
CHAPTER 1

Introduction

Patients seeking treatment for insomnia describe difficulties falling or staying asleep at night and stress the impact that their poor sleep has on their lives, often citing it as the reason for seeking help. Consistent with patients' experiences, the clinical entity *insomnia disorder* (American Psychiatric Association, 2013) is considered a 24-hour condition, consisting of disturbed sleep at night and associated distress or impaired functioning during the day. Cognitive-behavioral therapy for insomnia (CBT-I) is a brief and effective sleep-focused treatment that is anchored in the science of sleep and utilizes principles of general cognitive-behavioral therapy (CBT) to address both nocturnal and daytime symptoms of insomnia disorder. Despite strong empirical support, CBT-I is not available to most patients who could benefit from it. To a large extent, this is because there is a shortage of therapists trained to deliver it.

This book aims to guide therapists in helping their adult patients, including those with comorbidities, to sleep better. The goal is to allow therapists to apply CBT-I in a flexible, patient-tailored manner. To enable this flexible approach, and based on our extensive experiences of training clinicians to deliver CBT-I, we have decided to devote the first part of the book (Chapters 1–5) to background information about the nature of sleep and insomnia. We believe that information about how normal sleep is organized and regulated, how behaviors influence the sleep regulation process, and how comorbidities affect sleep and its regulation is essential for effective delivery of CBT-I. The second portion of the book (Chapters 6–13) is about the treatment itself. After discussing a sleep-focused assessment, we introduce the treatment components, and then discuss how to select the most relevant component, in what order to introduce them, and when to alter the standard guidelines to the unique needs of each patient.

We provide a case conceptualization framework for making these important clinical decisions, and we discuss cautions and contraindications. Comorbidities and consumptions of sleep medications are carefully considered, but they are not contraindications. This book discusses specific effects of the comorbidities and the use of sleep medications on insomnia, and when and how to modify the standard CBT-I guidelines accordingly. Also in the spirit of promoting implementation of CBT-I that meets the unique needs of patients, we do not present a rigid session-by-session protocol in this book. Nonetheless, to aid therapists as they learn to use this treatment, we do include a template for a six-session treatment plan (an insomnia assessment session plus five treatment sessions), and we demonstrate a full course of treatment for two cases, both of which are introduced at the end of this chapter.

DIAGNOSIS OF INSOMNIA DISORDER

The term *insomnia*, as it is often used colloquially in reference to poor sleep, is not the same as the clinical entity called *insomnia disorder*. According to the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5; American Psychiatric Association, 2013), the diagnosis of insomnia disorder includes the following two core criteria: (1) difficulties falling asleep or staying asleep (nighttime symptoms), and (2) associated distress and/or perceived negative impact on daytime functions and mood (daytime symptoms). In other words, as is the case with other disorders, a clinical diagnosis is not made unless symptoms are associated with clinically significant consequences.

Operationalizing Insomnia Disorder Criteria

The diagnosis of insomnia disorder is based entirely on the patient's report and the clinician's judgment. Objective sleep measurements, such as staying overnight in a sleep laboratory while being physiologically monitored, are not required or even recommended for the diagnosis. The core clinical criteria for insomnia disorder are not well operationalized. For instance, the DSM-5 criteria for poor sleep do not specify a cutoff for time to fall asleep or for time awake in the middle of the night. Quantitative criteria that operationalize sleep difficulties have been proposed (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003) and used in research. Specifically, the proposed quantitative criterion for poor sleep is having sleep onset latency or time awake after sleep onset greater than 30 minutes at least three nights a week for at least 6 months. However, there is evidence that morbidity can be similar in cases with less frequent but more severe sleep disruption (in term of number of minutes with unwanted wakefulness) and in cases with more frequent but milder sleep loss (Lineberger, Carney, Edinger, & Means, 2006). DSM-5 does provide a little more guidance on what is meant by *clinically significant* consequences of poor sleep, by listing examples of specific domains of daytime impairment; these are similar to those specified in the Research Diagnostic Criteria for insomnia (Edinger et al., 2004). The consequences of poor sleep include the following domains:

- Fatigue/ malaise
- Attention, concentration, or memory impairment
- Social/vocational dysfunction or poor school performance
- Mood disturbance /irritability
- Daytime sleepiness
- Motivation/energy/initiative reduction
- Proneness to errors/accidents at work or while driving
- Physical symptoms such as tension headaches and gastrointestinal symptoms
- Concerns or worries about sleep

DSM-5 includes a minimum frequency criterion (at least three nights a week), which was not present in DSM-IV-TR (American Psychiatric Association, 2000), and a minimum duration of 3 months, which is longer than was indicated in DSM-IV-TR. Another, and more major, revision

to the definitions of sleep–wake disorders in DSM-5 relative to DSM-IV-TR is the elimination of the distinction between primary insomnia and insomnia related to a medical or psychiatric condition, which was made in the earlier version. DSM-5 defines a single condition: insomnia disorder. The elimination of the distinction between primary insomnia and insomnia related to other disorders is based on the notion that the presence of comorbidities is a dimension, rather than a defining feature, of the disorder. This change reflects the growing recognition that, even when poor sleep emerges as a symptom of another disorder, it may develop into a separate comorbid disorder that is not ameliorated by the treatment of the comorbid condition. For example, in the context of depression, insomnia is a common, unresolved residual symptom among patients whose depression remits following antidepressant therapy (Nierenberg et al., 1999, 2010). The implication is that a diagnosis of insomnia disorder can be made when sleep disturbances emerge in the context of another psychiatric or medical condition, as long as the complaint of poor sleep is accompanied by clinically meaningful consequences, such as distress and perceived impairment in function.

AGREEMENT BETWEEN OBJECTIVE AND SUBJECTIVE SLEEP IN INSOMNIA

The “gold standard” objective measure of sleep is *polysomnography* (PSG). PSG is a method for studying brain waves and other physiological parameters, such as eye movements, muscle activity, respiration, and heart rate. Using such methods, research shows that compared to non-symptomatic control participants, individuals with insomnia take longer to fall asleep; they are awake for a longer time in the middle of the night; and their sleep efficiency (percentage of time asleep relative to time allotted for sleep) is lower. However, average differences between people with and without insomnia disorder are modest and of questionable clinical significance. For example, Perlis, Smith, Andrews, Orff, and Giles (2001) report that individuals with insomnia disorder sleep 25 minutes less and take 12 minutes longer to fall asleep than good sleeper controls.

In general, people with insomnia or insomnia disorder underestimate the time they sleep and overestimate the time it takes them to fall asleep, compared to their PSG sleep results (Carskadon et al., 1976; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Also, compared to people without insomnia (symptoms or disorder), those with insomnia are more likely to identify themselves as having been awake when awakened from PSG-defined sleep (Carskadon et al., 1976; Perlis, Giles, Mendelson, et al., 1997).

There are, however, individual differences in the extent of sleep state misperception among people with insomnia disorder. At the extreme is a group of people (about 5–9% of people with sleep disorders; American Academy of Sleep Medicine, 2005; Coleman et al., 1982) for whom there is a profound mismatch between subjective and objective sleep estimates. The *International Classification of Sleep Disorders*, second edition (ICSD-2; American Academy of Sleep Medicine, 2005), which is distinct from the DSM classification system, classifies this extreme group as having *paradoxical insomnia*. Clinically, people with paradoxical insomnia report a chronic pattern of little or no sleep on most nights; although their self-reported levels of day-

time impairment are similar to those of other people with insomnia disorder, their reported impairment appears much less severe than would be expected from the extreme level of sleep deprivation they report.

PREVALENCE AND COURSE

Estimates of *point prevalence* (i.e., the proportion of people in a population who have a disorder at a particular time) depend on the operational definition of insomnia or insomnia disorder, the population studied, and the method of assessment. Epidemiological studies estimate the point prevalence of poor sleep that is associated with daytime distress or impairment and lasts at least 1 month as 5–10% (Mai & Buysse, 2008; Morin, LeBlanc, Daley, Gregoire, & Merette, 2006; Ohayon, 2002; Ohayon & Roth, 2001). As expected, the prevalence decreases as the criteria become more restrictive. For instance, 25.3% of 2,001 individuals sampled in a Canadian epidemiological study were dissatisfied with their sleep, but only 17.2% also had difficulty initiating or maintaining sleep (Morin, LeBlanc, et al., 2006). Most (78.2%) of those with insomnia complaints (dissatisfaction with sleep and difficulty with falling or staying asleep) had the problem for over a month, and 38.3% also experienced negative daytime consequences, resulting in a 9.6% prevalence estimate of insomnia disorder in this study (Morin, LeBlanc, et al., 2006). The *incidence* (rate of new cases) of insomnia per year is between 3 and 6% (Ford & Kamerow, 1989; Jansson-Frojmark & Linton, 2008; LeBlanc et al., 2009). There is roughly a 2:1 ratio of women to men (e.g., Mai & Buysse, 2008; Ohayon & Roth, 2001), and an increased prevalence with advancing age (Bixler, Kales, Soldatos, Kales, & Healey, 1979). The prevalence of insomnia is higher among people with comorbid medical and psychiatric conditions; the most prominent among these comorbidities are depressive disorders (Ford & Kamerow, 1989) and chronic pain conditions (Ohayon, 2005).

There is a large body of retrospective and prospective studies (summarized by Jansson-Frojmark & Linton, 2008) suggesting that poor sleep tends to be a chronic problem. The retrospective studies report that most individuals complaining of poor sleep (insomnia symptoms) have had the problem for more than 1 year, and approximately 40% have had it for more than 5 years. The longitudinal prospective studies have similarly found that roughly half of individuals with insomnia symptoms continue to experience poor sleep at least 1 year later. There is less research about the course of insomnia disorder. Two population-based studies that did examine the course of insomnia as a disorder found that over the course of 1 year, insomnia disorder was persistent for 31–44% of the baseline cases (reviewed in Jansson-Frojmark & Linton, 2008). One of the two studies further reported that 11% of individuals whose insomnia disorder remitted eventually experienced a relapse of insomnia disorder during the 3-year follow-up period (Morin et al., 2009). These two naturalistic studies included a mix of individuals with treated (usually over-the-counter or prescription medications) and untreated insomnia disorder. It is possible that rates of persistence of insomnia disorder are even higher among individuals with untreated insomnia disorder. Interestingly, although poor sleep tends to be persistent or recurrent, in most cases it does not develop into insomnia disorder. It is estimated that only 10–14% of people with poor sleep who do not meet all diagnostic criteria for insomnia disorder end up meeting these criteria at some assessment point during a 3-year follow-up (Morin et al., 2009).

COST

Poor sleep and insomnia disorder are frequently encountered by mental health professionals and primary care physicians in their practices (Canals, Domenech, Carbajo, & Blade, 1997; Ford & Kamerow, 1989). Insomnia disorder carries significant personal costs, being associated with increased risk for other health conditions and mood disturbances, including a twofold increase in risk for depression (Baglioni et al., 2011; Ford & Kamerow, 1989). It also has a marked economic impact, which, when both direct and indirect costs are accounted for, is estimated at \$100 billion annually in the United States (Rosekind & Gregory, 2010; Stoller, 1994). Compared to a healthy sleeper, a person with insomnia incurs roughly \$4,500 more in costs per year, considering the costs of health care, absenteeism from work, disability, prescription medications, and over-the-counter medications (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009).

HELP-SEEKING BEHAVIORS

Although psychological distress is one of the main determinants prompting individuals with insomnia to seek treatment, only 6% of people with insomnia consult with a psychologist; most consult with a medical professional, primarily a primary care physician (Morin, LeBlanc, et al., 2006). In general, fewer than half of individuals with insomnia symptoms seek help over their lifetimes (Ancoli-Israel & Roth, 1999; Morin, LeBlanc, et al., 2006), and only 20% make an appointment to see a physician specifically about their sleep (Bartlett, Marshall, Williams, & Grunstein, 2008; Shochat, Umphress, Israel, & Ancoli-Israel, 1999). In addition to psychological distress, predictors of help-seeking behaviors for insomnia include insomnia severity (both poor sleep and perceived daytime impairment), short sleep duration (less than 6.5 hours), older age, poorer health, and higher income (Bartlett et al., 2008). Little is known about cross-referrals between medicine and psychology for the treatment of insomnia, or about help seeking for insomnia in the context of psychotherapy for other mental disorders.

EMPIRICAL SUPPORT FOR CBT-I

A large and consistent body of literature provides strong empirical support for the efficacy of CBT-I. The evidence is based on studies that compare CBT-I to a control therapy (Edinger, Wohlgemuth, Radtke, Marsh, & Quillian, 2001; Manber et al., 2008) and to delayed-treatment controls (Edinger et al., 2001; Lichstein, Riedel, Wilson, Lester, & Aguillard, 2001; Morin, Kowatch, Barry, & Walton, 1993). Direct comparison with hypnotic medications demonstrates equivalence of CBT-I to sleep medications, such as temazepam (Morin, Colecchi, Stone, Sood, & Brink, 1999), zolpidem (Jacobs, Pace-Schott, Stickgold, & Otto, 2004), and zopiclone (Sivertsen et al., 2006). There is also evidence that the effects of CBT-I are durable, lasting 1–2 years following discontinuation of treatment, and in that way are superior to the effects of medication treatments (Morin, Colecchi, et al., 1999; Sivertsen et al., 2006). The weight of evidence supporting CBT-I has led to its recognition as a first-line treatment for insomnia in a National Institutes of Health Consensus Statement (2005). The British Association of Psychopharmacology has also

recognized the importance of CBT-I in the management of insomnia and has issued a statement recommending CBT-I as a front-line treatment for insomnia (Wilson et al., 2010).

CBT-I Is Effective for Insomnia Comorbid with Other Disorders

Sleep difficulties are common among those with psychiatric comorbidities and are diagnostic symptoms of depressive and anxiety disorders. However, sleep problems often do not resolve with general psychotherapy (Kopta, Howard, Lowry, & Beutler, 1994), pharmacotherapy (McClintock et al., 2011; Nierenberg et al., 1999, 2010), or psychotherapy for the comorbid disorders (Manber et al., 2003; Thase et al., 2002; Zayfert & DeViva, 2004). In the past, it was assumed that sleep difficulties experienced by individuals with mental illnesses were symptoms of psychiatric disorders that would resolve when the underlying disorders were successfully treated. This belief led clinicians to ignore sleep difficulties. A similar state of neglect is present for people with medical comorbidities, including sleep disorders other than insomnia disorder. As a result, poor sleep has rarely received special psychotherapeutic attention. This is unfortunate, because poor sleep among people with psychiatric and medical conditions contributes to the severity of the comorbid conditions and hinders response to treatment of these conditions (Buysse et al., 1999; Thase, Simons, & Reynolds, 1996). Several clinical trials have indicated that CBT-I is indeed effective for people with depression (Lancee, van den Bout, van Straten, & Spoormaker, 2013; Manber et al., 2008; Morawetz, 2003), posttraumatic stress disorder (PTSD) (Germain, Shear, Hall, & Buysse, 2007; Zayfert & DeViva, 2004), and pain (Currie, Wilson, & Curran, 2002; Edinger, Wohlgemuth, Krystal, & Rice, 2005; Rybarczyk et al., 2005). Evidence also suggests that in the case of depression, simultaneous treatment of depression and insomnia with a hypnotic (Fava et al., 2006) or CBT-I (Manber et al., 2008) enhances depression outcomes. Thus, psychotherapists could improve care of their patients who present with comorbid insomnia disorder by treating the insomnia disorder with this brief, empirically validated, insomnia-focused therapy.

Can CBT-I Be Used When Hypnotic Medications Are Concomitantly Used?

Individuals with insomnia who take hypnotic medications but nonetheless have difficulty sleeping can benefit from CBT-I. Whereas randomized controlled trials of CBT-I usually exclude participants who use hypnotic medications, uncontrolled case series reports from clinic samples and studies that combine CBT-I with a medication taper protocol suggest that CBT-I is not less effective for hypnotic users than for nonusers (Morin et al., 2004; Rosen, Lewin, Goldberg, & Woolfolk, 2000).

Patients who meet criteria for insomnia disorder even though they use hypnotics may have developed tolerance to the medications or psychological dependence on them. *Tolerance* to a drug is a decrease in susceptibility to the effects of the drug, due to its continued administration; it results from cellular adaptation to the active substance in the drug, so that increasingly larger doses are required to produce the same physiological or psychological effect obtained earlier with smaller doses. *Psychological dependence* on a hypnotic medication is present when a patient does not believe that sleep can be attained without the use of the hypnotic medication. Abrupt discontinuation of a hypnotic medication is often associated with transient sleep difficulty immediately after discontinuation. Patients who interpret the experience of poor sleep immediately

after discontinuation as evidence that they cannot sleep well without the medication develop psychological dependence. Patients with insomnia disorder who use hypnotics regularly and have developed tolerance to or psychological dependence on hypnotic medications are classified in ICSD-2 as having *hypnotic-dependent insomnia* (American Academy of Sleep Medicine, 2005). Among patients receiving CBT-I at the Stanford School of Medicine, about half indicated worry that they would not be able to sleep if they did not take medications, that they had “become too dependent on the medication,” or that they had tried to stop taking medications and failed (Adler, Carde, Kuo, Ong, & Manber, 2008).

Some patients with hypnotic-dependent insomnia include hypnotic discontinuation as a treatment goal and others do not. In Chapter 12 we outline a treatment for Sam, introduced at the end of this chapter. Sam had been using a hypnotic medication for 20 years, and his physician who referred him to CBT-I described him as having hypnotic-dependent insomnia. Sam did not include hypnotic taper as a treatment goal. After five sessions of CBT-I, encouraged by the improvement in his sleep and daytime functioning, Sam spontaneously discontinued the sleep medication. Many patients with hypnotic-dependent insomnia who state that their goal is to sleep well without sleep medications express fear of not being able to sleep without the drugs. Psychologists in collaboration with physicians have developed and tested slow-taper protocols for patients with hypnotic-dependent insomnia, usually combining such protocols with CBT-I (Kirmil-Gray, Eagleston, Thoresen, & Zarcone, 1985). In Chapter 13, where we discuss collaboration between a CBT-I therapist and a prescribing clinician, we also briefly describe a few of these protocols.

Patient Preference

Most people seeking help for insomnia are treated with a sleep medication; few know about CBT-I as a treatment option. However, when provided a description of CBT-I, patients with insomnia rate it more favorably than they do descriptions of pharmacological treatments (Azad, Byszewski, Sarazin, McLean, & Koziarz, 2003; Morin, Gaulier, Barry, & Kowatch, 1992). Vincent and Lionberg (2001) further reported that psychological treatment was judged to be more acceptable than pharmacological treatment, more effective in the long term, more likely to improve daytime functioning, and less likely to produce negative side effects (Vincent & Lionberg, 2001). Among middle-aged and older adults with insomnia symptoms, predictors of interest in CBT-I were reporting greater negative impact of poor sleep in terms of interference with daily functioning and daytime fatigue, and reporting more mood problems and physical symptoms (Cahn et al., 2005).

Sleep Hygiene Alone Is Rarely Effective

Sleep hygiene is a set of popular recommendations for good sleep practices. A few examples are that patients should not drink coffee too late, should limit alcohol, should keep the bedroom at a comfortable temperature, and should always go to bed at the same time. However, sleep hygiene recommendations have limited efficacy and have often been used as control therapy in randomized controlled trials demonstrating the efficacy of CBT-I. Our experience has been that by the time patients seek our help, they have already tried these common recommendations. In such

cases, it is best to emphasize that CBT-I involves much more than sleep hygiene recommendations.

USING THIS BOOK

This book prepares therapists to deliver CBT-I effectively and flexibly, tailoring it to individual presentations (including those with comorbid conditions). This flexible approach to CBT-I requires readers to understand sleep regulation, presented in Chapters 2 and 3; to be familiar with models of insomnia, discussed in Chapter 3; and to be mindful of the impact of comorbid conditions on insomnia, the focus of Chapters 4 and 5. Thus Chapters 2–5 build a foundation for the assessment and effective implementation of CBT-I. Chapter 6 is devoted to the assessment of insomnia, and Chapters 7–9 present the behavioral and cognitive treatment components, including suggestions for when and how to alter these components to promote adherence and efficacy. In Chapter 10, we describe a treatment planning process that guides clinicians in how to select and sequence CBT-I components for each individual patient. Inherently, truly customized treatment is incongruent with a fixed session-by-session description of treatment. Nonetheless, we describe a general structure for a six-session treatment protocol, consisting of a comprehensive sleep assessment and five treatment sessions; we then demonstrate the full course of treatment with two case examples in Chapters 11 and 12. Chapter 13 discusses implementation and professional issues.

Below, we introduce the two case examples. We encourage our readers to keep these two cases in mind as they read the rest of the book, and to be curious about how the information being discussed applies to these cases.

CASE EXAMPLES

Sophie

Sophie was a 42-year-old unmarried woman who was having difficulty initiating sleep; she was also waking in the night and unable to fall back to sleep for hours. Sophie was an attorney specializing in corporate law. Her job was intermittently stressful when she was nearing project deadlines. She did not have a bed partner and lived alone. Sophie was very healthy and active; she loved to hike, swim, and ride her bicycle. She had regular wellness visits with her primary care physician and had never had any major health problems. She did not snore or have other symptoms of sleep apnea or other sleep disorders. She had never had a sleep study. Prior to the onset of her insomnia, she used to enjoy 1–2 cups of coffee in the morning, but now she did not drink any caffeinated drinks because she was concerned that they would negatively affect her sleep.

This was the first time Sophie had experienced problems sleeping for so long. Before the previous year, she had never had trouble sleeping. She said, “I used to love going to sleep and looked forward to it. I used to fall asleep when my head hit the pillow, and I never woke up until I had to in the morning.” Her typical sleep schedule was 10:30 P.M. to 6:30 A.M., but she would sometimes sleep in a couple of hours on the weekends or stay up later to work if she had a deadline.

Roughly 1 year ago, Sophie had gone through a very painful breakup. She remembered an incident that had happened around the time of the breakup: As she was drifting off to sleep, she woke up feeling that it was hard to breathe and fearing she might be having a heart attack. She went to the bathroom, splashed water on her face, and felt a little better, but went to the emergency room to make sure she was OK. Sophie was medically cleared and told that she had probably had a panic attack. After this memorable night, Sophie started having difficulties falling asleep. She found herself lying in bed for hours trying to fall asleep, “fighting with” her sleep, and growing more and more frustrated the longer she was awake. She would eventually fall asleep, but then awaken with a start about 4 hours later and would again have difficulty returning to sleep. Sophie had never experienced a second panic attack. Although initially she was afraid it would happen again, she said that now she had no concerns about having a panic attack; her concern now was about sleep.

Sophie estimated that she was getting approximately 4 hours of sleep per night. After dinner, she surfed the Internet and watched TV. She got into bed around 9:30 P.M., because she wanted to have “plenty of time to fall asleep.” She used to read in bed, but after she read somewhere that she should not do this, she stopped doing it; now she turned off the light right away. It took her 1–3 hours to fall asleep. She stayed in bed tossing and turning, checking the clock. She was not worried about anything in particular, but her mind skipped around aimlessly (“My mind is definitely not quiet”). She woke at around 2:00 A.M. and 4:00 A.M., and sometimes again around 5:00 A.M. When she woke up at 4, her mind would get going about what she needed to do, and she would start getting anxious that she was awake and only had a few hours left before she had to get up. She set an alarm for 7:00 A.M. on weekdays, but usually woke up a few minutes before it sounded. She did not set an alarm on the weekends, but she still woke up at about the same time. She estimated that she was awake for a total of about 30–90 minutes each night. Sophie had a very difficult time getting out of bed in the mornings, and as a result, she had discontinued her morning workout routine. On weekdays, Sophie “forced” herself out of bed by 7:30 A.M. so she could make it to work on time. On the weekends, she allowed herself to stay in bed until 9:00 A.M.; she found it “nice” to rest in bed, and she was occasionally able to fall back to sleep. On most days, Sophie would lie down for a nap when she got home from work, but she was usually unable to quiet her mind and would not actually fall asleep. On the weekends, she sometimes did sleep on the couch while watching a movie in the living room during the early afternoon.

Sophie reported feeling miserable during the day, finding it difficult to concentrate in meetings, and losing track of important work. Just last week, she had been arguing a case in front of a judge and completely lost her train of thought. She was also concerned that she was becoming more irritable with colleagues and with friends. She no longer went out much with friends in the evenings, because she did not feel that she had enough energy to do much, and she was concerned that if she went out in the evenings she would have problems sleeping at night.

When she first experienced difficulty with sleep, Sophie tried taking sleep medications. Eszopiclone was “not that helpful,” and clonazepam left her feeling “fuzzy and groggy” in the morning, so she stopped taking either drug. She then experimented with drinking a glass of wine before going to bed. This helped her fall asleep a little faster, but she would wake more often at night—so she did it only rarely, when she felt particularly “wired” at bedtime. Sophie expressed skepticism that anything would help her sleep well again, but stated that she would “give anything to go back to sleeping like I did before.”

Sam

Sam was a 60-year-old accountant. He had recently been having a very hard time falling asleep and was waking up many times in the middle of the night. He also had a diagnosis of sleep apnea that was not fully treated. He would wake up in the middle of the night noticing that the mask used to treat his sleep apnea (see Chapter 4) was off, but he often would not put it back on. For the past 6 months, he had been out of work on disability for chronic pain and fatigue related to a diagnosis of fibromyalgia. Sam lived with his wife and an adult daughter, with both of whom he enjoyed positive relationships. His second daughter lived nearby with her husband and their two young children. Sam enjoyed interacting with his grandkids and had been spending 2 days a week with them until recently, in part because his insomnia had worsened and his energy “to keep up with toddlers” was diminished.

Sam had suffered intermittent insomnia for the past 25 years. At first he did not recall any specific trigger, but upon further reflection he remembered that he had first experienced insomnia around the time his second daughter was born. His doctor prescribed zolpidem, and he had been taking it every night for the past 18 years. He would like to stop, but although he did not feel it helped that much, he was worried that his sleep would “be even worse” if he stopped. He was also taking gabapentin and duloxetine for chronic neuropathic pain and fibromyalgia, and his doctor had said that these medications might also help him sleep better.

Sam estimated that most nights he slept around 5 hours, but noted that about once a month he was “up for 20 hours straight” and had no idea why. He believed that this long-standing battle with sleep was having a great impact on him. During the day, Sam was experiencing pain, fatigue, depressed mood, difficulty concentrating, irritability, and memory impairment, all of which he attributed to his sleep difficulties. He had also stopped driving more than 3 miles from home, because he feared he would fall asleep at the wheel. He and his wife slept separately, because he felt that her soft snoring made it difficult for him to sleep.

Over the years, Sam had seen many doctors and therapists to help him sleep better. He had been keeping notes about his sleep and the advice he had received. He brought these notes to his initial appointment, along with a three-ring binder with medical records related to his sleep problems. The notes in the binder included details about when and how long he was awake in the middle of the night, what time he woke up in the morning, and how he felt throughout the following day.

When asked to describe a typical night, Sam reported that he got into bed around 10:30 P.M. It took him about 2 hours to fall asleep. He usually slept 4 or 5 hours and then woke up. He said that the rest of the night he was in and out of sleep, sleeping in chunks of half an hour or less. He woke up sometimes between 5:00 A.M. and 6:00 A.M., but continued to try to sleep until 7:30 to 8:30 A.M., when he would get up to start the day. He said that he sometimes dozed off during the period before he got up, but did not feel that this was “real sleep.” When he got out of bed in the morning, he felt as if he had been “fighting with sleep for hours.” After breakfast, he would lie on the couch watching TV for a couple of hours. He thought that sometimes he dozed off during these periods, because he sometimes realized he had missed a short segment of the show he was watching. He would try to do things out of the house in the afternoons, but often stayed at home because of pain. If he did not go out of the house, he would lie down again in the afternoon to “rest from the pain” until his wife got home from work. He spent the evenings with his wife. They

often watched a television program or a movie. He reported sometimes dozing off while watching television; when he did, his wife would wake him. Asked to describe his prior sleep schedule, Sam said that when he was still working, he used to sleep in on the weekends and on days off until 11:00 A.M.; he added that he used to be a bit of a “night owl” then, and on weekends he would go to bed after midnight.

Sam’s goals were to sleep more, get rid of his pain, and not feel sleepy during the day so that he could spend more time with his grandchildren.

We will revisit the cases of Sophie and Sam throughout the book, and we present their treatment plans in Chapters 11 and 12, respectively.

KEY PRACTICE IDEAS

- Insomnia disorder is a 24-hour disorder.
- People with insomnia tend to underestimate how long they sleep and overestimate how long it takes them to fall asleep.
- People with insomnia disorder comorbid with another physical or mental disorder can benefit from CBT-I.
- Hypnotic use is not a contraindication for using CBT-I.