Does Cognitive-Behavioral Insomnia Therapy Alter Dysfunctional Beliefs About Sleep?

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Study Objectives: This study was conducted to exam the degree to which cognitive-behavioral insomnia therapy (CBT) reduces dysfunctional beliefs about sleep and to determine if such cognitive changes correlate with sleep improvements.

Design: The study used a double-blind, placebo-controlled design in which participants were randomized to CBT, progressive muscle relaxation training or a sham behavioral intervention. Each treatment was provided in 6 weekly, 30-60-minute individual therapy sessions.

Setting: The sleep disorders center of a large university medical center.

Participants: Seventy-five individuals (ages 40 to 80 years of age) who met strict criteria for persistent primary sleep-maintenance insomnia were enrolled in this trial.

Interventions: N/A

Measurements and Results: Participants completed the Dysfunctional Beliefs and Attitudes About Sleep (DBAS) Scale, as well as other assessment procedures before treatment, shortly after treatment, and at a six-month follow-up. Items composing a factor-analytically derived DBAS short form (DBAS-SF) were then used to compare treatment groups across time points. Results showed CBT produced larger changes on the DBAS-SF than did the other treatments, and these changes endured through the follow-up period. Moreover, these cognitive changes were correlated with improvements noted on both objective and subjective measures of insomnia symptoms, particularly within the CBT group.

Conclusions: CBT is effective for reducing dysfunctional beliefs about sleep and such changes are associated with other positive outcomes in insomnia treatment.

Key words: Cognitive-behavioral therapy; primary insomnia; dysfunctional beliefs and attitudes about sleep

INTRODUCTION

DURING THE PAST 30 YEARS, INSOMNIA TREATMENT HAS BENEFITTED BY THE INCREASING RECOGNITION OF THOSE MECHANISMS WHICH SUSTAIN SUCH SLEEP DIFFICULTY. Although many insomnia complaints are traceable to underlying medical (e.g., thyroid, chronic pain disorders), psychiatric (e.g., depression), or primary sleep disorders (e.g., central sleep apnea, restless legs syndrome), approximately one out every five insomnia patients presents with persistent primary insomnia (PPI) which endures in the absence or independent of such causes.[1-4] PPI traditionally has been attributed to perpetuating factors such as conditioned bedtime arousal, adherence to erratic sleep-wake schedules, daytime napping, spending too much time in bed, and/or simply "trying too hard to sleep."[5-8] Moreover, various behavioral treatments designed to eliminate heightened anxiety/arousal (e.g., progressive relaxation, imagery training, meditation) or sleep-disruptive habits (e.g., stimulus control, sleep restriction therapy, sleep hygiene) have proven highly efficacious and served to support the importance of these emotional and behavioral mechanisms in sustaining PPI.

Over the past decade, efforts to improve our understanding and management of PPI have led to the scrutiny of additional cognitive factors which might perpetuate this condition. In this regard, Morin et al.[9] have suggested that dysfunctional beliefs and attitudes about sleep may underlie and support anxiety and habits that disrupt the sleep process. For example, the dysfunctional belief that there is little one can do about poor sleep may help sustain sleep-related anxiety, whereas the belief that one should try to catch up for lost sleep may lead to practices such as remaining in bed beyond the usual rising time or subsequent daytime napping. In either case, the dysfunctional belief leads to a response that contributes to the on-going sleep difficulty. Given this rationale, sleep-related cognitions may serve as important perpetuating mechanisms which merit specific attention in the omnibus management of PPI complaints.

Initial support for this contention comes from group comparative studies as well as a limited number of clinical trials which have included specific cognitive interventions. Several studies[9-11] have shown that PPI sufferers report more negative sleep-related cognitions on questionnaires designed to measure such constructs than do non-complaining normal sleepers. Moreover, the results of these studies suggest that sleep-related cognitions might contribute to actual sleep disturbance and subjective sleep appraisals. In addition, behavioral insomnia treatments, which include strategies (e.g., sleep education, cognitive restructuring) specifically designed to correct patients dysfunctional beliefs and attitudes about sleep, have proven more efficacious than pharmacotherapy, relaxation therapy, medication placebo, or sham behavioral therapy among samples of PPI patients.[12-15] Unfortunately, no studies to date have shown that dysfunctional sleep-related cognitions are specifically and favorably altered by these cognitive-behavioral treatments. As a result, the relative degree to which dysfunctional sleep-related beliefs change in response to cognitive-behavioral therapy (CBT) and the relation of such changes to improvements in core insomnia symptoms, per se, have yet to be explored.

The current study, performed in the context of a large clinical trial, was conducted to test our hypotheses concerning the nature and potential importance of cognitive changes occurring among patients treated with CBT. Specifically, we hypothesized: (1) CBT-treated PPI patients would show significantly larger pre

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Table 1—Demographic characteristics and therapist assignment for the three treatment groups

Characteristic	CBT	RT	Placebo	Total
Age - Years: Mean (SD)	55.8 (12.1)	54.5 (10.2)	55.7 (9.5)	55.3 (10.5)
Sex - Females / Males	11 / 14	11 / 14	13 / 12	35 / 40
Education - Years: Mean (SD)	16.4 (3.6)	16.3 (3.3)	16.7 (2.7)	16.5 (3.2)
Marital Status				
Married	18	19	17	54
Not Married	7	6	8	21
Yrs. of Insomnia: Mean (SD)	13.0 (12.2)	13.2 (12.3)	14.8 (11.5)	13.6 (11.9)
Current Hypnotic Use				
None	21	16	21	58
< once per week	0	2	3	5
1 - 4 times per week	4	6	1	11
> 4 times per week	0	1	0	1
Therapist Assignment				
Male Therapist - total (F,M)	12 (5, 7)	13 (5, 8)	13 (7, 6)	38 (17, 21)
< 55 yrs. old - total (F,M)	6 (3, 3)	7 (3, 4)	7 (3, 4)	20 (9, 11)
> 55 yrs. old - total (F,M)	6 (2, 4)	6 (2, 4)	6 (4, 2)	18 (8, 10)
Female Therapist - total (F,M)	13 (6, 7)	12 (6, 6)	12 (6, 6)	37 (18, 19)
< 55 yrs. old - total (F,M)	7 (3, 4)	7 (3, 4)	7 (3, 4)	21 (9, 12)
> 55 yrs. old - total (F,M)	6 (3, 3)	5 (3, 2)	5 (3, 2)	16 (9, 7)

to post-treatment reductions in their dysfunctional beliefs about sleep than would patients treated with either relaxation therapy (RT) or a sham behavioral placebo control (PC); (2) comparisons of CBT- and RT treated groups at a six-month post- treatment follow-up would show that CBT recipients achieved larger reductions (relative to pre-treatment levels) in dysfunctional beliefs about sleep than did RT recipients; and (3) treatment related changes in these dysfunctional cognitions would be significantly correlated with treatment-related improvements on other outcome measures. To test these hypotheses, we used a recently developed short form of the Morin et al.[9] Beliefs and Attitudes About Sleep Scale to compare the changes in dysfunctional sleep-related cognitions shown by groups of PPI patients treated with CBT, RT, or PC. Subsequently, we conducted correlational analyses to test whether changes on the shortened DBAS correlated with pre to post-treatment improvements noted from polysomnography, sleep logs and a global insomnia symptom questionnaire.

METHODS

Design

This study used a double-blind, placebo-controlled, randomized group design. Each patient enrolled in the study was randomly assigned to one of three treatment conditions (CBT, RT, or PC) and to one of two behavioral therapists (one female, one male). All study participants were kept blind to the study hypotheses. Although they were told that they had a one in three chance of being assigned to the placebo treatment, they were not told which treatment was the PC. The therapists were kept blind to the study hypotheses and to the fact that one of the treatments they were trained to administer was a sham intervention. Therapists were also blind to study participants' responses (scores) on the questionnaires used to measure changes in sleep-related cognitions and subjective insomnia symptoms. The study protocol was approved by the institutional review board of our university medical center, and all study patients provided written informed consent at the time of volunteering to participate. Upon enrollment, participants received information about the therapist blinding from the first author, who instructed them to avoid informing the therapists about the inclusion of a PC condition.

Study Participants

Study candidates were recruited through newspaper advertisements and via face-to-face solicitation of those patients who presented to our sleep disorders center for treatment of their insomnia. Willing participants were considered for inclusion if they: (a) were between 40 and 80 years of age; (b) met DSM-III-R16 criteria for PPI via a Structured Interview for Sleep Disorders (SIS-D);[17] (c) had a mean wake time after sleep onset (WASO) >60 minutes per night as shown by one week of sleep log monitoring; (d) experienced the onset of insomnia after age 10; (e) had a history of insomnia >6 months duration; (f) had a history of one or more poor sleep hygiene practices such as taking >3 naps/week, varying bed times or wake-up times by >2 hours from day to day, or lying in bed awake for extended periods (>30 min.) on three or more nights per week; and (g) produced a subjective estimate of total sleep time that was at least 50% of the actual sleep time recorded during an initial diagnostic polysomnogram.

Excluded from the study were (a) pregnant women; (b) individuals with terminal illnesses; (c) individuals with medical conditions (e.g., thyroid disease, rheumatoid arthritis) that compromise sleep; (d) individuals who, on the basis on a Structured Clinical Interview[18] (SCID- P) met DSM-III-R[16] criteria for a major Axis I psychiatric disorder; (e) habitual substance abusers and persons unwilling to abstain from sleep medications during the study; (f) patients on anxiolytics or antidepressants; (g) indi-

viduals with a periodic limb movement arousal index >15 arousals per hour of sleep (from diagnostic PSG); (h) individuals with 15 or more episodes of sleep apnea per hour (from PSG); and (i) persons who reported histories of little or no sleep and who under-estimated total sleep time by 50% or more during an initial diagnostic PSG study.

Through use of our recruitment procedures and selection criteria, we were able to enroll 75 (35 women, 40 men) individuals with primary sleep-maintenance insomnia. A stratified random assignment procedure (described later) was used to assign study patients to treatment conditions and therapists. Table 1 presents some basic demographic and clinical features of these individuals and shows that the subgroups assigned to the three treatment conditions and two therapists were comparable. Statistical comparisons (one-way ANOVA or chi square) showed no disproportionate distribution of participant types or age groups across the various treatment condition x therapist "cells" of the study.

Dysfunctional Sleep-Related Cognitions

The primary measures of interest for this report were derived from the Morin et al.[9] Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS) which participants completed at baseline (i.e., pre-treatment), during a post-treatment assessment, and again at a six month follow-up time point. In its original form, this instrument contains 28-items designed to reflect common myths and dysfunctional beliefs about sleep. Each of the items is accompanied by a 100 mm visual analog scale which respondents use to indicate their degrees of agreement. As originally proposed, the DBAS was assumed to contain five rationally derived subscales purported to measure: (1) attributions about the effects of insomnia (2) perceptions of loss of control and unpredictability of sleep; (3) perceived sleep needs and sleep expectations; (4) misattributions about causes of insomnia; and (5) misconceptions about sleep-promoting habits. However, several of the subscales have been shown to have poor internal consistency, and factor analytic studies have failed to support the originally proposed DBAS subscale structure.[19,20]

Nonetheless, our recent factor analytic work[20] with a large cohort (N = 586) of DBAS respondents showed that an abbreviated DBAS composed of 10 of the original DBAS items (i.e., items 1, 2, 5, 8, 10, 11, 12, 21, 25, and 28) maintained the thrust of the original instrument, reliably discriminated normal sleepers from insomnia sufferers, and included four factor analytically-supported subscales which appear to measure: (1) concerns about the negative effects of insomnia, (2) misconceptions about sleep needs/requirements; (3) excessive sleep preoccupations; and (4) a proneness toward medication dependence. Using pre-treatment data from the cohort enrolled in the current study, we found that the mean item score on this DBAS short form (DBAS-SF) correlated highly ($r = .81$, $p<.0001$) with the mean item score on the full DBAS score and the 10 items appeared to have a reasonable level of internal consistency (Cronbach's=0.62). In addition, the four DBAS-SF subscales had Cronbach alpha coefficients which varied from a low of 0.48 for the needs subscale to a high of 0.72 for the medication dependence subscale (Mean=0.62). Since this abbreviated form seemingly has better psychometric properties than the original DBAS, we extracted mean item scores for the entire DBAS-SF and its subscales from each administration of the full DBAS to assess changes in our patients' dysfunctional beliefs about sleep through the course of treatment and follow-up.

Primary Measures of Treatment Outcome

Polysomnography

All study participants underwent an initial polysomnographic (PSG) sleep recording for screening purposes and two subsequent recordings to assess pre to post-treatment sleep improvements. The first of the latter two PSG's served as the pre-treatment baseline and was conducted one to two weeks before treatment was initiated, whereas the second was conducted during a two-week post-treatment assessment period. All PSG's were conducted in study patients' homes using eight-channel Oxford Medilog 9000/9200 (Oxford Medical, Inc., Clearwater, Fl.) analogue-cassette recorders. The monitoring montage for the initial, screening PSG consisted of two EEG channels, one chin EMG channel, two channels of EOG, one respiration channel (nasal/oral thermistor) and two channels of anterior tibialis EMG (right and left leg). Tibialis and airflow monitoring were excluded during the subsequent "baseline" and post- treatment PSG's.

All recordings were scored directly on the screen of the Medilog Playback Unit by a sleep technician or polysomnographer using standard scoring criteria[21] and a previously validated screen-scoring methodology.[22] While scoring the PSG's, scorers were kept blind to the dates on which the recordings were conducted and the treatment condition to which participants had been assigned. Selected outcome measures derived from each baseline and post-treatment PSG included total sleep time (TST), wake time after sleep onset (WASO: all time awake between the initial sleep onset and the final AM rising time) and sleep efficiency (SE % = [total sleep time/total time in bed] x 100%). For the purposes of calculating WASO, sleep onset was defined as the time between "lights out" and the first 10 minutes of sleep containing no more than two minutes of wake time, stage 1 sleep, or movement time.

Sleep Logs

Subjective sleep changes were derived from sleep logs which participants completed during a two-week pre-treatment baseline, a two-week post-treatment assessment, and a two week follow-up six months later. Logs were designed so that a week's worth of sleep data could be recorded on each sleep log form. For each 24-hour period, the respondent completed questions about the time of retiring to and arising from bed as well as questions designed to obtain the respondent's estimates of sleep onset latency, number of nocturnal awakenings and wake time after sleep onset for each night of sleep. In addition, the log contained Likert-style items that allowed respondents to rate the perceived quality (1 = "extremely poor," 5 = "excellent") of each night's sleep as well as how well rested (1 = "not at all rested," 5 = "extremely well rested") they felt upon arising each day. Target subjective outcome measures derived from these logs included the estimates of TST, WASO, sleep efficiency, and both the sleep quality and restedness ratings.

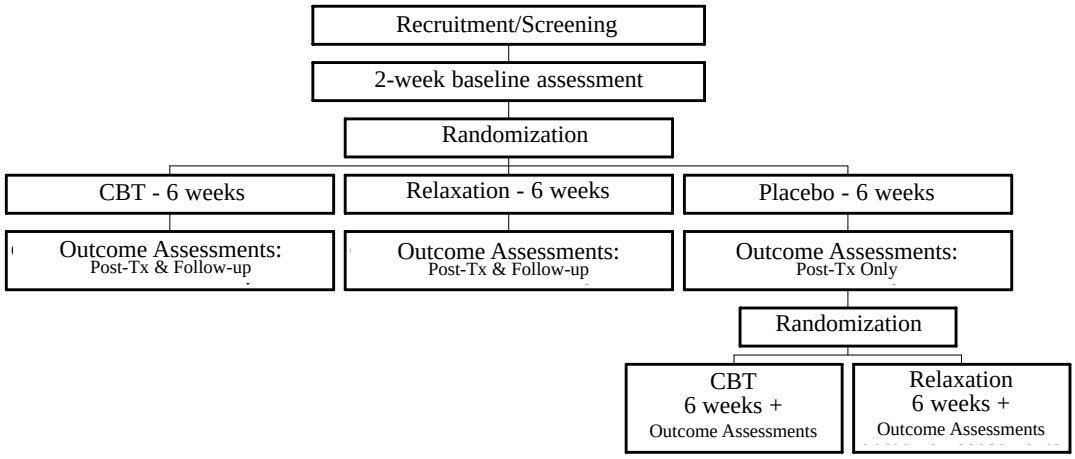

Figure 1—Study flowchart

Insomnia Symptom Questionnaire

An Insomnia Symptom Questionnaire (ISQ), completed by participants at baseline, post-treatment, and the six month follow-up time points, was used to assess treatment-related changes in global subjective insomnia symptoms. This instrument, developed by Spielman et al.,[6] contains 13 Likert-style items designed to assess respondents' perceptions about their daytime functioning and nighttime sleep. However, we altered the format of the ISQ so that each item was accompanied by a 100 mm visual analog scale (i.e. horizontal line) which was labeled "not at all" at its left extreme and "frequently" at its right extreme. In responding to this instrument, respondents were instructed to draw a vertical line through the point on each item's analog scale (i.e., 100 mm line) to indicate their responses. The distance from the left end of the line to the response line served as an analog measure of the degree to which the respondent had the symptom noted by the item. The average score across the 13 items represented the respondent's overall ISQ score for each administration. Using baseline ISQ responses of the 75 participants enrolled in this study, we found Cronbach's coefficient alpha to be 0.73 for the total ISQ score, thus attesting to the internal consistency of this measure. In support of this instrument's construct validity, Spielman et al.[6] have shown that scores on the ISQ improve as a function of behavioral sleep intervention.

Procedure

Prospective study patients first completed various screening procedures including structured interviews (i.e., SIS-D and SCID-P), one week of sleep log monitoring, a medical examination, a thyroid function test, and a screening/diagnostic PSG (conducted following two weeks of abstinence from sleep medication use). Consenting individuals who met study criteria then completed the DBAS, ISQ, the "baseline" PSG , and two weeks of sleep log monitoring before beginning treatment. Subsequently, a stratified randomization procedure was used to assign equal proportions of men and women in each of two age groups (i.e., those <55 years old and those >55 years of age or older) to each treatment/therapist combination.

Treatments were provided during six weekly, 30- 60-minute individual sessions by one of the two participating therapists, and all treatment sessions were conducted within our sleep disorders center. One male (age 30) and one female (age 29) doctoral level clinical psychologists, who were well trained in the administration of various types of behavioral therapies but who had no prior experience in behavioral insomnia treatment, served as the therapists in this trial. These therapists were provided manualized versions of the treatment protocols as well as training and ongoing supervision from the primary author to assure their competence in delivering these interventions.

All participants maintained nightly sleep logs throughout treatment and during the two weeks after the sixth treatment session. They were also administered the DBAS and ISQ measures and asked to undergo a single night of PSG monitoring sometime during the two-week post-treatment assessment period. Those initially assigned to the CBT or RT conditions additionally were asked to complete the DBAS, ISQ and two weeks of sleep log monitoring during a follow-up assessment scheduled six months after their post-treatment assessment. In contrast, individuals initially assigned to the PC condition were offered active treatment once they completed the initial post-treatment assessment. Those who accepted were then randomly assigned to either CBT or RT provided during six weekly, individual sessions by the same therapist who treated them initially. Subsequently these individuals were asked to complete the DBAS, ISQ, and 14 days of sleep log monitoring both immediately after the active treatment phase and again six months later. The overall sequence of screening, randomization, assessment, and treatment procedures is shown by the flowchart in Figure 1.

Treatments

Study patients assigned to CBT received a treatment designed to correct both dysfunctional sleep-related cognitions and sleep-disruptive habits. The "cognitive therapy" component of this treatment consisted of a sleep education module, prepared in lay terms, which was delivered via an audio tape recording during

the first treatment session and reinforced by each participant's assigned therapist over the course of treatment. The taped education module was designed to correct generic dysfunctional beliefs about sleep commonly reported by PPI patients. Specifically, this tape included information about the marked inter-individual variation in nightly sleep requirements, the effects of normal aging on sleep, the effects of mild sleep deprivation on subsequent sleep and daytime functioning, the influence of endogenous circadian rhythms, and the benefits of maintaining a regular sleep-wake cycle. During the remainder of the initial session as well as during subsequent sessions, the therapists referred to information presented on this tape to address relevant dysfunctional sleep-related cognitions subsequently presented by their assigned study patients during treatment. In addition to this cognitive intervention, participants assigned to CBT also received instruction in standard stimulus control and sleep-restriction instructions beginning with the first treatment session. Since our methods for implementing these latter interventions across sessions have been well-described elsewhere,[14,15,23] they will not be reiterated here.

Patients assigned to the other two treatment conditions received no treatments specifically designed to address dysfunctional sleep-related cognitions either via audio recording or from their respective therapists. Instead, those assigned to the RT condition received six weekly sessions of progressive muscle relaxation following the methods of treatment delivery outlined in the Bernstein and Borkovec[24] treatment manual. In contrast, those assigned to the PC were provided a quasi-desensitization treatment like that proposed by Steinmark and Borkovec.[25] In brief, this latter treatment consisted of the therapist assisting the study participant in the development of a chronological hierarchy of 12 activities in which he/she engaged upon awakening in the middle of the night (e.g., opening eyes, looking at the clock, using the restroom, etc). The therapist also helped the study patient develop six imaginal scenes of himself/herself engaged in neutral activities during the daytime (e.g., watching TV, reading the newspaper, preparing a meal, etc.). Subsequently, the therapist instructed the patient to pair images of each neutral scene with each item on the 12-item hierarchy. Two of the items from the 12-item hierarchy were presented during each treatment session so that by the end of the sixth session all items on the 12-item hierarchy had been practiced with therapist assistance. Both RT- and PC- assigned study patients were also provided audio tapes of the exercises conducted in each therapy session and were instructed to use these tapes to assist them in prescribed daily home practice sessions. Finally, those in the RT condition were instructed to use their relaxation skills to assist them in combating nocturnal awakenings whereas those assigned to the PC condition were told not to employ their desensitization technique during the night because this mental activity might "delay the return of sleep."

Treatment Credibility and Treatment Purity

Included in our research protocol were special measures or procedures to assure that participants perceived the treatments as equally credible and the therapists maintained strict adherence to treatment protocol. As these measures have been described at length in another report,[23] they will be described briefly herein. Treatment credibility was assessed at the end of the first treatment session and again after the final treatment session using the Therapy Evaluation Questionnaire (TEQ).[26] The first five questions of this instrument (rated on a seven-point scale) assessed patients' confidence in their assigned treatment, willingness to recommend it to a friend, willingness to repeat the treatment, etc. The final two items of this measure were designed to assess the quality of the therapeutic relationship throughout treatment. Participants completed the first five TEQ items at both time points and the final two items only at the end of treatment.

Treatment purity was assessed by requiring therapists to tape record all therapy sessions and then randomly selecting a small subset (10 CBT, 12 RT, and 7 PC sessions) of these for systematic review. In performing this review, a blinded rater (clinical psychology post-doctoral student), uninvolved with this study, reviewed these tapes and completed a specially designed checklist to indicate which of 10 listed components for each treatment were present during the taped session. A therapy "purity" index for a particular taped treatment session was then computed by dividing the number of items on the checklist for the treatment by the total number of items checked.

RESULTS

Treatment Credibility and Purity

One-way ANOVA's used to compare the treatment groups in regard to their responses to the initial administration of the TEQ items were all nonsignificant (p's>.05) suggesting that the three interventions were perceived as equally credible at the outset of treatment. However, at the conclusion of treatment, analyses of two TEQ items showed that those assigned to the PC reported significantly less willingness to recommend their treatment to a friend and significantly less confidence that the treatment would be effective for others in general than did those assigned to the other two treatments. Despite these latter findings, ANOVA comparisons suggested TEQ ratings of therapist warmth and competence obtained at the end of treatment did not differ across the three treatment groups (p's>.05). Hence, study participants seemingly found the three treatments equally credible, particularly at the beginning of treatment, and the therapists delivered each treatment with steady levels of warmth and competence.

In performing the ratings of therapy tapes, our blinded rater identified an average of 3.5 (SD=2.3) of the 10 therapy specific elements on the taped CBT sessions, 3.8 (SD=1.2) of the 10 therapy specific elements on the RT tapes, and 2.7 (SD=2.1) of the 10 treatment-specific elements during the taped PC sessions. Moreover, all of these sessions were rated as 100% "pure"; no "contaminating" elements of an unintended treatment were noted for any of these randomly selected sessions. Thus, to the extent that these tapes are representative, it appears that our therapists adhered to treatment protocols over the course of treatment.

Treatment Comparisons

Of the 75 participants enrolled, 70 (23 CBT, 23 RT and 24 PC) completed treatment and post-treatment administration of the DBAS. The remaining five participants failed to complete treatment and were excluded from tests of hypotheses since they failed both to receive the full "doses" of their respective treatments and to complete the post-treatment DBAS. To test our first

Table 2—Pre-and post-treatment DBAS-SF means/standard deviations and ANCOVA results

DBAS-SF Scale	CBT Pre-Tx	CBT Post-Tx	RT Pre-Tx	RT Post-Tx	PC Pre-Tx	PC Post-Tx	F Values for ANCOVA Terms Time	F Values for ANCOVA Terms Tx x Time
Total DBAS-SF	46.6 (14.1)	35.0 (12.7)	48.1 (12.8)	43.9 (17.8)	45.6 (12.5)	43.0 (12.6)	0.72	5.03**
Effects	56.1 (19.3)	45.0 (17.4)	54.0 (18.1)	47.8 (19.8)	54.6 (14.7)	49.5 (19.1)	3.76	0.84
Needs	48.8 (25.6)	37.2 (20.0)	46.7 (22.4)	47.0 (23.7)	49.5 (26.9)	47.0 (23.8)	10.35**	2.98_
Sleep Preoccupations	47.6 (27.6)	32.0 (21.3)	56.3 (19.9)	50.2 (25.5)	42.3 (22.1)	47.9 (21.1)	10.99**	5.88**
Medication Dependence	24.5 (21.8)	15.9 (15.0)	29.8 (22.6)	26.6 (20.2)	27.2 (26.6)	20.7 (23.2)	6.38*	1.44

Note: Numbers outside of parentheses are means whereas numbers within parentheses are standard deviations. _ connotes that the F value approaches significance (p<.06). * connotes that the F value is significant at the p<.05 level. ** connotes that the F value is significant at the p<.01 level.

hypothesis, we compared the pre to post-treatment changes in DBAS-SF total and subscale scores shown by CBT-treated patients with the DBAS-SF changes shown by patients initially assigned to RT or PC. We conducted these comparisons using analyses of covariance (ANCOVA's) which used initial values of the DBAS-SF total or subscales scores as covariates. Since an initial set of ANCOVA's showed no main or interaction effects attributable to therapist differences we used 3 (CBT vs. RT. vs. PC) x 2 (pre-treatment vs. post-treatment) ANCOVA's to compare the DBAS-SF changes shown by the three treatment groups.

Table 2 shows the results of these ANCOVA's as well as the group means and standard deviations for the full DBAS-SF and each subscale at each of the two time points. Pertinent to our first hypothesis, these data show significant treatment group x time interactions for the total DBAS-SF score and for the subscale presumed to measure excessive sleep preoccupations. In regard to the total DBAS-SF scores, Bonferroni-corrected post hoc comparisons showed that CBT-treated patients had significantly greater pre to post-treatment reductions in their dysfunctional beliefs about sleep overall than did each of the other two treatment groups. Similar comparisons of the sleep preoccupations subscale showed that CBT-treated patients showed significantly greater reductions in their scores on this subscale than did the PC treated patients. Additionally, the ANCOVA's showed the three treatment groups collectively displayed significant pre to post-treatment reductions in their scores on the DBAS-SF sleep needs and medication dependence subscales although, for the former of these subscales, there was a trend (p<.06) toward a significant group x time interaction with CBT patients showing greater changes than the other groups.

During the course of this trial, a total of 65 study patients received CBT or RT either as an initial treatment assignment (CBT = 25; RT = 25) or subsequent to completing the PC condition (CBT = 9; RT = 6). However, only data from the 46 (24 CBT and 22 RT) of these who completed treatment and returned for the six-month follow-up were used to test our second hypothesis. To determine if, as predicted, CBT recipients showed greater long-term reductions in their DBAS-SF scores than did RT recipients, we conducted a series of 2 (CBT vs RT) x 2 (pre-treatment vs. follow-up) ANCOVA's using the DBAS-SF total and subscale scores obtained at pre-treatment and follow-up time points as dependent measures. Each of these ANCOVA's statistically adjusted both for the pre-treatment levels of the DBAS-SF measures and for potential differences between those receiving CBT or RT treatment initially or subsequent to the PC.

Consistent with our prediction, results of these ANCOVA's showed CBT produced larger long-term reductions in DBAS-SF scores than did RT. Specifically, these analyses showed significant treatment x time interactions for both the DBAS-SF total score ($F [1, 43]=4.40$, $p<.05$) and for the sleep preoccupations subscale ($F [1, 43]=5.51$, $p<.05$). As suggested by Figure 2, the CBT group showed larger declines/improvements in these scores by follow-up than did the RT group. In addition, a significant main effect for time ($F [1, 43]=8.10$ $p<.01$) suggested that the two treatment groups collectively showed a significant reduction in their scores on the sleep needs DBAS-SF subscale from baseline to follow-up. All of the remaining main and interaction effects in this set of ANCOVA's were nonsignificant.

Correlations Other Outcome Measures

We also conducted two sets of correlational analyses to test our prediction (i.e., Hypothesis 3) that treatment-related reductions in dysfunctional beliefs about sleep would be correlated with sleep improvements reflected by other treatment outcome measures.

In the first set of these analyses we correlated pre to post-treatment changes shown in the total DBAS-SF score with pre-to-post treatment changes shown on the ISQ, PSG parameters, and sleep log measures. These correlations were computed for all three treatment groups combined and then for each group separately. In the second set of analyses, we correlated the changes from baseline to the follow-up time point noted in the DBAS-SF total score with changes noted on the ISQ and log measures over the same time period for all CBT- and RT-treated participants who actually completed the follow-up assessment. Again, we computed these correlations for the CBT and RT groups combined and separately.

Table 3 shows results of Pearson Product correlational analyses conducted to determine the relationship between DBAS-SF

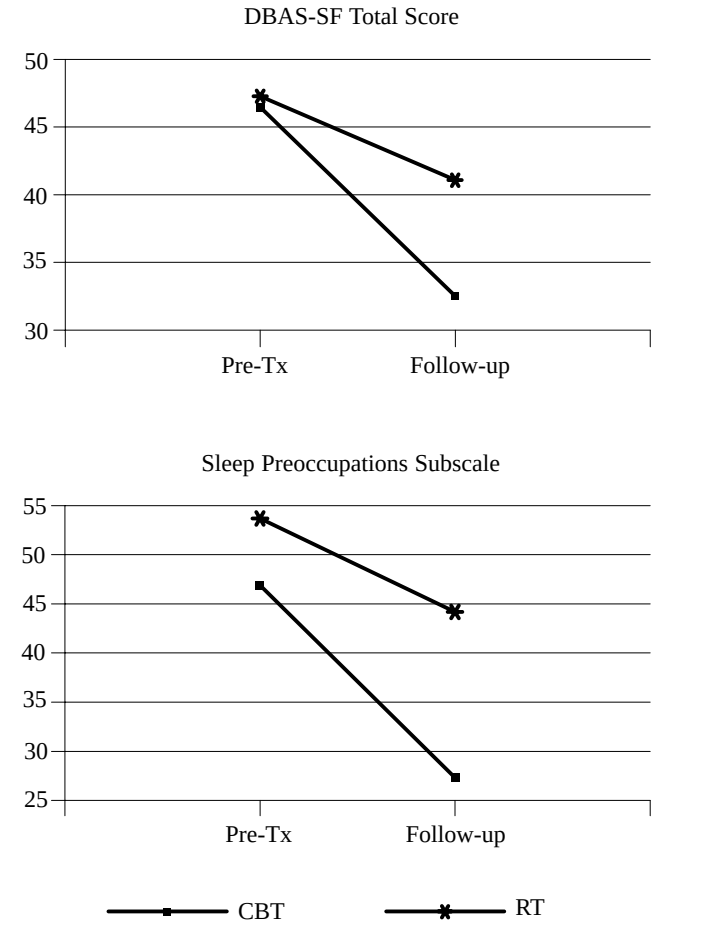

Figure 2—Mean changes in DBAS-SF scores for the CBT and RT groups from pre-treatment assessment to six-month follow-up

changes and changes in the other treatment outcome measures. Within the entire study sample, pre-to-post-treatment reductions in the DBAS-SF total score were significantly correlated with pre-to-post-treatment reductions in PSG values of wake time after sleep and global insomnia symptoms as measured by the ISQ and increases in PSG values of sleep efficiency. However, when the correlational analyses were conducted with each treatment group separately, only within the CBT group were all three of these correlations found to be significant. Within the PC group improvements in the DBAS-SF score were correlated with improvements in PSG values of WASO and sleep efficiency but were unrelated to changes in the ISQ scores. Conversely, within the RT group improvements in DBAS-SF scores were correlated with improvements in ISQ scores but they were unrelated to any of the PSG measures. Furthermore, only within the RT group were pre-to-post-treatment reductions/improvements in DBAS-SF correlated with worsening trends in sleep log measures reflective of sleep consolidation/ fragmentation (i.e., WASO, efficiency). Hence, short-term reductions in dysfunctional beliefs about sleep were most consistently related to short-term improvements in other outcome measures among those who received CBT treatment.

Correlations using follow-up data showed that pre-treatment to follow-up reductions/improvements in the DBAS-SF total score were correlated with reductions/improvements in ISQ scores both within the entire sample who completed follow-up and within the CBT and RT groups considered separately. However, only within the CBT group were improvements in DBAS-SF scores also related to improvements (i.e., increases) in patients' subjective ratings of restedness and sleep quality derived from sleep logs. Thus, as was noted for short-term changes, reductions in dysfunctional beliefs about sleep noted at the six-month follow-up were most consistently related to positive changes in other outcome measures within the CBT group.

DISCUSSION

This study was conducted to test the specific effects of cognitive-behavioral insomnia therapy (CBT) on dysfunctional beliefs and attitudes about sleep. Specifically, we conducted this study to determine if CBT is more effective in reducing dysfunctional beliefs about sleep than is either standard progressive muscle relaxation therapy (RT) or a sham placebo control (PC). In addition, we examined the relationship between reductions in dysfunctional beliefs/attitudes about sleep and improvements in various measures of insomnia symptoms. Consistent with predictions, our results showed CBT produced significantly larger pre-to-post-treatment reductions in dysfunctional sleep-related cognitions than did either RT or the PC. Furthermore, comparisons between our two "active" treatments showed that CBT produced larger reductions in dysfunctional sleep-related cognitions through the six-month follow-up than did RT. Moreover, such cognitive changes were significantly correlated with improvements in both subjective and objective outcome measures, particularly among those who received CBT. Considered collectively, these findings suggest CBT is particularly effective for reducing dysfunctional beliefs about sleep, and reductions in such beliefs have an important association with the overall insomnia treatment outcome.

Cognitive-behavioral insomnia therapies are presumed to have advantages over other behavioral strategies largely due to their specific corrective effects on dysfunctional sleep-related cognitions. Despite its intuitive appeal, this contention had previously received no empirical confirmation. The current study's results, however, provide initial support for the notion that dysfunctional sleep-related cognitions are specifically and favorably altered by cognitive-behavioral insomnia treatment, and such changes apparently remain robust over time. Hence, the degree to which CBT has been shown to out-perform other behavioral insomnia treatments, may, at least in part, be attributable to its specific and enduring effects on those underlying cognitive distortions which presumably perpetuate sleep difficulties.

The potential importance of such changes is highlighted by the results of our correlational analyses. As show by Table 3, reductions in dysfunctional beliefs about sleep were significantly related to improvements noted by primary subjective and objective treatment outcome measures particularly among CBT-treated individuals. This finding suggests that such cognitive changes are an important part of the overall change process that occurs in insomnia treatment in general and in CBT specifically. Of course, our findings fall short of demonstrating a cause-effect relationship. In fact, several interpretations of our findings seem tenable. Consistent with the speculation of Morin et al.,[9] it is possible that dysfunctional beliefs about sleep support and sustain sleep-disruptive practices which, in turn, perpetuate insom-

Table 3—Correlations between changes in the DBAS-SF total score and changes in other outcome measures

Measure	CBT		RT		Placebo		Total	
	Post-Tx	F/U	Post-Tx	F/U	Post-Tx	F/U	Post-Tx	F/U
ISQ	.60**	.63**	.47*	.68**	.03	-	.46**	.62***
PSG								
TST	-.16	-	.20	-	-.41	-	-.06	-
WASO	.45*	-	-.03	-	.55**	-	.39**	-
Efficiency	-.45*	-	.21	-	-.55**	-	-.31*	-
Sleep Logs								
TST	.07	-.41	.37	.22	.00	-	.21	.11
WASO	.18	.19	-.46*	-.06	.02	-	-.01	.07
Efficiency	-.25	-.20	.48*	.11	-.08	-	.04	-.02
Restedness	-.28	-.45*	.03	-.42	.14	-	-.05	-.30
Quality	-.10	-.46*	-.02	-.18	.08	-	-.08	-.25

Note:Post-Tx = Post-Treatment; F/U = Follow-up. Post-treatment correlations with ISQ based on 23 CBT, 23 RT, & 24 PC participants. Post-treatment correlations with PSG based on 23 CBT, 23 RT & 21 PC participants. Post-treatment correlations with Logs based on 23 CBT, 23 RT & 24 PC participants.The follow-up data are based on those who received CBT or RT either as their initial treatment or subsequent to the PC. Follow-up correlations with ISQ based on 24 CBT and 22 RT participants. Follow-up correlations with Logs based on 21 CBT and 20 RT participants. * = p<.05; ** = p<.01 ***=p<.001.

nia symptoms. Reductions in such beliefs may give way to elimination of sleep-disruptive habits and subsequent sleep improvements. Alternately, it is possible that beliefs about sleep change in response to objective and/or subjective sleep improvements. Indeed, this possibility may explain why our other treatment groups, which received no structured cognitive interventions, each showed a relationship between cognitive changes and improvements on one or two of the primary treatment outcome measures. However, it is possible that both of these speculations are accurate. That is to say, cognitive changes may contribute to sleep improvements and sleep improvements may help alter individuals' dysfunctional beliefs about sleep. Finally, inasmuch as some previous research[10,11] suggests that beliefs/attitudes about sleep may influence subjective sleep appraisals, it is possible that reductions in dysfunctional sleep-related cognitions may contribute to improvements on a subjective measure like the ISQ mainly by giving way to more favorable subjective sleep assessments. Whereas each of these speculations seems reasonable, further research is needed to determine their relative merits.

In addition to these implications, our results also suggest that reductions in dysfunctional beliefs about sleep may not always be associated with improvements implied by other symptomatic measures. Indeed, within the RT group, pre-to-post-treatment increases in sleep fragmentation (i.e., increases in WASO, decreases in sleep efficiency) reflected by sleep logs was correlated with reductions/improvements in patients' DBAS-SF scores over the same time period. This finding is perplexing and difficult to explain. However, it should be noted that these patients were instructed to use their newly learned relaxation techniques as a means of returning to sleep if they awakened during the night. Given this instruction, it is possible that study patients who followed this recommendation became less concerned about their nocturnal awakenings through their repeated pairing with relaxation. For such patients, this decreased concern may have led to reductions in scores on the DBAS-SF and ISQ while simultaneously allowing them to tolerate more wakefulness in bed. If this speculation is correct, it may explain why those RT patients who showed increased nocturnal wake time through treatment were able to still manifest reductions in their DBAS-SF and ISQ scores. However, additional research is required to determine if these speculations are correct.

Admittedly, this study had several limitations. Although, to date, this has been one of the largest placebo-controlled and well-blinded investigations involving CBT, our study sample was only moderate in size. This limitation may have reduced power to detect some potential differences among conditions. Also, our assessment of cognitive changes relied on a relatively brief measure which assesses only a few cognitive dimensions. In addition, this trial included well-screened and more highly selected patients than typically found in primary care settings. Hence, CBT's effectiveness for altering dysfunctional cognitions among more "real-world" patients is yet to be determined. Finally, follow-up data should be interpreted cautiously since a notable number of study patients did not complete the follow-up assessment. Nonetheless, since the investigation provides evidence that CBT has relative efficacy for altering dysfunctional sleep-related cognitions and such cognitive changes correlate with sleep improvements, the study's findings merit consideration.

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