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TREATMENT OPTIONS FOR SLEEP DISTURBANCES DURING ALCOHOL RECOVERY

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Abstract

Sleep disturbances are extremely common in the early stages of recovery from alcohol dependence and may persist for several months despite continued abstinence. Studies indicate that sleep disturbances independently increase the risk for relapse to alcohol, suggesting that targeting these problems during recovery may support continued abstinence. However, there is limited information in the addiction literature about available and effective treatments for sleep disturbances in recovering alcoholic patients. The primary goals of this article are to describe the phenomenology of sleep disturbances during recovery from alcohol dependence, to outline the evidence linking sleep problems with alcohol relapse, and to describe available pharmacological and nonpharmacological treatment options, including the evidence regarding their efficacy in recovering alcoholic patients. Recommendations for future research are provided along with special considerations for treating insomnia in this population, including avoiding cross-dependent sedatives, such as benzodiazepines and benzodiazepine receptor agonists (BzRAs).

Keywords

alcohol; alcoholism; sleep; insomnia; treatment; pharmacological; nonpharmacological; cognitive-behavioral treatment

Insomnia is a complaint of difficulties falling asleep, frequent and/or prolonged awakenings, and/or consistently unrefreshing sleep that occurs despite adequate sleep opportunity. Insomnia is exceedingly common: as many as 1/3 of adults report experiencing persistent insomnia symptoms during the year.[1–4] Insomnia symptoms persisting for more than one month that are associated with daytime impairment and/or psychological distress define an insomnia disorder, which occurs in one in ten individuals. Persistent insomnia is associated with wide ranging adverse consequences, including decreased quality of life, increased risk for psychiatric disturbances, increased work absenteeism, and poor interpersonal functioning.[1,2,5,6] Among patients in recovery from alcohol dependence, insomnia complaints are especially frequent and may persist for weeks to months post-abstinence.[7–14] Of particular importance, untreated insomnia may interfere with recovery from the primary alcohol addiction and contribute to relapse within the first several months of recovery.[12,15,16] Thus, treating insomnia in recovering alcoholic patients may aid in their recovery and support continued abstinence.

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Despite the putative importance of sleep in the recovery process, there is limited information in the addiction literature about appropriate treatment options for insomnia during recovery. This may exist for a variety of reasons: clinicians assume that co-morbid sleep problems will remit with treatment of the primary alcohol condition; sleep problems are de-emphasized as relatively less important in the early stages of treatment for alcohol dependence; or physicians may be reluctant to address sleep problems due to concerns about sleep medication addiction and limited awareness of other treatment options. This article has three primary goals: (1) to describe the nature and course of insomnia and sleep disturbances among recovering alcoholics; (2) to outline the evidence for the relationship between sleep variables and relapse; and (3) to describe available sleep treatments and the evidence regarding their use in alcoholic patients.

What are the rates of co-occurrence of insomnia and alcohol problems?

Alcohol is used by more than one in ten individuals as a hypnotic agent to self-medicate sleep problems.17 Among community samples, insomnia increases the likelihood of developing alcohol problems. The Epidemiologic Area Catchment (ECA) study, for example, found that the 6-month prevalence of DSM-III-defined alcohol abuse and dependence was 7.0% among those who responded yes to the question “Have you ever had a period of two weeks or more when you had trouble falling asleep, staying asleep or waking up too early?” compared to only 3.8% among those who responded no.5 Those who reported insomnia in the past year but no psychiatric condition were more than twice as likely to develop alcohol abuse over the subsequent year compared to those without either condition at baseline.18 A similar level of risk was found between “sleep disturbances because of worry” and alcohol problems 12 to 15 years later after controlling for select demographic (e.g., age of first intoxication) and psychiatric variables (OR=2.32, 95% CI=1.31–4.09).19 Chronic dependence was more commonly associated with insomnia than no history of dependence at 13-year follow-up.20 A recent study in adolescents found that maternal-rated sleep problems at 3 to 5 years of age (Child Behavior Checklist – Parent Version) in 257 boys from a community-recruited sample of high-risk families predicted early onset of any alcohol use by 12 to 14 years of age. This relationship was not mediated by attention problems, anxiety/depression, or aggression.21 Thus, insomnia may increase the risk for the development of alcohol problems and may contribute to initial substance use.

At least seven studies have evaluated the rates of insomnia among alcoholic patients in treatment. In an early observational study, 58% of a sample of 40 alcoholic male inpatients developed insomnia during the first 6 days of acute alcohol withdrawal.[7] Using single item rating scales, nearly three-quarters of 82 inpatients endorsed sleep problems immediately after detoxification[11] and slightly more than one-third of 294 outpatients reported sleep disturbance during the first month of abstinence.[8] Using a more rigorous standardized questionnaire, Caetano et al. found that ⅔ of a sample of more than 600 men admitted to detoxification and residential treatment programs endorsed insomnia as a withdrawal symptom.[10] Only two studies have used validated sleep disturbance scales to assess insomnia among alcoholic patients in recovery. One found that 61% of a heterogeneous sample of inpatient and outpatient alcoholics reported insomnia during the six months preceding treatment, as measured by a subset of items from the Sleep Disorders Questionnaire;[12] the other found that 91% of inpatient alcoholics scored above the clinical cutoff on the Pittsburgh Sleep Quality Index after one week of abstinence.[14]

Taken together, these seven studies suggest higher co-morbidity for insomnia symptoms and alcoholism than in the general population,[5,18] with comparable rates to more widely studied psychiatric subgroups (e.g., depression).[22] Prevalence estimates vary widely because insomnia was rarely operationally defined, few studies used validated instruments, none established

whether insomnia was a pre-existing condition or developed in the context of alcohol dependence, and sample characteristics and the assessment time frame varied from study to study. It was also not always clearly stated that subjects were abstinent from cross-tolerant sedatives in addition to alcohol. Studies also failed to differentiate insomnia symptoms from an insomnia disorder, which is characterized by the additional burden of daytime impairment and/or psychological distress attributable to the sleep problems. Insomnia disorders are more likely to have a chronic course, to require independent treatment, and may contribute more directly to relapse during alcohol recovery. Large-scale epidemiological studies with validated measures and good operational definitions of insomnia are clearly warranted to establish the incidence and prevalence of insomnia in the alcoholic population and its clinical course in relation to the alcohol disorder.

What is the course of recovery of insomnia and other sleep disturbances during abstinence?

Only a handful of studies have assessed the persistence of insomnia during alcohol recovery. In an early uncontrolled study, Alling and colleagues23 reported that insomnia symptoms in 56 recently abstinent alcoholic patients persisted for approximately 5 weeks. Cohn and colleagues14 administered the Pittsburgh Sleep Quality Index (PSQI),24 a validated instrument that measures general sleep disturbance, to 57 alcoholic inpatients (19 women, age range 23–69 years) monthly for 12 weeks. Despite improvement, PSQI total scores remained above the clinical cutoff of 5, suggesting persistent sleep disturbances: 11.7±4.3 at baseline, 9.1±4.4 at 4 weeks, 7.3±4.2 at 8 weeks, and 5.7 ±3.3 at 12 weeks. The subscales for sleep quality, sleep latency, and habitual sleep efficiency, which are most reflective of insomnia, showed no improvements.14 Another study found recovery of total PSQI scores by six months with continued abstinence (7.1±3.4 at baseline vs. 4.8±2.4 at 6 months).25

Several studies have evaluated sleep disturbances in abstinent alcoholics using overnight polysomnography (PSG). PSG provides objective information about sleep quantity and quality that are summarized in measures of sleep continuity, sleep architecture, and respiratory or other physiological parameters (e.g., limb movements). Sleep **continuity** refers to measures of sleep initiation and maintenance (i.e., insomnia) and include sleep latency (the length of time from "lights out" to sleep onset), the number and duration of nighttime awakenings, total sleep time, and sleep efficiency (time asleep/time in bed*100). Sleep **architecture** refers to the macrostructure of sleep that is divided into two separate states: Non Rapid Eye Movement (NREM; Stages 1, 2, 3, and 4) and Rapid Eye Movement (REM) sleep. Sleep architecture differences may be less obvious to patients than periods of extended wakefulness at night (sleep discontinuity), but they may relate to next-day reports of sleep quality and daytime functioning.

In young adults without sleep complaints, sleep latency occurs within 20 minutes, 75% of the night is spent in NREM sleep (15–20% in Stages 3 and 4 combined, also called "deep" or "slow wave" sleep (SWS), and sleep alternates between NREM and REM in roughly 90-minute cycles.[26] With increasing age, decreases in total sleep time, sleep efficiency, percentage of SWS and REM, and REM latency (the elapsed time between sleep onset and the first appearance of REM sleep) are evident; by contrast, there are increases in sleep latency, the percentage of Stages 1 and 2 (light sleep), and time awake during the night.27

Middle-aged alcoholic adults demonstrate severely disrupted sleep continuity and architecture during acute withdrawal (first 1–2 weeks), subacute withdrawal (weeks 2 to 8), and with sustained abstinence. Early largely uncontrolled studies in male alcoholics during the first few months of recovery reported increased sleep fragmentation (frequent awakenings, stage changes, and movements during sleep), a substantial reduction of SWS, particularly Stage 4, and increased REM sleep, due to shortened REM latency and REM-NREM cycles.9,28–32

More recent well-controlled studies largely supported these earlier findings, but continued to include only male subjects so the findings may not generalize to female alcoholics. Gillin and colleagues,33 for example, compared the sleep of 31 middle-aged male DSM-III-defined "primary" alcoholics (no pre-existing major psychiatric disorder prior to the onset of alcoholism) in the first two weeks of recovery with 34 healthy male controls. Compared to the controls, sleep latency was increased (24 ± 15 vs. 10 ± 8 minutes) and total sleep time (334 ± 51 vs. 368 ± 48 minutes) and SWS percentage (3.6 ± 6.1 vs. 8.1 ± 8.0 %) were decreased in the alcoholic group. Other studies have reported decreased sleep efficiency and REM latency, and increases in the percentage of Stage 1, the number of awakenings and stage shifts during the night, and REM density (number of eye movements in REM sleep per REM episode) in alcoholic patients compared with controls.[25,34,35] The effects on REM sleep typically persist during subacute withdrawal, particularly in alcoholics with depression.[29–32] Sleep disturbances during the first few months of recovery are more severe in alcoholic patients who are older,[36] of African American ethnicity,[37] and who have co-morbid depression.[38] Indeed, the sleep of non-depressed alcoholics in early recovery may be even more disturbed than the sleep of depressed non-alcoholic patients.[35]

Longitudinal studies of sleep in alcoholics beyond subacute withdrawal are largely lacking, although the available data suggest that sleep remains grossly disturbed with continued abstinence. In 6- and 12-month PSG evaluations of 11 (28%) abstinent subjects, Gann and colleagues25 reported continued abnormalities in sleep continuity and sleep architecture despite subjectively-rated sleep improvements. Two longitudinal studies evaluated the sleep of male alcoholic veteran inpatients with PSG follow-ups at 2 to 3 weeks, 3 to 6 months, 9 to 15 months, and 2 years post-abstinence.[39,40] Despite the methodological differences, some consistent findings emerged. Most measures of sleep continuity (total sleep time, sleep efficiency, sleep latency) and sleep architecture improved in those who remained abstinent during the first year, but increased fragmentation/arousals from sleep, elevated stage changes, and REM disruptions persisted even after two years. These findings need to be interpreted with extreme caution, however, in light of the low retention rates: only 5 of the original 48 patients were evaluated at final follow-up in the first study39 and 4 of 29 were studied at 27 months in the other.40

What role do sleep disturbances play in alcohol relapse?

Five published studies examined whether subjective sleep disturbances increased the likelihood of relapse. In an early observational study, Skoloda and colleagues41 reported that alcoholic inpatients with disturbed sleep and difficulty falling asleep were more likely to relapse by 4 weeks. Two more recent studies[15,42] extended these early findings to a 3–5 month follow-up. In one of these studies, 32 of 58 (62%) alcohol dependent subjects who completed an inpatient detoxification program relapsed by 12 weeks.15 Among the clinical and demographic variables collected, only the item "It takes me a long time to fall asleep" on the Nottingham Health Profile (NHP) questionnaire differentiated relapsers from abstainers. These results supported earlier findings that relapse was most strongly related to the sleep subscores on the NHP.42 Similarly, Brower and colleagues16 reported that alcoholic patients who relapsed by 5 months post-treatment were more likely to report difficulty falling asleep and abnormal sleep than patients who remained abstinent. In a follow-up study, 60% (28/47) of alcoholic patients who reported insomnia in the 6 months prior to quitting alcohol, compared to 30% (8/27) who did not, relapsed after 5 months. After controlling for alcohol dependence and depression severity, baseline insomnia was the only significant predictor of relapse.43

Objective sleep architecture (SWS and REM) and sleep continuity (sleep latency, total sleep time, sleep efficiency) variables have also been studied as potential predictors of relapse.[44] Reduced SWS percentage at baseline was implicated in early studies,[45,46] but these findings

were only partially replicated in a larger sample.16 Gillin and colleagues47 found that increased "REM pressure"—a composite of short REM latency, increased REM percentage, and increased REM density (a measure of eye movement activity during sleep)—at the time of admission to a VA inpatient program correctly predicted relapse in 80% of non-depressed alcoholics at 3-month follow-up. These findings were replicated in one study with a 6-month follow-up that used a cholinergic REM induction test to probe for REM sleep abnormalities. 25 Other studies have reported that short REM latency16 and increased REM density48 predicted relapse by 5 months, and in both cases sleep continuity measures (increased sleep latency and reduced total sleep time) were also strongly predictive.16,48 Objective predictors of drinking status at 27 months in the only long-term study were elevated sleep onset latency and decreased sleep efficiency.40

The evidence to date suggests that subjective and objective sleep continuity variables (insomnia) are robust predictors of relapse during recovery from alcohol dependence. On the other hand, the relationship between relapse and sleep architecture variables remains to be fully elucidated. Targeting insomnia during recovery from alcohol dependence may thus improve treatment outcome for the alcoholic patient.

What options exist for treating insomnia in alcoholic patients?

A number of effective pharmacological and nonpharmacological treatment options exist to manage insomnia. Most have been evaluated in non-alcoholic insomnia patients so their efficacy in alcoholic patients is uncertain. Moreover, treating insomnia in the alcoholic patients requires special consideration because of the abuse history and potential for overdose with some pharmacological agents when mixed with alcohol. Clinical guidelines have been recommended for addressing insomnia complaints in early recovery.[43] Good sleep practices should be discussed with all recovering alcoholic patients, but adjunctive insomnia treatment should be initiated if sleep problems persist beyond one month and are accompanied by daytime impairment and/or psychological distress.

Pharmacological treatments for insomnia

There are numerous hypnotic medications currently on the market for treating insomnia in nonalcoholic patients. A recent postal survey of addiction treatment specialists, however, revealed reticence in prescribing hypnotic medications to alcoholic patients with insomnia: only 64% of 311 respondents reported offering pharmacological treatment to a recovering alcoholic patient with insomnia and only 22% offered medication to more than half of these patients.[49] When a medication was prescribed, trazodone was the preferred hypnotic followed by other sedating antidepressants and antihistamines despite limited evidence of their efficacy in this population. Below, we outline the most commonly available hypnotic medications and their efficacy in alcoholic patients.

Benzodiazepines and benzodiazepine receptor agonists (BzRAs)— Benzodiazepines and other benzodiazepine receptor agonists (e.g., zolpidem) are safe, efficacious, sedative-hypnotics and often the medications of choice for treating transient insomnia in nonalcoholic patients.[50] They also may have beneficial effects on sleep during subacute alcohol withdrawal.51 Although the potential for abusing sedative-hypnotics in most patients is low,52 alcoholic patients are regarded as having an increased risk for sedative-hypnotic abuse.53–55 Accordingly, most addiction treatment specialists recommend against the use of sedative-hypnotics in alcoholic patients (except for benzodiazepines during acute alcohol withdrawal), because of their abuse potential, withdrawal effects, rebound insomnia, and potential for overdose when mixed with alcohol.56–59 Moreover, benzodiazepines suppress SWS, shift EEG activity to higher frequencies (signaling more arousal), and reduce homeostatic sleep drive.60

Anticonvulsants—The potential uses of anticonvulsants for treating alcohol dependence have recently been reviewed.[61] They have the obvious advantage of not lowering seizure threshold in alcoholics, a population already at risk for seizures. Kindling and sensitization have been suggested as mechanisms that contribute to protracted withdrawal symptoms such as craving. The anti-kindling effects of anticonvulsants may be important in their potential to prevent relapse. At least two anticonvulsants – carbamazepine and gabapentin – have been studied specifically for their effects on sleep in alcoholics. Carbamazepine was found in one study to be superior to lorazepam for treating sleep disturbance associated with acute alcohol withdrawal.[62] Gabapentin has the advantages of sleep promotion, non-liver metabolism, non-interference with metabolism or excretion of other medications, and it does not require blood monitoring for therapeutic concentrations, hepatotoxicity, and hematological toxicity. Furthermore, it has a favorable side effects profile and no known abuse potential, and is not protein-bound. It exerts its CNS effects by binding to alpha-2-delta receptors, resulting in voltage-sensitive calcium channel inhibition.[63]

In two studies, gabapentin improved the sleep of recovering alcoholic patients.64–66 Karam-Hage and Brower64 used gabapentin (mean dose 953 mg/day) to treat 15 of 17 consecutively evaluated abstinent alcoholic patients with persistent insomnia of at least four weeks in an open label study. Self-reported sleep quality improved over 4–6 weeks of treatment and all but two patients remained abstinent. Nightly gabapentin (mean dose 888 ± 418 mg/day) for 4–6 weeks was also found to improve scores on the Sleep Problems Questionnaire (SPQ)[67] more than trazodone (mean dose 105 ± 57 mg).65 Patients receiving gabapentin were less likely to report feeling tired and worn out upon awakening than those receiving trazodone. One recent placebo-controlled study, however, found no differences in sleep outcomes between gabapentin and placebo but patients taking gabapentin remained abstinent longer.68 More controlled studies in alcoholic patients with and without insomnia are necessary to evaluate the efficacy of gabapentin as a sleep aid and/or as an agent to reduce relapse.

Antidepressants—A number of antidepressants have sedative effects. Longo and Johnson[69] recommended trazodone or nefazodone as first-line agents for medicating insomnia in substance abusers, but nefazodone is rarely used today because of hepatic failure risk. Trazodone is likely effective acutely in the treatment of depressed patients with insomnia.[70–73] It has been used safely in several samples of alcoholics.[65,74] Liebowitz & El-Mallakh[75] posited that trazodone facilitated abstinence in alcoholics by reducing anxiety and insomnia.

Only one controlled study has evaluated the efficacy of trazodone as a hypnotic in alcohol dependent patients. Sixteen patients were randomized to receive 4 weeks of either trazodone (50–200 mg nightly, n=8) or placebo (n=8) following participation in an alcohol detoxification program and two-week washout period. Overnight sleep studies were conducted at baseline, (N1 and N2), the night following the first trazodone or placebo dose (N3), and after four weeks of treatment (N28). The Hamilton Rating Scale for Depression (HRSD) and Clinical Global Impression (CGI) scales were administered at baseline and at post-treatment. Compared to placebo, trazodone reduced wake time after sleep onset (WASO) and improved sleep efficiency with a trend for longer total sleep time on N3. Only WASO improvements were sustained at N28, but CGI and HRSD scores were more improved in the trazodone group.[76] Trazodone may improve sleep and other important indicators of daytime functioning in recovering alcoholic patients, but more large scale controlled trials with longer follow-up periods are needed.

Low doses of sedating tricyclic antidepressants (TCAs), such as amitriptyline and doxepin, are used frequently to treat insomnia.[53] However, their dosing is complicated by alcohol-induced changes in liver metabolism77 that can result in decreased blood concentrations and efficacy. 78 Another disadvantage is that the tricyclic antidepressants have considerable overdose

potential in a population that is prone to suicide. SSRIs, another class of antidepressants, can increase the number of awakenings and worsen sleep in both depressed and non-depressed individuals[79,80] making them unlikely candidates to treat insomnia. Moreover, studies of alcoholism treatment with TCAs or SSRIs in controlled trials have produced inconsistent results[81–86] and SSRIs may even worsen drinking outcomes in some patient subgroups.[87] The antidepressant mirtazapine was associated with somnolence in controlled clinical trials for major depression and it improves sleep in depressed patients, but has untoward side effects including weight gain.[88]

Antipsychotics—Quetiapine, an atypical antipsychotic with sedative effects[89], has been used to teat insomnia in alcohol dependent patients. Monnelly et al[90] found that alcoholic veterans with sleep complaints who received 25–200 mg of quetiapine had more days of abstinence and fewer hospitalizations, suggesting that quetiapine may be associated with improved abstinence through improvements in sleep.[90] Unfortunately, sleep was not directly assessed in this study. Moreover, any benefit of quetiapine must be weighed against its potential for akathisia[91] and increased periodic limb movements when used to promote sleep.[89] Thus, quetiapine should not be a first-line hypnotic for alcoholic patients, but additional studies examining its benefit as a sleep aid are warranted.

Other Hypnotics—Melatonin is a sleep promoting agent that is particularly useful for treating circadian rhythm disorders.[92] Given the evidence that melatonin levels are decreased in alcoholic patients,[93–95] supplemental melatonin should be investigated to treat alcoholic patients with sleep disturbances. Because the manufacturing and quality of melatonin is not currently regulated in the U.S., the melatonin receptor agonist, ramelteon, may be a better candidate for study. Disulfiram causes drowsiness more frequently than placebo,[96] but it decreases total REM time[97] and is compromised by low compliance rates.[96, 98]

Nonpharmacological treatments for insomnia

A recent NIH State-of-the-Science conference on the Manifestations and Management of Chronic Insomnia in Adults recently concluded that cognitive-behavioral treatment for insomnia (CBT-I) benefits 70–80% of non-alcoholic insomnia patients and has more sustained benefits than medications.[99] The efficacy of CBT-I has been replicated in numerous controlled trials of primary insomnia patients[100,101] and more recently in patients with insomnia that is comorbid to chronic pain,[102] breast cancer,[103–105] and hypnotic dependence.[106] Four separate meta-analysis found that CBT-I produced medium to large effect sizes for subjective sleep latency, nighttime wakefulness, and sleep efficiency in both young and older patients with insomnia.[100,101,107,108] Its efficacy is equivalent to or superior to pharmacological treatments over six to eight weeks,[107,109–111] with excellent maintenance of therapeutic gains.[109] The specific use of CBT-I in alcohol-dependent patients is reviewed below.

CBT-I is a multi-component therapy that includes behavioral and cognitive strategies to consolidate nighttime sleep and improve sleep quality and daytime functioning. Treatment targets the factors that maintain insomnia over time by disrupting the circadian timing system and/or reducing sleep drive. These factors include maladaptive behavioral strategies to compensate for poor sleep (e.g., alcohol use for sleep, napping), efforts to deal with the consequences of insomnia (e.g., excessive caffeine intake), pre-sleep cognitive arousal, and negative sleep-related beliefs and attitudes (e.g., worry about inability to sleep and daytime consequences as a result of sleep loss, unrealistic sleep expectations). Treatment is conducted in individual or group format and usually involves 6 to 8 treatment sessions, although briefer forms of the therapy have shown promise.[112] The primary components of CBT-I are briefly described below:

Sleep Restriction—Initially developed in the late 1980s, the goal of sleep restriction is to increase the "drive" or "pressure" to sleep by temporarily restricting sleep to shorter periods of time spent in bed (TIB) and by prohibiting sleep at times outside of this restricted sleep schedule. This technique circumvents the usual compensatory strategy of spending extra time in bed to "catch up" on lost sleep that is used almost universally by patients with insomnia.[113] The effect of reducing TIB is to create mild sleep deprivation during the daytime, which helps to consolidate sleep at night. Once sleep is consolidated over a shorter TIB, initially equivalent to the patient's typical sleep duration, TIB is gradually extended until a sleep/wake schedule that optimizes daytime alertness is achieved.113 Commonly, sleep restriction is implemented along with another behavioral strategy, stimulus control.

Stimulus Control—Another contributor to persistent insomnia is that environmental sleep cues (bed and bedroom) may become associated with wakefulness and with activities and behaviors that are incompatible with sleep, such as reading, watching television, or worrying about the inability to sleep. The major goals of stimulus control therapy are to re-establish the discriminative properties of sleep and sleep-compatible stimuli with the act of sleeping and to stabilize the sleep/wake timing system.[114] Patients are provided a set of instructions to follow consistently, shown in Table 1.

Sleep Hygiene Education—Sleep hygiene education provides knowledge about behavioral practices that can help or interfere with nighttime sleep.[115] The goal is to increase patients' awareness of and encourage change in daily activities and environmental factors that may be contributing to the sleep problem. Common topics covered include the detrimental influence of substances such as caffeine, alcohol, and nicotine on sleep, the importance of creating a comfortable, cool, dark, and quiet sleep environment, and the benefits of regular routines, including meal schedules, exercise, and winding down before bed. It is important to note that sleep hygiene in isolation has minimal benefit for patients with persistent sleep difficulties[116,117] but it is recommended as part of a multi-component treatment.[118,119] Patients need to understand that they may need to engage in changes for a period of time before they will derive sleep benefits. Table 2 highlights some of the most commonly covered sleep hygiene principles.

Relaxation Therapy—Relaxation therapy (RT) may be helpful for individuals who exhibit high levels of physiological or cognitive arousal at night and/or during the daytime. There are several RT techniques, some that focus on reducing physiological arousal (e.g., progressive muscle relaxation [PMR], biofeedback) and others that attempt to reduce cognitive arousal (e.g., visualization, thought stopping). Selection of the specific relaxation technique should be determined by the presenting complaints of the patient. Mastery of the relaxation strategies usually requires professional guidance and committed practice over the course of several weeks. Of all the relaxation strategies, PMR and biofeedback have received the most empirical support, demonstrating superiority to placebo, wait-list, and no-treatment controls.[120–125] Some studies, however, have found relaxation to be less effective than either sleep restriction[124,126] or stimulus control,[123] thus RT is usually used in conjunction with other CBT-I behavioral components rather than as a stand-alone treatment.[115]

Cognitive Therapy—Cognitive therapy for insomnia aims to alter dysfunctional beliefs about sleep that contribute to perpetuating sleep problems and helps patients to develop more realistic sleep expectations.[115] Dysfunctional beliefs common to insomnia patients (e.g., lack of sleep is totally disabling to functioning) can be addressed using psychoeducational techniques or with a more traditional cognitive therapy model.[127] Recent work has found a positive association between a reduction in sleep-related dysfunctional beliefs and attitudes and treatment outcome.[128–130]

There have been comparatively few studies of nonpharmacological sleep treatments in patients recovering from alcohol dependence. Greeff and Conradie[131] assessed the benefits of PMR for improving subjective sleep quality in 22 male alcoholic inpatients who met DSM-III-R criteria for an insomnia disorder. Half of the participants received two weeks of daily relaxation training while the other half received no treatment. At post-treatment, the relaxation group reported better sleep quality than the control group on a 10-point rating scale. The study had several methodological limitations, including no screening for occult sleep disorders, poor outcome measure selection, and no active control group.

More recent studies have evaluated the efficacy of CBT-based interventions on insomnia and relapse in alcoholic patients. Currie and colleagues[132] randomized sixty alcoholic outpatients (mean age 43.3 ± 10.9 years, 42 men) to individual CBT-I therapy (five sessions over seven weeks), self-help manual with five telephone support calls, or wait-list control. The CBT-I therapy included education about sleep, behavioral therapy (sleep restriction and stimulus control), imagery relaxation, and cognitive therapy. Sleep and drinking outcomes were collected at post-treatment and at 3- and 6-month follow-up with sleep diaries and questionnaires. Treated participants demonstrated greater post-treatment improvements than controls on diary measures of sleep quality, sleep efficiency, number of awakenings, and time to fall asleep. Follow-up assessments at three and six months revealed maintenance of treatment gains. Treatment appeared to have a minimal impact on relapse, however. Conversely, no one relapsed to drinking during a recent 8-week open trial of CBT-I. Moreover, treatment improved sleep quality and important measures of daytime functioning, such as fatigue, mood, and quality of life.[133] Finally, use of a six-session group CBT-based treatment focusing on insomnia improved sleep, reduced aggression, and decreased drug use after one year in adolescents who had received treatment for substance abuse.134,135

Taken together, these findings indicate that CBT-I may improve sleep and quality of life of recovering alcoholic patients. The effects of CBT-I on relapse remain to be determined. Studies are needed to compare the efficacy of pharmacological and nonpharmacological interventions for sleep in early alcohol recovery, both alone and in combination. Potential moderators and mediators of treatment outcome, including demographic, clinical, and physiological measures of alcohol dependence need to be considered to identify which alcoholic patients are most in need of adjunctive sleep treatment.

Conclusions and Directions for Future Research

Insomnia and other sleep disturbances are exceedingly common during early recovery from alcohol dependence and likely contribute to relapse in this population. Although sleep improves with continued abstinence, permanent alterations to the sleep centers of the brain from chronic alcohol exposure may produce persistent abnormalities that, in many cases, require independent treatment. When insomnia symptoms persist beyond four weeks and are accompanied by daytime impairments and/or psychological distress, adjunctive sleep treatments should be initiated. Both pharmacological and nonpharmacological treatments are available, but many are either inappropriate or have not been adequately tested in patients with a history of abuse. More well-controlled studies are needed to characterize the phenomenology of sleep during recovery, to determine the efficacy of monotherapy and combined approaches to sleep treatment in alcoholic patients, and to evaluate the impact of such treatments on relapse and recovery in alcohol dependence. Of particular importance are treatment studies that compare treatment-as-usual for addiction with and without adjunctive pharmacological and nonpharmacological insomnia treatments to determine the relative importance of focusing on sleep problems during alcohol recovery.

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Table 1

Stimulus Control Instructions[114]

1	Get into bed to go to sleep only when you are sleepy.
2	Avoid using the bed for activities other than sleep; for example, do not read, watch TV, eat, or worry in bed. Sexual activity is the only exception to this rule. On these occasions, follow the instructions afterward when you intend to go to sleep.
3	If you are unable to fall asleep within 15–20 minutes, get out of bed and go into another room. Remember, the goal is to associate your bed with falling asleep quickly! If you are in bed for more than about 15–20 minutes without falling asleep and have not gotten up, you are not following this instruction. Return to bed intending to go to sleep only when you are very sleepy.
4	While out of bed during the night, engage in activities that are quiet but of interest to you. Do not exercise, eat, smoke, or take warm showers or baths. Do not lie down or fall asleep when not in bed.
5	If you return to bed and still cannot fall asleep within 15–20 minutes, repeat Step 3. Do this as often as necessary throughout the night.
6	Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body to acquire a consistent sleep-wake rhythm.
7	Do not nap during the day.

Table 2

Common sleep hygiene recommendations.

1	Maintain a regular bedtime and wake time, including on weekends.
2	Do not use alcohol to help you sleep.
3	Do not consume caffeinated products (e.g., coffee, tea, sodas, chocolate) in the evening.
4	Smoking and other drugs will disrupt your sleep.
5	Eat regular meals every day.
6	Make the last hour before bed a wind-down time. Have a *light* carbohydrate snack before bed.
7	Limit evening liquid intake.
8	Avoid napping during the day.
9	Exercise regularly, but not within about 3 hours of bedtime.
10	Make sure that your bed is comfortable.
11	The temperature of your bedroom should be comfortable and on the cool side (around 65°F).
12	Make sure that your bedroom is dark and quiet.
13	Spend time outside in the light each day.