ERRATUM — In the March issue, two tables were omitted from this paper. This paper has been republished to include the two tables.

Identifying Critical Beliefs About Sleep in Primary Insomnia

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Subject Objective: Maladaptive beliefs about sleep are associated with insomnia and are assessed with the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS). Three studies explored which DBAS items (1) maximally differentiated people with insomnia from good sleepers, (2) declined with cognitive behavior therapy (CBT), and (3) were related to other clinical improvement indexes.

Design: Data from previous studies were analyzed to evaluate the above 3 hypotheses.

Participants: The total sample (N = 332) was comprised of experimental and treatment-seeking people with insomnia and good sleepers ranging from 20 to 79 years of age (mean ± SD 51.3 ± 14.7).

Results: The analyses of variance of the 30 items of the DBAS in Study 1 suggested that 16 items differentiated insomnia sufferers from good sleepers. In Study 2, 8 items showed significantly greater changes in response to CBT than alternate therapies. However, only 2 of these items were among the 16 items that discriminated insomnia sufferers from good sleepers in Study 1. In Study 3, declining scores on 15 of 30 DBAS items in response to CBT were related to 1 or more indexes of clinical improvement.

Conclusion: The 16 beliefs of the DBAS-30 that best discriminated insomnia sufferers from good sleepers related to helplessness and hopelessness in the insomnia group. CBT addressed some of these beliefs, although some beliefs relating to helplessness remained relatively elevated. These residual beliefs should be investigated further, as they may confer cognitive risk for future insomnia and imply ways to improve current CBT strategies.

Keywords: Beliefs about sleep, insomnia, cognitive behavior therapy

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INTRODUCTION

PRIMARY INSOMNIA IS A PREVALENT AND OFTEN DEBILITATING FORM OF SLEEP DIFFICULTY, TRADITIONALLY ATTRIBUTED TO SUCH SUSTAINING FACTORS as conditioned arousal at bedtime and sleep-disruptive habits.[1,2,3,4] Current cognitive-behavioral conceptualizations[5-7] of primary insomnia posit that rigidly held or self-defeating beliefs and attitudes about sleep also play important roles in sustaining this form of sleep difficulty. For example, unrealistic sleep expectations or beliefs that there is little one can do about poor sleep may heighten sleep-related "performance anxiety" and make sleep more difficult to achieve. Likewise, the belief that one should try to "catch up" for lost sleep may lead to sleep-disruptive compensatory practices such as subsequent daytime napping or remaining in bed well beyond the usual rising time. Because of their putative roles in spawning sleep-related distress or arousal and practices that perpetuate primary insomnia, maladaptive beliefs and attitudes about sleep arguably can be viewed as centrally and mechanistically important to this type of insomnia. Research designed to identify the specific beliefs most critical to sustaining primary insomnia, and their response to belief-targeted treatment, is of utmost importance to our understanding and management of this condition.

Recognizing the importance of such research, Morin and colleagues[6,7] developed the Dysfunctional Beliefs and Attitudes about Sleep questionnaire (DBAS) to provide a systematic method for assessing sleep-disruptive cognitions. The DBAS consists of 30 rationally derived items presumed to assess the range of maladaptive or self-defeating beliefs most integral to chronic insomnia. Specifically, the DBAS includes item subsets or subscales designed to measures 5 discrete cognitive themes, including (1) maladaptive beliefs about the effects of insomnia, (2) beliefs that sleep is unpredictable and uncontrollable, (3) unrealistic expectations about sleep needs, (4) misconceptions about the causes of insomnia, and (5) erroneous beliefs about sleep-promoting practices. Although the name of the scale implies that the presence of such beliefs is inherently "dysfunctional," it is probably more accurate to say that strong or rigid endorsement of these beliefs can be maladaptive. As such, good sleepers would not be expected to completely disagree with these beliefs; instead, their degree of agreement would be moderately low and reflect some flexibility in the beliefs. In contrast, strong endorsement of these beliefs may connote less flexibility and, consequently, more distress when faced with situations that appear to confirm such beliefs.

Since its advent, the DBAS has been employed in several types of studies designed to document the role of maladaptive beliefs in the protraction of primary insomnia. Given the presumed mechanistic importance of such beliefs to primary insomnia, those with this condition should show more rigidly held or self-defeating sleep-related beliefs than should those without sleep complaints. Studies[7,8] testing this assumption have employed the DBAS to compare the sleep-related beliefs of insomnia sufferers and noncomplaining normal sleepers. However, it is difficult to discern the specific beliefs that are important in primary insomnia from these studies because the former study[7] compared a mixed group of primary and secondary insomnia sufferers with normal sleepers, whereas the latter study[8] subdivided groups of

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primary insomnia sufferers and normal sleepers into subgroups of polysomnographically (PSG) defined good and poor sleepers prior to conducting DBAS comparisons. Thus, additional studies comparing pure primary insomnia groups with good sleepers groups would be helpful.

An alternative line of research has examined DBAS changes in response to cognitive behavioral therapy (CBT), a treatment that attempts to replace rigid and self-defeating sleep-related beliefs with more flexible or sleep-favorable cognitions (e.g., belief-targeted treatment). Although limited in number, studies of this nature have provided some support for the notion that sleep-related beliefs of primary insomnia sufferers do, indeed, change over the course of treatment with well-proven CBT. At least 2 investigations[9,10] have shown that DBAS total scores change from the beginning to the end of CBT treatment. However, since these studies did not conduct item-by-item analyses, they did not identify the specific beliefs that change with treatment. In contrast, Espie and colleagues[11] showed that 10 DBAS items measuring worries about acute and long-term insomnia effects and fears about loss of control over sleep changed significantly over the course of CBT. These findings provide some initial insight into potentially important and malleable cognitive targets in primary insomnia, but additional studies of this nature that control for natural regression to the mean and nonspecific effects of treatment would be useful. To the extent that rigid and self-defeating sleep-related beliefs sustain primary insomnia, it is perhaps most important to determine how changes in such beliefs relate to other indexes of clinical improvement. Given this consideration, some research has been conducted to relate the changes primary insomnia sufferers show in their sleep-disruptive beliefs to the changes they show on other important disease-specific outcome measures over the course of therapy. Studies[9,10] have suggested that reductions in selected subscale scores on the full DBAS or the total score on a short form of the DBAS are associated with both objective and subjective improvements on other sleep-related outcome measures. Because these studies analyzed changes in subscale or total DBAS score, little is known about which specific items are most germane to improvements on other indexes of clinical improvement. Thus, it is unknown as to which specific dysfunctional beliefs are most relevant to the overall treatment responsiveness of primary insomnia patients.

As suggested by this discussion, a number of potentially useful methodologic probes have been utilized to assess the role of maladaptive sleep-related beliefs in both the maintenance and remission of primary insomnia. Each of these probes may be useful for elucidating the roles of dysfunctional beliefs in the primary-insomnia process, yet each has its limitations. Moreover, previous studies using these strategies are somewhat limited in the specificity and comprehensiveness of their findings. As a result, investigations that address some of the limitations of previous studies, and simultaneously employ the 3 probes described to identify the specific beliefs instrumental in primary insomnia, could be useful.

The current report describes an investigation designed to address these objectives. Specifically, this investigation included 3 studies designed to identify the specific maladaptive beliefs most germane to the perpetuation and treatment of primary insomnia. In the initial study, DBAS item responses of age-matched groups of primary insomnia sufferers and normal sleepers were compared to identify the items that statistically discriminated these 2 groups. In Study 2, pretreatment-to-posttreatment DBAS item change scores of primary insomnia sufferers receiving either a belief-targeted therapy (e.g., CBT) or alternate treatment were compared to identify items most relevant to the insomnia-treatment process. In contrast with previous research,[11] use of a comparison group in study 2 controlled for regression to the mean and allowed for elucidation of those maladaptive beliefs that "normalize" specifically with CBT treatment. Finally, in Study 3, we explored the assumption that treatment-related reductions in beliefs should contribute to improvements in other indexes of sleep. To do so, we examined the association of several indexes of clinical improvement with item-by-item changes on the DBAS from before to after treatment. In conducting this series of investigations we hypothesized that a subset of items would be found that would (1) discriminate primary insomnia sufferers from normal sleepers, (2) change significantly through belief-targeted therapy, and (3) show pretreatment-to-posttreatment changes (improvements) that correspond to improvements on other clinically relevant measures. Results of this series of studies were, thus, used to test this global hypothesis.

Study 1

As noted above, previous studies[7-11] have shown that insomnia sufferers produce higher and presumably more pathologic scores on the DBAS overall and on various of the rationally derived DBAS subscales originally proposed by Morin.[6,7] Such findings are consistent with the notion that the global cognitive themes measured by the DBAS may play an important sustaining role in the overall insomnia process. However, it remains unclear how well each of the individual DBAS items discriminate primary insomnia sufferers from those without sleep complaints. In this initial study, we compared large samples of insomnia sufferers and noncomplaining normal sleepers on each item of the DBAS-30 to identify the specific dysfunctional beliefs that most strongly relate to insomnia complaints.

METHOD

Design

This study used a between-groups cross-sectional research design. Independent groups of age- and sex-matched primary insomnia sufferers and noncomplaining normal sleepers comprised the study sample. The participants for the current study were drawn from a series of studies[12-14] conducted to compare the home and laboratory sleep patterns of young, middle-aged, and older adult insomnia sufferers and normal sleepers. All study procedures were reviewed and approved by the institutional review boards of the VA Medical Center and Duke University Medical Center in Durham, NC. All participants were required to provide written informed consent prior to enrolling the research and undergoing study-related procedures.

Participants

All participants were recruited via (1) posted advertisements at the Durham (NC) VA Medical Center, (2) letters mailed to persons in the Duke University Center for the Study of Aging and Human Development Subject Pool, or (3) face-to-face solicitations of patients presenting to the Duke University Sleep Disorders Center. Prior to their acceptance into the research program, all partici-

pants underwent a thorough screening process that included structured psychiatric[15] and sleep interviews,[16] a medical exam, thyroid (thyroid-stimulating hormone, level) screening, and 1 to 2 nights of screening PSG to rule-out occult primary sleep disorders. The insomnia sufferers recruited were adults between the ages of 20 and 79 who reported sleep complaints consistent with Diagnostic and Statistical Manual for Mental Disorders (DSM) criteria for primary insomnia (e.g., 6 months or more of difficulty initiating or maintaining sleep or nonrestorative sleep at least 3 times per week, with accompanying daytime deficits).[17,18] The normal sleepers enrolled were an age-matched sample of adults who (1) reported no sleep complaints and (2) evidenced no major medical or psychiatric condition that might have contributed to an unreported, occult sleep disorder.

Excluded from the final sample were study participants who (1) had a medical condition (e.g., rheumatoid arthritis, thyroid disease) that compromises sleep; (b) had a current major psychiatric (Axis I) condition on the basis of a Structured Clinical Interview for Psychiatric Disorders (SCID)[19]; (c) showed sedative hypnotic dependence and were unwilling or unable to abstain from these medications while in the study; (d) were taking anxiolytics, antidepressants, or any other psychotropic medication; or (e) had an apnea-hypopnea index ≥ 15 or a periodic limb movement-related arousal index ≥ 15 during on screening PSG. In addition, we excluded prospective insomnia sufferers if they met structured interview criteria[16] for another sleep disorder in addition to primary insomnia, whereas we excluded normal sleepers who met criteria for any sleep disorder.

Through use of these selection criteria, a sample of 208 study participants were enrolled and subsequently completed the DBAS, other questionnaires, and a series of home- and laboratory-based evaluations of sleep and daytime performance. Three study enrollees failed to complete the DBAS properly and were excluded from the sample chosen for the current study. The final study sample, thus, consisted of 205 adults. Of these, 101 (52 women) met selection criteria for primary insomnia, whereas the remaining 104 (52 women) were noncomplaining normal sleepers. The mean age of the primary insomnia sample was 49.2 (SD = 17.1) years, whereas the mean age of the normal group was 47.3 (SD = 16.8) years. The primary insomnia sample was composed of 76 Caucasians, 17 African Americans, and 5 Asian Americans, and 1 Native American; the normal sleepers included 85 Caucasians, 14 African Americans, 4 Asian Americans, and 1 Native American.

PSG Screening

All participants underwent a total of 3 in-home and 3 in-lab nights of PSG monitoring with one half of the men and women in each group undergoing lab recording first, and the other half completing home monitoring first. Per preplanned study protocols, the first night (older cohort aged 60+ years) or initial 2 (remainder of the sample) PSG nights (home or lab) were used to screen out those exceeding the above-mentioned apnea-hypopnea index or periodic limb movement arousal index cutoffs for study inclusion. PSG monitoring included 2 electroencephalogram channels (C_3-A_2, O_z-C_z), bilateral electrooculogram, submental electromyogram, 2 channels of anterior tibialis electromyogram (right and left leg), and a nasal-oral respiration thermistor. All PSGs were scored using standard scoring criteria for assignment of sleep stages, identification of respiratory events (e.g., apneas, hypopneas) and identification of periodic limb movements and periodic limb movement-related arousals. Although PSG typically includes additional respiratory measures (respiratory effort, oximetry) to detect breathing abnormalities, it was thought that monitoring of nasal/oral airflow along with our thorough interview screening for apnea would be sufficient to identify most cases with an apnea-hypopnea index above the study's exclusionary cutoff.

Measure

The DBAS[6,7] scale was used to identify specific sleep-related beliefs that discriminated the insomnia sufferers and normal sleepers comprising this study's sample. Each of the 30 items consists of a statement that poses a sleep-related belief or attitude pertinent to 1 of these 5 cognitive themes. A 100-mm analog scale (i.e., horizontal line) labeled "strongly disagree" at its far left extreme and "strongly agree" at its far right extreme accompanies each item and is used by respondents to indicate their degree of endorsement. When completing the DBAS, respondents are required to draw a vertical line through the point on the 100-mm scale to indicate their degree of agreement or disagreement with each item. The distance in millimeters between the far-left extreme of the analog scale and the response line then is used at the item's "score." With 1 exception, all items are structured so that higher scores (i.e., stronger item agreement) connote more dysfunctional beliefs.

Procedure

The participants included in the current investigation completed the DBAS on 1 occasion while enrolled in the larger study from which they were obtained. Participants' scores for each of the 30 DBAS item were extracted and placed in an electronic file (spreadsheet database) for subsequent analyses. To control for type-I error, a 2 (insomnia vs normal sleeper) x 30 (DBAS items) multivariate analyses of variance (MANOVA) was first conducted. Group comparisons were then conducted via analyses of variance (ANOVAs) for each of the 30 DBAS items to follow-up significant MANOVA effects.

RESULTS

There was a statistically significant group effect on the MANOVA, $F_{30,206} = 9.85$, $p < .001$. Follow-up ANOVAs found a statistically significant group effect on 16 of the 30 pretreatment DBAS items; thus, there were 16 items that discriminated good sleepers from those with insomnia. Examination of the means revealed that insomnia sufferers had higher scores (e.g., more-rigid or sleep-disruptive beliefs) than good sleepers on each of the discriminating items. Four of the 5 thematic subscales of the DBAS contained discriminating items. All 9 of the items of the Control/Predictability scale differentiated normal sleepers from insomnia sufferers. Five of the 9 items of the Effects scale demonstrated discriminative value. In contrast, only 1 of the 3 items relating to Sleep Needs scale and 1 of the 7 items of the Sleep-Promoting Practice scale significantly discriminated those with insomnia from those without. Neither of the 2 items of the Causal Attributions scale showed a statistically significant group effect on ANOVA. Table 1 contains group means and SEM and the resultant F statistics and p values from the ANOVAs.

Table 1—Comparisons of Insomnia Sufferers' and Normal Sleepers' DBAS Item Responses

Theme/items	Insomnia Sufferers		Normal Sleepers		Group Comparisons	
	Mean	SEM	Mean	SEM	F	p value
Effects of Insomnia						
5. Insomnia seriously affects health	50.2	3.0	32.0	3.5	15.6	.0001
12. Poor sleep disturbs daytime mood.	50.0	3.0	34.5	2.6	15.1	.0001
15. Afraid of dying in sleep	9.9	2.1	5.1	1.0	4.5	.04
21. Lack of energy due to poor sleep	62.2	2.5	46.1	2.7	19.6	.0001
30. Cancel obligations after poor night's sleep	20.3	2.6	12.3	1.9	6.2	.01
Control/Predictability						
8. Worried may lose control of sleep	31.3	2.8	9.7	1.5	45.8	.0001
16. Bad night follows good night's sleep	23.3	2.8	5.2	.9	39.1	.0001
17. Poor night's sleep affect whole week	17.8	2.1	9.9	1.7	8.5	.004
19. Can't predict sleep	69.5	2.8	35.4	3.2	64.8	.0001
20. Can't manage negative sleep consequences	43.3	2.6	27.9	2.5	18.5	.0001
22. No control over racing mind	48.0	3.1	19.3	2.2	57.6	.0001
23. Can lead satisfactory life despite insomnia	70.2	2.3	60.0	2.8	8.38	.004
25. Insomnia prevents enjoying life	28.5	2.9	12.2	2.1	20.4	.0001
28. Sleep is worsening and no one can help	14.6	1.7	7.2	1.3	12.56	.0005
Sleep-Promoting Practices						
11. Better off taking sleeping pills	25.7	2.7	16.0	2.1	7.9	.006
Sleep Needs						
13. Should sleep as well as partner	31.5	3.0	19.0	2.3	11.0	.001

DBAS refers to Dysfunctional Beliefs and Attitudes about Sleep Scale

Study 2

Study 2 employed a method similar to that reported by Espie[11] for identifying mechanistically important beliefs and attitudes measured by the DBAS. Specifically, in this study we attempted to identify DBAS items that changed significantly over the course of a CBT designed in part to alter insomnia sufferers' rigid or otherwise maladaptive sleep-related thinking. To control for normal regression to the mean, DBAS item score changes shown by a CBT-treated group were compared with the DBAS item change scores of similar patients who received alternate forms of behavioral insomnia therapy that did not specifically target sleep-related cognitions.

METHOD

Design

This study used a randomized parallel-group design. The participants were selected from 3 different randomized clinical trials[9,20,21] designed to evaluate the efficacy or effectiveness of CBT for insomnia. These clinical trials were approved by either the Duke University or VA Medical Center Institutional Review Board, and all participants provided written informed consent to participate at the time of their study enrollments. All participants received study-related screening and therapy services free of charge and were either provided parking free of charge or were compensated for parking expenses while enrolled in the study.

Participants

Participants included in this study were selected from those who underwent CBT or an alternate active insomnia therapy (relaxation training, sleep hygiene therapy) as part of their research participation. Participants drawn from 2 of the clinical trials[9,21] were obtained largely through advertisements in local newspapers, whereas those drawn from the third trial[20] were predominantly physician referred. Standard study screening procedures were used for selection of all participants and included structured sleep[16] and psychiatric interviews[15,19] and 1 week of sleep-log monitoring. In addition, participants drawn from the 2 larger trials underwent 1 night of PSG monitoring in their homes to rule out sleep apnea and periodic limb movements. The PSG equipment, monitoring montage, and scoring procedures for these studies were similar to those described in Study 1 above.

The inclusion criteria were similar to those used for the primary insomnia group from study 1 (e.g., 6 months or more of difficulty initiating or maintaining sleep or nonrestorative sleep at least 3 times per week, with accompanying daytime deficits). Those enrolled in the larger 2 trials[9,21] were additionally required to be 40 years of age or older and have a mean wake time after sleep onset (WASO) ≥ 60 minutes during 1 screening week of sleep-log monitoring. Participants drawn from the third trial[20] were all adults over the age of 20 who had mean total WASO ≥ 60 minutes during a 1-week screening sleep-long monitoring period.

Excluded from these trials were individuals (1) with medical or physical conditions that compromise sleep, (2) who, on the basis on a Structured Clinical Interview,[15] met DSM-IV[18] criteria for a major psychiatric disorder, (3) who obtained a score < 27 on the Folstein Mini-Mental Status Exam[22] conducted during screening, (4) who were unwilling to abstain from sleep medications during the study, and (e) taking anxiolytics or antidepressants. Additional criteria used in the larger 2 trials excluded individuals (1) with periodic limb movements during sleep that were associated with ≥ 15 arousals per hour (from diagnostic PSG), (2) with 15 or more episodes of sleep apnea per hour (from PSG), and (3) who reported chronic histories of little or no sleep or who underestimated total sleep time by 50% or more during an initial diagnostic PSG study.

A total of 128 (54 women) individuals met all selection criteria

Table 2—Comparisons of CBT and Other Treatment on DBAS Item Change Scores Before and After Treatment

Theme/items	CBT		Other Treatment		Group Comparisons	
	Mean	SEM	Mean	SEM	F	p value
Effects of Insomnia						
10. Poor sleep will interfere with daytime activities	-22.11	2.58	-7.25	4.09	7.73	.006
12. Poor sleep disturbs daytime mood.	-13.43	2.33	0.14	4.04	7.69	.006
Control/Predictability						
8. Worried may lose control of sleep	-20.48	2.83	0.75	5.9	11.75	.0008
19. Can't predict sleep	-20.17	3.14	-7.25	4.01	4.2	.04
Sleep-Promoting Practices						
2. Need to catch up on poor sleep	-13.4	3.19	0.96	5.51	4.36	.04
6. Spend more time in bed to get more sleep	-7.23	2.92	7.12	4.45	5.71	.02
7. Should try harder when having sleep problems	-19.98	2.75	1.07	4.48	13.57	.0003
Sleep Needs						
1. Need 8 hours of sleep to function	-20.03	3.19	-1.61	3.94	8.31	.005

CBT refers to cognitive behavior therapy; DBAS, Dysfunctional Beliefs and Attitudes about Sleep Scale.

for enrollment in their original investigation and had sufficient DBAS data to be included in the current study. The mean age of the sample was 54.3 (SD = 10.7) years. One hundred nineteen of the participants were Caucasian, 8 were African American, and 1 was Hispanic. The majority (n = 100) of these participants received CBT as their assigned treatment, whereas the remaining 28 participants received progressive muscle relaxation training (n = 20) or generic sleep-hygiene instructions (n = 8) per random assignment in their respective studies.

Treatment Protocols

Doctoral-level clinical psychologists served as therapists in all 3 clinical trials from which participants were drawn for the current investigation. In delivering the various treatments, therapists strictly followed manualized therapy protocols designed to standardize each treatment's presentation. In all cases, treatment was delivered in individual sessions with the initial sessions lasting 45 to 60 minutes and subsequent sessions (if any) lasting 15 to 30 minutes.

Individuals comprising the CBT group were drawn from 3 different clinical trials. Twenty-three (9 women) were drawn from a study[9] designed to test the general efficacy of CBT. These individuals all were provided 6 CBT sessions scheduled at weekly intervals. A total of 70 (35 women) were drawn from a second study[21] designed to compare various CBT "doses." These individuals received 1 session (n = 16), 2 sessions (n=16), 4 sessions (n= 23) or 8 sessions (n = 15) of CBT scheduled over an 8-week period. The remaining 7 individuals were drawn from a third study[20] designed to tested the clinical effectiveness of an abbreviated CBT intervention. These individuals each received two 25-minute CBT sessions scheduled 2 weeks apart.

Individuals comprising the comparison treatment group were drawn from the first or third studies described above. Twenty (9 women) of these were enrolled in the first study[9] and received progressive muscle relaxation training[21] delivered in 6 weekly sessions. The remaining 8 (1 woman) individuals were enrolled in the latter study and were provided sleep hygiene instructions in two 25-minute sessions schedule 2 weeks apart. Weighted averages showed that the average number of treatment sessions (mean = 4.86 sessions) received by the 28 patients in this comparison group was similar to the average number of sessions (mean = 4.12 sessions) received by the CBT group.

Procedure

The study participants all completed the DBAS prior to treatment and again on 1 or more subsequent occasions as one of several study-related treatment-outcome measures. Those enrolled in the initial treatment-efficacy study[9] and dose-response study[21] were asked to complete the DBAS midway through treatment, immediately following the completion of treatment, and again at a 6-month follow-up assessment. Those drawn from the remaining study were asked to complete the DBAS immediately after completing their 2-session therapy and again at a follow-up scheduled 3 months later. DBAS item change scores (treatment endpoint – baseline value) were then computed for each of the 30 DBAS items. To control for type-I error, a 2 (CBT vs other treatment) x 30 (DBAS items) MANOVA was first conducted using the DBAS change scores as the dependent variables. Treatment-group comparisons (CBT vs comparison treatment) were then conducted via ANOVAs for each of the 30 DBAS item change scores to follow-up significant MANOVA effects.

RESULTS

The MANOVA on DBAS change scores between groups (CBT vs comparison treatment) revealed a statistically significant group effect, $F_{1,126} = 10.72$, $p = .001$. There was a significant group effect on follow-up ANOVAs for 8 of the 30 DBAS item change scores. Of the 8 items with a significant ANOVA group effect, 3 were characterized as discriminating items in Study 1. There were 2 items or beliefs relating to the Effects of Insomnia, 2 relating to Control/Predictability, 3 relating to Sleep-Promoting Practices, and 1 relating to Sleep Needs. Table 2 contains the change scores for the 8 beliefs that showed significant group effects.

Study 3

Implicit to CBT approaches is the assumption that reduction in dysfunctional beliefs and attitudes about sleep should contribute to improvements in insomnia-related symptoms or measures. As noted earlier, there is evidence that global improvements on the DBAS are associated with improvements in other important outcome measures among primary insomnia sufferers undergoing CBT intervention. However, previous studies of this nature did not examine the relationship between changes on individual

Table 3—Summary of Group Comparisons (Improved Versus Unimproved on ISQ, SES, and WASO)

Theme/items	ISQ-defined Improved		ISQ-defined Unimproved		Group Comparisons	
	Mean	SEM	Mean	SEM	F	p value
Effects of Insomnia						
12. Poor sleep disturbs daytime mood	-18.12	3.24	-8.74	3.24	4.18	.04
Control/Predictability						
8. Worried may lose control of sleep	-29.02	3.84	-11.94	3.83	9.91	.002
16. Bad night follows good night's sleep	-7.06	3.37	4.26	3.34	5.65	.02
17. Poor night's sleep affect whole week	-8.96	2.56	0.94	2.56	7.5	.007
20. Can't manage negative sleep effects	-22.08	4.63	-6.3	4.63	5.81	.02
22. No control over racing mind	-22.18	3.69	-4.2	3.69	11.87	.0008
25. Insomnia prevents enjoying life	-43.96	4.24	-24.38	4.43	9.78	.002
28. Sleep is worsening; no one can help	-6.56	2.67	3.92	2.67	7.72	.007
Sleep-Promoting Practices						
7. I should stay in bed and try harder	-26.58	3.79	-13.38	3.79	6.06	.02
27. Medication is the only solution	-8.08	2.48	-1.22	2.48	7.72	.007
Causal Attributions						
14. Insomnia is due to aging	-6.5	3.42	3.9	3.42	4.64	.04
Sleep Needs						
1. I need 8 hours to function	-28.88	4.35	-11.18	4.35	8.25	.005

	SES-defined Improved		SES-defined Unimproved		Group comparisons	
	Mean	SEM	Mean	SEM	F	P value
Effects of Insomnia						
12. Poor sleep disturbs daytime mood.	-19.5	3.19	-7.36	3.19	7.22	.008
Control/Predictability						
8. Worried may lose control of sleep	-26.4	3.93	-14.56	3.93	4.53	.04
17. Poor night's sleep affect whole week	-8.68	2.57	0.66	2.57	6.62	.01
20. Can't manage negative sleep effects	-21.18	4.66	-7.2	4.66	4.5	.04
22. No control over racing mind	-21.72	3.71	-4.66	3.71	10.56	.002
Causal Attributions						
24. Insomnia due to a chemical imbalance	-16.98	3.45	0	3.45	12.13	.001
Sleep-Promoting Practices						
7. I should stay in bed and try harder	-25.86	3.82	-14.1	3.82	4.75	.03
27. Medication is the only solution	-5.3	2.71	2.66	2.71	4.31	.04

	WASO-defined Improved		WASO-defined Unimproved		Group comparisons	
	Mean	SEM	Mean	SEM	F	P value
Effects of Insomnia						
18. Can't function without adequate sleep	-8.93	2.79	-0.76	2.57	4.65	.04
Control/Predictability						
20. Can't manage negative sleep effects	-23.11	4.81	-6.59	4.44	6.36	.01
28. Sleep is worsening; no one can help	-12.17	3.44	-0.76	3.17	5.95	.02

ISQ refers to Insomnia Symptom Questionnaire; SES, Sleep Self-Efficacy Scale; WASO, wake after sleep onset.

DBAS items and changes in other clinically important treatment-outcome measures. As a result, the specific sleep-related beliefs that are most germane to positive clinical outcomes remain relatively unexplored. In this study, we related pretreatment-to-posttreatment change scores on each of the 30 DBAS items to several indexes of clinical improvement among a sample of CBT-treated primary insomnia sufferers.

METHODS

Design and Participants

This study used a between-group cross-sectional research design. The 100 CBT-treated participants employed in study 2 were retained for this study.

Measures

Sleep Log

Participants completed paper-and-pencil sleep logs each morning during a 2-week pretreatment baseline, the treatment phase itself, a 2-week posttreatment assessment, and a 2-week follow-up scheduled either 3 or 6 months later. Sleep-log items included questions about the previous night's bedtime, rising time, sleep-onset latency, and WASO (middle and end of the night).

Outcome Questionnaires

All participants completed the Insomnia Symptom Questionnaire (ISQ)[2] and Sleep Self-Efficacy Scale (SES)[23] prior to treat-

Table 4—Summary of Significant Findings Across Studies 1, 2, and 3

Item	Study 1	Study 2	Study 3		
	Insomnia vs Normal	CBT change	ISQ change	SES change	WASO change
Sleep Needs					
1. I need 8 hours to function[a,b]		√	√		
3. Because getting older, need less sleep					
13. Should sleep like bed partner	√				
Sleep-Promoting Practices					
2. Need to catch up on poor sleep[a,b]		√			
6. Spend more time in bed to get more sleep and feel better		√			
7. I should stay in bed and try harder[a]		√	√	√	
9. Because getting older, should go to bed earlier					
11. Better off taking sleeping pills[b]	√				
26. Nightcap helps sleep					
27. Medication is the only solution[b]			√	√	
Effects of Insomnia					
4. Without sleep, may have a nervous breakdown					
5. Insomnia seriously affects health[a,b]	√				√
10. Poor night's sleep will interfere with activities[a,b]		√			
12. Poor sleep disturbs daytime mood[a,b]	√	√	√	√	
15. Afraid of dying in sleep	√				
18. Can't function without adequate sleep[b]					√
21. Poor energy/functioning due to poor sleep[a,b]	√				
29. Poor sleep affects physical appearance					
30. Avoid/cancel obligations after poor sleep[b]	√				
Control/Predictability					
8. Worried may lose control of sleep[a,b]	√	√	√	√	
16. Bad night follows good night's sleep	√		√		
17. Poor night's sleep affect whole week[a,b]	√		√	√	
19. Can't predict sleep[b]	√	√			
20. Can't manage negative sleep consequences[b]	√		√	√	√
22. No control over racing mind[a]	√		√	√	
23. Can lead satisfactory life despite insomnia	√				
25. Insomnia prevents enjoying life[b]	√		√		
28. Sleep is worsening and no one can help	√		√		√
Causal Attributions					
14. Insomnia is due to aging			√		
24. Insomnia due to a chemical imbalance[b]				√	

[a]Denotes items contained in the 10-item version of the DBAS
[b]Denotes items contained in the 16-item version of the DBAS
CBT refers to cognitive behavior therapy; ISQ, Insomnia Symptom Questionnaire; SES, Sleep Self-Efficacy Scale; WASO, wake after sleep onset.

ment, at a midtreatment assessment, during a posttreatment assessment, and again at a final follow-up time point. The ISQ is a 13-item questionnaire designed to assess the presence or absence of nocturnal and diurnal insomnia symptoms. A 100-mm horizontal response line labeled "not at all" at the left extreme and "frequently" at its right extreme accompanies each item. The SES contains 9 items designed to assess perceived control over sleep. The SES items include similar 100-mm analog scales labeled "Not at all [confident]" at their left extremes and "Very [confident]" at their right extremes. For both instruments, respondents draw a vertical line through the point on each item's analog scale to indicate their responses. Both instruments are scored in a manner similar to the DBAS, and a mean score across all items is used to represent the global score. Higher scores on the ISQ reflect more-pronounced insomnia symptoms, whereas higher scores on the SES connote more-perceived control over sleep.

Procedure

Pretreatment Pearson product-moment correlation coefficients for DBAS, ISQ, SES, and mean sleep-log WASO were calculated to ensure independence of the constructs. Data retained for the current study included (1) DBAS item difference scores (treatment endpoint – baseline value) for each of the 30 DBAS items, (2) ISQ difference scores calculated by subtracting participants' pretreatment ISQ score from the ISQ score obtained at the study endpoint, (3) SES difference scores computed in a manner similar to the ISQ difference scores, and (4) the change in WASO from pretreatment to the study endpoint expressed as a percentage of the pretreatment or baseline value. In the event that follow-up data were missing, a standard intent-to-treat data imputation (i.e., last value carried forward) was used to estimate endpoint data for the DBAS items, the ISQ, the SES, and sleep-log WASO. These data were obtained for each participant and placed in an electronic database for analyses.

Subsequently, a series of 3 sets of analyses were conducted. In each analysis, the sample was first divided into "improved" and "unimproved" groups based on the degree of change they showed on the selected outcome measures over their study participation.

For the first set of analyses, the sample was dichotomized on the basis of their ISQ difference scores. Those 50% showing the greater amount of decline in ISQ scores were labeled "improved," whereas the remainder were labeled "unimproved." For the second set of analyses, a median split of the SES change scores was used to dichotomize the sample into "improved" and "unimproved" groups. The 50% with the greatest increase in SES scores were labeled "improved," whereas the remaining participants were classified as "unimproved." For the final set of analyses, change in sleep-log WASO was used to dichotomize the sample. Those who showed a 50% or greater reduction in WASO from pretreatment to their study endpoint were labeled "improved," whereas the remaining individuals were labeled "unimproved." For each of these classification methods, we conducted a 2 (improved vs unimproved) x 30 (DBAS items) MANOVA using the DBAS difference scores as the dependent variables. Group comparisons (improved vs unimproved) were then conducted via ANOVAs for each of the 30 DBAS item change scores to follow up significant MANOVA effects.

RESULTS

The DBAS was significantly correlated with the ISQ ($r = .49$, $p < .001$) and SES ($r = -.35$, $p < .001$). The R^2 coefficients were quite low, thus the linear relationship between the DBAS and the ISQ and the SES accounted for relatively little of the shared variance (24% and 12%, respectively). The MANOVA showed a statistically significant group effect (ISQ-improved vs ISQ-unimproved) on DBAS difference scores, $F_{1,98} = 26.19$, $p < .001$. Follow-up ANOVAs revealed that ISQ-defined improved patients had significantly greater pretreatment-to-posttreatment DBAS item reductions on 13 of the 30 DBAS items than did those classified as unimproved on the ISQ. All 5 subscales of the DBAS were represented among these 13 items. The MANOVA comparing those categorized as improved on the SES versus those unimproved on SES revealed a statistically significant group effect, $F_{1,98} = 14.40$, $p = .0003$. Follow-up ANOVAs revealed that those classified as improved on the SES after treatment had significantly greater pretreatment-to-posttreatment increases on 8 of the 30 DBAS items than did those classified as unimproved on self-efficacy. Of these 8 items, 4 related to Control/predictability, 2 related to Sleep-Promoting Practices, 1 related to the Effects of Insomnia, and 1 related to Causal Attributions. Lastly, the MANOVA comparing those categorized as improved according to a 50% pretreatment-to-posttreatment decrease in WASO versus those unimproved on WASO (less than a 50% decrease) likewise revealed a significant group effect, $F_{1,98} = 4.14$, $p = .05$. On follow-up ANOVAs, WASO-improved patients had significantly greater DBAS change scores than those unimproved on the WASO index on 3 items. One item related to the Effects of Insomnia, and the other 2 items were related to Control/predictability.

Table 4 contains a summary of the results across all 3 studies. There were only 2 items that were significant across each of the 3 investigatory probes and 5 items that were nonsignificant across all probes used. The former 2 items related to insomnia effects and the controllability/predictability of sleep, whereas the latter 5 items related to beliefs about sleep needs, sleep-promoting practices, and the potential deleterious effects of sleep loss.

DISCUSSION

This study was conducted to identify the beliefs that are key in sustaining insomnia. We hypothesized that key items would be those that (1) discriminated those with primary insomnia from those without, (2) declined with belief-targeted therapy, and (3) related to other measures of clinical improvement following belief-targeted treatment. This omnibus hypothesis was confirmed for only 2 items related to the themes of control/predictability of sleep and the consequents of insomnia. Partial support for the omnibus hypothesis was found with 11 items that evidenced significant findings in at least 2 of the 3 probes. These items represented all DBAS thematic subscales except the subscale assessing presumed causes of insomnia. The current findings have implications for understanding the mechanisms underlying both insomnia and CBT for insomnia, identifying key targets for maximizing CBT outcomes, and considerations for future scale refinement.

Understanding the beliefs that are key in insomnia can aid our understanding of how cognition contributes to insomnia. In Study 1, 16 DBAS items were identified that discriminated primary insomnia sufferers from good sleepers. The majority of these items connoted beliefs pertaining to the adverse consequences after poor sleep, or to losing control of sleep, and the primary insomnia sufferers showed stronger endorsement of these themes than did our normal sleepers. These 2 sets of beliefs approximate the 2 core pathologic beliefs of hopelessness (e.g., "There's little chance of getting better") and helplessness (e.g., "There's nothing I can do…") found across other disorders, such as clinical depression or generalized anxiety disorder.[5,24] In these other disorders, maladaptive cognitions are presumed to be activated by underlying mood states and serve to perpetuate the illness. Interestingly, the constructs of hopelessness and helplessness were postulated as core cognitive insomnia-perpetuating agents in the first DBAS paper,[7] and our findings from Study 1 appear consistent with that notion.

Previous studies have supported the idea that insomnia sufferers' global DBAS scores. as well as scores on selected items. decline over the course of CBT.[9-11] Given that CBT is designed, in part, to modify sleep-interfering attitudes, examination of those items that change with this treatment provides us useful information about the cognitive processes in primary insomnia and the mechanisms of CBT. Our results from Study 2 showed 8 items that declined significantly more through CBT than they did in response to alternate behavioral therapies. Of these 8 items, 6 were included in the 10-item pool previously reported to be sensitive to CBT effects.[11] Several of the items we found that declined specifically with CBT were those closely related to both stimulus control and sleep-restriction instructions (e.g., "I need 8 hours to function," "Need to catch up on lost sleep," "Spend more time in bed to get more sleep"). Other items related to control and predictability, or managing the effects of insomnia (e.g., "Worried may lose control over sleep," "Can't predict sleep," or "Poor sleep disturbs daytime mood"). CBT appears to have beneficial effects in all areas of maladaptive sleep cognitions except for patients' beliefs about what causes insomnia. However, the effect of CBT on primary insomnia patients' beliefs relating to the causes of their sleep problems remains questionable, since the DBAS includes only 2 items to assess this theme.

The results from Study 3 add to those of Studies 1 and 2 by identifying potentially important targets for maximizing CBT outcomes. It was expected that a pretreatment-to-posttreatment CBT decline in beliefs important in the protraction of primary insomnia would relate to other indexes of clinical improvement.

There were 8 items that declined through our CBT intervention and related to clinical outcomes on a global insomnia rating, self-efficacy rating, and/or sleep-log WASO. Many of these items were related to the theme of control/predictability, suggesting that declines in patients' sleep-specific sense of helplessness are associated with insomnia-symptom improvement. There were also improvements on 2 items relating to beliefs about sleep-promoting habits that were related to improvements in both global insomnia symptoms and self-efficacy.

Curiously, there were 6 items that related to indexes of clinical improvement but did not differentiate insomnia patients from good sleepers. These items were "Need 8 hours to function" (# 1), "I should stay in bed and try harder" (# 7), "Insomnia is due to aging" (# 14), "Can't function without adequate sleep" (# 18), "Insomnia due to a chemical imbalance" (# 24), and "Medication is the only solution" (# 27). This is a perplexing finding, as disease-sustaining beliefs are presumably activated during the active phase of the disorder. In the case of these 6 items, declining scores related to clinical improvement, but they did not appear to be activated during the acute phase (e.g., they did not distinguish those with from those without the disorder).

Perhaps these unexpected results were due to demand characteristics associated with CBT. In CBT, beliefs are assessed at pretreatment, followed by extensive psychoeducation about how certain beliefs about sleep are maladaptive, and then the same beliefs are reassessed after treatment. The implicit expectation that these beliefs should change may lead some patients to acquiesce and rate items relevant to the sleep education received as very low after treatment. This may be especially true for the 3 DBAS items that showed CBT-specific changes but neither discriminated primary insomnia sufferers from normal sleepers nor related to any other index of clinical improvement. Given this possibility, future research both to assess the demand characteristics of CBT and to determine the mechanistic relevance of the 3 DBAS items mentioned to primary insomnia may prove useful.

An alternative explanation for our unexpected results is that the beliefs assessed by these 6 items mentioned above serve as mediators of treatment change through increasing treatment adherence. For example, if a poor sleeper believes that 8 hours is necessary for optimal functioning (# 1) or that increasing time spent in bed or sleep effort promotes sleep (# 2 and # 6), it could be difficult to adhere to sleep-restriction or stimulus-control instructions without prior modification of these beliefs. Likewise, the belief that insomnia is due to a chemical imbalance could conceivably undermine adhering to CBT, as it would be reasonable for such patients to assume that a chemical imbalance would be better remedied by pharmacotherapy.

We suspected that changes in key beliefs that are present during the active phase of the disorder (e.g., those items that distinguish good sleepers from primary insomnia sufferers) would occur in response to CBT or at least discriminate CBT responders (i.e., patients classified as improved) from nonresponders (i.e., those rated unimproved). However, 7 of the 16 DBAS items (i.e., items 4, 11,13, 15, 21, 23, 30) that discriminated primary insomnia sufferers from good sleepers neither changed significantly in response to CBT nor related to posttherapy improvement status of those receiving this treatment. Since these items are useful discriminators of primary insomnia sufferers, the specific beliefs they assess should be investigated as possible cognitive risk factors for future problems with insomnia. The identification of such items may also highlight a shortcoming of our CBT intervention. Specifically, these items may connote "gaps" in the cognitive therapy our CBT offers, so it may be useful to augment our cognitive module to address these beliefs.

In addition to suggesting the mechanistic relevance of various DBAS items, our results also suggested that 5 of the DBAS items (i.e., items 3, 4, 9, 26 and 29) may have no relevance to primary insomnia or its treatment. These items did not discriminate primary insomnia and normal sleeper groups, change significantly with CBT, or relate to indexes of improvement. However, it is possible that some of these items may be relevant to insomnia in selected populations. For example, 2 of the items relating to aging (e.g., "Because getting older, need less sleep" and "Because getting older, should go to bed earlier") have proven[7] useful in discriminating older adults with and without insomnia. Similarly, another 1 of the items relating to needing a nightcap to sleep (item 26) may have utility with those who use alcohol as a sleep aid. Hence, further scrutiny of these items may prove useful in certain settings.

The present results also may be used to guide clinicians and researchers in their decisions as to which of the currently available DBAS versions to use. In addition to the 30-item DBAS used herein, abbreviated 10-item[11] and 16-item[25] versions are available. For the reader's convenience, we have noted the items contained in these abbreviated versions, along with the other summary information provided in Table 4. Given the findings summarized in this table, we recognize that 1 version of the DBAS may appear to be most efficient or appropriate for some applications, whereas another version may be viewed as most useful for others. However, the use of varied versions of DBAS in research may make cross-study comparisons difficult and, thus, slow progress in our understanding of the relevance of sleep-related beliefs to insomnia. Thus, rather than encouraging the use of varied DBAS versions, perhaps the findings reported herein could contribute to a consensus among insomnia researchers as to which of these versions represents the most theoretically and pragmatically useful instrument. In our view, the most omnibus version of the DBAS would have good psychometric properties but would also differentiate the beliefs important in insomnia from sleep beliefs that are found in those without insomnia (e.g., good sleepers) and would detect treatment response to a treatment that specifically targets maladaptive cognitions.

The findings reported herein must be considered within the context of a few methodologic caveats. Despite its strengths, the DBAS includes a small number of items that assess beliefs relating to sleep needs and causal attributions, and expansion of these scales may be important. There may be additional beliefs that are important in insomnia but currently are not included on the DBAS. Studies 2 and 3 were conducting using our laboratory's CBT model, and it is possible that somewhat different findings would have been obtained with other versions of this treatment. There were only a limited number of CBT outcomes considered, and it is possible that other indexes (e.g., sleep-onset latency or daytime sleepiness ratings) may better relate to posttreatment belief change. Whereas the DBAS was modestly correlated with 2 of the clinical outcome measures (ISQ and SES), the DBAS does share method variance (e.g., they all are paper-and-pencil measures) with these other instruments. As such, use of such measures may provide a somewhat inflated view of the relevance of cognitive changes to clinical improvement in insomnia therapy.

Finally, our microanalytic approach of examining changes in individual items over time admittedly is encumbered with possible measurement error due to the inherent unreliability of single items. Although our large sample and conservative statistical approach (e.g., use of MANOVA) may have offset this problem somewhat, our findings clearly warrant replication. Despite these various limitations, our results support the utility of the DBAS in insomnia research and provide some insights into the specific sleep-related beliefs that warrant treatment consideration.

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