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Cognitive Behavioral Therapy for Insomnia in Posttraumatic Stress Disorder: A Randomized Controlled Trial

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Study Objectives: Examine whether cognitive behavioral therapy for insomnia (CBT-I) improves sleep in posttraumatic stress disorder (PTSD) as well as nightmares, nonsleep PTSD symptoms, depression symptoms, and psychosocial functioning.

Design: Randomized controlled trial with two arms: CBT-I and monitor-only waitlist control.

Setting: Department of Veterans Affairs (VA) Medical Center.

Participants: Forty-five adults (31 females: [mean age 37 y (22-59 y)] with PTSD meeting research diagnostic criteria for insomnia, randomly assigned to CBT-I (n = 29; 22 females) or monitor-only waitlist control (n = 16; nine females).

Interventions: Eight-session weekly individual CBT-I delivered by a licensed clinical psychologist or a board-certified psychiatrist.

Measurements and Results: Measures included continuous monitoring of sleep with diary and actigraphy; prepolysomnography and postpolysomnography and Clinician-Administered PTSD Scale (CAPS); and pre, mid, and post self-report questionnaires, with follow-up of CBT-I participants 6 mo later. CBT-I was superior to the waitlist control condition in all sleep diary outcomes and in polysomnography-measured total sleep time. Compared to waitlist participants, CBT-I participants reported improved subjective sleep (41% full remission versus 0%), disruptive nocturnal behaviors (based on the Pittsburgh Sleep Quality Index-Addendum), and overall work and interpersonal functioning. These effects were maintained at 6-mo follow-up. Both CBT-I and waitlist control participants reported reductions in PTSD symptoms and CAPS-measured nightmares.

Conclusions: Cognitive behavioral therapy for insomnia (CBT-I) improved sleep in individuals with posttraumatic stress disorder, with durable gains at 6 mo. Overall psychosocial functioning improved following CBT-I. The initial evidence regarding CBT-I and nightmares is promising but further research is needed. Results suggest that a comprehensive approach to treatment of posttraumatic stress disorder should include behavioral sleep medicine.

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INTRODUCTION

As many as 90% of individuals with posttraumatic stress disorder (PTSD) report nightmares and insomnia[1,2] and even when nightmares are excluded, sleep disturbance is the most frequently reported symptom of PTSD.[3] A survey of Vietnam combat veterans with PTSD showed that 59% to 73% of subjects with PTSD report insomnia and nonrestorative sleep.[4] Moreover, self-reported poor sleep quality in PTSD appears to be minimally influenced by age, sex, and psychiatric comorbidity.[5] Due to the frequency and severity of sleep disturbance, more than 50% of patients with PTSD on psychopharmacologic medications are prescribed trazodone or sedative hypnotic drugs.[6,7]

In addition to experiencing poor sleep as disturbing, individuals with PTSD may suffer from numerous sleep related consequences including a worse course of PTSD, physical health problems, and declined overall functioning. More specifically, sleep disturbance strongly correlates with PTSD symptom severity,[8] worsens daytime PTSD symptoms,[9] and may contribute to comorbid psychiatric problems, given that untreated insomnia is associated with prospective risk for major depression.[10-13] Insomnia in PTSD is also associated with an increased risk for physical health complaints,[14,15] which is not unexpected given that in the general population chronic and severe insomnia are associated with increased risk for hypertension and/or cardiovascular disease[16] and immunosuppression.[17] Finally, sleep disturbance in PTSD is also associated with a reduced capacity to carry out daily activities,[18-20] again aligning with insomnia-related problems in the general population including functional impairment, cognitive impairment, reduced quality of life,[17,21-25] and doubled risk of accidents.[26]

Notably, sleep disturbance frequently does not improve after otherwise successful first- line PTSD treatment,[27,28] and disturbed sleep is one of the two most reported residual symptoms.[28] In particular, insomnia is highly prevalent in individuals who have received treatment for PTSD,[29-32] with one report demonstrating that residual insomnia is found in approximately 50% of patients treated with PTSD-specific cognitive behavior therapy.[28]

The persistence of sleep disturbance in PTSD and its consequences indicate that treatments targeting sleep are necessary.

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Cognitive behavioral therapy for insomnia (CBT-I), a psychological and behavioral treatment with well-established efficacy,[33-36] is a promising candidate for several reasons. First, CBT-I is indicated for the treatment of chronic and severe insomnia,[37,38] as it is delivered as a short-term intervention and produces long-term clinical gains.[39,40] In contrast, pharmacologic treatments appear to treat insomnia less effectively,[29-32] do not have much efficacy data beyond 6 mo,[41] and have not been tested as maintenance therapies. Second, CBT-I has received substantial empirical support for the treatment of insomnia that co-occurs with psychiatric or medical disorders,[42-48] including depression, cancer, alcoholism, and chronic pain. Third, Spielman and colleagues' behavioral model[49] likely applies to PTSD in that experiencing acute trauma exposure may act as a precipitating factor for acute insomnia, but the chronic form of the disorder is likely maintained in part by behavioral factors (e.g., sleep extension) independent from PTSD-specific phenomena such as trauma-initiated fear conditioning and hyperarousal.

Few studies have examined the efficacy of nonpharmacological interventions for insomnia experienced by individuals with PTSD. DeViva and colleagues examined the effectiveness of a five-session CBT-I trial in a case series of five patients with PTSD who had completed a trial of PTSD-specific CBT.[50] CBT-I treatment resulted in improvements in sleep onset latency, wake after sleep onset, total sleep time, sleep efficiency, and sleep quality on subjective measures. Several other studies have examined the effect of imagery rehearsal (IR) therapy (a therapy that involves rescripting of nightmares with the use of imagery)[51] with added CBT-I components in a group or individual format.[52] Several of these studies demonstrated some improvement in subjective sleep and PTSD symptoms. One recent study examined a combined CBT-I and IR therapy in which the first three individual sessions focused on CBT-I in a sample of 22 veterans with PTSD.[53] The CBT-I/IR group demonstrated large treatment effects for subjective insomnia severity and sleep quality compared to the waitlist control group and improvement in self-reported PTSD symptoms, but no improvement in PTSD-specific disruptive nocturnal behaviors. A second recent study compared a behavioral sleep intervention that included nightmare education and IR to prazosin and placebo in 50 military veterans with sleep disturbance and stress-related psychiatric symptoms.[54] Both the prazosin and behavioral sleep treatment groups showed reductions in subjective insomnia severity and nightmare frequency posttreatment, and all three treatment groups (including placebo) had improvements in PTSD symptoms.

To date, no studies have examined the effect of CBT-I in PTSD in a pure format; that is, without the addition of a nightmare-targeted treatment. If such a treatment were effective in treating sleep disturbance, nightmares, and PTSD symptoms, it might confer several practical advantages over a treatment that involves a focus on nightmares. First, and most importantly, IR may be specific to a subgroup of patients with PTSD with stereotypic repetitive nightmares. Individuals without nightmares or without stereotypic repetitive nightmares are unlikely to benefit from IR therapy and the inclusion of nonrelevant information in the protocol could decrease their treatment adherence or completion. Moreover, for individuals with stereotypic repetitive nightmares, it is possible that CBT-I could diminish nightmares more broadly by altering individuals' capacity to remember nightmares (e.g., due to changes in sleep depth or number of awakenings). Second, sleep disturbance often remains at clinically significant levels following IR therapy,[52] whereas CBT-I has demonstrated well-established effects on sleep disturbance. Third, IR therapy requires expertise and sensitivity to trauma experiences that require specialized skills generally found only in PTSD specialty clinics. In contrast, CBT-I is a treatment that can be disseminated to nonspecialist providers.[55-57] Fourth, the dream narrative aspect is not always well tolerated in individuals with PTSD.[58]

Hence, the current study sought to assess the efficacy of unaltered CBT-I in PTSD. Specifically, the CBT-I administered did not contain any quasi-exposure components that could result from the dream narrative aspect inherent to IR or any trauma-specific cognitive components, such as discussions of the safety of the bedroom. The objectives of the current study were to examine whether an 8-w course of CBT-I would improve sleep disturbance in PTSD, as measured by sleep diary, polysomnography, questionnaires assessing subjective sleep quality, and actigraphy. We also assessed whether CBT-I would demonstrate clinical effects that extend beyond the amelioration of sleep disturbance. Specifically, we hypothesized that participants randomized to CBT-I would show improvements in nightmares, nonsleep PTSD symptoms, depression symptoms, and psychosocial functioning and that these effects would be durable as indicated by 6-mo follow-up data. To address these objectives, participants with PTSD were randomized to a CBT-I or waitlist control group with subjective and objective measurement of sleep and other symptoms collected before and after treatment and, for CBT-I participants, repeat assessments conducted at 6 mo posttreatment.

METHODS

Participants

Study participants were recruited from May 2009 to March 2012 through Internet postings and contact with relevant clinicians and community resources in the San Francisco Bay area. Study participants included individuals between the ages of 18 and 65 y who (1) had chronic PTSD of at least 3 mo duration based on Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) diagnostic criteria or partial PTSD operationalized as a past diagnosis of PTSD plus at least one current B symptom and either the C cluster criteria or the D cluster criteria (n = 40 met full criteria for PTSD and n = 5 met partial criteria, of which three were in CBT-I and two in waitlist control); (2) were currently in treatment for PTSD that could include medication therapy (see exceptions in the following paragraphs) or enrollment in a specialized PTSD program or individual psychotherapy with a licensed clinician and had been in one of more of these treatments for at least 3 mo; additionally, participants' medication must have been stable for at least 1 mo prior to baseline assessments and participants in psychotherapy needed to have no plans to discontinue psychotherapy or start new psychotherapy during the course of CBT-I; and (3) had persistent insomnia as defined by meeting research diagnostic criteria (RDC) for insomnia.[59]

Exclusion criteria were (1) presence of conditions or substances associated with comorbid insomnia independent to PTSD, including lifetime history of any psychiatric disorder with psychotic features and bipolar disorder and alcohol or substance abuse or dependence in the past year; (2) current exposure to a recurrent trauma or exposure to a traumatic event within the past 3 mo; (3) pregnancy; (4) diagnosis of sleep apnea, neurologic disorder, systemic illness affecting central nervous system function, and/or anemia; (5) prominent suicidal or homicidal ideation; (6) reports that insomnia began or worsened after starting selective serotonin reuptake inhibitor therapy; (7) history of sleep restriction therapy or cognitive restructuring therapies of beliefs related to sleep; (8) current prescriptions for benzodiazepine or benzodiazepine receptor agonists, opiates, or trazodone, or the use of over-the-counter sleep aids; (9) termination of benzodiazepine or benzodiazepine receptor agonists, anticonvulsants, atypical antipsychotic medication, antidepressant medications in the past 2 w or plans to start these medications during the course of CBT-I; (10) night shift work, in order to avoid the effect of circadian factors on evaluating insomnia; (11) unstable housing; and (12) nonclinically significant or sub-threshold insomnia, as indicated by a score of 0-14 on the Insomnia Severity Index (ISI).[60]

Of the 893 individuals who contacted the recruitment line, 321 participants completed the institutional review board-approved telephone screen. Two hundred forty-one participants were ineligible after telephone screening for the following reasons: medication type or unstable current medication (n = 63); not currently in treatment for PTSD via either specialized PTSD program or individual therapy with a licensed clinician for at least three months (n = 55); pregnancy or diagnosis of sleep apnea, neurologic disorder, or central nervous system illness (n = 36); no longer interested in the study (n = 30); alcohol or substance abuse or dependence in the past year (n = 22); lifetime history of a psychiatric disorder with psychotic features or bipolar disorder (n = 11); no trauma history (n = 8); nonclinically significant insomnia (n = 6); unstable housing (n = 3); out of age range (n = 2); current exposure to a recurrent trauma or exposure to a traumatic event within the past 3 mo (n = 2); history of sleep restriction therapy or cognitive restructuring therapies of beliefs related to sleep (n = 1); night shift work (n = 1); and prominent suicidal ideation (n = 1). See Figure 1.

Eighty participants met initial eligibility criteria based on the telephone screen and were invited for comprehensive second-stage screening. Second-stage screening included the Structured Clinical Interview for DSM-IV (SCID), the Clinician-Administered PTSD Scale (CAPS), a portion of the Duke Structured Interview for Sleep Disorders (DSISD), a medical history interview, a blood draw, and a urine screen. Thirty-five individuals were excluded after this second-stage screening for the following reasons: did not meet aforementioned criteria for PTSD (n = 12), did not meet RDC criteria for insomnia (n = 1), met criteria for alcohol or substance abuse or dependence in the past year (n = 4), met criteria for bipolar disorder (n = 2), met criteria for psychotic disorder (n = 1), had unstable prescription medication use (n = 3); had circadian day-night reversal (n = 1), had diagnosis of sleep apnea (n = 1), experienced severe head trauma (n = 1), did not have enough time to commit to the study (n = 4), and declined to participate (n = 5). See Figure 1.

Study Design

The study was a parallel-groups randomized controlled trial comprised of an 8-w CBT-I treatment arm and an 8-w monitor-only waitlist control arm. Two-thirds of participants were randomized to the treatment group and one-third to the waitlist control group, with blind assignment determined by a computer-generated random allocation schedule operated by the study statistician. Group allocation was provided to the study coordinator in opaque, sealed envelopes that were opened by the study coordinator with the participant following the completion of baseline measures. Clinical interviewers and the polysomnography technician were blind to participants' treatment conditions during both pretreatment and posttreatment administration and scoring. Moreover, the clinical interviewers did not conduct any of the CBT-I treatment sessions, worked in the research program only 1 day per week, and worked in a different building from the research trial, thus ensuring the integrity of their blind status.

Randomization was stratified by sex, age (younger than 45 y versus 45 y or older), and use of antidepressant medication (yes or no). Eight separate block randomization lists for each combination of sex, age, and medication status were used. Within each list, conditions were randomized in blocks of n = 6 (i.e., four participants assigned to CBT-I and two assigned to the monitor-only waitlist control group in each block).

Clinical interviews were conducted at the San Francisco VA Medical Center during the eligibility period and after the 8-w treatment or monitor-only period. Polysomnography in the participant's home environment was used with all participants at baseline and after the 8-w treatment or monitor-only period. All participants maintained a daily sleep diary (with morning and evening entries) and wore wrist actigraphs during the 1-w baseline period, for the duration of the 8-w treatment or monitor-only periods, and during the posttreatment 1-w assessment period. Self-report measures of sleep quality, nightmares, PTSD symptoms, depression symptoms, and psychosocial functioning were obtained in all participants at baseline, after 4 w, and again after 8 w. Participants randomized to CBT-I had repeat assessments and procedures (except polysomnography) at 6 mo posttreatment. All research was approved by the Committee on Human Research at the University of California, San Francisco and at the San Francisco Veterans Affairs Medical Center, and informed consent was obtained from all participants.

Assessment Measures

Clinician-Administered PTSD Scale

Current PTSD was assessed with the CAPS.[61] The CAPS measures frequency and intensity of PTSD-related symptoms. Possible scores range from 0 to 136. The CAPS has excellent test-retest reliability (r = 0.92-0.99) and internal consistency (alpha = 0.80-0.90).[62] Additionally, the CAPS item B2, "recurrent distressing dreams", has face validity for assessment of trauma nightmares.[63]

Structured Clinical Interview for DSM-IV

Diagnoses other than PTSD were assessed with the SCID.[64] The SCID is a semistructured interview designed to assess DSM-IV diagnostic criteria for Axis I disorders. The SCID has been shown to have good reliability.[65]

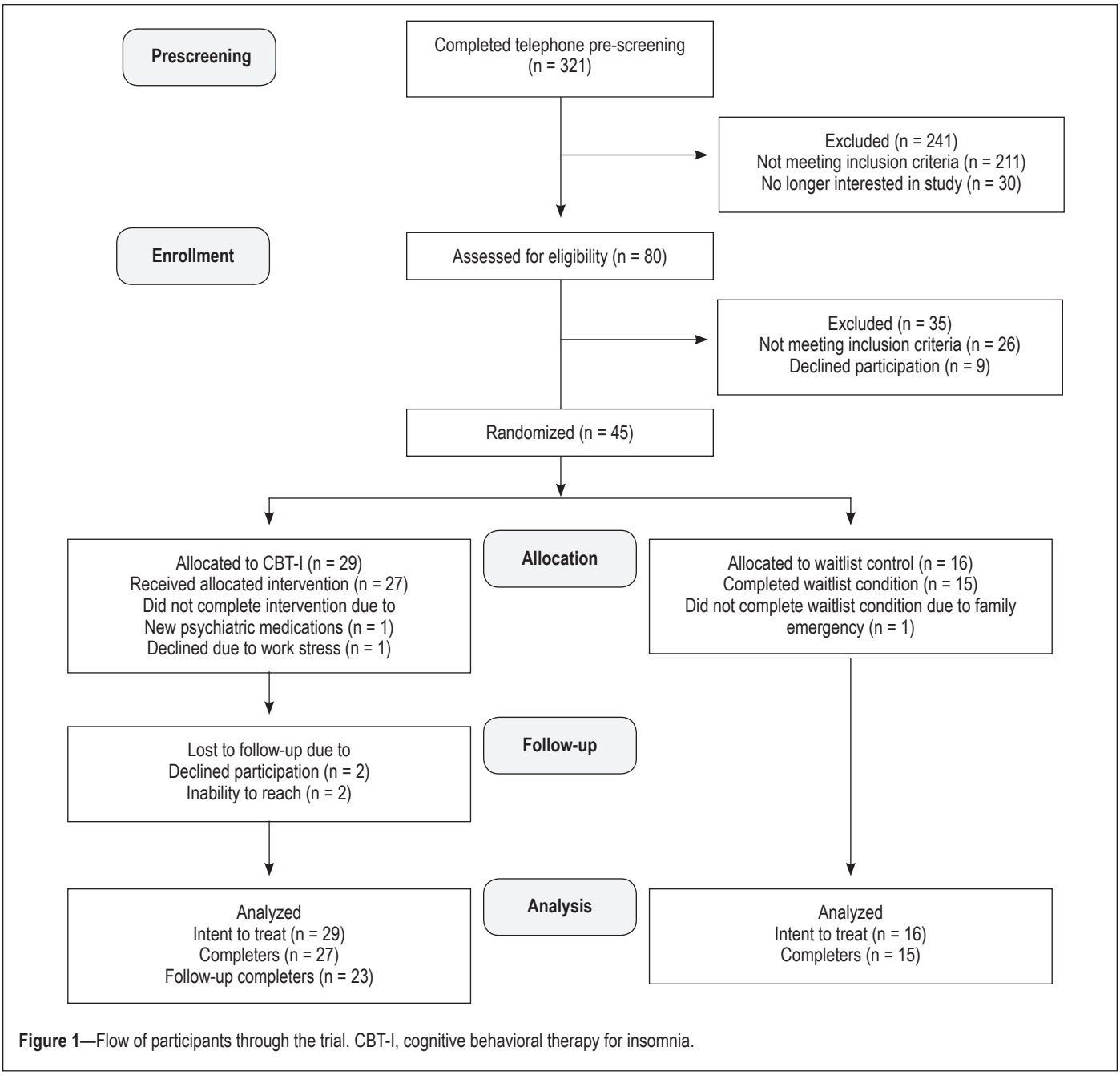

Figure 1—Flow of participants through the trial. CBT-I, cognitive behavioral therapy for insomnia.

Duke Structured Interview for Sleep Disorders

Research diagnostic criteria for insomnia were assessed using a portion of the DSISD.[59] The DSISD is a semistructured interview that assesses research diagnostic criteria for sleep disorders. The DSISD has been shown to have good reliability and validity.[66]

All diagnoses were made by trained clinical interviewers who calibrated their assessments at weekly case consensus meetings, supervised by an experienced PhD-level clinical psychologist.

Sleep Diary

Participants recorded their sleep throughout the study using the sleep diary.[67] The sleep diary was used by the study therapists on a weekly basis to monitor progress and was also an outcome measure. The sleep diary followed the standard recommendations for sleep research.[68] Questions included in the diary allowed for the assessment of sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST), sleep efficiency (SE), and energy level. The sleep diary has been shown to be a reliable estimate[69] and is considered the gold standard subjective measure of sleep.[68]

Polysomnography

Polysomnography recordings were obtained with ambulatory polysomnography using ambulatory recorders (Trackit; Lifelines Ltd., Stockbridge, United Kingdom). These recorders filter and amplify the raw electroencephalogram (EEG) signals, then digitize the signals at 256 HZ and record to a removable hard disk in the EDF file format. The Trackit and associated recording software contain an internal calibration routine to insure that the values recorded in the EDF files truly represent the EEG amplitude. The parameters recorded included an EEG at leads C3, C4, O1, and O2, left and right

electrooculograms (EOG), submental electromyogram (EMG), bilateral anterior tibialis EMGs, and electrocardiogram (EKG) in accordance with standardized guidelines. Electrode impedances were < 5 kohm at the start of the recording. The EEG and EOG leads were referenced to linked mastoids, A1 and A2. Participants were screened for periodic limb movements (using the bilateral anterior tibialis EMG measurements) as well as obstructive sleep apnea, which involved measuring reductions in oronasal airflow with a thermistor, pulse oximetry for detection of oxygen desaturation events, and two channels of respiratory effort using strain gauges to measure chest and abdominal movement during breathing. No participants had an apnea/hypopnea index of 10 or greater (mean = 1.88, standard deviation [SD] = 2.34, range 0.0-7.20). Digitized polysomnography data were imported in TWin software (Grass Technologies, Middleton, WI) for visual scoring, and the data were scored in 30-sec epochs using standard scoring criteria[70] by an experienced registered polysomnography technician who was unaware of participant group and time point. The results were used to generate the following polysomnography indices used in the analyses: WASO, TST, and sleep maintenance (SM) percentage [100 × TST/(TIB-sleep onset)]. We note that SOL was omitted; the polysomnography technician was not in the participants' homes at the time of lights out in order to denote this event for accurate calculation of SOL. Correspondingly, SM rather than SE was used in order to eliminate SOL from the equation. Two nights of recording were obtained at baseline and two consecutive nights after 8 w. In both cases, data were analyzed only from the second night in order to avoid the first-night effect.[71]

Insomnia Severity Index

The ISI is a seven-item measure of perceived insomnia severity.[60] The ISI assesses sleep difficulties and distress and impairment related to the sleep disturbance. Total scores range from 0-28, with a higher score indicative of greater insomnia severity. The ISI has excellent internal consistency (Cronbach $\alpha = 0.74$) and temporal stability ($r = 0.80$), has been validated with both sleep diary and polysomnography,[60] and is sensitive to clinical treatment response.[72] Scoring guidelines consist of: score of 0-7 (no clinical insomnia), 8-14 (subthreshold insomnia), 15-21 (insomnia of moderate severity), and 22-28 (severe insomnia). Remitters are defined as those with a final score below 8.

Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index (PSQI) is a widely-used 19-item broad measure of sleep quality and disturbances over the past month.[73] Scores range from 0-21, with higher scores indicating worse sleep quality. The PSQI has been validated in both healthy and psychiatric patients and has strong psychometric properties.[74]

Epworth Sleepiness Scale

The Epworth Sleepiness Scale (ESS) is an eight-item measure of daytime sleepiness.[75] It assesses the likelihood of falling asleep in common daily situations, with higher scores indicating greater sleepiness. The ESS is a validated measure with high specificity and sensitivity.[75,76]

Pittsburgh Sleep Quality Index-Addendum

The Pittsburgh Sleep Quality Index-Addendum (PSQI-A) assesses disruptive nocturnal behaviors related to PTSD, such as sleep disturbances related to hot flashes, nightmares, and episodes of terror during sleep.[77] The total score ranges from 0 (normal) to 21 (severe). The PSQI-A has demonstrated good internal consistency and convergent validity.

PTSD Checklist

The 17-item PTSD checklist (PCL) is a validated self-report scale for assessing PTSD symptoms.[78] Items correspond to the DSM-IV symptoms of PTSD. Scores range from 17 to 85, with higher scores indicating more severe PTSD symptoms.

Beck Depression Inventory

The Beck Depression Inventory (BDI) is a widely-used 21-item measure assessing the subjective intensity of depression symptoms in the past week, with established validity and reliability.[79-82] The total score ranges from 0-63, with a higher score indicative of more depression symptoms. This measure was included because of the frequent co-occurrence of depression symptoms with both PTSD[83] and insomnia.[10]

Work and Social Adjustment Scale

The Work and Social Adjustment Scale (WSAS) is a five-item measure that assesses functioning in work, home management, social leisure activities, private leisure activities, and relationships with others.[84] Each item is rated on a scale of 0 to 8, with higher scores reflecting greater impairment attributable to insomnia. The WSAS has demonstrated good internal consistency and test-retest correlation

Actigraphy

Participants had their sleep-wake schedule monitored throughout the study with wrist actigraphy (Micro Motionlogger; Ambulatory Monitoring, Inc., Ardsley, NY). Actigraphy is an important adjunctive measure in the diagnosis and treatment of insomnia that can improve the reliability of self-report estimates of sleep.[85] Actigraphy was used by the study therapists on a weekly basis to ensure that participants' self-report was generally accurate (i.e., actigraphy served as a measure of participant accountability). If there was great discrepancy between sleep diary and actigraphy measures, therapists could inquire about the difference, but actigraphy data did not inform interventions or sleep restriction titration. Actigraphy also served as a secondary outcome measure. Actigraphs were initialized and downloaded with the ActMe program (Ambulatory Monitoring, Inc., Ardsley, NY) using the PIM/ZCM/TAT sampling mode in 1-min epochs. The PIM UCSD algorithm was used in ActionW Version 2.7 (Ambulatory Monitoring, Inc.) software to estimate sleep parameters including WASO, SM, and TST. SOL was not included (see rationale in polysomnography description in previous paragraphs).

Treatment Conditions

Cognitive Behavioral Therapy for Insomnia

CBT-I was administered, as is described in detail elsewhere,[86] at the San Francisco VA Medical Center. CBT-I is a highly

structured intervention with core behavioral components of stimulus control and sleep restriction, along with sleep hygiene instructions, a cognitive intervention focused on catastrophic beliefs and attitudes related to sleep, and relapse prevention.

Stimulus control therapy focuses on eliminating environmental cues associated with arousal.[87,88] Individuals are provided with a detailed rationale for the intervention and are instructed to use their bed only for sleep and intimacy, to go to bed only when sleepy, to get out of bed and leave the bedroom when unable to sleep, to return to bed only when ready to fall asleep, and to arise at the same time each morning regardless of previous night's sleep. The goal is to limit the amount of wake time spent in bed, thereby reestablishing a strong association between the bed and sleep.

Sleep restriction therapy also requires individuals to reduce the amount of time they spend in bed, based on the premise that excessive time in bed perpetuates insomnia.[89] Individuals are asked to record in a sleep diary the amount of time they estimate was spent asleep each night. They are then instructed to restrict their time in bed to a degree commensurate with their average total sleep time. In this study, a minimum time in bed rule of 4 h was applied, though therapists prescribed a time in bed restriction of less than 5 h with only one participant. Individuals often experience their usual difficulties with sleep fragmentation during the first few nights and become sleep deprived. Sleep deprivation helps consolidate sleep on subsequent nights, thereby improving SE. As participants show improvements in sleep efficiency as measured by the sleep diary, allowable time in bed can be systematically titrated upward on a week-to-week basis.

Study therapists included three licensed clinical psychologists and one board-certified psychiatrist. The clinical psychologists each provided CBT-I to approximately eight to 10 participants. The psychiatrist provided CBT-I to three participants. Study therapists were trained in person by one of the coauthors (MP), who is also one of the authors of the published treatment manual used for this study. Initial didactic training (covering all core components of CBT-I, session-by- session procedures, common challenges, case examples, etc.) lasted 3 days and was immediately followed by training cases supervised by another treatment manual coauthor (DP). DP continued to provide group supervision (approximately monthly) throughout the duration of the trial, along with ongoing as-needed individual consultation. MP provided an in-person refresher didactic approximately halfway through the trial.

Waitlist Control

The monitor-only waitlist control condition consisted of continuous monitoring of sleep using diary and actigraphy in addition to data collection at baseline, after 4 w, and after 8 w as described previously under Study Design. Participants received weekly telephone or email check-ins from the study coordinator and were offered CBT-I following their completion of the research protocol.

Statistical Analysis

Analysis was carried out using SPSS statistical software Version 19 (IBM Corp., Armonk, NY). The sample size was determined using a power analysis with 80% power to detect a medium effect size in diary-measured outcomes at $P = 0.05$. These criteria yielded a proposed sample size of 45, with randomization of two-thirds of participants to the treatment group and one-third to the waitlist control group, and a consequent recruitment stop rule at n = 45.

Primary outcomes included sleep diary, polysomnography, subjective sleep quality, nightmares, nonsleep PTSD symptoms, depression symptoms, and psychosocial functioning. The secondary outcome was actigraphy. All tests were planned and used two-tailed tests of significance, with $P < 0.05$ values indicating statistical significance. In measures with multiple outcomes (i.e., diary, polysomnography, and actigraphy), a P-value of $0.05/n$ of comparisons was applied to control for the family-wise error rate.

For measures collected daily (i.e., sleep diary and actigraphy) we used linear mixed models in order to treat time as a continuous variable and to include all available data regardless of sporadic missing daily observations. These models included random intercepts for subjects and fixed effects for treatment condition, time, and treatment condition by time interaction.

Repeated-measures analysis of variance (ANOVA) was carried out on measures collected at three time points (baseline, midtreatment, and posttreatment) in both conditions. Analysis of covariance, controlling for baseline score, was conducted to assess posttreatment group differences on measures collected at baseline and posttreatment (i.e., polysomnography, CAPS). Paired *t*-tests were used to compare baseline data to 6-mo follow-up data in the CBT-I group.

RESULTS

Participants included 45 individuals with chronic PTSD and chronic insomnia of whom 29 were randomly assigned to receive CBT-I and 16 to waitlist control. Demographic data for the two groups is presented in Table 1. Participants included 31 women and 14 men between 22 and 59 y old (mean age 37.2 y). Participants were primarily white and most were single, with a mean (SD) education duration of 16.2 (2.78) y. There were more veterans in the waitlist group (n = 6) compared to the control group (n = 3). No other significant differences were seen on any of the demographic variables. The mean (SD) PTSD duration was 18.48 (2.05) y. Forty percent of participants were taking a stable dose of antidepressant medication throughout the study. Twenty percent of participants had comorbid depression, and 51% had another psychiatric comorbidity. The mean (SD) number of comorbidities was 1.09 (0.19).

Treatment Attrition and Adherence

Three participants did not complete the treatment or waitlist period (two in CBT-I, of whom n = 1 dropped out after session two and n = 1 after session 3, and one in waitlist control, with reasons cited as work stress, n = 1; psychiatric crisis/new psychiatric medications, n = 1; and family emergency, n = 1). See Figure 1. We note that the participant in psychiatric crisis chose to complete five additional sessions of CBT-I but data were not collected due to the introduction by the participant's psychiatrist of several new medications. The participants who did not complete the treatment or waitlist period did not differ on demographic variables including age, sex, years of education, race, marital status, or veteran status compared to completers. Four additional participants did not complete 6-mo follow-up (unable to reach, n = 2; declined, n = 2). They did not

Table 1—Sociodemographic and clinical characteristics of participants

Characteristic	CBT-I (n = 29)	Waitlist (n = 16)	Test statistic	All participants
Age, mean (SD), y	37.1 (10.4)	37.3(11.0)	$t(43) = -0.05$	37.2 (10.5)
Sex, no. (%)			$\chi^2(1) = 1.85$	
Male	7 (24.1)	7 (43.8)		14 (31.1)
Female	22 (75.9)	9 (56.3)		31 (68.9)
Education duration, mean (SD), y	16.4 (3.1)	15.9 (2.1)	$t(43) = 0.52$	16.2 (2.8)
Race, no. (%)			$\chi^2(3) = 1.21$	
African American	5 (17.2)	1 (6.3)		6 (13.3)
Asian American	3 (10.3)	2 (12.5)		5 (11.1)
Caucasian	20 (69.0)	12 (75.0)		32 (71.1)
Other	1 (3.4)	1 (6.3)		2 (4.4)
Marital status, no. (%)			$\chi^2(3) = 2.81$	
Single	20 (69.0)	10 (62.5)		30 (66.6)
Married/partnered	6 (20.7)	2 (12.5)		8 (17.8)
Divorced	3 (10.3)	3 (18.8)		6 (13.3)
Separated	0 (0.0)	1 (6.3)		1 (2.2)
Veterans, no. (%)	3 (10.3)	6 (37.5)	$\chi^2(1) = 4.75$	9 (20.0)
PSTD duration, mean (SD), y	20.4 (13.6)	15.0 (13.8)	$t(43) = 0.21$	18.5 (2.1)
Current depression, no. (%)	5 (17.2)	4 (25.0)	$\chi^2(1) = 0.39$	9 (20.0)
Other psychiatric comorbidity, no. (%)	13 (44.8)	10 (62.5)	$\chi^2(1) = 1.29$	23 (51.1)
Total number of comorbidities	0.9 (1.3)	1.4 (1.3)	$t(43) = -1.38$	1.1 (0.2)
Psychotropic medication use, no. (%)	11 (37.9)	7 (43.8)	$\chi^2(1) = 0.15$	18 (40.0)

SD, standard deviation; CBT-I, cognitive behavioral therapy for insomnia; PTSD, posttraumatic stress disorder.

differ on any of the demographic variables compared to those who completed the follow-up.

Digital audio recordings of therapist sessions were assessed for treatment fidelity by DP, a PhD-level licensed clinical psychologist with 25 y of experience in behavioral sleep medicine and a coauthor of the published treatment manual used in this trial. Therapists received high ratings across the constructs assessed (therapist delivery of the individual treatment components, knowledge, attentiveness to the participants, skillfulness, and adherence to the protocol). Mean rating of overall delivery of the therapy components on a 0-10 scale was 9.65 (SD = 0.06, range 7-10), indicating excellent delivery of CBT-I. Therapists were also rated on whether they conducted trauma exposure and/or trauma event related cognitive therapy, on a scale from 0 ('very true') to 10 ('not true'). Mean ratings were 9.99 (SD = 0.01, range 9-10), indicating that therapists did not stray into trauma content during the therapy.

Primary Outcomes: Sleep Diary

CBT-I was superior to the waitlist condition in our sleep diary outcomes (Table 2). Statistical analyses showed significant condition × time interactions. CBT-I produced significantly greater baseline to posttreatment improvements in diary-measured SOL ($F(1,2697) = 20.59$, $P < 0.001$, $d = 0.82$), WASO ($F(1,2695) = 22.75$, $P < 0.001$, $d = 0.93$), SE ($F(1,2710) = 35.89$, $P < 0.001$, $d = 1.06$), TST ($F(1,2711) = 5.25$, $P = 0.022$, $d = 0.30$), and energy level ($F(1,2606) = 68.15$, $P < 0.001$, $d = 0.67$) compared to the waitlist control group (see footnote A). When a P-value cutoff of P = 0.01 was applied to control for the family-wise error rate, all outcomes remained significant except for TST.

Participants in CBT-I also showed significant reductions in mean diary-measured SOL ($t(22) = 6.31$, $P < 0.001$, $d = 1.31$) and WASO ($t(22) = 4.96$, $P < 0.001$, $d = 1.03$), and increases in SE ($t(22) = -7.10$, $P < 0.001$, $d = -1.48$), TST ($t(22) = -3.63$, $P = 0.001$, $d = -0.76$) and energy ($t(22) = -2.19$, $P = 0.039$, $d = -0.46$) from the baseline assessment to the 6-mo follow-up. When a P-value cutoff of P = 0.01 was applied to control for the family-wise error rate, all outcomes remained significant except for energy.

Polysomnography

A univariate analysis of covariance (ANCOVA) was conducted on polysomnography-measured TST with condition (CBT-I, waitlist control) as the between-subjects variable, with baseline polysomnography-measured TST as the covariate (Table 2). There was a significant effect of condition ($F(1,32) = 5.48$, $P = 0.008$, $\eta\rho^2 = 0.15$), with the CBT-I group demonstrating more TST at posttreatment. Univariate ANCOVAs on polysomnography-measured WASO and SM did not yield effects of condition. When a P-value cutoff of P = 0.02 was applied to control for the family-wise error rate, TST remained significant.

Subjective Sleep Quality

Insomnia Severity Index

CBT-I was superior to the waitlist condition in our primary self-report sleep outcomes (Table 3). A repeated-measures ANOVA was conducted on the ISI total score with condition (CBT-I, waitlist control) as the between-subject variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was a significant condition × time

Table 2—Means and standard errors for sleep parameters from sleep diaries, polysomnography, and actigraphy

	Initial treatment			Follow-up
	Baseline	Midtreatment	Posttreatment	6-mo
Sleep diary data				
SOL, min				
CBT-I	49.24 (6.13)	20.74 (3.68)	14.27 (2.27)	12.52 (1.95)
Waitlist control	63.11 (17.31)	47.92 (12.11)	44.31 (11.72)	
WASO, min				
CBT-I	56.35 (7.81)	16.80 (2.61)	13.821 (2.03)	18.71 (4.52)
Waitlist control	71.85 (11.96)	45.85 (8.88)	47.25 (11.45)	
SE, %				
CBT-I	78.68 (2.15)	91.12 (1.42)	93.72 (0.93)	93.72 (1.30)
Waitlist control	73.47 (3.45)	79.89 (2.80)	81.77 (2.47)	
TST, h				
CBT-I	6.56 (0.23)	6.59 (0.29)	7.29 (0.24)	7.70 (0.26)
Waitlist control	6.11 (0.26)	6.49 (0.26)	6.57 (0.19)	
Energy, from 0-100				
CBT-I	41.94 (2.60)	40.05 (4.22)	55.83 (4.24)	53.89 (4.61)
Waitlist control	48.89 (4.14)	46.17 (4.75)	42.73 (5.17)	
Polysomnography data				
WASO, min				
CBT-I	43.75 (7.12)		39.25 (7.73)	
Waitlist control	39.34 (7.48)		57.63 (18.28)	
SM, %				
CBT-I	89.02 (2.26)		91.35 (1.91)	
Waitlist control	91.45 (1.56)		88.14 (3.59)	
TST, min				
CBT-I	6.35 (0.41)		6.94 (0.28)	
Waitlist control	6.77 (0.35)		6.38 (0.33)	
Actigraphy data				
WASO, min				
CBT-I	124.67 (14.80)	107.66 (14.52)	104.88 (14.91)	112.25 (17.28)
Waitlist control	129.40 (18.61)	132.35 (18.45)	118.84 (18.25)	
SM, %				
CBT-I	74.08 (3.02)	75.83 (2.93)	76.93 (3.11)	77.50 (3.04)
Waitlist control	71.55 (3.97)	71.56 (3.89)	73.74 (4.31)	
TST, min				
CBT-I	5.85 (0.31)	5.52 (0.29)	5.84 (0.32)	6.29 (0.30)
Waitlist control	5.57 (0.43)	5.70 (0.50)	5.79 (0.52)	

SOL, sleep onset latency; CBT-I, cognitive behavioral therapy for insomnia; WASO, wake after sleep onset; SE, sleep efficiency; TST, total sleep time; SM, sleep maintenance.

interaction, ($F(2,80) = 19.75$, $P < 0.001$, $\eta\rho^2 = 0.33$). Follow-up tests yielded no significant group difference at baseline but significantly lower scores in the CBT-I participants at midtreatment ($t(40) = -2.27$, $P = 0.029$) and posttreatment ($t(40) = -6.82$, $P < 0.001$). Using the ISI cutoff score of 7 or less indicating no clinically significant insomnia, 41% of participants in CBT-I were classified as remitters whereas 0% of participants in the waitlist control group were classified as remitters. CBT-I participants also showed significant reductions in the ISI score from baseline to the 6-mo follow-up ($t(22) = 7.62$, $P < 0.001$, $d = 1.59$).

The Pittsburgh Sleep Quality Index

A repeated-measures ANOVA was conducted on the PSQI score with condition (CBT-I, waitlist control) as the between-subjects variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was a significant condition × time interaction for PSQI, ($F(2,80) = 22.13$, $P < 0.001$, $\eta\rho^2 = 0.36$; Figure 2). Follow-up tests yielded no significant group difference at baseline, but significantly lower scores in the CBT-I group at midtreatment ($t(40) = -2.13$, $P = 0.039$) and posttreatment ($t(40) = -7.62$, $P < 0.001$). CBT-I participants also showed significant reductions in the PSQI score from the baseline assessments to the 6-mo follow-up ($t(22) = 6.86$, $P < 0.001$, $d = 1.43$).

Epworth Sleepiness Scale

A repeated-measures ANOVA was conducted on the ESS score with condition (CBT-I, waitlist control) as the between-subjects

Table 3—Means and standard errors for self-reported sleep measures, nonsleep posttraumatic stress disorders symptoms, nightmares, and depression symptoms

	Initial treatment			Follow-up
	Baseline	Midtreatment	Posttreatment	6-mo
Sleep measures				
ISI				
CBT-I	18.58 (0.59)	14.07 (0.80)	8.00 (0.76)	8.17 (1.13)
Waitlist control	17.94 (0.60)	17.00 (0.97)	16.60 (0.99)	
ESS				
CBT-I	7.69 (0.81)	10.50 (1.09)	6.13 (0.95)	5.04 (0.98)
Waitlist control	8.44 (0.89)	9.27 (1.08)	10.14 (1.11)	
Nonsleep PTSD symptoms				
PCL				
CBT-I	43.69 (1.85)	37.19 (1.76)	32.96 (1.65)	32.91 (2.05)
Waitlist control	46.19 (2.40)	42.60 (2.88)	39.43 (2.87)	
Nightmares				
CAPS Distressing Dreams Item				
CBT-I	4.38 (0.47)		1.48 (0.43)	2.23 (0.48)
Waitlist control	4.25 (0.48)		1.07 (0.48)	
Depression symptoms				
BDI				
CBT-I	18.55 (1.43)	14.56 (1.19)	13.15 (1.68)	11.82 (1.37)
Waitlist control	21.75 (2.14)	19.00 (2.04)	19.20 (1.80)	

ISI, Insomnia Severity Index; CBT-I, cognitive behavioral therapy for insomnia; ESS, Epworth Sleepiness Scale; PTSD, posttraumatic stress disorder; PCL, PTSD checklist; CAPS, Clinician-Administered PTSD Scale; BDI, Beck Depression Inventory. Lower scores indicate less severe clinical symptoms.

variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was a significant condition × time interaction, ($F(2,70) = 5.74$, P = 0.005, $\eta\rho^2 = 0.14$). Follow-up tests yielded no significant group difference at baseline or midtreatment but significantly lower scores in the CBT-I participants at posttreatment ($t(35) = -2.68$, P = 0.011). CBT-I participants also showed significant reductions in the ESS score from the baseline assessments to the 6-mo follow-up ($t(22) = 3.23$, P = 0.004, $d = 0.67$).

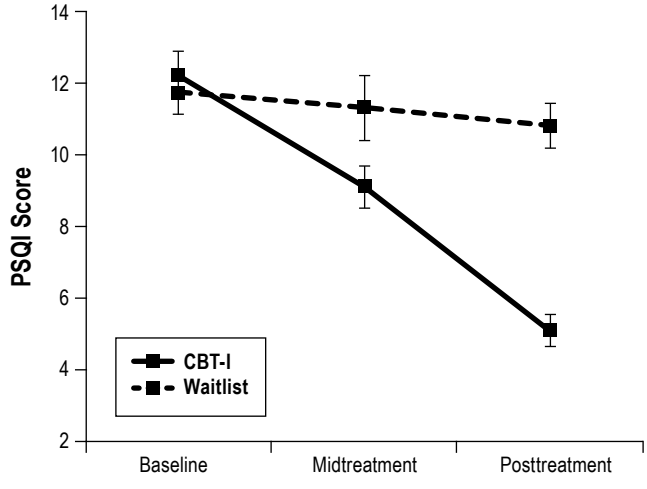

Figure 2—Pittsburgh Sleep Quality Index (PSQI) scores. CBT-I, cognitive behavioral therapy for insomnia. Condition × time interaction, P < 0.001.

Nightmares

Disruptive nocturnal behaviors were assessed using the PSQI-A and the CAPS distressing dreams item. A repeated-measures ANOVA was conducted on the PSQI-A score with condition (CBT-I, waitlist control) as the between-subjects variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was a significant condition × time interaction ($F(2,80) = 9.64$, P < 0.001, $\eta\rho^2 = 0.19$; Figure 3). Follow-up tests yielded a significant difference at baseline ($t(43) = 2.68$, P = 0.004), with the CBT-I participants starting with higher PSQI-A scores, but no difference at posttreatment ($t(40) = -1.69$, P = 0.099). CBT-I participants also showed significant reductions in the PSQI-A score from the baseline assessments to the 6-mo follow-up ($t(22) = 5.14$, P < 0.001, $d = 1.07$; Figure 4).

A univariate ANCOVA was conducted on the posttreatment CAPS distressing dreams item (sum of frequency and intensity of item B2) with condition (CBT-I, waitlist control) as the between-subjects variable, with baseline CAPS distressing dreams item score as the covariate. There was no effect of condition. CBT-I participants showed significant reductions in this item score from the baseline assessments to the 6-mo follow-up ($t(21) = 3.40$, P = 0.003, $d = 0.73$).

Nonsleep PTSD Symptoms

CBT-I was not superior to the waitlist control condition in either the rater or self-report measure of symptom severity.

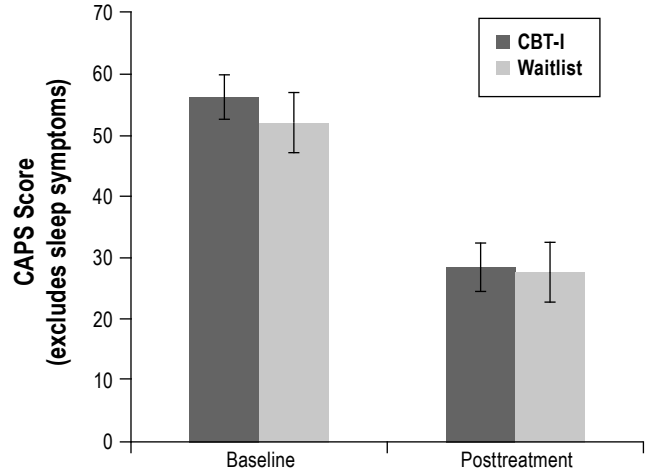

Figure 3—Clinician-Administered PTSD Scale (CAPS) scores. CBT-I, cognitive behavioral therapy for insomnia. Condition × time interaction, P < 0.001.

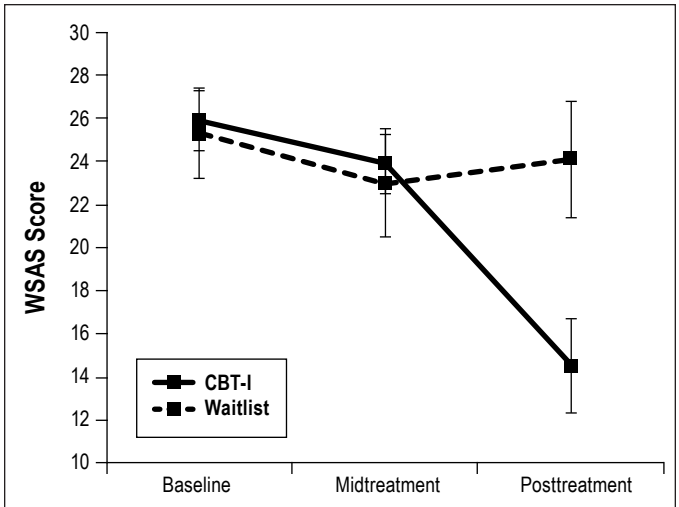

Figure 5—Work and Social Adjustment Scale (WSAS) (impairment) scores. CBT-I, cognitive behavioral therapy for insomnia. Condition × time interaction, P = 0.007.

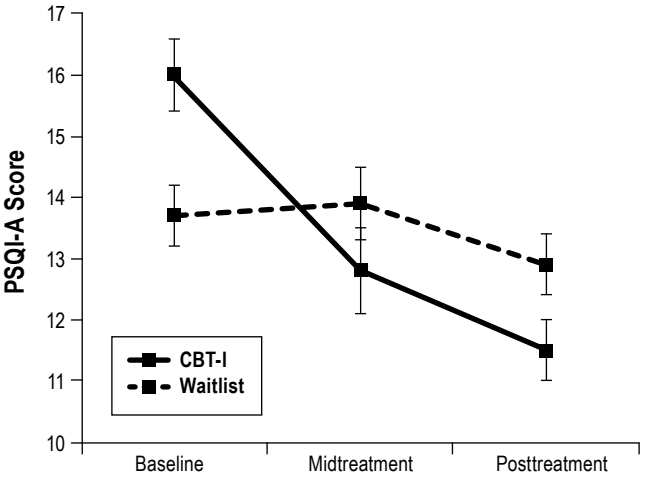

Figure 4—Pittsburgh Sleep Quality Index-Addendum (PSQI-A) scores. CBT-I, cognitive behavioral therapy for insomnia. Condition × time interaction, P < 0.001.

To assess overall PTSD symptoms without the influence of sleep symptoms, CAPS total score was computed without the two sleep symptoms (B2: distressing dreams and D1: difficulty falling or staying asleep). A univariate ANCOVA was conducted on the total CAPS score (excluding sleep symptoms) with condition (CBT-I, waitlist control) as the between-subjects variable, with baseline total CAPS score (excluding sleep symptoms) as the covariate. There was no effect of condition (Figure 2). CBT-I participants showed significant reductions in total CAPS score (excluding sleep symptoms) from the baseline assessments to the six-month follow-up ($t(23) = 6.06$, $P < 0.001$, $d = 1.23$).

Next, overall PTSD symptoms without the influence of sleep symptoms were examined using the PCL (excluding the sleep-related items, #2 and #13). A repeated-measures ANOVA was conducted on the PCL score (excluding sleep items) with condition (CBT-I, waitlist control) as the between-subjects variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was no significant condition × time interaction. There was a main effect of condition ($F(1,39) = 4.41$, $P = 0.042$, $\eta\rho^2 = 0.10$; Table 3), with the participants in CBT-I reporting lower mean PCL scores across the time points compared to the waitlist control group. There was also a main effect of time, with all participants demonstrating lower scores at the midtreatment and posttreatment time points compared to baseline ($F(2,78) = 12.19$, $P < 0.001$, $\eta\rho^2 = 0.24$). CBT-I participants also showed significant reductions in the PCL score from the baseline assessments to the 6-mo follow-up ($t(22) = 3.96$, $P = 0.001$, $d = 0.83$).

Depression Symptoms

A repeated-measures ANOVA was conducted on the BDI total score (excluding sleep item) with condition (CBT-I, waitlist control) as the between-subjects variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was no condition × time interaction. There was a main effect of condition ($F(1,40) = 5.53$, $P = 0.024$, $\eta\rho^2 = 0.12$; Table 3), with the CBT-I participants demonstrating lower scores across the three time points. There was also a main effect of time ($F(1,40) = 5.37$, $P = 0.026$, $\eta\rho^2 = 0.12$), with both groups demonstrating lower scores at midtreatment (but only the CBT-I group demonstrating lower scores posttreatment). CBT-I participants also showed significant reductions in this item score from the baseline assessments to the 6-mo follow-up ($t(22) = 3.30$, $P = 0.003$, $d = 0.69$).

Psychosocial Functioning

A repeated-measures ANOVA was conducted on the WSAS total score with condition (CBT-I, waitlist control) as the between-subjects variable and time (baseline, midtreatment, posttreatment) as the within-subject variable. There was a condition × time interaction ($F(1,40) = 8.13$, $P = 0.007$, $\eta\rho^2 = 0.17$; Figure 5). Follow-up tests yielded no group differences at baseline or midtreatment, but the CBT-I participants had significantly lower scores at posttreatment ($t(40) = -2.71$, $P = 0.010$). CBT-I participants also showed significant reductions from

the baseline assessments to the 6-mo follow-up ($t(22) = 4.90$, $P < 0.001$, $d = 1.02$).

Secondary Outcome: Actigraphy

Statistical analyses showed no significant treatment × time interactions or main effects of condition or time. Participants in CBT-I did not show any differences in mean WASO, SM, or TST from baseline to 6-mo follow-up.

DISCUSSION

We examined the efficacy of CBT-I in individuals with PTSD compared to a waitlist control group. In support of our hypothesis, we observed that CBT-I improved sleep outcomes across sleep diary and polysomnography. According to the sleep diary, individuals randomized to CBT-I demonstrated reduced SOL and WASO and increased SE, TST, and energy compared to waitlist control participants, though we note that TST did not remain significant after applying a strict alpha to control for the family-wise error rate. These effects remained robust at 6-mo follow-up (e.g., TST continued to increase, from 7.3 to 7.7 h). These findings are noteworthy given that insomnia is currently defined based on self-report[59] and some research suggests that sleep diaries more accurately distinguish individuals with insomnia from good sleepers compared to actigraphy.[90]

As measured by polysomnography, the CBT-I group demonstrated more TST at posttreatment compared to the waitlist control group but no difference in WASO or SM. The finding that this objective estimate indicated a mean TST increase of 30 min by the end of the 8-w treatment is compelling, particularly given that one of the core behavioral components of CBT-I (sleep restriction) often results in a temporarily reduced TST that gradually returns to baseline levels or higher after 6 mo.[36] Nonetheless, we are cautious in our enthusiasm given that the data are based on 1 night of measurement (subsequent to acclimation nights) at baseline and posttreatment.

CBT-I participants also reported significant improvements in subjective sleep quality at posttreatment, which remained at 6 mo, based on the three self-report measures: ISI, ESS, and PSQI. These indicate that CBT-I participants experienced improved functioning across a broad array of sleep related constructs including sleep disturbance, daytime sleepiness, and overall sleep quality. In particular, the CBT-I participants had a remission rate (based on an ISI score of less than 7, indicating no clinically significant insomnia) of 41%. We note that this rate is comparable with the 39% remission rate demonstrated in a recent trial of 6-w CBT-I in a sample of adults with persistent insomnia (of which 15% had a comorbid psychiatric disorder).[36] The encouraging remission rates in the current study suggest that CBT-I in PTSD may be as clinically useful as CBT-I delivered to individuals without psychiatric comorbidities.

We included actigraphy as a secondary sleep outcome. In contrast to diary and polysomnography, based on actigraphy the CBT-I group did not show differences in sleep. However, we note that the means were in the predicted direction and that a paired *t*-test on the means in the CBT-I group indicated a significant decrease in WASO from baseline to posttreatment (while the same comparison was not significant in the waitlist condition). In addition, mean TST increased by more than 30 min from posttreatment to 6-mo follow-up.

Hence, CBT-I participants demonstrated improvements across numerous measures of sleep, though there were some variations across diary, polysomnography, and actigraphy. These differences align with previous research that has frequently demonstrated some discrepancies between objective and subjective measures of sleep, particularly in individuals with psychiatric disorders including PTSD.[91-93] Numerous possibilities may explain these differences, such as sleep hyperarousal.[94] As such, it is considered the gold standard to collect data from both objective and subjective sources and regard both as important.[68]

Individuals in the waitlist control group showed smaller improvements in sleep than did the CBT-I group and only demonstrated these improvements based on the sleep diary. A set of *post hoc* paired *t*-tests on mean values of diary variables in the waitlist group indicated that individuals reported significantly increased TST and SE and decreased WASO and marginally decreased SOL ($P = 0.07$) after the 8-w monitor-only period compared to baseline. It is possible that individuals in the waitlist condition reported improvements in these sleep measures as a result of (1) having self-monitored their sleep on a daily basis for 8 w, (2) feeling their sleep was being "observed" via actigraphy, and (3) anticipating the upcoming sleep treatment. The first two possibilities indicate that daily monitoring of sleep could serve as an intervention for insomnia.

The overall finding that CBT-I appears to be a useful treatment that will likely improve the highly prevalent sleep disturbance that occurs in PTSD is noteworthy for several reasons. First, the data answer the theoretical question pertaining to whether the potential unique features of sleep disturbance inherent to PTSD would render individuals unresponsive to CBT-I. For example, nightmares are frequent in PTSD and they differentiate the sleep disturbance in PTSD from the sleep disturbance of other disorders. In addition, it is common for the trauma of PTSD to have occurred at night or in the bedroom, potentially leading individuals to feel unsafe in the sleeping environment. We emphasize that in our trial therapists intentionally did not address any beliefs about the safety of the bed/bedroom in order to avoid introducing elements of CBT for PTSD. Despite these unique aspects of sleep in PTSD, CBT-I was efficacious. These findings augment the theoretical foundation of CBT-I, which presumes a conditioned association between being in bed and feeling anxiously aroused. The findings are in accord with accruing research demonstrating the success of CBT-I in the context of comorbid disorders.[45,95]

Second, CBT-I as a nonpharmacologic treatment may be particularly beneficial to individuals with PTSD given that many individuals are already taking numerous medications and as such CBT-I would not result in further polypharmacy or drug interaction effects. At the same time, data suggest that CBT-I is efficacious regardless of whether individuals are taking medications so it could be applied even when individuals are unable or unwilling to reduce hypnotic medications.[96-98]

Third, CBT-I is a very disseminable treatment.[57] In particular, the CBT-I in our trial emphasized the behavioral components of stimulus control and sleep restriction. Previous research suggests that behaviorally focused versions of CBT-I can be delivered effectively by providers without a background in sleep medicine.[56] Along these lines, the Veterans Health

Administration has begun to implement training of a broad spectrum of clinicians in CBT-I,[55] and as such many providers could use CBT-I to treat the large number of veterans with PTSD.

Fourth, many individuals with PTSD do not seek treatment due to such issues as stigma[99] and avoidance of discussing the trauma. Treatment of insomnia with CBT-I would likely carry less stigma. In addition, CBT-I could serve as an introduction to further treatment (e.g., exposure-based) or potentially even improve some of the nightmare and nonsleep PTSD symptoms, as we discuss in the following paragraphs.

We examined whether CBT-I would improve nightmares, nonsleep PTSD symptoms, depression symptoms, and psychosocial functioning. In regard to nightmares, we observed that active treatment was superior to the waitlist control condition for disruptive nocturnal behaviors as indicated by changes in the PSQI-A scores. The PSQI-A is a broad measure of PTSD-related sleep disturbances, including nightmare items. However, based on the CAPS distressing dreams item, both the CBT-I and waitlist control groups improved posttreatment. The CBT-I group's improvement was durable as measured 6 mo later. Hence, the findings in regard to nightmares are mixed overall. There is initial evidence that CBT-I may be beneficial for nightmares, but the waitlist control group's substantial improvement on the specific nightmare item suggests it is difficult to draw unequivocal conclusions. Nonetheless, given the promising PSQI-A results, clinicians may consider offering a short course of CBT-I as an initial treatment for nightmares that co-occur with insomnia, particularly if there are concerns about the use of a nightmare-targeting medication or a nightmare protocol with some exposure elements.

We also examined whether CBT-I would improve nonsleep PTSD symptoms. Here, both the CBT-I and waitlist control groups showed a large improvement from baseline to posttreatment as evidenced by the CAPS (mean score decrease of 27.5 for the CBT-I group and 24.1 for the waitlist control group) and the PCL, again making it difficult to draw conclusions about the effect of the treatment on non-PTSD symptoms. It is not uncommon for participants in inactive or waitlist control treatments to demonstrate improvements in outcomes such as PTSD symptoms.[100] There are a number of possible explanations for the improvement in the waitlist control group. The waitlist control participants may have derived some relief from the anticipation of the CBT-I treatment they would be offered at the conclusion of the study. In addition, they may have experienced satisfaction from contributing to the development of a treatment for their symptoms. They may also have derived a sense of belonging or social support through the connection to the study, which included meeting with a clinical interviewer, visiting the laboratory at baseline, after 4 w and 8 w, and receiving weekly calls or emails from the study coordinator. Indeed, posttreatment feedback forms indicated a high degree of satisfaction with study staff. Moreover, as discussed earlier, participants in the waitlist control group reported significantly improved diary-measured sleep at the end of the 8 w, raising the possibility that monitoring via sleep diary and actigraphy may be an active treatment for sleep disturbance (though not as potent as CBT-I). If this is the case, the sleep gains may have negated potential differences in nonsleep PTSD outcomes between the CBT-I and waitlist control groups.

Overall, further research is necessary to address whether CBT-I improves nonsleep PTSD symptoms. Future research could include a waitlist control group without self-monitoring, though this design presents data limitations. A future trial could instead include an attention-control condition, though this design raises some of the same nonspecific factors as a waitlist control condition as well as potential issues pertaining to the ethics of disingenuous treatment and treatment credibility. Notwithstanding, a combination of additional research, including varying methods and larger trials, will inform clinicians whether to begin treatment of PTSD with CBT-I in order to address both sleep disturbance and other symptoms, or whether to use CBT-I concurrently with or subsequent to PTSD-focused treatment in order to address the sleep disturbance that commonly remains following first-line PTSD treatment.[27]

Our final questions pertained to whether CBT-I might improve depression symptoms, given the high comorbidity between PTSD and depression, as well as overall psychosocial functioning. In terms of depression symptoms, both groups improved by midtreatment, likely for the same reasons that PTSD symptoms improved in both groups, but only the CBT-I group continued to demonstrate a decrease in scores at posttreatment. These data tentatively suggest that CBT-I may ameliorate depression symptoms that co-occur with PTSD, in accord with previous CBT-I research demonstrating benefits for depression.[101]

In terms of overall psychosocial functioning, the CBT-I group demonstrated a significant drop in psychosocial impairment from baseline to posttreatment that continued to drop at the 6-mo follow-up, whereas the waitlist control group's scores remained flat. This finding that treating sleep disturbance effectively addressed the overall impairment associated with PTSD has important clinical implications, given that individuals with PTSD frequently report difficulties in psychosocial functioning,[102] such as difficulties with close relationships.[103]

Strengths of the current study include the multimethod measurement of sleep, two-arm design, and longitudinal follow-up. Limitations include the small sample size and limited number of nights of polysomnography. In addition, the sample may not have been completely representative of the general PTSD population (e.g., considering participant requirements of current mental health treatment and no alcohol or substance abuse or dependence in the past year).

In summary, CBT-I was efficacious in the treatment of insomnia and disruptive nocturnal behaviors in PTSD, and the improvements in sleep were sustained. The initial evidence regarding CBT-I and nightmares is promising but further evaluation is needed. Most importantly, overall psychosocial functioning improved following CBT-I. Further research that includes larger sample sizes is needed to definitively determine whether nonsleep PTSD symptoms improve as a result of CBT-I. Combined, the results suggest that a comprehensive approach to treatment of PTSD should include behavioral sleep medicine.

FOOTNOTE

A. Cohen's *d* for mixed model comparisons are calculated as mean group difference at posttreatment divided by pooled standard deviation, where means and standard deviations are estimated from the mixed model and therefore are adjusted for

random effects and covariates, including pretreatment scores on the outcome variable.

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