Research Article

RESIDUAL SLEEP BELIEFS AND SLEEP DISTURBANCE FOLLOWING COGNITIVE BEHAVIORAL THERAPY FOR MAJOR DEPRESSION

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Background: *Sleep disturbance is a commonly reported residual symptom after effective depression treatment. This residual sleep impairment, as well as the presence of problem levels of certain sleep beliefs, may be important for depressive relapse prevention, and as such should be addressed in treatment. The following study examined residual sleep disturbance and residual maladaptive sleep beliefs in those treated with Cognitive Behavior Therapy for depression.* **Methods:** *Participants (N = 24) were clinic patients seeking treatment for depression at a community clinic. Repeated measures analyses of variance tested pre- to posttreatment change on depression symptoms, general negative beliefs, sleep quality, and maladaptive sleep beliefs.* **Results:** *As expected, significant time effects were found for depressive symptoms and general negative beliefs. Sleep quality scores also decreased significantly at posttreatment; however, 92% of those no longer meeting depressive criteria continued to endorse residual sleep disturbance, according to an established clinical cutoff score of > 5 on a validated measure of sleep quality (the Pittsburgh Sleep Quality Index). There were no significant pre- to posttreatment changes for maladaptive sleep beliefs.* **Conclusions:** *The results indicate that sleep disturbance and maladaptive sleep-related beliefs remain a problematic residual symptom of remitted depression. These findings are discussed with reference to improving cognitive behavioral treatments for depression in order to help reduce rates of residual sleep problems. Depression and Anxiety 28:464–470, 2011.*

Key words: *depression; sleep disturbance; cognitive behavioral therapy for depression; beliefs about sleep; residual insomnia*

Depression is a devastating and debilitating disorder, partly because of its recurrent nature. Fortunately, managing and ameliorating depression is possible with psychological and pharmacological intervention. Although both forms of treatment are similarly effective,[1,2] the relapse rates following these treatments demonstrate a need to implement long-term or maintenance therapy for depression. For example, pharmacotherapy has associated relapse rates of approximately 50–80% and Cognitive Behavioral Therapy (CBT) for depression has associated relapse rates ranging from 20–45%.[3–7] It is evident that the durability of these treatments leaves room for improvement and, as a result, these rates have prompted focus on relapse prevention

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strategies, such as maintenance pharmacotherapy and Mindfulness-Based Cognitive Therapy.[8]

Another approach to improving outcomes is to focus on addressing common residual problems, so that such symptoms do not remain after depression treatment or lead to subsequent depressive episodes. Such residual problems can lead to increased subjective distress[9] as well as higher likelihood of depressive relapse.[10] Specifically, insomnia has been reported as a commonly occurring residual symptom that remains a problem after both pharmacotherapy and CBT for depression.[11] Within the context of depression, insomnia has been linked to poorer treatment outcomes[12] and increased suicide risk.[13] Furthermore, the variability of residual insomnia predicts depressive relapse and recurrence, and insomnia and fatigue are among the most variable residual symptoms.[10] Given the deleterious consequences that result from insomnia in depression, there may be a need to address sleep disturbances within depression treatment to optimize outcome and maintenance of therapeutic gains. Moreover, Manber et al. demonstrated that insomnia can easily be addressed during depression treatment, and that adding CBT for insomnia to an antidepressant produces almost double the remission rate to using an antidepressant medication alone.[14]

CBT for insomnia is widely considered to be an effective treatment for primary insomnia,[15–18] and preliminary research suggests that it is also effective in the treatment of comorbid depression and insomnia.[14,19,20] The main behavioral components (i.e., stimulus control and time-in-bed restriction) of CBT for insomnia address the circadian, homeostatic, and arousal systems, which are thought to regulate our day-to-day sleep schedules. Cognitive restructuring is primarily designed to target rigid, and often inaccurate, beliefs about sleep (i.e., "I need 8 hours of sleep to function"), which are thought to perpetuate insomnias.[21,22] Rigidly held beliefs about sleep can include unrealistic expectations about one's sleep, such as one night of poor sleep will interfere with the remaining week of sleep, and the need to cope with the daytime consequences of sleep loss by engaging in sleep-specific safety behaviors (i.e., spending excessive time in bed, increased consumption of caffeine) and sleep-related anxiety (i.e., worrying about daytime fatigue) at night.[22–25] These maladaptive beliefs are problematic in that they can increase distress and arousal regarding sleep, which then reinforce the unhelpful beliefs and behaviors, and ultimately maintain sleep difficulties.[21,26] Such maladaptive sleep beliefs have been shown to distinguish between good and poor sleepers, and these beliefs have been shown to improve with CBT for insomnia to a significantly greater extent than they do with other behavior-based therapies.[27] This study also revealed that decreases in maladaptive sleep beliefs from pre-to posttreatment were associated with clinically relevant improvements in other sleep indices. As a result, addressing these maladaptive sleep beliefs in treatment are an important aspect of recovery from insomnia. Indeed, these maladaptive beliefs have been shown to decrease, along with the insomnia symptoms themselves, with effective belief-targeted CBT for insomnia.[26–28]

Whereas those with insomnia tend to have maladaptive beliefs about sleep and their ability to cope with sleep loss, those with depression tend to have maladaptive negative beliefs about themselves, the world, and the future. Modifying such beliefs with CBT to include more realistic or helpful appraisals is associated with improvements in depression.[29] Because insomnia and depression so commonly co-occur, one might think that the excessive levels of negative sleep beliefs held by those individuals with depression may be due to or inflated by the globally negative beliefs typical of a major depressive episode. We recently tested this hypothesis in several patient groups with both insomnia and depression, and found that controlling for levels of depression did not eliminate these unhelpful sleep beliefs;[27] that is, they remained as pathologically high on maladaptive sleep beliefs as those with diagnoses of primary insomnia only. Thus, there is something unique about the content of maladaptive beliefs across these two commonly comorbid disorders. However, this study only examined actively depressed patients in a cross-sectional sample, and thus it would be informative to test whether such patients continue to have beliefs known to perpetuate insomnia posttreatment when the depressive episode is no longer present.

The following study examined levels of residual sleep disturbance and problematic sleep beliefs in individuals treated for depression with CBT. Given that CBT for depression currently does not actively target insomnia, it is important to further understand the rate of residual sleep disturbance after undergoing CBT for depression, as residual insomnia can lead to depressive relapse. Residual insomnia rates following remission suggest that between 30 and 55%[11,30] of those who recover from depression have residual insomnia. Using Hamilton Rating Scale for Depression (HRSD) sleep items, Carney et al.[11] found that such rates of residual insomnia does not differ between those who remitted after CBT for depression or pharmacotherapy for depression. Although previous studies[31] have shown that using sleep items on a depression measure, such as the HRSD, is a valid approach to assessing insomnia in this population, it would be preferable to use a validated measure for sleep disturbance. As such, to improve upon existing residual insomnia studies, the recommended (see Buysse et al.[32]) Pittsburg Sleep Quality Index (PSQI)[33] was used to assess residual sleep quality problems. Thus, the first hypothesis of this study is that while depression is expected to decrease significantly post-treatment, insomnia will not significantly improve and high rates of the sleep disturbance will persist post CBT for depression.

The second study aim is to examine whether there are residual maladaptive sleep-related beliefs post-CBT treatment. Because CBT for depression targets general negative beliefs rather than maladaptive beliefs specifically related to sleep, sleep-specific maladaptive beliefs are not likely to decrease with treatment.

METHODS

PARTICIPANTS

Participants ($N = 24$) were clinic patients seeking treatment for depression at the Cognitive Behavior Therapy (CBT) Clinic at the Centre for Addiction and Mental Health (CAMH) in Toronto, Canada. The sample was 66.7% female and ranged in age from 20 to 69 years ($M = 40$, $SD = 12.61$). All participants had a primary diagnosis of Major Depressive Disorder (MDD) according to the Structured Clinical Interview for Axis I Mental Disorders (SCID-I)[34] and had pre-treatment BDI-II scores ≥ 17. The majority of participants (80%) had recurring episodes of MDD, whereas 15% had a single episode and 5% were diagnosed with a chronic single episode (i.e., the episode has lasted for two years or greater). Patients were admitted for treatment with a concurrent Axis I or Axis II diagnosis, provided that the primary diagnosis was MDD (e.g., MDD was the patient's chief complaint and was viewed by the clinical interviewer as the most prominent problem). Secondary psychiatric diagnoses and patient medications are displayed in Table 1.

MEASURES

The Beck Depression Inventory, Second Edition (BDI-II)[35] is a 21-item self report measures that assesses common depressive symptoms, such as depressed mood, hopelessness, suicidal ideation, sleep disturbance, and appetite change. Suggested guidelines for the interpretation of scores: scores of 0–13 suggest minimal depression, scores of 14–19 indicate suggest depression, scores of 20–28 indicate moderate depression, and scores of 29 or above suggest severe depression. Scores greater than 17 are suggestive of clinical depression.[35] The BDI-II has good internal consistency (Cronbach's $\alpha = .92$ in outpatient populations).[36] It also has well established content validity and is good at differentiating between depressed and non-depressed individuals.[35,37]

The HRSD[38] is a 17-item clinician-administered scale that assesses depression severity, and includes symptoms such as depressed mood, guilt and insomnia. Items on the HRSD are measured on 3- or 5-point scales, and scores range from 0 to 52, with higher values representing greater symptoms. Scores greater than 14 are suggestive of clinical levels of depression.[38] The HRSD has both good internal reliability and inter-rater reliability.[39] The HRSD has high convergence of depressive symptoms with the BDI, with correlations ranging from .70 to .80.[39,40]

The Pittsburgh Sleep Quality Index (PSQI)[33] is a recommended self-report measure for assessing sleep quality disturbance. It includes 19 items that query aspects of subjective sleep quality. The PSQI is comprised of seven components, measuring sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction.[32,33] A global score, ranging from 0 (good sleep quality) to 21 (poor sleep quality), are obtained by summing the seven component scores. Scores greater than five are suggestive of sleeping difficulties.[33,41] The PSQI accurately discriminates between good and poor sleepers, and is highly correlated with other subjective sleep measures, such as daily sleep diaries.[41] The PSQI is highly reliable with good internal reliability (Cronbach's $\alpha = .83$) and test–retest reliability ($r = .87$).[41] Past studies have used the PSQI to assess sleep quality in those diagnosed with depression.[42]

The Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16)[43] is a 16-item self-report measure assessing unhelpful sleep-related beliefs. It consists of four domains: consequences of insomnia, worry about sleep, sleep expectations, and medication. The individual rates his/her level of agreement with each statement on a 10-point scale ranging from 0 ("strongly disagree") to 10 ("strongly agree"). The DBAS-16 is a short version of the original DBAS, which contains 30 items. The DBAS-16 is scored by calculating a mean-item score across all 16 items. The DBAS-16 demonstrates adequate internal consistency (Cronbach's $\alpha = .79$) and has appropriate convergent validity with the Insomnia Severity Index, sleep diaries, and polysomnography.[43] The DBAS-16 can effectively discriminate between those who do and do not have clinical levels of unhelpful sleep beliefs via a cutoff score of 3.8, which maximized both sensitivity (80%) and specificity (76%) based on a Receiving Operator Characteristic curve.[44]

The Dysfunctional Attitudes Scale (DAS)[45] was used to measure maladaptive patterns of thinking held by patients with depression.[45,46] This study used the 40-item version with ratings options from 1 ("totally agree") to 7 ("totally disagree"). Higher scores indicate more maladaptive attitudes and cognitive distortions.[45] The DAS has very good internal consistency (Cronbach's $\alpha = .93$) and good concurrent validity, as it correlates highly with the BDI (rs ranging from .53 to .65) and other measures of depression.[45]

TABLE 1. Comorbid psychiatric diagnoses and antidepressant medication use

Secondary diagnoses	%
Social phobia	21.1
Dysthymic disorder	10.5
Panic with or without agoraphobia	5.3
Substance use/dependence	5.3
Other	26.3
None	31.5
Antidepressant medication	**%**
SSRI	29.2
Tricyclics	4.2
Other	20.8
None	45.8

PROCEDURES

A clinic sample of patients seeking depression treatment from CAMH were screened by a Master's level clinician using the SCID,[34] to confirm the primary diagnosis of MDD. Pre- and posttreatment scores were collected for the following measures: BDI-II, HRSD, PSQI, DBAS-16, and DAS. The structure of the CBT treatment sessions followed a 20-week protocol based on "Mind over Mood."[47] Before each CBT session, participants completed BDI-II inventories to monitor weekly depression levels. A posttreatment evaluation that included the SCID and the abovementioned inventories were administered after completing the course of therapy.

ANALYSES

To determine whether responses to self-report measures changed across the course of treatment, a series of repeated measures Analyses of Variance (ANOVAs) were conducted, using the baseline and posttreatment scores for each of the following measures: BDI-II, HRSD, PSQI, DBAS-16, and DAS.

RESULTS

Pre- and posttreatment scores on the BDI-II, HRSD, PSQI, DBAS-16, and DAS are presented in Table 2. The ANOVA revealed a significant time effect, thus posttreatment scores on both the BDI-II and HRSD decreased significantly at posttreatment. Following CBT for depression, 36.8% of participants remitted from depression, meaning that they no longer met criteria for MDD on the SCID, and had a posttreatment HRDS score ≤ 7.[48] There was a 30.4% remission rate, according to a BDI-II score ≤ 8 and not meeting full MDD criteria on the SCID.[48] Posttreatment maladaptive depressive beliefs, as measured by the DAS, significantly decreased from pretreatment scores (see Table 2). Remission rates, as defined by the BDI-II or HRSD, did not vary according to whether or not participants were on antidepressant medication at pretreatment, $\chi^2(1) = .68$, $P = .41$; $\chi^2(1) = .69$, $P = .41$, respectively.

PSQI scores also changed significantly from pre- to posttreatment (see Table 2). However, 91.3% of the total sample continued to endorse having clinically significant sleep disturbances following treatment, compared to 96.4% at pretreatment, according to a score > 5 on the PSQI.[33] Furthermore, posttreatment sleep disturbances continued to be endorsed by 92.3% of participants no longer meeting depressive criteria, according to the SCID (BDI: $M = 12.92$, $SD = 11.09$; HRSD: $M = 7.23$, $SD = 4.93$) (i.e., residual sleep impairment). Maladaptive sleep beliefs, as measured by the DBAS-16, did not change significantly from pre- to posttreatment (see Table 2). At posttreatment, 82.6% of participants, compared to 84% at pretreatment, continued to have DBAS-16 scores above the suggested clinical cutoff of 3.8.[44] The sample included both males and females across a wide age span, so sex and age were controlled in each of the repeated measures analyses. There were no effects for gender or age, and no significant interactions on any of the measures (BDI-II, HRSD, PSQI, DBAS, DAS) at the $P < .05$ level.

TABLE 2. Means and standard deviations for self-report measures pre- and posttreatment

Measure	Pretreatment mean (*SD*)	Posttreatment mean (*SD*)	*F*-value	*P*	η^2
BDI-II	32.00 (10.56)	16.63 (11.76)	50.14	$<.001^{**}$	.71
HRSD	14.95 (6.92)	9.63 (6.34)	17.44	$<.001^{**}$	.49
PSQI	17.40 (8.17)	14.35 (7.76)	13.05	$.002^{**}$	.41
DBAS	5.45 (1.61)	5.45 (1.53)	0.001	.981	
DAS	4.46 (1.60)	3.99 (0.89)	5.22	$.035^{*}$	.22

BDI-II, Beck Depression Inventory; HRSD, Hamilton Rating Scale for Depression; PSQI, Pittsburgh Sleep Quality Index; DBAS, Dysfunctional Beliefs and Attitudes about Sleep Scale; DAS, Dysfunctional Attitudes Scale.
$^{*}P < .05$; $^{**}P < .01$.

DISCUSSION

This study examined rates of residual sleep disturbance, using a validated sleep measure in a clinic sample for those treated with CBT for depression. Consistent with the literature,[2,49,50] the study found that rates of depression decreased significantly over the course of treatment. Sleep quality also improved after CBT; that is, scores on the PSQI significantly decreased from pre- to postintervention. However, the vast majority of the sample (approximately 92%) who no longer met criteria for depression continued to have significant sleep impairment following treatment. That sleep quality did not improve even after the depression remitted indicates that the traditional approach to treating depression (i.e., treat the depression and the insomnia will disappear) is not supported. Instead, these results suggest that while sleep quality does seem to improve with CBT for depression, this treatment is not sufficient to address the sleep disturbance. Thus, although the improvement in reported sleep quality may have been statistically significant, one may question the clinical significance of this improvement, given the high rate of reported residual sleep difficulties. This finding raises concern because, left untreated, insomnia can have serious consequences.[51]

This study also demonstrates that along with decreases in posttreatment depression, general negative beliefs decreased with treatment, lending further validation to the effectiveness of CBT for depression in treating MDD. However, as expected, sleep-specific negative beliefs did not significantly decrease following treatment, suggesting that CBT for depression does account for patients' unhelpful beliefs about sleep. This finding is problematic because previous research has demonstrated that maladaptive sleep beliefs play an integral role in the perpetuation of the insomnia problem.[21,22] Insofar as these maladaptive beliefs can maintain the sleep disturbance, the reduction of such beliefs is considered to be important in the treatment of insomnia. Indeed, previous studies have demonstrated that maladaptive sleep beliefs improve with belief-targeted CBT for insomnia to a significantly greater extent than they do with pharmacotherapy[26] or nonbelief-targeted behavioral therapy.[27] Given that maladaptive sleep beliefs are also commonly found among individuals with depression,[24] treatment beyond CBT for depression is required to adequately address the sleep disturbance among both those with depression and insomnia.

Whereas one might assume that learning to challenge maladaptive thoughts more generally in CBT for depression would be effective for alleviating sleep-related thoughts too, this might be difficult without some psychoeducation about sleep. For example, if one was preoccupied with the idea that 8 hr is needed to adequately function, but they were unaware that quantity of sleep can be less important than the quality

and continuity of sleep, it may be difficult for them to challenge what seems to be a factual belief. However, even with the addition of cognitive and psychoeducational modules, one would expect that behavioral elements, such as stimulus control and sleep restriction, would be important adjuncts to CBT for depression given that cognitive therapy for insomnia is not a supported monotherapy.[15]

Taken together, the findings of this study highlight the importance of regarding insomnia as an autonomous disorder, often requiring treatment independent from or in conjunction with depression treatment. As discussed above, CBT for insomnia is one such treatment which has been shown to be efficacious[15–18] and has been designed to specifically target those factors that perpetuate the insomnia.[52] Findings from Manber et al.[14] support the addition of CBT for insomnia to depression therapy for alleviating both conditions. Although further studies are needed to corroborate these findings, this study provides preliminary support for the efficacy of CBT for insomnia in the treatment of comorbid insomnia and depression.

The PSQI results indicated that 91% of participants still complained of sleep disturbances. The exceptionally high rates (i.e., more than 90%) of residual sleep impairment according to the PSQI was surprising, especially compared to previous examinations of residual symptoms of depression, which have reported residual insomnia rates of 30–55%.[11,30] The high rate of residual sleep disturbance in our study suggests that untreated insomnia remains a major problem—one that might be even greater than was previously imagined. Other explanations include that the high rate was due to the small sample size or that the PSQI may be too sensitive for use in this population, and thus may not be the most appropriate tool to assess insomnia. Indeed, although the PSQI shows good discriminability for identifying poor sleepers versus good sleepers, the PSQI assesses sleep quality rather than the narrowly defined insomnia symptoms, and thus may overestimate residual insomnia. In addition, the PSQI items have some overlap with depression symptoms[53] and may be elevated in this population; although the analyses focused on the remitted group, so this may not be an issue. In contrast, previous estimates of residual insomnia using HRSD items may have been too low. Future studies must address this issue and explore other insomnia-specific measures, such as the Insomnia Severity Index or prospective sleep monitoring, in order to determine which measures best identify and evaluate sleep disturbances in depressed populations.

Although it is advantageous from a generalizability standpoint to have data from real-world treatment, there are drawbacks with such studies. There are potential confounds that could have been controlled for with the addition of a control group; without a control group, these results must be considered preliminary only. In addition, another limitation of this study is the small sample size; thus, the analyses may be underpowered. Nevertheless, despite the small sample, we did observe the expected results in the depression measures. Furthermore, the fact that more than 90% of participants endorsed having sleeping difficulties demonstrates the persistent and chronic nature of untreated insomnia, which clearly remains a major problem even after successful treatment of depression. In addition, although the literature has established a clear pattern between residual sleep disturbance and depressive relapse,[10] unfortunately this study does not have follow-up data to definitively conclude that this sample would have had greater rates of relapse. This study is also limited in that some participants were on antidepressant medication, whereas others were not taking any medication at the time. Patients were not required to start, end, or change their medication during the study; so, it is unknown as to whether antidepressant medications played a role in these patients' sleep or depression.

This study replicates previous research, which has also found that residual insomnia remains a problem amongst individuals treated for and remitted from depression.[11] In contrast to previous studies, this study examined rates of residual sleep problems using a validated sleep measure in a clinic sample for those treated with CBT for depression. Using the PSQI to measure residual sleep difficulties presents a notable strength over previous studies, which used the individual items from depression measures (see Carney et al.[11]). This study also highlights the problematic nature of maladaptive sleep beliefs. Indeed, maladaptive sleep beliefs are an integral component of CBT for insomnia that is not addressed in treatment for depression. Furthermore, these unhelpful sleep beliefs also remain cognitive risk factors for the development of subsequent depressive episodes, a finding that highlights the importance of addressing these beliefs in treatment. Taken together, the study results suggest that our depression treatment strategies are not effective enough with regard to sleep. Incorporating effective insomnia strategies, particularly belief-targeted insomnia treatment strategies into CBT for depression, may be a worthwhile treatment endeavor. Addressing residual sleep problems during treatment (i.e., so they do not become residual symptoms) may be an important inroad toward preventing recurrent depressive episodes.

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