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Comparative Efficacy of Behavior Therapy, Cognitive Therapy and Cognitive Behavior Therapy for Chronic Insomnia: A Randomized Controlled Trial

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Abstract

Objective—To examine the unique contribution of behavior therapy (BT) and cognitive therapy (CT) relative to the full cognitive behavior therapy (CBT) for persistent insomnia.

Method—Participants were 188 adults (117 women; *M* age = 47.4 years old, *SD*=12.6) with persistent insomnia (average of 14.5 years duration). They were randomized to eight, weekly, individual sessions consisting of BT (*n* = 63), CT (*n* = 65), or CBT (*n* = 60).

Results—Full CBT was associated with greatest improvements, the improvements associated with BT were faster but not as sustained and the improvements associated with CT were slower and sustained. The proportion of treatment responders was significantly higher in the CBT (67.3%) and BT (67.4%) relative to CT (42.4%) groups at post treatment, while 6-months later CT made significant further gains (62.3%), BT had significant loss (44.4%) and CBT retained its initial response (67.6%). Remission rates followed a similar trajectory, with higher remission rates at post treatment in CBT (57.3%) relative to CT (30.8%), with BT falling in between (39.4%); CT made further gains from post treatment to follow up (30.9% to 51.6%). All three therapies produced improvements of daytime functioning at both post treatment and follow up, with few differential changes across groups.

Conclusions—Full CBT is the treatment of choice. Both BT and CT are effective, with a more rapid effect for BT and a delayed action for CT. These different trajectories of changes provide unique insights into the process of behavior change via behavioral versus cognitive routes.

Keywords

sleep; insomnia; behavior therapy; cognitive therapy; CBT; behavior change

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Insomnia is among the most frequent complaints brought to the attention of health-care practitioners and is the most prevalent of all sleep disorders in the general population (Ohayon & Reynolds, 2009). Relative to good sleepers, individuals with insomnia report more psychological distress, more impairments of daytime functioning and accidents, take more frequent sick leave and utilize more health care resources (Daley et al., 2009; Sivertsen, Øverland, Bjorvatn, Mæland, & Mykletunb, 2009). Moreover, insomnia heightens the risk of developing subsequent depression, anxiety, and substance-related problems (Baglioni et al., 2011; Breslau, Roth, Rosenthal, & Andreski, 1996).

Despite its high prevalence and negative impact, insomnia often goes unrecognized and remains untreated. Most individuals with insomnia who initiate treatment do so without professional consultation and often resort to self-help remedies (e.g., alcohol, over-the-counter drugs) of limited benefit and questionable safety (Morin & Benca, 2012). When insomnia is brought to professional attention, typically to a primary care physician, treatment is usually limited to pharmacotherapy. Hypnotic medications are effective for the short-term management of insomnia but there is limited evidence about sustained efficacy with long-term use (Krystal, 2009). Recognition that psychological factors play an important role in maintaining sleep disturbances has led to increased interest in the use of a cognitive behavior therapy for insomnia (CBT). CBT targets maladaptive sleep habits and irregular sleep-wake schedules, unhelpful beliefs about sleep, sleep-related worry and attentional bias and hyperarousal (Buysse, Germain, Hall, Monk, & Nofzinger, 2011; Harvey, 2002; Lundh & Broman, 2000; Morin & Espie, 2003; Spielman & Glovinsky, 1991). There is a large body of evidence regarding the efficacy of CBT (e.g., Morin et al., 2006; Morin, Culbert, & Schwartz, 1994; Smith et al., 2002) and clinical benefits are well sustained over time (Morin, Colecchi, Stone, Sood, & Brink, 1999). Despite positive outcomes for the majority of patients, not everyone achieves full remission, and patients often continue experiencing residual sleep disturbances after treatment (Buysse, 2013; Espie, Inglis, & Harvey, 2001; Harvey & Tang, 2003; Morin, et al., 1994). In addition, there are two gaps in knowledge that the present study was designed to address.

First, the efficacy of behavior therapy (BT) components of CBT, which usually includes stimulus control and sleep restriction, is well established (Morin, et al., 2006). While cognitive therapy (CT) is typically incorporated within CBT programs, its unique contribution is not yet known (Morin, et al., 2006). There is initial evidence pointing to the potential importance of CT in the management of insomnia. Two research teams have reported that change to dysfunctional beliefs about sleep predicts a better outcome (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Morin, Blais, & Savard, 2002). Moreover, one open trial of CT for chronic insomnia yielded promising results (Harvey, Sharpley, Ree, Stinson, & Clark, 2007). However, as there was no control group, we cannot rule out the possibility that the improvement was due to the passage of time or to non-specific therapy effects (e.g., expectation of improvement).

Second, daytime impairment is an essential feature of the diagnosis of insomnia (American Academy of Sleep Medicine, 2005; American Psychiatric Association, 2013; Edinger et al., 2004). Yet, the vast majority of the research, theory, and treatment evidence focuses on night time symptoms and processes (Riedel & Lichstein, 2000). Moreover, there is very

limited evidence that insomnia treatment improves daytime functioning, psychological well-being, and quality of life (NIH, 2005). This is an important gap in knowledge given that it has typically been assumed that treatments for insomnia that address sleep will also effectively address daytime impairment but the limited data currently available are equivocal. To date, one study of relaxation therapy improved sleep but noted modest or no effects of this insomnia treatment on daytime outcomes (Means, Lichstein, Epperson, & Johnson, 2000). However, moderate to large effect sizes on daytime functioning outcomes were reported following four weeks of sleep restriction on the Daytime Functioning and Sleep Attribution Scale, Glasgow Sleep Impact Index, Occupational Impact of Sleep Questionnaire as well as on three domains of the SF-36 (Kyle, Morgan, Spiegelhalder, & Espie, 2011). Other studies have highlighted that the nighttime and daytime aspects of insomnia may be functionally independent (Lichstein, Durrence, Riedel, & Bayen, 2001; Neitzert Semler & Harvey, 2005). Hence, perhaps the daytime aspects of insomnia will require specific treatment.

The present study was designed to establish the comparative efficacy of BT and CT, relative to their combination (CBT) and to evaluate their effects on nighttime *and* daytime outcomes. Based on the exclusive focus of BT on sleep-related behaviors and scheduling factors, we hypothesized that the BT group would exhibit greater sleep improvement, relative to the CT group for sleep/nighttime measures. Conversely, as CT targets *both* nighttime sleep disturbance *and* daytime impairment, but not directly sleep-wake behaviors and scheduling factors, we hypothesized that CT will be more potent, relative to BT in reducing daytime functional impairment. Another aim was to evaluate the effects of treatment on day and nighttime functioning from post to 6 month follow-up. It was expected that all three treatment arms would produce improvements at the end of treatment that would be sustained at the 6 month follow-up.

Method

Participants

Patients were recruited from March, 2008 to November, 2011 through advertisements and referrals from health care practitioners. Participants were recruited from two sites: Laval University in Quebec City, Canada and University of California, Berkeley. A telephone interview was completed to initially screen for eligibility. Eligible individuals were then invited to participate in an extensive diagnostic interview session.

Inclusion criteria were: (a) 25 years of age or older and (b) meeting criteria for persistent insomnia: (i) difficulty initiating and/or maintaining sleep, defined as a sleep onset latency and/or wake after sleep onset greater or equal to 30 min, with a corresponding sleep time of less than or equal to 6.5 hours per night, as ascertained by daily sleep diaries kept for a two-week baseline period; (ii) presence of insomnia more than 3 nights per week and for more than 6 months; (iii) the sleep disturbance (or associated daytime fatigue) causes significant distress or impairment in social, occupational, or other areas of functioning as measured by a rating of at least 2 on item no. 5 or 7 on the Insomnia Severity Index (Morin, 1993). This definition represents a combination of the Research Diagnostic Criteria (Edinger, et al., 2004), the International Classification of Sleep Disorders' criteria (ICSD; American

Academy of Sleep Medicine, 2005) and the Diagnostic and Statistical Manual of Mental Disorders' criteria (DSM-IV-TR; American Psychiatric Association, 2013) along with quantitative cutoffs typically used in insomnia research.

Exclusion criteria were: (a) presence of a progressive or unstable physical illness (e.g., cancer, acute pain) or neurological degenerative disease (e.g., dementia) directly related to the onset and course of insomnia, (b) use of hypnotics and other medications known to alter sleep (e.g., steroids, anxiolytics) (patients on SSRI for at least 3 months were included), (c) evidence of sleep apnea (apnea/hypopnea index > 15), restless legs or periodic limb movements during sleep (PLMS with arousal > 15 per hour), or a circadian-based sleep disorder (e.g., delayed or advanced sleep phase syndrome); or body mass index (BMI) of 35 or above, or BMI of 32 or above and reporting at least 3 symptoms of breathing-related sleep disorder on the Duke Structured Interview for Sleep Disorders (Edinger et al., 2009), (d) irregular sleep schedules, with usual bedtimes earlier than 9:00pm or later than 2:00am or rising time earlier than 5:00am or later than 10:00am, occurring more than twice/week or working on night or rotating shifts within the last year, (e) current or past psychological treatment of insomnia within the past 5 years, (f) individuals consuming more than two alcoholic beverages or more than four caffeinated beverages per day were required to reduce their intake below or equal to two and four respectively for the duration of the study or be excluded from the study, (g) a lifetime diagnosis of a psychotic or bipolar disorder or more than two lifetime episodes of major depressive disorder or an untreated current major depressive disorder or alcohol or drug abuse within the past year. When other comorbidities were present, we ensured that insomnia was the disorder currently most distressing and disabling (Di Nardo et al., 1993) or that participant were still suffering significant insomnia despite receiving treatment for the comorbid condition (e.g., major depression). Of the total 188 patients, 45 (23.9%) had at least one current comorbid Axis I disorder (ranging from 1 to 4 diagnoses, $M = 1.4$). Most frequent comorbid disorders were generalized anxiety disorder ($n = 18$), specific phobia ($n = 10$), adjustment disorder ($n = 5$), dysthymia ($n = 4$), obsessive-compulsive disorder ($n = 4$), social phobia ($n = 3$), panic disorder ($n = 3$), and major depression disorder ($n = 3$). Of the total sample, 35.1% had used a prescribed hypnotic medication and 18.6% had used an over the counter product for sleep in the last month before the study.

Study Design

A total of 188 adults with persistent insomnia were randomly assigned to one of three groups: (a) behavior therapy (BT; $n = 63$), (b) cognitive therapy (CT; $n = 65$), or (c) cognitive-behavior therapy (CBT; $n = 60$). Randomization was stratified by age (25–49 versus 50+) and presence of a comorbid psychiatric disorder (absence vs. depression, dysthymia, generalized anxiety disorder, social phobia, panic disorder, adjustment disorders). Group allocation concealment was achieved by sequentially numbered, opaque, sealed envelopes opened by the project coordinator at each study site. Treatment lasted 8 weeks for all three groups. Outcome measurements were taken at baseline (Time 1), at the end of treatment (Time 2), and at 6-month follow-up (Time 3). Figure 1 summarizes the flow through the study. All participants provided written informed consent and received financial compensation to cover their travel expenses.

Assessment Measures

Diagnostic Measures

Structured Clinical Interview for DSM-IV: (SCID; First, Spitzer, Gibbon, & Williams, 1995) is a semi-structured interview designed to assess *DSM-IV-TR* diagnostic criteria for Axis I disorders. The SCID has good reliability. Trained psychology doctoral students and postdoctoral fellows administered the SCID to assess current and lifetime Axis I disorders.

Duke Structured Interview for Sleep Disorders: (DSISD; Edinger, et al., 2004) is a semi-structured interview that assesses research diagnostic criteria for sleep disorders. The DSISD has good reliability and validity (Edinger, et al., 2009).

Sleep Measures

Insomnia Severity Index: (ISI; Bastien, Vallieres, & Morin, 2001; Morin, Belleville, Bélanger, & Ivers, 2011) is a 7-item scale assessing nighttime (difficulties falling asleep, staying asleep, early morning awakenings) and daytime variables (satisfaction with sleep, degree of impairment with daytime functioning, noticeability of impairments, distress or concern with sleep). Each item is rated on a 5-point scale and the total score ranges from 0 to 28. The ISI has adequate internal consistency (Cronbach's alpha =.91) and temporal stability (r = .80), and is sensitive to therapeutic changes (Morin et al., 2004; Morin, Beaulieu-Bonneau, LeBlanc, & Savard, 2005; Morin et al., 2009). The following interpretation guidelines are recommended: score of 0–7 (no clinical insomnia), 8–14 (sub threshold insomnia), 15–21 (insomnia of moderate severity), and 22–28 (severe insomnia). The total score, as well as rates of treatment responders (defined as achieving a change of 8 points or more) and remitters (defined as a final score below 8) were the primary outcome measures for this study.

Sleep Diary: Participants kept daily sleep diaries during a 2-week baseline period, the 8-week treatment phase, and for 2 weeks at the post-treatment and 6-month follow up assessments. The primary dependent variables derived from the diaries were: sleep onset latency, wake time after sleep onset, total sleep time, time in bed, and sleep efficiency (dividing total sleep time by time in bed and multiplying this value by 100). The sleep diary has been shown to be a reliable estimate and is considered the gold standard subjective measure of sleep (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006).

Polysomnography (PSG): All participants underwent a total of 5 nights of evaluation in the sleep laboratory, including 1 screening/adaptation night, 2 baseline nights and 2 nights after the end of treatment. Bedtime and arising time in the sleep laboratory were kept as close as possible (i.e., within 30 min) to the participant's habitual sleep schedule at home (as determined by sleep logs kept during the two weeks preceding recording). Participants were allowed the same amount of time in bed during the PSG before versus after the treatment phase. Also, we did not encourage patients to get out of bed if unable to sleep on the PSG nights. A standard montage including electroencephalographic, electromyographic (EMG), and electro-oculographic monitoring was used (Rechtschaffen & Kales, 1968). Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis EMG was also monitored during the first (screening) night to evaluate sleep apnea and periodic limb movements

during sleep. All recordings were scored by experienced technicians, blind to participants' condition, and according to standardized criteria (Rechtschaffen & Kales, 1968). The primary dependent variables were sleep onset latency (time from lights out to persistent sleep), wake after sleep onset (time awake from initial sleep onset until last awakening), total sleep time, time in bed and sleep efficiency. These variables were averaged over two nights for each assessment phase. Persistent sleep was defined as 10 consecutive epochs or the first 5 min of any stage of sleep (Kushida et al., 2005). To reduce the potential for variability in scoring, all PSGs were scored at the Laval site.

Daytime Measures

Multidimensional Fatigue Inventory: (MFI; Smets, Garssen, Bonke, & De Haes, 1995) is a 20-item measure with five factors assessing general fatigue, physical fatigue, mental fatigue, reduced motivation and reduced activity. Total score ranged from 20 to 100. The MFI has good internal consistency and established construct and convergent validity.

Work and Social Adjustment Scale: (WSAS; Mundt, Marks, Shear, & Greist, 2002) assessed functioning across work, home management, social leisure activities, private leisure activities and relationships with others. Total score of 5 items ranged from 0 to 10. The psychometric properties are adequate.

SF-36 Health Survey: (SF-36; Ware & Sherbourne, 1992), Version 2 (Jenkinson, Stewart-Brown, Petersen, & Paice, 1999) is a self-rated measure of functioning, health status, and well-being. Eight subscales (Physical functioning, Role-Physical, Bodily Pain, General Health, Vitality, Social Functioning, Role-Emotional and Mental Health) aggregate two to ten items each and two summary measures (Physical Component Scale and Mental Component Scale) aggregate the subscales. Only T scores ($M = 50$, $SD = 10$) for the summary measures are reported. Reliability estimates of the different scales vary between . 76 and .90. The SF-36 has been validated against numerous other health questionnaires (Ware & Sherbourne, 1992).

The rationale for selecting these daytime measures was: (a) the MFI and SF-36 were recommended by Buysse et al. (2006) for the standard research assessment of insomnia and (b) the WSAS was selected as a short, well validated assessement of very specific domains of life functioning, which are not covered by the MFI or SF-36.

Credibility, Expectancy Measures

Credibility/Expectancy Questionnaire: (CEQ; Devilly & Borkovec, 2000) was administered at the end of the first therapy session. After receiving a description of the treatment procedures and their rationale, participants provided ratings (1–5 point scale) of treatment acceptability, treatment plausibility, and expectancies for success. This questionnaire has demonstrated high internal consistency (standardized alpha = .84–.85) and good test-retest reliability over one week (0.83; Devilly & Spence, 1999).

Treatments

Treatments were provided in the context of eight weekly individual therapy sessions, with BT and CT sessions lasting 45–60 minutes and CBT sessions lasting 75 minutes long. Therapy sessions followed a structured agenda including: (a) review of sleep diary data, (b) discussion/implementation of clinical procedures and rationale, (c) compliance issues and problem-solving and (d) homework assignments. Treatment elements in common across all three arms were: providing a generic overview of the CBT approach and the 'Self-Management Approach' in which the patient assumes an active role in his/her treatment, keeping a sleep diary, introducing the 3 P Model of Insomnia (Spielman, Caruso, & Glovinsky, 1987), setting treatment goals, reviewing sleep hygiene information and progress and goal attainment. The week-by-week content of sessions is presented in an online supplement.

Behavior Therapy—(BT) included a combination of stimulus control and sleep restriction procedures. Stimulus control (Bootzin, 1979) is derived from the proposal that conditioning has occurred between temporal and environmental stimuli (the bed, bedroom, bedtime) normally conducive to sleep and sleep incompatible behaviors (e.g. worry/frustration at not being able to sleep), such that the bed, bedroom and bedtime are no longer discriminative stimuli for sleep. The intervention aims to reverse this maladaptive association by limiting the sleep incompatible behaviors engaged in within the bedroom environment thereby decreasing cues for sleep incompatible behaviors while increasing cues for sleep compatible behaviors. This intervention involves the therapist providing a detailed rationale for and assisting the patient to achieve the following: (a) go to bed only when sleepy at night; (b) use the bed and bedroom only for sleep and sex (i.e. no reading, TV watching, or worrying either during the day or at night); (c) get out of bed and go to another room whenever you are unable to fall asleep or return to sleep within 15–20 minutes and return to bed only when sleepy again; (d) repeat this last step as often as necessary throughout the night; (e) arise in the morning at the same time regardless of the amount of sleep obtained on the previous night (Bootzin, 1972; Bootzin, Epstein, & Wood, 1991). Limited daytime napping (< 1 hour) before 03:00 p.m. was made optional early in the treatment.

The second component of BT, sleep restriction (Spielman, Saskin, & Thorpy, 1987), is derived from the proposal that excessive time in bed perpetuates insomnia. The intervention involved curtailing time in bed to the actual time slept and gradually increasing it back to an optimal sleep time. Based on sleep diary data, each patient was prescribed a specific amount of time in bed (sleep window) not to be exceeded. The duration of this sleep window was reviewed weekly and increased or decreased contingent upon the sleep efficiency for the previous week. The goal was to maximize sleep efficiency [(total sleep time divided by time in bed) x 100] to more than 85%. A lower limit of 5 hours was set on the time in bed recommendation.

Cognitive Therapy—(CT) was first described by Aaron T. Beck and colleagues (Beck, 1979; Beck, Emery, & Greenberg, 1985). The CT approach used was an enhanced program, relative to that included in most previous trials of CBT for insomnia. First, based on accruing evidence for cognitive maintaining processes, CT sought to reverse a broader range

of cognitive maintaining mechanisms; namely, (1) unhelpful beliefs about sleep (Morin, et al., 2002), (2) sleep-related or sleep-interfering worry (Tang & Harvey, 2004), (3) attentional bias and monitoring for sleep-related threat (Neitzert Semler & Harvey, 2005), and (4) misperception of sleep (Harvey & Tang, 2012). These treatment approaches are described elsewhere (Harvey, et al., 2007; Morin, 1993; Perlis, Aloia, & Kuhn, 2011). Second, the therapy time and homework assignments were equally split between working on reversing these cognitive maintaining mechanisms during the daytime and the nighttime (Harvey, 2002). Third, CT included individually formulated experiments to test beliefs. A minimum of four experiments were conducted across the 8 sessions: a monitoring/ attentional bias experiment, the sleep survey experiment, the energy generating experiment and the fear of poor sleep experiment (Harvey, et al., 2007; Perlis, et al., 2011; Ree & Harvey, 2004).

Cognitive-Behavior Therapy—*(CBT)* consisted of a combination of both the BT and CT components delivered in an integrated fashion. A case formulation driven approach (Harvey, 2006; Persons, 2006) was used to determine the relative time and ordering of CT vs. BT. The formulation was guided by the symptoms that were present and the approach that elicited the most optimal response from the patient. For CBT to truly combine, and cover all elements of CT and BT, we elected to devote more time to CBT.

Therapists: All treatments were administered by licensed clinical psychologists (n = 39 patients) or advanced graduate students in clinical psychology (*n* = 149 patients) who had completed all of their clinical training requirements. Therapists had attended joint training workshops with the study principal investigators (A. Harvey and C. Morin). Treatment manuals were also available to therapists and ongoing joint supervision from both study sites were provided during the course of the study.

Treatment integrity and contamination was carefully managed via three strategies. First, two multi-day therapist training workshops were conducted. One prior to the beginning of the study. The second after the first year of data collection. Both workshops involved a specific focus on promoting adherence and on delivering each individual treatment with a high level of fidelity. In addition, sessions within these workshops focused on identifying specific methods to avoid contamination across the three treatments. Specifically, the therapists were instructed to gently disengage the patient's attention from a question or tangent not allowed within the intervention being delivered and redirect attention back to the allowable session content. Second, fidelity and contamination were major topics within the weekly supervision sessions. One hour per week was devoted to a conference call involving therapists and supervisors across both sites. Additional site-specific supervision sessions were also provided. In sum, if there was even the smallest doubt that a given intervention may involve contamination the issue was addressed quickly.

Data Management and Analyses

All data were double-entered in an Access data warehouse (one per site) and missing or aberrant data were verified for maximal integrity of the database. Sleep diary and PSG data

were computed as nightly means averaged over the two-week (diary) or two-night (PSG) periods for each assessment phase.

Analyses for the main hypotheses were performed using an intent-to-treat approach, such that all randomized participants were included in the analyses. No data imputation was performed. Site and stratification variables (age and comorbidity) were included in all main analyses as fixed effects (Chow & Liu, 1998).

To study changes on sleep and daytime variables within and between conditions, 3 (Groups) X 3 (Time: Pre, Post, 6-month followup) split-plot mixed model analyses were computed to test Group, Time, and Interaction effects. Linear mixed model analysis was preferred to least-squares ANOVA with the last-observation carried forward approach, since the former analysis ensures an unbiased intent-to-treat approach to deal with attrition. Empirical ("sandwich") estimates of the standard errors of fixed effects were computed since they are typically more robust to small sample size, non-normality and mispecification of the variance-covariance matrix. Group X Time interactions (significant or not) were decomposed using simple effects in order to compare pre to post changes associated with each treatment condition, as well as averaged change scores between conditions. Following APA recommendations to avoid conclusions based strictly on statistical significance testing (p-values) (Wilkinson, 1999), effect sizes for pre to post (temporal) changes were computed as the difference between means, divided by the root mean squared error (RMSE) of the mixed model. Raw *p*-values for all simple effects are reported in tables but the multiplicity problem was addressed by computing adjusted *p*-values using the Hochberg and Benjamini (1990) adaptive step-down Bonferroni method. All analyses were performed using SAS 9.3 (SAS Institute Inc., 2011) using standard two-tailed 5% alpha level unless otherwise specified.

Results

Sample and Treatment Attendance

There were no significant differences between groups at baseline on demographic variables, medical or psychological comorbidity, insomnia duration, and baseline insomnia severity (ISI) (see Table 1).

Figure 1 summarizes the flow of participants through the study. The overall attrition rate was 7.5% (14/188) during treatment and 10.6% (20/188) at the 6-month followup. Attrition was not significantly different across treatment groups at posttreatment (CBT = 3.3%, CT = 9.2%, BT = 9.5%, $p = .37$) or at 6-month followup (CBT = 5.0%, CT = 13.9%, BT = 12.7%, $p = .25$). There was only one significant difference between treatment completers and those who dropped during treatment, with dropouts reporting longer insomnia duration (22.2 yrs vs. 13.9 yrs), $t(186) = 2.37$, $p = .02$.

Treatment attendance and credibility

All three conditions were rated as highly acceptable and credible and generated high expectancies for success. There were no significant group differences for total CEQ (BT $M = 73.9$; $SD = 12.5$; CT $M = 72.9$; $SD = 15.3$; CBT $M = 75.8$; $SD = 16.2$; all $p > .05$). Overall,

94.2% of the patients attended the planned number of eight therapy sessions (M = 7.8, range = 1 to 8) and this high attendance was not different across conditions.

Power Analysis

Sensitivity analyses using G*Power 3.1.7 revealed that a total sample of 188 subjects, with 3 assessments and an observed overall dropout rate of 6% (df error for group x time interaction = 323), allow the detection of a very small effect size (f = 0.086) under standard power conditions (80% power, two-tailed alpha 5%). According to Cohen (1992), a small ES in ANOVA is f = 0.10, a moderate ES = 0.25 and a large ES = 0.40. Thus, the study appears to be appropriately powered to detect even very small effects.

Insomnia Severity Index (ISI)

ISI adjusted means across groups and time are displayed in Table 2, as are the percentage of treatment responders and remitters based on ISI scores. For the total ISI score, a significant time effect was observed, $F(2,323) = 347.74$, $p < .001$, indicating that all three groups reduced their insomnia symptoms over time, but the group x time interaction failed to reach significance, $F(4,323) = 2.05$, $p = .09$. Simple effects showed that pre to post-treatment ISI reductions among CBT patients ($M = -10.6$ units, $d = -2.50$) were significantly higher than those for CT patients ($M = -8.2$ units, $d = -1.94$) but not those for BT patients ($M = -9.3$ units, $d = -2.21$), $F(2,323) = 3.39$, $p = .03$.

Percentage of treatment responders were significantly different across conditions at post-treatment compared to FU-6 month (interaction), $F(2,153) = 10.07$, $p < .001$. Simple effects indicated that there were significantly more treatment responders at post-treatment among the CBT (67.3%) and BT groups (67.4%) compared to the CT group (42.2%), $F(2,153) = 4.84$, $p = .009$, but these differences failed to reach significance at FU-6, $F(2,153) = 2.94$, $p = .06$. There was a significant increase in the percentage of treatment responders from post-treatment to FU-6 in the CT group (+20.2%), with a similar decrease among the BT group (−22.9%), suggesting a late response for CT patients and a loss of benefits for BT at FU-6. To understand this pattern of results we calculated odds ratios for pair-wise comparisons by time. At post-treatment, CBT patients were OR = 2.79 times more likely to show a treatment response compared to CT patients (67.3% vs 42.4%, p = .01), and BT patients were OR = 2.80 times more likely to show the same response (67.4% vs 42.4%, p = .009). At 6-month follow-up, CBT patients were OR = 2.60 times more likely to show treatment response compared to BT patients (67.6% vs 44.4%, p = .02), and CT patients were OR = 2.09 times more likely to show the same response (62.6% vs 44.4%, p = .07). Thus, it appears that the size of the difference between BT and CT at FU6 is indeed smaller that the similar effect size at post-treatment.

Remission rates followed a similar trajectory for CBT and CT, with a significant group x time interaction, $F(2,153) = 3.86$, $p = .02$. In this case, there was a larger proportion of patients who remitted at post-treatment in the CBT group (57.3%) compared to CT (30.8%), $F(2,153) = 3.81$, $p = .02$, but these differences failed to reach significance at FU-6, $F(2,153) = 2.21$, $p = .11$. There was a significant increase in the proportion of CT patients who went

into remission from post-treatment to FU-6 (+20.8%), while remission rates did not change significantly in the BT group (−2.9%) or CBT group (−1.5%) for the same period.

Sleep Diary Variables

Overall (all conditions and assessments), participants completed an average of 12.3 (SD = 4.0) out of 14 scheduled days of diary at each assessment period and no significant differences were found between conditions. However, average number of days with available diary was significantly lower at post-treatment (M = 11.3) compared to FU6 (M = 12.2) or baseline (M = 13.3), p < .001. Perfect compliance (14/14 days for a specific assessment phase) was found for 72.0% of the assessments (all groups and time confounded). No significant differences were found between conditions (CBT = 67.6%, CT = 70.2%, BT = 78.2%, p=.08) but compliance at post-treatment was lower (overall 56.4%) compared to baseline (82.5%) and FU6 (76.4%), p<.001.

Adjusted means across groups and time for the sleep diary variables are displayed in Table 3. Significant group x time interactions were obtained for both sleep onset latency (SOL) and wake after sleep onset (WASO), F(4,289) = 3.51, *p*=.008 and 3.13, *p* = .02, respectively. BT patients reported a higher pre to post treatment SOL reduction (*M* = −19.1 min, *d* = −0.86) compared to CT patients (*M* = −9.3 min, *d* = −0.42) but not CBT patients (*M* = −14.0 min, *d* = −0.63), *F*(2,289) = 4.52, *p* = .01; and a higher WASO reduction (*M* = −38.4 min, *d* = −1.29) compared to CT (*M* = −20.4 min, *d* = −0.68) but not CBT patients (*M* = −29.1 min, *d* = −0.98), *F*(2,289) = 5.99, *p* < .001.

Total sleep time increased significantly from baseline to post-treatment and to FU-6 in all three groups (changes of 34 min in CT, 45 min in CBT and 49 min in BT), *F*(2,289) = 54.26, *p* < .001, but no significant differences were found between groups for any assessment times.

Sleep efficiency increased significantly over time, *F*(2,289) = 177.54, *p* < .001, but these improvements varied across groups as suggested by a significant group x time interaction, *F*(4,289) = 6.29, *p* < .001. BT and CBT patients showed higher pre to post-treatment sleep efficiency increases (*M* = 16.9%, *d* = 1.53, and 13.7%, *d* = 1.24, respectively) compared to CT patients (*M* = 8.6%, *d* = 0.78), *F*(2,289) = 12.47, *p* < .001. Compared to baseline, these changes were still significant at 6-month followup but the difference between BT (pre to FU-6 = 13.5%) and CT (pre to FU-6 = 7.2%) was no longer significant after correction for multiplicity (*p* = .06).

Polysomnography

Adjusted means across groups and time (pre and post only) for polysomnographic variables are displayed in Table 4. For sleep onset latency, no significant interaction was observed (*p* = .13), only a significant overall reduction similar across groups, *F*(1,165) = 7.93, *p* = .005. Simple effects revealed lower sleep latencies among CBT and BT patients (*M* = 9.5 min and 12.4 min, respectively, *p* < .001) relative to CT (*M* = 17.9 min) at post-treatment. A significant interaction was found for WASO, *F*(2,165) = 4.41, *p* = .01, with BT patients showing higher pre to post-treatment reductions (*M* = −13.5 min, *d* = −0.46) compared to

CT patients (M = 2.1 min, d = 0.07) or CBT patients (M = −4.0 min, d = −0.13), $F(2,165)$ = 4.41, p = .01.

There was no significant time, $F(1,165)$ = 0.02, p = .90, or interaction effects, $F(2,165)$ = 1.94, p = .15, for total sleep time. There was a significant overall increase of sleep efficiency with treatment (p = .001) and a significant group x time interaction, $F(2,165)$ = 6.36, p = .002. Simple effects revealed that BT patients (3.7%, d = 0.49) and CBT patients (2.8%, d = 0.37) had greater sleep efficiency increase from pre to post treatment relative to CT patients (−0.7, *NS*, d = −0.09), $F(2,165)$ = 6.36, p < .001.

Daytime Functioning

Adjusted means across conditions and time for the daytime variables are displayed in Table 5. On the Work and Social Adjustment Scale, a significant time effect was observed, $F(2,324)$ = 89.38, p < .001 (pre to post-treatment changes ranging from −1.4, d = −0.87, in the BT condition to −1.9, d = −1.21, in the CT condition), but these changes were not significantly different across conditions, $F(4,324)$ = 0.95, p = .44. No significant time or group x time interaction was obtained for the Multimensional Fatigue Inventory.

On the SF-36 physical component, no groups or time effects were observed, and the group x time interaction failed to reach significance, $F(2,312)$ = 2.20, p = .07. A significant time effect was observed for the mental component, $F(2,312)$ = 33.47, p < .001, but no significant interaction was found (p = .33), suggesting that all three conditions showed similar improvements in the mental aspects of the SF-36 (between 4 and 6 T scores from baseline to FU-6).

Discussion

The goal was to establish the comparative efficacy of BT and CT, relative to their combination (CBT) and to evaluate their effects on nighttime *and* daytime outcomes. Several studies have shown that CBT is effective for various forms of persistent insomnia (e.g., younger and older adults, with and without comorbidities, medication-free and chronic hypnotic users) (e.g., Morin, et al., 2006). The present study extends these findings through a dismantling strategy to document the unique contribution of the key therapeutic components of CBT. The results add to the substantial existing evidence that CBT is an effective treatment for persistent insomnia and that one of its key therapeutic components, BT used singly, is also effective (e.g., Morin, et al., 2006). It also extends previous research with the finding that CT used singly is also effective. Indeed, significant improvements across all three treatment conditions were obtained on measures of insomnia symptom severity, nighttime sleep disturbances, and daytime functioning and these improvements were generally sustained at 6-month follow up.

On the primary end point of the Insomnia Severity Index, at post treatment, there were higher rates of responders to CBT (67%) and BT (67%) relative to CT (42%) and more patients in remission in CBT (57%) and BT (39%) relative to CT condition (31%). While initial treatment response was more modest for CT patients, outcome for CT improved significantly at 6-month followup, as evidenced by higher rates of both responders (62% vs.

44%) and remitters (51% vs. 36%) in CT relative to BT, while CBT still had the best response and remission rates (67% and 62%) rates than either of its single components. Hence, BT produced faster improvements but the improvements were not as well sustained, whereas CT produced slower but better sustained improvements. This different trajectory of changes are perhaps expected given that BT directly targets behavioral and sleep scheduling factors with best effectiveness while they are actively used and/or supervised by a therapist. In contrast, CT targets cognitive processes (e.g., sleep-related worries, unhelpful beliefs, attentional processes), which may take longer to modify but once modified the changes are sustained without further therapist guidance.

Two other features of the Insomnia Severity Index results warrant comment. First, the percentage of responders declined from post-treatment to follow-up in the BT group, but the percentage of remitters did not change. This intriguing finding raises the possibility that response, which implies the presence of residual insomnia symptoms for at least some patients, constitutes a risk for falling back into vicious cycles and insomnia worsening. Second, the non-significant (exact $p = .056$) simple effect comparing response rates between conditions at FU6 likely arises from a combination of two factors: the difference between the two means (CT vs BT) at FU6 is smaller than the difference between the same means at post-treatment and there were larger standard errors due to a smaller number of observations at FU6. Moreover, generalized mixed models dealing with binary data are inherently less powerful than mixed models.

Our hypothesis that the BT group would exhibit greater sleep improvement, relative to the CT group and that full CBT would be at least equal to BT was generally supported as evidenced by improvements on sleep onset latency, wake after sleep onset, and sleep efficiency at post treatment. On the polysomnographic outcomes, BT also showed several small advantages in sleep onset latency and wake after sleep onset at post treatment compared to CT and improved sleep efficiency relative to CT, while CBT was not different from BT on these latter outcomes. These findings make sense given that BT focuses exclusively on nighttime sleep, whereas CT includes interventions for the daytime symptoms. It is also of interest to note that despite relatively small changes on sleep/wake variables, these changes proved clinically significant for a large proportion of patients based on the primary ISI outcome measure. Finally, eight sessions of treatment is longer than previous studies of BT. Perhaps the longer TST noted at post-treatment, relative to previous studies, reflects the longer period available for the week-by-week increases in TIB prescribed by sleep restriction.

Our second hypothesis that CT would be more potent in reducing daytime functional impairments, relative to BT, and produced equivalent outcomes to CBT was not supported. All three therapies produced significant improvements of daytime measures over time. The results highlight that a treatment which focuses entirely on improving nighttime sleep (BT) appears to be sufficient to improve daytime functioning. These results may also speak to the effectiveness of the specific interventions included in CT and CBT for daytime symptoms reported by patients with insomnia. Together these findings add to the existing dialogue on whether daytime symptoms of insomnia are independent of or a consequence of nighttime sleep disturbances.

With regard to the long-term impact of insomnia therapies, there was a significant increase in the percentage of treatment responders from post-treatment to 6 month follow-up in the CT group (+20.2%) and a decrease (−22.9%) in the BT group. Hence, full CBT is the treatment of choice to promote both short- and long-term outcomes. This pattern of findings provides a unique window into more general processes of behavior change, a key emerging interest in our field (Mabry, Olster, Morgan, & Abrams, 2008; Michie, Rothman, & Sheeran, 2007). The findings point to a need for future research to identify why an intervention targeting behavioral change generates faster improvement but is not as well sustained, while an intervention targeting cognitive processes generates slower but more sustained change. Perhaps the behavioral adjustments that are core to BT are easier to implement when a therapist is available for 'coaching'. Or perhaps more emphasis needs to be placed on establishing the behavioral recommendations as habits that the patient automatically reinitiates if/when insomnia recurs. Perhaps there are features of the procedures used in CT that are more conducive to habit formation.

The findings reported here must be interpreted in the light of several methodological issues, each pointing to important domains for future research. First, CBT sessions were 75 minutes while BT and CT sessions were 45–60 minutes. Hence, we cannot exclude the possibility that duration of treatment sessions contributed to the advantage associated with CBT. A question for future research is whether the inclusion of 75 minutes of CT would yield the same response as CBT. Given that session time was constant for BT and CT, one perspective is that the BT vs CT comparison is clearer. Also, we cannot know whether the enhanced version of CT employed in the present study would yield significantly different results relative to the CT traditionally added to CBT-I. Second, eight sessions of CT is shorter than the only prior test of CT for insomnia (average sessions = 14; Harvey, et al., 2007) and for CT for other disorders. For example, CT for depression and social phobia is up to 16 sessions (Clark et al., 2006; DeRubeis et al., 2005), posttraumatic stress disorder is up to 15 sessions (Ehlers et al., 2003) and for schizophrenia is up to 50 sessions (Grant, Huh, Perivoliotis, Stolar, & Beck, 2012), although briefer forms are effective (Clark et al., 1999). Hence, we cannot exclude the possibility that an adequate initial dose of CT requires more than 8 sessions. Third, graduate students delivered the majority of sessions (79%). On the one hand, two intensive 1 day workshops and weekly supervision may not have been a sufficient dose of training for CT delivery. On the other hand, if CBT and BT are easier to disseminate it may confer a benefit to these approaches in terms of dissemination, an enormous problem in our field (Kazdin & Blase, 2011). Fourth, the daytime intervention was focused on a limited number of domains. Coverage of a broader set of domains may have yielded a better outcome. Fifth, on the one hand reviewing a portion of the therapy sessions delivered via audiotape for fidelity to the BT or CT protocols is an important issue for future research. One the other hand, we recognize that it may be difficult to truly isolate behavioral versus cognitive change since improvements in sleep through behavioral means may well improve cognitions and vice versa. The measurement of cognitions in the BT group and behavior in the CT group would allow this issue to be evaluated directly. Sixth, less than 12% of individuals who enquired about the study were enrolled. As evident in Figure 1, the reasons for exclusion highlight the need for insomnia treatment among individuals with psychiatric, medical and substance-related disorders. Also the rigors of

participating in a research study, with the interviews, questionnaires and nights of PSG, may create a disincentive for potential patients and reduce the generalizability of research samples. Seventh, another important question arising from the present study, and relevant for future research, includes whether there are specific patient profiles that would lead a clinician to select CT or BT over CBT. As such, the present study sets the stage to conduct research on the potential for personalized care, based on individual responses, to improve treatment outcome (Insel, 2009). Also, this is the first RCT involving a CT only condition. Accordingly, replication studies will be important additions to the literature. Finally, several additional domains for future research arise from the present study including a need to: assess the comparative adherence and side effects of CT, BT and CBT (Kyle, et al., 2011), include non-self-report assessments of alcoholic and caffeine (key inclusion criteria), determine if men and women differ in their response to treatment and include sufficient samples to compare the outcomes from licensed versus non-licensed therapists as well as possible effects of therapist allegiance.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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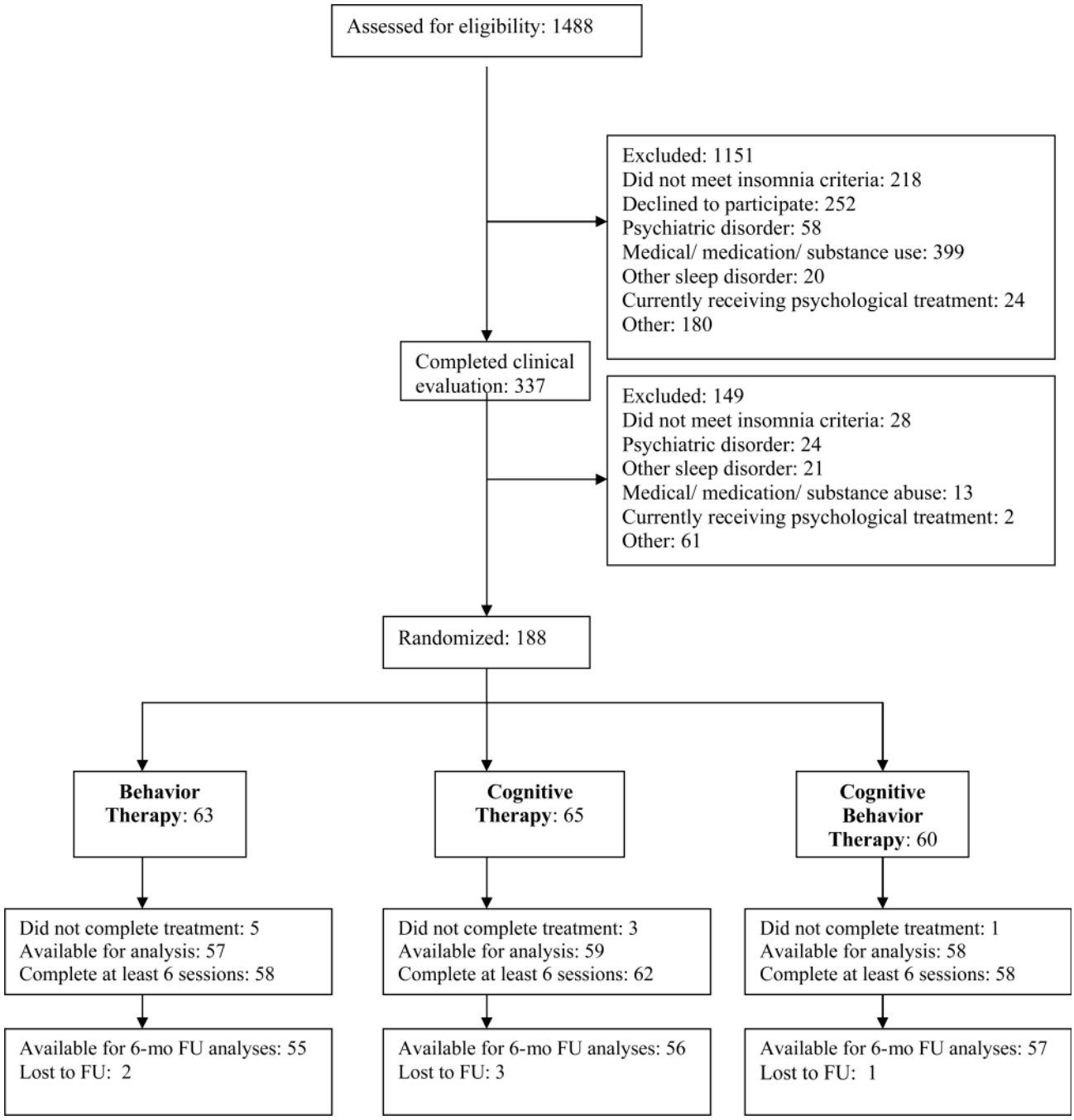

Figure 1.
Flow Diagram of Participants Through Each Stage of the Study

Table 1

Participant Characteristics at Baseline

	CBT (*n* = 60)	**CT (*n* = 65)**	**BT (*n* = 63)**	**Total (*N* = 188)**	**Statistic**
Gender (female) - % (*n*)	53.3 (32)	69.2 (45)	63.5 (40)	62.2 (117)	3.42, *p* = .18
Age (yrs) – *M* (*SD*)	46.9 (11.3)	46.7 (12.8)	48.5 (13.6)	47.4 (12.6)	0.40, *p* = .67
Ethnicity (non-caucasian) - % (*n*)	8.5 (5)	9.2 (6)	3.2 (2)	6.9 (13)	3.61, *p* = .23
Marital Status - % (*n*)					1.73, *p* = .78
Single	33.3 (20)	24.6 (16)	27.0 (17)	28.2 (53)	
Married/Partnered	51.7 (31)	53.9 (35)	55.6 (35)	53.7 (101)	
Divorced/Separated/Widowed	15.0 (9)	21.5 (14)	17.5 (11)	18.1 (34)	
Education (yrs) – *M* (*SD*)	16.7 (3.0)	15.9 (3.4)	15.5 (3.3)	16.0 (3.2)	2.00, *p* = .14
Employment - % (*n*)					7.42, *p* = .12
Full-part time/student	82.8 (48)	76.6 (49)	67.7 (42)	75.5 (139)	
Unemployed	12.1 (7)	6.3 (4)	12.9 (8)	10.3 (19)	
Retired	5.2 (3)	17.2 (11)	19.4 (12)	14.1 (26)	
Insomnia duration (yrs) – *M* (*SD*)	13.8 (11.9)	14.8 (12.9)	14.8 (13.6)	14.5 (12.8)	0.11, *p* = .90
Insomnia Severity Index– *M* (*SD*)	17.9 (3.4)	17.6 (3.5)	18.3 (3.4)	17.9 (3.4)	0.70, *p* = .50
Type of insomnia - % (*n*)					9.53, p = .30
Initial	6.7 (4)	4.6 (3)	6.4 (4)	5.9 (11)	
Middle	21.7 (13)	29.2 (19)	12.7 (8)	21.3 (40)	
Late	3.3 (2)	9.2 (6)	6.4 (4)	6.4 (12)	
Mixed	65.0 (39)	56.9 (37)	73.0 (46)	64.9 (122)	
Non-restorative	3.3 (2)	0.0 (0)	1.6 (1)	1.6 (3)	
Number of apnea/hypopnea /hour (PSG) – *M (SD)*	0.26 (0.86)	0.15 (0.39)	0.14 (0.38)	0.18 (0.58)	0.74, p = .48
Number of PLM associated with arousals (PSG) – *M (SD)*	0.97 (2.64)	0.74 (1.55)	0.52 (1.58)	0.74 (1.98)	0.78, p = .46
Prior medication - % (*n*)					
Antidepressant	1.7 (1)	6.2 (4)	3.2 (2)	3.7 (7)	2.66, p = .26

	CBT (*n* = 60)	CT (*n* = 65)	BT (*n* = 63)	Total (*N* = 188)	Statistic
Hypnotic	35.0 (21)	30.8 (20)	39.7 (25)	35.1 (66)	1.17, p = .57
OTC	20.0 (12)	15.4 (10)	20.6 (13)	18.6 (35)	0.69, p = .71
Comorbidity - % (*n*)					
Medical (any)	59.3 (35)	52.3 (34)	69.8 (44)	60.4 (113)	4.16, *p* = .13
Psychiatric (any)	25.0 (15)	23.1 (15)	23.8 (15)	23.9 (45)	0.06, *p* = .97
Anxiety	18.3 (11)	21.5 (14)	23.8 (15)	21.3 (40)	0.55, *p* = .76
Mood	6.7 (4)	6.2 (4)	6.4 (4)	6.4 (12)	0.01, *p* = .99

Table 2

Adjusted Means and Changes Scores on the Primary Outcome (Insomnia Severity Index) According to Group and Time

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,323)[a]**	**Post-hoc[a]**
Insomnia Severity Index (ISI)						
t1 (Pre)	18.51 (0.49)	18.16 (0.48)	18.91 (0.43)	cond/t1	0.88, $p = .42$	
t2 (Post)	7.94 (0.58)	9.95 (0.63)	9.57 (0.69)	cond/t2	3.71, $p = .03$ (.03)	CBT<CT
t3 (FU6)	8.10 (0.62)	9.25 (0.76)	10.16 (0.73)	cond/t3	2.73, $p = .07$	
Change t1-t2 (*ES*)	−10.56*** (−2.50)	−8.21*** (−1.94)	−9.34*** (−2.21)	cond/t1-t2	3.39, $p = .05$ (.04)	CBT>CT
Change t1-t3 (*ES*)	−10.40*** (−2.46)	−8.91*** (−2.11)	−8.75*** (−2.07)	cond/t1-t3	1.91, $p = .15$	
Change t2-t3 (*ES*)	0.16 NS (0.04)	−0.69 NS (−0.16)	0.59 NS (0.14)	cond/t2-t3	1.08, $p = .34$	
ISI Response - % (reduction of at least 8 points from baseline)						
t1 (Pre)						
t2 (Post)	67.25 (6.99)	42.42 (6.65)	67.35 (7.00)	cond/t2	4.84, $p = .01$ (.02)	CBT=BT>CT
t3 (FU6)	67.55 (6.98)	62.59 (7.12)	44.44 (7.74)	cond/t3	2.94, $p = .06$	
Change t2-t3	0.30 NS	20.17 ***	−22.91 ***	cond/t2-t3	10.07, $p = .00$ (.00)	CT>CBT>BT
ISI Remission - % (ISI < 8)						
t1 (Pre)						
t2 (Post)	57.29 (7.40)	30.84 (6.33)	39.37 (6.78)	cond/t2	3.81, $p = .02$ (.02)	CBT>CT
t3 (FU6)	55.82 (7.29)	51.62 (7.34)	36.45 (7.08)	cond/t3	2.21, $p = .11$	
Change t2-t3	−1.47 NS	20.78 **	−2.92 NS	cond/t2-t3	3.86, $p = .02$ (.02)	CT>CBT=BT

* $p < .05$

** $p < .01$

*** $p < .001$

Note. All means (standard errors) and change scores are adjusted for site and stratification effects.

[a] *p*-values in parentheses are corrected for multiplicity. *df* = 2,153 for ISI Response and Remission. *ES* = effect size (Cohen's *d*) for change scores.

Table 3

Adjusted Means and Changes Scores on the Sleep Diary Variables According to Group and Time

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,289)[a]**	**Post-hoc[a]**
Sleep Onset Latency (in min)						
t1 (Pre)	30.73 (3.01)	32.21 (3.35)	38.39 (4.63)	cond/t1	1.40, $p = .25$	
t2 (Post)	16.76 (2.34)	22.90 (2.79)	19.24 (3.66)	cond/t2	1.86, $p = .16$	
t3 (FU6)	18.99 (2.38)	21.07 (2.89)	24.80 (4.04)	cond/t3	1.20, $p = .30$	
Change t1-t2 (*ES*)	−13.97***(−0.63)	−9.31***(−0.42)	−19.14***(−0.86)	cond/t1-t2	4.52,$p = .01$ (.04)	BT>CT
Change t1-t3 (*ES*)	−11.74***(−0.53)	−11.14***(−0.50)	−13.58***(−0.61)	cond/t1-t3	0.27, $p = 0.76$	
Change t2-t3 (*ES*)	2.23*(0.10)	−1.83 NS (−0.08)	5.56**(0.25)	cond/t2-t3	4.71, $p = .01$ (.03)	CBT=BT<CT
Wake After Sleep Onset (in min)						
t1 (Pre)	55.74 (4.36)	58.81 (4.47)	62.20 (4.99)	cond/t1	0.53, $p = .59$	
t2 (Post)	26.62 (3.33)	38.45 (3.95)	23.76 (3.17)	cond/t2	6.59, $p = .00$ (.00)	CBT=BT<CT
t3 (FU6)	32.29 (4.69)	38.35 (5.38)	29.74 (3.56)	cond/t3	1.09, $p = .34$	
Change t1-t2 (*ES*)	−29.12***(−0.98)	−20.36***(−0.68)	−38.44***(−1.29)	cond/t1-t2	5.99, $p = .00$ (.01)	BT>CT
Change t1-t3 (*ES*)	−23.45***(−0.79)	−20.47***(−0.69)	−32.45***(−1.09)	cond/t1-t3	1.79, $p = .17$	
Change t2-t3 (*ES*)	5.67 NS (0.19)	−0.10 NS (0.00)	5.99**(0.20)	cond/t2-t3	0.77, $p = .46$	
Total Sleep Time (in min)						
t1 (Pre)	348.14 (8.18)	333.79 (7.98)	334.37 (8.34)	cond/t1	1.22, $p = .30$	
t2 (Post)	380.15 (8.05)	366.15 (9.32)	374.89 (7.55)	cond/t2	0.86, $p = .42$	
t3 (FU6)	393.07 (8.91)	367.37 (11.87)	383.13 (7.51)	cond/t3	1.82,$p = .16$	
Change t1-t2 (*ES*)	32.01***(0.53)	32.36***(0.54)	40.52***(0.67)	cond/t1-t2	0.48, $p = .62$	
Change t1-t3 (*ES*)	44.93***(0.75)	33.58***(0.56)	48.76***(0.81)	cond/t1-t3	0.75, $p = .48$	
Change t2-t3 (*ES*)	12.92*(0.21)	1.22 NS (0.02)	8.24 NS (0.14)	cond/t2-t3	0.57, $p = .57$	
Total Time in Bed (in min)						

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,289)[a]**	**Post-hoc[a]**
t1 (Pre)	492.27 (6.63)	478.51 (5.65)	488.79 (7.70)	cond/t1	1.70, $p = .19$	
t2 (Post)	449.84 (6.91)	466.54 (6.23)	438.14 (5.85)	cond/t2	6.51, $p = .00$ (.00)	CBT=BT<CT
t3 (FU6)	476.33 (6.22)	476.35 (6.42)	466.35 (7.36)	cond/t3	0.79, $p = .46$	
Change t1-t2 (*ES*)	−42.43*** (−0.92)	−11.97* (−0.26)	−50.65*** (−1.09)	cond/t1-t2	13.70, $p = .00$ (.00)	CBT=BT>CT
Change t1-t3 (*ES*)	−15.93** (−0.34)	−2.16 NS (−0.05)	−22.44*** (−0.48)	cond/t1-t3	3.60, $p = .03$ (.03)	BT>CT
Change t2-t3 (*ES*)	26.49*** (0.57)	9.81* (0.21)	28.21*** (0.61)	cond/t2-t3	3.87, $p = .02$ (.02)	CBT=BT>CT
Sleep Efficiency (%)						
t1 (Pre)	71.14 (1.57)	70.21 (1.78)	68.65 (1.48)	cond/t1	0.80, $p = .45$	
t2 (Post)	84.84 (1.18)	78.83 (1.88)	85.56 (1.30)	cond/t2	6.73, $p = .00$ (.00)	CBT=BT>CT
t3 (FU6)	82.53 (1.47)	77.37 (2.33)	82.18 (1.36)	cond/t3	2.45, $p = .09$	
Change t1-t2 (*ES*)	13.70*** (1.24)	8.61*** (0.78)	16.91*** (1.53)	cond/t1-t2	12.47, $p = .00$ (.00)	CBT=BT>CT
Change t1-t3 (*ES*)	11.39*** (1.03)	7.16*** (0.65)	13.52*** (1.23)	cond/t1-t3	3.51, $p = 0.03$ (.06)	BT>CT
Change t2-t3 (*ES*)	−2.31* (−0.21)	−1.46 NS (−0.13)	−3.39*** (−0.31)	cond/t2-t3	0.58, $p = .56$	

* $p < .05$

** $p < .01$

*** $p < .001$

Note. All means (standard errors) and change scores are adjusted for site and stratification effects.

[a] p-values in parentheses are corrected for multiplicity. *ES* = effect size (Cohen's *d*) for change scores.

Table 4

Adjusted Means and Changes Scores on PSG-Defined Sleep Variables According to Group and Time (pre and post only)

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,180)[a]**	**Post-hoc[a]**
Sleep Onset Latency (in min)						
t1 (Pre)	13.27 (1.93)	17.74 (2.20)	17.24 (1.55)	cond/t1	1.98, $p = .14$	
t2 (Post)	9.45 (1.36)	17.85 (2.50)	12.36 (1.34)	cond/t2	6.04, $p = .00$ (.00)	CBT=BT<CT
Change t1-t2 (*ES*)	−3.82*(−0.29)	0.12 NS (0.01)	−4.88***(−0.37)	cond/t1-t2	2.10, $p = .13$	
Wake After Sleep Onset (in min)						
t1 (Pre)	47.15 (4.15)	46.65 (3.92)	54.73 (4.35)	cond/t1	1.44, $p = .24$	
t2 (Post)	43.19 (3.97)	48.73 (4.61)	41.22 (3.99)	cond/t2	0.93, $p = .39$	
Change t1-t2 (*ES*)	−3.96 NS (−0.13)	2.08 NS (0.07)	−13.51***(−0.46)	cond/t1-t2	4.41, $p = .01$ (.01)	BT>CT
Total Sleep Time (in min)						
t1 (Pre)	387.61 (5.14)	388.60 (5.31)	381.97 (5.59)	cond/t1	0.49, $p = .61$	
t2 (Post)	389.88 (5.76)	380.72 (6.28)	388.81 (4.66)	cond/t2	0.80, $p = .45$	
Change t1-t2 (*ES*)	2.27 NS (0.06)	−7.88 NS (−0.20)	6.84 NS (0.17)	cond/t1-t2	1.94, $p = .15$	
Total Time in Bed (in min)						
t1 (Pre)	457.57 (2.85)	459.14 (2.39)	461.54 (2.81)	cond/t1	0.55, $p = .58$	
t2 (Post)	448.41 (2.74)	454.83 (3.70)	450.40 (2.39)	cond/t2	0.96, $p = .38$	
Change t1-t2 (*ES*)	−9.16**(−0.43)	−4.31 NS (−0.20)	−11.14**(−0.52)	cond/t1-t2	0.78, $p = .46$	
Sleep Efficiency (%)						
t1 (Pre)	84.27 (1.06)	84.56 (1.06)	82.71 (1.15)	cond/t1	0.93, $p = .40$	
t2 (Post)	87.08 (1.02)	83.85 (1.17)	86.43 (0.99)	cond/t2	2.87, $p = .06$	
Change t1-t2 (*ES*)	2.81*(0.37)	−0.71 NS (−0.09)	3.71***(0.49)	cond/t1-t2	6.36, $p = .00$ (.00)	CBT=BT>CT

*$p < .05$

**$p < .01$

*** $p < .001$

Note. All means (standard errors) and change scores are adjusted for site and stratification effects.

[a] p-values in parentheses are corrected for multiplicity. *ES* = effect size (Cohen's *d*) for change scores.

Table 5

Adjusted Means and Changes Scores on Daytime Functioning According to Group and Time

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,323)[a]**	**Post-hoc**
Multidimensional Fatigue Inventory						
t1 (Pre)	57.91 (0.59)	58.04 (0.69)	58.22 (0.61)	cond/t1	0.08, $p = .92$	
t2 (Post)	58.24 (0.56)	57.63 (0.58)	57.51 (0.60)	cond/t2	0.50, $p = .61$	
t3 (FU6)	57.49 (0.56)	57.96 (0.82)	57.90 (0.57)	cond/t3	0.18, $p = .83$	
Change t1-t2 (*ES*)	0.33 NS (0.07)	−0.41 NS (−0.09)	−0.71 NS (−0.15)	cond/t1-t2	0.74, $p = .48$	
Change t1-t3 (*ES*)	−0.42 NS (−0.09)	−0.08 NS (−0.02)	−0.32 NS (−0.07)	cond/t1-t3	0.06, $p = .94$	
Change t2-t3 (*ES*)	−0.75 NS (−0.16)	0.33 NS (0.07)	0.39 NS (0.08)	cond/t2-t3	1.20, $p = .30$	
Work and Social Adjustment Scale						
t1 (Pre)	3.18 (0.22)	3.58 (0.23)	3.68 (0.23)	cond/t1	1.56, $p = .21$	
t2 (Post)	1.68 (0.20)	1.67 (0.19)	2.31 (0.23)	cond/t2	3.13, $p = .05$ (.18)	
t3 (FU6)	1.41 (0.19)	1.59 (0.24)	1.98 (0.22)	cond/t3	2.31, $p = .10$	
Change t1-t2 (*ES*)	-1.50^{***}(−0.95)	-1.91^{***}(−1.21)	-1.38^{***}(−0.87)	cond/t1-t2	1.64, $p = .19$	
Change t1-t3 (*ES*)	-1.77^{***}(−1.12)	-1.99^{***}(−1.26)	-1.70^{***}(−1.08)	cond/t1-t3	0.35, $p = .71$	
Change t2-t3 (*ES*)	−0.27 NS (−0.17)	−0.08 NS (−0.05)	−0.32 NS (−0.20)	cond/t2-t3	0.52, $p = .60$	
Short Form Health Survey (SF-36) – Physical component						
t1 (Pre)	49.22 (1.10)	50.29 (0.87)	49.51 (1.15)	cond/t1	0.40, $p = .67$	
t2 (Post)	52.08 (0.79)	49.43 (1.06)	50.38 (1.06)	cond/t2	2.74, $p = .07$ (.33)	
t3 (FU6)	50.22 (0.91)	50.55 (1.06)	49.59 (1.31)	cond/t3	0.19, $p = .83$	
Change t1-t2 (*ES*)	2.86^{**}(0.38)	−0.86 NS (−0.12)	0.87 NS (0.12)	cond/t1-t2	4.00, $p = .02$ (.10)	
Change t1-t3 (*ES*)	1.00 NS (0.13)	0.26 NS (0.04)	0.08 NS (0.01)	cond/t1-t3	0.22, $p = .81$	
Change t2-t3 (*ES*)	−1.86a (−0.25)	1.12 NS (0.15)	−0.78 NS (−0.10)	cond/t2-t3	2.30, $p = .10$	
Short Form Health Survey (SF-36) – Mental component						
t1 (Pre)	45.41 (1.10)	43.77 (1.32)	44.28 (1.07)	cond/t1	0.60, $p = .55$	

Means (standard errors), by time and change scores				Comparison between groups		
Time (or change)	**CBT**	**CT**	**BT**	**Effect**	***F*(2,323)[a]**	**Post-hoc**
t2 (Post)	49.15 (1.10)	48.67 (1.29)	48.93 (1.27)	cond/t2	0.05, $p = .95$	
t3 (FU6)	51.46 (0.95)	47.87 (1.36)	48.79 (1.06)	cond/t3	3.62, $p = .03$ (.17)	
Change t1-t2 (*ES*)	3.74** (0.44)	4.90*** (0.58)	4.66*** (0.55)	cond/t1-t2	0.28, $p = .75$	
Change t1-t3 (*ES*)	6.05*** (0.72)	4.10*** (0.49)	4.51*** (0.53)	cond/t1-t3	0.79, $p = .46$	
Change t2-t3 (*ES*)	2.31* (0.27)	−0.80 NS (−0.10)	−0.15 NS	cond/t2-t3	2.29, $p = .10$	

* $p < .05$

*** $p < .01$

*** $p < .001$

Note. All means (standard errors) and change scores are adjusted for site and stratificationeffects.

[a] p-values in parentheses are corrected for multiplicity. *df* = 2,312 for SF-36. *ES* = effect size (Cohen's *d*) for change scores.