CHEST Postgraduate Education Corner
CONTEMPORARY REVIEWS IN SLEEP MEDICINE

Cognitive Behavioral Treatment of Insomnia

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Chronic insomnia (symptoms for ≥6 months) is the most common sleep disorder, affecting 6% to 10% of adults in the general population, with even higher rates in patients with comorbid conditions (eg, hypertension, 44%; cardiac disease, 44.1%; breathing problems, 41.5%). Traditionally, chronic insomnia occurring with another condition has been considered secondary and rarely received direct treatment because treatment of the primary condition was expected to improve the insomnia. However, this approach often failed because chronic insomnia is maintained by behaviors, cognitions, and associations that patients adopt as they attempt to cope with poor sleep but that end up backfiring (eg, increasing caffeine, spending more time in bed, trying harder to sleep). Cognitive behavioral treatment of insomnia (CBTi) targets those behaviors, cognitions, and associations and is effective across a variety of populations, including those with medical and psychologic comorbidities. Thus, in 2005, a National Institutes of Health expert consensus panel on chronic insomnia recommended dropping the term "secondary insomnia" in favor of the term "comorbid insomnia." Because CBTi does not carry the risks associated with some sleep medications (eg, dependency, polypharmacy, cognitive and psychomotor impairment), it is an attractive option for patients with other conditions. Through the Society of Behavioral Sleep Medicine (www.behavioralsleep.org) and the American Board of Sleep Medicine (www.absm.org), it is possible to find practitioners with expertise in CBTi (as well as other aspects of behavioral sleep medicine) and other behavioral sleep resources. Given the currently limited number of trained practitioners, exploration of alternative delivery methods (eg, briefer protocols, self-help, Internet) to improve access to this highly effective treatment and expanded training in these treatments are warranted.
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Abbreviations: CBTi = cognitive behavioral treatment of insomnia; ICD = implantable cardioverter-defibrillator; NIH = National Institutes of Health; SDB = sleep-disordered breathing

Insomnia is defined as complaints of difficulty initiating sleep, maintaining sleep, waking too early, and nonrestorative sleep despite adequate opportunity plus a complaint of impaired daytime functioning (eg, fatigue, depressed mood, poor concentration).[1] Acute insomnia lasts <4 weeks and can be linked to a specific cause,[1] whereas chronic insomnia lasts for at least 6 months and may not have an easily identifiable cause.[2] Instead, chronic insomnia is believed to be perpetuated over time by changes in behaviors, cognitions, and associations that patients make as they attempt to compensate for poor sleep. (See "Etiology" section for more details.) Comorbid insomnia accounts for the majority of chronic insomnia cases. Thus, this review focuses on comorbid chronic insomnia with special emphasis on medical comorbidities (hypertension, cardiac disease, and breathing disorders).[3,4]

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Prevalence

General estimates vary depending on the criteria used to define insomnia, and prevalence rates tend to decrease as the stringency of the criteria increases.[5] Thirty percent of adults have insomnia when defined as reporting at least one insomnia symptom.[6] When

daytime impairment or distress is a required criterion, prevalence drops to 10%.[1] When the most stringent diagnostic criteria are applied,[7] prevalence remains substantial, but further drops to about 6% of adults.[5,8] The few studies examining racial differences in prevalence have reported rates of 16.4% to 28.3% in whites, 15.3% to 23.7% in blacks, and 13.4% to 17.1% in Hispanics.[9] When separated into age categories, blacks appear to have a greater prevalence of insomnia in middle age (30-59 years), whereas whites have a greater prevalence of insomnia across the life span (Table 1).[10] Overall, sex, age, and health and mental conditions appear to be the most significant risk factors for insomnia.[1,15] Older age has been associated with increased risk of insomnia.[15,16] However, research suggests that age itself is not the risk; instead, the risk is related to inactivity, sleep changes, decreased social activities, and increases in health conditions associated with aging.[17,18] Women are at least two times more likely to have insomnia than age-matched men,[10] and an increased prevalence of insomnia has been seen in adolescent girls compared with age-matched boys (Table 1).[11] The presence of a health or mental condition increases the risk, with insomnia seen in 37.8% of individuals with a comorbid condition but in only 8.4% of those without a comorbid condition.[12] Although there are numerous epidemiologic studies of insomnia, criteria for insomnia classification are highly varied among these studies. Therefore, the summary of prevalence data in Table 1 relies heavily on data collected from a single epidemiologic study by Lichstein[10] in 2004. Some of the data generated by that study have appeared in the peer-reviewed literature,[12] but the bulk of findings to date have been published in Lichstein's book. Thus, it is important to note that the methodology used in Lichstein's study was highly rigorous and provides one of the best sources of information currently available on the prevalence of insomnia. In terms of methodology, Lichstein[10] used random-digit dialing to sample 50 men and women in each age decade from 20 to ≥ 80 years. Additionally, unlike the bulk of studies in this area, Lichstein's study used prospective data collection methods (2 weeks of sleep diaries) and stringent criteria for diagnosing insomnia.[10,12] The other studies in Table 1 were also selected because they used the strongest methodology and most stringent criteria available.

Etiology

Prior to a 2005 National Institutes of Health (NIH) state of the science conference, insomnia was defined as primary or secondary.[1,4] If insomnia symptoms were related to another physical or psychologic condition, that condition was considered the primary diagnosis and insomnia the secondary diagnosis.[4] Thus, treatment focused on the primary diagnosis, and it was assumed that successful treatment of the primary condition would resolve the secondary insomnia. Currently, no evidence supports this assumption, and some evidence suggests that treating the primary condition of depression does not result in remission of insomnia.[19]

Table 1—*Prevalence of Insomnia by Sex, Age, Race, Health Condition, and Mental Disorder*

	Prevalence of Insomnia, %				
Grouping	Men	Women	Blacks	Whites	All
Age					
Adolescents (13-16 y; n = 1,014)[11]	8.9	12.4	…	…	…
20-29 y (n = 772)[10]	6.0	12.0	2.4	9.8	…
30-39 y (n = 772)[10]	22.0	12.0	23.4	11.1	…
40-49 y (n = 772)[10]	11.0	20.0	20.6	12.7	…
50-59 y (n = 772)[10]	10.0	21.0	21.9	13.1	…
60-69 y (n = 772)[10]	9.0	17.0	11.1	13.9	…
70-79 y (n = 772)[10]	23.0	26.0	18.8	25.3	…
80-89+ y (n = 772)[10]	23.0	41.0	22.2	35.1	…
Health condition					
Hypertension (n = 772)[12]	…	…	…	…	44
Cancer (n = 772)[12]	…	…	…	…	41.4
Heart disease (n = 772)[12]	…	…	…	…	44.1
Diabetes (n = 772)[12]	…	…	…	…	47.4
Chronic pain (n = 772)[12]	…	…	…	…	48.6
Breathing problems (n = 772)[12]	…	…	…	…	59.6
Urinary problems (n = 772)[12]	…	…	…	…	41.5
GI problems (n = 772)[12]	…	…	…	…	55.4
Sleep-disordered breathing (n = 225)[13]	…	…	…	…	54.9
Neurologic disorders (n = 772)[12]	…	…	…	…	7.3
Mental disorder					
Depressive (n = 14,915)[14]	…	…	…	…	64.6
Anxiety (n = 14,915)[14]	…	…	…	…	43.6

However, there is a critical need for additional research on the effects of nonsleep interventions across the variety of presenting comorbid conditions. Prevalence studies have estimated that secondary insomnia accounts for up to 90% of insomnia cases.[15,20] The NIH conference recommended that the term "secondary" be dropped in favor of the term "comorbid." This recommendation was made for several reasons. First, when insomnia co-occurs with another condition, it can be difficult to establish which condition is causing the other. Additionally, the relationship between insomnia and some conditions is likely reciprocal in nature (eg, pain contributes to poor sleep and vice versa). Second, the factors that perpetuate insomnia over time often are different from those that precipitated it in the first place. For example, poor sleep may begin in response to cardiac disease but is maintained over time by the behaviors (increased caffeine use) and thought patterns (worrying about one's ability to get a good night's sleep) that patients develop in an attempt to cope with their sleep difficulties. Finally, when insomnia is conceptualized as secondary, it is unlikely to receive direct treatment. This is unfortunate because effective treatment of chronic insomnia often requires direct intervention to correct the behaviors, thought patterns, and associations that maintain it.

Spielman 3-P Model of Insomnia

The most commonly used model in understanding the development of chronic insomnia includes predisposing conditions, precipitating circumstances, and perpetuating factors (3-P model) (Fig 1).[21] In this theoretical model, predisposing conditions do not produce chronic insomnia but precede its onset and increase the likelihood for its occurrence. For example, anxiousness personality traits may result in hyperarousal, predisposing a person to sleep problems. Precipitating circumstances co-occur with the onset of acute insomnia (eg, stressful personal events, declines in health). According to this theory, insomnia is maintained by perpetuating factors, which include the changes in daytime behaviors, cognitions, or sleep/wake schedules that patients adopt to compensate for poor sleep. The development of chronic insomnia is conceptualized as related to a combination of predisposing (anxious personality type) and precipitating factors (eg, cardiac disease). However, over time, a positive feedback loop of perpetuating factors (eg, compensatory behaviors, cognitions) may develop that maintain or even exacerbate the sleep problem. Predisposing and precipitating factors of insomnia decline as the perpetuating factors exert a more direct impact on a nightly basis. These perpetuating factors are the targets of cognitive behavioral treatment of insomnia (CBTi), which, as will be discussed later, has extensive empirical support. Less well researched is the direct relationship of the 3-P model in the development and maintenance of insomnia.

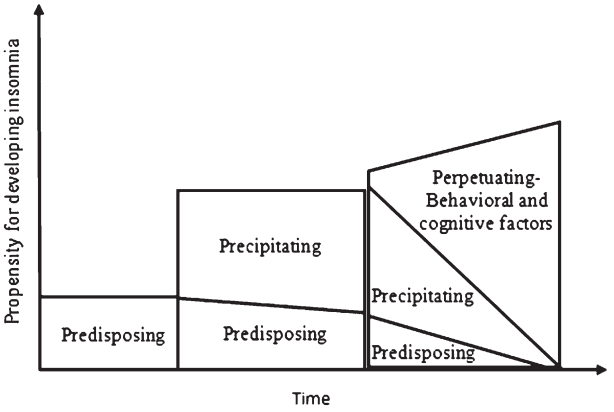

Figure 1. The 3-Ps model of the development of chronic insomnia.

Perpetuating Factors

Behaviors

Compensatory changes in behavior, such as daytime naps, excessive time in bed, and consuming stimulants, maintain insomnia over the long term.[22,23] Specifically, daytime naps may meet some of a patient's sleep needs and as a result, disrupt the natural daytime increase in the homeostatic sleep drive.[24] As a result, the patient's drive for sleep is weakened, and he or she may take longer to fall asleep that night. Stimulants (eg, caffeine, nicotine) act on the CNS and reduce sleep drive. Reduced daytime activity among patients with chronic illness[25] could lead to a number of potential consequences, including modification of the circadian rhythm due to reduced light exposure and increased opportunity for napping with increased time in bed. However, the role of daytime activity in insomnia needs more extensive research.

Associations

As patients experience sleep problems over time, they may begin to associate bed, bedtime, and the bedroom environment with difficulty falling asleep.[26] After repeated negative experiences, the bed and bedroom become cues for arousal rather than sleep. Spending an excessive amount of wakeful time in bed (during the night, while attempting to sleep in during the morning, or during the day) is a common response to poor sleep. Intuitively, this makes sense, but unfortunately, it may backfire because the additional wake time strengthens the association between the bed/bedroom and wakefulness, anxiety, and frustration.

Cognitions

Cognitive distortions related to sleep are related to elevated emotional arousal and worsening sleep problems.[27] Negative beliefs, attitudes, and interpretations related to sleep problems exacerbate and maintain insomnia by stimulating the sympathetic nervous system and increasing arousal. Worries about sleep, physical health, or other stressors may increase both cognitive and physiologic arousal.[28]

Chronic Comorbid Insomnia

As mentioned previously, chronic insomnia is commonly comorbid with other conditions. Comorbidity between chronic insomnia and depression or anxiety is high,[29] and insomnia is a risk factor for both.[30,31] Controlling for psychologic disorders and other sleep disorders, chronic insomnia is also highly comorbid with most major medical conditions (Table 1).[12] This review focuses on chronic insomnia comorbid with hypertension, cardiovascular events, and sleep-disordered breathing (SDB). The literature pertaining to insomnia in these specific medical conditions is limited but does indicate an increased rate of insomnia among these conditions when compared with the general population.

Hypertension and Cardiovascular Disease

Comorbidity between insomnia and hypertension ranges from 25% to 44%, depending on the measurement tools and definitions used.[12,32] Table 2 summarizes the data of recent epidemiologic studies on the interaction among hypertension, cardiovascular disease, and insomnia. Schwartz and colleagues[35] and, more recently, Spiegelhalder and colleagues[36] conducted reviews of the literature. Unfortunately, the studies to date have used a wide range of definitions from unvalidated questionnaires that assessed sleep complaint and not daytime complaints, which are necessary for an insomnia diagnosis. Thus, it is difficult to determine the true rates of insomnia among patients with hypertension and cardiovascular disease.

Consistently sleeping < 6 h a night is associated with an increased risk for hypertension.[37] Although some research suggests that insomnia does not increase mortality risk,[38] other data indicate that increased frequency of insomnia symptoms is associated with an increased risk for all-cause mortality and cardiovascular events.[39] Frequent difficulty in initiating and maintaining sleep may be related to increased risk for acute myocardial infarction and coronary cardiovascular disease-related death.[35,40] The biologic mechanisms for this link remain uncertain but may be related to increased sympathetic nervous system activity or increased inflammatory activity in the hypothalamic-pituitary-adrenal axis.[41,42] Patients with implantable cardioverter-defibrillator (ICD) devices also show clinically significant levels of sleep disruption.[34] Patients with ICDs are at an increased risk for clinically significant anxiety, which is related to hypervigilance, shock anxiety, and fear of device malfunction,[34,43,44] placing these patients at increased risk for insomnia.

Sleep-Disordered Breathing

SDB disorders are highly comorbid with insomnia, with prevalence estimates ranging from 22% to 54.9%.[45] As with cardiovascular disease, the wide range in prevalence rates of comorbid insomnia and SDB is likely related to the diversity of definitions and measurement tools used. For a review of the prevalence and interaction of comorbid insomnia and SDB, see Al-Jawder and BaHammam.[45] SDB is most often related to increased rates of sleep maintenance insomnia, and higher apnea-hypopnea indices are risk factors for sleep maintenance problems.[46,47] Older adults with insomnia, patients with long-term medication-resistant insomnia, and women with insomnia have elevated rates of comorbid SDB,[45,48] highlighting the importance of polysomnographic screening for patients with insomnia and symptoms of SDB. The overlap between the conditions may render identification of SBD in these patients more difficult for the nonsleep specialist. Despite a lack of empirical links between SDB and insomnia, possible mechanisms include increased nighttime awakenings due to nocturia, sleep fragmentation due to oxygen desaturation-related arousals, and decreased neck and throat muscle tone due to sleep deprivation.[45]

Assessment

The standard assessment of insomnia should include a thorough review of current insomnia symptoms, including nighttime complaints and daytime consequences (Table 3).[49] A sleep history should be taken to identify other conditions that may lead to poor sleep, such as psychologic factors, other sleep disorders, substance abuse, and current medications. For an example of a structured sleep history, see Morin and Espie.[53] Anxiety and depression are common in the context of chronic insomnia but do not necessarily preclude the separate treatment of insomnia symptoms because significant improvements in insomnia have been found without direct treatment of mood symptoms.[54] The use of sleep diaries is essential for documenting sleep/wake patterns and tailoring treatments to the patient's symptoms. Although assessment tools for documenting sleep quality and symptom severity exist, none are independently adequate for making a differential diagnosis.[49] The use of actigraphy

Table 2—*Prevalence and Symptoms of Insomnia in Hypertension and Cardiovascular Disease*

Authors/Year	Insomnia Definition	Measures	Sample	Results
Taylor et al[12]/2007	Research diagnostic criteria for chronic insomnia (consistent with DSM-IV criteria)	Sleep diary for 14 d; health survey	772 community-dwelling individuals in Tennessee contacted through stratified random sampling. Sampled at a rate of 50 men and 50 women for each decade of life from 20 to ≥ 80 y	Hypertension: 44% met criteria for insomnia; 43% of patients with insomnia had hypertension. Heart disease: 44% met criteria for insomnia; 22% of patients with insomnia had heart disease.
Redeker et al[33]/2010	Difficulty initiating and maintaining sleep or waking too early; frequency of problem often or almost always	Sleep Habits Questionnaire; Pittsburgh Sleep Quality Index	173 patients with stable heart failure from the northeastern United States. Mean age, 60.3 y; 65% men	Fifty-one percent of sample met insomnia definition (47% maintaining sleep, 42% falling asleep, 24% waking too early). No effect of demographic or medical characteristics was found.
Cross et al[34]/2010	Not defined	Actigraphy; self-report diary	30 patients with ICDs; 30 patients with CAD. Mean age, 66.9 y; 61.5% men	Both groups had an average of > 31 min of self-report sleep onset latency; patients with ICDs averaged > 31 min of self-report wake after sleep onset. Both groups had an average of > 31 min of actigraphic wake after sleep onset; patients with CAD averaged > 31 min on actigraphic sleep onset latency.
Budhiraja et al[32]/2011	DSM-IV criteria for insomnia	Self-report sleep questionnaire; polysomnography	3,247 community-dwelling adults in Michigan with a variety of comorbid medical conditions. Mean age, 41.7 y; 49.2% men	Hypertension: 24.7% met criteria for insomnia; 42.4% had a sleep efficiency < 25th percentile of healthy individuals. Heart disease: 26.8% met criteria for insomnia; 62.8% had a sleep efficiency < 25th percentile of healthy individuals.

CAD = coronary artery disease; DSM-IV = *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*; ICD = implantable cardioverter-defibrillator.

(wrist-worn activity monitoring) is not a standard assessment tool for insomnia but can be a useful complement to sleep diaries, particularly when circadian rhythm disorders are suspected.[55] Polysomnography and multiple sleep latency testing use electrophysiological measurement of neurologic and muscular activity and are standard assessments for SDB and narcolepsy. These tests are not recommended in the routine assessment of insomnia but should be considered when another sleep disorder is suspected or when treatment of the insomnia is unsuccessful.

Treatment Overview

Insomnia treatments fall into two categories: pharmacologic and cognitive behavioral. Medications typically are indicated for short-term use, although CBTi may be more beneficial for chronic insomnia. Although a range of classes of medications are indicated for the treatment of insomnia (eg, sedating antidepressants, melatonin, benzodiazepine receptor agonists), there is no single reliable pharmacologic method to treat insomnia[56]; however, hypnotic medications are the most commonly prescribed medication. Although effective for short-term use, some hypnotic medications can lose effectiveness over time while retaining the potential for dependence and rebound insomnia.[57] More recently formulated medications retain effectiveness over a longer period; however, these drugs can still lead to tolerance, physical and psychologic dependence, and hypnotic-dependent insomnia. Nevertheless, hypnotic medications should be

Table 3—*Insomnia Assessment*[49]

Category	Assessments
Recommended for all patients	Review of current nighttime symptoms and daytime complaints
	In-depth sleep history, related medical and psychologic conditions, and current medications
	Sleep diaries to determine daily patterns of sleep problems
Supplementary information	Sleep questionnaires (eg, Epworth Sleepiness Scale,[50] Pittsburgh Sleep Quality Index)
	Mood questionnaires (eg, Beck Depression Inventory,[51] State Trait Anxiety Inventory[52])
Not recommended for standard assessment of insomnia	Actigraphy
	Polysomnography (recommended when sleep-disordered breathing is suspected)
	Multiple sleep latency tests (recommended when narcolepsy is suspected)

considered for acute bouts of insomnia or when behavioral treatments are unsuccessful.

CBTi comprises several well-established therapeutic strategies that target a broad range of symptoms and etiologic factors,[56] including sleep education, sleep hygiene, stimulus control, sleep restriction, relaxation training, and cognitive therapy. The efficacy of CBTi is well documented (Table 4), and given that it offers several significant advantages over hypnotic medications, both researchers and clinicians, including the NIH, consider it to be a, if not the, first-line treatment of chronic insomnia.[4]

Older adults[58] and patients with chronic comorbid insomnia (eg, insomnia with concurrent SDB, cardiac disease, hypertension, or other breathing disorder) can significantly benefit from CBTi (Table 5). An added benefit of CBTi for these populations is that unlike medications, behavioral treatments do not contribute to polypharmacy and are unlikely to cause side effects or exacerbate the comorbid diagnosis. Additionally, remediating insomnia may result in improvements in the comorbid condition.

Table 4—*Average Effect Sizes Across 18 CBTi Trials*

Outcome	Effect Size
Self-report sleep onset latency	0.90[a]
Objective sleep onset latency	0.33[b]
Self-report wake after sleep onset	1.05[a]
Objective wake after sleep onset	0.46[b]
Self-report total sleep time	0.24[b]
Objective total sleep time	0.13
Subjective sleep efficiency	0.99[a]
Objective sleep efficiency	0.44[b]
Mood (depression, anxiety)	2.31[a]

CBTi = cognitive behavioral treatment of insomnia.
[a] Interpretation of large effect size using Cohen[8] standards.
[b] Interpretation of small effect size using Cohen[8] standards.

Cognitive Behavioral Treatments

Sleep Education

Education regarding normative sleep practices often is helpful. In patients with an associated medical or psychologic condition, information regarding sleep disturbances is important. For example, patients with ICDs and coronary artery disease may be informed that insomnia is a highly comorbid condition.[27] More general education would inform patients that sleep needs vary from person to person and from night to night and that taking ≤ 30 min to fall asleep or awakening for ≤ 30 min during the night is normal.[57]

Sleep Hygiene

Sleep hygiene aims to increase behaviors and environmental conditions that promote improved quality or quantity of sleep while reducing or eliminating practices that interfere with sleep (Table 6). For some behaviors, sleep hygiene recommendations may focus less on reducing and eliminating the behavior than on limiting those behaviors to certain times of the day (eg, avoiding liquids within 2 h of bedtime).[57] There may be individual differences in sensitivity and adherence to sleep hygiene rules that need to be tailored to individuals (eg, differential sensitivity to caffeine use). Although sleep hygiene is widely prescribed, the Standards Practice Committee of the American Academy of Sleep Medicine suggests that there is insufficient evidence to conclude that it should be used as a standalone treatment.[54]

Stimulus Control

Stimulus control therapy is based on learning theory and the underlying premise that sleep difficulties may be due to the bed and bedroom becoming conditioned cues for anxiety and arousal associated with unsuccessfully trying to fall asleep.[56] This technique aims to strengthen the association between the bed/bedroom and sleep and to weaken the associations between the bed/bedroom and arousing activities that interfere with sleep. There are six primary instructions for stimulus control (Table 7).[26] Stimulus control has been identified as a well-established treatment of insomnia,[54,64] and CBTi that includes it has been shown to be more effective in improving sleep onset latency, wake time after sleep onset, sleep efficiency (ratio of time spent asleep to time spent in bed), and slow-wave sleep than pharmacotherapy,[65,66] with improvements maintained at 1-year follow-up.[67]

Table 5—*CBTi Trials in Patients With Comorbid Illness*

Authors/Year	Study Design	Study Sample	Intervention	Outcome
SDB				
Krakow et al[59]/2004	Assessed sequential CBTi and SDB therapy in patients with concurrent chronic insomnia and SDB	17 patients (mean age, 42.9 y) with psychophysiologic insomnia who completed CBTi (but did not fully respond to therapy) and received a diagnosis of SDB after CBTi completion	Sequential CBTi and CPAP therapy (following CBTi, PSG was conducted to confirm SDB and titrate CPAP; SDB treatment of 3 mo)	Eight of 17 participants had nonclinical insomnia levels post-CBTi. Fifteen of 17 participants reached nonclinical insomnia levels post-SDB treatment.
Guilleminault et al[60]/2008	Evaluated subjective and objective outcomes of different sequences of two treatments for OSA: surgical intervention and CBTi	30 patients (mean age, 31.8 y [surgical intervention]; 30.9 y [CBTi]) with both insomnia and OSA	Participants randomized to one of two groups: (1) CBTi followed by surgical intervention or (2) surgical intervention followed by CBTi	Surgical intervention alone eliminated insomnia complaint in five of 15 patients; no patients receiving CBTi alone were remitted. Surgical intervention had greater initial improvements in subjective (sleepiness, fatigue), objective (TST, slow-wave sleep, WASO), and respiratory measures (AHI, RDI, Spo_2).
COPD				
Kapella et al[61]/2011	Evaluated effectiveness of CBTi in patients with comorbid insomnia and COPD	23 patients (mean age, 65 y [CBTi]; 60 y [WE]) with COPD and concurrent insomnia symptoms (difficulty either initiating or maintaining sleep)	Participants randomized to one of two groups: (1) six weekly CBTi sessions or (2) six weekly WE sessions	After CBTi, significant improvements were observed in subjective insomnia severity, global sleep quality, SE, WASO, and attitudes about sleep. Significant improvements in depressed mood were observed after WE.
CAD				
Rybarczyk et al[62]/2002	Compared effectiveness of group CBTi to self-guided audio relaxation in older adults with comorbid insomnia and chronic health conditions	35 older adults (mean age, 66.5 y [CBTi]; 65.6 y [home relaxation]; 71.4 y [control]) with comorbid chronic health conditions (either CAD, osteoarthritis, or type 2 diabetes mellitus)	Participants randomized to one of three groups: (1) eight weekly group CBTi sessions or (2) home-based audio relaxation program. Delayed treatment control (given home relaxation intervention later)	CBTi significantly improved compared with control on SE, WASO, TIB, global quality of sleep, and beliefs and attitudes about sleep at 4 mo follow-up. Home relaxation improved SE, WASO, and global sleep quality compared with control at 4-mo follow-up.
Rybarczyk et al[63]/2011	Evaluated two self-help methods of CBTi with older adults with comorbid insomnia and chronic health conditions, including CHD	106 older adults (mean age, 68 y) with insomnia and either CHD, osteoarthritis, or no health condition	Participants randomized to one of two groups: (1) CBTi book or (2) CBTi multimedia	Across both treatments, participants experienced moderate to significant clinical changes in sleep that were durable at 1-y follow-up.

AHI = apnea-hypopnea index; CHD = coronary heart disease; OSA = obstructive sleep apnea; PSG = polysomnography; RDI = respiratory disturbance index; SDB = sleep-disordered breathing; SE = sleep efficiency; Spo_2 = oxygen saturation as measured by pulse oximetry; TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; WE = wellness education. See Table 2 and 4 legends for expansion of other abbreviations.

Sleep Restriction and Sleep Compression

Sleep restriction and sleep compression both focus on reducing the amount of time a patient spends awake in bed during the night by prescribing a time in bed that more closely resembles the actual amount of time the patient spends sleeping. The primary distinction between these two approaches is how quickly the amount of time the patient currently spends in bed is reduced. Sleep restriction sharply reduces the

Table 6—*Sleep Hygiene Activities That Interfere With Sleep*[24]

Activity	Examples
Frequent napping	…
Variable bedtimes and wake times	…
Frequently spending an excessive length of time in bed	…
Use of stimulants near bedtime	Alcohol, tobacco, caffeine
Stimulating activities too close to bedtime	Working, talking about stressful topics, paying bills
Use of bed for nonsleep-related activities	Watching television, reading, snacking
Poor bedroom environment	Uncomfortable bed, too much light or noise, too cold or too warm
Allowing oneself to persist in sleep-preventing mental activities while in bed	Thinking, planning, reminiscing

amount of time spent in bed, whereas sleep compression takes a more gradual approach; Table 8 provides detailed instructions on how to apply both methods. Both approaches help patients to achieve a consolidated block of higher-quality sleep and work by (1) weakening the association between the bed/bedroom and being awake and (2) inducing partial sleep deprivation that builds sleep debt and activates the sleep drive,[56] facilitating more-rapid sleep onset, increased slow-wave sleep, and fewer awakenings during the night. Following successful consolidation of sleep time (measured by tracking sleep efficiency), time in bed can be gradually expanded to allow for greater sleep opportunity.

Sleep restriction has received greater research attention and is the technique most commonly used in CBTi protocols. Across various studies, sleep restriction alone has been shown to decrease sleep onset latency and wake time during the night as well as to increase total sleep time and sleep efficiency.[68,69] However, sleep restriction may not be appropriate for some patients. For example, sleep restriction is contraindicated for patients with a history of mania or seizures because sleep deprivation (even if it is only mild) increases the risk for episodes. Sleep compression may also be preferred where a drastic reduction in sleep or increase in sleep deprivation is not recommended (eg, frail older adults, patients with severe or multiple medical comorbidities, patients resistant to sleep restriction).[70]

Table 7—*Stimulus Control Instructions*[26]

Step	Instructions
1	Lie down to go to sleep only when you are sleepy.
2	Do not use the bed for anything except sleep and sex. Do not eat, read, watch television, or worry in bed.
3	If you cannot fall asleep within 10 min, get up and go to another room. Only return to bed when you feel sleepy again.
4	If you return to bed and still cannot fall asleep, repeat step 3. Do this as often as necessary throughout the night.
5	Set your alarm and get up at the same time every morning regardless of how much you slept during the night. This will help your body to acquire a constant sleep rhythm.
6	Do not nap during the day.

Relaxation

A variety of relaxation techniques can be used in the treatment of insomnia, including diaphragmatic breathing, biofeedback, imagery, and meditation. The goal of relaxation is to reduce the patient's levels of physiologic and cognitive arousal. One approach acknowledged as an empirically supported treatment by the American Academy of Sleep Medicine is progressive muscle relaxation. This method involves leading patients through a deep-breathing exercise followed by alternating tensing and relaxation of muscle groups (eg, arms, neck, back, legs) throughout the body. Patients are instructed to pay attention to the feelings of relaxation after the process compared with feelings of tension before and to practice this technique once during the day and before bedtime. Relaxation can also be integrated with stimulus control. Prior to leaving the bed during nighttime awakenings, patients can practice a relaxation technique (one time per awakening) to see whether it helps them to fall back to sleep. For patients with concomitant pain or joint disorders, a passive relaxation procedure that does not involve tensing and relaxing muscles, which could exacerbate their conditions, may be preferred.

Cognitive Therapy

Patients with insomnia frequently express attitudes and beliefs about sleep that can prompt worry and anxiety about one's ability to get the sleep he or she needs.[56,71] This anxiety and worry contributes to arousal that interferes with sleep, which prompts further anxiety and worry and sets up a self-fulfilling cycle that is difficult to break in the absence of direct intervention. Cognitive therapy focuses on identifying these beliefs and replacing them with more-adaptive beliefs and attitudes.[57,71] Sleep education (discussed previously) assists cognitive therapy because learning about normative sleep patterns can be helpful in addressing mistaken beliefs. Although cognitive therapy has not been evaluated as a singular therapy

Table 8—*Sleep Restriction and Sleep Compression Instructions*

Instructions	Details
Common	Sleep diaries: ask patients to complete daily sleep diaries starting 1-2 wk prior to initiating treatment and throughout treatment. At a minimum, the diaries should provide daily information on TIB, TST, TWT, and SE.
	Avoid daytime napping.
Technique specific	
Sleep restriction	Calculate average TIB and TST for the previous 1-2 wk.
	Determine initial TIB prescription, which is the average TIB during baseline + 30 min. (Do not go below 5.5 h.)
	Work with patient to set a regular bedtime and wake time consistent with the TIB prescription.
	Monitor TIB prescription during each treatment session and adjust as follows:
	If average SE > 90%, add 30 min to averaged reported TIB.
	If average SE < 85%, subtract 30 min from average reported TIB.
	If SE is between 85% and 90%, do not adjust.
	Do not go to bed before set bedtime. Get out of bed at the set wake time every morning.
	Avoid daytime napping.
Sleep compression	Calculate average TIB and TWT for the previous 1-2 wk.
	Divide TWT by number of proposed treatment sessions.
	Determine TIB prescription by reducing TIB slowly by the calculated increment above.
	Work with patient to set a regular bedtime and wake time consistent with the TIB prescription.
	Go to bed and get out of bed at set bedtime and wake time.

See Table 5 legend for expansion of abbreviations.

for insomnia, CBTi with a specific cognitive component has been shown to improve both objectively measured sleep and subjective sleep satisfaction[72] and to decrease dysfunctional sleep-related thoughts.[73]

Cognitive Behavioral Therapy for Insomnia

Given that multiple factors can contribute to insomnia, using a multicomponent approach such as CBTi enhances the likelihood that one or more of the treatment elements will target the factors contributing to a patient's poor sleep. CBTi has been shown to be more effective than stand-alone treatments[74] and to provide a more durable treatment response than medication.[75] There is considerable evidence for the effectiveness of CBTi across patient populations. For example, CBTi has been found to produce clinically significant improvement in insomnia symptoms in patients with breast cancer, chronic pain, fibromyalgia, and other medical comorbidities.[76-80] Additionally, behavioral interventions have been shown to be effective for middle-aged and older adults and older caregivers, populations in which comorbid medical conditions (eg, SDB, hypertension, cardiac disease) are more likely.[58]

Tailoring CBTi

For some populations, the standard CBTi recommendations should be tailored. For example, among caregivers for individuals with dementia, standard CBTi recommendations have been combined with stress management, linkage to community resources, management of problem behaviors in patients with dementia, and communication skills.[81] Caregivers of patients with dementia and Alzheimer disease have been trained in the application of sleep hygiene, sleep-wake scheduling, early morning bright light exposure, and exercise.[82] There is very limited literature pertaining to the treatment of insomnia in the context of comorbid medical disorders, and far more research is needed in this area. Some empirical evidence[59,60] suggests that concurrent medical treatment of SDB and psychologic treatment of chronic insomnia can result in clinically significant improvements in both conditions (Table 5). However, these two studies had relatively small samples sizes and lacked true control groups. When addressing insomnia comorbid with symptoms of another psychologic disorder, it may be appropriate to pursue adjunctive treatment of both conditions, but this remains to be thoroughly investigated. Sleep-related education can specifically address common symptom patterns. Within each patient population may be specific cognitions or emotions that disrupt sleep, and addressing these specific concerns is important in tailoring cognitive treatment. For example, among patients with ICDs, concerns about receiving a shock in the middle of the night may lead to increased emotional and physiologic arousal, which can interfere with sleep.[34]

Finding a Therapist

Presently, the number of patients who could benefit from behavioral sleep treatment far exceeds the current number of certified providers (160 in 2010). Of these certified providers, 8.6% are doctors of psychology, 26.5% are medical doctors, and 64.9% are doctors of philosophy (71 of 103 are confirmed psychologists).[83,84] Increasing the number of providers

Table 9—*Finding a Behavioral Sleep Medicine Therapist or Other Behavioral Specialist*

Organization	Website Address	Website Path
American Board of Sleep Medicine	www.absm.org	Behavioral Sleep Medicine Certification Exam, Certified Behavioral Sleep Medicine Specialists
American Psychological Association	www.apa.org	Psychology Help Center, Find a Psychologist
Association of Behavioral and Cognitive Therapies	www.abct.org	Find a Therapist
Society of Behavioral Sleep Medicine	www.behavioralsleep.org	Find a Behavioral Sleep Medicine Provider

is a major goal of the American Board of Sleep Medicine and the newly established Society of Behavioral Sleep Medicine. In the meantime, resources are available for providers who may not have sleep-specific expertise but who have expertise in related psychologic and cognitive behavioral therapies. Table 9 lists resources for locating a certified behavioral sleep specialist as well as other behavioral specialists who may provide services for patients with insomnia.

Future Directions

Given that most studies examine the discrete cognitive behavioral treatments as part of a comprehensive treatment package (ie, CBTi), conducting dismantling trials to evaluate the effectiveness of the specific components of CBTi is warranted. Increased efforts to determine the optimal combination, sequence, and duration of treatments are needed to provide improved care for specific conditions (eg, medical or psychologic problems, primary insomnia). Furthermore, because of the lack of trained specialists, alternative forms of behavioral treatment delivery should be explored, including developing briefer treatments, the use of self-guided treatments, or alternative methods of treatment delivery (eg, therapy sessions delivered through the Internet or by telephone).

Summary

Comorbid insomnia accounts for the majority of chronic insomnia cases. It often requires direct treatment to address the behaviors, cognitions, and associations that patients adopt to cope with poor sleep but which backfire (eg, caffeine use, spending more time in bed, trying harder to sleep). CBTi offers effective treatment of comorbid insomnia and represents a particularly attractive treatment option because it does not carry some of the risks associated with medications (eg, dependency, polypharmacy, cognitive and psychomotor impairment). Given the currently limited number of trained practitioners, alternative methods of delivery (eg, briefer protocols, Internet, videoconferencing) should be explored to improve access to this highly effective treatment.

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