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# Cognitive–behavioral therapy for primary insomnia

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**Abstract**

Primary insomnia (PI) is a prevalent form of sleep difficulty that impairs diurnal functioning, reduces quality of life and enhances health care utilization/costs for millions worldwide. Whereas the underlying pathophysiology of PI remains poorly understood, it is widely accepted that a host of cognitive and behavioral factors play important roles in perpetuating this condition. As such, a multi-factorial, cognitive–behavioral therapy (CBT) has emerged as a "treatment of choice" for managing the sleep/wake complaints of PI sufferers. This article considers the nature and relative merits of CBT for treating PI patients. In addition, this article reviews studies supporting the general efficacy and clinical effectiveness of CBT for treating PI complaints. Issues related to treatment implementation as well as factors that mediate patients' responses to CBT and predict treatment acceptance/outcome are also considered. Finally, remaining questions regarding CBT's application to PI are considered, and suggestions for future research are provided.
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*Keywords:* Cognitive–behavioral therapy; CBT; Primary insomnia

Primary Insomnia (PI) is a prevalent and potentially serious condition that adversely affects the functioning, health status, and quality of lives of millions worldwide. Currently, both the nature and roles of underlying neural mechanisms and hereditary factors involved in this form of sleep difficulty remain poorly understood. However, it has long been recognized that a host of psychological and behavioral factors play central roles in perpetuating this condition. As such, PI is particularly well suited for the behavioral therapies that address these psychological and behavioral perpetuating mechanisms. Given

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this observation, it is no surprise that the bulk of the studies conducted to test and refine behavioral insomnia therapies have been conducted with samples of PI patients.

The current article discusses the nature and significance of primary insomnia (PI) as well as the behavioral therapies designed for its management. In the initial section of this article we review the definition of PI and discuss its prevalence and associated morbidity. Subsequently, we consider PI's etiology and provide a conceptual rationale for use of behavioral treatments with PI by considering those psychological and behavioral factors presumed to sustain this disorder. Following a brief review of historically popular behavioral insomnia therapies, we describe current-day cognitive–behavioral insomnia therapy (CBT) and consider both its general efficacy and its clinical effectiveness with "real-world" patients. In addition, we discuss various issues related to treatment implementation such as optimal treatment "dosing", cost-effectiveness, and methods of delivery, and we consider those factors that mediate acceptance of and predict response to CBT. We conclude our discussion by outlining the current limitations in the CBT insomnia literature and suggesting directions for future research.

## 1. Primary insomnia: definition and epidemiology

PI is a diagnostic term specific to the American Psychiatric Association's sleep disorder nosology outlined in recent versions of its Diagnostic and Statistical Manuals. This diagnostic category first appeared as a formal insomnia diagnosis in the revised, third edition of the Association's Diagnostic and Statistical Manual (American Psychiatric Association, 1987) and has been maintained through subsequent revisions of this text (American Psychiatric Association, 1994, 2000). PI's diagnostic criteria listed in Table 1 highlight the primary or central role sleep/wake disturbance serves in defining this condition. In fact, these criteria specify that a PI diagnosis is assigned when the insomnia does not occur only during the course of another primary sleep or psychiatric disorder and is not the direct result of a general medical disorder or substance use/abuse. As such, PI is perhaps best conceptualized as a diagnosis established by exclusion of other primary and secondary forms of sleep disturbance. Nonetheless, PI can usually be discerned from clinical interview, as expensive and time-consuming laboratory tests are seldom needed for diagnosis of insomnia (Chesson et al., 2000).

Table 1
Diagnostic criteria for primary insomnia

A. The predominant complaint is difficulty initiating or maintaining sleep or nonrestorative sleep for at least 1 month.
B. The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The sleep disturbance does not occur exclusively during the course of narcolepsy, breathing-related sleep disorder, circadian rhythm sleep disorder, or a parasomnia.
D. The disturbance does not occur exclusively during the course of another mental disorder (e.g., major depressive disorder, generalized anxiety disorder, delirium).
E. The disturbance is not due to the direct physiologic effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Diagnostic criteria taken from the Diagnostic and Statistical Manual, Fourth Edition—Text Revision published by the American Psychiatric Association.

Epidemiologic studies suggest that 9% to 15% of the general population suffer from some form of chronic insomnia, whereas between 1% and 2% meet criteria for PI (Ohayon, 1997, 2002; Ohayon & Partinen, 2002). Insomnia diagnoses in general are most common in middle-aged and older adults, but PI is the most frequent insomnia diagnosis found in adolescents and young adults (Ohayon, 2002; Ohayon & Roberts, 2001). As such, PI's prevalence is relatively stable across age groups. Although many insomnia sufferers go undetected (Ancoli-Israel & Roth, 1999), PI is common in primary care settings and accounts for over 20% of all insomnia sufferers who present to specialty sleep disorders centers (Coleman et al., 1982; Simon & VonKorff, 1997). Thus, PI appears sufficiently prevalent and disturbing so as to frequently come to the attention of both sleep specialists and general medical practitioners.

Since PI is devoid of secondary causes, there is, perhaps, a temptation to view this problem as less serious than those insomnias arising from medical, psychiatric, or substance abuse problems. However, epidemiologic evidence suggests insomnia, uncomplicated by co-morbid psychiatric, substance abuse, or medical disorders, substantially increases health care utilization/costs and accounts for as many as 3.5 disability days per month among affected individuals (Hajak, 2001; Simon & VonKorff, 1997; Weissman, Greenwald, Nino-Murcia, & Dement, 1997). Also, several studies have shown that PI complaints reported during an initial interview predicted subsequent depressive illnesses ascertained 1 to 45 years later even after other significant predictors were statistically controlled (Breslau, Roth, Rosenthal, & Andreski, 1996; Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997; Ford & Kamerow, 1989; Livingston, Blizard, & Mann, 1993; Vollrath, Wicki, & Angst, 1989). In addition, PI contributes to reduced productivity, work-related accidents, increased alcohol consumption, serious falls among the elderly, and a general sense of being in poor health (Brassington, King, & Bliwise, 2000; Gislason & Almqvist, 1987; Johnson, Roehrs, Roth, & Breslau, 1998; Johnson & Spinweber, 1983; Katz & McHorney, 1998). Thus, when encountered clinically, PI warrants timely, effective, and enduring treatment.

## 2. Etiology

Currently, mechanisms underlying the pathophysiology of PI remain poorly understood. Deficiencies in endogenous melatonin or benzodiazepine receptors and hyperactivity of corticotrophin releasing factor neurons have been cited as potentially important to the etiology of PI (Richardson & Roth, 2001). However, the etiological importance of these factors has yet to be empirically documented. Likewise, recent findings (Yves et al., 2003) showing a pronounced familial propensity toward the development of PI imply that genetic or hereditary factors may play a role in the etiology of this condition. To date, genetic aberrations specific to PI have not been identified, so it is possible that familial trends toward this sleep difficulty are due to learning/modeling rather than to genetic transmission.

Despite limited information concerning PI's underlying pathophysiology and heritability, there is a general agreement that this condition is perpetuated by the interplay of cognitive and behavioral mechanisms as illustrated in Fig. 1. Setting the stage for sustained sleep difficulty is a host of cognitive aberrations including misattributions about the causes of insomnia, misconceptions about sleep needs and the effects of sleep loss, propensities toward catastrophizing about the consequences of poor sleep, and dysfunctional beliefs about sleep promoting practices (Edinger et al., 2000; Morin, 1993; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993). These cognitions, in turn, support and

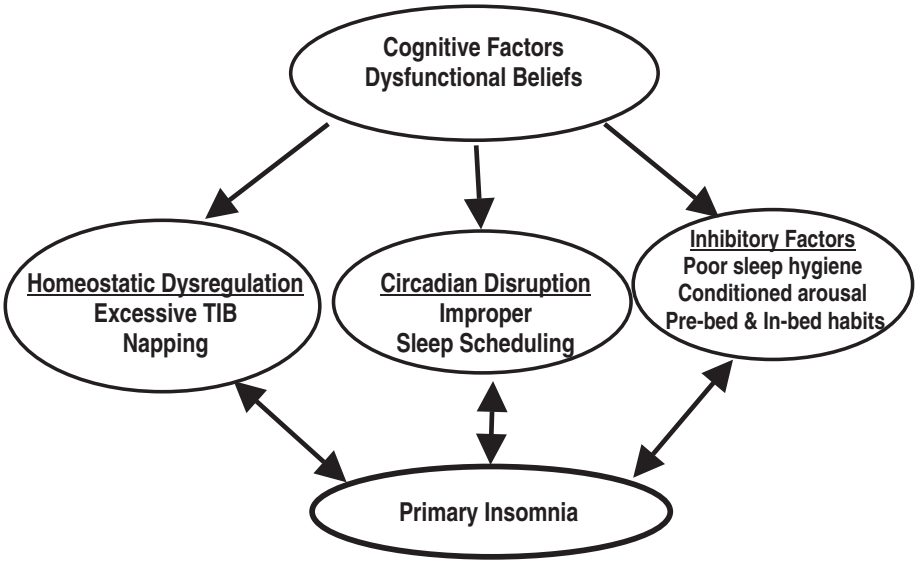


Fig. 1. Sleep-disruptive pre-bed habits include engaging in physically, psychologically, or emotionally arousing activities shortly before bed. Sleep disruptive in-bed habits include watching TV, eating, reading, ruminating or spending long times awake in bed at night.

sustain sleep-disruptive habits and conditioned emotional responses that either interfere with normal sleep drive or timing mechanisms or serve as environmental/behavioral inhibitors to sleep. (Bootzin, 1977; Morin, 1993; Spielman, Saskin, & Thorpy, 1987; Webb, 1988). For example, daytime napping or spending extra time in bed in pursuit of elusive, unpredictable sleep may only serve to interfere with the body's homeostatic mechanisms that operate automatically to increase sleep drive in the face of increasing periods of wakefulness (i.e., sleep debt). Alternately, the habit of remaining in bed well beyond the normal rising time following a poor night's sleep may disrupt the body's circadian or "clock" mechanisms that control the timing of sleep and wakefulness in the 24-h day. Additionally, the repeated association of the bed and bedroom with unsuccessful sleep attempts may eventually result in sleep-disruptive conditioned arousal in the home sleeping environment. Finally, failure to discontinue mentally demanding work and allot sufficient "wind-down" time before bed may serve as a significant sleep inhibitor during the subsequent sleep period. In sum, all of these factors may contribute to and perpetuate PI (Bootzin & Epstein, 2000; Edinger & Wohlgemuth, 1999; Hauri, 2000; Morin, Savard & Blais, 2000).

## 3. The behavioral therapies for primary insomnia

Given the important roles that the above-mentioned cognitive and behavioral factors play in perpetuating PI, a variety of behavior therapies have been proposed for this condition. A detailed overview of the most widely used therapies for PI is provided in Table 2. As shown in this table, a host of formal *relaxation therapies* have been applied to PI since such therapies reduce the sleep-related performance anxiety and bedtime arousal common to this condition (Borkovec & Fowles,

Table 2
Common behavioral therapies for primary insomnia

| Type of treatment | Treatment description |
|---|---|
| Relaxation therapy | ■ Various techniques used to treat PI including progressive muscle relaxation, passive relaxation, autogenic training, biofeedback, imagery training, meditation, and hypnosis.<br>■ The therapy goal is to reduce or eliminate sleep-disruptive physiological (e.g., muscle tension) and/or cognitive (e.g., racing thoughts) arousal.<br>■ Regardless of the specific relaxation strategy employed, treatment typically entails conducting specific treatment exercises and teaching relaxation skills over multiple treatment sessions. |
| Stimulus control therapy | ■ This approach is based on the assumption that both the timing (bedtime) and sleep setting (bed/bedroom) are associated with repeated unsuccessful sleep attempts and, over time, become conditioned cues for arousal that perpetuate insomnia. As a result, the goal of this treatment is that of re-associating the bed and bedroom with successful sleep attempts.<br>■ In practice, this therapy requires instructing the patient to: (a) go to bed only when sleepy; (b) establish a standard wake-up time; (c) get out of bed whenever awake for long periods; (d) avoid reading, watching TV, eating, worrying and other sleep-incompatible behaviors in the bed/bedroom; and (e) refrain from daytime napping.<br>■ Stimulus control instructions usually can be administered in one visit, but follow-up visits to facilitate compliance are beneficial. |
| Sleep restriction therapy | ■ Sleep restriction therapy reduces nocturnal sleep disturbance primarily by restricting the time allotted for sleep each night so that, eventually, the time spent in bed closely matches the individual's presumed sleep requirement.<br>■ This treatment typically begins by calculating the individual's average total sleep time (ATST) from a sleep log that is kept for 1 to 2 weeks.<br>■ An initial time-in-bed (TIB) prescription may either be set at the ATST or at a value equal to the ATST plus an amount of time that is deemed to represent normal nocturnal wakefulness (e.g., ATST + 30 min). The initial TIB prescription is seldom set below 5 h per night.<br>■ On subsequent visits TIB may be adjusted up or down in 15 to 30 min increments dependent upon the patient's sleep performance and waking function.<br>■ Therapy typically entails an initial visit to introduce treatment instructions and follow-up visits to alter TIB prescriptions. |
| Sleep hygiene | ■ Patients are educated about healthy sleep behaviors and sleep-conducive environmental conditions. Typically they are encouraged to exercise daily, eliminate the use of caffeine, alcohol, and nicotine, eat a light snack at bedtime, and ensure that the sleeping environment is quiet, dark, and comfortable.<br>■ Sleep hygiene is seldom used as a primary intervention, but is often included with other interventions. |

1973; Jacobson, 1964; Nicassio & Bootzin, 1974; Schultz & Luthe, 1959). *Stimulus control therapy* (SCT: Bootzin, 1972, 1977), a simple, straightforward classical conditioning approach, also has been popular since this treatment is well suited to address the conditioned timing (bedtime) and setting (home bedroom) cues for arousal that may perpetuate sleep difficulty. In contrast, more global lifestyle and environmental factors that serve as sleep inhibitors are targeted in *sleep hygiene therapy* (Hauri, 1982). Finally, *sleep restriction therapy* (SRT) designed primarily to restore normal [homeostatic] sleep drive has enjoyed wide popularity as well.

Over the years, each of these first generation behavioral therapies has shown some promise for treating PI, although SCT and SRT have proven to be the most efficacious (Morin, Culbert, & Schwartz, 1994; Morin, Hauri et al., 1999; Murtagh & Greenwood, 1995). Nonetheless, as illustrated in Fig. 2, none of these interventions addresses all PI perpetuating mechanisms. Relaxation approaches target conditioned arousal and sleep-related anxiety but largely ignore

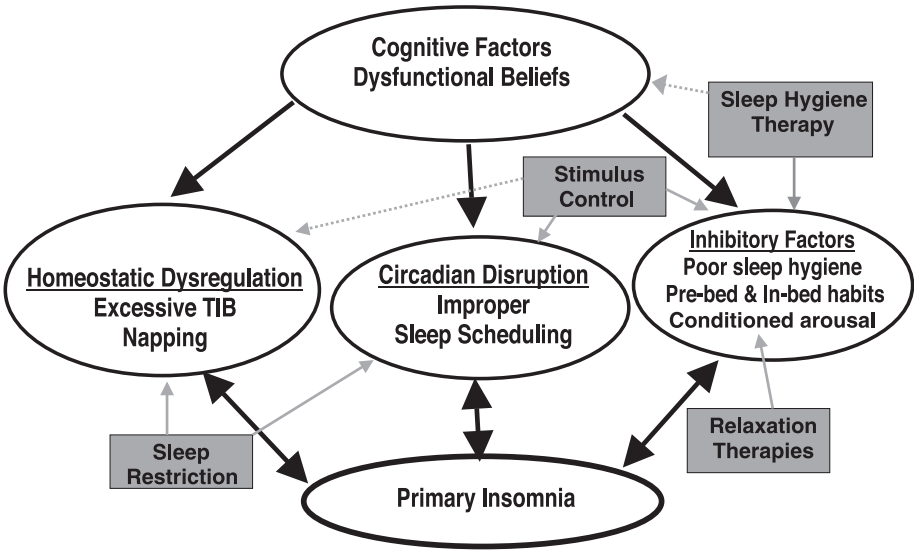


Fig. 2. Sleep-disruptive pre-bed habits include engaging in physically, psychologically, or emotionally arousing activities shortly before bed. Sleep disruptive in-bed habits include watching TV, eating, reading, ruminating or spending long times awake in bed at night. Note that arrows drawn from the treatment "boxes" with solid lines indicate a primary effect whereas dotted lines indicate a possible incidental or secondary effect of the treatment.

cognitive, homeostatic, and circadian aberrations that sustain sleep difficulty. Sleep hygiene directly addresses some inhibitory mechanisms and indirectly may alter some dysfunctional sleep-related beliefs through the sleep education it typically includes. SCT directly addresses both circadian and inhibitory mechanisms (e.g., conditioned arousal) involved in PI but has less direct effects on homeostatic sleep drive since it places no limits on time spent in bed. Furthermore, this therapy does not include specific interventions for cognitive factors that sustain many sleep-disruptive attitudes and behaviors. Finally, SRT addresses the homeostatic and circadian mechanisms that sustain PI but ignores many inhibitory factors and maladaptive beliefs and attitudes about sleep.

Due to the limitations of these first generation approaches, a more omnibus hybrid approach currently called cognitive–behavioral therapy (CBT) has become popular for the management of PI (Edinger, Hoelscher, Marsh, Lipper, & Ionescue-Pioggia, 1992; Hoelscher & Edinger, 1988; Morin, 1993; Morin, Kowatch, Barry, & Walton, 1993). Although various renditions of this approach have been described, included in all of these are treatment components that address the array of cognitive, homeostatic, circadian, and sleep-inhibitory factors that sustain PI. Common to all forms of CBT is a form of cognitive therapy such as formal cognitive restructuring (Morin, 1993) or a standardized sleep education package (Edinger et al., 1992; Hoelscher & Edinger, 1988) to correct dysfunctional beliefs and attitudes about sleep common to PI. Moreover, in order to address the host of homeostatic, circadian, and conditioning factors that may perpetuate PI, CBT combines SCT and SRT resulting in a modified set of behavioral prescriptions. With the modified regimen, patients are instructed to: (a) adhere to a standard rising time; (b) avoid extended periods in bed awake; (c) eliminate sleep-incompatible behaviors in the bed and bedroom; (d) avoid daytime napping; and (e) limit total time in bed (TIB) each night to a specific, individually prescribed amount. The initial TIB prescription is set to approximate the patient's average sleep time (estimated from a pre-treatment

sleep log). TIB subsequently is adjusted up or down (in 15- to 30-min increments) on a weekly basis contingent upon observed sleep improvements or decrements. When sleep becomes well consolidated and waking function appears improved or at least unimpaired, no further TIB adjustments are effected. Thus, CBT provides a comprehensive treatment for PI by eliminating common sleep-disruptive behaviors and correcting the beliefs/attitudes that support such practices.

## 4. Efficacy of CBT for treating PI

The usefulness of a treatment for a particular disorder is typically first established by efficacy studies in which the treatment is tested with well-characterized and carefully selected patient samples using a rigorous experimental design. The initial studies designed to test CBT for treating PI utilized single subject multiple baseline designs to test the efficacy of CBT with middle-aged and older patients who presented sleep-maintenance difficulties (Edinger et al., 1992; Hoelscher & Edinger, 1988). In the earlier of these studies, four PI sufferers were given 4 weeks of CBT after varying baseline periods. In the later study, seven PI patients completed varying baseline periods and 4 weeks of progressive relaxation treatment prior to completing 4 weeks of CBT. In both studies the majority of patients showed clinically significant subjective and objective improvements in common measures of sleep consolidation (e.g., sleep efficiency, nocturnal wake time) only after the introduction of CBT. Furthermore, these patients maintained these improvements through 3- to 6-month follow-up periods.

Since the time of these early reports, four well controlled randomized clinical trials have been conducted specifically to test CBT's efficacy with PI patients. Table 3 provides a listing and brief description of these studies. The findings from these studies collectively suggest that CBT produces significantly greater subjective and objective sleep improvements than do no treatment, pharmacologic and non-pharmacologic placebo interventions, and progressive relaxation therapy. These studies also demonstrated that sleep improvements resulting from CBT tend to endure across post-treatment follow-up periods varying from 3 to 24 months. In addition, findings from one of these studies (Morin, Colecchi, Stone, Sood, & Brink, 1999) suggest that combined CBT and pharmacotherapy (temazepam) may produce slightly greater short-term sleep improvements than does CBT alone. However, the advantages of this combined approach over the long-term seems less clear since many patients receiving this treatment regress significantly over long-term follow-up. In fact, on average, those receiving CBT alone showed better maintenance of their sleep improvements at the end of a 2-year follow-up than did those receiving combined CBT and pharmacotherapy.

It also should be noted that findings from these and related studies show CBT leads to improvement in clinically important sleep-related metrics in addition to subjective/objective measures of sleep. Compared to PI patients receiving relaxation therapy, placebo treatment, or no treatment, CBT-treated PI patients show larger improvements on questionnaire measures of their sleep/wake insomnia symptoms, personal self-efficacy in regard to sleep, and dysfunctional sleep-related cognitions (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001a; Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001b; Morin, Blais & Savard, 2002; Morin, Colecchi et al., 1999). Likewise, when improvement rates are used as a metric, CBT appears to fair well against other treatments. Edinger et al. (2001a), for example, found that a significantly greater percentage of their CBT-treated patients with sleep-maintenance PI met predetermined improvement criteria (i.e., a 50% pre- to post-treatment reduction in wake time after sleep onset) than did patients treated with either relaxation

Table 3
CBT treatment efficacy studies conducted with primary insomnia patients

| Citation | *N* | Mean age (years) | Treatment groups | Treatment duration (weeks) | Treatment format | Follow-up period (months) | Findings |
|---|---|---|---|---|---|---|---|
| Morin et al., 1993 | 24 | 67.1 | CBT<br>WL | 8 | Group | 12 | CBT significantly better than WL across most measures.<br>For CBT, percentage reductions in WASO were 53.7% (diaries) and 51.3% (PSG). For WL, percentage reductions in WASO were 12.3% (diaries) and 10.4% (PSG).<br>Improvements from CBT persisted over 12-month follow-up. |
| Morin et al., 1999 | 78 | 65.0 | CBT<br>PCT<br>CBT+PCT<br>Placebo drug | 8 | Group | 24 | 3 active treatments improved sleep better than placebo drug by post-treatment.<br>Combined therapy slightly better than single treatments in the short-term.<br>Percentage reductions in WASO were 63.5% for CBT+PCT, 55% for CBT, 46.5% for PCT, and 16.9% for placebo.<br>As a group, those treated with CBT alone maintained improvements across 2-year follow-up better than those receiving combined therapy although considerable variability was noted in the latter group. |
| Mimeault & Morin, 1999 | 54 | 50.8 | SHCBT<br>SHCBT+P<br>WL | 6 | Self-help manual ± Phone support from therapist | 3 | Both active treatments improved SE% and TWT whereas WL did not.<br>Phone assistance provided slight short-term benefit but this advantage did not persist through follow-up. By follow-up the SHCBT group showed 36% and 19.1% improvements in TWT and SE%, respectively. SHCBT+P showed 32.5% and 13.4% improvements in TWT and SE%. |
| Edinger et al., 2001a, 2001b | 75 | 55.8 | CBT<br>PMR<br>Placebo | 6 | Individual therapy | 6 | CBT produced a 54% reduction in subjective WASO compared to 16% for PMR and 12% for placebo.<br>PSG differences smaller but supported CBT over other treatments.<br>CBT significantly better than other treatments for reducing subjective insomnia symptoms and dysfunctional beliefs about sleep. |

Note: CBT=cognitive–behavioral therapy; PMR=progressive muscle relaxation therapy; WL=waiting list condition; PSG=polysomnography; PCT=pharmacotherapy; SHCBT=self-help CBT treatment; P=phone therapy; SE%=sleep efficiency; TWT=total wake time; WASO=wake time after sleep onset.

therapy or a non-drug placebo therapy. Thus, the efficacy studies show that CBT produces marked changes across a range of clinically important treatment outcome measures.

## 5. Clinical effectiveness of CBT with PI

The various well-designed efficacy studies reviewed above provide strong support for CBT's usefulness with PI. However, one shortcoming of these studies is their use of highly screened and selected research volunteers that may not represent the typical PI patient encountered in clinical practice. As a result, it is questionable whether the very promising findings of these studies are likely to generalize to the population of PI sufferers as a whole. To make this determination, additional clinical effectiveness studies conducted with less carefully screened "real-world" patients are necessary.

Fortunately, a number of studies have evaluated the clinical effectiveness of CBT with clinic samples of PI patients. Table 4 provides an overview of such studies. Included in this table are only those studies that evaluated CBT's effectiveness specifically and exclusively with PI sufferers. Excluded from the table are effectiveness studies conducted with multicomponent behavioral treatments other than CBT as well as studies of mixed insomnia subtypes that lacked separate evaluation of CBT's effectiveness within the PI subgroup. The studies listed all included cohorts composed largely of unrecruited and/or physician referred patients treated in primary or tertiary treatment settings.

Considered collectively, these studies confirm impressions derived from the efficacy studies and suggest that CBT is highly effective with "real-world" patients across a range of important outcome measures. Specifically, these studies confirm that CBT results in important improvements in subjective measures of sleep including sleep onset latency, wake time after sleep onset, sleep efficiency, and total sleep time. A subset of these studies (Edinger & Sampson, 2003; Espie, Inglis, Tessier, & Harvey, 2001) show that clinic patients treated with CBT manifest larger improvement in these sleep measures than do either untreated patients or patients treated with an alternate behavioral approach. The cumulative findings of the studies listed also suggest that CBT enhances sleep-related self-efficacy, reduces depressive and anxiety symptoms, corrects dysfunctional beliefs about sleep, reduces use of sleep medications, and leads to improvements in insomnia-related sleep/wake symptoms. Given such findings, CBT can be considered a highly effective treatment for the sleep and related complaints of the PI patients encountered in clinical practice.

## 6. Treatment implementation

Although current data support both the efficacy and effectiveness of CBT for PI, a number of factors impact the successful implementation of this treatment. The following sections discuss these factors, which include methods of delivering CBT, treatment dosage, cost-effectiveness, and accessibility.

### *6.1. Treatment delivery*

CBT for insomnia was developed to be provided to patients in individual sessions (Morin, 1993). In efforts to improve cost-effectiveness and increase accessibility, however, a number of alternative delivery methods have been investigated. Studies employing a group CBT format of 6 to 8 sessions (4 to

Table 4
CBT treatment effectiveness studies conducted with primary insomnia patients

| Citation | *N* | Mean age (years) | Study design/ Patient types | Treatment duration (weeks) | Treatment format | Follow-up period (months) | Findings |
|---|---|---|---|---|---|---|---|
| Morin, Stone, McDonald & Jones, 1994 | 31 | 42.7 | Case series/PI patients from sleep center; 67.7% used sleep aid at study entry. | 14 | Individual | 24 | On average, PI patients showed > 50% reductions in SOL and WASO by treatment end whereas SE% increased from 70.2% to 84%.<br>Patients significantly reduced medication use from the beginning to the end of treatment.<br>Follow-up showed treatment gains maintained. |
| Perlis et al., 2000 | 47 | 39.2 | Case series/PI patients from sleep center; 53% comorbid Psychiatric or medical disorders. | 4 to 9 | Individual | N/A | 61% completed at least 4 sessions and showed global improvement in symptoms.<br>Patients showed a 65% reduction in SOL, a 46% reduction in number of nocturnal awakenings, a 48% reduction in WASO, and a 13% increase in TST. |
| Backhaus et al., 2001 | 20 | 43.0 | Single group/PI patients from sleep clinic; 60% on hypnotics | 6 | Group | 36 | Measures of SOL, TST, and SE% all improved.<br>By the 3-year follow-up SOL, TST, and SE% were improved by 26%, 28%, and 33%, respectively.<br>CBT reduced dysfunctional cognitions, depression, and anxiety. |
| Espie et al., 2001 | 139 | 51.4 | CBT vs. WL/PI patients from medical clinics; >50% took sleep medications. | 6 | Group | 12 | CBT produced significant reductions in SOL and WASO but WL did not. Within the CBT group, 64.2% has SOL values and 63.3% has WASO values <30 min by 12-month follow-up.<br>Significantly increased TST shown at follow-up for CBT group.<br>84% of hypnotic users were drug free at follow-up. |
| Edinger & Sampson, 2003 | 20 | 51.0 | CBT vs. SH/PI patients in primary care at VA hospital; 65% had comorbid medical or psychiatric diagnoses. | 2 | Individual | 3 | Abbreviated 2-session CBT superior to SH for improving subjective sleep measures and measures of sleep-related self-efficacy, insomnia symptoms, and dysfunctional beliefs about sleep.<br>52% of those receiving CBT reported at least a 50% reduction in their WASO.<br>55.6% of CBT-treated patients who entered the study with pathological scores on an Insomnia Symptom Questionnaire (ISQ), achieved normal ISQ scores by their final outcome assessment. |

Note: CBT=cognitive–behavioral therapy; SH=sleep hygiene therapy; WL=waiting list condition; SE%=sleep efficiency; SOL=sleep onset latency; WASO=wake time after sleep onset; TST=total sleep time. The Morin et al. (1994) study included 100 mixed insomnia patients but only findings for the 31 primary insomnia (PI) patients are considered here.

12 individuals per group) for PI have found significant improvements in subjective and objective sleep patterns (Backhaus, Hohagen, Voderholzer, & Riemann, 2001; Espie, Inglis, Tessier et al., 2001; Morin, Colecchi et al., 1999; Morin, Kowatch et al., 1993). Backhaus et al. (2001) reported that at a long-term follow-up of almost 3 years, individuals treated with group CBT had greater total sleep time and sleep efficiency than prior to treatment. Furthermore, group CBT was associated with reductions in depressed mood and negative cognitions such as focusing on and ruminating about sleep. Individuals treated with this approach have also reported greater satisfaction with and less distress about sleep compared to control groups (Morin, Colecchi et al., 1999; Morin, Kowatch et al., 1993). Although group CBT is a popular approach, studies directly comparing the relative benefits of individual versus group formats are lacking.

There have been some recent evaluations of CBT through self-help approaches such as books, audio/video tapes, telephone support, and the Internet. However, very limited data exist on the effectiveness of these approaches specifically with PI. Mimeault and Morin (1999) tested self-help CBT bibliotherapy with and without supportive phone consultations against a wait list control group. Compared to the control condition, both treatment groups showed substantial sleep improvements that were maintained at a 3-month follow-up. The addition of therapist guidance conferred some advantage over the bibliotherapy-only group at post-treatment, but these benefits disappeared by follow-up. Morin, Bastien, and Savard (2003) also report finding comparable effectiveness of CBT for PI provided in individual, group, and telephone consultation formats. Recently, Strom, Pettersson, and Andersson (2004) tested a 5-week self-help CBT program delivered via the Internet to individuals with PI. Although the treatment group demonstrated many sleep improvements at post-treatment, gains were obscured by the wait-list control group, which also showed improved sleep. The treatment group showed significantly less dysfunctional cognitions regarding sleep after treatment, whereas the control group did not improve on this measure.

### *6.2. Treatment dosage*

Little is known about the dose–response curves of CBT for PI. Despite ample models of such research in the pharmacological treatment literature, behavioral therapists have generally failed to explore what dose of CBT is needed to achieve minimally acceptable improvement. As such, no guidelines exist for how many treatment sessions and how many days or weeks in treatment are required to achieve the desired treatment outcomes. As few as two individual sessions of CBT have been shown to produce significant improvements in insomnia symptoms and subjective sleep (less wake time during the night, higher sleep efficiency, and improved sleep quality) compared to sleep hygiene treatment in primary care patients diagnosed with PI (Edinger & Sampson, 2003). Other preliminary data suggest that 4 individual sessions conducted at 2-week intervals may optimize clinical outcome for PI patients (Edinger, Wohlgemuth, Radtke, & Marsh, 2004).

### *6.3. Cost-effectiveness*

Demonstrating the cost-effectiveness of CBT for insomnia is crucial given the well-established capitation and managed care models of health care delivery. Pharmacologic interventions are currently the most widely used treatments for PI. While recent comparisons indicate that pharmacotherapy and behavior therapy produce similar short-term treatment outcomes in PI,

information regarding long-term outcomes is lacking (Smith et al., 2002). Chronic hypnotic use is associated with costs of medications in addition to repeated physician visits for medication prescriptions. Comparatively, CBT is time-consuming and expensive to administer at least initially. However, CBT may be more cost-effective since such treatment entails a short-term investment and is likely to result in long-term benefits. Furthermore, CBT addresses mechanisms that ostensively sustain PI whereas pharmacologic treatments represent a more symptom-focused treatment. As discussed above, alternative delivery methods such as group sessions, abbreviated individual sessions, and self-help interventions show promise for reducing costs associated with CBT implementation. Unfortunately, systematic cost-effectiveness and cost-benefit comparisons have not yet been conducted.

Another important economic consideration is the reduction in healthcare costs and utilization associated with CBT. PI may be a significant risk factor for serious psychiatric and medical disease (Riemann & Voderholzer, 2003). Since CBT produces long-term sleep improvements, it may reduce healthcare utilization and/or reduce risks for disease. Preliminary data with PI patients suggest that compared to pharmacotherapy, CBT is associated with less frequent use of healthcare services after treatment (Morin et al., 2003).

### *6.4. Treatment accessibility*

Despite the high prevalence of PI and the effectiveness of CBT, very few individuals with PI actually receive CBT, due to a number of barriers inhibiting access to treatment. First, there exists a general lack of knowledge on the part of the public and healthcare professionals regarding CBT for insomnia. Many chronic insomnia sufferers do not seek treatment, perhaps because they are unaware that successful treatments exist or because they believe that pharmacotherapy is the only option. In one United States population survey, only 5% of individuals with insomnia reported seeking help from their physician specifically for a sleep problem; instead, most are self-medicated with alcohol or non-prescription sleep aids (Ancoli-Israel & Roth, 1999). Insomnia sufferers usually do not mention sleep difficulties to their physicians, and physicians usually do not inquire about sleep problems (Hajak, 2000; Sateia, Doghramji, Hauri, & Morin, 2000; Shochat, Umphress, Israel, & Ancoli-Israel, 1999). Those individuals who do seek treatment for insomnia typically present initially to their primary care physician (Ancoli-Israel & Roth, 1999; Hajak, 2000). Unfortunately, these front line healthcare providers typically lack sufficient training and expertise to appropriately address PI (Sateia et al., 2000). Thus, patients with PI may be ineffectively treated if their physicians lack awareness about behavioral interventions such as CBT.

Another significant barrier to treatment is the lack of trained sleep specialists (Perlis, Smith, Cacialli, Nowakowski, & Orff, 2003). Considering the prevalence of PI, very few sleep training programs exist, and even fewer provide CBT training. Additionally, not all sleep disorders centers employ staff trained in CBT. In fact, it is quite unrealistic to expect that the current number of CBT-trained sleep specialists could possibly treat the significant proportion of the population that suffers from PI. Furthermore, in the United States, lack of insurance coverage for CBT may present a financial barrier to many patients (Perlis et al., 2003).

To increase accessibility of behavioral treatments for PI, some researchers have trained non-specialist healthcare professionals to provide these interventions. Espie, Inglis, Tessier et al. (2001) demonstrated that primary care nurses could effectively administer CBT to PI patients, and that sleep

improvements were maintained after 1 year. Similarly, Baillargeon, Demers, and Ladouceur (1998) reported success in training family physicians to administer SCT in primary care settings. Self-help approaches (described above) hold promise for providing affordable access to CBT. For example, treatment delivery via the Internet can be widely disseminated and is experienced by individuals as convenient (Strom et al., 2004).

## 7. Treatment acceptability and adherence

The ultimate success of CBT depends on patients' willingness, ability, and motivation to learn and implement behavioral changes at home. Patients must first accept CBT, finding it an attractive solution to their sleep difficulties. Secondly, patients must enact and adhere to CBT recommendations, transferring skills learned in the therapeutic environment to their daily lives. Historically, there has been a paucity of systematic research on how these factors affect the success of CBT for PI. However, as reflected in more recent literature, investigators are increasingly recognizing the importance of these factors (Bouchard, Bastien, & Morin, 2003; Harvey, Inglis, & Espie, 2002; Riedel & Lichstein, 2001; Vincent & Hameed, 2003; Vincent & Lionberg, 2001).

### *7.1. Acceptance of CBT*

Treatment preferences may affect patients' willingness to follow recommendations (Vincent & Lionberg, 2001). Studies that have investigated preferences for insomnia treatments have found that most insomnia sufferers rate CBT as more acceptable than long-term pharmacotherapy for sleep problems (Morin, Gaulier, Barry, & Kowatch, 1992; Vincent & Lionberg, 2001). Furthermore, patients are more satisfied with CBT and rate it as more effective compared to medications (Morin, Colecchi et al., 1999). Individuals with chronic insomnia not only prefer CBT to pharmacotherapy, but also expect that CBT produces greater improvements in daytime functioning, better long-term effects, and fewer negative side effects (Morin et al., 1992). Collectively, these data suggest that patients accept CBT as a viable treatment alternative for insomnia.

### *7.2. Adherence to CBT*

Because CBT requires considerable investment of time and effort in the home setting to realize desired levels of sleep improvement, adherence to treatment recommendations is important in establishing the overall success of CBT. At present, no standardized methodology for measuring adherence exists. Investigations measuring adherence to CBT with a variety of methods have appeared in recent literature. Two studies have reported adherence to group CBT for PI. In the first study, adherence was reported by participants and their significant others; both rated compliance as high (Morin, Colecchi et al., 1999). More recently, Vincent and Hameed (2003) measured adherence with therapist and spouse ratings, as well as sleep logs (to provide a measure of sleep schedule consistency). Therapists rated 48% of the sample as "very much" or "extremely" adherent to CBT components. Spouses reported that participants were most adherent with completing sleep logs, followed by changes to the bedtime routine, SRT, and relaxation practice. Higher adherence as rated by therapists was associated with better sleep quality, fewer dysfunctional beliefs regarding sleep, and

less sleep-related impairment at post-treatment. Interestingly, neither sleep schedule consistency nor attendance was related to outcome. Bouchard et al. (2003) also measured adherence from sleep logs of PI patients over 8 weeks of CBT. Seven daily criteria reflecting SCT and SRT components were rated. On average, participants complied with at least 6 criteria each day. Adherence increased over the course of treatment, but declined slightly after the last treatment session. Furthermore, perceptions of self-efficacy were positively related to adherence, suggesting that self-efficacy may provide another avenue through which to promote adherence behaviors.

Studies of adherence to components of CBT also have been undertaken. Harvey et al. (2002) asked patients to rate their home use of behavioral treatment components 1 year after treatment. Typical CBT components such as SRT, SCT, and cognitive restructuring were used by about 40% of participants. Reported adherence to SRT and SCT components emerged as the strongest predictor of clinical sleep improvements in sleep latency and nighttime wakefulness. Adherence to cognitive strategies predicted reductions in wakefulness. Use of sleep hygiene strategies, however, was unrelated to sleep outcome. Interestingly, Vincent and Lionberg (2001) reported that sleep hygiene was the most liked and used by participants, whereas SRT was the least liked and used. Adherence to SRT may be compromised because the recommendations are counterintuitive and often result in increased levels of sleepiness initially (Riedel & Lichstein, 2001). In a sample of older adults with primary insomnia, adherence to SRT recommendations was "reasonably good", although participants were spending almost 30 min longer in bed than prescribed (Riedel & Lichstein, 2001). In this study, consistency in the sleep schedule predicted sleep improvements at post-treatment, whereas the degree of bedtime reduction did not. Considered collectively, research on adherence to CBT is encouraging, but much more work in this area is needed to determine definitions of adherence as well as its impact on outcome.

## 8. Predictors and mediators of treatment outcome

As discussed above, CBT for PI has proven to be an efficacious and effective treatment. As such, the next step in promoting its successful application is to determine what factors influence and/or predict a positive outcome. This section reviews studies that have investigated these factors. Because CBT is a relatively new behavioral intervention, the information presented below comes from a handful of studies awaiting further replication and refinement.

### *8.1. Demographic variables*

To determine whether older adults' responsiveness to treatment differs from younger adults, some researchers have investigated the effect of age on CBT treatment outcome. Espie, Inglis, Tessier et al. (2001) found that at a long-term (1 year) follow-up of PI patients treated with CBT, older patients (≥60 years old) showed a greater reduction in wake time during the night compared to younger patients (≤44 years old). This finding suggests that older adults actually benefited more from treatment. However, when this sample was categorized into treatment responders and non-responders, patients who, at the end of treatment, had normative levels of wake time at night (≤30 min) were significantly younger than non-responders (Espie, Inglis, & Harvey, 2001). In both of these analyses, age did not predict improvements in other sleep variables such as sleep latency and total sleep time (Espie, Inglis, & Harvey, 2001; Espie,

Inglis, Tessier et al., 2001). In a study specific to older adults with PI, advancing age was associated with poorer sleep efficiency after CBT (Gagne & Morin, 2001). This finding suggests that among adults ≥55 years old, those at the older end of the spectrum show less treatment-related improvement in sleep efficiency. Interestingly, this finding emerged only when sleep efficiency was measured objectively by polysomnography (PSG) and not when measured by subjective self-report. Considered collectively, these preliminary studies suggest that age is not consistently related to CBT treatment response in individuals with PI. Studies of mixed insomnia samples have also found that age is not a significant predictor of CBT treatment response, indicating that older adults can benefit from CBT as much as their younger counterparts (Chambers & Alexander, 1992; Verbeek, Schreuder, & Declerck, 1999).

In general, other demographic variables such as gender, marital status, education, and occupational status, are not significantly related to CBT outcome (Espie, Inglis, & Harvey, 2001; Espie, Inglis, Tessier et al., 2001; Gagne & Morin, 2001). These findings are consistent with reports from other behavioral treatments for insomnia (Morin, Culbert et al., 1994; Verbeek et al., 1999).

### *8.2. Severity of insomnia*

Questionnaire ratings of greater insomnia severity at baseline are associated with poorer outcome on objectively (but not subjectively) measured sleep efficiency at post-treatment (Gagne & Morin, 2001). When insomnia severity is measured by self-reported sleep disturbance, individuals exhibiting greater insomnia severity at pre-treatment demonstrate larger sleep improvements (Espie, Inglis, & Harvey, 2001). However, these patients are less likely to achieve normative endpoint scores because their initial values are so high (Espie, Inglis, & Harvey, 2001). Analyses of insomnia duration and its impact on outcome have yielded inconsistent findings (Espie, Inglis, & Harvey, 2001; Espie, Inglis, Tessier et al., 2001; Gagne & Morin, 2001). The use of sleep medications, however, does not appear to affect successfulness of CBT (Espie, Inglis, & Harvey, 2001; Espie, Inglis, Tessier et al., 2001).

### *8.3. Medical and psychological factors*

Studies that have assessed medical and psychological predictors of outcome in PI, have, by definition, excluded patients with medical or psychiatric disorders that could be contributing to the sleep disturbance. Thus, it is not surprising that some studies have not found a relationship between treatment outcome and either medical history (Espie, Inglis, Tessier et al., 2001) or psychopathology (Espie, Inglis, Tessier et al., 2001; Gagne & Morin, 2001). One small study reported a perplexing finding that a higher number of active medical illnesses predicted improved sleep efficiency measured by sleep log but not by PSG (Gagne & Morin, 2001).

Compared to normal sleepers, individuals with insomnia score higher on personality measures of neuroticism, rumination, anxiety, and depressed mood (Coursey, Buchsbaum, & Frankel, 1975; Kales, Caldwell, Soldatos, Bixler, & Kales, 1983; Shealy, Lowe, & Ritzler, 1980). The effect of these personality characteristics on treatment outcome is unclear. Some data suggest that there are subgroups of PI patients that differ in their responsiveness to behavioral treatments (Bliwise, Friedman, Nekich, & Yesavage, 1995; Edinger, Stout, & Hoelscher, 1988). Furthermore, Espie, Inglis, and Harvey (2001) found that individuals with higher levels of pre-treatment depression and anxiety experienced greater sleep improvements. However, proneness toward worrying seemed to negatively affect certain outcome measures. Higher worry at pre-treatment was associated with poorer sleep quality after CBT; the authors

interpret this finding to suggest that worry may compromise sleep quality improvement but not sleep pattern improvement (Espie, Inglis, & Harvey, 2001).

### *8.4. Cognitive mediators*

As CBT distinguishes itself from other behavioral treatments by the inclusion of techniques designed to alter dysfunctional cognitions regarding sleep, it is important to determine not only whether CBT actually alters cognitions, but also whether cognitive changes are associated with treatment outcome. Edinger et al. (2001b) tested these questions directly in a sample of patients with PI. Indeed, patients treated with CBT had significantly greater reductions in dysfunctional beliefs about sleep compared to a relaxation treatment group and a placebo group. Furthermore, these reductions were correlated with pre- to post-treatment improvements in PSG sleep measures (reduced wake time at night, increased sleep efficiency) as well as a reduction in global insomnia symptoms. At a 6-month follow-up, reductions in dysfunctional cognitions were also associated with subjective patient ratings of improved restedness and sleep quality. Morin et al. (2002) replicated these findings in a sample of older adults with PI. They demonstrated that dysfunctional beliefs were reduced only for those individuals receiving CBT and not for those receiving sleep medication or placebo. Reductions in dysfunctional beliefs were associated with improved subjective sleep efficiencies at follow-up periods as long as 2 years. Espie, Inglis, and Harvey (2001) reported that stronger beliefs concerning the negative consequences of insomnia were associated with greater clinical improvements in sleep latency, suggesting that dysfunctional thinking may be a good clinical indicator for CBT. Lastly, more support for the positive outcome of altering dysfunctional sleep-related cognitions comes indirectly from a clinical study of mixed insomnia patients. Verbeek et al. (1999) found that treatment non-responders were less likely to have received cognitive therapy as part of their behavioral treatment package than treatment responders.

## 9. Conclusions

PI is a prevalent and significant health concern that is perpetuated by dysfunctional beliefs about sleep, heightened anxiety, and a host of sleep-disruptive compensatory practices. CBT addresses these perpetuating mechanisms and has emerged as a front line treatment choice for PI. CBT produces subjective and objective improvements in sleep and other clinical outcome measures that are durable over time. Furthermore, it is effective with clinical samples and easily adapted to the primary care setting. Although it requires a greater investment of patients' time and effort initially, it is well accepted and preferred over pharmacological approaches.

Further research will advance our understanding of the mechanisms underlying CBT and maximize its effectiveness with patients. Future studies are needed to determine the effects of CBT on outcome measures other than sleep, such as daytime functioning, and quality of life. Little is known about the effects of CBT on daytime fatigue, concentration difficulties, reduced work performance and compromised social functioning often reported by chronic insomnia sufferers. Other areas deserving greater scrutiny are factors that predict or moderate treatment outcome as well as methods to promote adherence to CBT. The optimal number of treatment sessions and mode of treatment delivery have yet to be determined; these findings will play an important role in efforts to disseminate CBT to the large population of individuals suffering from PI. Research demonstrating the cost-effectiveness of CBT and

the usefulness of this treatment for reducing or preventing long-term morbidity associated with chronic insomnia is critical to assure the continued vitality of CBT given the current emphasis on evidenced-based medical decision-making and cost-containment in healthcare delivery. In conclusion, CBT is a promising and relatively new treatment approach for PI that will likely continue to benefit from future refinement.

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