

William K. Wohlgemuth
Ana Imia Fins

Advances in Psychotherapy –
Evidence-Based Practice

Insomnia



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Advances in Psychotherapy – Evidence-Based Practice

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Insomnia

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Preface

Insomnia is a widespread problem. Estimates suggest that, within a given year, about 40% of the population will experience difficulty falling or staying asleep, while about 10% experience chronic insomnia. Sleeping pills have been used for decades, but physicians are wary about the consequences of long-term use. Fortunately, efficacious nondrug, behavioral methods have been developed and tested over the past 2 decades. These treatments were developed with knowledge of the biological underpinnings of sleep. Additionally, during this time, investigators gained a better understanding of common beliefs about sleep and the disruptive habits which develop as a result of those beliefs. This knowledge has been incorporated into a treatment called *cognitive behavioral therapy for insomnia* (or CBT-I). Treatment guidelines based on reviews of the evidenced-based literature, published by both the American Academy of Sleep Medicine and the American College of Physicians, support CBT-I as first-line therapy for insomnia.

Insomnia is a common symptom of many medical, psychiatric, and other sleep disorders, and proper evaluation is necessary to rule out other potential causes of the sleep difficulty. Consultation with a sleep specialist may be needed to determine if a comorbid sleep disorder is present. Consultation with a physician or psychiatrist may be needed to rule out either medical or psychiatric causes of insomnia. Sometimes it may be necessary to work in tandem with a physician or sleep specialist to coordinate medical treatment (e.g., hypnotic medication) with CBT-I.

When learning any new therapeutic technique, therapists can be assisted by supervised practice for several cases to gain confidence in effective implementation of the therapy. We suggest that therapists seek to consult when beginning to use CBT-I, as clinical cases are varied and can be quite complex.

Our goal in this book is to provide a general overview of definitions, prevalence, impact, and theories of insomnia. We then provide a more specific, detailed description of the evaluation and treatment of insomnia. We also review more recent developments in the treatment of insomnia, such as the online implementation of CBT-I and interventions which focus more directly on cognitive aspects of insomnia. Recently, clinical trials have effectively combined CBT-I with other therapies (e.g., antidepressants) in patients with comorbid conditions (e.g., insomnia and depression). Positive results in these trials demonstrate the flexibility and strength of CBT-I with more complex presentations of insomnia.

Finally, we present a sample case of insomnia which includes the use of CBT-I. This case was not complicated with comorbidities and demonstrates many prototypical issues that arise when using CBT-I. The appendices include useful resources for assessment and treatment of insomnia, which readers are free to use in their practice.

Dedication

To my family – Mom and Dad, Kathy, Greg, and Mark – for their unconditional support and continued interest in my professional work.

W. K. W.

To my husband, Tony, who has always encouraged me to go outside my comfort zone and has steadfastly supported me in all of my professional endeavors; to Katrina and Anthony, whose love and support mean the world to me; and to my parents, who from an early age taught me to work hard and persevere in reaching my goals.

A. I. F.

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We want to acknowledge our mentor and friend, Jack Edinger, PhD. Jack has been a pioneer and industrious investigator in behavioral sleep medicine. He introduced us to the world of insomnia during our internships and continued to train us in behavioral sleep medicine after hiring each of us as research coordinators for his insomnia grants. Since then, Jack has continued to mentor and serve as a consultant in our own work. We are both indebted to him for the fundamental role he has played in our professional development.

We are also grateful for the extensive encouragement and support received from Linda Sobell, PhD, even when our progress was impeded by unexpected events. Moreover, from the initial idea for this book and its inception, as well as throughout the writing process, her editorial feedback and comments have been invaluable and have greatly enhanced the clarity of the book.

Finally, we would like to acknowledge our students and their interest and excitement in learning how to diagnose and treat insomnia. Their energy has made it easy for us to “pay it forward” and emulate Jack’s mentorship to train future behavioral sleep medicine specialists. We would also like to recognize Shantha Gowda and Danielle Millen for their contribution to the preparation of this book.

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1

Description of Insomnia

1.1 Terminology

The term *insomnia* can be used to characterize a symptom, a cluster of symptoms, or a disorder. In broad terms, *insomnia* refers to difficulty sleeping. However, the complaints of insomnia can present in a variety of ways. Insomnia is characterized by difficulty either falling asleep or maintaining sleep (e.g., waking frequently during the night, difficulty falling asleep after waking, or awakening early in the morning without the ability to return to sleep). Sleep that is not restorative (in the absence of nighttime wakefulness) has historically been included as part of the diagnostic criteria. However, in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) the criteria for insomnia do not include nonrestorative sleep.

1.2 Definition

1.2.1 Classification of Insomnia

The characteristics of the symptoms can aid with the classification of the disorder and, in turn, can inform treatment planning. There are a number of different ways that symptoms of insomnia can be classified.

Insomnia associated with difficulty falling asleep, or initiating sleep, is classified as sleep-onset insomnia, whereas difficulty remaining asleep is considered sleep-maintenance insomnia. Most commonly, however, patients present with a combination of these sleep complaints.

Insomnia can also be categorized by considering the duration of symptoms. *Acute insomnia* symptoms generally occur at least 3 times a week, last a brief period of time (less than 3 months; American Psychiatric Association, 2013), and are often easily linked to a precipitating cause (e.g., a significant life event). Symptoms associated with an acute episode often resolve without any type of intervention. Sometimes, however, the insomnia may be treated with a short trial of hypnotic medication to help the person manage troublesome symptoms. To be considered as chronic or persistent, insomnia complaints must be experienced at least three times per week for a minimum of 3 months. However, patients with *chronic insomnia* typically report symptoms that persist over a longer period of time.

Acute insomnia occurs at least 3 times per week and lasts less than 3 months

Chronic insomnia lasts 3 months or more

When comorbidities exist, the diagnosis of insomnia can be more complicated

Insomnia most often presents concurrently with medical or psychiatric conditions. In such cases, the insomnia disorder can be classified as a comorbid disorder. The term *primary insomnia* has been used to describe insomnia symptoms that cannot be attributed to another condition. However, the DSM-5 no longer utilizes the term *primary* to distinguish insomnia symptoms that are not linked to other conditions, from insomnia symptoms that occur concurrently with other disorders. When psychiatric, medical, or other sleep comorbidities exist, DSM-5 requires clinicians to specify and code the comorbid condition concurrently with the insomnia diagnosis (American Psychiatric Association, 2013). It is important to recognize that, in the case of comorbid insomnia, it is often difficult to ascertain the relationship between the insomnia symptoms and the concurrent disorder; as a result, establishing which condition presented first can be challenging. Differential diagnoses and comorbidities will be discussed further in Chapter 3 (Diagnosis, Assessment, and Treatment Indications).

Diagnostic criteria have consolidated many previous diagnoses into one of insomnia disorder

Three separate classification systems with diagnostic criteria for insomnia exist. These are the DSM-5, the *International Classification for Sleep Disorders* (3rd ed.; ICSD; American Academy of Sleep Medicine, 2014), and the *International Classification of Diseases* (11th ed., ICD-11; World Health Organization, 2018). Differences in the diagnostic criteria across these classification systems have varied over the years. Currently the DSM-5, ICSD-3, and ICD-11 share similar diagnostic criteria for insomnia.

1.3 Epidemiology

1.3.1 Prevalence

The prevalence of insomnia can be evaluated by examining the rates of insomnia as a symptom or as a diagnosable disorder. The operational definitions used to define insomnia can lead to highly variable prevalence findings. In fact, prevalence rates can vary dramatically and have been reported to range anywhere between 4% and 50% (Wade, 2011). In an epidemiological survey of community-dwelling residents, approximately 42% of respondents reported at least one symptom of insomnia (sleep-onset, sleep-maintenance, early morning awakenings, or nonrestorative sleep; Walsh et al., 2011). When considering prevalence rates of insomnia as a disorder, rates can also vary as a result of the diagnostic criteria and classification system used, with rates between 3% and 22% reported (Ohayon & Reynolds, 2009; Roth et al., 2011).

Insomnia disorder is more prevalent among women, older people, and those with comorbid conditions

Certain patient characteristics are also associated with greater prevalence of insomnia, including being female or older, as well as having comorbid medical or psychiatric conditions or being employed as a shift worker (Morin & Jarrin, 2013a, 2013b; Ohayon, 2002).

1.3.2 Economic Impact of Insomnia

Insomnia can have a significant impact on costs associated with health care utilization, medication use, and other direct costs, as well as indirect costs,

Theories and Models of Insomnia

Multiple theoretical models exist to explain how insomnia develops and is maintained

Insomnia's development and maintenance has been conceptualized in various theoretical models. These models can assist in providing a rationale for a specific treatment formulation. This chapter will briefly describe early behavioral models of insomnia and then present newer models that have refined the earlier theories by incorporating cognitive perspectives. Early biological perspectives explaining insomnia that focus on physiological hyperarousal will also be described. These initial models serve as the foundation for a discussion of more recent neurobiological models of insomnia that are being proposed as advances in neuroimaging technology permit us to better understand brain function. Finally, a framework that integrates these multiple approaches to understanding insomnia will be presented. Before discussing the insomnia models, a brief description of the normal sleep–wake process will be presented.

2.1 Fundamentals of Sleep–Wake Regulation

Behavioral interventions often incorporate a psychoeducational module that provides a rationale for treatment to facilitate patients' acceptance of, and adherence to, treatment. Similarly, CBT-I integrates a psychoeducational component that incorporates concepts associated with the sleep process. The most widely used model of sleep–wake regulation, called the *two-process model*, is presented here to facilitate a basic understanding of the sleep process and factors that can impede normal sleep.

Borbély (1982) originally described a model that considers the effect of homeostatic processes and circadian influences on sleep and wakefulness. While the two separate processes independently contribute to sleepiness and wakefulness, they occur simultaneously and interact to produce regularity in the sleep–wake cycle.

2.1.1 Homeostatic Process

Sleep drive is an important biological regulator of the sleep process

The homeostatic process (called *Process S* by Borbely) is a mechanism by which a “sleep drive” or a propensity to fall asleep gradually develops. The longer an individual is awake, the greater is the drive to fall asleep. In turn, once a sleep period is initiated, the sleep drive gradually dissipates. In this context, an increase in the drive to sleep occurs as we spend more time awake.

This homeostatic process is driven by sleep-regulating substances, of which adenosine is the most widely studied. Adenosine is a by-product produced as energy is utilized by an active, waking brain. The longer an individual is awake, the more pronounced is the buildup of adenosine and other sleep-regulating substances. Eventually the drive to sleep is overwhelming, and under normal circumstances and without any external influences that might hinder this process, an individual falls asleep.

2.1.2 Circadian Process

The second process described by Borbely is the circadian process (referred to as *Process C*) that maintains the sleep–wake cycle occurring regularly in an overall period that lasts approximately 24 hrs. This process is driven by an internal clock residing in the suprachiasmatic nucleus of the hypothalamus. This internal clock drives many biological functions that oscillate in 24-hr cycles – for example, internal body temperature, level of arousal, and the release of cortisol. Daily synchronization or *entrainment* of this internal clock helps to ensure that this 24-hr regularity is maintained. This entrainment process occurs via exposure to sunlight and other cues that can provide synchronization (such as alarm clocks or meal times). Under normal circumstances, the circadian process influences sleep by synchronizing the sleep period to occur at approximately the same time every day.

An internal clock regulates many biological functions including sleep

2.1.3 Interaction of Homeostatic and Circadian Processes

Homeostatic and circadian processes interact with each other to either maintain wakefulness or induce sleep (Figure 1). As the homeostatic drive builds up throughout the day, the propensity to sleep increases. However, arousal level – driven by circadian influence – is also building up during the day, counterbalancing the sleep drive and helping to maintain wakefulness. Into the evening hours, the alertness provided by circadian factors begins to wane, and concomitantly with the increasing sleepiness fostered by the homeostatic sleep drive, sleep eventually is induced. Over the nighttime period as sleep progresses, the sleep drive associated with homeostatic factors diminishes and the rhythmicity of the circadian process begins to build up once more to facilitate wakefulness.

Sleep drive and circadian factors work together to regulate our alertness and sleepiness

While these two biological processes work automatically to maintain a regular sleep–wake cycle, many behaviors or activities can disrupt each process. For example, in the case of homeostatic sleep drive, consumption of caffeine can inhibit the effects of adenosine on the sleep system, and napping late in the day can reduce the buildup of sleep drive. Both of these actions can, in turn, delay the onset of sleep. In turn, the circadian process can be affected by significant daily schedule changes that routinely alter bedtimes and wake-times and, in turn, negatively affect the ability to get sleepy at the same time every night. Dysregulation of these underlying sleep–wake processes may interfere with the ability to fall asleep and stay asleep, and ultimately precipitate insomnia.

Dysregulation of sleep drive and circadian factors can interfere with a normal sleep rhythm

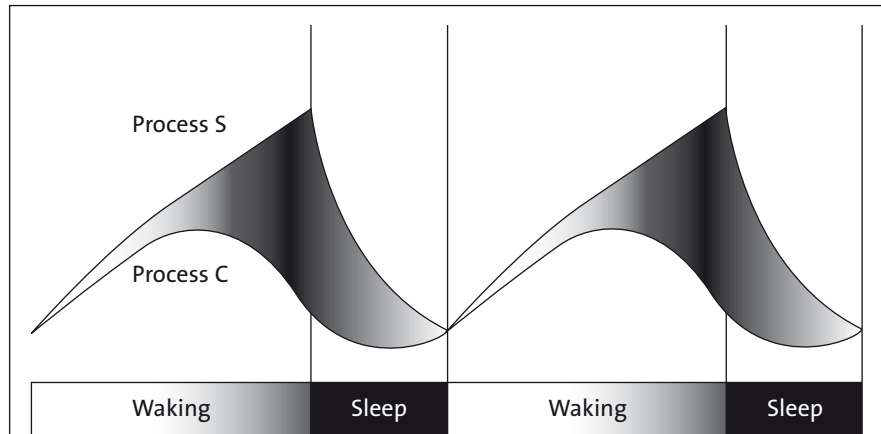


Figure 1

The two-process model for regulation of the sleep–wake cycle. Process S represents the need for sleep, which increases during wake and decreases during sleep. Process C represents the circadian system, which oscillates with a period of about 24 hrs. In this view, the times of sleep and wakefulness occur at the peak and trough of these additive processes, respectively. Reprinted with permission from “Circadian and Homeostatic Factors in Arousal” by R. Silver and J. LeSauter, 2008, *Annals of the New York Academy of Sciences*, 1129, p. 268. © 2008 by Wiley-Blackwell

2.2 Behavioral Model of Insomnia

The popularity of behaviorism in the 1960s and 1970s influenced thinking about the development of insomnia symptoms. Bootzin (1972) proposed a model of insomnia based on principles of learning theory, and more specifically, operant conditioning. His conceptualization proposed that falling asleep is an instrumental act designed to yield reinforcement – that is, sleep. Any stimulus associated with sleep (e.g., bedroom, bed), therefore, can become a discriminative stimulus for sleep. Bootzin proposed that in insomnia, the stimulus control for sleep is lacking, or alternatively, there are discriminative stimuli present that are not compatible with sleep behavior. In essence, the environment and stimuli that are traditionally associated with sleep are no longer discriminative stimuli for sleep behaviors and instead are associated with wakefulness. For individuals experiencing insomnia, being awake in bed increases arousal and frustration which, in turn, leads to greater concern and anxiety about falling asleep and/or staying asleep. Moreover, behaviors such as tossing and turning in bed, watching the clock constantly, and catastrophizing about lack of sleep occur frequently. During this process, the surrounding environment begins to be associated with the unpleasant experience of being awake, and stimuli that normally should be discriminative for sleep (such as the bed or bedroom) are no longer soporific and become discriminative cues for wakefulness. Often individuals with insomnia will report feeling sleepy

High arousal combined with incompatible sleep behaviors can become cues for wakefulness in bed

Diagnosis, Assessment, and Treatment Indications

3.1 Diagnosis of Insomnia

The diagnostic criteria for insomnia have evolved over the past several iterations of the psychiatric (i.e., DSM) and sleep disorder nosologies (i.e., ICSD), and the most recent volumes have introduced a significant simplification in the criteria for insomnia. The current criteria for *insomnia disorder* in the DSM-5 (American Psychiatric Association, 2013) and the ICSD-3 (American Academy of Sleep Medicine, 2014) were developed in parallel so there would be consistency between the nosologies. At the most basic level, the diagnosis of insomnia disorder necessarily requires a subjective complaint of difficulty falling or staying asleep, or of waking up earlier than desired. Mixed insomnia, which is some combination of difficulty falling asleep or staying asleep, or of waking up too early, is the most commonly reported type of insomnia (Morin, LeBlanc, Daley, Gregoire, & Merette, 2006). Isolated sleep-onset insomnia is less common than mixed insomnia. These nighttime symptoms should occur at least three times per week. Insomnia disorder is called persistent if it lasts 3 months or longer, and considered acute if it lasts less than 3 months. Physiological evidence of difficulty sleeping (i.e., a sleep study) is not necessary for an insomnia diagnosis to be made (Littner et al., 2003).

Insomnia can include combined complaints of difficulty falling/staying asleep or waking up early

Clinical Pearl **When a Sleep Study Is Indicated**

It is not uncommon for patients with insomnia to expect that spending the night in the sleep lab to record their sleep would be beneficial for gaining a better understanding of their insomnia. However, an overnight sleep study is not required for a diagnosis of insomnia. Patients come to the clinic complaining about being awake during the night, and a sleep study most likely will confirm that they are awake during the night. Therefore, typically no new information is gained from the sleep study to help with diagnosis and treatment, and conducting a sleep study in patients with insomnia is not an efficient use of sleep center resources. However, if patients do not improve after their insomnia is treated, or if they are at risk for sleep apnea, a sleep study is appropriate. Some patients may be more focused on physical symptoms or have health anxiety and may strongly push for a sleep study, believing that it will reveal the source of the insomnia. Providing information regarding the purpose of a sleep study (e.g., detecting breathing problems during sleep) can be useful with these patients.

The diagnosis of insomnia also requires a complaint of daytime dysfunction

Sufficient opportunity to sleep must be allotted before a diagnosis of insomnia can be considered

Clinical judgment is important when comorbid conditions exist, to choose best treatment for patient

Besides the nighttime complaints, the sleep difficulty must cause some daytime distress for the diagnosis to be made. Daytime symptoms commonly include fatigue or sleepiness, difficulty with attention and concentration, irritability, reduced motivation or energy, error proneness, or concerns about or dissatisfaction with sleep. Poor sleepers must also provide themselves with an adequate opportunity for sleep (e.g., reporting that “I’m not sleeping, and I’m tired during the day,” would usually justify a diagnosis of insomnia, but if the complaint derives from only allocating 4 hrs for daily sleep, the individual would not be diagnosed with insomnia).

Lastly, a diagnosis of insomnia requires that the sleeping difficulty cannot be adequately explained by another psychiatric, medical, or sleep disorder. Unfortunately, this criterion may not always be clear or straightforward and can require clinical judgment. For example, patients may decline treatment for PTSD because they fear that therapy may reduce their hypervigilance to their environment. From the patient’s perspective, while hypervigilance is important for safety, the PTSD symptom (i.e., hypervigilance) is interfering with their ability to fall or stay asleep. In this case, insomnia would not be diagnosed, because the trouble sleeping would be better explained by the psychiatric disorder (i.e., in this case, the PTSD). However, if such patients receive treatment and appreciate symptom relief from PTSD but continue to have trouble sleeping, then insomnia could be diagnosed.

In depressed patients who also complain of sleep difficulty, deciding to make the diagnosis of insomnia is less clear. For patients with an untreated mood disorder, while the priority will most likely be initiating psychiatric care for that condition, a question arises about whether a comorbid insomnia disorder diagnosis should be considered. In such cases, decisions are best made on a case-by-case basis. If the mood disorder is severe or the patient is in crisis or unstable, depression may better account for the patient’s insomnia. However, for a patient with mild or moderate depressive symptoms who is seriously complaining about difficulty staying asleep at night, a comorbid diagnosis of insomnia may be appropriate.

The above definitional changes related to the insomnia disorder represent a significant simplification and consolidation of the diagnostic criteria. In previous editions of both the psychiatric (DSM) and sleep nosologies (ICSD), diagnosticians had a more complicated task of deciding among several insomnia diagnoses. Previous versions of the DSM separated insomnia into primary and secondary subtypes. Primary insomnia was thought to “have a life of its own” with no other primary diagnosis as the cause. Secondary insomnia was thought to be caused by some other primary psychiatric, medical, or sleep disorder diagnosis (e.g., depression, restless legs syndrome, chronic pain); if the primary condition was treated, it was assumed that the insomnia would resolve.

Previous editions of the ICSD, designed by sleep disorder specialists, used a different approach. Several different diagnoses of insomnia were created. Because sleep specialists created this nosology, the diagnoses tended to correspond to theoretical models of insomnia. For example, a diagnosis of *psychophysiological insomnia* from the ICSD would have applied to someone appearing to have cognitive or physiological hyperarousal as the main feature of their sleep difficulty. *Inadequate sleep hygiene* may have been best applied to someone taking daily naps and frequently changing bedtimes and

Treatment of Insomnia

4.1 Methods of Treatment

4.1.1 Sleep Psychoeducation

Sleep education is an important component of CBT-I

Essential to most treatment approaches presented here is providing a basic understanding about sleep needs, normal changes in sleep that accompany aging, the effects of sleep deprivation, and the role of the sleep drive.

The recommendation for 8 hrs of sleep each night is not universal, because sleep needs vary

Sleep Needs

When queried about personal sleep needs, most patients will automatically respond that they need 8 hrs of sleep nightly. The National Sleep Foundation (2015) has published sleep duration recommendations that cover the lifespan. According to their recommendations, young adults and adults require approximately 7–9 hrs of sleep, while the recommended sleep duration for older adults is between 7 and 8 hrs (Hirshkowitz et al., 2015). However, the authors note that sleep needs can vary for different individuals, with some people needing more or less sleep than that indicated by these recommendations. The critical point to emphasize when addressing this item with a patient is the importance of avoiding preconceived beliefs that 8 hrs is the “magic number.” Much more important is the need to determine each patient’s required sleep duration based on individual data rather than relying on a recommended average. With this discussion, patients will often raise concerns about how they will be able know what is right for them. The clinician can explain that individual sleep needs are determined based on the amount of time that will allow them to *wake up feeling refreshed*. One of the therapeutic goals is to ascertain this amount of sleep. Through a collaborative process between the clinician and the patient, the initial amount of TIB prescribed is gradually adjusted until the patient reports feeling well-rested. This process is not always straightforward and usually requires several weeks of refining the TIB prescription before this goal is met.

Sleep and the Aging Process

Often patients’ sleep quality and quantity expectations are based on sleep experiences from their youth. As we age, however, sleep changes are normal. Specifically, sleep becomes more fragmented, with more arousals and awakenings possible. Moreover, with age, there can be a slight reduction in sleep duration needs as well as a circadian shift toward earlier bedtimes and waketimes. A discussion that explains these normal aging-related changes and addresses related sleep misconceptions can modify unrealistic sleep expectations of patients and will facilitate treatment.

Time Spent in Bed

Individuals with chronic insomnia are frequently distressed by the thought that they are not sleeping enough, worry about the potential negative consequences of sleep loss, and compensate by increasing the time they allocate for sleep. The assumption that they are not sleeping enough is partially reinforced by their extended periods of wakefulness at night. Patients, however, may not understand that the experienced sleep fragmentation can be a result of spending too much time in bed – that is, they may be getting about 7 hrs of sleep, but the consistently extended time that is spent in bed breaks up the 7-hr sleep period across the 8, 9, or 10 hrs they may be lying in bed. This fragmented sleep is commonly perceived as poor-quality sleep. As described above, TIB is gradually adjusted during treatment to facilitate a more consolidated sleep period.

Clinical Pearl

Conditioned Arousal Leading to Poor Sleep

The sleep fragmentation that occurs as a result of extended time in bed often leads to increased distress and anxiety if the patient remains in bed during the periods of wakefulness. This increased arousal can evolve into a conditioned arousal state so that over time, the bed, and even the bedroom, can become associated with wakefulness and arousal. The conditioned arousal can further compound the sleep difficulties that the patient experiences as a result of extended time in bed. In extreme cases, some patients have called this a “bedroom phobia” or come to refer to their bedroom as a “torture chamber.” Patients use these descriptions because the powerful association of arousal and wakefulness with the bedroom is in conflict with the desire to sleep, which leads to frustration, anxiety, and anger.

Sleep Drive

Patients with insomnia also routinely focus on the nights they sleep poorly and frequently fail to recognize that, interspersed between poor sleep nights, there are nights of better sleep. It is important for clinicians to explain the body’s innate ability to experience a good night’s sleep after a couple of bad nights. The concept of the natural (i.e., homeostatic) sleep drive that helps us to sleep after we have been awake for many hours can reinforce this point by illustrating that a good night’s sleep will typically follow a couple of poor nights. Sleep diaries completed prior to treatment can facilitate the demonstration of this concept. Furthermore, the buildup of the sleep drive with longer periods of wakefulness should also be addressed in the context of napping, as a daytime nap taken in an attempt to “make up for” the previous night’s lost sleep only hinders the body’s natural ability to fall asleep at a desired time the following night.

Sleep diaries are useful to demonstrate sleep concepts such as sleep drive

Circadian Rhythm

Our internal clocks play a significant role in our ability to sleep. At night, this internal clock ramps down the alerting signal as we prepare to sleep. In the morning, the clock ramps up and sends out an alerting signal to wake up and begin our day. This decrease in alertness at night and increase in the morning provides a *sleep window*, which is when optimal sleep will occur. Attempting to sleep outside this window (i.e., when the internal clock is sending out a

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Appendix: Tools and Resources

Appendix 1: Recommended Resource Websites

Appendix 2: Insomnia Assessment

Appendix 3: Sleep Diary and Instructions

Appendix 4: Sample Sleep Diary of Patient L.F. and Calculation Instructions

Appendix 5: Clinician Psychoeducation Sample Script

Appendix 6: Guidelines for Better Sleep

Insomnia History

1. Describe your current sleep problem:

How long have you had this problem? _____

How long have you been concerned with this sleep problem? _____

Has the severity of the sleep problem changed over time? No Yes

If yes, **describe** _____

When you were a child, did you experience any sleep difficulties? No Yes

If yes, **describe** _____

2. What do you think contributes to your sleep problem? Can you identify the cause of your sleep difficulties?

3. How many hours of sleep do you feel you need each night to feel rested? _____

Generally, how many hours of sleep do you get a night? _____

Think about nights when you consider you slept well, how many hours would you say you sleep on those good nights? _____

What about on bad nights, how many hours would you say you sleep on those nights? _____

4. What would you estimate is the length of time you generally take to fall asleep? _____

How long might that be on a good night? _____

How long might that be on a bad night? _____

5. Consider your awakenings throughout the night. After falling asleep, how many awakenings would you estimate you experience on a bad night? _____

What about on a good night? _____

How long would you say each of your awakenings last? _____

6. How many times per week do you have difficulty:

Falling asleep _____ (times/week)

Returning to sleep _____ (times/week)

Waking up too early _____ (times/week)

7. How do you generally feel during the day (e.g., concentration or memory difficulties, fatigue or irritability)?

Is it different depending on whether you've had a good night or a bad night of sleep?

Describe: _____

Sleep Habits

8. What time do you normally get into bed? _____

What time do you normally try to go to sleep? _____

What time do you normally wake up? _____

Guidelines for Better Sleep

For sounder, more reliable sleep, follow all of the guidelines below.

- 1. Establish a standard wake-up time AND stick to it every day no matter what your sleep is like on any particular night.**
 - a. You cannot force yourself to go to sleep, BUT you can control the time when you wake up.
 - b. The goal is to try to avoid different wake-up times and to establish a standard sleep–wake schedule because constantly changing your sleep–wake schedule can disrupt your sleep.
 - c. By waking up at the same time every day, you’ll notice that you begin to get sleepy at about the same time each night, and over the course of time, this will help you to obtain the sleep you need.

- 2. Never spend long periods of time awake in bed. When you are unable to sleep, get up and go to another room. Return to your bed only when you feel sleepy again. Continue to do this each time you find yourself spending long periods of time awake in bed.**
 - a. Being awake for a long period of time in bed can cause you to feel frustrated, and you may find yourself worrying about your lack of sleep. This inevitably makes it harder for you to fall asleep.
 - b. Give yourself *20 minutes* to fall asleep. If you do not fall asleep within 20 minutes, get up, go to another room and only return to the bed once you feel sleepy again.

- 3. Avoid naps.**
 - a. Naps during the day may reduce your sleepiness and fatigue but also can delay the time when you start to feel sleepy at night.

- 4. Use the bed only for sleeping. Do NOT read, watch TV, use electronic devices, eat, etc. Sexual activity or illness are the exceptions to this guideline.**
 - a. By doing these activities in bed, you are training yourself (not intentionally) to be awake in bed when you engage in these wakeful activities.
 - b. If you consistently avoid these activities, the bed will gradually become linked with sleep, and you will find it easier to fall asleep.
 - c. If your bedpartner does wakeful activities in bed ask them to alter these activities during the treatment.

- 5. Avoid worrying, thinking, planning, etc. in bed. If such mental activities come on automatically in bed, then get up and go to another room, stay up until you feel sleepy and the mental activities don’t disrupt your sleep. Once in bed, get up again if sleep does not come quickly (20 minutes).**
 - a. Remember, doing wakeful activities in bed involves unintentionally training yourself to be awake in bed.
 - b. Try to schedule time during the day (or in the early evening) to focus on these mental activities with the goal of anticipating, planning ahead, and working through some of the worries. If these worries are reviewed during the day, there is less chance of having them in bed or that they may keep you up at night.

Clear, concise, and expert guidance on evidence-based assessment and treatment of insomnia

About 40% of the population experiences difficulty falling or staying asleep at some time in a given year, while 10% of people suffer chronic insomnia. This concise reference written by leading experts for busy clinicians provides practical and up-to-date advice on current approaches to assessment, diagnosis, and treatment of insomnia. Professionals and students learn to correctly identify and diagnose insomnia and gain hands-on information on how to carry out treatment with the best evidence base: cognitive behavioral therapy for insomnia (CBT-I). The American Academy of Sleep Medicine (AASM) and the American College of Physicians (ACP) both recognize CBT-I as the first-line treatment approach to insomnia. Appendices include useful resources for the assessment and treatment of insomnia, which readers can copy and use in their clinical practice.

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“Insomnia provides a coherent, accurate, and comprehensive overview of the prevalence, diagnosis, assessment, and treatment of insomnia. Based on the latest science and with detailed patient-therapist dialogues and specific clinical tools, this book offers a clear blueprint for providing evidence-based care to the millions of people suffering from this highly common and debilitating sleep disorder.”

J. Todd Arnedt, PhD, Associate Professor of Psychiatry and Neurology, Director, Behavioral Sleep Medicine Program, Michigan Medicine, University of Michigan, Ann Arbor, MI

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