

New developments in cognitive behavioral therapy as the first-line treatment of insomnia

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Abstract: Insomnia is the most common sleep disorder. Psychological, behavioral, and biological factors are implicated in the development and maintenance of insomnia as a disorder, although the etiology of insomnia remains under investigation, as it is still not fully understood. Cognitive behavioral therapy for insomnia (CBTI) is a treatment for insomnia that is grounded in the science of behavior change, psychological theories, and the science of sleep. There is strong empirical evidence that CBTI is effective. Recognition of CBTI as the first-line treatment for chronic insomnia (National Institutes of Health consensus, British Medical Association) was based largely on evidence of its efficacy in primary insomnia. The aim of this article is to provide background information and review recent developments in CBTI, focusing on three domains: promising data on the use of CBTI when insomnia is experienced in the presence of comorbid conditions, new data on the use of CBTI as maintenance therapy, and emerging data on the delivery of CBTI through the use of technology and in primary care settings.

Keywords: insomnia, CBTI, nonpharmacological treatment

Introduction

Insomnia is the most common sleep disorder. It may present as the primary issue or in parallel with a coexisting medical or psychiatric condition. Psychological, behavioral, and biological factors are implicated in the development and maintenance of insomnia as a disorder, although the etiology of insomnia remains under investigation, as it is still not fully understood. Two types of treatment for insomnia disorder have received adequate empirical support: hypnotic medications and cognitive behavioral therapy for insomnia (CBTI). The aim of this article is to provide background information and review recent developments in CBTI, focusing on three domains: promising data on the use of CBTI when insomnia is experienced in the presence of comorbid conditions, new data on the use of CBTI as maintenance therapy, and emerging data on the delivery of CBTI through the use of technology and in primary care settings.

Overview of insomnia Insomnia as a diagnosis

Estimates of the prevalence of insomnia disorder in the US range between 6% and 10%.^{1,2} Insomnia disorder is characterized by night-time symptoms (difficulties initiating and/or maintaining sleep, or nonrestorative sleep) and daytime symptoms (distress and/or impairment in daytime functioning, such as difficulty with concentration, memory, fatigue, and/or mood). The diagnostic criteria also require that symptoms are present for at least 1 month.^{3,4} The word “insomnia” has been used interchangeably

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in the literature to refer to insomnia symptoms and insomnia disorder. To eliminate this ambiguity, sleep researchers have reserved the term “insomnia symptoms” to distinguish it from insomnia as a sleep disorder.⁵ We have adapted this distinction in the present manuscript. Insomnia can be classified by its duration: transient (less than a month), short term (between 1 month and 6 months), and chronic (more than 6 months).

Sleep specialists use the International Classification of Sleep Disorders (ICSD-II) as nosology. It includes general insomnia criteria and lists 10 types of insomnia: adjustment insomnia, psychophysiologic insomnia, paradoxical insomnia, idiopathic insomnia, inadequate sleep hygiene, insomnia due to mental disorder, insomnia due to drug or substance, insomnia due to medical condition, insomnia unspecified, and physiological insomnia.³ Mental health practitioners typically use the Diagnostic and Statistical Manual of Mental Disorders, which, at present (4th edition), includes criteria for primary insomnia or insomnia related/due to a substance or another medication, a psychiatric or medical condition, or another sleep disorder.⁴ The DSM V advisory committee on sleep nosology has proposed a single diagnosis of “insomnia disorder” that includes what was previously primary insomnia and insomnia related to medical or psychiatric conditions, and proposed the use of qualifiers to specify the presence of medical and/or psychiatric comorbidities.⁶

Insomnia development and maintenance

As previously stated, the etiology of insomnia remains under investigation, as it is still not well understood. There are various models of insomnia, none of which completely explains the etiology of insomnia disorder, as it is a complex and most likely multifactorial phenomenon. Insomnia can involve some level of physiological hyperarousal that can interfere with sleep initiation and/or maintenance. Studies have demonstrated a relationship between those with primary insomnia and those with physiologic factors such as increased whole-body metabolic activation, abnormal hormone secretion, variable heart rate, increased high-frequency electroencephalography activation, and sympathetic nervous system activation during sleep.^{7–11} It remains unclear whether physiological hyperarousal predisposes people to the development of insomnia disorder or whether physiological hyperarousal is a result of the disorder.

A cognitive model of the maintenance of insomnia was proposed by Harvey.¹² In this model, it is suggested that the initial development of acute insomnia can be due to a life stressor. When worry about the insomnia and daytime consequences related to not obtaining enough

sleep becomes present, this anxiety can trigger emotional distress and autonomic activation. As the anxiety related to sleep continues, selective attention to sleep-related threats, both internal (feelings of bodily sensations such as fatigue during the day, alertness when attempting to fall asleep) and external (watching the clock and calculating how much time is left to sleep, how long they were actually asleep), and inaccurate perceptions of daytime impairment (such as perceived work or school performance deficits) can occur. “Safety behaviors” can get put into place, such as canceling or avoiding daytime activities/obligations due to the sleep issue or drinking alcohol before bedtime to assist with sleep onset. This can lead to physiologic arousal, which may perpetuate the insomnia.¹²

Another model of the development and maintenance of insomnia was put forth by Spielman et al¹³ in 1987. They proposed a behavioral model of insomnia that identifies three factors: predisposing factors, precipitating events, and perpetuating attitudes and practices. Predisposing factors lower the threshold for the potential development of insomnia disorder and can include biological and psychological characteristics. Predispositions increase the risk for developing insomnia when precipitating events emerge. An example of a predisposition is a tendency for excessive worry, which is likely to increase basal sympathetic activation. A precipitating event is usually associated with distress. Examples of precipitating events include concern about job loss, illness, and death of a loved one. Around 75% of people with insomnia can identify what triggered the episode.¹⁴ A person’s response to the experience of poor sleep may perpetuate the problem. Responding to sleep disruptions with distress about sleep often leads to increased efforts to sleep and engagement in behaviors that, although they aim to facilitate sleep, lead to worsening of the sleep problem. For example, trying to improve sleep by staying in bed longer than before the insomnia developed rarely increases the actual amount of sleep obtained. Instead, it increases the time spent frustrated and tossing and turning and renders the bed a cue for sleeplessness, thus worsening and potentially prolonging the problem. If a person responds to poor sleep with distress, this can also lead to adopting maladaptive and inaccurate beliefs and cognitions that create increased anxiety, such as “performance anxiety”, anxiety about being able to sleep. Increases in anxiety in turn make it harder to sleep.

CBTI targets these and other compensatory behaviors and maladaptive cognitions. It includes behavioral components, specifically stimulus control instructions and sleep restriction therapy, cognitive therapy, relaxation and other

stress reduction techniques, and sleep hygiene education (see Table 1).^{15–21} CBTI is anchored in the science of behavior change, psychological theories, and the science of sleep.

CBTI as first-line treatment of insomnia

There is strong empirical evidence that CBTI is effective.²² CBTI demonstrates comparable efficacy with more durable long-term maintenance of gains after treatment discontinuation in randomized controlled trials of direct comparisons of CBTI with sleep medication.^{23–25} It has been proposed that skills are learned in CBTI that the patient can implement on their own long term beyond discontinuation of CBTI treatment, whereas medication use needs to continue in order to retain the benefit.

Meta-analyses have found robust effect sizes for the treatment components of CBTI.^{22,26–28} A meta-analysis of 23 randomized controlled trials, comparing studies that enrolled participants aged 55 years and older with studies that enrolled those, on average, younger than 55 years, found significant effects of behavioral interventions for sleep latency (adults Cohen's $d = -0.52$, older adults Cohen's $d = -0.51$), sleep quality (adults Cohen's $d = 0.89$, older adults Cohen's $d = 0.60$), wakefulness after sleep onset (adults Cohen's $d = -0.57$, older adults Cohen's $d = -0.73$), and sleep efficiency (adults Cohen's $d = 1.00$, older adults Cohen's $d = 0.38$) in both groups and for total sleep time in the younger cohort (adults Cohen's $d = 0.42$, older

adults Cohen's $d = -0.19$). The behavioral interventions appeared more effective in the younger cohort for sleep efficiency.²⁶ A meta-analysis of the short-term efficacy of pharmacotherapy (benzodiazepines or benzodiazepine receptor agonists) was compared with behavioral therapy (stimulus control and sleep restriction) for primary insomnia in 21 studies using prospective measures and within-subject designs. Comparable short-term outcomes were seen for both pharmacotherapy and behavioral therapy except in sleep latency where behavioral therapy revealed a great reduction in sleep latency. Post-treatment weighted-effect sizes included sleep latency (pharmacotherapy Cohen's $d = 0.45$, behavioral therapy Cohen's $d = 1.05$), sleep quality (pharmacotherapy Cohen's $d = 1.20$, behavioral therapy Cohen's $d = 1.44$), wakefulness after sleep onset (pharmacotherapy Cohen's $d = 0.89$, behavioral therapy Cohen's $d = 1.03$), and total sleep time (pharmacotherapy Cohen's $d = 0.84$, behavioral therapy Cohen's $d = 0.46$).²⁷

The American Academy of Sleep Medicine publishes practice parameter guidelines for sleep disorders.²⁹ These are accompanied by systematic reviews of the evidence supporting the recommendations. Two such reviews were conducted for insomnia and published by task forces commissioned by the American Academy of Sleep Medicine. The first was published in 1999 and reviewed 48 clinical trials, and the second, published in 2006, reviewed 37 additional treatment studies published since the publication of the first review. Both concluded that CBTI leads to significant improvements in the primary presenting sleep complaint (sleep initiation and/or maintenance) with sustained improvement seen for 6–24 months post-treatment.³⁰

Table 1 Description of cognitive behavioral therapy for insomnia components

Therapy component	Description
Stimulus control	Set of instructions aimed at breaking conditioned arousal and strengthening the bed and bedroom as stimuli for sleep
Sleep restriction	Limiting the time allowed in bed to the patient's average reported actual sleep time and subsequently slowly increasing the time allowed in bed as sleep improves
Cognitive therapy	Targets beliefs and thoughts that directly interfere with sleep by increasing arousal in bed or indirectly by interfering with adherence to stimulus control and sleep restriction
Relaxation techniques	Diaphragmatic breathing, progressive muscle relaxation, and visual imagery to reduce psychic and somatic anxiety related to sleep
Sleep hygiene education	Limiting caffeine intake, avoiding alcohol before bed, incorporating daily exercise, and keeping the bedroom quiet, dark, and at a comfortable temperature

CBTI treatment in the presence of comorbidities: recent developments

In the last decade, attention has been given to the application of CBTI when experienced in the context of medical and psychiatric comorbidities. Insomnia disorder has a high comorbidity rate with chronic medical conditions (ranges from 20% to 80%).^{31,32} The challenge in treating insomnia that is comorbid with medical conditions is that it is complicated by the direct impact of the comorbid disease and/or its treatment on sleep. For example, chronic pain and many HIV medications interfere with sleep. The comorbid medical disease may also hinder adherence to changes in behaviors that are introduced by CBTI. For example, pain and fatigue, which are common in many medical diseases, may make it difficult, and sometimes unsafe, to get out of bed when one is unable to sleep, a key recommendation of stimulus

control. Nonetheless, CBTI appears to be effective in treating insomnia in the context of cancer and chronic pain.^{33,34} Some adaptations for CBTI to the specific comorbid diseases have been used. For example, when treating insomnia in cancer and HIV patients, CBTI was augmented with counterfatigue measures such as scheduled short naps, exercise, and judicious use of caffeine.^{35–37}

Insomnia disorder also has a high comorbidity with psychiatric conditions, with estimated prevalence rates between 26% and 32%.^{38,39} Historically, it has been thought that sleep disturbances are caused by the psychiatric disorder and that when the parent disorder is treated, the sleep disturbance will resolve. There is recent data that suggest that the relationship between the psychiatric condition and sleep disturbances is more complex.^{40,41} The data also demonstrate that disturbed sleep can be a risk factor for the development of psychiatric disorders.⁴² The challenges in treating insomnia that is comorbid with psychiatric conditions is similar to the challenges in treating insomnia that is comorbid with medical conditions, in that the symptoms of the comorbid psychiatric disease and/or its treatment may have a direct impact on sleep and adherence to CBTI recommendations. Among the psychiatric disorders, depressive disorders and post-traumatic stress disorder (PTSD) have received the greatest attention. Uncontrolled and controlled pilot studies provide initial evidence that CBTI leads to significant improvements in sleep among patients with comorbid depressive disorders.^{43–45} The effects of CBTI in depression extend beyond sleep improvement and include improvement in depressive symptom severity.^{41,45,46} For example, a randomized controlled pilot study found that, among patients with comorbid insomnia and major depressive disorder (MDD), CBTI enhanced the antidepressant effects of escitalopram when concomitantly administered. This study reported remission of MDD in 61.5% of participants receiving a combination of escitalopram and CBTI, in comparison with those who received the medication combined with a control insomnia therapy (33.3%).⁴¹ This study also found a significantly greater remission of insomnia in association with CBTI (50%) than with a control insomnia therapy (7.7%). This is a clinically important finding because insomnia symptoms are the most common residual symptoms of antidepressant treatments that do not target sleep improvement, and patients who experience residual insomnia are at higher risk for relapse.^{40,47} This suggests that adding treatment for insomnia to standard antidepressant treatment in patients with insomnia may need to be considered in the clinical management of MDD.⁴⁷ It has yet to be systematically tested whether CBTI is a preventive measure for those with

insomnia disorder and who are at risk for the development of depression.

In those with PTSD, disturbed sleep is one of the most frequent symptoms.^{48,49} As is similarly found in MDD, despite remission of PTSD, disturbed sleep remains unresolved in 48% of patients treated with CBT for PTSD. This is noteworthy because hypervigilance and nightmares, two hallmark symptoms of PTSD, remain unresolved in only 33% of patients.⁵⁰ We found three studies that provide evidence that CBTI may be effective for insomnia that is comorbid with PTSD. One of the three studies targeted insomnia in veterans with comorbid PTSD or other psychiatric or medical comorbidities, and the other two included only patients who experienced insomnia comorbid with PTSD.^{51–53}

In two studies that focused on PTSD samples, CBTI was adapted to the specific needs of PTSD patients who commonly experience nightmares. Nightmares not only disrupt sleep but also may lead to anticipatory anxiety about experiencing nightmares.^{53,54} To address these issues, CBTI was combined with exposure to the nightmare content or imagery rehearsal therapy (IRT).⁵⁴ IRT suggests to patients that, over time, nightmares become a learned experience and recommends altering (rescripting) the content of dreams by activating the imagery system.^{55,56} Combined CBTI and nightmare exposure leads to large effect sizes for sleep efficiency (Cohen's $d = 1.01$), sleep onset latency (Cohen's $d = 0.89$), and nightmare distress (Cohen's $d = 1.14$).⁵⁴ Randomized controlled trials with larger samples are needed to confirm these promising preliminary findings.

Another adaptation of CBTI to PTSD was proposed by Haynes et al⁵⁷ but is not yet tested. Standard stimulus control guidelines dictate that insomnia sufferers should not get into bed until they are sleepy, that they get out of bed if they are unable to sleep, and that they return to bed only when they are sleepy. Haynes et al suggest that hypervigilance associated with PTSD may preclude the experience or awareness of feeling sleepy and therefore recommend altering stimulus control instructions by recommending a set bedtime, even if they are not sleepy. They also recommend that when patients follow the recommendation to get out of bed when they are unable to sleep, they should return to bed after a set amount of time (20 minutes), rather than wait until they feel sleepy.⁵⁷ Research is needed to test the proposed modifications to CBTI and combination of CBTI with nightmare therapies.

CBTI as maintenance therapy

To the best of our knowledge, only one study has examined the role of CBT as a maintenance therapy for insomnia.⁵⁸

This study had a complex design, consisting of essentially two separate randomized maintenance therapy trials. In one study, participants with persistent insomnia who completed six weekly sessions of group CBTI were randomized to receive six monthly sessions of individual CBTI as a maintenance therapy or 6 months of assessment only. Although continued therapy increased the remission rate from 44% to 57%, the improvement was similar among those who received no maintenance therapy (the observation only group), suggesting no added benefit of maintenance therapy among those who have previously received CBTI at the acute phase of treatment. This may be because CBTI has durable effects, as was previously documented, possibly because patients utilized skills learned in the acute phase of treatment.²³⁻²⁵

The second of the two maintenance studies randomized participants with persistent insomnia who completed 6 weeks of acute treatment with combined group CBTI and zolpidem (10 mg qhs) to receive either six monthly individual CBTI sessions plus medication (to be used as needed) or six monthly individual CBTI sessions with no additional medication.⁵⁸ Maintenance therapy with combined CBTI and zolpidem as needed increased remission from 44.4% at the end of the acute treatment phase to 59.8% at the end of the maintenance phase, as did maintenance therapy with CBTI alone (remission increased from 44.4% at the end of the acute phase to 56.9% at the end of the 6-month maintenance phase). This suggests no advantage for continued medication as needed over CBTI among those receiving combination treatment during the acute phase. Six-month follow-up data indicate that the best long-term trajectory was for those who received combined therapy during the acute phase and CBTI alone during the maintenance phase. Remission rates in this group continued to increase at 6-month follow-up to 68%, whereas the remission rate at the 6-month follow-up in the group who continued medication as needed dropped to 42%.

One interpretation of the results may be that hypnotic discontinuation while still receiving CBTI may have created an opportunity for patients to successfully handle transient worsening of sleep that often accompanies discontinuation. This experience might have increased self-efficacy regarding the ability to deal with disturbed sleep.

Expanding the mode of delivery: recent developments

Despite the existence of an effective and well-tolerated therapy for insomnia, the availability of CBTI to those who need it is limited by the small number of qualified behavioral sleep

medicine specialists and their restricted geographic region.⁵⁹ Increasing attention has been given to assessing the efficacy of CBTI when delivered in contexts that increase access and availability. These include Internet-based CBTI and delivery of CBTI by nurses in primary care.

Internet delivery

The evolution of the Web has brought about opportunities for behavioral medicine treatments to be delivered in an efficient manner. There are many advantages to the use of the Internet as a vehicle for treatment delivery, such as convenience for the patient, a decrease in expense, and accessibility. At the same time, it is important to acknowledge limitations related to Internet-delivered CBT, such as individualization of treatment to patients' presenting problems, a lack of provider support and guidance, and proper patient diagnosis and access to the appropriate online treatment.⁶⁰ Self-help CBTI has previously been found effective, as its focused and structured nature make it theoretically suitable for adaption to Internet delivery.⁶¹ The question that arises in delivering CBTI via the Internet is whether it is efficacious. This question was initially explored in a Swedish population in a randomized controlled trial of 109 participants who were randomized into a wait-list control group (assessment only) or a 5-week online CBTI intervention (sleep restriction, stimulus control, and cognitive restructuring). Participants receiving the CBTI intervention would read the treatment information each week, submit a sleep diary, and receive a new time in bed prescription calculated by an algorithm. The results revealed significant improvement in the treatment group on measures such as total sleep time, time awake in the middle of the night, and sleep efficiency, although the control group also showed improvement. Effect sizes between groups were low (Cohen's $d = -0.03$).⁶² Ritterband et al⁶³ have also started exploring this area. In a randomized pilot trial, 45 adults were randomized to a wait-list control (assessment only) group or to receive an Internet CBTI intervention, Sleep Healthy Using the Internet (SHUTi). The CBTI intervention included sleep restriction, stimulus control, cognitive restructuring, sleep hygiene education, and relapse prevention. The participants completed a daily sleep diary and based on that information received a new time in bed prescription weekly. Quizzes were used to test knowledge of the material presented, and vignettes were used to enhance engagement. The study revealed a significant reduction in Insomnia Severity Index in the CBTI Internet intervention and no significant change for the wait-list group. Improvements were also seen in decreased time awake after sleep onset and increased

self-reported sleep quality, and the gains were maintained at 6-month follow-up. The study suggests that an Internet-based approach is appealing to participants.⁶⁴ SHUTi is now being tested on a larger scale in a more representative sample that includes individuals with comorbid psychological or medical conditions.⁶⁵ These findings are also in line with an Internet randomized controlled trial of 118 participants randomized to wait-list control (assessment only) or a 5-week CBTi online intervention.⁶⁶ The results included significant improvement in insomnia severity, general fatigue, and sleep quality. Due to lower adherence to later modules in the active treatment group, the question of how to maintain engagement in online treatment was raised by the researchers as a future area for exploration.

Internet interventions may be a particularly attractive mode of delivery to the younger generation. In preparation for testing Internet delivery of a sleep intervention to college students, Trockel et al⁶⁷ tested email intervention that consisted of eight pdf files, each emailed weekly. The pdf files included sleep education, sleep restriction therapy with an emphasis on anchoring wake time, stimulus control instructions, relaxation and mindfulness training, and restructuring sleep-interfering beliefs and cognitions. The pilot study compared a sleep health intervention (Refresh), consisting of the same CBTi components as SHUTi, and an emotional health (Breathe) intervention, based on CBT for depression principles. The interventions were delivered in two separate freshmen dorms, to avoid cross-contamination of the interventions. The study revealed that the sleep health intervention was associated with greater improvements in sleep quality and depression severity than was the emotional health intervention among those with self-reported poor sleep quality (Pittsburgh Sleep Quality Index) at baseline.⁶⁸ Dropout rates among students with poor sleep quality were very low, suggesting a high level of acceptability. The results, if replicated in a larger sample and for Internet delivery, suggest that a self-guided CBTi may offer a nonstigmatizing approach for improving sleep and reducing depression in college students.

Stepped care model

Another approach to increasing access to CBTi that has been receiving increased attention is delivery by nurses in primary care settings where patients usually present with other comorbid conditions. Espie et al⁶⁹ first conducted a preliminary randomized controlled effectiveness study with 139 patients with insomnia in Scotland. The study tested the effectiveness of six sessions of group CBTi delivered by primary care nurses (health visitors) in a primary care setting.

The results revealed that, among completers, CBTi resulted in a substantial reduction of sleep latency and duration of wakefulness during the night, which was significantly greater than that observed in a self-monitoring control. However, effect sizes were smaller than those observed in efficacy studies of primary insomnia. These benefits were sustained a year later with the additional benefit of a significant increase in total sleep time. The intervention included support in withdrawal from hypnotics and a schedule of gradual tapering negotiated with the patient to maximize adherence, but reduction rate was not greater than one therapeutic dose per week. Of the 74 participants taking hypnotic medication at baseline, 76% were medication free post-treatment, and most remained medication free at 1-year follow-up. In a follow-up larger randomized controlled effectiveness study, this research group randomized 201 participants to five sessions of group CBTi or treatment as usual.⁷⁰ The results were similar. Effect size for reduction in sleep onset latency was moderate (Cohen's $d = 0.58$) and for minutes awake after sleep onset the effect size was small (Cohen's $d = 0.35$).

A more recent study of primary care delivery of CBTi focused on a different delivery model.⁷¹ Treatment consisted of brief behavioral therapy for insomnia. It included two in-person sessions delivered by mental health nurses and two telephone calls. The first session was 45–60 minutes in length and the second session was 30 minutes in length and scheduled 2 weeks later along with two telephone calls scheduled 1 and 3 weeks after the in-person sessions. The content of the sessions consisted of a brief behavioral therapy session, which included recommendations regarding reducing time in bed, keeping the wake time consistent, not getting into bed until sleepy, getting out of bed if not sleeping, and discouraging naps. The sample consisted of 79 older adults in the United States. The control therapy consisted of reading three brochures published by the American Academy of Sleep Medicine: *Insomnia*, *Sleep and Aging*, and *Sleep Hygiene*. The results also revealed a large effect size (Cohen's $d = 0.96$) for reduction in latency to sleep onset and a moderate effect size for minutes awake after sleep onset (Cohen's $d = 0.59$). The benefits were maintained at the 6-month follow-up.

Conclusions: place in therapy

Recognition of CBTi as the first-line treatment for chronic insomnia (National Institutes of Health consensus, British Medical Association) was based largely on evidence of its efficacy in primary insomnia.^{72,73} This manuscript has reviewed evidence that CBTi is effective in specific and mixed comorbid samples and when delivered in a primary care setting. We also

reviewed emerging data that Internet delivery of CBTI may be an effective method of delivery that is acceptable to patients and could provide access to care by patients without access to behavioral sleep medicine specialists. Future areas of research may include Internet-available CBTI for those with MDD or other comorbidities, such as those with chronic pain. Finally, we reviewed new evidence on the role of CBTI as a maintenance therapy for insomnia. When CBTI is delivered in combination with a hypnotic medication, long-term outcome is enhanced when medications are discontinued during maintenance therapy with CBTI. When CBTI is delivered alone, maintenance therapy may not be needed. Future research is needed to explore in what other situations maintenance CBTI is beneficial. These new directions of research inform the application of CBTI to “real-world” patients with medical and psychiatric comorbidities who may be taking hypnotic medications and to individuals with limited access to care.

Disclosure

The authors report no conflicts of interest in this work.

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