sleep disturbance of at least two weeks duration was a significant risk factor for developing alcohol use disorders (OR 2.7; 95% CI 1.4–5.2) over a year of follow-up but this study did not answer whether alcohol abuse had an independent association with insomnia after controlling for depression (6). Only one study has reported that the severity of both alcohol dependence and depressive symptoms were significantly associated with insomnia among alcohol treatment patients (41).

Problem drinkers may suffer from depression and/or anxiety independently of their drinking, or during or after heavy drinking (6,8,34,68–73). Disturbed sleep from alcohol use may be difficult to differentiate from signs and symptoms of psychiatric disorders. Difficulty falling asleep, early morning awakening, fatigue, and decreased concentration complicate the assessment of affective symptoms. Moreover, sleep problems often exacerbate primary psychiatric symptoms (74). For example, even small amounts of alcohol have been associated with the development of anxiety and mood symptoms among asymptomatic persons (75). Alcohol exerts effects on next-day behavior and mood that outlast its blood levels (9,76). Alcohol-induced cognitive disruption may also impair previously successful coping mechanisms. Patients and clinicians might interpret these alterations in alertness, judgement, impulse control, decision-making and mood as symptoms of recurrent primary psychiatric disorders, rather than considering the effects of alcohol.

The sleep of individuals with depressive and/or anxiety disorders resembles the sleep of persons with alcoholism (71). Similar REM sleep changes occur during withdrawal in alcoholics with and without secondary depression (54). Polysomnography is not helpful in differentiating among these disorders or their treatment, and thus should not be considered a primary diagnostic instrument for patients with psychiatric disorders and sleep complaints. Overall, an accurate clinical history is a better indicator of alcohol-related insomnia. Polysomnography may be useful to verify sleep difficulties or to diagnose other sleep pathology such as sleep apnea.

The interactions between alcohol use disorders, affective disorders, and sleep disturbance remain understudied, but it is clear that sleep problems in substance-using patients often have more than one cause. Whether chronic insomnia causes depression, anxiety, or alcoholism, or whether such disorders cause sleep problems, remains unclear. Perhaps longitudinal studies could disentangle whether sleep disturbance is a non-causal indicator of an underlying process that predisposes individuals to affective disorders and/or alcoholism, or whether insomnia has a causal role in the development of these disorders. At present, we can only say that in some patients, alcohol use, psychological and sleep disorders co-exist. Because insomnia is a risk factor for return of depression as well as alcohol relapse (77), prudent practice would advise that patients with sleep problems receive a thorough evaluation for both mental health and substance use disorders.

Treatment of Insomnia

Anestimated 10 million people consult health care practitioners for sleep disorders and of these, half receive prescriptions for sleep medications (4). For those patients with chronic insomnia who have an underlying problem of alcohol abuse, clinicians need to prescribe particularly carefully (78).

Non-pharmacological therapies, such as muscle relaxation, biofeedback, cognitive therapy, stimulus control, acupuncture, yoga, meditation, sleep restrictive therapies, and sleep hygiene techniques, should be considered first (4). Removing stimulant use six hours prior to sleep, and avoiding late meals and naps are simple advice that may be helpful. In progressive relaxation therapy, training muscle tension and breathing are used to create a restful state. Greeff and Conradie reported that 10 sessions of progressive relaxation training in 11 alcoholic men with insomnia showed significant improvement in self-reported sleep quality (79).

Despite non-pharmacological interventions, many alcoholic patients continue to have sleep problems and request pharmacotherapy. Many agents previously used for insomnia are relatively contraindicated because of their interaction with alcohol. Alcohol potentiates the effects of barbiturates and chloral hydrate, which are almost never clinically indicated for insomnia among alcoholics. Though safer, benzodiazepines raise concerns because of their abuse potential. Alprazolam and diazepam demonstrate greater rewarding effects of single challenge doses in abstinent alcoholic subjects compared to those without alcohol dependence (80). Not all alcoholics have a euphoric response, and clinicians are unable to predict who may misuse benzodiazepines. The studies of abuse liability of benzodiazepines in alcoholic persons suffer from the bias of only studying alcoholics seeking treatment. Some have argued that misuse of this class of medications is most common in severely dependent patients, and that less dependent patients may have little abuse potential (81,82).

Differences in the rewarding properties of various benzodiazepines in alcoholics have not been extensively studied. Many believe that rapid onset-rapid offset medications present the greatest risk for misuse (81), but these pharmacokinetic properties are desirable for sleep agents to allow rapid induction of sleep while avoiding morning lethargy. Relatedly, sleep experts commonly recommend nonbenzodiazepine agents like zaleplon and zolpidem because of their rapid action and clearance, although little is known about their abuse liability among alcohol dependent persons. Suffice it to say that more research is needed regarding the safety and effectiveness of benzodiazepine receptor agonists for the short-term management of insomnia in alcohol dependent patients (13).

However, it is for persistent insomnia, which can lead to alcohol relapse, that better and safer treatments are needed. Benzodiazepines produce tolerance and lose their sleep-promoting properties within 2 weeks. Physical dependence and withdrawal phenomena occur with long-term use of benzodiazepines, and all medications in this class can cause rebound insomnia following discontinuation. No studies have demonstrated the hypnotic efficacy of benzodiazepines beyond 12 weeks. For these reasons, benzodiazepines should probably be considered only after alternative therapies have proven ineffective (84). These cautions probably apply to the nonbenzodiazepines as well, although studies of their chronic use and abuse in recovering alcoholic populations are limited.

Pharmacological alternatives commonly prescribed for sleep disorders in alcohol-dependent patients include sedating antidepressants, antihistamines, and low-potency neuroleptics, but these agents have not been rigorously studied as sleep agents (85). Trazodone, a sedating antidepressant, is the medication most commonly prescribed by addiction experts for insomnia among sleep-disturbed alcoholics (85). We are performing an ongoing clinical trial to examine its effect on sleep and alcohol outcomes in early recovery. Ritanserin, a specific 5-

hydroxytryptamine antagonist was tested at three doses against placebo over 6 months in a randomized trial with 493 detoxified alcohol-dependent individuals (86). None of the three dosages revealed significant improvement over placebo in sleep quality in persons without psychiatric disorders. During the first two weeks after detoxification, five days of carbamazapine was superior to lorazepam in improving sleep for patients with mild to moderate alcohol withdrawal (87). Gabapentin, an anticonvulsant, in an open label, uncontrolled study, also showed promise as a safe and effective treatment for alcohol-dependent patients with insomnia during early recovery (88).

CONCLUSIONS

In low to moderate doses, alcohol initially promotes sleep. However, scientific consensus maintains that chronic use ultimately disrupts sleep-related physiology–even among those who do not meet diagnostic criteria for alcohol dependence.

Better prospective clinical and laboratory sleep studies of alcohol use and insomnia are clearly warranted. Future research should: (1) use rigorous definitions and standardized measures of both sleep disturbance and alcohol use; (2) investigate whether subjective or objective measures of sleep are most associated with alcohol use disorders; (3) evaluate the stimulant-like effects of acute alcohol intoxication, its relationship to sleep disturbance and the risk of developing alcohol problems; (4) assess whether routine inquiry about sleep disturbance in medical and mental health settings might lead to greater recognition of alcohol use disorders; (5) determine the best procedure for screening for sleep disturbance among people who drink alcohol; (6) assess gender and other demographic differences in the relationships between alcohol use, psychiatric disorders and sleep disturbance; and (7) examine the interactions between alcohol use, obesity and sleep apnea. Fine-grained longitudinal assessments of the relationship between the level of alcohol consumption and sleep problems on a night-to-night basis are also needed to inform medical practitioners about the proper advice (e.g., how much to cut down) to offer to heavy-drinking-patients who complain of sleep disturbance. Future research should also determine the efficacy of behavioral and/or pharmacological interventions for sleep disturbance among problem drinkers, their effect on drinking behavior and the most favorable phase of recovery to institute sleep interventions.

Acknowledgments

This review was funded by the Department of Veterans Affairs, Veterans Health Administration, Health Services Research and Development Services (TRP 04-179), the Robert Wood Johnson Foundation, and the National Institute on Alcohol Abuse and Alcoholism (1R01 AA13243). Dr. Stein is a recipient of a National Institute on Drug Abuse Mid-Career Investigator Award. Dr. Friedmann is Director of the Target Research Enhancement Program at the Providence Veterans Affairs Medical Center. The views expressed in this article are those of the authors and do not necessarily represent the views of the National Institute on Alcohol Abuse and Alcoholism, National Institute on Drug Abuse, or Department of Veterans Affairs.

References

1. Katz DA, McHorney CA. Clinical correlates of insomnia in patients with chronic illness. Arch Int Med 1998;58:1099–1107. [PubMed: 9605781]
2. Kuppermann M, Lubeck D, Mazonson P, et al. Sleep problems and their correlates in a working population. J Gen Int Med 1995;10:25–32.
3. Wiley J, Camacho T. Life-style and future health: Evidence from the Alameda County Study. Prev Med 1980;9:1–21. [PubMed: 7360725]
4. Kupfer D. Management of insomnia. NEJM 1997;336:341–6. [PubMed: 9011788]
5. Klink M, Quan S, Kaltenborn W, Lebowitz M. Risk factors associated with complaints of insomnia in a general adult population. Arch Int Med 1992;152:1634–7. [PubMed: 1497397]

6. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. JAMA 1989;262(11):1479–84. [PubMed: 2769898]
7. Dufour MC, Archer L, Gordis E. Alcohol and the elderly. Clin Ger Med 1992;8:127–41.
8. Schuckit MA, Irwin M. Diagnosis of alcoholism. Med Clin N Am 1988;72:1133–53. [PubMed: 2900917]
9. Castenada R, Sussman N, Westrich L. A review of the effects of moderate alcohol intake on the treatment of anxiety and mood disorders. Clin Psych 1996;57:207–12.
10. Reite M, Buysse D, Reynolds C, Mendelson W. The use of polysomnography in the evaluation of insomnia. Sleep 1995;18:58–70. [PubMed: 7761745]
11. Roehrs T, Papineau K, Rosenthal L, Roth T. Ethanol as a hypnotic in insomniacs: Self administration and effects on sleep and mood. Neuropsychopharm 1999;20:279–86.
12. Gresham SC, Webb WB, Williams RC. Alcohol and caffeine: Effect on inferred visual dreaming. Science 1963;140:1226–7. [PubMed: 13950328]
13. Prinz P, Roehrs T, Vitaliano P, Linnoila M, Weitzman E. Effect of alcohol on sleep and nighttime plasma growth hormone and cortisol concentrations. J Clin Endoc Metab 1980;1:759–64.
14. Petrucelli N, Roehrs TA, Wittig RM, Roth T. The biphasic effects of ethanol on sleep latency. Sleep Research 1994;23:75.
15. Roehrs T, Zwyghuizen-Doorenbos A, Timms V, Zorick F, Roth T. Sleep extension, enhanced alertness and the sedating effects of ethanol. Pharm Biochem Behav 1989;34:321–4.
16. Zwyghuizen-Doorenbos A, Roehrs T, Lamphere J, Zorick F, Roth T. Increased daytime sleepiness enhances ethanol's sedative effects. Neuropsychopharm 1988;1:279–86.
17. Maclean AW, Cairns J. Dose-response effects of ethanol on the sleep of young men. J Stud Alc 1982;43:434–44.
18. Landolt HP, Roth C, Dijk DJ, Borberly AA. Late-afternoon ethanol intake affects nocturnal sleep and the sleep EEG in middle-aged men. J Clin Psychopharm 1996;16:428–36.
19. Roehrs T, Claiborue D, Knox M, Roth T. Residual sedating effects of ethanol. Alc Clin Exp Res 1994;18:831–4.
20. Rundell OH, Lester BK, Griffiths WJ, Williams HL. Alcohol and sleep in young adults. Psychopharm 1972;26:201–18.
21. Ancoli-Israel S, Roth T. Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation Survey. Sleep 1999;22(Suppl 2):S347–53. [PubMed: 10394606]
22. Johnson EO, Roehrs T, Roth T, Breslau N. Epidemiology of alcohol and medication as aids to sleep in early adulthood. Sleep 1998;21(2):178–86. [PubMed: 9542801]
23. Costa E, Silva JA, Chase M, Sartorius N, Roth T. Special report from a symposium held by the World Health Organization and the World Federation of Sleep Research Societies: An overview of insomnias and related disorders–recognition, epidemiology, and rational management. Sleep 1996;19:412–16. [PubMed: 8843532]
24. Walsh JK, Humm T, Muehlback MJ, Sugerman JL, Schweitzer PK. Sedative effects of ethanol at night. J Stud Alc 1991;52:597–600.
25. Ohayon MM, Caulet M, Philip P, Guilleminault C, Priest RG. How sleep and mental disorders are related to complaints of daytime sleepiness. Arch Int Med 1997;157:2645–52. [PubMed: 9531234]
26. Aldrich MS. Automobile accidents in patients with sleep disorders. Sleep 1989;12:487–94. [PubMed: 2595172]
27. Johnson JE. Insomnia, alcohol, and over-the-counter drug use in old-old urban women. J Comm Health Nursing 1997;14:181–8.
28. Baum-Baicker C. The psychological benefits of moderate alcohol consumption: A review of the literature. Drug Alc Depend 1985;15:305.
29. Bixler EO, Kales A, Soldatos CR, Kales JD, Healey S. Prevalence of sleep disorders in the Los Angeles Metropolitan Area. Am J Psych 1979;136(1):1257–62.
30. Carskadon M, Demendt W, Mitler M, Guilleminault C, Zarcone V, Spiegel R. Self-report versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. Am J Psych 1976;133:1382–8.
31. Baekeland F, Hoy P. Reported vs. recorded sleep characteristics. Arch Gen Psych 1971;24:548–51.

32. Janson C, Lindberg E, Gislason T, Elmasry A, Boman G. Insomnia in men–A 10 year prospective population based study. Sleep 2001;24(4):425–30. [PubMed: 11403527]
33. Tachibana H, Izumi T, Honda S, Horiguchi I, Manabe E, Takemoto T. A study of the impact of occupational and domestic factors on insomnia among industrial workers of a manufacturing company in Japan. Occup Med 1996;46(3):221–7.
34. Harma M, Tenkanen L, Sjoblom T, Alikoski T, Heinsalmi P. Combined effects of shift work and lifestyle on the prevalence of insomnia, sleep deprivation and daytime sleepiness. Scand J Work Environ Health 1998;24(4):300–7. [PubMed: 9754862]
35. Fabsitz RR, Sholinsky P, Goldberg J. Correlates of sleep problems among men: The Vietnam Era Twins Registry. J Sleep Res 1997;6:50–6. [PubMed: 9125699]
36. Baekeland F, Lundwall L, Shanahan TJ, Kissin B. Clinical correlates of reported sleep disturbance in alcoholics. Q J Stud Alcohol 1974;35:1230–41. [PubMed: 4445463]
37. Feuerlein W. The acute alcohol withdrawal syndrome: findings and problems. Br J Addict 1974;69:141–8.
38. Caetano R, Clark RL, Greenfield TK. Prevalence, trends, and incidence of alcohol withdrawal symptoms: Analysis of general population and clinical samples. Alc Health Res World 1998;22:73–9.
39. Foster JH, Marshall EJ, Peters TJ. Predictors of relapse to heavy drinking in alcohol dependent subjects following alcohol detoxification–The role of quality of life measures, ethnicity, social class, cigarettes and drug use. Addict Biol 1998;3:333–43.
40. Mello NK, Mendelson JH. Behavioral studies of sleep patterns in alcoholics during intoxication and withdrawal. J Pharmacol Exp Ther 1970;175(1):94–112. [PubMed: 4319478]
41. Brower KJ, Aldrich MS, Robinson EAR, Zucker RA, Greden JF. Insomnia, self-medication, and relapse to alcoholism. Am J Psych 2001;1568:399–404.
42. Allen RP, Wagman AM, Funderburk FR, Wells DT. Slow wave sleep: A predictor of individual differences in response to drinking? . Biol Psych 1980;15:345–8.
43. Gillin JC, Smith TL, Irwin M, Kripke DF, Schuckit M. EEG sleep studies in "pure" primary alcoholism during subacute withdrawal: Relationships to normal controls, age and other clinical variables. Biol Psych 1990;27:477–88.
44. Thompson PM, Gillin JC, Golshan S, Irwin M. Polygraphic sleep measures differentiate alcoholics and stimulant abusers during short-term abstinence. Biol Psych 1995;38:831–6.
45. Johnson LC, Burdick JA, Smith J. Sleep during alcohol intake and withdrawal in the chronic alcoholic. Arch Gen Psych 1970;22:406–18.
46. Segal BM, Kushnarev VM, Urakov IG, Misionzhnik EU. Alcoholism and disruption of the activity of deep cerebral structures: Clinical-laboratory research. Q J Stud Alcohol 1970;31:587–601. [PubMed: 5458470]
47. Begleiter H, Porjesz B. Persistence of a "subacute withdrawal syndrome" following chronic ethanol intake. Drug Alc Depend 1979;4:353–7.
48. Wellman M. The late withdrawal symptoms of alcoholic addiction. Can Med Assoc J 1954;70:526–59. [PubMed: 13160879]
49. Kissin B. Biological investigation in alcoholics. J Stud Alcohol 1979;8:S146–81.
50. Drummond SPA, Gillin JC, Smith TL, DeModena A. The sleep of abstinent pure primary alcoholic patients: Natural course and relationship to relapse. Alc Clin Exp Res 1998;22:1796–1802.
51. Williams HL, Rundell OH. Altered sleep physiology in chronic alcoholics; reversal with abstinence. Alc Clin Exp Res 1981;2:318–25.
52. Mackenzie A, Funderburk FR, Allen RP. Controlled drinking and abstinence in alcoholic men: Beliefs influence actions. Int J Addict 1994;29:1377–92. [PubMed: 7829276]
53. Gillin JC, Smith TL, Irwin M, Butters N, DeModena A, Schukit M. Increased pressure of rapid eye movement sleep at time of hospital admission predicts relapse in non-depressed patients with primary alcoholism at 3-month follow-up. Arch Gen Psych 1994;51:189–97.
54. Clark C, Gillin J, Golshan S, et al. Increased REM sleep density at admission predicts relapse by three months in primary alcoholics with a lifetime diagnosis of secondary depression. Biol Psych 1998;43:601–7.

55. Brower K, Aldrich M, Hall J. Polysomnographic and subjective sleep predictors of alcoholic relapse. Alc Clin Exp Res 1998;22:1864–71.
56. Allen RP, Wagman AMI, Fund ER, Burke FR. Slow wave sleep changes: Alcohol tolerance and treatment implications. Adv Exp Med Biol 1977;85:629–40. [PubMed: 920496]
57. Block AJ, Hellard DW, Slayton PC. Effect of alcohol ingestion on breathing of asymptomatic subjects. Arch Intern Med 1987;147:1145–7. [PubMed: 3592880]
58. Robinson RW, White DP, Zwillich CW. Moderate alcohol ingestion increases upper airway resistance in normal subjects. Am Rev Resp Dis 1985;132:1238–41. [PubMed: 4073664]
59. Dawson A, Lehr P, Bigby BG, Mitler MM. Effect of bedtime ethanol on total inspiratory resistance and respiratory drive in normal nonsnoring men. Alc Clin Exp Res 1993;17:256–62.
60. Krol RC, Knuth SL, Bartlett D. Selective reduction of genioglossal muscle activity by alcohol in normal human subjects. Am Rev Resp Dis 1984;129:247–50. [PubMed: 6421210]
61. Scrima L, Broudy M, Nay KM. Increased severity of obstructive sleep apnea after bedtime alcohol ingestion: diagnostic potential and proposed mechanism of action. Sleep 1982;5:318–28. [PubMed: 7163721]
62. Aldrich MS, Shipley JE, Tandon R, Kroll PD, Brower KJ. Sleep-disordered breathing in alcoholics: Association with age. Alc Clin Exp Res 1993;17:1179–83.
63. Mamdani M, Hollyfield R, Ravi SD, Dorus W, Borge GF. Prevalence of sleep apnea among abstinent chronic alcoholic men. Sleep Res 1989;18:349.
64. Bassetti C, Aldrich MS. Alcohol consumption and sleep apnea in patients with TIA and ischemic stroke. Sleep Res 1996;25:400.
65. Aldrich MS, Chervin RD. Alcohol use, obstructive sleep apnea, and sleep-related motor vehicle accidents. Sleep Res 1997;26:308.
66. Aldrich MS, Shipley JE. Alcohol use and periodic limb movement of sleep. Alc Clin Exp Res 1993;17:192–6.
67. Berlin RM, Qayyum U. Sleepwalking: Diagnosis and treatment through the life cycle. Psychosomatics 1986;27:755–60. [PubMed: 3797607]
68. Roberts RE, Shema SJ, Kaplan GA. Prospective data on sleep complaints and associated risk factors in an older cohort. Psychosomatic Med 1999;61:188–96.
69. Kales JD, Kales A, Bixler EO, et al. Biopsychobehavioral correlates of insomnia, V: Clinical characteristics and behavioral correlates. Am J Psych 1984;141:1371–6.
70. Jacobs EA, Reynolds CF, Kupfer DJ, Lovine PA, Ehrenpreis AB. The role of polysomnography in the differential diagnosis of chronic insomnia. Am J Psych 1988;145:346–9.
71. Benca RM, Obermeyer WH, Thisted RA, Gillin C. Sleep and psychiatric disorders. A meta-analysis. Arch Gen Psych 1992;49:651–68.
72. Merikangas KR, Angst J, Eaton W, et al. Comorbidity and boundaries of affective disorders with anxiety disorders and substance misuse: Results of an international task force. Br J Psych 1996;30:58–67.
73. Davidson KM. Diagnosis of depression in alcohol dependence; changes in prevalence with drinking status. Br J Psych 1995;166:199–204.
74. Bliwise NG. Factors related to sleep quality in healthy elderly women. Psychol Aging 1992;71:83–8. [PubMed: 1558709]
75. Chait LD, Perry JL. Acute and residual effects of alcohol and marijuana, marijuana alone and in combination, on mood and performance. Psychopharm 1994;115:340–9.
76. Haack MR, Harford TC, Parker DA. Alcohol use and depression symptoms among female nursing students. Alc Clin Exp Res 1988;12:365–7.
77. Chang PP, Ford DE, Mead LA, Cooper PL, Klag MJ. Insomnia in young men and subsequent depression. The John Hopkins Precursor Study. Am J Epi 1997;146:105–14.
78. Longo LP, Johnson B. Treatment of insomnia in substance abusing patients. Psych Annals 1998;28:154–9.
79. Greeff AP, Conradie WS. Use of progressive relaxation training for chronic alcoholics with insomnia. Psych Rep 1998;82:407–12.

80. Ciraulo DA, Barnhill JG, Ciraulo AM, et al. Alterations in pharacodynamics of anxiolytsic in abstinent alcoholics: subjective responses, abuse liability, and electroencephalographic effects of alprazolam, diazepam and buspirone. J Clin Pharm 1997;37:64–73.

81. Ciraulo DA, Nace EP. Benzodiazepine treatment of anxiety or insomnia in substance abuse patients. Am J Addict 2000;9:276–84. [PubMed: 11155783]

82. Sellers EM, Ciraulo DA, Dupont RL, et al. Alprazolam and benzodiazepine dependence. J Clin Psych 1994;S54:64–75.

83. Mueller TI, Godenberg IM, Gordon AL, Keller MB, Warshaw MG. Benzodiazepine use in anxiety disordered patients with and without a history of alcoholism. J Clin Psych 1996;57:83–9.

84. Miller NS, Gold MS. Introduction-benzodiazepines: A major problem. J Sub Abuse Treatment 1991;8:3–7.

85. Friedmann PD, Herman DS, Freedman S, Lemon SC, Ramsey S, Stein MD. Treatment of sleep disturbance in alcohol recovery: A national survey of addiction medicine physicians. J Addict Dis 2003;22(2):91–103. [PubMed: 12703672]

86. Wiesbeck GA, Weihers HG, Chick J, Boening J. The effects of ritanserin on mood, sleep, vigilance, clinical impression, and social functioning in alcohol-dependent individuals. Alc Alcoholism 2000;35:384–89.

87. Malcolm R, Myrick H, Roberts J, Wang W, Anton RF. The differential effects of dedication on mood, sleep disturbance, and work ability in outpatient alcohol detoxification. Am J Addictions 2002;11:141–50.

88. Karam-Hage M, Brwoer K. Gabapentin treatment for insomnia associated with alcohol dependence. Am J Psych 2000;157:151.

TABLE 1

General Effects of Alcohol Use and Cessation on Sleep Architecture.

Drinking Behavior	**REM Sleep**	**Slow Wave Sleep**	**Sleep Continuity**	**Sleep Latency**	**Total Sleep Time**
Acute Use					
High dose	↓↓	↑↑	↓	↓	↓
Low dose	↓↑	↓↑	↓↑	↑	↑
Chronic Use	↓	↑	↓	↑	↓
Cessation After Chronic Use	↑↑	↓	↓	↑↑	↓

TABLE 2

Association Between Alcohol Use and Insomnia.

Authors/Year	Country	Sample/N	Insomnia Definition	Insomnia Prevalence	Alcohol Definition	Alcohol-Insomnia Association
Bixler et al. 1979 (29)	U.S.A.	Los Angeles household survey, N = 1006	Trouble falling asleep, waking up during night, waking up too early.	32.2% current	Need for help in past year for problems with alcohol	N.S.
Kales et al. 1984 (70)	U.S.A.	Sleep disorders clinic patients and volunteer controls without sleep complaints, N = 200	Difficulty falling asleep or maintaining sleep for one year	100% in sleep disorders clinic	Drinking alcoholic beverages	Unadjusted OR, 5.0
Ford et al. 1989(6)	U.S.A.	Epidemiological Catchment Area study, a 5-site household probability survey, N = 7954	Two or more weeks with trouble falling or staying asleep or waking too early in prior 6 months, not always associated with alcohol use.	10.2% overall	DSM-III alcohol abuse or dependence	Adjusted OR 2.4 (95% CI, 1.0–6.1) for insomnia at two interviews 1-year apart
Kuppermann et al. 1995 (2)	U.S.A.	Telecommunication firm employee volunteers, N = 588	Current “problems with sleep”	29.6%	Daily alcohol use	OR 1.04 (N.S.)
Tachibana et al. 1996 (33)	Japan	Male industrial workers, N = 271	Difficulty falling asleep, mid-sleep awakenings, early morning awakening in last month.	27.7%	Alcohol use 4 or more days per week	OR, 2.6 (95% CI, 1.2–5.7)
Fabsitz 1997 (35) et al.	U.S.A.	Male veterans, N = 8870	Trouble falling asleep, trouble staying asleep at least one day per month	48.1% falling asleep, 48.6% staying asleep	Alcohol use 13 or more drinks in past two weeks	OR, 1.2 (95% CI 1.0–1.4) for trouble falling asleep; OR 1.5 (95% CI 1.3–1.7) for trouble staying asleep
Katz & McHorney, 1998 (1)	U.S.A.	Medical Outcomes Study, a 3-city clinical sample, N = 3445	Difficulty initiating or maintaining sleep in the last 4 weeks on a 6-point Likert-type scale.	16% sever 34% mild	Alcohol use:no history, current or past use	“No significant trends”
Harma et al. 1998 (34)	Finland	Employed men aged 45–60 in Helsinki Heart Study, N = 3020	Difficulty falling asleep, waking up too early, disturbed or restless sleep in past 3 months	39–53% depending on shift worked	250 or more grams of alcohol per year	OR, 1.3 (95% CI, 1.08–1.52)
Janson et al., 2001 (32)	Sweden	Random population survey of men from Upsala, N = 2606	Severe or very severe difficulty falling asleep and/or maintaining sleep in the last months.	12.8% in 1994	CAGE score 2 or greater	OR 1.75 (95% CI, 1.2–2.5)

Subst Abus. Author manuscript; available in PMC 2009 November 10.

TABLE 3

Prevalence of Insomnia Among Alcohol Treatment Populations.

Authors/Year	Country	Sample/N	Insomnia Definition	Insomnia Prevalence
Mello & Mendelsohn, 1970(40)	U.S.A.	Inpatient alcohol treatment, N = 50	Decreased sleep duration from a baseline abstinent period	25%
Baekeland et al. 1974 (36)	U.S.A.	Alcohol dependent outpatients, N = 294	Single item, self-rating	36%
Feuerlein 1974 (37)	U.S.A.	Alcoholic inpatients and outpatients, N = 184	Current sleep disturbance by self-report	Inpatients, 39% Outpatients, 36%
Caetano et al. 1998 (38)	U.S.A.	Detoxification and residential alcohol treatment, N = 748 Driving-Under-Influence program, N = 445	Unable to sleep last 12 months	Detox/residential, 67% DUI, 42%
Foster et al. 1998 (39)	London, England	Inpatient alcohol detoxification, DSM-IV alcohol dependent, N = 82	Score of 1 to 3 (poor) on the 7-point sleep item of the Life Situation Survey (LSS)	72%
Brower et al. 2001 (41)	U.S.A.	Inpatient alcohol treatment, N = 172	1 or more positive responses on the Sleep Disorders Questionnaire for the past 6 months	61%