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Article *in* Journal of Counseling Psychology · April 2015

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Journal of Counseling Psychology
2015, Vol. 62, No. 2, 115–123

0022-0167/15/$12.00 http://dx.doi.org/10.1037/cou0000059

A Randomized Controlled Trial of Cognitive Behavioral Therapy for Insomnia: An Effective Treatment for Comorbid Insomnia and Depression

Damon K. Ashworth and Tracey L. Sletten
Monash University

Moira Junge
Monash University and Melbourne Sleep Disorders Centre, East Melbourne, Australia

Katrina Simpson and David Clarke
Monash University

David Cunnington
Melbourne Sleep Disorders Centre, East Melbourne, Australia

Shantha M. W. Rajaratnam
Monash University

Insomnia and depression are highly comorbid conditions that show a complex, bidirectional relationship. This study examined whether cognitive–behavioral therapy for insomnia (CBT-I) delivered by a therapist compared with self-help CBT-I (written materials only) reduces insomnia and depression severity in individuals with comorbid insomnia and depression. A total of 41 participants (18–64 years; 25 females) with comorbid depression and insomnia, treated with antidepressants for at least 6 weeks, were randomized to receive 4 sessions of either CBT-I or self-help CBT-I over 8 weeks. Insomnia (Insomnia Severity Index [ISI]) and depression (Beck Depression Inventory-II [BDI-II]) were assessed at baseline, following each session, and at 3-month follow-up. Secondary outcomes were sleep quality and duration (actigraphy and diaries), anxiety, fatigue, and daytime sleepiness. Compared with self-help CBT-I, BDI-II scores in the CBT-I group dropped by 11.93 (95% confidence interval [CI] [6.60, 17.27], $p < .001$) more points, and ISI scores dropped by 6.59 (95% CI [3.04, 10.15], $p = .001$) more points across treatment. At 3-month follow-up, 61.1% of CBT-I participants were in clinical remission from their insomnia and depression, compared with 5.6% of the self-help group. ***Conclusions:*** CBT-I administered by a therapist produced significant reductions in both insomnia and depression severity posttreatment and at follow-up, compared with a control condition in which participants received only written CBT-I material. Targeting insomnia through CBT-I is efficacious for treating comorbid insomnia and depression, and should be considered an important adjunct therapy for patients with depression whose symptoms have not remitted through antidepressant treatment.

Keywords: insomnia, depression, CBT-I, self-help CBT-I, antidepressant

Supplemental materials: http://dx.doi.org/10.1037/cou0000059.supp

By the year 2030, unipolar major depression is projected to be the leading cause of disease burden worldwide (World Health Organization, 2004). The burden of major depressive disorder (MDD) includes an increased risk of mortality from cardiovascular death and stroke, as well as 20-fold increased risk of mortality from suicide compared with the general population (Lepine & Briley, 2011). The cognitive impairments and psychosocial dysfunction commonly found in MDD contribute to a lower quality of life and greater disability (Lepine &

Damon K. Ashworth and Tracey L. Sletten, School of Psychological Sciences, Monash University; Moira Junge, School of Psychological Sciences, Monash University, and Melbourne Sleep Disorders Centre, East Melbourne, Australia; Katrina Simpson, School of Psychological Sciences, Monash University; David Clarke, Southern Clinical School, Monash University; David Cunnington, Melbourne Sleep Disorders Centre; Shantha M. W. Rajaratnam, School of Psychological Sciences, Monash University.

Tracey L. Sletten reports her institution has received equipment donations or other support from Phillips Lighting, Phillips Respironics, Optalert™, and Compumedics. Tracey L. Sletten serves as a Project Leader in the Cooperative Research Centre for Alertness, Safety and Productivity. Shantha M. W. Rajaratnam has served as a consultant through his institution to Vanda Pharmaceuticals, Phillips Respironics, EdanSafe, The Australian Workers' Union, National Transport Commission, Transport Accident Commission, and New South Wales Department of Education and Communities and has, through his institution, received research grants from Vanda Pharmaceuticals, Takeda Pharmaceuticals North America, Phillips Lighting, Phillips Respironics, Cephalon, and ResMed Foundation and reimbursements for conference travel expenses from Vanda Pharmaceuticals. Monash University has received equipment donations or other support from Optalert™, Compumedics, and Tyco Health Care. He has also served as an expert witness and/or consultant to shift work organizations. Shantha M. W. Rajaratnam also serves as a Program Leader in the Cooperative Research Centre for Alertness, Safety, and Productivity.

Correspondence concerning this article should be addressed to Shantha M. W. Rajaratnam, School of Psychological Sciences, Monash University, Building 17, Wellington Road, Clayton VIC 3800, Australia. E-mail: shantha.rajaratnam@monash.edu

Briley, 2011). Higher rates of both absenteeism and presenteeism in MDD have been estimated to result in a loss of $36.6 billion per year in the United States (Kessler et al., 2006).

Many patients undergoing pharmacological treatment for depression fail to improve, with an even lower percentage reaching stable remission (Walsh, Seidman, Sysko, & Gould, 2002). A meta-analysis of studies treating MDD with selective serotonin reuptake inhibitors (SSRIs), the most commonly prescribed antidepressant, reported a mean response rate of only 50.1% (Walsh et al., 2002). Over 60% of remitted MDD patients experience a recurrence of symptoms after 5 years and 85% after 15 years when treated in specialized mental health settings (Hardeveld, Spijker, De Graaf, Nolen, & Beekman, 2010). Consequently, there remains a need for treatments for MDD that will not only improve remission rates, but also decrease recurrence.

Insomnia and depression are highly comorbid conditions, with 67% of individuals with MDD also meeting criteria for chronic insomnia (Franzen & Buysse, 2008). The traditional view has been that insomnia, as a symptom of MDD, is expected to develop following the onset of MDD, not require specific treatment, and resolve with effective treatment of the depressive symptoms. There is now substantial evidence challenging this view. First, between 40 and 69% of individuals with MDD report that their insomnia appeared before any other depressive symptom (Johnson, Roth, & Breslau, 2006). Second, when comorbidities occur, prior insomnia significantly predicts the onset of future MDD, but prior MDD does not predict the onset of future insomnia (Johnson et al., 2006). For this reason, it is thought that poor sleep quality is a precursor of depression. Third, untreated insomnia symptoms increase the duration and the severity of a depressive episode (Staner, 2010), including increased frequency of suicidal ideation in depressed individuals with insomnia (Thase, 1998). Fourth, chronic insomnia remains in nearly half of cases when MDD remits through antidepressant treatment (Franzen & Buysse, 2008). This ongoing poor sleep subsequently increases the likelihood of depressive relapse (Perlis, Giles, Buysse, Tu, & Kupfer, 1997). Finally, combined treatment of a hypnotic for insomnia and an antidepressant for depression significantly improved insomnia and depression severity compared with antidepressant treatment alone (Fava et al., 2006). Cumulatively, these lines of evidence demonstrate a need for targeted treatment of insomnia in individuals with comorbid insomnia and MDD.

Cognitive–behavioral therapy for insomnia (CBT-I) has equal or superior efficacy to hypnotics, as well as fewer side effects and longer-lasting benefits following the discontinuation of treatment (Butler, Chapman, Forman, & Beck, 2006; Riemann & Perlis, 2009). CBT-I combines several techniques demonstrated to be efficacious as stand-alone treatments for insomnia (Edinger & Means, 2005) including stimulus control, sleep restriction, relaxation, and cognitive therapy. Meta-analysis of CBT-I for primary insomnia reveals that it significantly improves sleep quality and sleep efficiency (SE), shortens sleep onset latency (SOL), and decreases wake after sleep onset (WASO), with large effect sizes in both SOL (1.05) and WASO (0.92; Morin, Culbert, & Schwartz, 1994). These improvements are typically maintained through to follow-up, even 1 year later (Edinger & Means, 2005). CBT-I is also efficacious in the treatment of insomnia associated with medical and psychiatric comorbidities (Morin et al., 2006).

To date, eight studies have investigated the efficacy of CBT-I in individuals with comorbid depression. Although these studies are promising, none provides conclusive evidence for the efficacy of CBT-I in reducing depression severity. Five of the studies were not randomized controlled trials (RCTs) and did not include a control condition for the intervention (Lancee, van den Bout, van Straten, & Spoormaker, 2013; Manber et al., 2011; Maroti, Folkeson, Jansson-Frojmark, & Linton, 2011; Morawetz, 2003; Taylor, Lichstein, Weinstock, Sanford, & Temple, 2007); thus causal effects from the CBT-I cannot be strongly inferred. Two of the remaining studies that were RCTs (Manber et al., 2008; Wagley, Rybarczyk, Nay, Danish, & Lund, 2013) showed trends toward reduced depression ratings in the CBT-I group compared with control, but these effects were not statistically significant, in one study possibly due to concurrent commencement of antidepressant therapy (Manber et al., 2008). The final study was an assessor-blind RCT that demonstrated significant reductions in depression scores. However, the control condition was "treatment as usual," and the authors acknowledge that the study cannot assess whether the psychological therapy itself or careful monitoring of patients resulted in improvements in insomnia and depression (Watanabe et al., 2011).

This study used a randomized, parallel groups design to investigate the efficacy of CBT-I as an adjunctive therapy to pharmacological antidepressant treatment for individuals with comorbid insomnia and depression. The primary aim of the study was to test the efficacy of CBT-I in reducing depression and insomnia severity in participants with comorbid insomnia and MDD whose depressive symptoms have not remitted with pharmacological antidepressant treatment. Specifically, the study examined whether CBT-I would significantly reduce insomnia severity (Insomnia Severity Index) and depression severity (Beck Depression Inventory-II) when compared with self-help CBT-I in nonremitted MDD participants currently undergoing antidepressant treatment. In order to inform the direction of treatment effect, the study also examined whether insomnia improvement predicted depression improvement, and conversely whether improvements in depression predicted improvements in insomnia. As CBT-I was designed to target insomnia symptoms, it was hypothesized that insomnia improvement would significantly predict depression improvement through CBT-I treatment, but depression improvement would not significantly predict insomnia improvement. As a secondary aim, the study examined the efficacy of CBT-I on measures of sleep quality, anxiety, fatigue, and sleepiness, as well as subjective and objective sleep variables.

Method

Participants

From 159 enquiries received and 61 participants screened, 41 participants (25 female, 16 male) between the ages of 18 and 64 years were recruited from the community via advertisements and through referrals from primary care physicians and mental health clinicians. Participants were required to have been previously diagnosed with MDD and be taking antidepressant medication at adequate dose according to label (including at least one dose increase as permitted by label) for at least 6 weeks prior to the screening visit. Participants were also required to meet the *DSM–IV–TR* (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*) criteria for pri-

mary insomnia, which includes reporting difficulties initiating or maintaining sleep for at least 1 month contributing to either significant daytime functional impairment or distress (American Psychiatric Association, 2013). Participants were required to have a Beck Depression Inventory (BDI-II) score of at least 17, which has high sensitivity (81%) and specificity (79%) in identifying clinically significant depression in individuals with insomnia (Carney, Ulmer, Edinger, Krystal, & Knauss, 2009), and a reported SOL or WASO of greater than 30 min at least 3 nights per week, which is considered to be the most accurate criteria for correctly identifying those with insomnia (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003).

Participants had a Body Mass Index (BMI) between 19.3 and 34.0 kg/m^2, had not taken sleep medication in the 14 days prior to screening, and did not have a current diagnosis of another sleep disorder, all confirmed by self-report. They had adequate English language fluency, agreed not to undergo any other treatment for depression or insomnia during their participation in the study, and did not exhibit any active suicidal potential or psychotic features, as determined through a semistructured clinical interview at the screening visit. Participants did not have MDD with a seasonal pattern, any other comorbid psychiatric diagnoses except for anxiety spectrum disorders, or any uncontrolled medical conditions. Participants were allowed to take medication to help control their medical conditions, and were required to remain on their antidepressant medication during their participation in the study. All participants gave written informed consent and were not paid for their participation in the study. The study protocol was evaluated by an Internal Review Board and received approval at all relevant institutions.

Protocol

Participants underwent a screening visit, completed baseline data to ensure eligibility, and were randomized to either CBT-I or the self-help CBT-I (written materials only) control condition (Figure 1). The CBT-I group received written materials and a brief psychotherapy intervention, whereas the self-help group received only written materials. Each condition consisted of four sessions administered over an 8-week interval (one session every 2 weeks).

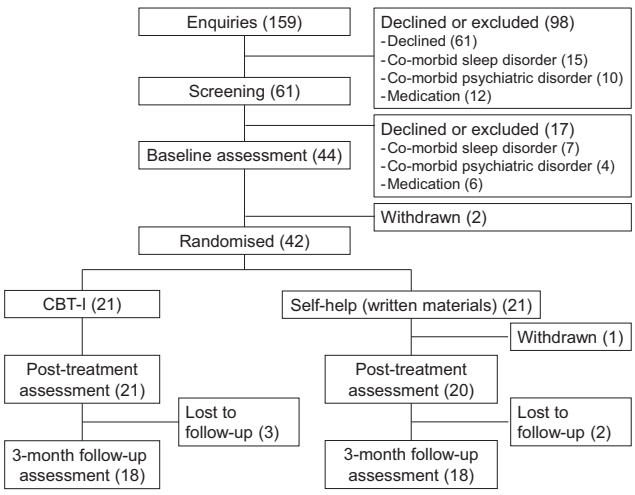

Figure 1. Participant disposition.

Session content was as follows: sleep information, sleep hygiene recommendations and abdominal breathing (Session 1), stimulus control, sleep restriction, and progressive muscle relaxation (Session 2), cognitive restructuring and imagery relaxation (Session 3), and self-management, relapse prevention, and mindfulness relaxation (Session 4). See supplemental materials for further details about session content.

For the self-help group, each session consisted of approximately 15 min contact time with the therapist. During this time, the participants' sleep diary and activity monitor were collected, and they completed assessment questionnaires for the particular session. Participants were provided with written materials describing the CBT-I content relevant to the particular session for them to read in a nondirective, self-help format with no guided practice, feedback, or verbal instruction. Casual conversation was permitted, but sleep-related issues were not discussed.

For the CBT-I group, each session consisted of approximately 50 min contact time with the therapist, and included discussion of the written information provided and any sleep-related questions or concerns. Feedback was provided based on sleep diary and actigraphy data. The CBT-I treatment and self-help sessions were conducted by a single therapist (DA), who was a postgraduate level clinical psychology student who had observed and underwent 12 hr of CBT-I training by a practicing psychologist specializing in CBT-I. A semistructured CBT-I therapist manual was developed in collaboration with CBT-I specialists to ensure that the treatment was delivered as thoroughly and as consistently as possible. To determine integrity of the CBT-I intervention, all CBT-I sessions were video-recorded, and 25% of these sessions were randomly selected for review by a supervisor to ensure that all critical components of the protocol were followed in the correct order as set out in the CBT-I therapist manual. The CBT-I participants were also encouraged to implement the strategies between their sessions and discuss which techniques they found most helpful in improving their sleep. Conversely, participants in the self-help CBT-I group were only given the written information and were not given the opportunity for discussion or feedback about their sleep during the treatment phase.

The posttreatment assessment took place at the conclusion of the fourth treatment session. A follow-up assessment was conducted with the participants 3 months after the final treatment visit to determine longer-term changes following CBT-I treatment and self-help CBT-I. After follow-up, the self-help CBT-I participants were offered up to four additional sessions of traditional CBT-I. Data were not collected during this time.

Primary Outcome Measures

The Insomnia Severity Index (ISI) and the Beck Depression Inventory, Second Edition (BDI-II) were the primary insomnia and depression outcome measures, respectively, assessed at baseline (Week 0), following each session (Weeks 2, 4, 6), at posttreatment (Week 8), and at 3-month follow-up (Week 24). The ISI is a seven-item self-report measure that produces an overall score from 0 to 28, with 0–7 indicating absence of insomnia, 8–14 indicating not clinically significant (or subthreshold) insomnia, 15–21 indicating a moderate severity of clinical insomnia, and 22–28 indicating severe insomnia (Morin, 1993). The ISI has good internal consistency (Cronbach's alpha 0.90 and 0.91; Morin, Belleville,

Belanger, & Ivers, 2011) and is sensitive in detecting perceived improvements across treatment (Bastien, Vallières, & Morin, 2001). The BDI-II (Beck, Steer, & Brown, 1996) is a 21-item self-rated measure that is considered the gold-standard in assessing depression severity and determining changes made through treatment. The sum of all items produces an overall depression severity score (range = 0–69), with 0–13 indicating minimal depression, 14–19 indicating mild depression, 20–28 indicating moderate depression, and 29 and above indicating severe depression (Beck et al., 1996).

Secondary Outcome Measures

The Pittsburgh Sleep Quality Index (PSQI), Depression Anxiety Stress Scales (DASS-21), Fatigue Severity Scale (FSS), and the Epworth Sleepiness Scale (ESS) were the secondary outcome measures, and were assessed at baseline (Week 0), posttreatment (Week 8), and 3-month follow-up (Week 20) to determine changes in subjective sleep quality, anxiety, fatigue, and daytime sleepiness. These instruments and the respective results are described and discussed in the supplemental materials.

Sleep Measures

A modified version of the Pittsburgh Sleep Diary (Monk et al., 1994) was completed by participants in 2-Week intervals at baseline (Weeks 0–2), throughout treatment (Weeks 2–8), and for 2 Weeks before the 3-month follow-up assessment (Weeks 18–20) to determine subjective changes in SE, SOL, and total sleep time (TST).

Wrist activity was monitored by actigraphy (Actiwatch-2, Phillips Respironics, Bend, Oregon) at baseline (Weeks 0–2) and throughout the treatment (Weeks 2–8) in 2-week intervals to determine objective changes in SE, SOL, and TST, calculated using Actiware software version 5.70. Participants continuously wore the actigraph device on their nondominant wrist. Wrist activity levels were collected in 1-min epochs and were downloaded following each treatment session using the medium sensitivity threshold (40 counts per epoch), as this setting is reported to moderately correlate with polysomnographic sleep variables in individuals with depression and insomnia (McCall & McCall, 2012). Bedtime and rise-time were determined using a combination of the marker on the actigraph device and the times recorded on the sleep diary, with the marker being used if there was less than 30 min discrepancy between the two sources or if the sleep diary data were unavailable. If the marker was not pressed or was more than 30 min different to the sleep diary times and did not correspond with an increase or decrease in activity levels, the sleep diary times were used.

Individual actigraphy results were provided to CBT-I participants within 24 hr of each session, to provide feedback on their progress in improving their sleep. If average subjective SOL, SE, or TST values were poorer than the objective sleep values, these discrepancies were also provided to CBT-I participants to target unhelpful sleep state misperception. At the conclusion of the follow-up assessment, participants in the self-help group were given their objective sleep results, and encouraged to ask questions about their data.

Data Analysis

Data were analyzed using the Statistical Package for Social Sciences Version 20 (SPSS, Chicago, IL). Mixed model analysis of covariance (ANCOVA) was conducted to assess the impact of the CBT-I and self-help interventions on outcome measures across time points, with baseline (Week 0) entered as a covariate: Week 2, 4, 6, 8, and 20 for BDI-II and ISI; Week 4, 6, 8, and 20 for subjective sleep variables; and Week 4, 6, and 8 for objective sleep variables, and Week 8 and 20 for PSQI, DASS-A, FSS, and ESS. To reduce experiment-wise error, the critical p value was adjusted to $p = .025$ for the primary outcome measures (ISI and BDI-II). Critical p values were not adjusted for secondary outcomes. The self-help participant who withdrew following their first treatment session was excluded from the analysis so that their baseline data did not skew the overall group results at posttreatment. Any other missing data was replaced using the group mean at each time point, including the participants who dropped out between the posttreatment and 3-month follow-up assessments. Where the ANCOVA findings indicated a significant interaction effect, post hoc independent variable t tests were conducted to determine significant group differences at each time period. To determine levels of reliable and clinically significant change on an individual level between groups at follow-up, the reliable change criterion (Jacobson & Truax, 1991) was calculated to be 3.54 for ISI and 6.85 for BDI-II. The previously established cut-off scores for determining remission from insomnia (ISI < 8) and depression (BDI-II < 14) were used to establish clinically significant change. Chi-square analysis was conducted to assess group differences on reliable and clinically significant change outcomes. Indirect effects of insomnia improvement on depression outcome and depression improvement on insomnia outcome at follow-up were assessed for mediation using a series of regression analyses, as outlined in the stepwise approach to mediation testing (Baron & Kenny, 1986). All analyses were performed with 1,000 bootstrap samples to correct for bias and increase power (Hayes, 2009). Indirect effects and 95% bias corrected bootstrap confidence intervals were checked for significance using the Indirect Mediation Effect Confidence Interval Calculator (http://www.danielsoper.com/statcalc3/calc.aspx?id=88). If these confidence intervals did not include zero, there was 95% likelihood that at least partial mediation was supported (Hayes, 2009). If the direct effect was still significant after controlling for the mediator, the finding supported partial mediation. If the direct effect was no longer significant after controlling for the mediator, the finding supported full mediation (Baron & Kenny, 1986).

Results

At baseline, no significant group differences were found for age, BMI, gender, highest level of education, occupational status, marital status, and all primary and secondary outcome measures ($p > .05$; Table 1).

Primary Outcome Measures

ISI was found to have good internal consistency: Cronbach's alpha 0.76 for baseline assessment and 0.90 for all time points.

CBT-I was more effective than self-help CBT-I in reducing both depression and insomnia severity (Figure 2). Significant main effects of treatment group were observed for BDI-II, $p < .001$, and ISI, $p < .001$, and a significant interaction effect between treat-

Table 1
Participant Demographic Characteristics

	CBT-I	Self-help	*p*
n	21	20	
Age, years (mean ± SD)	37.1 ± 12.8	36.4 ± 13.8	0.95
BMI, kg/m^2 (mean ± SD)	25.6 ± 4.1	26.5 ± 4.7	0.54
Females (%)	61.9	60.0	0.90
Level of education (%)			
High school	33.3	30.0	0.96
Vocational training	4.8	5.0	
Undergraduate degree	47.6	55.0	
Postgraduate degree	14.3	10.0	
Employment status (%)			
Employed	76.1	75.0	0.53
Unemployed	9.5	15.0	
Student	14.3	5.0	
Retired	0	5.0	
Marital status (%)			
Single	38.1	45.0	0.67
Partner	9.5	15.0	
Married	47.6	30.0	
Separated/divorced	4.8	10.0	

Note. BMI = Body Mass Index.

ment group and time was also found for ISI, $p = .01$. Post hoc analyses revealed that, compared with the self-help CBT-I group, the CBT-I group showed significantly lower scores on the ISI at the end of Session 3, $p < .05$, posttreatment, $p < .01$, and follow-up, $p < .001$. Comparing the treatment groups at the posttreatment assessment, large effect sizes were found for both depression (Cohen's $d = 1.24$) and insomnia severity ($d = 0.92$). At the 3-month follow-up, effect sizes were even larger: depression severity ($d = 1.65$) and insomnia severity ($d = 1.87$).

At follow-up, 94% (17/18) of the CBT-I participants achieved reliable insomnia improvements from baseline of at least 4 ISI points, compared with 50% (9/18) of those in the self-help CBT-I group, $p < .005$. Sixty-seven percent (12/18) of those in the CBT-I group had reached clinical remission for their insomnia at follow-up, compared with only 11% (2/18) of those in the self-help CBT-I group, $p = .001$. In both groups, 6% (1/18) showed evidence of reliably worsened insomnia severity, with ISI scores worsening by at least 4 points from baseline, $p = 1.00$.

For depression severity, 94% (17/18) of the CBT-I participants reported reliable reductions of at least 7 BDI-II points from baseline to follow-up compared with 39% (7/18) of the self-help CBT-I group, $p < .001$, and 78% (14/18) of CBT-I participants reached clinical remission compared with 17% (3/18) of self-help CBT-I participants, $p < .001$. None of the participants in the CBT-I group showed reliable worsening of depression symptoms (at least 7 BDI-II points) compared with 6% (1/18) of those in the self-help CBT-I group, $p = .31$.

At the 3-month follow-up, 61% (11/18) of CBT-I participants were in clinical remission from both insomnia and depression, compared with only 6% (1/18) of the self-help CBT-I participants, $p < .001$. This equates to a number needed to treat of 1.8 participants that need to undergo this intervention, for 1 participant to achieve clinical remission from both insomnia and depression.

Mediation Testing

The difference between treatment groups in the magnitude of change in depression severity from baseline to follow-up (Weeks 0–20) was significantly mediated by decreases in insomnia severity (Figure 3). We used Jose's medgraph package (http://pavlov.psyc.vuw.ac.nz/paul-jose/medgraph/instruction.php) to impute values from the three regression analyses to test the direct, indirect, and total effects. Sobel's test at follow-up showed a reduction in depression severity in the CBT-I group of 12.50 (3.20) unstandardized coefficients (95% CI [−18.59, −6.52]), $p < .005$, larger on the BDI-II compared with the change in scores in the self-help CBT-I group. When insomnia severity was included as a mediating variable, the reduction in insomnia severity from baseline to follow-up accounted for .98 (.26) unstandardized coefficients (95% CI [11.77, 1.22]), Sobel's *z* value for the mediation model = 3.52, $p < .0005$. The standardized effect size indicated that the Indirect to Total Ratio was .75 or 75% of the reduction in depression severity, with the direct effect of CBT-I on depression outcome no longer reaching significance ($p = .39$; see standardized regression weights in Figure 3A).

Conversely, the effect of the treatment group on the magnitude of change in insomnia severity from baseline to follow-up was partially mediated by changes in depression severity (Figure 3B).

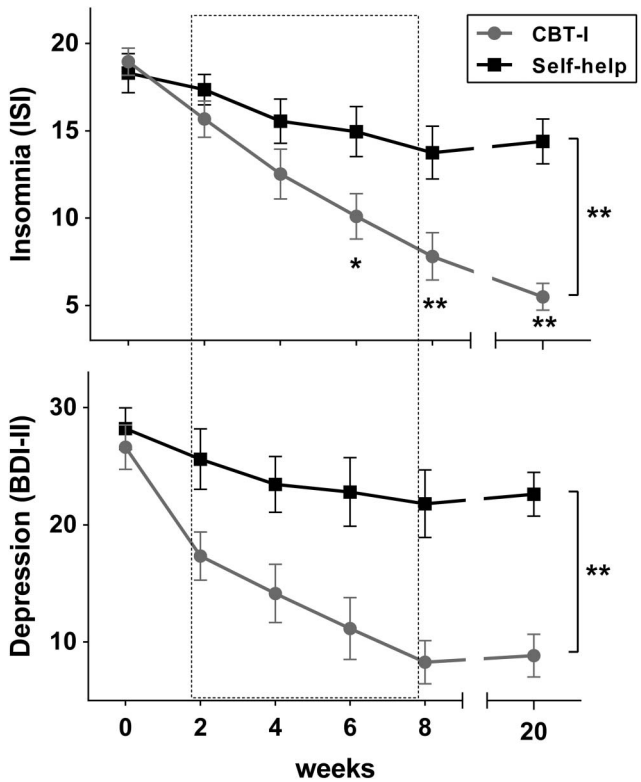

Figure 2. Mean ± SEM group (CBT-I, self-help CBT-I) insomnia severity (ISI) and depression severity (BDI-II) scores at baseline (Week 0), end of Session 1 (Week 2), end of Session 2 (Week 4), end of Session 3 (Week 6), posttreatment (Week 8), and 3-month follow-up (Week 20). Treatment phase is indicated by the dotted lines (Week 2 through Week 8). * $p < .05$. ** $p < .01$ between treatment groups.

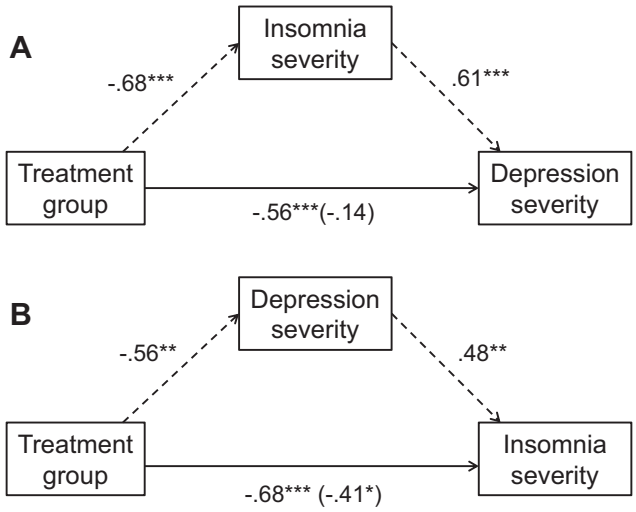

Figure 3. Standardized regression weights for baseline to follow-up change when depression severity was regressed on treatment group and insomnia severity (A). Standardized regression weights for baseline to follow-up change when insomnia severity was regressed on treatment group and depression severity (B). Solid lines show direct effects, dotted lines show indirect effects. Total effects are shown in parentheses (change in direct effect after mediator is included in the model).

Sobel's test, which compared the self-help CBT-I group with a reduction in insomnia severity from baseline to follow-up, was 9.56 points (95% CI [−12.92, −6.13], $p < .001$) greater on the ISI. When depression severity was tested as a mediating variable, reduction in depression severity from baseline to follow-up accounted for .30 points (95% CI [.76, .50], $p < .02$). The standardized effect size indicated that the Indirect to Total Ratio was .40 or 40% of the reduction in insomnia severity, while the direct effect of the CBT-I treatment continued to account for 5.77 points (95% CI [−10.81, −1.23], $p < .05$). (Standardized regression weights in Figure 3).

Sleep Measures

For the subjective (sleep diary) measures, the CBT-I group showed significantly higher sleep efficiency, $p < .001$, and total sleep time, $p < .05$, and significantly shorter sleep onset latency, $p < .005$, compared with the self-help CBT-I group (Figure 4). For the objective (actigraphic) measures, sleep efficiency, $p < .05$, was significantly higher, and sleep onset latency, $p < .001$, was significantly shorter in the CBT-I group compared with the self-help CBT-I group.

Discussion

This study showed that, compared with self-help CBT-I, four sessions of CBT-I delivered by a therapist markedly reduced the severity of depression and insomnia, and improved both subjective and objective sleep quality in individuals with comorbid insomnia and major depression who had not remitted from antidepressant treatment. Effect sizes for improvements in depression and insomnia were large and were found to persist at 3-month follow-up. Remission rate from both insomnia and depression at follow-up was 10 times greater in the CBT-I group compared with the self-help CBT-I group. Reductions in depression severity were mediated by improvements in insomnia at follow-up.

The finding that CBT-I improved insomnia and depression supports previous studies showing improvements in primary insomnia (Morin et al., 1994) and insomnia comorbid with depression (Manber et al., 2008). Previous studies report significant reductions in depression severity through CBT-I (Taylor et al., 2007; Watanabe et al., 2011), although this study is the first to show such reductions relative to an active control treatment for insomnia. The CBT-I treatment in the current study was provided at the optimal frequency and duration of therapeutic sessions, based on a previous study showing that four biweekly sessions of CBT-I was preferable to even eight weekly sessions (Edinger, Wohlgemuth, Radtke, Coffman, & Carney, 2007). Furthermore, because both CBT-I and self-help CBT-I consisted of the same factual information, our study highlights the importance of other therapeutic elements as being essential for optimal insomnia and depression outcomes through CBT-I treatment.

Improvements in insomnia contributed to the observed reductions in depression severity by follow-up, suggesting a directional relationship between insomnia and depression (Franzen & Buysse, 2008). Previous studies have shown that sleep disturbances precipitate depressive relapse (Perlis et al., 1997) and perpetuate or worsen already existing MDD (Staner, 2010). When insomnia is successfully treated alongside antidepressant treatment, the antidepressant effect is hastened (Fava et al., 2006). Treatment of insomnia through CBT-I often results in improvements in psychiatric comorbidities (Smith, Huang, & Manber, 2005). The finding in the present study that significant improvements in insomnia predicted longer-term remission from both conditions provides a justification for targeting insomnia through CBT-I in those with comorbid insomnia and depression, especially in individuals who are yet to remit through antidepressant treatment.

Both subjective (as measured by sleep diary) and objective (as measured by actigraphy) assessments of SE and SOL were improved with CBT-I, demonstrating that the improvements observed in this study are not limited to self-report measures. However, subjective TST improved without a corresponding improvement in objective TST. These findings are not unexpected, as previous studies link insomnia severity with perception of sleep difficulties more than objectively assessed sleep disturbances (Edinger et al., 2000). Individuals with insomnia often underestimate their TST (Harvey, Sharpley, Ree, Stinson, & Clark, 2007). If sleep-state misperception was reduced across CBT-I treatment, it could help explain improvements in subjective but not objective TST.

The CBT-I intervention was compared with self-help CBT-I, an insomnia intervention without the financial and availability issues that often limit access to CBT-I (Perlis, Smith, Cacialli, Nowakowski, & Orff, 2003), particularly in those with mobility and transport difficulties and those in remote or rural settings (Espie, 2009). We found that therapist-led CBT-I was more efficacious than self-help CBT-I. This is consistent with findings from a meta-analysis of self-help treatments for insomnia, which concluded that once publication bias was controlled, self-help interventions did not significantly improve sleep outcomes (van Straten & Cuijpers, 2009). However, a more recent study has shown that

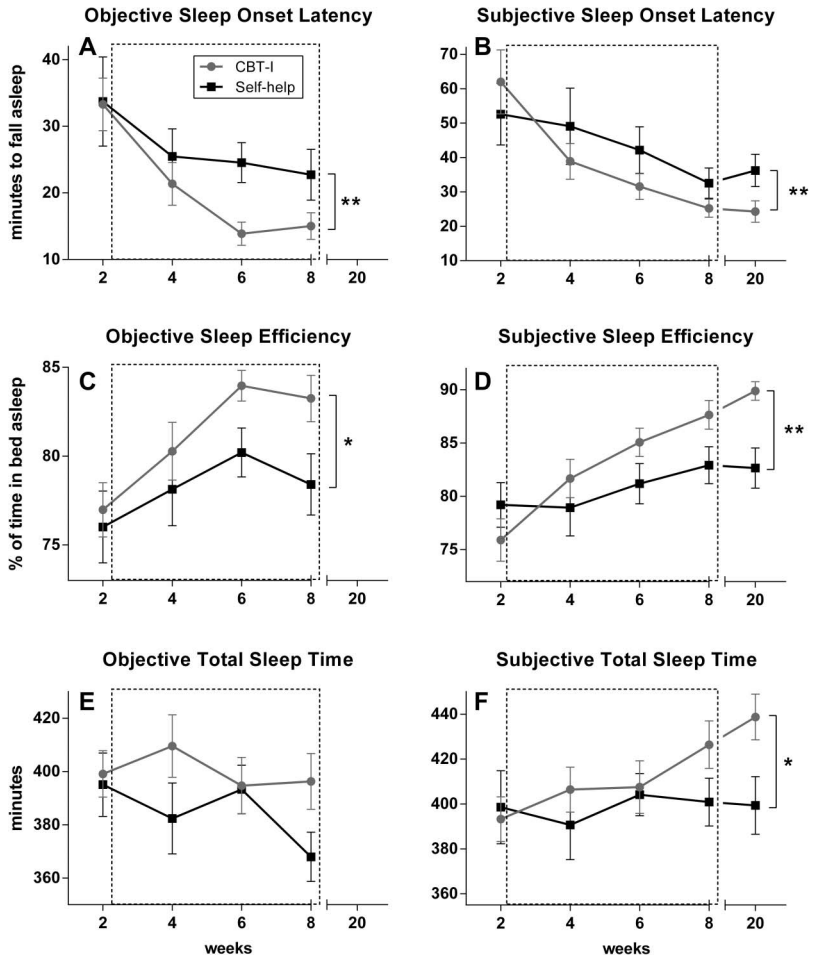

Figure 4. Mean ± SEM group (CBT-I, self-help CBT-I) objective (derived from actigraphy data) and subjective (derived from sleep diary data) SOL, SE, and TST at baseline (Weeks 1 and 2), after Session 1 (Weeks 3 and 4), after Session 2 (Weeks 5 and 6), and after Session 3 (Weeks 7 and 8). Treatment phase is indicated by the dotted lines (Week 2 through Week 8). * $p < .05$. ** $p < .01$ between groups.

self-help CBT-I could produce significant insomnia and depression improvements (Lancee et al., 2013).

There are several possible reasons why CBT-I was superior to self-help CBT-I in the current study. Self-help treatments for insomnia are reported to be less effective when comorbidities are present (van Straten & Cuijpers, 2009). Depression has been found to lower adherence to CBT-I behavioral interventions and reduce the likelihood of changes in expectations about sleep across the treatment (Manber et al., 2011). Some form of support and therapeutic alliance seem necessary for optimal benefits through CBT-I, especially for individuals with depression (Lancee et al., 2013). This is because a positive therapeutic alliance can improve adherence to CBT-I interventions (Trockel et al., 2014). Furthermore, nonspecific factors of psychological treatment, including expectancy of treatment effects, therapeutic alliance, and life circumstances, personal resources, or readiness to change in the participant, typically account for a much larger percentage (up to 85%) of overall outcome variance in comparison with the specific factors of a model of treatment (as little as 15% of outcome variance; Hubble & Miller, 2004). Therefore individualized guidance, feedback, support, and a sense of alliance seem crucial to an optimally effective CBT-I intervention.

If nonspecific psychological treatment factors could be successfully incorporated into a self-help CBT-I intervention, it may be possible to produce similar results to the CBT-I findings in the current study. Preliminary evidence suggests that Sleep Healthy Using the Internet (SHUTi), an interactive Internet-delivered CBT-I program, is more effective in reducing insomnia severity than typical self-help CBT-I interventions (Ritterband et al., 2009), and also improves depression, anxiety, and fatigue (Thorndike et al., 2013). However, it will need to be further assessed in individuals who have been diagnosed with comorbid insomnia and depression to determine its effectiveness in comparison with CBT-I.

Limitations of the study are noted. Although significant effects and large effect sizes were observed, the sample size is modest, and generalizability of findings should be tested in a larger study. Many of the outcomes assessed in the study were self-report, which rely on individuals' perceptions of their symptom severity. While perceived improvement in symptom severity is clinically important, cognitive bias may influence such measures (Kahne-

man, 2003). However, improvements in sleep outcomes were confirmed using an objective measure, and are thus less likely to be explained by cognitive or perceptual bias. Actigraphy data was not collected at follow-up. Future studies should supplement self-report depression measures with clinician assessments of depression severity. The nature of the intervention was such that participants could not be blinded. This may have contributed to larger expectancy effects, which should be assessed and controlled for in future studies. The self-help intervention was described to participants as a comparative treatment rather than a control condition to reduce expectancy effects. Although the CBT-I participants were provided additional feedback and support, the same frequency of face-to-face contact with the researcher was provided and self-help CBT-I participants were also given all written materials included in the CBT-I intervention. Future studies might also benefit from examining the impact of training in the CBT-I model and therapist-specific attributes and skills, which we could not examine as our study used a single therapist for the intervention. Although the intervention was semistructured and manualized, which reduces the need for extensive training, individual differences in implementation of the intervention may still occur. Finally, the CBT-I elements could not be separated from nonspecific therapeutic elements. To unequivocally attribute the efficacy to CBT-I, a future study may consider comparing CBT-I with another brief therapy consisting of nonspecific therapeutic elements without the specific CBT-I interventions. However, unless the perpetuating factors of insomnia are specifically targeted through psychological treatment, it is unlikely that sleep complaints will improve (Carney, Harris, Freedman, & Segal, 2011).

Depression and sleep disturbance are the first and third most common psychological reasons, respectively, for patient encounters in general practice (Britt et al., 2010). The substantial burden associated with these conditions, and their high degree of comorbidity calls for a coordinated approach to the management of insomnia and depression and widespread implementation of effective treatments. This study shows that the CBT-I intervention was very effective, with 1 participant achieving remission of both insomnia and depression for every 1.8 participants. These study findings can be readily translated into clinical practice because the CBT-I treatment was developed for psychologists to use without the need for additional sleep disorders training. Recent evidence suggests that manual-guided CBT-I could also be effectively administered by primary care personnel (Bothelius, Kyhle, Espie, & Broman, 2013). Developing a stepped-care model would enable wider dissemination (Espie, 2009), which is essential for highly prevalent disorders such as depression and insomnia.

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Received May 18, 2014
Revision received December 3, 2014
Accepted December 4, 2014 ■