Chapter 7
Management of Sleep Disorders – Cognitive Behavioral Therapy for Insomnia

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Abstract Insomnia is a common complaint in the general population. Hypnotics are the most common treatment for insomnia, despite some unwanted effects such as tolerance and dependence. Cognitive Behavioral Therapy for Insomnia (CBT-I) was shown to be effective in the treatment of chronic insomnia. Recent studies comparing CBT-I treatment with hypnotics further demonstrated that the CBT-I treatment was at least as effective as hypnotics during acute treatment, and may be more effective than medication during long-term follow-ups. This chapter describes the role of neurophysiological and psychological factors in the regulation and dysregulation of sleep. Further, the rationales and procedures of CBT-I are introduced. Lastly, the application of CBT-I with modern information technologies is discussed.

Keywords Insomnia • Cognitive behavioral therapy for insomnia • Internet

Insomnia is a common complaint in the general population. The prevalence of insomnia varies depending on its definition. In a survey study conducted in seven European countries (France, the United Kingdom, Germany, Italy, Portugal, Spain, and Finland) with a representative sample consisting of 25,579 subjects, approximately 37% of the subjects had sleep complaints, 9.8% had sleep symptoms and daytime consequences, and 6.6% satisfied the DSM-IV diagnostic criterion for insomnia [49]. The disorder is more common among women, older adults, and those with medical and psychological disorders [8, 26].

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Hypnotics are the most common treatment for insomnia, despite some unwanted effects such as tolerance and dependence [45]. Many psychological interventions have been shown to be effective in the treatment of chronic insomnia. In several meta-analyses and reviews, it was shown that psychological treatments for insomnia could benefit around 70–80% of patients with insomnia [30, 39–41, 43, 44]. The 2005 State-of-the-Science Conference of the National Institutes of Health of United States also recognized the effectiveness of psychological and behavioral therapies to treat chronic insomnia in adults [45]. These treatment techniques are usually combined into Cognitive Behavioral Therapy for Insomnia (CBT-I). Many studies have supported the efficacy of CBT-I. Recent meta-analyses reported moderate to large effects of CBT-I in improving sleep latency, duration of wakening, and sleep quality in patients with primary insomnia [39, 44]. Recent studies comparing CBT-I treatment with hypnotics further demonstrated that the CBT-I treatment was at least as effective as hypnotics during acute treatment and may be more effective than medication during long-term follow-ups [27, 40, 41]. In addition, the subjects were more satisfied with CBT-I than with medication [40, 41]. Therefore, there is a great need to introduce insomnia patients to this evidence-based treatment option.

In this chapter, we will begin with an introduction of the neurophysiological mechanisms of normal sleep regulation, and then provide a framework to conceptualize the contribution of behavioral factors for the development of insomnia. Further, we will describe the rationales and procedures of CBT-I. Lastly, we will discuss the application of CBT-I with modern information technologies.

7.1 The Pathological Model of Insomnia

7.1.1 Neurophysiological Regulation of Sleep

Insomnia can be construed as a disruption of the neural systems that normally regulate the sleep and waking cycle. The neural systems that regulate sleep and waking may include a homeostatic system that accumulates sleep propensity with each passing hour of wakefulness, a circadian process that generates a biological rhythm of sleep and wake tendency over the course of a day, and an arousal system that promotes wakefulness and counteracts sleep drive [6, 33, 55].

The function of the homeostatic system is to maintain an adequate amount of total sleep over successive nights. The level of sleep drive present on the basis of this homeostatic mechanism is, at any given time, determined by prior durations of sleep and wakefulness. Sleep deprivation leads to an augmented sleep drive and an increased likelihood of accumulating extra recovery sleep during subsequent night(s), thereby restoring the balance. Oversleeping, by contrast, reduces the homeostatic sleep drive, leading to shorter or lighter stints of sleep.

The circadian system is regulated primarily by an internal clock in the hypothalamus that generates a rhythm of sleepiness and alertness independent of prior sleep history. Studies with both animals and humans have identified the genetic basis of

this cycle [29, 63]. The typical endogenous circadian cycle in human beings has a period of slightly over 24 h [11, 12]. Therefore, there is an innate tendency for our bedtimes and rising times to slowly drift later in time. This propensity is often revealed during vacation periods, when we are not as constrained by externally imposed schedules. Exposure to environmental time cues, especially daylight in the morning, can stabilize an endogenous circadian rhythm that would otherwise "free run." The circadian and the homeostatic systems interact reciprocally with each other to initiate and maintain sleep. Under normal conditions, homeostatic drive is built up during the day when circadian propensity for wake is high. Circadian wake propensity then falls off in the evening that allows sleep to occur. Circadian sleep tendency reach its peak and maintain the sleep in the middle of night when homeostatic sleep drive has been partially satiated. Sleep disturbances thus may occur when the homeostatic system mis-phased with the timing of the circadian system.

The arousal system counteracts the sleep drive by promoting arousal. The system could be activated by internal thoughts and emotions as well as by external stimulation. It can be viewed as a mobilizing system intended to arouse an individual when he or she is at risk. In contrast to the homeostatic mechanism that gradually strengthens the sleep drive as our waking hours pass, alertness can soar in a moment, as required in an emergency. While this arrangement may be adaptive from an evolutionary standpoint, it does work against the prospects for sleep in insomnia patients. Their careful preparations for sleep can be overturned in an instant by an errant thought.

Several models highlighting the role of arousal as a cause of insomnia conceptualize the disorder as a manifestation of hyperarousal [4, 50]. Individuals with insomnia have been shown to have elevated activities of autonomic nervous system, indicated by higher metabolic rate, body temperature, heart rate, urinary cortisol and adrenaline excretion, skin conduction, and muscle tension [3, 62], as well as increased cortical arousal, as indicated by faster EEG frequencies [17, 32, 51], enhanced activating components, and decreased inhibitory components in event-related potentials [66] and PET [48] during sleep initiation. These results further suggest that insomnia may result from an inability to lower general attention or the arousal process and an impairment in the sleep-specific inhibitory process associated with sleep initiation.

7.1.2 Psychological and Behavioral Factors Affecting Sleep

Although the systems that regulate sleep are physiologically based, they are susceptible to interruption by psychological and behavioral factors. Sleep is readily deferred, at least in the short run, in the efforts to complete tasks deemed sufficiently important or to maintain vigilance in the face of real or perceived threats. In patients with chronic insomnia, the efforts to fall asleep and the threat of an upcoming sleepless night may lead to increased arousal that further exacerbates the already fragile sleep.

Transient insomnia resulting from acute stress is a nearly universal experience. It may be associated with autonomic nervous system (ANS) activation and hormone release subserving the normal stress reaction [62]. Arousal in chronic insomnia patients is however often associated with excessive cognitive activities around bedtime. Previous studies have provided evidence suggesting that anxious or dysphoric thoughts are not conducive to sleep [15, 16, 19, 38, 46, 64]. Insomnia is clearly associated with worrying that has a "real world" basis, or with exposure to traumatic events. Furthermore, in chronic insomnia patients, arousal is often associated with concern that centers on sleep itself. After experiencing frequent sleepless nights, recalling long hours of nocturnal wakefulness and consequent daytime listlessness as well as anticipating more of the same may elicit further arousal and disrupt sleep. Arousal can also be a learned response through classical conditioning. After repeated pairings of bedroom environment with the experience of sleeplessness, bedtime, with its attendant rituals, begins to offer contextual cues for arousal rather than sleep [5].

Some misconceptions about sleep may also be adverse to poor sleepers. Patients who have unreasonable expectations about sleep may be frustrated by not being able to fulfill these expectations. Poor sleepers who feel that their insomnia is "inevitable" and will necessarily lead to dire health consequences may also become frenzied by bedtime. Directly challenging dysfunctional thinking about sleep through education and cognitive therapy has been shown to improve sleep [21, 42]. Furthermore, the extent to which the anxiety-provoking cognition decreased following CBT-I was associated with the level of improvement after treatment [42]. Misconceptions about sleep may disrupt normal sleep regulation through maladaptive sleep practices. Over-concern about the consequences of poor sleep often leads to pernicious behavior practices that originate in the desire to compensate for poor sleep or its daytime effects [20]. It is common to hear patients sleeping into the morning following a disrupted night when their schedules allow or of taking an afternoon nap. These responses may bring short-term relief from the effects of sleep loss, but at the cost of reducing the amount of sleep drive available to induce sleep at bedtime or disrupting the circadian processes that regulate sleep and wakefulness. Figure 7.1 provides a simplified framework that shows how cognitive and behavioral factors may affect sleep through the mediation of the neurophysiological systems that regulate sleep and wakefulness. The techniques of CBT-I are aimed at adjusting these factors to help patients regain the balance of the neurophysiological regulation of sleep and wakefulness.

7.1.3 The 3P Model of Insomnia

Over time, the physiological, psychological, and behavioral factors may all contribute, in varying degrees, to the development of insomnia in an individual patient. The waxing and waning of these factors along with their interaction can complicate

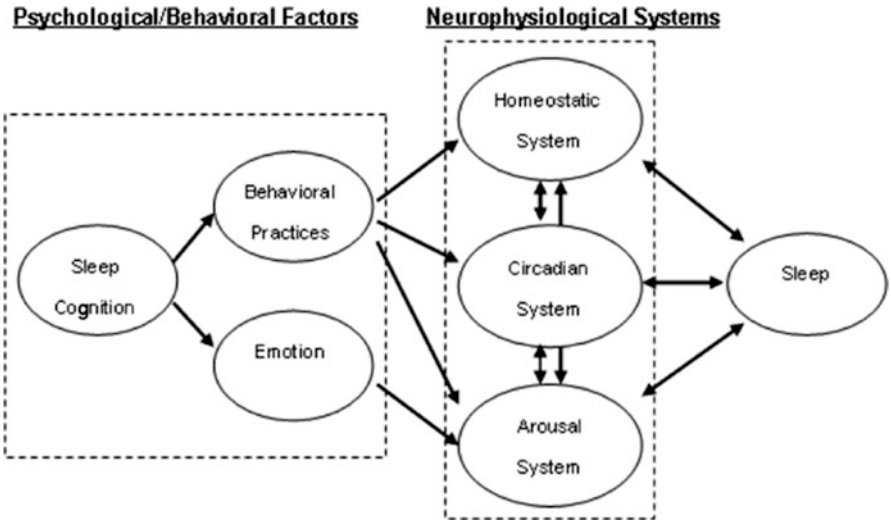

Fig. 7.1 A conceptual model shows that psychological/behavioral factors influence sleep mediated by the neurophysiological systems for sleep regulation (Adapted from Yang et al. [67])

the clinical assessment of the disorder. Spielman introduced a model that is very useful for understanding the genesis of insomnia in a patient. The 3P Model of insomnia groups the etiological factors of the disorder temporally into the Predisposing factor that sets the stage for its development, the Precipitating factor that triggers its onset, and the Perpetuating factor that maintains the sleep difficulty in the long term [56] (see Fig. 7.2).

Difficulty sleeping can arise from inherited predispositions pertaining to each of the three neurophysiological systems subserving sleep and wakefulness described above. These predisposing characteristics are often present for years before chronic insomnia takes hold. Some individuals possess an inherently weak homeostatic sleep drive. Others are under the sway of an atypical circadian system. For example, the endogenous circadian clock of those with a "night owl" sleep pattern under typical circumstances may have a greater tendency to be shifted later in time; thus, they may experience sleep initiation difficulties following occasions that require staying up late. Finally, the sleep of those who have hyperaroused constitutions may be especially vulnerable to disruption when encountering stressful events [13]. Previous studies have also shown that those who tend to internalize conflicts through self-inhibition, denial, or suppression seem to be more susceptible to the impact of acute stress [28].

The events or stressors that trigger the onset of insomnia in these examples are the precipitating factors. Patients tend to label these precipitating events as the "cause" of their insomnia since they usually appear just prior to or concurrently with the sleep disturbance. These factors may be as dire as divorce or serious illness, or as

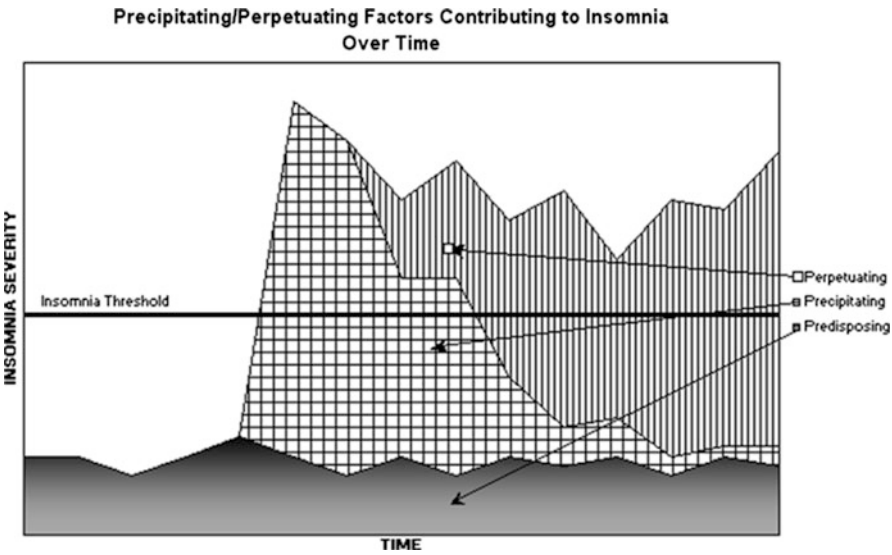

Fig. 7.2 The 3P model of insomnia, in this case illustrating the major contributions of Precipitating and Perpetuating factors and the minor contribution of Predisposing factors (Adapted from Spielman et al. [58])

elating as a newborn or a job promotion. However, in chronic insomnia, the sleep disturbance lasts even after the impact of the precipitating event has subsided or resolved. The cognitive and behavioral factors described above often become the major contributing factors and maintain the sleep disturbance.

These perpetuating factors are the major targets of CBT-I in patients with chronic insomnia. However, other factors in the 3P Model should not be overlooked. As predisposing characteristics increase the risk of developing insomnia, any mitigation of their contribution would be helpful. Similarly, addressing the precipitating factors of sleep loss directly, such as marital conflicts or performance anxiety, can prevent the recurrence of sleep disturbance after the perpetuating factors are controlled.

7.2 The Structure of CBT-I

CBT-I is usually conducted in groups with a structured program or is administered individually with selected techniques tailored for an individual. In order to facilitate the treatment process, it is suggested that patients be prepared before instituting the treatment techniques. First, just like all types of psychotherapies, a good conceptualization of the patients' problem may add to the effectiveness of the treatment. Prior to initiating CBT-I, it is important to conduct a thorough evaluation to identify possible contributing factors. Patients with sleep disturbances often

have some misconceptions regarding the causes of their sleep problem or may be puzzled by their condition. The feeling of being out of control and the mystery of what is causing the problem could generate anxiety that may further disrupt their sleep. Therefore, sharing the conceptualization with the patient is therapeutic in that such an approach restores some modicum of control and reduces unnecessary anxiety. This understanding, furthermore, may motivate the patients to practice the behavioral techniques.

In addition to the evaluation and etiological formulation, providing a description of the treatment procedure as well as the rationale of the treatment may comprise a comprehensive first treatment session. It is useful for patients to know what to expect from the treatment. A series of office visits or group sessions are scheduled on a weekly or biweekly base while maintaining a sleep log in between sessions. For both individual and group CBT-I, the entire program usually takes 4–8 weeks, although an abbreviated, 2-session CBT-I was also reported to be effective in primary care settings [14]. In addition, the patients should expect the effects of the behavioral techniques to be manifest in a few weeks. Unrealistic expectations of immediate improvement may lead to premature demoralization. The patient should also understand that reliably carrying out behavioral practices is crucial for the treatment to be effective.

Different treatment techniques will be introduced during the course of the treatment sessions. As mentioned earlier, the procedure of CBT-I could be a standardized structured program or could be tailored according to the etiological formulation of an individual session. The patients may be required to change their sleep habits and daily life routines and to practice some behavioral techniques in between sessions. In subsequent sessions, the "prescribed" sleep and behavioral practices may be adjusted depending on the outcomes following the treatment and the difficulties encountered in carrying out the techniques. Table 7.1 provides an example of a structured CBT-I program that is conducted in our treatment outcome studies.

7.3 CBT-I Techniques

CBT-I comprises three components: an educational, a cognitive, and a behavioral component. The educational component aims to enhance the understanding of basic mechanisms of sleep regulation, etiological factors of insomnia, and sleep and daily life practices that are compatible with good quality sleep. The behavioral component includes specific techniques that enhance the quality of sleep by adjusting the sleep schedule and employing relaxation training that can reduce tension and anxiety. Finally, the cognitive component aims to correct beliefs and attitudes about sleep that may be anxiety-provoking and may lead to maladaptive sleep practices. In the following section, we will describe the rationale and principles of the techniques.

Table 7.1 A sample of a structured CBT-I program

Week 1	Education:
	Mechanism of sleep regulation
	Neurophysiological model of insomnia
	The 3P model of insomnia
Week 2	Education:
	Sleep hygiene education and pre-sleep behaviors
	CBT-I techniques:
	Stimulus control
	Sleep restriction: introduction
	Relaxation training: diaphragmatic breathing
Week 3	Education:
	Circadian rhythm and sleep
	CBT-I techniques:
	Sleep restriction: sleep schedule titration
	Relaxation training: progressive muscle relaxation
Week 4	Education:
	Hypnotic introduction and tapering
	Sleep disturbance
	CBT-I techniques:
	Sleep restriction: sleep schedule titration
Week 5	CBT-I techniques:
	Cognitive restructuring
	Sleep restriction: sleep schedule titration
	Relaxation training: direct relaxation
Week 6	Education:
	Relapse prevention
	CBT-I techniques:
	Review of CBT-I techniques

7.3.1 Sleep Hygiene Education

Sleep hygiene refers to the practices of everyday living and sleep-related activities that promote good quality sleep and those that make sleep more resistant to disruption. The objectives of sleep hygiene education are to improve basic knowledge about sleep and modify counterproductive sleep practices [24]. Understanding the mechanisms of sleep regulation and the etiology of their sleep problem empowers the patient and eliminates unnecessary worry about their insomnia. It also provides the rationale for sleep promoting behavioral practices. In terms of sleep hygiene practices, the clinician reviews daily life practices and sleep-wake habits with the patient and identifies a set of practices that are not consistent with good sleep hygiene. The patient is asked to refrain from maladaptive activities and, in some cases, engage in sleep promoting behaviors.

Sleep hygiene education is usually a standard part of a more comprehensive treatment program. Sleep hygiene education alone has been shown to be less effective than the other behavioral treatments [30]. Many patients are aware of

sleep hygiene practices, but they do not believe that the practices will produce significant changes in their sleep. It is important to convey to such individuals that insomnia is the result of the interaction of multiple factors. Eliminating maladaptive sleep hygiene practices may not solve the problem. A successful treatment result, however, may not be achieved without changing poor sleep hygiene practices.

7.3.2 Cognitive Therapy

As described above, misconceptions about sleep may lead to increased arousal and/or sleep-disruptive behavioral practices. Changes of dysfunctional thoughts can reduce worries, and therefore, break the vicious cycle that leads to arousal. Part of the disruptive cognitions about sleep may be corrected with sleep hygiene education. Cognitive restructuring, on the other hand, addresses sleep-disturbing cognitions directly, and replaces these thoughts with more realistic thoughts and positive ideas [22, 37]. In conducting this part of CBT-I, dysfunctional sleep cognition should be identified by discussing the worries of patients prior to sleep or with the assistance of self-report questionnaires [37]. After the belief has been identified, the validity of the belief can be challenged with evidence from scientific knowledge as well as the observation of the experiences of the patient—for example, if a patient believes that "if I don't get enough sleep, I will perform poorly on my job." The clinician may educate patients about the scientific facts of the effect of a few nights of poor sleep on performance and ask the patients to rate the level of job performance along with maintaining a sleep log. The association between job performance and sleep quality may not be as consistent as expected by the patient. If the belief is proved to be valid, the utility of the thoughts can be examined. This can be done by querying the consequences of maintaining this belief. For example, although poor sleep usually leads to poor performance on the job, constantly thinking about it does not help matters. The clinician can question the patient about how he/she feels and what he/she will do if the patient continues ruminating about this belief. It is very likely that the belief may increase anxiety and negative mood that may further disrupt his/her sleep.

7.3.3 Stimulus Control Therapy

The objective of stimulus control therapy is to disrupt the association of bedtime cues with wakefulness and arousal. The patients are instructed to get out of bed if unable to fall asleep and return to bed when ready to sleep. The following are specific instructions followed in stimulus control therapy: (1) go to sleep only when feeling sleepy; (2) do not use the bed or bedroom for other activities except for sleep (sexual activity is the only exception); (3) if you do not fall asleep within approximately 20 min, go into another room and do something relaxing; (4) go

back to bed only when feeling sleepy again; (5) repeat the procedure of getting out of bed if you still cannot fall asleep rapidly; (6) get up at the same time each morning regardless of how much you have slept; and (7) avoid napping in the daytime. Over time, the repeated association of bedroom cues with rapid sleep onset brings sleep under the stimulus control of the bedroom environment [5].

These seemingly simple instructions are difficult to carry out regularly among many patients. Initially, patients will spend considerable time out of bed, and thus, suffer some sleep loss. It is important to motivate the patients by letting them fully understand the rationale of this treatment procedure. Although daytime functioning and mood may be impaired temporarily, the partial sleep deprivation will foster both rapid sleep onset and increased sleep. Further, the clinician may need to prescribe activities to perform during the night that are not too taxing or activating for the patients who do not know what to do when they are unable to sleep during the night.

7.3.4 Sleep Restriction Therapy

The basic assumption underlying sleep restriction therapy is that the homeostatic sleep process can self-correct sleep loss. Sleep restriction therapy promotes sleep by inducing mild sleep loss initially and gradually increasing sleep time after sleep is stabilized. On the other hand, this procedure, which is the same as the stimulus control therapy, can also break the maladaptive association between anxiety and bedtime cues by decreasing the chance of lying awake in bed [18, 57]. The specific procedures of this therapy are as follows:

- Patients complete a sleep log that records the daily sleep pattern over a 2-week period.
- The average total sleep time per night during this 2-week period is then prescribed as the time in bed for the following week. To avoid the effects of severe sleep deprivation, the minimum time in bed is never set below 4.5 h. Lying down or napping outside the scheduled bedtimes is not permitted.
- Patients fill out a sleep daily log to record bedtimes and estimated total sleep time. Sleep efficiency (SE) (estimated total sleep time/time in bed × 100%) is evaluated every week.
- Prescribed time in bed is adjusted by three criteria: (1) when the mean SE is equal to or more than 90% (85% in older individuals), then the subject's time in bed is increased by 15 min by setting the retiring time earlier; (2) if the mean SE is less than 85% (80% in seniors), the time in bed is decreased by 15 min; and (3) when the SE is between 85 and 90% (80–85% in seniors), then the time in bed remains the same.

Some modified procedures to adjust time in bed have been proposed and utilized. One way is to increase the time in bed progressively by 15 or 30 min each week until the patient is spending 7 h in bed. Further changes of time in bed may be made based on daytime functioning, fatigue, and sleepiness [54]. Further, a sleep compression

procedure has been utilized to gradually reduce time in bed in 5 weeks instead of curtailing it abruptly. This procedure was found to be helpful for elderly patients with insomnia [31].

Similar to the instructions for stimulus control therapy, patients should be told to expect mild sleep loss and daytime deficits at the initiation of the treatment. It should be stressed that short-term sacrifice will produce long-term gains. Patient may have difficulty resisting the temptation to go to bed earlier than the preset schedule. Clinicians should provide the rationale and encourage the patient to continue following the instructions. For some patients, it may be necessary to plan specific activities to increase their time in the evening before going to bed.

7.3.5 Relaxation Training

As described earlier, thoughts and behavior resulting in arousal may interfere with sleep. For the same reasons, relaxation training, aiming to reduce arousal, may facilitate sleep. Various relaxation techniques have been developed to assist in the reduction of tension and anxiety. Many of them have been utilized in the treatment of insomnia, such as progressive muscle relaxation that reduces muscle tension by sequential tensing and relaxing of the main muscle groups [7, 47], autogenic training that produces somatic relaxation by inducing sensations of warmth and heaviness of the body [47], and guided imagery that aims to channel mental processes into a vivid story line [65]. Biofeedback has also been utilized to assist the mastering of relaxation techniques and has generated positive results [23, 25]. Relaxation training usually starts with a demonstration of how the procedure is carried out. The patients are then instructed to practice the technique once or twice a day at home in between sessions. The instructions of the relaxation procedures can be recorded during the session for the patients to practice at home. Commercial relaxation training CDs are also available to facilitate practice at home. It is suggested that a patient's level of relaxation should be assessed before and after each practice session in order to monitor the progress of the training. It may take weeks for some individuals to be able to relax on cue. Only after mastering the procedure is the patient told to use it to facilitate falling asleep at the beginning of sleep or following a nocturnal awakening. It is crucial to help motivate the patients to continue practicing the technique and to help them deal with obstacles they encounter.

7.3.6 Light Therapy

Light therapy is often included as part of a comprehensive CBT-I program especially for those patients whose contributing factors include the misalignment of endogenous circadian rhythms with environmental time. Exposure to bright light has been shown to shift the phase of circadian rhythms. The magnitude and direction of the

phase shift depend on the intensity and timing of the exposure [10, 35]. In general, morning light exposure in individuals following a nocturnal sleep schedule will tend to advance the circadian sleep phase. Evening light exposure, by contrast, tends to delay the circadian sleep phase.

7.4 Information Technology and CBT-I

Although the treatment efficacy of CBT-I has been well established by empirical studies, the application of CBT-I in the clinical field is limited by an insufficient number of well-trained professionals to administer this treatment. In addition, the arrangement to see a therapist once or twice a week for several consecutive weeks is not feasible for individuals with busy schedules. Self-help approaches or telephone consultations to reduce costs and improve accessibility have been evaluated in research. One study demonstrated that group therapy and telephone consultations were as effective as individual therapy [2]. Many forms of self-help approaches, such as bibliotherapy with or without minimal professional guidance, have also shown to be effective [1, 34, 36]. The effect of self-help treatments was found to be nearly equal to individual and group treatments, and was improved by adding telephone consultations [30, 39].

With the rapid development of information technologies and the accessibility of the Internet, several studies have evaluated self-help treatment for various conditions through the Internet (e.g. [9, 52, 61]). In one study of insomnia, 109 insomnia patients were randomly assigned to an Internet-based self-help CBT-I treatment or a waiting-list control condition. The CBT-I treatment includes sleep restriction, stimulus control, and cognitive restructuring. Sleep log data were completed for 2 weeks before and after the 5 weeks treatment. It showed that total sleep time, total wake time in bed, and SE were significantly improved in the CBT-I self-help treatment [59]. Another study evaluated the effect of a self-help sleep-improving program that adopted part of the concept and technique of CBT-I. Forty-three subjects recruited through a website were randomly assigned to two groups—an intervention group and a waiting list group. The intervention group participated in a 2 weeks Internet-based program that provided advice for sleep-related daily practices and monitored those behaviors along with sleep quality. The results showed that the Internet-based self-help programs improved subjective sleep quality and sleep-onset latency [60]. A recent study further confirmed the efficacy of intervention through the Internet for patients with insomnia. Forty-five insomnia patients were randomly assigned to an Internet intervention or a wait-list control group. The Internet intervention program incorporated the primary components of CBI-I, including sleep restriction, stimulus control, sleep hygiene, cognitive restructuring, and relapse prevention. The results showed that scores on the Insomnia Severity Index, wake after sleep onset, and SE were significantly improved in the Internet group but not in the control group [53].

7.5 Summary

Insomnia is a condition with a high prevalence. Although nonpharmacological treatment is available, most insomnia sufferers may not have access to the treatment. This limitation may be resolved by the rapid development of information technology. Although preliminary studies have shown promising results with Internet-based CBT-I programs, those studies lacked good controls. Future studies with better controls and more rigorous protocols are needed to clarify those questions and substantiate its effectiveness. Further, these programs primarily used the Internet to provide information and contact patients. Future research should utilize advanced technology not only to deliver the CBT-I material but also to evaluate the patients and facilitate the practice of CBT-I techniques at home.

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