Understanding and Treating Insomnia

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Annu. Rev. Clin. Psychol. 2011. 7:435–58

First published online as a Review in Advance on January 3, 2011

The *Annual Review of Clinical Psychology* is online at clinpsy.annualreviews.org

This article's doi:
10.1146/annurev.clinpsy.3.022806.091516

1548-5943/11/0427-0435$20.00

Keywords

sleep, cognitive-behavioral therapy, stimulus control, sleep restriction, mindfulness meditation

Abstract

Sleep disturbance is intricately entwined with our sense of well-being, health, emotion regulation, performance and productivity, memory and cognitive functioning, and social interaction. A longitudinal perspective underscores the conclusion that persistent sleep disturbance, insomnia, at any time during the life span from infancy to old age has a lasting impact. We examine how insomnia develops, the evidence for competing explanations for understanding insomnia, and the evidence about psychological and behavioral treatments that are used to reduce insomnia and change daytime consequences. There are new directions to expand access to treatment for those who have insomnia, and thus a critical analysis of pathways for dissemination is becoming increasingly important.

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INTRODUCTION

In the past decade, the importance of sleep and sleep disorders has been increasingly recognized. Sleep has been found to have broad, systemic effects on cognition, emotion, performance, and physical functioning. Consequently, insomnia and sleep disturbance are often comorbid with other disorders, including other sleep disorders (such as sleep apnea, narcolepsy, and sleep-wake circadian rhythm disorders), physical disorders (such as chronic pain and cancer), and psychological and psychiatric disorders (such as substance abuse, anxiety, and mood disorders).

In this review, we summarize the evidence for the consequences of sleep disturbance and how insomnia develops. We further review the current status of the psychological and cognitive-behavioral therapies that have been developed to treat insomnia, with attention to alternative methods of delivering treatment, and we discuss future directions for therapy.

CONSEQUENCES OF SLEEP DISTURBANCE

Strong interrelationships exist between sleep disturbance, daytime sleepiness, cognitive functioning, emotion regulation, social problems, and substance abuse. Sleep disturbance is so interrelated with overall functioning that it has been considered transdiagnostic (Harvey 2008); i.e., a core disturbance that increases the risk for other problems and disorders. Persistent sleep disturbance, at any particular point in our life span, starting in infancy, has been found to have effects that influence the likelihood of negative consequences for years to come.

Infants

Between 15% and 35% of infants have sleep disturbances. Many develop as a result of parental attempts to soothe and comfort infants who arouse during sleep. A mutually reinforcing cycle may develop in which infants are reinforced for crying during the night by parental attention, and parents are reinforced by a reduction in the infant's immediate crying and distress. In the long run, however, this cycle leads to increased sleep disturbance (Blampied & France 1993).

Other factors influence poor sleep in infants as well. The mother's depression during pregnancy and family disorganization predict infant sleep disturbance at 12 and 18 months of age, but not at 6 months of age (O'Connor et al.

2007). The delay in development of disturbed sleep suggests that some degree of maturation of the sleep-wake circadian rhythm is necessary before persistent disturbed sleep can be detected reliably. As infants mature and if disturbed sleep is a problem, parental training to improve sleep is often effective (Blampied & Bootzin 2011).

Toddlers

Naps are an important part of the toddler's daily schedule. In a study of emotional reactions to an unsolvable puzzle, 11 two- and three-year-olds were videotaped when playing with a puzzle both after being deprived of a nap and after having a nap. Infants showed more worry or anxiety (repetitive lip biting, fist clenching, and sucking on fingers) while completing the unsolvable puzzle after being deprived of a nap than after taking a nap (Berger et al. 2009). Inadequate or poorly timed sleep appears to result in insufficient emotional resources when toddlers face challenges. As seen below, if poor sleep persists, emotional and cognitive consequences can result.

A wide range of sleep problems is commonly seen in toddlers, including trouble sleeping, too little sleep, too much sleep, being overtired, nightmares, walking or talking in sleep, and wetting the bed. In a longitudinal study of 916 twins whose sleep was rated by parents annually from age 4 through 16 (except at ages 6 and 8), the sleep problems, listed above, decreased over time. About 70% of the children had one or more sleep problems at age 4, which decreased to about 33% at age 16. Those children whose number of sleep problems decreased the most over time had the best performance on cognitive executive functioning measures taken at age 17 (Friedman et al. 2009). This suggests that there are more negative consequences from persistent sleep problems than from a pattern of sleep problems that improves as the child matures.

In a long-term longitudinal study of a sample at risk for the development of substance abuse—sons of alcoholic men (Wong et al. 2004)—mothers rated their 3- to 5-year-old boys on sleep and overtiredness problems. Boys with sleep and overtiredness problems were more likely to have early onset of cigarette, alcohol, and other drug use a decade later, in early adolescence, than were boys without sleep problems. Sleep problems in toddlers also predicted the development of attention problems and anxiety or depression in adolescence.

Children

Sleep problems in childhood can persist and have effects years later. Persistent sleep problems at 5, 7, and 9 years of age predicted anxiety disorders at ages 21 and 26 in a longitudinal study of more than 900 children in New Zealand (Gregory et al. 2005). In a follow-up to the toddler study, daughters were recruited to participate when they were between the ages of 6 and 11. A majority of the eligible daughters joined the study. Analysis of the full sample, 292 boys and 94 girls, found that those with trouble sleeping in childhood were twice as likely to have the same sleep problems in adolescence (Wong et al. 2010). In addition, childhood overtiredness predicted poor response inhibition in adolescence and predicted alcohol use and alcohol-related problems in young adulthood.

There is also evidence for poor cognitive outcomes and behavior problems due to inadequate sleep in children. In a study of 135 second-, fourth-, and fifth-graders, those with fragmented sleep, defined by more night awakenings and lower sleep efficiency using actigraphy, had more parent-rated behavior problems and poorer cognitive functioning on a battery of cognitive assessments than those with undisturbed sleep (Sadeh et al. 2002). Evidence that insufficient sleep is related to academic and attention problems was also provided in a study of restricted and optimal sleep in children (Fallone et al. 2005). Seventy-four children between the ages of 6 and 12 participated in a three-week, within-subject, counterbalanced study during the school year. Children completed three experimental conditions: (*a*) their usual amount

Actigraphy: an objective sleep measurement process that uses an actigraph, which is about the size of a sports watch and is typically worn on the nondominant wrist, to detect movement and immobility. Specialized software translates movement and immobility into sleep parameters. Primarily used in research, although some sleep disorders centers utilize it as well

REM: rapid eye movement

of time in bed, (*b*) restriction to 8 hours a night in bed for those in second grade or lower and 6.5 hours a night for those in third grade or higher, and (*c*) "optimal" sleep of at least 10 hours in bed each night. Teachers were blind to the experimental condition of the school children. Restricting sleep led to significantly increased teacher ratings of academic and attention problems even though none of the children previously had a history of difficulty in school. For a review of the relationship between sleep and learning in children, see Gomez and colleagues (2011).

Adolescents

In recent years, the technology use in bedrooms at night, such as television, computer games, Internet social networks, cell phones, and instant messaging, has produced additional disruptions of sleep (e.g., Van den Bulck 2007). The increased arousal from gaming not only delays sleep, but also reduces slow-wave sleep, verbal memory performance (Dworak et al. 2007), and the amount of rapid eye movement (REM) sleep (Higuchi et al. 2005).

Many teenagers have a sleep-wake circadian rhythm problem, delayed sleep phase disorder, in which the individual's biological time to fall asleep occurs later, and thus is delayed, in comparison with the time the individual tries to go to sleep. Teenagers who have a delayed sleep phase have difficulty both falling asleep at night and waking up in the morning to go to school (Lack et al. 2009). They are likely to exhibit excessive daytime sleepiness, particularly in the morning. Excessive daytime sleepiness produces increased vulnerabilities to a number of poor outcomes (Carskadon 1990, Wolfson & Carskadon 1998), including academic performance failures, increased irritability, depressive mood, and automobile accidents.

Further evidence of the effect of sleep on cognitive performance has been seen in high school students in sleep extension studies. Nineteen high school students who extended their sleep at least 60 minutes on three consecutive nights showed reduced daytime sleepiness, improved backward digit span, and improved trail-making B (a cognitive task in which students draw lines connecting letters with numbers in order) in comparison with controls who did not extend their sleep (Cousins 2008).

The effects of sleep disturbance on health and interpersonal and psychological functioning were the focus of a one-year longitudinal study of more than 3,000 adolescents sampled from managed care rosters. Roberts and colleagues (2002) found that insomnia at time one predicted measures of life satisfaction, depression, self-esteem, and social support at time two. These predictions were reduced when insomnia at time two was controlled, indicating that persisting insomnia had an important role in maintaining problems in functioning. Thus, the treatment of sleep disorders may be of benefit, both as prevention and as treatment for interpersonal and emotional problems (e.g., Haynes et al. 2006).

Adults

Disturbed sleep in adults has both immediate and longer-term consequences in many domains of functioning. Disturbed sleep results in daytime impairments on memory, attention, alertness, and performance, and it has bidirectional causal effects on a large number of medical and psychiatric disorders (Rosekind & Gregory 2010).

Particularly noteworthy effects have been found between sleep and depression. In an epidemiological study from Zurich, Switzerland, participants were interviewed six times over the course of 20 years. Those who experienced two weeks or more of insomnia were at higher risk for later developing depression (Buysse et al. 2008).

In an earlier epidemiological study, the odds of new cases of depression were 3.95 for those assessed three years earlier with insomnia and 2.91 for those assessed earlier with hypersomnia compared to those with no sleep disturbance (Breslau et al. 1996). This suggests that both too little and too much sleep can lead to negative consequences. U-shaped curves were

found between sleep duration and mortality in a large population study that found that the lowest mortality risk was for those who reported sleeping seven hours a night (Kripke et al. 2002). Thus, more and less than seven hours of sleep a night increased risk for earlier death, even after a number of other variables were controlled. Similar curvilinear relationships have been found for sleep duration and health problems including heart disease, stroke, hypertension, diabetes, obesity, metabolic syndrome, and depression (Youngstedt & Kripke 2004).

Older Adults

Characteristics of sleep change with age throughout adulthood. The Sleep Heart Health Study assessed 5,407 community-dwelling adults older than 39 years of age and found different patterns of aging and sleep for men and women (Unruh et al. 2008). When sleep was measured objectively with polysomnography, men and women had more frequent awakenings and more wake after sleep onset as they aged, but only men showed linear decreases in the percentage of slow wave sleep and increases in the percentage of stage 1 sleep. The different patterns of findings for men and women were maintained even after the data were adjusted for the presence of sleep apnea and chronic health conditions. When sleep was measured subjectively, women, but not men, reported increasing sleep onset latencies and increasing difficulty falling sleep as they aged (Unruh et al. 2008). As is discussed later in the section on diagnosis, women are more likely to have complaints of insomnia than are men.

Estimates of the prevalence of insomnia among those over 65 from a large epidemiological survey of more than 9,000 participants found that more than 50% had insomnia, with most participants having difficulty maintaining sleep (Foley et al. 1995). Although these figures indicate a high prevalence of sleep problems in the elderly, comorbid medical and mental health problems contribute substantially to these figures, with the result that the prevalence of insomnia in healthy older adults is low (Ancoli-Israel 2009, Bliwise 2005, Vitiello 2009). A meta-analysis of sleep architecture across the entire life span found that most changes in adult sleep occur before age 60 and that after age 60 sleep changes only gradually due to age (Ohayon et al. 2004).

The consequences of poor sleep in older adults include poor health and cognitive impairment as seen in other age groups, but also increased risk for falls and mortality (Ancoli-Israel 2009). To study falls and fractures, Stone and colleagues (2008) had 3,000 community-dwelling women over 70 years of age wear an Actiwatch activity monitor for five days to obtain measures of sleep. Self-reported falls and fractures were recorded during the following year. Even after controlling for age, medical condition, and medication, women who had total sleep times of less than seven hours a night or sleep efficiency of 65% or less had a 30% to 40% increased risk for falls and fractures during the following year.

As previously mentioned, risk for mortality has been found to be increased with both short and long sleep times (Youngstedt & Kripke 2004). In a study of 184 community-dwelling older adults drawn from eight different protocols, individuals with polysomnographically measured sleep were followed up an average of 12.8 years later to determine whether they were deceased or living (Dew et al. 2003). Three background variables were related to mortality—age at entry to the original study, sex (male), and medical burden. After controlling for these variables, individuals with sleep onset latency >30 minutes, sleep efficiency less than 80%, and extreme high and low values of percentage of REM sleep had increased risk of mortality of 2.14, 1.93, and 1.71, respectively. Neither time in bed nor total sleep time increased risk for mortality. Thus, poor sleep, not short sleep, increased risk for earlier mortality.

In this section on the consequences of sleep disturbance, many of the findings come from large epidemiological studies. Although these results are intriguing and generally consistent in showing that sleep disturbance predicts

subsequent negative consequences, a major disadvantage is that the measurement of sleep is often a retrospective, single-item self report. Use of both subjective and objective assessments of sleep and sleep disturbance is desirable. The studies that experimentally evaluate sleep restriction and sleep extension are welcome additions to the literature because they permit direct assessment of the hypotheses of the effects of sleep on cognitive, interpersonal, and emotional functioning.

WHAT CAUSES INSOMNIA?

The most widely accepted overarching framework for the development and maintenance of chronic insomnia describes predisposing conditions, precipitating circumstances, and perpetuating factors (the 3-P model) as proposed by Spielman (Spielman 1986, Spielman & Glovinsky 1991) and further elaborated by Morin (1993). Predisposing conditions or traits such as hyperarousal, or familial or genetic tendency may play a role in increasing one's vulnerability to develop insomnia. Continuing advances in the genetics of sleep disorders (Taheri & Mignot 2002) show promise for enhancing our understanding of circadian rhythm disorders, narcolepsy, and sleep apnea. They may also expand our knowledge of predispositions, vulnerabilities, and invulnerabilities for developing insomnia.

One presumed vulnerability factor that has received considerable attention by researchers is hyperarousal, particularly cognitive hyperarousal (e.g., Bonnet & Arand 1995, Morin 1993, Perlis et al. 1997). Those with insomnia have been found to be more cognitively aroused and less sleepy in the bedroom before sleep than normal sleepers (Robertson et al. 2007). Evidence from functional neuroimaging has found that those with insomnia show a pattern of increased activation during sleep from subcortical brain areas but decreased prefrontal cortical activation during wakefulness (Nofzinger et al. 2004). That is, the brains of those with insomnia are too active during sleep and not active enough during wake. This matches the complaints of insomnia at night and fatigue during the day. Daytime fatigue may result from the lack of sleep and incomplete restoration of the prefrontal cortex the night before (Nofzinger 2005). Importantly, decreased prefrontal cortical activation during wakefulness in those with insomnia is reversible. Multicomponent, nonpharmacological treatment of insomnia has been found to increase prefrontal activation during daytime cognitive performance (Altena et al. 2008).

Espie (2002) considers hyperarousal to be an incomplete explanation for the central mechanism for developing insomnia. He proposes instead that falling asleep is an automatic process, one that can be disrupted and inhibited by the many variables discussed in this section. Thus hyperarousal and variables to be discussed as precipitating and maintaining insomnia are mechanisms that inhibit the normal automatic processes contributing to good sleep. Espie proposes that effective treatment re-establishes the normal mechanisms of sleep and wake.

Beyond predisposing conditions, precipitating factors such as illness, family, work and school, and other stressors may adversely influence a person's sleep (Bastien et al. 2004). An individual's initial response to sleep difficulty, in the form of worry and rumination about insomnia and its consequences, can determine whether an acute problem becomes a chronic condition (Bélanger et al. 2006). As the insomnia continues, individuals experience behavioral and cognitive responses that, with time, become maladaptive and feed a vicious cycle of insomnia. These factors can lead to increased arousal.

Maladaptive sleep habits (e.g., extended time in bed, irregular sleep-wake schedules, irregular napping, sleep-incompatible activities in bed), dysfunctional cognitions (e.g., worry, unrealistic expectations, misattributions), and arousal (physiologic, emotional, and cognitive) are the perpetuating or maintaining factors that become the targets of treatment (Bootzin et al. 1996).

In addition to maladaptive sleep behaviors, dysfunctional cognitions, and increased

arousal, the interaction of circadian and sleep homeostatic processes plays a role in insomnia and its treatment. Together, these processes generate sleep and wake timing (Achermann 2004, Borbely 1994). Homeostatic mechanisms work to balance sleep and wake states and are driven by the duration of wakefulness. With increasing wakefulness or sleep debt, the drive to sleep is increased, and when sleep occurs, the drive is reduced. Therefore, under usual conditions, wakefulness should lead to the onset and maintenance of sleep throughout the night. Although daytime wakefulness in persons with insomnia appears to be normal, the transition to sleep is difficult, and sleep is fragmented. Thus, sleep homeostasis may be dysregulated (Pigeon & Perlis 2006). The circadian clock, located in the suprachiasmatic nucleus of the hypothalamus, regulates sleep and wakefulness, among other biological rhythms (Zee & Manthena 2007). Cues from the environment such as light and dark, regular schedules, meal times, and social interactions help to regulate circadian rhythms. Maladaptive sleep behaviors, developed in response to insomnia, may disrupt the environmental cues the circadian clock requires to keep sleep and wake regulated (Pigeon & Perlis 2006).

DIAGNOSIS AND PREVALENCE OF INSOMNIA

There is considerable overlap in the general statements of what constitutes insomnia in formal diagnostic systems, including the *International Classification of Sleep Disorders-2* (ICSD-2; Am. Acad. Sleep Med. 2005), the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; Am. Psychiatr. Assoc. 2000), and Research Diagnostic Criteria for Insomnia (Edinger et al. 2004). There is consensus that the primary symptoms of insomnia are complaints of difficulty initiating sleep, difficulty maintaining sleep, and/or sleep that is nonrestorative or poor in quality. Although these different symptoms are targets for treatment, they are not stable across the life span (Buysse 2008). Younger adults are more likely to have sleep onset problems, whereas older adults are more likely to have sleep maintenance problems.

To diagnose insomnia, complaints of daytime impairment are required in addition to those of sleep symptoms. The list of daytime consequences in ICSD-2 and Research Diagnostic Criteria for Insomnia includes fatigue/malaise; attention, concentration, or memory impairment; social/vocational dysfunction or poor school performance; mood disturbance/irritability; daytime sleepiness; motivation/energy/initiative reduction; proneness for errors/accidents at work or while driving; tension headaches and/or gastrointestinal symptoms in response to sleep loss; and concerns or worries about sleep (Am. Acad. Sleep Med. 2005, Edinger et al. 2004).

Duration criteria differ somewhat across diagnostic systems. A complaint of insomnia must persist for one month to receive the diagnosis of primary insomnia in the DSM-IV-TR (Am. Psychiatr. Assoc. 2000). The most common duration for diagnosis of chronic insomnia in research studies is six months or longer (Edinger et al. 2004). Different durations between patients are often an indication of the causes of the insomnia (Buysse 2008). Acute or short-term insomnia is often due to life events or acute illnesses. Chronic insomnia is more likely due to learned and psychological factors or to the relationship with comorbid physical and mental health disorders.

The most commonly used quantitative values on sleep variables to characterize insomnia (Edinger et al. 2004) are more than 30 minutes for sleep onset latency, more than 30 minutes of wake after sleep onset, and total sleep time of less than six hours. The most common nightly frequency for identifying insomnia in research studies is three nights or more per week.

Sleep and insomnia can be assessed in many ways including structured interviews, questionnaires, daily sleep diaries or logs, observer ratings, actigraphy, and polysomnography in a lab or at home. There are advantages and disadvantages for every method of assessment, and consequently multiple methods of assessment are desirable (Buysse et al. 2006).

Primary insomnia: occurs independently of medical, psychological, or psychiatric disorders

Polysomnography: an objective sleep measure conducted overnight in a sleep disorders center or research laboratory. Multiple physiologic parameters are measured to diagnose sleep disorders. In CBT-I research, polysomnography may be used at baseline, after treatment, and at follow-up phases of the study

The prevalence of insomnia depends upon whether symptoms or a diagnosis is required (Ohayon 2002). The average prevalence for having insomnia symptoms is 30% to 48% and decreases to 9% to 15% for those who also report daytime complaints. The prevalence for those who have a diagnosis of insomnia is 6%. There is also a difference in prevalence between women and men. Women are more likely than men to report having insomnia complaints at a ratio of about 1.4 to 1.0 (Ohayon 2002). In the Zurich study described previously, the prevalence of one month of insomnia for women and men was 27% and 12%, respectively, across all measurement occasions (Buysse et al. 2008). Persistence of insomnia also depends on whether symptoms or a diagnosis were present at baseline. In a three-year natural history study of adults with insomnia (Morin et al. 2009), those who had severe insomnia and qualified for a diagnosis were most likely to maintain their sleep problems. Those with some symptoms, but who lacked all the criteria to receive a diagnosis, were more likely to improve, but about half of those who remitted relapsed later.

TREATMENT OF INSOMNIA

Pharmacological Therapies

Although the focus of this review is on nonpharmacological therapies of insomnia, pharmacological treatment continues to be the most widely available form of treatment for insomnia (Rosekind & Gregory 2010). There are a wide variety of medications and over-the-counter substances that are sedating. The most commonly prescribed hypnotics affect the gamma-aminobutyric acid ($GABA_A$) neurotransmitter. These include benzodiazepines that are anxiolytic or hypnotic or both, and nonbenzodiazepines (including newer hypnotics, commonly called the "z" medications for zolpidem, zaleplon, zopiclone and eszopiclone, that—like benzodiazepines—selectively affect the $GABA_A$ neurotransmitter) (Bateson 2010). There is a substantial literature on the efficacy and side effects of the GABA medications (Krystal 2010, Roehrs & Roth 2010). Gaps in the literature include evidence that hypnotics improve measures of quality of life and daytime functioning in addition to improving sleep; and hypnotics have not been evaluated for use with children (Krystal 2010). Side effects are usually dose dependent, and thus the lowest effective dose should be used (Roehrs & Roth 2010). To reduce daytime impairment and sedation, it is important to be attentive to duration of action of the medication being prescribed. Hypnotics with a half-life of more than five hours are likely to produce daytime sedation beyond the sleep period (Roehrs & Roth 2010).

Included among the newer nonbenzodiazepine hypnotics are melatonin-agonists that affect the timing of sleep but do not produce the same feeling of sleepiness as do the GABA agonists (Zee & Reid 2010). Other medications prescribed off-label for insomnia because of their sedating properties are antidepressant, antipsychotic, and anticonvulsant medications. There are limited efficacy data for these medications for the treatment of insomnia, and all have substantial side effects (McCall 2010). In addition to prescription medication, many individuals use alcohol, antihistamines, and a variety of complementary and alternative medicine preparations to treat insomnia. Although there are endorsements for such products by those who use them, there are limited efficacy data available (Neubauer & Flaherty 2010).

The Elements of Cognitive-Behavioral Therapy for Insomnia

Psychological and behavioral treatments for insomnia cover a broad range of interventions. This section describes the elements of cognitive-behavioral treatment for insomnia and the evidence supporting their use individually and in combination.

In the 1970s and 1980s, relaxation, paradoxical intention, and stimulus control therapy were the commonly used treatments for insomnia. At the time of the American Academy of Sleep Medicine (AASM) 1999 review (Morin et al.

1999a), evidence for sleep restriction therapy and multicomponent treatment studies was limited, thereby relegating them to the status of probably efficacious. Currently, multicomponent treatment packages are popular and typically include stimulus control therapy, sleep restriction therapy, and sleep hygiene and education. Researchers have also added relaxation therapy and cognitive therapy to the package. Some cognitive-behavioral therapy treatments for insomnia (CBT-I) studies devote specific sessions to cognitive restructuring (e.g., Morin et al. 1999b), during which dysfunctional beliefs and attitudes are identified, challenged, and reframed. Other CBT-I studies include cognitive components in educational instruction that address misconceptions about sleep, sleep requirements, sleep and aging, sleep loss, sleep drive, and circadian rhythms (e.g., Edinger et al. 2001, Epstein & Dirksen 2007). Cognitive therapy has not received testing in a controlled trial as a single-component intervention for insomnia (Bélanger et al. 2006), but has been tested in an open clinical series (Harvey et al. 2007).

Stimulus Control Therapy

Proposed by Bootzin (1972, 1977), stimulus control therapy (SCT) was the first nonpharmacological treatment developed specifically for insomnia. SCT has become the gold standard against which new interventions are tested. SCT is based on an operant paradigm within which the bed and bedroom no longer function as discriminative stimuli for sleep (Blampied & Bootzin 2011, Bootzin & Nicassio 1978, Bootzin et al. 2010). For the person with insomnia, the bed and bedroom become associated with behaviors that are incompatible with sleep such as watching television, eating, reviewing the day's events, planning, worrying, lying awake, and becoming anxious and frustrated from trying to fall asleep or fall back to sleep. There is also a Pavlovian conditioning component in the development of insomnia in that the bed and bedroom become conditioned stimuli for stress and frustration associated with being unable to fall asleep. The aims of SCT are to strengthen the bed and bedroom as cues for sleep, to weaken them as cues for behaviors that are incompatible with sleep, and to develop a consistent sleep-wake pattern.

CBT-I: cognitive-behavioral treatment for insomnia

SCT: stimulus control therapy

The stimulus control instructions are (Bootzin 1972, 1977):

1. Lie down to go to sleep only when you are sleepy.
2. Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward, when you intend to go to sleep.
3. If you find yourself unable to fall asleep, get up and go into another room. Stay up as long as you wish and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep immediately. Remember the goal is to associate your bed with falling asleep quickly! If you are in bed more than about 10 minutes without falling asleep and have not gotten up, you are not following this instruction.
4. If you still cannot fall asleep, repeat step 3. Do this as often as is necessary throughout the night.
5. Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will help your body acquire a consistent sleep rhythm.
6. Do not nap during the day.

Reserving the bed for sleep and sex helps to establish new sleep habits. Staying out of the bed and bedroom when unable to sleep decreases sleep anticipatory anxiety, dysfunctional sleep-related cognitions, and arousal (Morin & Epsie 2003). Getting out of bed during the night if unable to fall asleep is likely to increase the patient's sleep debt and homeostatic sleep drive. Following stimulus control instructions makes it more likely that patients will fall asleep quickly and stay asleep, thereby strengthening the bed and bedroom as discriminative stimuli

SRT: sleep restriction therapy

SHE: sleep hygiene and education

for sleep. Getting up at the same time each day impacts the circadian clock by helping to develop a regular sleep-wake schedule.

Detailed rationales for each of the stimulus control instructions are provided to patients (Bootzin & Epstein, 2000, Bootzin et al. 2010). Meta-analyses and systematic reviews (e.g., Morin et al. 1999a, 2006) indicate that SCT is one of the most effective, if not the most effective, single-component intervention for insomnia. Although SCT has received less testing as a single intervention in the past 10 years (e.g., Pallesen et al. 2003, Reidel et al. 1998), its inclusion in multicomponent interventions has increased (e.g., Lichstein et al. 2000; Morin et al. 1999b, 2004; Rybarczyk et al. 2002).

Sleep Restriction Therapy

Sleep restriction therapy (SRT) is a behavioral treatment for insomnia developed by Spielman and colleagues (1987). The theoretical underpinnings of SRT emphasize that individuals with insomnia spend too much time in bed attempting to sleep, which leads to increased wakefulness, fragmented sleep, and variability in the timing of sleep and wake. The aims of SRT are the consolidation of sleep and the establishment of a consistent sleep-wake schedule by limiting time spent in bed.

An individualized sleep-wake schedule is developed to limit the patient's amount of time in bed to the estimated mean time spent asleep. The nightly mean total sleep time is typically determined through two weeks of pretreatment sleep diaries. A wake time, which is followed throughout treatment, is agreed upon by the therapist and the patient. A bedtime is then established that gives the patient an amount of time in bed equivalent to the baseline mean total sleep time. For instance, if the mean total sleep time is 5.5 hours, and the patient agrees to awaken each day at 5:30 AM, then a bedtime is set for about 11:30 or midnight for the first week of treatment. During each week of treatment, the bedtime is advanced about 15 to 30 minutes based on the previous week's sleep. The prescribed time in bed is probably less than is actually needed since total time slept is often underestimated by persons with insomnia (Wohlgemuth & Edinger 2000). As a result, some partial sleep deprivation is induced, the homeostatic sleep drive is increased, and sleep consolidation occurs on subsequent nights. Patients develop a regular sleep-wake rhythm through consistent scheduling. Patients are not prescribed less than five hours of time in bed. Length of treatment is six weeks to obtain improvement (Rubenstein et al. 1990), although eight weeks have also been recommended (Spielman & Glovinsky 1991). Further details on the use of SRT are provided in Epstein & Bootzin (2002), Spielman and colleagues (2010), and Wohlgemuth & Edinger (2000). SRT has been used as a single treatment (e.g., Friedman et al. 2000) and included in multicomponent interventions (e.g., Edinger et al. 2001, Espie et al. 2001). Sleep compression is a modification of SRT that gradually reduces time in bed over the treatment period rather than the immediate reduction approach used in SRT. This more gentle variation has been used successfully with older adults (Lichstein et al. 2001, Reidel et al. 1995).

Sleep Hygiene and Education

Sleep hygiene and education (SHE) is composed of both information about sleep and a group of lifestyle recommendations under the label of sleep hygiene. Sleep education consists of knowledge about sleep processes and function, developmental changes in sleep, sleep homeostasis, circadian rhythms, and individual sleep needs (Bootzin et al. 1996, Lacks 1987). Sleep hygiene consists of rules to improve sleep, for example, put the bedroom clock where you can't see it and avoid coffee, nicotine, and alcohol (Hauri 1991). There is a lack of agreement among experts regarding a definition of sleep hygiene (Stepanski & Wyatt 2003), and the term is often used incorrectly by healthcare providers to refer to SCT (Edinger & Wohlgemuth 1999). Stepanski and Wyatt were unable to find any studies that used the same sleep hygiene

instructions. Although a core set of instructions was identified, the specific recommendations differed across studies.

SHE, particularly the education component, is a fundamental part of insomnia intervention (Morin et al. 1999a) that can lay the basis for patients' understanding of theory-based interventions such as SCT and SRT. As such, it is usually delivered at the beginning of the CBT intervention period. Although SHE has limited efficacy as a single-component intervention (Morin et al. 1999a), it is included in the recent surge of multicomponent intervention studies (e.g., Edinger & Sampson 2003, Espie et al. 2007, Morin et al. 2004, Reidel 2000).

Cognitive Therapy

There are two types of cognitive therapy for insomnia. Cognitive therapy developed by Morin (1993) and based on Beck's therapy for depression (Beck et al. 1979) is a cognitive restructuring approach that focuses on dysfunctional beliefs and attitudes about sleep. Harvey's (2005) approach targets cognitive processes that maintain insomnia such as monitoring for sleep-related threat, misperception of sleep and daytime deficits, and the use of safety behaviors that maintain unhelpful beliefs. Both therapies address dysfunctional beliefs about sleep, but the difference lies in the strategy used to accomplish the therapeutic goal.

Cognitive Restructuring

Cognitions play an important role in the development and maintenance of insomnia (Morin 1993). Dysfunctional cognitions surrounding insomnia (unrealistic sleep expectations, misperceptions about the causes of insomnia, distorted perceptions of insomnia's consequences, faulty beliefs about sleep-promoting practices, and other sleep disturbing thoughts) are the focus of cognitive restructuring (Bélanger et al. 2006). The patient's problem areas are identified through examining specific examples of trouble sleeping and the patient's response to the questionnaire Dysfunctional Beliefs and Attitudes about Sleep (Morin et al. 1993). The validity of the dysfunctional cognitions are challenged and reframed. Typically, cognitive therapy is delivered over several sessions. Multicomponent intervention studies for insomnia have included cognitive restructuring as part of the treatment package in a small-group format (e.g., Morin et al. 1999b, 2004).

CT-I: cognitive therapy for chronic insomnia

Cognitive Therapy for Insomnia

Based on prior theoretical work regarding cognitive processes in insomnia and success in the use of theory-driven cognitive therapy in other psychological disorders, Harvey developed a cognitive model (Harvey 2002) and therapy (Harvey 2005) for insomnia. The model of insomnia proposes cognitive processes that maintain insomnia and extends perpetuating factors to include daytime variables. The five processes that maintain insomnia are worry, selective attention to and monitoring for sleep-related threats, misperception of sleep and daytime deficits, unhelpful beliefs about sleep, and counterproductive safety behaviors. Cognitive therapy for chronic insomnia (CT-I) includes three phases. In the conceptualization phase of CT-I, the therapist and patient work together to develop an individualized cognitive model of daytime and nighttime problems that elucidate the vicious cycles experienced by the patient. The daytime and nighttime models are used in the intervention phase, where individualized behavior experiments are developed and used by the patient to directly test and reverse the insomnia-maintaining processes. The final phase of CT-I includes discoveries made during the behavioral experiments, identification of treatment gains, relapse prevention, and development of goals to sustain benefits.

An open trial of Harvey's approach (Harvey et al. 2007) with 19 patients provided individual treatment in from 6 to 22 sessions (average 14) and found significant improvement in sleep and daytime impairment that was maintained through a 12-month follow-up. Although these are encouraging results, there was no control group, and the large number of sessions required is in the opposite direction of societal

pressure to provide treatment gains more efficiently to reduce the cost of treatment.

MBSR: mindfulness-based stress reduction

Arousal Reduction Using Relaxation or Meditation

Relaxation training has a long history as a treatment for insomnia. If hyperarousal is a primary determinant of insomnia, it would be expected that methods to reduce arousal would be among the treatments to produce the most improvement. There are a number of different methods for reducing arousal, including progressive muscle relaxation, diaphragmatic breathing, autogenic training, electromyography biofeedback, meditation, yoga, and hypnosis (Bootzin & Rider 1997). Progressive muscle relaxation (Bernstein & Borkovec 1973, Jacobson 1938) is the most widely researched single psychological and behavioral treatment for insomnia (Lichstein & Riedel 1994). Relaxation has been designated as an effective treatment in practice guidelines (Morgenthaler et al. 2006). Nevertheless, relaxation has not been found to be as effective in producing improved sleep as a single treatment as other treatments described in this section (Morin et al. 2006). Multicomponent treatment that includes relaxation training has been found to be effective (e.g., Jacobs et al. 2004).

Mindfulness meditation is a recently added component in multicomponent treatments for insomnia. Mindfulness is defined as the awareness that arises out of intentionally attending in an open, accepting, and discerning way (Shapiro & Carlson 2009), and it involves formal meditation practices as well as principles for applying this awareness to one's moment-to-moment experience. It promises to be particularly effective at reducing mind-racing and ruminative worrying (Bootzin et al. 2010).

Bootzin & Stevens (2005) developed a manualized, small-group treatment to improve sleep, daytime sleepiness, and emotional distress in teens with substance-abuse treatment histories. The multicomponent treatment consisted of six 90-minute weekly small-group sessions, with the first session dedicated to sleep education and sessions 2–6 divided equally into segments for the cognitive-behavioral sleep intervention and a modified mindfulness-based stress reduction (MBSR) program. The multicomponent cognitive-behavioral sleep treatment consisted of SCT, emphasis on regularizing sleep-wake schedules across school and weekend days, the use of bright light therapy to advance circadian sleep-wake schedules, and cognitive therapy.

The multicomponent treatment program was successful in improving the sleep of adolescents who attended four or more of the six treatment sessions. The frequency of mindfulness meditation practice, but not duration of practice, was significantly related to improvement in total sleep time and to improvement in self-efficacy about sleep problems (Britton et al. 2010).

Mindfulness principles and practices have also been integrated with CBT-I for adults with insomnia (Ong et al. 2008, Ong & Sholtes 2010). In a treatment development study, 27 adults with insomnia completed a multicomponent group treatment that consisted of SCT, SRT, sleep education, sleep hygiene, and MBSR in an integrative framework. There were significant changes in both sleep measures and measures of arousal (Ong et al. 2008).

Similar to the results from the study of substance-abusing adolescents, frequency, but not duration of meditation practice, was significantly related to reductions in arousal. Both studies suggest that emphasis should be placed more on the frequency than the duration of mindfulness meditation practice. Furthermore, both studies indicate that mindfulness meditation may make a contribution to improvement of sleep problems and reduction in arousal in multicomponent treatment studies for insomnia (Bootzin et al. 2010).

EVIDENCE FOR PSYCHOLOGICAL AND BEHAVIORAL TREATMENTS

A strong evidence base supports the efficacy and durability of psychological and behavioral

treatments for insomnia. Reviews and meta-analyses have contributed to current knowledge and treatment approaches in the field. (e.g., Irwin et al. 2006, Morgenthaler et al. 2006, Morin et al. 2006, Pallesen et al. 1998).

The National Institutes of Health (NIH) State-of-the-Science Conference on the Manifestations and Management of Chronic Insomnia in Adults (Natl. Inst. Health 2005) presented a summary of the current knowledge regarding treatments for the management of chronic insomnia. The conference statement supports the use of pharmacological, behavioral, and cognitive-behavioral treatments for insomnia. Industry was called upon to help support comparisons of nonpharmacological and pharmacological treatment studies. The statement recognizes that most insomnia is comorbid and recommends the use of that term rather than "secondary insomnia," which may lead to undertreatment. An important area of the statement acknowledges the limited research on the consequences of insomnia, such as daytime functioning and quality of life, and calls for more studies addressing these variables.

Whereas the NIH State-of-the-Science statement is a summary of the current knowledge, a recent systematic review of psychological and behavioral interventions for chronic insomnia provides guidance regarding specific insomnia treatments (Morin et al. 2006). The 2006 review is an update of the evidence from an earlier review (Morin et al. 1999a). Both reviews were conducted by task forces commissioned by the AASM to develop practice parameters in the psychological and behavioral treatment of chronic insomnia. Similar to the 1999 review, the updated report found that stimulus control therapy, progressive muscle relaxation, and paradoxical intention met criteria for empirically validated treatments. Sleep restriction therapy and multicomponent cognitive-behavior therapy moved from probably efficacious to well-established intervention status. Psychological and behavioral treatment was found to maintain its effectiveness over time, although there are few studies with follow-up measurement greater than one year. Treatment was delivered mostly in individual sessions followed by group format and several alternative delivery methods (e.g., telephone, Internet; see later section on New Directions in Therapy). Treatment length averaged six weeks. In both reviews, the majority of studies used daily sleep diaries to measure sleep outcomes. Similar to the 2005 NIH State-of-the-Science Conference, both reviews stressed the need to examine clinically meaningful outcomes of daytime functioning and quality of life.

From 1999 to 2006, the AASM reviews showed an increase in treatment studies of older adults. This trend indicates a change from conventional beliefs that older adults cannot benefit from behavioral treatment compared to young and middle-aged adults. A meta-analysis by Irwin and colleagues (2006) examined the efficacy of 23 behavioral treatment studies on primary insomnia and the moderating effects of age cohort (less than 55 years and at least 55 years) and treatment type (CBT, relaxation-based treatment, and behavioral-only treatment). Medium to large effect sizes were obtained for sleep outcomes except for total sleep time. Improvement was seen in sleep onset latency, wake after sleep onset, sleep efficiency, and sleep quality. Sleep efficiency differed from the other sleep outcomes in response to treatment type. CBT and behavioral-only treatment showed significant effect sizes for improvement. Subjects in both age cohorts improved on sleep onset latency, wake after sleep onset, and sleep quality. The meta-analysis lends support to the use of psychological and behavioral interventions in older persons. Once again, the lack of outcomes addressing daytime performance was noted.

Another trend identified in the 2006 AASM review was the use of multicomponent treatment. The reason for the increase in multicomponent treatment is unclear but may be driven by clinical reasoning: the inclusion of several treatments may increase the likelihood of affecting multiple aspects of insomnia, for example, excessive time in bed, conditioned responses, cognitive arousal, and dysfunctional beliefs. Comparative and dismantling studies

Comorbid insomnia: associated with a medical, psychological, or psychiatric disorder. Comorbid insomnia was often referred to as secondary insomnia, but because both problems usually require treatment, the term "comorbid" is recommended

are limited. Several comparison studies indicate stimulus control, sleep restriction, and CBT-I have better sleep outcomes than does relaxation alone (Morin et al. 2006).

The AASM reviews are consistent in conclusions reached regarding studies combining and comparing behavioral and pharmacological treatments: Both are effective in the short-term, but behavioral treatment maintains its effect over time. A new area reviewed in 2006 was the treatment of insomnia in hypnotic-dependent persons. CBT-I has been combined with a tapering schedule to reduce drug use successfully. The effect on sleep seems to occur when the CBT-I sleep treatments are introduced (Vallieres et al. 2005).

A practice parameter paper from an AASM task force reviewed and graded psychological and behavioral treatment studies of primary and secondary (comorbid) insomnia using the AASM Classification of Evidence and Levels of Recommendations (Morgenthaler et al. 2006). The goal of the practice parameters is to influence provider behavior, patient outcomes, and possibly healthcare costs. The practice parameter task force found psychological and behavioral treatments to be effective and recommended them for use with primary and secondary (comorbid) insomnia. Specifically, the task force recommended stimulus control therapy, sleep restriction therapy, relaxation therapy, multicomponent therapy (without cognitive therapy; using combinations of stimulus control therapy, sleep restriction therapy, and relaxation therapy), cognitive behavior therapy (with or without relaxation; using combinations of cognitive and behavior therapy), paradoxical intention, and biofeedback. The use of psychological and behavioral therapy for insomnia was recommended as effective for older adults and chronic hypnotic users.

A meta-analysis by Smith and colleagues (2002) compared the short-term efficacy of behavioral and pharmacological treatments for primary insomnia. Pharmacological treatment included benzodiazepines and nonbenzodiazepine medications that affect the GABA neurotransmitter. The behavioral interventions were stimulus control therapy and sleep restriction therapy. Both types of treatments reduced sleep onset latency and nighttime awakenings in the short term. Large mean effects sizes for all sleep outcome variables were found for both treatments. When effect sizes of individual sleep variables were examined, sleep onset latency was significantly different, with the advantage going to behavioral treatments. The authors note that variability in effect sizes and differences in experimental design may have contributed to the difference in sleep onset latency, but they also suggest that manipulation of the homeostatic sleep drive through behavioral treatment may have stronger effects on sleep initiation than the manipulation of the neurotransmitter affected by pharmacotherapy.

Treating Patients with Comorbid Disorders

The studies in the 1999 AASM review focused on primary insomnia. By the time of the 2006 review, a shift in the literature was emerging. Several comorbid insomnia treatment studies indicated that sleep difficulty in the context of medical and psychological or psychiatric conditions may require separate treatment for sleep, and positive outcomes can be achieved. These include sleep problems associated with pain (Edinger et al. 2005), cancer (see below), major depression or other mood disorders (Harvey 2011), mixed psychiatric disorders (Edinger et al. 2009), mixed medical and psychiatric disorders (Lichstein et al. 2000), and even other sleep disorders such as obstructive sleep apnea (Beneto et al. 2009).

Particularly interesting findings addressing whether insomnia is secondary to depression are reported in recent treatment studies of comorbid major depression and insomnia. In these studies, treatment for insomnia is added to medication for depression (either by addition of a sleep medication or CBT-I). The addition of treatment for insomnia produced more improvement in both insomnia and depression (Fava et al. 2006, Manber et al. 2008). Furthermore, a study by Taylor and colleagues (2007) found that treating comorbid insomnia and

depression with only CBT-I resulted in improvement in both insomnia and depression.

Insomnia is a common sleep complaint in persons with chronic medical problems. There has been considerable sleep treatment research on cancer, a medical problem in which patients have frequent sleep disturbance (Savard & Morin 2001) and that is associated with pain, depression, and fatigue (Stepanski & Burgess 2007). Similar to the attempts of persons in the general population to cope with insomnia, persons with cancer engage in maladaptive behaviors to manage sleep difficulty and fatigue such as resting, napping, going to bed earlier or sleeping later, reading, and watching television (Berger & Farr 1999). These behaviors, particularly those that include excessive time awake in bed and an irregular sleep-wake schedule, contribute to the development of a chronic insomnia problem for persons with sleep difficulty. Altered rest/activity patterns are also present in cancer patients (Ancoli-Israel et al. 2001). Furthermore, the mood disorders commonly seen in cancer patients (Cimprich 1999, Speigel 1997) may also contribute to insomnia.

Treatment studies of insomnia in patients with cancer have reported significant improvements in sleep. Five studies use multicomponent interventions in a CBT-I approach (Davidson et al. 2001, Epstein & Dirksen 2007, Espie et al. 2008, Quesnel et al. 2003, Savard et al. 2005). The latter two studies impressively use polysomnography as one of the multiple measures of improvement of sleep and the most recent two studies use both actigraphy and sleep diaries. These studies are the exception. Many treatment studies of sleep problems in comorbid patients have relied on single self-report sleep inventories, such as the Pittsburgh Sleep Quality Index (Buysse et al. 1989), to evaluate the success of sleep treatment.

NEW DIRECTIONS IN THERAPY

The high prevalence of insomnia coupled with the paucity of therapists trained to deliver the empirically supported treatments have restricted accessibility and influenced the current direction of research. Treatment outcome studies have largely used six to eight weekly individual or group treatment sessions. Alternative delivery strategies have the potential to improve access to insomnia treatment. Several have been tested using multicomponent treatments, including brief interventions, the telephone, self-help (television, Internet, videos, and books), delivery within the primary care setting, and masters-level clinicians as therapists.

Several abbreviated treatments have recently received testing. Primary care may hold the potential for the greatest accessibility to treatment and is well suited to accommodate brief interventions for insomnia. A brief, two-session (25 minutes each) CBT-I intervention combined with take-home pamphlets and audiotapes that reinforced the information provided during the sessions in a Veterans Affairs primary care setting showed significant outcomes compared to a sleep hygiene control group (Edinger & Sampson 2003). Germain and colleagues examined a brief behavioral intervention in older adults (Germain et al. 2006) and civilians with posttraumatic stress disorder (PTSD) (Germain et al. 2007). In the older adult study, patients received one individual session (45 minutes) followed by a booster session (30 minutes) two weeks later. Significant improvement in sleep outcomes was found in comparison with the information-only control group. The patients with PTSD received one individual session (90 minutes), focused on insomnia and nightmares, and a follow-up telephone call three weeks after the session. Clinically significant changes in sleep and daytime PTSD symptom severity occurred.

Other abbreviated treatment protocols include a three-session multicomponent insomnia treatment in a family medicine clinical setting (Goodie et al. 2009), a brief (two individual sessions and two telephone follow-ups) multicomponent intervention for older adults provided by rural care providers who were trained in a two-day workshop (McCrae et al. 2007), and a brief multicomponent intervention for family caregivers of cancer patients (Carter 2006).

A study of different dosing protocols (one, two, four, or eight sessions) on insomnia outcomes found that four sessions produced the most favorable results (Edinger et al. 2007). However, the success of the varied abbreviated protocols suggests that further work is needed to determine the optimal frequency and duration of sessions to produce improvement in insomnia, depending on comorbidities, the best fit within a primary care model, and whether treatment gains are maintained over time.

Primary care is a setting in which nurses as interveners could improve access to treatment. Two studies conducted in the United Kingdom by Espie and colleagues (2001, 2007) found that trained and supervised primary care nurses were able to successfully implement CBT-I in a group format. In the more recent study, 201 insomniacs participated in an effectiveness trial that compared general practitioner treatment as usual (TAU) with TAU plus multicomponent CBT-I. No improvements on sleep variables were found in the TAU condition. The addition of CBT-I produced significantly reduced sleep latency on sleep diaries and significantly reduced wakefulness during the night on both sleep diaries and actigraphy compared to TAU. Improvements were maintained at a six-month follow-up (Espie et al. 2007).

To increase access to insomnia treatment, healthcare providers other than psychologists will be needed. Issues regarding the education, training level, and supervisory requirements of persons who provide CBT-I are controversial topics in the field (Perlis & Smith 2008). Outcomes between healthcare providers who did not have mental health training produced outcomes comparable to those who did (Morin et al. 2006). Nevertheless, recent studies in the United States have employed masters-level nurses with mental health training and background to deliver treatment to cancer survivors in a group format (Epstein & Dirksen 2007) and to older adults in individual sessions (Germain et al. 2006).

Many people who have insomnia symptoms seek self-help methods instead of seeking professional help. Self-help methods in a recent meta-analysis were defined as those that a person can use independently in the home setting and included books, booklets, audiotapes, video, television programs, and Internet (van Straten & Cuijpers 2009). Small to moderate effect sizes were found, which are smaller than effect sizes in the usual individual and group insomnia treatment formats (e.g., Irwin et al. 2006, Smith et al. 2002). Seven of the 10 self-help studies offered additional support via email, telephone, or face-to-face encounters. When subgroup analyses were performed, studies that included persons with psychiatric comorbidities did not fare as well as those in studies in which they were excluded. The support offered was limited and may not have allowed for a thorough and ongoing assessment of mental health status.

One issue that faces all self-help methods is whether the individual knows that the treatment being offered is the correct treatment for the individual's problem. If the person examining Internet programs or books or videos for insomnia does not have insomnia but rather has sleep apnea or narcolepsy or a severe medical or emotional problem, then treating only the insomnia symptoms may delay treatment for the other problems and could raise safety issues. One Internet program that had strong results in improving sleep provides interesting data for how to handle this issue of match between the program and the patient (Ritterband et al. 2009). Rather than having broad inclusion criteria, the investigators provided access to the Internet program only as part of their research study and recruited a small sample of individuals who all went through a three-step screening process. Potential participants filled out an online interest form, then had a 15-minute telephone interview followed by a semistructured personal interview. Those with sleep disorders other than insomnia or who had major medical or psychiatric problems were ineligible to participate. Those who met all the criteria for inclusion were randomly assigned to a nine-week CBT-I Internet program or to a no-treatment control condition. Remarkably, there were very few dropouts from either condition.

In many ways, this research Internet program kept professional help close at hand both during screening and then later for monitoring progress. It may be wise to consider the need for screening and professional support to maximize effectiveness, particularly as those with comorbid disorders seek to use the self-help materials.

THE CHALLENGES AHEAD

As this review illustrates, much has been learned about the consequences of insomnia, how insomnia develops, and the successful treatment of insomnia. The challenges ahead fall into five broad areas. First is to increase the knowledge across multiple levels of analysis about what insomnia is and how it develops. Second is to develop new treatments that apply the knowledge being generated by advances in our understanding of insomnia. Third is to focus on mechanisms of action about how established treatments of insomnia produce improvement rather than engaging in ever more horse-race clinical trials. Fourth is to disseminate what is already known about successful treatment so that more can be helped. And fifth, because the consequences of poor sleep affect so many domains of functioning, increasing attention must be given to prevention, early intervention, and public policy initiatives. This is a full agenda and much remains to be done.

To focus on the dissemination agenda item, the high prevalence of insomnia underscores the need for treatment, yet the lack of trained providers remains one of the greatest obstacles to CBT-I dissemination. Potential strategies to address the problem include training with certification examination for different types of providers (Perlis & Smith 2008) and a stepped-care model in which providers might use different methods and increased individual personal support for those who have more severe or resistant problems (Espie 2009).

If the high demand for services by those with insomnia is to be met, the challenge for CBT-I, as Espie points out, "... is no longer to prove its credentials, but to punch its weight" (p. 1549). The Veterans Health Administration (VHA), the largest integrated health care system in the United States, is taking the contender into the ring. The VHA is currently undertaking an evidence-based psychotherapy dissemination program (Zeiss & Karlin 2008). This national initiative is training VA mental health clinicians in the delivery of CBT-I, among other evidenced-based treatments. The CBT-I dissemination has the potential to impact a tremendous comorbid insomnia problem, thereby improving treatment outcomes for Veterans. Furthermore, the CBT-I initiative program could be an exemplar for other integrated health systems such as HMOs.

The future has begun, and there are many opportunities for both generating and applying knowledge. For dissemination and public policy initiatives to succeed, the knowledge base upon which they depend must also expand to meet the challenge.

SUMMARY POINTS

1. Sleep disturbance at any development stage from infancy through old age affects health and cognitive, emotional, and social functioning.
2. Certain individuals may be predisposed to develop insomnia under stressful conditions that lead to arousal, maladaptive sleep habits, and dysfunctional cognitions. These perpetuating factors are the targets of treatment.
3. Circadian and sleep homeostatic processes play an important role in insomnia development, maintenance, and treatment.

4. Insomnia is a highly prevalent problem that is comorbid with a range of psychological, psychiatric, and medical conditions.
5. The primary symptoms of insomnia are complaints of difficulty initiating sleep, difficulty maintaining sleep, and/or sleep that is nonrestorative or poor in quality. In addition to sleep symptoms, complaints of daytime impairment are required to diagnose insomnia.
6. Psychological and behavioral treatments for insomnia are efficacious for primary and comorbid insomnia. The common elements of CBT-I are cognitive therapy, typically in the form of cognitive restructuring, stimulus control therapy, sleep restriction therapy, and relaxation therapy.
7. Adding treatment for insomnia, in the form of sleep medication or CBT-I, to antidepressant therapy produces more improvement in depression and insomnia.
8. Alternative delivery methods and innovative treatments hold promise for increasing access to care and accelerating improvement. Gaps in insomnia treatment include an understanding of the mechanisms underlying the efficacious interventions, trained providers, and dissemination models.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

Dana Epstein's contribution to this review was supported with resources and the use of facilities at the Phoenix Veterans Affairs Health Care System. The contents of this article do not represent the views of the Department of Veterans Affairs or the United States government.

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Annual Review of
Clinical Psychology

Volume 7, 2011

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