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ORIGINAL ARTICLE

Insomnia Patients With Objective Short Sleep Duration Have a Blunted Response to Cognitive Behavioral Therapy for Insomnia

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Study Objectives: This study examined whether individuals with insomnia and objective short sleep duration <6 h, a subgroup with greater risks of adverse health outcomes, differ in their response to cognitive–behavioral therapy for insomnia (CBT-I) when compared to individuals with insomnia and normal sleep duration ≥6 h.

Methods: Secondary analyses of a randomized, clinical trial with 60 adult participants (n = 31 women) from a single academic medical center. Outpatient treatment lasted 8 weeks, with a final follow-up conducted at 6 months. Mixed-effects models controlling for age, sex, CBT-I treatment group assignment, and treatment provider examined sleep parameters gathered via actigraphy, sleep diaries, and an Insomnia Symptom Questionnaire (ISQ) across the treatment and follow-up period.

Results: Six months post-CBT-I treatment, individuals with insomnia and normal sleep duration ≥6 h fared significantly better on clinical improvement milestones than did those with insomnia and short sleep duration <6 h. Specifically, individuals with insomnia and normal sleep duration had significantly higher insomnia remission (ISQ < 36.5; χ^2[1, N = 60] = 44.72, p < .0001), more normative sleep efficiency (SE) on actigraphy (SE > 80%; χ^2[1, N = 60] = 21, p < .0001), normal levels of middle of the night wake after sleep onset (MWASO) <31 minutes (χ^2[1, N = 60] = 37.85, p < .0001), and a >50% decline in MWASO (χ^2[1, N = 60] = 60, p < .0001) compared to individuals with insomnia and short sleep duration. Additionally, those with insomnia and normal sleep duration had more success decreasing their total wake time (TWT) at the 6-month follow-up compared to those with insomnia and short sleep duration (χ^2[2, N = 60] = 44.1, p < .0001). Receiver–operating characteristic curve analysis found that using a 6-h cutoff with actigraphy provided a 95.7% sensitivity and 91.9% specificity for determining insomnia remission, with the area under the curve = 0.986.

Conclusions: Findings suggest that individuals with insomnia and objective short sleep duration <6 h are significantly less responsive to CBT-I than those with insomnia and normal sleep duration ≥6 h. Using an actigraphy TST cutoff of 6 hours to classify sleep duration groups was highly accurate and provided good discriminant value for determining insomnia remission.

Keywords: cognitive–behavioral therapy, insomnia, short sleep, actigraphy.

Statement of Significance

Chronic insomnia is typically treated with medication, cognitive–behavioral therapy for insomnia (CBT-I), or a combination of the two treatments. Previous reports have shown that individuals with insomnia who sleep less than 6 hours per night on average have significantly greater risks of adverse health outcomes. Some researchers have also hypothesized that these individuals might not respond as well to CBT-I compared to individuals with insomnia sleeping 6 or more hours per night. The current study showed poorer outcomes at the end of treatment and at the 6-month follow-up for individuals with insomnia and short sleep duration <6 h than for those with insomnia and normal sleep duration ≥6 h. An actigraphically measured 6-h cutoff was highly accurate in determining insomnia remission; actigraphy is a useful, cost-effective substitute if PSG is not available for classifying individuals into sleep duration groups. Future research might test whether insomnia patients with short sleep respond better to medication alone or a combination of medication and CBT-I.

INTRODUCTION

While chronic insomnia (10%–15% prevalence in adults)[1] or short sleep (estimates of 9.3%[2]–29.2%[3] prevalence in adults) has each been associated with adverse health outcomes, the combination of insomnia with short sleep duration appears particularly detrimental to individuals' health. Compared to normal sleepers who sleep ≥6.5 h on average, individuals with insomnia who also have short sleep durations <6.5 h have nearly a 3-fold increased risk of mortality; in contrast, relative mortality risk does not appear to be elevated among individuals reporting insomnia and sleep duration ≥6.5 h.[4] Similarly, Vgontzas et al.[5] have shown that men (but not women) with insomnia and objective short sleep duration <6 h have significantly increased mortality rates after adjusting for diabetes, hypertension, and other confounders when compared to individuals with normal sleep duration and no insomnia complaints. Furthermore, in our recently published study examining objective short sleep and associated hypertension risk, we found that individuals with insomnia and objective (but not subjective) sleep durations <6 h were associated with a 3.59 increased risk of reporting hypertension as a current medical problem when compared to individuals with insomnia who had objective sleep durations

≥6 h.[6] Therefore, it appears that untreated insomnia coupled with objective short sleep duration may be associated with significant adverse health consequences.

Based on such findings, Vgontzas and colleagues have proposed the existence of two insomnia phenotypes that differ in etiology, pathophysiology, biological severity, natural course, psychological characteristics, and, possibly, their specific treatment needs.[7,8] The first insomnia phenotype is characterized by physiological hyperarousal (eg, short objective sleep duration), significant medical sequelae, and a persistent course. The second insomnia phenotype is characterized by cognitive–emotional and cortical arousal and a remitting course, without the physiological hyperarousal or significant medical sequelae. Objective short sleep duration <6 h (ie, physiological hyperarousal) appears to be an important distinguisher between these two phenotypes and an important factor to consider during their respective treatments.

The most efficacious treatments for insomnia tend to be biologically based (eg, pharmacotherapy with benzodiazepine receptor agonists), behaviorally based (eg, cognitive–behavioral therapy for insomnia; CBT-I), or a combination of the two treatments.[9,10] Vgontzas et al. have hypothesized that treatment

outcomes might differ as a function of insomnia phenotype, although few studies have examined these differences.[7,8] For instance, individuals with the insomnia phenotype associated with short sleep duration (ie, physiologic hyperarousal) might respond better to biologically based treatments, since selected medications aim to reduce physiological hyperarousal and increase sleep duration.[7,8] On the other hand, individuals with the insomnia phenotype associated with normal sleep duration and increased cognitive arousal might be better served with a behaviorally based approach aimed at decreasing cognitive–emotional arousal, altering unhealthy sleep-related behaviors and beliefs and changing sleep misperceptions.[11–13] Unfortunately, we currently lack studies that have tested whether this hypothesized differential treatment response actually exists in clinical samples.

Insomnia sufferers with objective short sleep duration <6 h seem to represent a biologically more severe insomnia phenotype associated with increased risk of cardiometabolic morbidity.[14–17] It has been surmised that insomnia patients with short sleep duration might not receive the same treatment gains in CBT-I compared to insomnia patients with normal sleep duration (≥6 h).[7,8] This study was conducted to test this assumption; we hypothesized that individuals with insomnia and objective short sleep duration <6 h, hereafter called the "short sleep duration group," would have a poorer treatment response to CBT-I than would individuals with insomnia and objective normal sleep duration ≥6 h, hereafter called the "normal sleep duration group." Additionally, this study looked at whether an actigraphy-determined sleep time provides an equally useful alternative to polysomnography (PSG) to define objective sleep duration groups with high accuracy and good discriminate value for determining insomnia remission.

METHODS

Design
The data presented here were collected as part of a parent study conducted at Duke Medical Center's Sleep Disorder Center (Durham, NC), examining the optimal number of therapist-guided CBT-I sessions required for treating primary sleep maintenance insomnia in a sample of participants without a comorbid mental health or sleep-interfering condition.[18] Participants were randomly assigned to a waiting list (WL) or a treatment condition consisting of 1, 2, 4, or 8 CBT-I sessions with a study therapist. Participants and study therapists were initially blinded to the number of treatment sessions they would receive/deliver to encourage maximum use of the initial CBT-I session. After the initial session, participants and therapists were informed about the treatment condition to which they were assigned. This randomized, parallel group study was reviewed and approved by the Institutional Review Boards of Duke University's Medical Center.

Participants
Participants of the original parent sample (*n* = 86) were recruited between October 1998 and August 2002 primarily through newspaper advertisements. Participants met the following inclusion criteria: (1) between the ages of 40–75 years

with sleep maintenance complaints; (2) met diagnostic criteria for primary insomnia as described by the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition (*DSM*-IV);[19] (3) had a nightly mean wake time after sleep onset (WASO) > 60 min on his or her screening sleep diary; (4) had suffered insomnia >6 months with onset after age 10; and (4) reported one or more poor sleep hygiene practices (eg, taking 3 or more naps/week, varying bed/rising times by >2 h from day to day). Since participants met *DSM*-IV criteria for primary insomnia and reported symptoms for at least 6 months, we can simulate that they would meet *Diagnostic Statistical Manual for Mental Disorders*, fifth edition, criteria for insomnia disorder, which requires at least 3 months of sleep difficulty.[20] Exclusion from the study occurred if any of the following criteria was met: (1) pregnancy; (2) a medical condition that compromises sleep (eg, rheumatoid arthritis); (3) Structured Clinical Interview finding suggesting a major psychiatric disorder[21]; (4) a score <27 on the Folstein Mini-Mental Status Exam (MMSE)[22]; (5) habitual substance abuse or unwillingness to abstain from sleep medications during the study; (6) current use of anxiolytics or antidepressants; (7) periodic limb movements during sleep associated with >15 arousals per hour or an apnea–hypopnea index (AHI) >15 on diagnostic PSG; (8) structured sleep interview findings suggesting a primary sleep disorder other than or in addition to primary insomnia; or (9) PSG-measured sleep time ≥2 times higher than sleep time estimated by the patient for the PSG night. The last item was included because we wanted to rule out individuals with significant subjective sleep misperceptions compared to their objective data, as we have found CBT-I is not particularly effective in those patients. Additionally, we required participants in the current study to have complete actigraphy data at baseline and at least some actigraphy data during or posttreatment. We did not include participants on the WL in this study's analyses. Based on the aforementioned criteria, 26 of the original 86 participants were excluded from our analyses (11 people were assigned to the WL and 15 people did not have adequate actigraphy data), leaving a final study sample of 60 participants.

Sleep Assessments

Polysomnography
Per requirements of the parent study, participants meeting initial selection criteria completed a screening PSG in their homes with an 8-channel Oxford Medilog® analogue recorder. PSG was used for screening purposes only; monitoring included electroencephalography, submental electromyography (EMG), bilateral electro-oculography, nasal/oral airflow, and bilateral anterior tibialis EMG. Although PSG typically includes additional respiratory measures (eg, respiratory effort and oximetry), we felt that airflow monitoring in conjunction with the screening interview would provide reasonable likelihood of excluding sleep apnea sufferers, particularly those with more than mild sleep-disordered breathing. PSG screening measures included the AHI (number of apneas and hypopneas per hour of sleep) and periodic limb movement arousal index (PLMAI; number of periodic limb movement-related EEG arousals per hour of sleep). Participants were included if their AHI and

PLMAI were <15. Total sleep time (TST) data from the screening PSG were not available for use in our analyses; PSGs that did not have respiratory or periodic limb movement concerns were not formally scored, thereby rendering us unable to get an accurate TST for all participants from PSGs.

Actigraphy

Per requirements of the parent study, Actitrac® actigraphs (IM Systems, Baltimore, MD), which monitor movement and activity, were used to assess objective sleep parameters throughout the study. Participants wore the actigraph nightly on their nondominant wrists throughout a 2-week baseline, the 8-week treatment, and 2 follow-up periods (3 and 6 months posttreatment). Previous research has shown that the Actitrac estimates of sleep and wake time in insomnia patients correlate moderately well with analogous measures derived from PSG (Rs = 0.52–0.71) and are sufficiently sensitive to detect sleep improvements resulting from behavioral insomnia therapy.[23] The manufacturer's sleep scoring algorithm was used to derive estimates of time in bed (TIB), sleep onset latency (SOL), total wake time (TWT), TST, and sleep efficiency (SE: [TST/TIB] × 100%) for each night of monitoring.

Sleep Diary

Participants were also asked to maintain records of their sleep each morning during 1 screening week, a 2-week baseline, the 8-week treatment phase, and for 2-week periods at the 3- and 6-month follow-up assessments. Diary entries were hand entered into a computer database for subsequent analyses. The database contained information about each participant's SOL, TWT, TST, SE, and middle of the night wake after sleep onset (MWASO: total time awake between initial sleep onset and the final morning awakening). Participants also indicated on a 5-point scale how rested they felt upon awakening (REST) and their perceived sleep quality (QUAL) based on their previous night's sleep (higher scores indicating more restedness and better sleep quality).

Questionnaire Instruments

Per requirements of the parent study, all participants completed a number of self-report measures designed to provide basic demographic information and assessment of sleep history, sleep complaints, and mood during pretreatment (2-week baseline), midtreatment (end of week 4), the end of treatment (end of week 8), and at two follow-up points posttreatment (3 and 6 months). Data from the following instruments were used in this study.

Insomnia Symptom Questionnaire

The Insomnia Symptom Questionnaire (ISQ) is a 13-item self-report questionnaire designed to assess sleep (eg, sleep onset difficulty and wakefulness during sleep) and waking (eg, daytime fatigue and sleep worries) symptoms of insomnia.[24] Each item is accompanied by a 100-mm visual analog scale (ie, horizontal line) that is labeled "not at all" on the left extreme and "always" on the right extreme. Participants draw a vertical line through the point on each item's analog scale to indicate their responses. The distance from the left end of the line to the participant's vertical mark serves as the analog measure of the degree to which the participant has the symptom noted by the item. The mean score across all 13 items was used to indicate overall insomnia severity. In our previous work, we have found that the ISQ has acceptable internal consistency (Cronbach's α = 0.72) and is sensitive to treatment-related sleep improvements.[13,25] Our previous studies showed a clinical cutoff of a total ISQ score <41 was sufficient to connote insomnia remission with 92% sensitivity and 64% specificity for discriminating normal sleepers from primary insomnia sufferers.[13,25] However, in a more recent unpublished work with a large validation sample, a receiver–operator curve analysis revealed that an ISQ total score <36.5 may be a better benchmark, since this cutoff had 89% sensitivity and 86.5% specificity for discriminating patients with primary insomnia from normal sleepers. A copy of the ISQ has been included in Appendix 1 in supplemental material.

Therapy Evaluation Questionnaire

The Therapy Evaluation Questionnaire (TEQ) is a 7-item scale that uses a 7-point Likert-type scale to assess the credibility of varying CBT therapy "doses" and therapist warmth/competence.[26] Participants completed the first 5 items, which focused on treatment credibility and willingness to participate after their first therapy session prior to knowing how many therapy sessions they would receive (higher scores indicating greater confidence and willingness to undergo treatment). These same five questions were answered at the end of the 8-week treatment as well as 2 additional items addressing therapist warmth and competence (higher scores indicating more therapist warmth and competence). A copy of the TEQ has been included in Appendix 2 in supplemental material.

Treatment

Two licensed male clinical psychologists provided individual CBT-I sessions guided by the study's treatment manual. When the study commenced, therapist 1 (WKW) and therapist 2 (JDE), respectively, had 5 and 17 years of experience working with sleep-disordered patients. The first CBT-I session that each participant received lasted 45–60 min; subsequent sessions provided to those receiving more than one session lasted 15–30 min. Depending on their treatment assignment, participants randomized to CBT-I met with their assigned therapist on 1 (Week 1 only), 2 (Weeks 1 and 5), 4 (Weeks 1, 3, 5, and 7), or 8 (weekly) occasions during the study's 8-week treatment.

All participants assigned to CBT-I treatment had an initial therapy session that included education to correct dysfunctional beliefs about sleep and a behavioral regimen to correct sleep-disruptive behaviors. First, participants listened to an audiocassette recording that provided information about sleep needs and the effects of aging, circadian rhythms, and sleep loss on daily sleep–wake functioning. Next, the therapist provided verbal and written (pamphlet) stimulus control instructions encouraging (1) a standard rising time; (2) exiting bed during extended awakenings, (3) using the bedroom only for sleep and sex; and (4) avoiding daytime naps. The therapist provided the participant with an initial TIB prescription, derived from the average baseline sleep time (from diaries) + 30 min. Each

participant was also given instructions for modifying this prescription throughout treatment as a function of the sleep performance achieved. Participants assigned to more than one CBT-I session received therapist guidance in modifying TIB prescriptions and therapist encouragement of treatment adherence.

Procedures

As part of procedures for the parent study, all recruited candidates had an initial visit with a Research Assistant (RA) who described study procedures, obtained informed consent, and assessed the candidate's cognitive status using the MMSE.[22] Candidates with MMSE scores <27 were excluded and referred for further cognitive evaluation. Candidates also completed pretreatment screening measures, including structured sleep[27] and psychiatric[21] interviews administered by a licensed clinical psychologist, a medical examination by a study physician, a 1-week sleep diary, and a diagnostic PSG.

For those meeting the parent study's inclusion criteria during the pretreatment screen, the RA used a computerized randomization program to assign participants to treatment × therapist "cells" within sex and age (<55 vs. >55 years) strata. The program was designed to limit assignment to the WL control condition so that 8 of every 9 enrollees were initially assigned to an active CBT-I treatment during an 8-week period. Participants were randomly assigned to receive 1, 2, 4, or 8 CBT-I sessions. The randomization program was structured to assign 60% of the treatment recipients to therapist 1 (WKW) and the remaining 40% to therapist 2 (JDE). Of the 60 participants selected for this particular study, 36 (60%) were assigned to receive treatment with therapist 1 and 24 (40%) were assigned to receive treatment with therapist 2.

The RA was responsible for collecting all outcome measures. Participants' age, sex, race, education level, and marital status were obtained from initial demographic forms. Participants' objective sleep was measured using an actigraph worn nightly on their nondominant wrist; subjective sleep was measured using sleep diaries. Objective and subjective sleep data were collected during a 2-week baseline (pretreatment), the 8-week treatment period, and for 2-week periods at 3 and 6 months posttreatment. Previous research by our group has demonstrated that using objective sleep measures to determine sleep duration, rather than subjective measures, is more useful in detecting comorbidity risk associated with insomnia.[6] Therefore, insomnia participants were classified as having short sleep duration <6 h (n = 35) or normal sleep duration ≥6 h (n = 25) using their average total sleep duration gathered from the 2-week baseline period of actigraphy. The cutoff of <6 h, derived from objective sleep monitoring, was used because it has proven optimal for predicting morbidity and mortality among insomnia sufferers.[5,6,15,16]

Global insomnia severity was assessed using mean item scores on the ISQ during the 2-week baseline period (pretreatment), midtreatment (Week 4), the end of treatment (Week 8), and for 2-week periods at 3 and 6 months posttreatment. Insomnia remission criteria at the 6-month follow-up was considered an ISQ total score <36.5.

Treatment credibility of varying CBT-I therapy "doses" was obtained from the mean score on TEQ questions 1–5 at baseline (pretreatment) and at the end of treatment (Week 8); therapist warmth and competence (TEQ questions 6 and 7) was obtained at the end of treatment (Week 8).

Data from actigraphy was used to calculate sleep efficiency, which was examined at the 6-month follow-up to determine whether the treatment resulted in clinically acceptable levels of SE (≥80%). We chose an SE cutoff of 80% based on previous research comparing PSG and different actiwatches,[23,28,29] which found that the actigraph used herein tended to overestimate TWT and underestimate TST. We also examined a number of additional sleep outcomes at the 6-month follow-up. Because previous research has suggested an SOL and MWASO cutoff of ≤31 min is optimal for discriminating insomnia cases from normal sleepers in middle-aged and older adults,[30,31] we computed the proportions of individuals in the short and normal sleep duration groups with mean diary SOL <31 min and MWASO <31 min. We also computed the proportions individuals within each of these groups showing ≥50% decline in diary MWASO and decreases in diary TWT declines <25%, 25%–33%, and 33+%.

Statistical Analyses

Chi-square tests were used to determine whether the two sleep duration groups differed in regard to sex, race, marital status, therapist assignment, or CBT treatment group assignment. One-way analysis of variance analyses were used to determine whether there were significant mean differences between sleep duration groups in regard to their mean ages, education levels, insomnia duration, and baseline ratings on the ISQ and mean of TEQ question (1–5) scores. A cutoff of <6 hours using PSG has been shown to be optimal for predicting morbidity and mortality among individuals with insomnia[5,6,15,16]; however, it is unclear whether this cutoff also applies to actigraphic estimates of TST. Therefore, a receiver–operating characteristic (ROC) curve analysis was used to test the sensitivity and specificity an actigraphically measured 6 h TST cutoff derived from baseline assessment for predicting insomnia remission (ISQ score <36.5) at the 6-month follow-up.

Linear mixed models, using the SAS PROC MIXED procedure (v9.3; SAS Institute, Cary, NC) were used to analyze the effects of the CBT-I intervention on sleep diary and actigraphy measures of SOL, TWT, TST, and SE by objective sleep duration group during a 2-week baseline (pretreatment), the 8-week treatment period, and at 3 and 6 months posttreatment. Sleep diaries also contained information on MWASO, perceived sleep quality, and how rested participants felt upon awakening during the same measurement intervals. Each model controlled for a number of covariates including age, sex, CBT treatment group assignment (1, 2, 4, or 8 sessions), and treatment provider. We tested for the main effects of sleep duration group and time as well as the interaction effect between sleep duration group and time. All available data, including those from participants who subsequently discontinued from the study, were used for the longitudinal mixed-effect analyses. This statistical approach is well suited to manage missing observations, as estimated mixed-effects parameters have shown to yield unbiased estimates of model parameters when outcomes are missing at random.[32] To reduce type 1 error, we applied Bonferroni corrections to the

multiple analyses conducted with subjectively and objectively defined sleep parameters. For the seven analyses conducted with sleep diary data (TWT, SOL, MWASO, TST, SE, QUAL, and REST), we used an adjusted $\alpha = 0.007$ (ie, 0.05/7). For the four analyses conducted with actigraphy (SOL, TWT, TST, and SE) we used an adjusted $\alpha = 0.0125$ (ie, 0.05/4).

Chi-square analyses using baseline and model estimates at the 6-month follow-up were used to determine whether there were significant proportional differences between objective sleep duration groups on clinical improvements such as insomnia remission (ISQ score <36.5), actigraphy SE $\geq$80%, and diary MWASO <31 min. We also used the 6-mo follow-up estimates to determine whether there were group differences between objective sleep duration groups on diary SOL <31 minu, $\geq$50% decline in diary MWASO, and decreased diary TWT (grouped as TWT declines <25%, 25%–33%, and $\geq$33%).

RESULTS

Table 1 provides descriptive characteristics of the 60 enrolled participants. Participants tended to be female (51.7%), white

Table 1—Demographic Characteristics of Insomnia Participants.

Characteristic	Overall sample, N = 60	Objective[a] short sleep (<6 h per night), N = 35	Objective[a] normal sleep (≥6 h per night), N = 25	Test values		
Frequencies, N (%)				χ^2	df	p
Sex				2.61	1	.11
Female	31 (51.7)	15 (42.9)	16 (64)			
Male	29 (48.3)	20 (57.1)	9 (36)			
Race				1.52	3	.68
White	56 (93.3)	32 (91.3)	24 (96)			
Black or African American	2 (3.3)	1 (2.9)	1 (4)			
Hispanic	1 (1.7)	1 (2.9)	0 (0)			
Asian	1 (1.7)	1 (2.9)	0 (0)			
Marital status				3.03	3	.39
Married	42 (72.4)	23 (67.7)	19 (79.2)			
Divorced/separated	7 (12.1)	5 (14.7)	2 (8.3)			
Single, never married	6 (10.3)	5 (14.7)	1 (4.2)			
Widowed	3 (5.2)	1 (2.9)	2 (8.3)			
Therapist assignment				1.14	1	.29
JDE	24 (40)	16 (45.7)	8 (32)			
WKW	36 (60)	19 (54.3)	17 (68)			
CBT-I treatment group				3.69	3	.30
1 session	13 (21.6)	5 (14.3)	8 (32)			
2 sessions	15 (25)	8 (22.9)	7 (28)			
4 sessions	16 (26.7)	11 (31.4)	5 (20)			
8 sessions	16 (26.7)	11 (31.4)	5 (20)			
Means (SD)				F	df	p
Age	56.2 (10.1)	55.7 (10.4)	56.8 (10)	0.30	1,59	.66
Years of formal education	16.4 (2.1)	16.4 (2.1)	16.5 (2.2)	0.09	1,57	.76
Insomnia duration, years	11.9 (11.3)	12.5 (11.5)	11.1 (11.3)	0.20	1,52	.66
ISQ rating at baseline	55.3 (13.3)	56.8 (12.4)	53.2 (14.4)	1.12	1,59	.29
TEQ mean rating at baseline[b]	5.9 (0.7)	6.0 (0.6)	6.0 (0.8)	0.03	1,55	.86

JDE, Jack D. Edinger; WKW, William K. Wohlgemuth; CBT-I, Cognitive Behavioral Therapy for Insomnia; ISQ, Insomnia Symptom Questionnaire; TEQ, Therapist Evaluation Questionnaire.
Missing data on education from two participants, insomnia duration from seven participants, and TEQ from four participants. Mean and SD calculations exclude subjects with missing data.
[a] Based on 2 weeks of actigraphy at baseline (pre-treatment).
[b] TEQ mean at baseline comprised of the average score of the first five TEQ questions focusing on treatment credibility and willingness to participate.

(93.3%), well educated ($M_{education}$ = 16.4 ± 2.1 years), middle-aged (M_{Age} = 56.2 ± 10.1 years), and married (72.4%). Using the TST duration average across a 2-week baseline of actigraphy, participants were divided into two groups: short sleep duration <6 h (n = 35, 58.3%) and normal sleep duration ≥6 h (n = 25, 41.7%). No significant differences were observed between the short sleep and normal sleep duration groups on sex, race, marital status, assigned therapist, CBT-I treatment group, age, years of formal education, years of insomnia duration, or baseline ratings of ISQ, or TEQ. Likewise, posttreatment ratings of the therapist were uniformly positive across both the groups. Figure 1 shows the participant flow from the parent study into this study, including study attrition. The proportion of participants who did and did not return for follow-up did not differ across CBT-I groups (Fisher's Exact p = .25).

Given the systemic differences between measuring TST using PSG versus actigraphy, it is plausible that the 6-h TST cutoff point that dictates "short sleep" might be different for PSG and actigraphy measures. To test this assumption, we conducted an ROC curve analysis to examine the association between baseline actigraphy TST and insomnia remission (ISQ score <36.5) at the 6-month follow-up. The ROC curve is plotted for all TST values; the further the ROC curve lies above the diagonal reference line, the more accurate the test.[33] Assuming it is desirable to obtain a balance between correct identification of true insomnia remission cases and correct exclusion of false positives (ie, people without insomnia remission that are classified as having remitted), our ROC curve showed that as we approach 6 hours, we achieve both high sensitivity (95.7%) and high specificity (91.9%). Another index of accuracy is the area under the curve (AUC); this is the probability that a test result for a randomly chosen positive case will exceed the result for a negative case.

Swets[35] has suggested that AUC values under 0.7 have "low" accuracy, values between 0.7 and 0.9 have "moderate" accuracy, and values greater than 0.9 have "high" accuracy. Our model had high accuracy (AUC = 0.986), which suggests that baseline actigraphy TST was able to reliably predict the probability of insomnia remission at the 6-month follow-up.

Actigraphy

Table 2 provides the least squares means (LSM) and standard errors for all actigraphy measures collected during baseline, Week 8, and the 3- and 6-month follow-ups. Four mixed-model analyses examining actigraphy measures of SOL, TWT, TST, and SE were conducted controlling for the effects of age, sex, CBT treatment group assignment (1, 2, 4, or 8 sessions), and treatment provider. To reduce type 1 error, we applied a Bonferroni correction for a more conservative estimate of significance (adjusted α = 0.0125; ie, 0.05/4). Significant sleep duration group main effects were noted for actigraphy measures of TWT (F = 21.15, p < .0001), TST (F = 86.26, p < .0001), and SE (F = 43, p < .0001). We expected to see a group difference on TST, as we defined our subgroups based on their actigraphy-measured TST. The sleep duration group main effect was not significant for SOL; however, this was expected since participants were selected for the study based on having sleep maintenance concerns rather than sleep onset concerns. In general, participants in the short sleep duration group showed less TST (318.5 min) and more TWT (138.0 min) at baseline compared to those in the normal sleep duration group (TST = 408 min, TWT = 59.4 min). Additionally, those in the short sleep duration group had lower sleep efficiency (70.5%) at baseline compared to those in the normal sleep duration group (79%). Significant time effects were noted for actigraphy
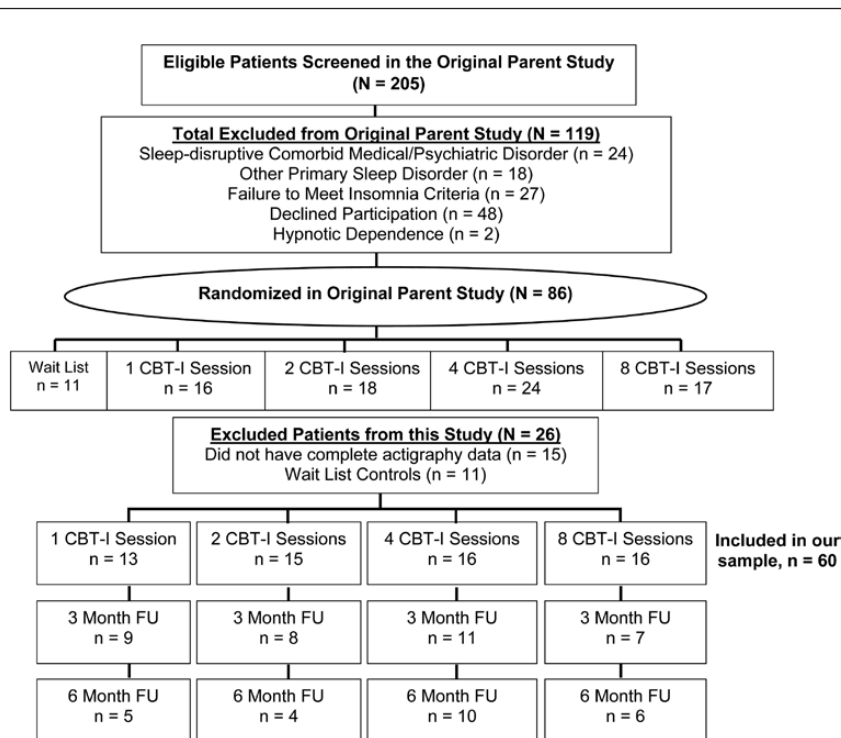

Figure 1—Study flowchart.

Table 2—Least Squares Means and Standard Errors for Sleep Measures Across Time Points.

Measure	Objective[a] short sleep <6 h, N = 35		Objective[a] normal sleep ≥6 h, N = 25	
	LSM estimate	*SE*	LSM estimate	*SE*
Efficiency, %				
Sleep diary				
Baseline	78.3	1.4	72.3	1.6
Week 8	83.4	1.4	82.8	1.8
3-Month Follow-up	84.0	1.9	81.3	2.6
6-Month Follow-up	83.6	2.3	80.1	2.9
Actigraphy				
Baseline	70.5	1.2	79.0	1.4
Week 8	73.3	1.2	80.0	1.5
3-Month Follow-up	72.7	1.6	79.7	1.9
6-Month Follow-up	71.8	1.8	78.8	2.6
Sleep onset latency, min				
Sleep diary				
Baseline	18.0	2.4	26.6	2.7
Week 8	16.0	2.4	18.9	3.0
3-Month follow-up	15.2	3.1	16.3	4.3
6-Month follow-up	15.0	3.9	20.2	4.8
Actigraphy				
Baseline	7.6	1.0	7.4	1.2
Week 8	8.4	1.0	7.4	1.3
3-Month follow-up	6.8	1.3	5.5	1.6
6-Month follow-up	9.7	1.4	6.4	2.1
Total wake time, minutes				
Sleep diary				
Baseline	99.4	6.6	137.9	7.8
Week 8	73.5	6.8	77.6	8.4
3-Month follow-up	70.3	8.9	90.7	12.3
6-Month follow-up	74.4	11.2	91.5	13.8
Actigraphy				
Baseline	138.0	6.5	107.5	7.7
Week 8	118.0	6.6	91.7	8.1
3-Month follow-up	123.7	8.4	98.9	10.3
6-Month follow-up	129.8	9.3	99.0	13.6
Middle wake after sleep onset,[b] min				
Sleep diary				
Baseline	48.0	4.0	59.4	4.6
Week 8	37.5	4.0	29.8	5.0

Table 2—Continued

Measure	Objective[a] short sleep <6 h, N = 35		Objective[a] normal sleep ≥6 h, N = 25	
	LSM estimate	SE	LSM estimate	SE
3-Month follow-up	37.8	5.3	30.1	7.3
6-Month follow-up	40.4	6.7	24.8	8.2
Total sleep time, min				
Sleep diary				
Baseline	358.1	8.3	359.3	9.7
Week 8	362.4	8.5	374.2	10.5
3-Month follow-up	368.2	11.1	384.5	15.5
6-Month follow-up	376.2	14.0	384.0	17.3
Actigraphy				
Baseline	318.5	6.6	408.0	7.9
Week 8	319.0	6.8	363.4	8.4
3-Month follow-up	318.0	8.6	385.8	10.5
6-Month follow-up	318.4	9.6	365.3	13.9
Perceived sleep quality[b,c]				
Sleep diary				
Baseline	2.9	0.11	3.0	0.12
Week 8	3.2	0.11	3.5	0.13
3-Month follow-up	3.2	0.14	3.6	0.20
6-Month follow-up	3.3	0.18	3.6	0.22
Restedness upon awakening[b,c]				
Sleep diary				
Baseline	2.8	0.11	2.9	0.13
Week 8	3.2	0.11	3.3	0.14
3-Month follow-up	3.1	0.15	3.6	0.21
6-Month follow-up	3.4	0.19	3.6	0.23

LSM, least squares mean; SE, standard error.
[a] Based on 2 weeks of actigraphy at baseline (pretreatment).
[b] Collected only on sleep diary, not with actigraphy.
[c] Measured on a 5-point scale (higher scores indicate better sleep quality/more restedness upon awakening).

measures of TST (F = 9.91, p <.0001) and TWT (F = 6.64, p < .0001). Those with short sleep duration continued to have less TST (318.4 min) and more TWT (129.8 min) at the 6-month follow-up compared to those with normal sleep duration (TST = 365.3 min, TWT = 99 min). The time effect was not significant for SOL or SE, and there were no significant sleep duration group × time interactions for any of the actigraphy measures. In general, the short sleep duration group started out at baseline with greater nocturnal wakefulness, less TST, and lower sleep efficiency compared to the normal sleep duration group. This difference persisted to the 6-mo follow-up, suggesting that those in the short sleep duration group had worse end points on objective sleep parameters, which helps explain why we see group effects for TWT, TST, and SE but no group × time interactions.

Sleep Diaries

Table 2 provides the LSM and standard errors for all sleep diary measures collected during baseline, Week 8, and the 3- and 6-month follow-ups. Seven mixed-model analyses examining TWT, SOL, MWASO, TST, SE, QUAL, and REST measured with sleep diaries were examined, controlling for the effects of age, sex, CBT-I treatment group assignment (1, 2, 4, or 8 sessions), and treatment provider. To reduce type 1

error, we applied a Bonferroni correction for a more conservative estimate of significance (adjusted $\alpha = 0.007$; ie, 0.05/7). Significant sleep duration group main effects were noted for sleep diary measures of QUAL ($F = 9.4$, $p = .003$). The sleep duration group main effect was not significant for TWT, SOL, MWASO, TST, SE, or REST. In general, participants in the short sleep duration group had poorer sleep quality ratings than those in the normal sleep duration group. Significant time effects were noted for sleep diary measures of TWT ($F = 12.7$, $p < .0001$), MWASO ($F = 9.17$, $p < .0001$), TST ($F = 3.47$, $p = .0002$), SE ($F = 7.26$, $p < .0001$), QUAL ($F = 2.72$, $p = .003$), and REST ($F = 3.21$, $p = .0005$). The time effect was not significant for SOL. In general, participants' TWT and MWASO decreased significantly from baseline to the 6-month follow-up, whereas TST, SE, sleep quality, and restedness upon awakening significantly increased from baseline to the 6-month follow-up. Significant sleep duration group × time effects were noted for sleep diary measures of TWT ($F = 3$, $p = .001$) and MWASO ($F = 9.17$, $p = .0006$). There were no significant sleep duration group × time interactions for sleep diary measures of SOL, TST, SE, QUAL, or REST. In general, participants in the short sleep duration group reported less TWT at baseline, Week 8, and follow-up time points than did those in the normal sleep duration group; however, the latter group showed a 46.4-min decline in TWT from baseline to the 6-month follow-up, whereas the former group reported only a 25-min decline in this measure over the same time period. With regard to MWASO, those in the short sleep duration group had less MWASO (48.0 min) at baseline compared to those with normal sleep duration (59.4 min), but this pattern was reversed during treatment and follow-up. Those in the short sleep duration group reported only a 7.6-min decline in MWASO from baseline to the final follow-up assessment, whereas those in the normal sleep duration group reported a 34.6-min decline in MWASO over the same time period.
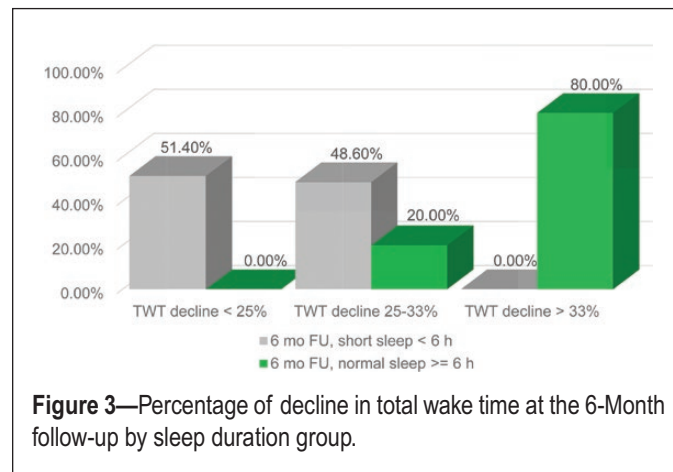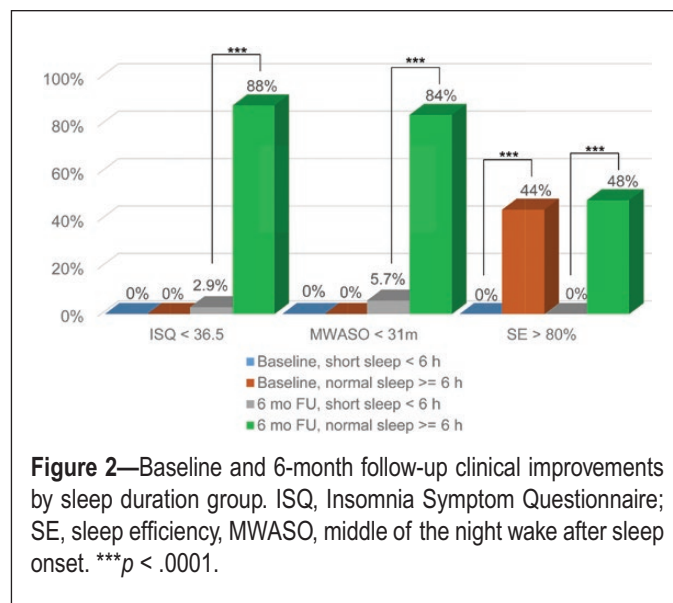
Clinical Improvements From Baseline to Follow-Up

At baseline, no one in the short and normal sleep duration groups had an ISQ score <36.5 or MWASO <31 min; however, at the 6-month follow-up, individuals in the normal sleep duration group showed significantly more insomnia remission, defined as an ISQ score <36.5 ($\chi^2[1, N = 60] = 44.72$, $p < .0001$), and significantly more MWASO improvement, defined as MWASO <31 min ($\chi^2[1, N = 60] = 37.81$, $p < .0001$) and a ≥50% decline in MWASO ($\chi^2[1, N = 60] = 60$, $p < .0001$), compared to the short sleep duration group. With regard to sleep efficiency at baseline, the normal sleep duration group had significantly more individuals with actigraphy SE ≥80% ($\chi^2[1, N = 60] = 18.86$, $p < .0001$) compared to the short sleep duration group and continued to have significantly more individuals with actigraphy SE ≥80% ($\chi^2[1, N = 60] = 21$, $p < .0001$) at the 6-month follow-up. Of particular interest was that at the 6-month follow-up, 0% of the short sleep duration group had actigraphy SE ≥80%, but 48% (12 of 25) of the normal sleep duration group achieved actigraphy SE ≥80%. Figure 2 shows baseline and 6-month follow-up clinical improvements on ISQ-determined insomnia remission, diary MWASO, and actigraphic sleep efficiency for each sleep duration group.

TWT improvements at the 6-month follow-up were significantly different between the sleep duration groups, $\chi^2(2, N = 60) = 44.1$, $p < .0001$. As seen in Figure 3, the normal sleep duration group had more success decreasing their diary TWT by the 6-month follow-up than did those in the short sleep duration group. With regard to SOL, all participants in both the groups reported a mean diary SOL of less than 31 min by their study end points. This result was expected, as participants were selected for sleep maintenance (and not SOL) issues at the start of the study.

DISCUSSION

Vgontzas et al. have proposed that varying insomnia phenotypes might respond differently to insomnia treatment; those experiencing insomnia with short sleep duration might respond better to biological interventions,[7,8] whereas those with insomnia and a more normal sleep duration might have a more positive response to a behavioral interventions targeting cognitive–emotional arousal, altering unhealthy sleep-related behaviors and beliefs, and changing sleep misperceptions.[11–13] However, this hypothesis has remained untested. This study provides initial support for this contention through a comparison of CBT-I responsiveness of individuals with insomnia and objective


Figure 2—Baseline and 6-month follow-up clinical improvements by sleep duration group. ISQ, Insomnia Symptom Questionnaire; SE, sleep efficiency, MWASO, middle of the night wake after sleep onset. ***$p < .0001$.

Figure 3—Percentage of decline in total wake time at the 6-Month follow-up by sleep duration group.

short sleep duration <6 h ("short sleep duration group") or insomnia and objective normal sleep duration ≥6 h ("normal sleep duration group"). This study also provides support that TST obtained from actigraphy can be used to accurately classify individuals into sleep duration groups using the previously established PSG cutoff of 6 h.

Our results showed that the short sleep duration group was generally less responsive to CBT-I than was the normal sleep duration group. Six months after treatment, those in the short sleep duration group reported significantly less insomnia symptom remission, fewer improvements in sleep efficiency and TWT, and more difficulties with MWASO compared to the normal duration group. Thus, in most respects, the short sleep duration group showed a blunted treatment response. Additionally, we found the actigraphically measured 6 h TST cutoff provided good discriminant value in determining insomnia remission (95.7% sensitivity, 91.9% specificity; AUC = 0.986) and may represent a useful, cost-effective substitute for classifying individuals into sleep duration groups if PSG is not available.

Individuals in the short sleep duration group began and ended treatment with less overall TST and saw no significant change in their posttreatment TST when compared to those in the normal sleep duration group. This is consistent with Vgontzas' description of the first insomnia phenotype that is associated with physiological hyperarousal, short sleep duration, and a persistent, unremitting course. Additionally, the short sleep duration group tended to exhibit a consistent misperception of their sleep (as measured by sleep diaries) compared to their objective, actigraphy reports. As seen in Figure 4, those with short sleep duration overestimated their TST at the beginning of treatment and continued to overestimate their TST through the end of treatment and follow-up. The lack of TST change in the short sleep duration group might also reflect a "floor effect" of sleep restriction, as sleep restriction guidelines do not suggest restricting sleep below 5 h/night. Given the TST measurement differences between sleep diaries and actigraphy, one might question whether using actigraphic TST would be more useful when applying time-in-bed prescriptions. However, as mentioned earlier, clinical convention usually places a lower limit of 5 h in bed. We also know from previous data that insomnia with short sleep duration is associated with multiple medical morbidity outcomes,[6,15] so further sleep restriction might augment this risk. Previous research has also shown that strictly applied sleep restriction can lead to notable daytime impairments,[35] so there would be potential safety risks if we were to restrict our short sleep group even further. Finally, sleep restriction has always been based on sleep diaries, with no evidence supporting the use of actigraphy to make such recommendations. Thus, using actigraphy TST to guide sleep restriction/TIB prescriptions for individuals with insomnia and short sleep duration remains a questionable practice.

On the other hand, actigraphy readings from individuals in the normal sleep duration group showed a TST decrease over time, while sleep diary reports showed a TST increase over time. This discrepancy is likely due to sleep misperception and is best represented in Figure 5. In the beginning of the treatment, the normal sleep duration group underestimated their TST; however, as treatment progressed through follow-up, their TST reports became more accurate with a slight overestimation.
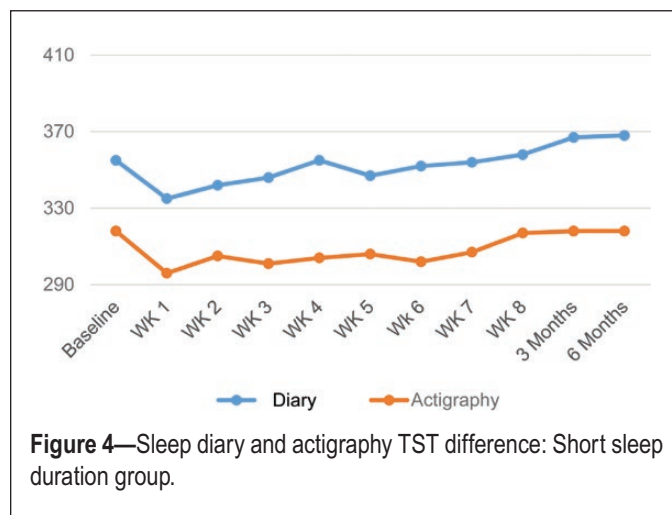
Figure 4—Sleep diary and actigraphy TST difference: Short sleep duration group.
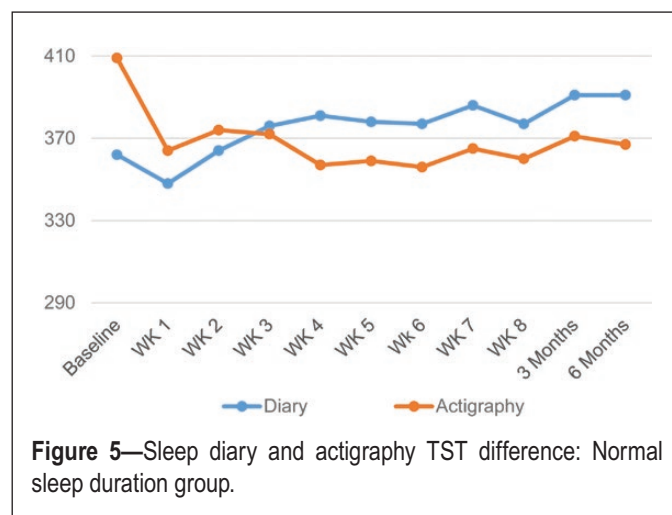
Figure 5—Sleep diary and actigraphy TST difference: Normal sleep duration group.

Although having a decreased TST during treatment may seem counterintuitive to achieving greater insomnia remission, past research supports this trend of decreased TST during treatment. In a 2011 study comparing brief behavioral treatment to an information control condition in older adults with chronic insomnia, researchers found that those receiving brief behavioral treatment had greater insomnia remission despite having a significantly greater reduction in actigraphy-measured TST from pre- to posttreatment.[36]

Our findings using actigraphy align with previous hypotheses that insomnia patients with objective short sleep duration of less than 6 h/night might not receive the same treatment gains in CBT-I compared to insomnia patients with a more normal sleep duration of at least 6 h/night.[7,8] It is plausible that biologically based treatments such as pharmacotherapy, used alone or in combination with CBT-I, might provide greater treatment benefits to individuals with insomnia and short sleep duration <6 h. As such, future research to test this hypothesis as an extension of findings presented in this article seems warranted.

Past and current insomnia nosologies have failed to find a reliable way to discriminate among different types of insomnia. This may be, in part, due to our lack of biomarkers or objective measures we might consistently employ to make such a distinction. Practice guidelines currently dissuade the use of PSG for routine differential diagnosis or severity assessment

of insomnia[37]; however, emerging research evidence supports the use of objective measures of sleep to differentiate insomnia phenotypes associated with morbidity risks.[5–7,15,16,38] This study provides further support for the use of objective sleep measures to aid in the diagnosis and the matching of treatment to distinctive insomnia disorder subtypes.

Admittedly, this study had several limitations that merit consideration. Recruitment for this study was limited to middle-aged and older adults with primary sleep maintenance insomnia. As a result, findings may not generalize to younger adults, those with exclusively sleep onset complaints, or those with comorbid medical/psychiatric conditions. The CBT-I model we tested was delivered using individual sessions; different results may have been obtained if we delivered the CBT treatment as group sessions or individual sessions supplemented by either interactive Internet-based interventions or programmed self-help. Our screening PSG lacked the full respiratory montage commonly used to screen out those with moderate to severe sleep disordered breathing. It is, therefore, possible that this montage was not effective for screening out all with moderate to severe sleep apnea. Additionally, the objective sleep parameters were collected using actigraphy without PSG confirmation of obtained measures. We recognize that in-home test recordings not restricted to 8 hours of TIB (as is typically done in PSG studies) can make for substantially different methodology compared to PSG and may result in somewhat different TST measurements. When looking at previous research comparing PSG to different actiwatches,[23,28,29] we found that the particular actiwatch used in this study tended to *underestimate* sleep duration and overestimate total wake time compared to PSG. It is plausible that some insomnia individuals with normal PSG sleep duration (eg, sleeping 6.25 h/night) could have been misclassified into our short sleep duration group based on their actigraphy reading (eg, their watch underestimating their sleep and recording less than 6 h of sleep). However, if they were misassigned in this way, our short sleep duration group might actually represent a more conservative estimate of individuals with short sleep, thereby making our group difference findings at the 6-month follow-up underestimate the differential treatment responses of short and normal sleep duration groups. Furthermore, previous reports support that actigraphy may be a useful, ecologically valid way to assess sleep patterns in one's habitual sleep environment[39–42] and that actigraphy might be a cost-effective, simpler method (compared to PSG) to obtain sleep duration in clinical settings.[7] Finally, our follow-up data at 3- and 6 months suffered from a high rate of attrition. We suspect this was due to a lack of participant incentives for completing the follow-up assessments. To account for this missing data, we used a mixed-effect model that uses a technique called maximum likelihood estimation to estimate the model parameters and standard errors.[43] Previous work examining how to best handle missing data in sleep disorder trials found that mixed effect models were able to better accommodate missing data compared to other common approaches, such as complete-case analysis and last observation carried forward.[44] Nonetheless, we feel that this study has merit, since it provides initial insights into differential treatment response to CBT-I based on objective sleep duration group.

In conclusion, ours results indicate that individuals with insomnia and objective short sleep duration <6 h tended to have a blunted treatment response to CBT-I compared to individuals with insomnia and objective normal sleep duration ≥6 h. Results also suggest that actigraphy was highly accurate and provided good discriminant value in determining insomnia remission and may represent a useful, cost-effective substitute if PSG is not available for classifying individuals into sleep duration groups. This study extends the previous literature that perhaps individuals with insomnia and objective short sleep duration represent a more biologically severe insomnia phenotype such that they are associated with adverse health outcomes and more specific treatment needs compared to those with insomnia that have longer average sleep durations. As such, our findings seem important to future revisions of our insomnia nosologies, as objectively measured sleep might find a place in their diagnostic criteria for differentiating phenotypes with distinctive characteristics, clinical courses, and treatment needs.

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SUPPLEMENTARY MATERIAL

Supplementary Material is available at *SLEEP* online.

SUBMISSION & CORRESPONDENCE INFORMATION

DISCLOSURE STATEMENT