

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

Allison G. Harvey  
University of California, Berkeley

Lynda Bélanger  
Université Laval

Lisa Talbot  
University of California, San Francisco

Polina Eidelman  
Cognitive Behavior Therapy and Science Center,  
Oakland, California

Simon Beaulieu-Bonneau, Émilie Fortier-Brochu,  
Hans Ivers, and Manon Lamy  
Université Laval

Kerrie Hein  
University of California, Berkeley

Adriane M. Soehner  
University of Pittsburgh Medical Center,  
Pittsburgh, Pennsylvania

Chantal Mérette and Charles M. Morin  
Université Laval

**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,  $SD$  = 12.6) with persistent insomnia (average of 14.5 years duration). They were randomized to 8 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 3 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, CBT, behavior change

**Supplemental materials:** <http://dx.doi.org/10.1037/a0036606.supp>

This article was published Online First May 26, 2014.

Allison G. Harvey, Department of Psychology, University of California, Berkeley; Lynda Bélanger, Department of Psychology, Université Laval; Lisa Talbot, Department of Psychiatry, University of California, San Francisco; Polina Eidelman, Cognitive Behavior Therapy and Science Center, Oakland, California; Simon Beaulieu-Bonneau, Émilie Fortier-Brochu, Hans Ivers, and Manon Lamy, Department of Psychology, Université Laval; Kerrie Hein, Department of Psychology, University of California, Berkeley; Adriane M. Soehner, Department of Psychiatry, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; Chantal Mérette and Charles M. Morin, Department of Psychology, Université Laval.

This project was supported by National Institute of Mental Health Grant RO1MH079188 to Allison G. Harvey and Charles M. Morin.

Allison G. Harvey and Charles M. Morin contributed to this research equally. Charles M. Morin is a Consultant/Advisory Board member for Merck, Valeant, and Novartis and has a Research Contract with Merck and Novartis.

The authors wish to acknowledge Amélie Bernier, Leslie-Ann Boily, Béatrice de Montigny, Marilyn Vigneault, Vincent Moreau, Cristina Perozzo, Christine Gagnon, Sonia Petit, Bernard Guay, Teresa Caffé, Jennifer Kanady, Jason Lee, Kate Kaplan, Eleanor McGlinchey, and Jamie Rifkin.

Correspondence concerning this article should be addressed to Allison G. Harvey, University of California, Berkeley, Department of Psychology, 2205 Tolman Hall #1650, Berkeley, CA 94720-1650. E-mail: [aharvey@berkeley.edu](mailto:aharvey@berkeley.edu)

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 nonspecific 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 & Lich-

stein, 2000). Moreover, there is very limited evidence that insomnia treatment improves daytime functioning, psychological well-being, and quality of life (National Institutes of Health, 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, Epstein, & Johnson, 2000). However, moderate to large effect sizes on daytime functioning outcomes were reported following 4 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, Quebec, 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 hr per night, as ascertained by daily sleep diaries kept for a 2-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 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 Interna-

tional Classification of Sleep Disorders' criteria (ICSD; American Academy of Sleep Medicine, 2005), and the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) criteria, 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 selective serotonin reuptake inhibitor [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), a circadian-based sleep disorder (e.g., delayed or advanced sleep phase syndrome), body mass index (BMI) of 35 or above, or BMI of 32 or above and reporting at least three 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 9:00 p.m. or later than 2:00 a.m. or rising time earlier than 5:00 a.m. or later than 10:00 a.m., 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, Moras, Barlow, & Rapee, 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 vs. 50+ years) 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 semistructured interview designed to assess *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000) 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 semistructured 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, Valières, & Morin, 2001; Morin, Belleville, Bélanger, & Ivers, 2011) is a seven-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 (*subthreshold 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 posttreatment 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 five nights of evaluation in the sleep laboratory, including one screening/adaptation night, two baseline nights and two 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 2 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

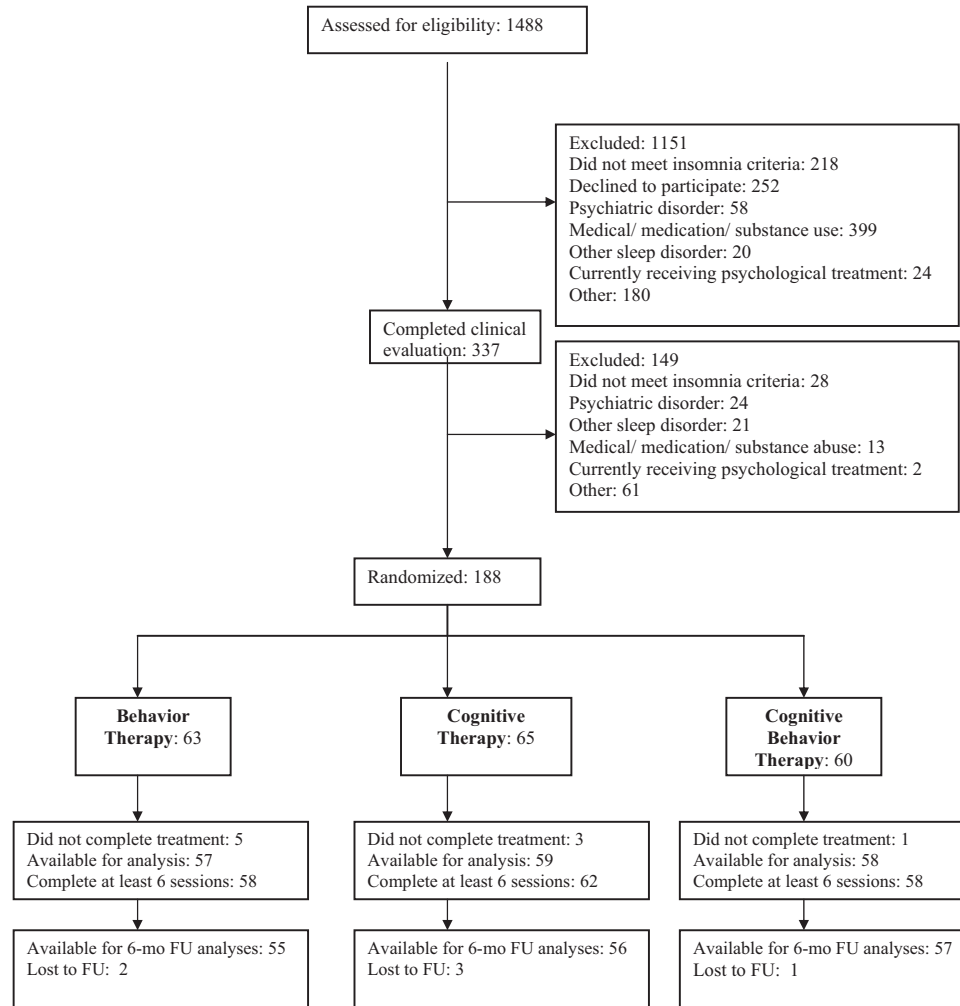


Figure 1. Flow diagram of participants through each stage of the study. FU = follow-up.

(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 five 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 10 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 as follows: (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 assessment 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- to 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 1 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 min and CBT sessions lasting 75 min 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 or 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 the online supplemental materials.

*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 min 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 hr) before 3:00 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)  $\times$  100] to more than 85%. A lower limit of 5 hr 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, (a) unhelpful beliefs about sleep (Morin et al., 2002), (b) sleep-related or sleep-interfering worry (Tang & Harvey, 2004), (c) attentional bias and monitoring for sleep-related threat (Neitzert Semler & Harvey, 2005), and (d) 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 eight 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 multiday 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 2-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)  $\times$  3 (Time: Pre, Post, 6-month follow-up) split-plot mixed model analyses were computed to test Group, Time, and Interaction effects. Linear mixed model analysis was preferred to least-squares analysis of variance (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, nonnormality, and misspecification of the variance-covariance matrix. Group  $\times$  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 American Psychological Association 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 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 follow-up. 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 follow-up (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 years vs. 13.9 years),  $t(186) = 2.37$ ,  $p = .02$ .

### 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 three assessments and an observed overall dropout rate of 6% ( $df$  error for group  $\times$  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  $\times$  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 posttreatment compared to follow-up (FU)-6-month (interaction),  $F(2, 153) = 10.07$ ,  $p < .001$ . Simple effects indicated that there were significantly more treatment responders at posttreatment 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 posttreatment 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 posttreatment, CBT patients were odds ratio (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

Table 1  
Participant Characteristics at Baseline

Variable	CBT ( <i>n</i> = 60)				CT ( <i>n</i> = 65)				BT ( <i>n</i> = 63)				Total ( <i>N</i> = 188)				Statistic
	%	<i>n</i>	<i>M</i>	<i>SD</i>	%	<i>n</i>	<i>M</i>	<i>SD</i>	%	<i>n</i>	<i>M</i>	<i>SD</i>	%	<i>n</i>	<i>M</i>	<i>SD</i>	
Gender (female)	53.3	32			69.2	45			63.5	40			62.2	117			3.42, <i>p</i> = .18
Age (years)			46.9	11.3			46.7	12.8			48.5	13.6			47.4	12.6	0.40, <i>p</i> = .67
Ethnicity (non-Caucasian)	8.5	5			9.2	6			3.2	2			6.9	13			3.61, <i>p</i> = .23
Marital Status																	1.73, <i>p</i> = .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 (years)			16.7	3.0			15.9	3.4			15.5	3.3			16.0	3.2	2.00, <i>p</i> = .14
Employment																	7.42, <i>p</i> = .12
Full- or 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 (years)			13.8	11.9			14.8	12.9			14.8	13.6			14.5	12.8	0.11, <i>p</i> = .90
Insomnia Severity Index			17.9	3.4			17.6	3.5			18.3	3.4			17.9	3.4	0.70, <i>p</i> = .50
Type of insomnia																	9.53, <i>p</i> = .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			
Nonrestorative	3.3	2			0.0	0			1.6	1			1.6	3			
Number of apnea/hypopnea/ hour (PSG)			0.26	0.86			0.15	0.39			0.14	0.38			0.18	0.58	0.74, <i>p</i> = .48
Number of PLM associated with arousals (PSG)			0.97	2.64			0.74	1.55			0.52	1.58			0.74	1.98	0.78, <i>p</i> = .46
Prior medication																	
Antidepressant	1.7	1			6.2	4			3.2	2			3.7	7			2.66, <i>p</i> = .26
Hypnotic	35.0	21			30.8	20			39.7	25			35.1	66			1.17, <i>p</i> = .57
OTC	20.0	12			15.4	10			20.6	13			18.6	35			0.69, <i>p</i> = .71
Comorbidity																	
Medical (any)	59.3	35			52.3	34			69.8	44			60.4	113			4.16, <i>p</i> = .13
Psychiatric (any)	25.0	15			23.1	15			23.8	15			23.9	45			0.06, <i>p</i> = .97
Anxiety	18.3	11			21.5	14			23.8	15			21.3	40			0.55, <i>p</i> = .76
Mood	6.7	4			6.2	4			6.4	4			6.4	12			0.01, <i>p</i> = .99

Note. CBT = cognitive behavior therapy; CT = cognitive therapy; BT = behavior therapy; PSG = polysomnography; PLM = periodic limb movements during sleep; OTC = over-the-counter.

(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 than the similar effect size at posttreatment.

Remission rates followed a similar trajectory for CBT and CT, with a significant Group  $\times$  Time interaction,  $F(2, 153) = 3.86$ ,  $p = .02$ . In this case, there was a larger proportion of patients who remitted at posttreatment 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 posttreatment 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 posttreatment ( $M = 11.3$ ) 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 posttreatment 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  $\times$  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 pretreatment to posttreatment SOL reduction ( $M = -19.1$  min,  $d = -0.86$ ) compared to CT patients ( $M = -9.3$  min,  $d =$

Table 2  
Adjusted Means and Changes Scores on the Primary Outcome (Insomnia Severity Index) According to Group and Time

Time (or change)	Means (SEs), by time and change scores			Comparison between groups		
	CBT	CT	BT	Effect	$F(2, 323)^a$	Post hoc <sup>a</sup>
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 <i>ns</i> (0.04)	–0.69 <i>ns</i> (–0.16)	0.59 <i>ns</i> (0.14)	cond/t2–t3	1.08, $p = .34$	
ISI Response % (reduction of at least 8 points from baseline)						
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 <i>ns</i>	20.17***	–22.91***	cond/t2–t3	10.07, $p = .00$ (.00)	CT > CBT > BT
ISI Remission % (ISI < 8)						
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 <i>ns</i>	20.78**	–2.92 <i>ns</i>	cond/t2–t3	3.86, $p = .02$ (.02)	CT > CBT = BT

Note. CBT = cognitive behavior therapy; CT = cognitive therapy; BT = behavior therapy; FU = follow-up; ES = effect size (Cohen's  $d$ ) for change scores. All means (standard errors) and change scores are adjusted for site and stratification effects.

<sup>a</sup>  $p$ -values in parentheses are corrected for multiplicity.  $df = 2, 153$  for ISI Response and Remission.

\*\*  $p < .01$ . \*\*\*  $p < .001$ .

–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  $\times$  Time interaction,  $F(4, 289) = 6.29$ ,  $p < .001$ . BT and CBT patients showed higher pre to posttreatment 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 follow-up, 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

posttreatment 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  $\times$  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 posttreatment 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  $\times$  Time interaction was obtained for the Multidimensional Fatigue Inventory.

On the SF-36 physical component, no groups or time effects were observed, and the Group  $\times$  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).



Table 3  
Adjusted Means and Changes Scores on the Sleep Diary Variables According to Group and Time

Time (or change)	Means (SEs), by time and change scores			Comparison between groups		
	CBT	CT	BT	Effect	$F(2, 289)^a$	Post hoc <sup>a</sup>
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, $p = .76$	
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)						
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, $p = .03$ (.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, $p = .56$	

Note. CBT = cognitive behavior therapy; CT = cognitive therapy; BT = behavior therapy; FU = follow-up; ES = effect size (Cohen's  $d$ ) for change scores. All means (standard errors) and change scores are adjusted for site and stratification effects.

<sup>a</sup>  $p$ -values in parentheses are corrected for multiplicity.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

## 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 posttreatment, 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 follow-up, 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

Table 4  
Adjusted Means and Changes Scores on PSG-Defined Sleep Variables According to Group and Time (Pre and Post Only)

Time (or change)	Means (standard errors), by time and change scores			Comparison between groups		
	CBT	CT	BT	Effect	$F(2, 180)^a$	Post hoc <sup>a</sup>
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 <i>ns</i> (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 <i>ns</i> (-0.13)	2.08 <i>ns</i> (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 <i>ns</i> (0.06)	-7.88 <i>ns</i> (-0.20)	6.84 <i>ns</i> (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 <i>ns</i> (-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 <i>ns</i> (-0.09)	3.71*** (0.49)	cond/t1–t2	6.36, $p = .00$ (.00)	CBT = BT > CT

Note. PSG = polysomnography; CBT = cognitive behavior therapy; CT = cognitive therapy; BT = behavior therapy; ES = effect size (Cohen's  $d$ ) for change scores. All means (standard errors) and change scores are adjusted for site and stratification effects.

<sup>a</sup>  $p$ -values in parentheses are corrected for multiplicity.

\*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

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 posttreatment 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 nonsignificant (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 posttreatment, 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 posttreatment. 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 posttreatment, 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 that 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

Table 5  
Adjusted Means and Changes Scores on Daytime Functioning According to Group and Time

Time (or change)	Means (SEs), by time and change scores			Comparison between groups	
	CBT	CT	BT	Effect	F(2, 323) <sup>a</sup>
Multidimensional Fatigue Inventory					
t1 (Pre)	57.91 (0.59)	58.04 (0.69)	58.22 (0.61)	cond/t1	0.08, <i>p</i> = .92
t2 (Post)	58.24 (0.56)	57.63 (0.58)	57.51 (0.60)	cond/t2	0.50, <i>p</i> = .61
t3 (FU6)	57.49 (0.56)	57.96 (0.82)	57.90 (0.57)	cond/t3	0.18, <i>p</i> = .83
Change t1–t2 (ES)	0.33 <i>ns</i> (0.07)	−0.41 <i>ns</i> (−0.09)	−0.71 <i>ns</i> (−0.15)	cond/t1–t2	0.74, <i>p</i> = .48
Change t1–t3 (ES)	−0.42 <i>ns</i> (−0.09)	−0.08 <i>ns</i> (−0.02)	−0.32 <i>ns</i> (−0.07)	cond/t1–t3	0.06, <i>p</i> = .94
Change t2–t3 (ES)	−0.75 <i>ns</i> (−0.16)	0.33 <i>ns</i> (0.07)	0.39 <i>ns</i> (0.08)	cond/t2–t3	1.20, <i>p</i> = .30
Work and Social Adjustment Scale					
t1 (Pre)	3.18 (0.22)	3.58 (0.23)	3.68 (0.23)	cond/t1	1.56, <i>p</i> = .21
t2 (Post)	1.68 (0.20)	1.67 (0.19)	2.31 (0.23)	cond/t2	3.13, <i>p</i> = .05 (.18)
t3 (FU6)	1.41 (0.19)	1.59 (0.24)	1.98 (0.22)	cond/t3	2.31, <i>p</i> = .10
Change t1–t2 (ES)	−1.50*** (−0.95)	−1.91*** (−1.21)	−1.38*** (−0.87)	cond/t1–t2	1.64, <i>p</i> = .19
Change t1–t3 (ES)	−1.77*** (−1.12)	−1.99*** (−1.26)	−1.70*** (−1.08)	cond/t1–t3	0.35, <i>p</i> = .71
Change t2–t3 (ES)	−0.27 <i>ns</i> (−0.17)	−0.08 <i>ns</i> (−0.05)	−0.32 <i>ns</i> (−0.20)	cond/t2–t3	0.52, <i>p</i> = .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, <i>p</i> = .67
t2 (Post)	52.08 (0.79)	49.43 (1.06)	50.38 (1.06)	cond/t2	2.74, <i>p</i> = .07 (.33)
t3 (FU6)	50.22 (0.91)	50.55 (1.06)	49.59 (1.31)	cond/t3	0.19, <i>p</i> = .83
Change t1–t2 (ES)	2.86** (0.38)	−0.86 <i>ns</i> (−0.12)	0.87 <i>ns</i> (0.12)	cond/t1–t2	4.00, <i>p</i> = .02 (.10)
Change t1–t3 (ES)	1.00 <i>ns</i> (0.13)	0.26 <i>ns</i> (0.04)	0.08 <i>ns</i> (0.01)	cond/t1–t3	0.22, <i>p</i> = .81
Change t2–t3 (ES)	−1.86a (−0.25)	1.12 <i>ns</i> (0.15)	−0.78 <i>ns</i> (−0.10)	cond/t2–t3	2.30, <i>p</i> = .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, <i>p</i> = .55
t2 (Post)	49.15 (1.10)	48.67 (1.29)	48.93 (1.27)	cond/t2	0.05, <i>p</i> = .95
t3 (FU6)	51.46 (0.95)	47.87 (1.36)	48.79 (1.06)	cond/t3	3.62, <i>p</i> = .03 (.17)
Change t1–t2 (ES)	3.74** (0.44)	4.90*** (0.58)	4.66*** (0.55)	cond/t1–t2	0.28, <i>p</i> = .75
Change t1–t3 (ES)	6.05*** (0.72)	4.10*** (0.49)	4.51*** (0.53)	cond/t1–t3	0.79, <i>p</i> = .46
Change t2–t3 (ES)	2.31* (0.27)	−0.80 <i>ns</i> (−0.10)	−0.15 <i>ns</i>	cond/t2–t3	2.29, <i>p</i> = .10

Note. CBT = cognitive behavior therapy; CT = cognitive therapy; BT = behavior therapy; FU = follow-up; ES = effect size (Cohen's *d*) for change scores. All means (standard errors) and change scores are adjusted for site and stratification effects.

<sup>a</sup> *p*-values in parentheses are corrected for multiplicity. *df* = 2,312 for SF-36.

\* *p* < .05. \*\* *p* < .01. \*\*\* *p* < .001.

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 posttreatment 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 min, while BT and CT sessions were 45–60 min. 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 min 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 eight

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 that arises from the present study (and is relevant for future research) is 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.

## References

- American Academy of Sleep Medicine. (2005). *International classification of sleep disorders (ICSD): Diagnostic and coding manual* (2nd ed.). Westchester, IL: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Baglioni, C., Battagliese, G., Feige, B., Spiegelhalter, K., Nissen, C., Voderholzer, U., . . . Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders, 135*, 10–19. doi:10.1016/j.jad.2011.01.011
- Bastien, C. H., Vallieres, A., & Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine, 2*, 297–307. doi:10.1016/S1389-9457(00)00065-4
- Beck, A. T. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.
- Beck, A. T., Emery, G., & Greenberg, R. L. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York, NY: Basic Books.
- Bootzin, R. R. (1972). Stimulus control treatment for insomnia. *Proceedings of the American Psychological Association, 7*, 395–396.
- Bootzin, R. R. (1979). Effects of self-control procedures for insomnia. *American Journal of Clinical Biofeedback, 2*, 70–77.
- Bootzin, R. R., Epstein, D., & Wood, J. M. (1991). Stimulus control instructions. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 19–28). New York, NY: Plenum. doi:10.1007/978-1-4757-9586-8\_2
- Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: A longitudinal epidemiological study of young adults. *Biological Psychiatry, 39*, 411–418. doi:10.1016/0006-3223(95)00188-3
- Buysse, D. J. (2013). Insomnia. *Journal of the American Medical Association, 309*, 706–716. doi:10.1001/jama.2013.193
- Buysse, D., Ancoli-Israel, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2006). Recommendations for a standard research assessment of insomnia. *Sleep: Journal of Sleep and Sleep Disorders Research, 29*, 1155–1173.
- Buysse, D. J., Germain, A., Hall, M., Monk, T. H., & Nofzinger, E. A. (2011). A neurobiological model of insomnia. *Drug Discovery Today: Disease Models, 8*, 129–137. doi:10.1016/j.ddmod.2011.07.002
- Chow, S.-C., & Liu, J.-P. (1998). *Design and analysis of clinical trials: Concepts and methodologies*. New York, NY: Wiley.
- Clark, D. M., Ehlers, A., Hackmann, A., McManus, F., Fennell, M., Grey, N., . . . Wild, J. (2006). Cognitive therapy versus exposure and applied relaxation in social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 74*, 568–578. doi:10.1037/0022-006X.74.3.568
- Clark, D. M., Salkovskis, P. M., Hackmann, A., Wells, A., Ludgate, J., & Gelder, M. (1999). Brief cognitive therapy for panic disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 67*, 583–589. doi:10.1037/0022-006X.67.4.583
- Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*, 155–159. doi:10.1037/0033-2909.112.1.155
- Daley, M., Morin, C. M., LeBlanc, M., Gregoire, J. P., Savard, J., & Baillargeon, L. (2009). Insomnia and its relationship to health-care utilization, work absenteeism, productivity and accidents. *Sleep Medicine, 10*, 427–438. doi:10.1016/j.sleep.2008.04.005
- DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., . . . Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry, 62*, 409–416. doi:10.1001/archpsyc.62.4.409
- Devilley, G. J., & Borkovec, T. D. (2000). Psychometric properties of the credibility/expectancy questionnaire. *Journal of Behavior Therapy and Experimental Psychiatry, 31*, 73–86. doi:10.1016/S0005-7916(00)00012-4
- Devilley, G. J., & Spence, S. H. (1999). The relative efficacy and treatment distress of EMDR and a cognitive-behavior trauma treatment protocol in the amelioration of posttraumatic stress disorder. *Journal of Anxiety Disorders, 13*, 131–157. doi:10.1016/S0887-6185(98)00044-9
- Di Nardo, P. A., Moras, K., Barlow, D. H., & Rapee, R. M. (1993). Reliability of DSM-III-R anxiety disorder categories: Using the Anxiety Disorders Interview Schedule-Revised (ADIS-R). *Archives of General Psychiatry, 50*, 251–256.
- Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., . . . American Academy of Sleep Medicine Work Group. (2004). Derivation of research diagnostic criteria for insomnia: Report of an American Academy of Sleep Medicine Work Group. *Sleep: Journal of Sleep and Sleep Disorders Research, 27*, 1567–1596.
- Edinger, J. D., Wohlgemuth, W. K., Radtke, R. A., Marsh, G. R., & Quillian, R. E. (2001). Does cognitive-behavioral insomnia therapy alter dysfunctional beliefs about sleep? *Sleep, 24*, 591–599.
- Edinger, J. D., Wyatt, J. K., Olsen, M. K., Stechuchak, K. M., Carney, C. E., Chiang, A., . . . Radtke, R. A. (2009). *Reliability and validity of*

- the Duke Structured Interview for Sleep Disorders for insomnia screening. Paper presented at the 23rd Annual Meeting of the Associated Professional Sleep Societies, LLC, Seattle, WA.
- Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., Fennell, M., Herbert, C., & Mayou, R. (2003). A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. *Archives of General Psychiatry*, *60*, 1024–1032. doi:10.1001/archpsyc.60.10.1024
- Espie, C. A., Inglis, S. J., & Harvey, L. (2001). Predicting clinically significant response to cognitive behavior therapy for chronic insomnia in general medical practice: Analyses of outcome data at 12 months posttreatment. *Journal of Consulting and Clinical Psychology*, *69*, 58–66. doi:10.1037/0022-006X.69.1.58
- First, M. B., Spitzer, M. B., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders—Patient Edition (SCID-I/P, Version 2.0)*. New York, NY: Biometrics Research Department, New York State Psychiatric Institute.
- Grant, P. M., Huh, G. A., Perivoliotis, D., Stolar, N. M., & Beck, A. T. (2012). Randomized trial to evaluate the efficacy of cognitive therapy for low-functioning patients with schizophrenia. *Archives of General Psychiatry*, *69*, 121–127. doi:10.1001/archgenpsychiatry.2011.129
- Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, *40*, 869–893. doi:10.1016/S0005-7967(01)00061-4
- Harvey, A. G. (2006). What about patients who can't sleep? Case formulation for insomnia. In N. Tarrier (Ed.), *Case formulation in cognitive behaviour therapy: The treatment of challenging and complex clinical cases* (pp. 293–311). New York, NY: Brunner-Routledge.
- Harvey, A. G., Sharpley, A. L., Ree, M. J., Stinson, K., & Clark, D. M. (2007). An open trial of cognitive therapy for chronic insomnia. *Behaviour Research and Therapy*, *45*, 2491–2501. doi:10.1016/j.brat.2007.04.007
- Harvey, A. G., & Tang, N. K. J. (2003). Cognitive behavior therapy for insomnia: Can we rest yet? *Sleep Medicine Reviews*, *7*, 237–262. doi:10.1053/smr.2002.0266
- Harvey, A. G., & Tang, N. K. Y. (2012). (Mis)perception of sleep in insomnia: A puzzle and a resolution. *Psychological Bulletin*, *138*, 77–101. doi:10.1037/a0025730
- Hochberg, Y., & Benjamini, Y. (1990). More powerful procedures for multiple significance testing. *Statistical Medicine*, *9*, 811–818. doi:10.1002/sim.4780090710
- Insel, T. R. (2009). Translating scientific opportunity into public health impact: A strategic plan for research on mental illness. *Archives of General Psychiatry*, *66*, 128–133. doi:10.1001/archgenpsychiatry.2008.540
- Jenkinson, C., Stewart-Brown, S., Petersen, S., & Paice, C. (1999). Assessment of the SF-36 Version 2 in the United Kingdom. *Journal of Epidemiology and Community Health*, *53*, 46–50. doi:10.1136/jech.53.1.46
- Kazdin, A. E., & Blase, S. L. (2011). Rebooting psychotherapy research and practice to reduce the burden of mental illness. *Perspectives on Psychological Science*, *6*, 21–37. doi:10.1177/1745691610393527
- Krystal, A. D. (2009). A compendium of placebo-controlled trials of the risks/benefits of pharmacological treatments for insomnia: The empirical basis for U.S. clinical practice. *Sleep Medicine Reviews*, *13*, 265–274. doi:10.1016/j.smr.2008.08.001
- Kushida, C. A., Littner, M. R., Morgenthaler, T., Alessi, C. A., Bailey, D., Coleman, J., Jr., . . . Wise, M. (2005). Practice parameters for the indications for polysomnography and related procedures: An update for 2005. *Sleep: Journal of Sleep and Sleep Disorders Research*, *28*, 499–521.
- Kyle, S. D., Morgan, K., Spiegelhalter, K., & Espie, C. A. (2011). No pain, no gain: An exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Medicine*, *12*, 735–747. doi:10.1016/j.sleep.2011.03.016
- Lichstein, K. L., Durrence, H. H., Riedel, B. W., & Bayen, U. J. (2001). Primary versus secondary insomnia in older adults: Subjective sleep and daytime functioning. *Psychology and Aging*, *16*, 264–271. doi:10.1037/0882-7974.16.2.264
- Lundh, L.-G., & Broman, J.-E. (2000). Insomnia as an interaction between sleep-interfering and sleep-interpreting processes. *Journal of Psychosomatic Research*, *49*, 299–310. doi:10.1016/S0022-3999(00)00150-1
- Mabry, P. L., Olster, D. H., Morgan, G. D., & Abrams, D. B. (2008). Interdisciplinarity and systems science to improve population health: A view from the NIH Office of Behavioral and Social Sciences Research. *American Journal of Preventive Medicine*, *35*(2 Suppl), S211–S224. doi:10.1016/j.amepre.2008.05.018
- Means, M. K., Lichstein, K. L., Epperson, M. T., & Johnson, C. T. (2000). Relaxation therapy for insomnia: Nighttime and daytime effects. *Behaviour Research and Therapy*, *38*, 665–678. doi:10.1016/S0005-7967(99)00091-1
- Michie, S., Rothman, A. J., & Sheeran, P. (2007). Current issues and new direction in *Psychology and Health*: Advancing the science of behavior change. *Psychology & Health*, *22*, 249–253. doi:10.1080/14768320701233582
- Morin, C. M. (1993). *Insomnia: Psychological assessment and management*. New York, NY: Guilford Press.
- Morin, C. M., Bastien, C., Guay, B., Radouco-Thomas, M., Leblanc, J., & Vallieres, A. (2004). Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. *American Journal of Psychiatry*, *161*, 332–342. doi:10.1176/appi.ajp.161.2.332
- Morin, C. M., Beaulieu-Bonneau, S., LeBlanc, M., & Savard, J. (2005). Self-help treatment for insomnia: A randomized controlled trial. *Sleep: Journal of Sleep and Sleep Disorders Research*, *28*, 1319–1327.
- Morin, C. M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The Insomnia Severity Index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep: Journal of Sleep and Sleep Disorders Research*, *34*, 601–608.
- Morin, C. M., & Benca, R. (2012). Chronic insomnia. *The Lancet*, *379*, 1129–1141. doi:10.1016/S0140-6736(11)60750-2
- Morin, C. M., Blais, F., & Savard, J. (2002). Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of insomnia? *Behaviour Research and Therapy*, *40*, 741–752. doi:10.1016/S0005-7967(01)00055-9
- Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: An update of recent evidence (1998–2004). *Sleep*, *29*, 1398–1414.
- Morin, C. M., Colecchi, C., Stone, J., Sood, R., & Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *Journal of the American Medical Association*, *281*, 991–999. doi:10.1001/jama.281.11.991
- Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: A meta-analysis of treatment efficacy. *American Journal of Psychiatry*, *151*, 1172–1180.
- Morin, C. M., & Espie, C. A. (2003). *Insomnia: A clinical guide to assessment and treatment*. New York, NY: Kluwer Academic/Plenum Press. doi:10.1002/0471264385.wei0914
- Morin, C. M., Vallieres, A., Guay, B., Ivers, H., Savard, J., Merette, C., . . . Baillargeon, L. (2009). Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: A randomized controlled trial. *JAMA: Journal of the American Medical Association*, *301*, 2005–2015. doi:10.1001/jama.2009.682
- Mundt, J. C., Marks, I. M., Shear, M. K., & Greist, J. H. (2002). The Work and Social Adjustment Scale: A simple measure of impairment in functioning. *British Journal of Psychiatry*, *180*, 461–464. doi:10.1192/bjp.180.5.461

- Neitzert Semler, C., & Harvey, A. G. (2005). Misperception of sleep can adversely affect daytime functioning in insomnia. *Behaviour Research and Therapy*, *43*, 843–856. doi:10.1016/j.brat.2004.06.016
- National Institutes of Health. (2005). National Institutes of Health State of the Science Conference Statement: Manifestations and management of chronic insomnia in adults. June 13–15, 2005. *Sleep*, *28*, 1049–1057.
- Ohayon, M. M., & Reynolds, C. F. (2009). Epidemiological and clinical relevance of insomnia diagnosis algorithms according to the *DSM-IV* and the International Classification of Sleep Disorders (ICSD). *Sleep Medicine*, *10*, 952. doi:10.1016/j.sleep.2009.07.008
- Perlis, M. L., Aloia, M., & Kuhn, B. (2011). *Behavioral treatment for sleep disorders: A comprehensive primer of behavioral sleep medicine*. Amsterdam, the Netherlands: Elsevier.
- Persons, J. B. (2006). Case formulation-driven psychotherapy. *Clinical Psychology: Science and Practice*, *13*, 167–170. doi:10.1111/j.1468-2850.2006.00019.x
- Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. Bethesda, MD: U.S. Dept. of Health Education and Welfare.
- Ree, M., & Harvey, A. G. (2004). Insomnia. In J. Bennett-Levy, G. Butler, M. Fennell, A. Hackman, M. Mueller, & D. Westbrook (Eds.), *Oxford guide to behavioural experiments in cognitive therapy* (pp. 287–305). Oxford, England: Oxford University Press.
- Riedel, B. W., & Lichstein, K. L. (2000). Insomnia and daytime functioning. *Sleep Medicine Reviews*, *4*, 277–298. doi:10.1053/smr.1999.0074
- Sivertsen, B., Øverland, S., Bjorvatn, B., Mæland, J. G., & Mykletun, A. (2009). Does insomnia predict sick leave? The Hordaland Health Study. *Journal of Psychosomatic Research*, *66*, 67–74. doi:10.1016/j.jpsychores.2008.06.011
- Smets, E. M., Garssen, B., Bonke, B., & De Haes, J. C. (1995). The Multidimensional Fatigue Inventory (MFI) psychometric qualities of an instrument to assess fatigue. *Journal of Psychosomatic Research*, *39*, 315–325. doi:10.1016/0022-3999(94)00125-O
- Smith, M. T., Perlis, M. L., Park, A., Smith, M. S., Pennington, J., Giles, D. E., & Buysse, D. J. (2002). Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *The American Journal of Psychiatry*, *159*, 5–11. doi:10.1176/appi.ajp.159.1.5
- Spielman, A. J., Caruso, L. S., & Glovinsky, P. B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America*, *10*, 541–553.
- Spielman, A. J., & Glovinsky, P. (1991). The varied nature of insomnia. In P. J. Hauri (Ed.), *Case studies in insomnia* (pp. 1–15). New York, NY: Plenum Press. doi:10.1007/978-1-4757-9586-8\_1
- Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep: Journal of Sleep Research & Sleep Medicine*, *10*, 45–56.
- Tang, N. K., & Harvey, A. G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep: Journal of Sleep and Sleep Disorders Research*, *27*, 69–78.
- Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-item Short-Form Health Status Survey (SF-36): 1. Conceptual framework and item selection. *Medical Care*, *30*, 473–483. doi:10.1097/00005650-199206000-00002
- Wilkinson, L. (1999). Statistical methods in psychology journals: Guidelines and explanations. *American Psychologist*, *54*, 594–604. doi:10.1037/0003-066X.54.8.594

Received April 19, 2013

Revision received February 14, 2014

Accepted March 3, 2014 ■