ELSEVIER

Sleep Medicine 8 (2007) 176–180

SLEEP MEDICINE

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Brief communication

An open trial of cognitive-behavioral treatment for insomnia comorbid with alcohol dependence

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Received 9 April 2006; received in revised form 30 May 2006; accepted 8 June 2006
Available online 18 January 2007

Abstract

Objective: We evaluated the efficacy of cognitive-behavioral treatment for insomnia in recovering alcoholic patients in an open pilot study.
Methods: Seven abstinent alcoholic patients (3 women, mean age 38.6 ± 10.8 years) recruited from outpatient and residential treatment facilities met the Diagnostic and Statistic Manual of Mental Disorders – Fourth edition (DSM-IV) criteria for insomnia comorbid with alcohol dependence and participated in eight individual treatment sessions. Participants were free of other medical, psychiatric, and sleep disorders. Daily sleep diaries were completed beginning two weeks before treatment until two weeks after treatment. Measures of sleep, daytime functioning, and drinking were collected.
Results: Diary-rated sleep latency [$F(2,10) = 14.4$, $p < .001$], wake after sleep onset [$F(2,10) = 7.7$, $p = .009$], and sleep efficiency [$F(2,10) = 28.3$, $p < .001$] improved as did patient-rated and clinician-rated Insomnia Severity Index (ISI) and the Dysfunctional Beliefs and Attitudes about Sleep – Short Form (DBAS-SF). Compared to pre-treatment, significant post-treatment improvements were found on scales measuring depression and anxiety symptoms, fatigue, and quality of life. No one relapsed to alcohol during treatment.
Conclusions: Cognitive-behavioral insomnia therapy may benefit recovering alcoholics with mild to moderate insomnia by improving sleep and daytime functioning. Effects on relapse remain to be determined. Findings need to be interpreted cautiously due to the uncontrolled design and lack of follow-up assessments.

Keywords: Alcoholism; Insomnia; Cognitive-behavioral treatment

1. Introduction

Insomnia during recovery from alcohol dependence is prevalent, persistent, and may contribute to relapse. Prevalence estimates of insomnia among alcoholics in early recovery range from 36–72% [1], with one recent study finding that up to 91% of alcoholic inpatients reported clinically significant sleep disturbance [2]. Objective and subjective sleep difficulties persist for months despite continued abstinence [3,4], and naturalistic treatment outcome studies indicate that these precipitate a return to drinking [5,6]. Thus, treatment of insomnia during alcohol recovery may improve sleep and decrease the likelihood of relapse.

Surprisingly, there are only a handful of insomnia treatment studies using either hypnotics [7–9] or

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doi:10.1016/j.sleep.2006.06.007

cognitive-behavioral therapy [10] in patients recovering from alcohol dependence and none has evaluated treatment changes in daytime functioning.

The primary aim of this study was to pilot the efficacy of an eight-week cognitive-behavioral treatment for insomnia specific to patients in recovery from alcohol dependence (CBTI-A). Our primary endpoints of interest included pre/post changes in subjective sleep quality, daytime functioning, and drinking. We hypothesized that CBTI-A would improve subjective sleep and daytime functioning and reduce the frequency and severity of drinking in these abstinent alcoholic patients with comorbid insomnia.

2. Methods

Participants were recruited from flyers placed at outpatient and residential substance abuse treatment facilities in Ann Arbor, MI. Interested volunteers self-referred and were initially screened by phone to determine minimal eligibility of alcohol dependence in remission and mild insomnia (self-reported sleep latency or wake after sleep onset >30 min on 3+ nights per week for 1+ month *plus* an Insomnia Severity Index (ISI; [11]) score $\geqslant 8$). Of the 40 volunteers screened between January and August 2005, 14 (35%) underwent in-person screening to confirm Diagnostic and Statistical Manual of Mental Disorders – Fourth edition (DSM-IV) lifetime alcohol dependence and insomnia due to alcohol dependence and to exclude chronic medical conditions, psychotic and bipolar disorder, current major depressive disorder (dysthymia and substance-induced mood disorder were *not* exclusionary), and current dependence on substances other than alcohol. Eligible participants underwent overnight polysomnography to rule out occult sleep disorders. Following screening, seven participants were excluded due to no alcohol dependence ($n = 1$), insufficient insomnia ($n = 3$), current major depressive disorder ($n = 1$), post-traumatic stress disorder ($n = 1$), and other sleep disorder ($n = 1$). The final sample included three men and four women. The study was approved by the University of Michigan Medical School Institutional Review Board.

The eight-week individual CBTI-A was developed and manualized by the lead author in consultation with recognized experts in insomnia and alcohol dependence. Four face-to-face sessions and four phone consultations were delivered by two PhD-level clinical psychologists with insomnia treatment experience (J.T.A. and D.C.). Treatment components included sleep restriction, stimulus control, sleep hygiene, cognitive strategies, and sleep maintenance/relapse prevention as well as education regarding the role of alcohol in precipitating and maintaining insomnia during alcohol dependence and withdrawal. The sleep hygiene session additionally included a review of alcohol/drug intoxication and withdrawal sleep effects.

Participants completed daily sleep diaries beginning two weeks pre-treatment until two weeks post-treatment. The following primary sleep variables were derived: sleep onset latency (SOL), wake after sleep onset (WASO), and sleep efficiency (SE; total sleep time/planned sleep time* $\times$ 100); secondary diary measures included frequency of nighttime awakenings, total sleep time, and ratings of sleep quality and restedness. The ISI (patient and clinician versions; [11]), and the Dysfunctional Beliefs and Attitudes about Sleep – Short Form (DBAS-SF; [12]) were completed pre-, mid-, and post-treatment. Daytime impairment was measured pre- and post-treatment with the Multidimensional Fatigue Inventory (MFI-20; [13]), the Inventory of Depressive Symptomatology – Self-Rated (IDS-SR; [14]), the State-Trait Anxiety Inventory (STAI; [15]), and the 36-item Short Form Health Survey from the Medical Outcomes Study (SF-36; [16]). Relapse to drinking was assessed with the Time Line Follow-Back Interview (TLFB; [17]) at pre-, mid-, and post-treatment, and the variables percent of heavy drinking days (PHDD) and the number of drinks per drinking days (DDD) were derived.

Sleep variables (SOL, WASO, SE, ISI, and DBAS-SF scores) and drinking variables (PHDD, DDD) were analyzed separately with one-way repeated measures analysis of variance (ANOVA) with time (pre-, mid-, and post-treatment) as the within-subjects factor. Significant main effects were further assessed with Bonferroni-corrected paired *t*-tests. Changes in daytime functioning measures were evaluated with paired *t*-tests.

3. Results

The seven participants (6 Caucasian, 1 African-American) were 38.6 ± 10.8 years of age. Four were unmarried (2 worked full-time, 2 were unemployed) and three were married but separated (3 worked full-time, 1 was unemployed). They reported abstinence for a median of 246 days (range 27 to 433 days) after being dependent on alcohol for a median of 7.5 years (range 2 to 14 years; Alcohol Dependence Scale (ADS; [18]) score of 16.0 ± 8.0). All continued in outpatient or residential alcohol treatment during the study. They self-reported mild to moderate insomnia (mean baseline ISI score of 13.1 ± 3.7, range 9 to 19) of a median 42 months' duration (range 1 to 398 months). Five had received no past treatment for insomnia, one previously took trazodone, and one took nortriptyline nightly during the study.

One male participant was lost to follow-up after the first treatment session; the other six participants completed the protocol in an average of 66.8 ± 35.3 days. The participant who discontinued treatment was within one standard deviation of the completers for age,

baseline ISI and ADS score, number of days sober, and duration of insomnia. All participants were abstinent at the beginning of treatment and none reported relapses at sessions 5 or 8.

Sleep and daytime functioning outcomes are summarized in Table 1. Over the eight-session treatment, the primary sleep variables SOL [$F(2,10) = 14.4$, $p < .001$], WASO [$F(2,10) = 7.7$, $p = .009$], and SE [$F(2,10) = 28.3$, $p < .001$] improved significantly. Follow-up analyses indicated that SOL and WASO improved significantly by mid-treatment ($p < .05$) and remained stable between mid-treatment and post-treatment ($p > .05$). The same was true for SE (pre vs. mid $p = .004$; mid vs. post $p > .05$). Improvements were additionally evident in the number of nighttime awakenings [$F(2,10) = 4.8$, $p = .03$] and morning ratings of restedness [$F(2,10) = 8.1$, $p = .008$] but not in total sleep time [$F(2,10) = 1.7$, $p = .23$] or ratings of sleep quality [$F(2,10) = 3.0$, $p = .20$].

Diary-reported improvements in sleep continuity were reflected in reductions in patient-rated [$F(2,10) = 32.8$, $p < .001$] and clinician-rated [$F(2,10) = 42.6$, $p < .001$] insomnia severity, both of which were evident by the midpoint of treatment (pre vs. mid $p < .01$ for both ISI measures). Scores on the DBAS-SF indicating dysfunctional sleep-related cognitions improved over the eight-week treatment, but significant improvements were not noted until the latter part of treatment (pre vs. mid $p = .06$, mid vs. post $p = .003$).

We defined clinical significance [19] as a post-treatment SOL *and* WASO < 30 min, SE > 90%, and TST > 6.5 h. By these criteria, five of the six completers experienced clinically significant improvements in their sleep, with an average reduction of 38 ± 6 (range 27 to

Table 1
Sleep and daytime functioning measures pre- and post-treatment

Measure	Pre-treatment	Mid-treatment	Post-treatment	*p*-Value
Sleep measures				
Insomnia Severity Index (patient-rated)[a]	12.7 ± 3.8	5.5 ± 3.8	3.3 ± 3.4	<0.001
Insomnia Severity Index (clinician-rated)[a]	14.5 ± 1.5	7.7 ± 2.3	3.2 ± 1.5	<0.001
Dysfunctional Beliefs and Attitudes about Sleep Scale – Short Form[b]	53.2 ± 17.2	39.7 ± 7.6	22.3 ± 8.6	0.001
Sleep diary				
Sleep onset latency (min)	23 ± 7	8 ± 3	8 ± 4	< .001
Wake after sleep onset (min)	20 ± 11	9 ± 6	8 ± 4	.009
Number of awakenings	2.3 ± 0.9	1.5 ± 0.9	1.4 ± 1.0	0.03
Total sleep time (min)	371 ± 55	410 ± 68	429 ± 43	0.23
Sleep efficiency (%)	86.0 ± 4.0	94.9 ± 2.2	95.4 ± 1.2	<0.001
Sleep quality (1–5)[c]	3.1 ± 0.6	3.4 ± 0.4	3.4 ± 0.4	0.09
Restedness (1–5)[d]	2.9 ± 0.6	3.4 ± 0.4	3.4 ± 0.4	0.008
Daytime functioning measures				
Inventory of Depressive Sympomatology – Self-Rated[e]	25.4 ± 15.3	–	12.4 ± 11.9	0.02
State trait anxiety inventory				
State	40.7 ± 8.8	–	31.8 ± 8.3	0.009
Trait	46.8 ± 11.9	–	37.8 ± 14.1	0.004
Multidimensional Fatigue Inventory-20				
General fatigue	12.8 ± 1.6	–	7.0 ± 2.6	0.003
Physical fatigue	9.3 ± 2.0	–	7.3 ± 2.3	0.17
Reduced activity	9.0 ± 3.3	–	7.3 ± 2.2	0.05
Reduced motivation	11.5 ± 4.0	–	7.0 ± 1.8	0.04
Mental fatigue	11.5 ± 5.3	–	9.7 ± 4.4	0.10
Short Form-36[f]				
Physical functioning[e]	53.7 ± 2.4	–	54.5 ± 2.7	0.65
Role-physical	53.9 ± 5.8	–	53.1 ± 6.7	0.84
Bodily pain	42.4 ± 11.5	–	50.8 ± 9.9	0.10
General health	48.4 ± 3.6	–	53.6 ± 7.5	0.12
Vitality	39.8 ± 9.6	–	49.5 ± 11.2	0.002
Social functioning	41.6 ± 8.2	–	43.0 ± 8.0	0.36
Role-emotional	37.7 ± 13.2	–	41.5 ± 11.4	0.26
Mental health	39.5 ± 13.4	–	45.6 ± 11.4	0.02

[a] Scores range from 0 to 28, with higher scores indicative of more severe insomnia.
[b] Scores range from 0 to 100, with higher scores indicative of greater sleep-related dysfunctional thinking.
[c] Sleep quality anchors: 1, very poor; 2, poor; 3, fair; 4, good; 5, excellent.
[d] Restedness anchors: 1, not at all; 2, slightly; 3, somewhat; 4, rested; 5, well rested.
[e] Data missing from one participant for this scale and subscale.
[f] Three participants received version 2 of the SF-36.

44) minutes of wake time during the night, an increase of 57 ± 79 (range −9 to 202) minutes to TST, and a 9.4 ± 3.7 (4.0 to 14.2) percent improvement in SE at the end of treatment.

Depression and anxiety symptoms improved significantly with treatment as measured by the IDS-SR [$t(4) = 3.7$, $p = .02$] and State [$t(5) = 4.1$, $p = .009$] and Trait [$t(5) = 5.0$, $p = .004$] subscales of the STAI, respectively. Post-treatment improvements were also noted on the General Fatigue [$t(5) = 5.6$, $p = .003$], Reduced Activity [$t(5) = 2.5$, $p = .05$], and Reduced Motivation [$t(5) = 2.8$, $p = .04$] subscales of the MFI-20, but not on the Mental Fatigue or Physical Functioning subscales. The Vitality [$t(5) = -6.0$, $p = .002$] and Mental Health [$t(5) = -3.5$, $p = .02$] SF-36 subscales also demonstrated post-treatment changes.

4. Discussion

In this uncontrolled study, we found that subjectively reported sleep continuity and some collateral measures of daytime impairment improved in recovering alcoholics with mild to moderate insomnia following an eight-session individual non-pharmacological insomnia treatment. Subjective improvements in SOL, WASO, and SE were both statistically and clinically significant with changes evident by session 5 of treatment. The magnitude of change in our primary outcome variables is consistent with previous non-pharmacological [10] and pharmacological [9] trials. Despite evidence of improved sleep consolidation, sleep quality ratings unexpectedly showed no significant post-treatment changes, but participants did report feeling more rested in the morning. Interestingly, sleep-related cognitions did not show marked changes until the latter part of treatment, although this may simply be a function of increased attention to these factors as treatment progressed. These findings require replication and need to be interpreted cautiously given the lack of control group and follow-up assessments.

Our findings extend those of Currie and colleagues [10] by indicating that a non-pharmacological insomnia treatment may benefit the sleep of patients with milder insomnia and contribute to improvements in daytime functioning. On validated scales, we found post-treatment improvements in depression and anxiety symptoms, physical fatigue, motivation, vitality, and mental health. Depression and anxiety scores fell from the moderate to mild range (e.g., IDS-SR 25.4 ± 15.3 pre-treatment to 12.4 ± 11.9 post-treatment), although these scales are not diagnostic and our participants had no DSM-IV-defined mood or anxiety disorder. It is unclear whether these daytime improvements are due to the insomnia treatment *per se* or reflect a non-specific treatment effect. Changes in daytime functioning are important to consider in this population because impairments in mood and quality of life may contribute to relapse.

The most critical outcome in alcohol treatment trials is drinking relapse. Those who completed our study remained abstinent throughout treatment, and we failed to collect sleep or drinking information from the participant who dropped out. Thus, we cannot draw conclusions about the efficacy of our intervention for altering relapse. Our theoretical position is that sleep improvement early in recovery may reduce future relapses, which could manifest as prevention, delay, or reduction in drinking severity if relapse occurs. The only controlled study to examine relapse as an outcome found no treatment effect [10], but only eight participants (15%) relapsed. These findings require replication given the potentially important treatment implications for alcoholic patients.

In summary, we have found in an uncontrolled study that sleep and daytime functioning in recovering alcoholics improved after a non-pharmacological sleep intervention. The degree to which this treatment also affects alcohol relapse remains an unresolved question.

Acknowledgements

This research was supported by National Institute on Alcohol Abuse and Alcoholism (R21 AA014408 and T32 AA07477). We are grateful to the following consultants for their guidance in the development of this intervention: Charles Morin Ph.D., Jack Edinger Ph.D., Timothy Roehrs Ph.D., Donn Posner Ph.D., Damaris Rohsenow Ph.D., and Peter Friedmann Ph.D. We also appreciate the work of Marc Stemmer in helping to prepare the manuscript.

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