Non-Pharmacological Treatment of Insomnia

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Abstract Insomnia is one of the most common sleep disorders, which is characterized by nocturnal symptoms of difficulties initiating and/or maintaining sleep, and by daytime symptoms that impair occupational, social, or other areas of functioning. Insomnia disorder can exist alone or in conjunction with comorbid medical and/or psychiatric conditions. The incidence of insomnia is higher in women and can increase during certain junctures of a woman's life (e.g., pregnancy, postpartum, and menopause). This article will focus on an overview of cognitive behavioral therapy for insomnia, evidence of effectiveness for this treatment when insomnia disorder is experienced alone or in parallel with a comorbidity, and a review with promising data on the use of cognitive behavioral therapy for insomnia when present during postpartum and menopause.

Key Words Insomnia · cognitive behavioral therapy · non-pharmacological treatment · women · postpartum · menopause

Introduction

Insomnia is one of the most common sleep disorders characterized by nocturnal symptoms of difficulties initiating and/or maintaining sleep, and by daytime symptoms that

impair occupational, social, or other areas of functioning. Insomnia disorder can exist alone or in conjunction with comorbid medical and/or psychiatric conditions. The incidence of insomnia is higher in women and can increase during certain junctures of a woman's life (e.g., pregnancy, postpartum, and menopause). The etiology of insomnia remains unknown, but several models have been put forth in the biological, behavioral, and psychological domains to explain the development and maintenance. Treatments that have received empirical support for insomnia disorder are hypnotic medications and cognitive behavioral therapy for insomnia (CBTI). This article will focus on an overview of CBTI treatment components, the empirical evidence for CBTI when insomnia disorder is experienced alone or in parallel with a comorbidity, and a review of promising data on the use of CBTI when insomnia is present during postpartum and menopause.

Insomnia Overview

Definition

Two classification systems exist that define insomnia disorder. The first classification system is The *International Classification of Sleep Disorders, 2nd edition* published by the American Academy of Sleep Medicine is used by sleep specialists and includes general criteria for insomnia disorder. The general criteria for insomnia disorder includes difficulty initiating or maintaining sleep, or nonrestorative sleep accompanied by daytime impairment such as fatigue or difficulties with memory or concentration that are related to the sleep difficulty. The *International Classification of Sleep Disorders, 2nd edition* [1] further specifies 10 subtypes if the patient meets the general insomnia disorder criteria: adjustment insomnia, psychophysiologic insomnia,

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paradoxical insomnia, idiopathic insomnia, inadequate sleep hygiene, insomnia due to a mental disorder, insomnia due to a drug or other substance, insomnia due to a medical condition, insomnia unspecified, and physiological insomnia.

The second classification system is the *Diagnostic and Statistical Manual for Mental Disorders, 4th edition* (DSM)-IV, which is published by the American Psychiatric Association [2] and used by mental health professionals in diagnostic assessments. This system classifies insomnia into 4 categories: 1) primary insomnia disorder, 2) insomnia related to another mental disorder, 3) insomnia due to a general medical condition, and 4) substance-induced insomnia. Diagnosis of primary insomnia disorder based on the DSM-IV criteria include difficulty initiating or maintaining sleep, or non-restorative sleep for at least 1 month, and sleep disturbance that leads to distress or impairment in daytime functioning in either social, occupational, or other areas of functioning.

The DSM, 5th edition (DSM-V) is scheduled to be published in 2012 and the DSM-V sleep advisory committee on sleep nosology proposes changing primary insomnia disorder to a single diagnosis of insomnia disorder with use of specifiers for clinical comorbidities. The proposed discontinuation of insomnia related to a mental condition or due to a medical condition moves away from causal attribution [3]. Additional proposed changes to the insomnia diagnosis using DSM-V include the presence of sleep disturbance for at least 3 nights a week, for a duration of at least 3 months [3] (Table 1).

Insomnia is a term that has been used interchangeably in the literature indicating both insomnia symptoms and insomnia disorder. To decrease ambiguity, the term insomnia

in this article will be used to indicate disorder distinct from insomnia symptoms. This is consistent with sleep researchers proposed use of an operationalized definition of insomnia disorder making it distinct from symptoms [4].

Prevalence Estimates

Epidemiologic studies indicate prevalence of insomnia disorder in the United States to be between 6 and 10 % [5, 6], and based on population surveys there were approximately 30 % of Americans who reported experiencing at least 1 insomnia symptom [7, 8]. Prevalence estimates of insomnia disorder vary based on definition and methodology [9]. Ohayon and Reynolds [10] examined insomnia disorder prevalence rates in a cross sectional study of 25,579 participants in Spain, France, United Kingdom, Germany, Italy, Portugal, and Finland, and found 6.6 % met DSM-IV primary insomnia disorder criteria, 9.8 % met criterion level (sleep symptom and daytime impairment), and 34.5 % reported at least 1 insomnia symptom.

Economic Impact

There is a significant economic burden and societal cost associated with insomnia due to the impact on healthcare utilization, impact in the work domain, and quality of life. Early estimates of direct costs (prescribed and over-the-counter supplements and healthcare services associated with insomnia) were in the range of 13.9 billion dollars in 1995.

Table 1 Summary of insomnia diagnoses across classification systems

	DSM-IV	ICSD-2	DSM-V (Proposed)
Diagnosis for primary insomnia	Primary insomnia (307.42)	Psychophysiological insomnia (307.42) Paradoxical insomnia (307.42) idiopathic insomnia (307.42)	Insomnia disorder
Comorbid insomnia disorder	Sleep disorder due to general medical condition, insomnia type (327.01) Insomnia related to another mental disorder (327.02)	Insomnia due to medical condition (327.01) Insomnia due to mental disorder (327.02)	No separate category; includes specifier to clarify comorbid physical or mental illness
Duration	Difficulty initiating or maintaining sleep, or nonrestorative sleep for at least 1 month	Not specified	Sleep difficulty occurs at least 3 nights per week, for at least 3 months
Other subtypes of insomnia	Substance-induced insomnia	Adjustment insomnia, inadequate sleep hygiene, insomnia due to drug or substance, insomnia unspecified and physiological insomnia	Not yet specified

The ICSD-2 classifies insomnia disorders as subtypes based on presumed etiology

ICSD-2 = *International Classification of Sleep Disorders, 2nd edition*; DSM-IV = *Diagnostic and Statistical Manual for Mental Disorders, 4th edition*

In 1994, annual estimates of direct costs (utilization of hospitalization, inpatient services, and outpatient healthcare services, and pharmaceuticals, including over-the-counter supplements) and indirect costs (workplace absenteeism, lost productivity, traffic and workplace accidents) totaled more than 30 billion dollars [11]. Kessler et al. [12] estimated insomnia-related economic burden in the workplace setting based on impacted productivity to be approximately 63 billion dollars, whereas more recent estimates of direct and indirect costs are upwards of 100 billion dollars annually in the United States [13]. Insomnia is a highly prevalent disorder, yet not adequately addressed in clinical settings. Only 1 in 5 patients report making an appointment to discuss sleep problems with their physician [14]. Reasons why patients are not discussing issues of sleep with their physician has not been empirically examined, but may include reluctance to take hypnotic medications, fear of not being taken seriously, and being unaware of an effective non-pharmacological treatment for insomnia.

Cognitive Behavioral Therapy for Insomnia: Treatment Components Overview

It is important to note that general psychotherapy is not an effective intervention for insomnia [15]. For effective delivery of CBTI, having specialized training in sleep medicine is an important factor among other essential prerequisites. Historically, psychologists who have been trained, in cognitive behavioral interventions and have received education in the science of behavioral change, psychological assessment, and sleep medicine, have been the forerunners in developing and providing CBTI. The use of CBTI aims to reduce maintaining factors that perpetuate insomnia. CBTI is a short-term treatment that includes 4 to 8 sessions on average and adopts a multi-component approach, which typically includes sleep restriction, stimulus control, cognitive therapy, sleep hygiene, and relaxation training [16].

During the first session, the clinician conducts a comprehensive clinical interview to gather information on current sleep patterns and impact of the sleep disturbance, history, and developmental course of the sleep problem, comorbid and complicating factors, and specific etiological features that may have precipitated the insomnia episode. In addition, the clinician also provides the patient with sleep education and specific instructions on how to complete sleep diaries. Using a sleep diary is considered to be a standard of practice for monitoring progress during treatment, and is frequently used in CBTI to derive sleep parameters, such as sleep onset latency, wake after sleep onset, number of awakenings during the night, total time spent in bed, total amount of sleep, wake time, and sleep efficiency. Parameters, such as number and duration of naps, use and timing of sleep medication, ratings

of sleep quality, daytime sleepiness, and daytime fatigue are also frequently included in sleep diaries.

Subsequent sessions are designed to deploy multiple modalities along the course of several sessions. Although there is no particular order that has been specified in which these modalities are delivered, stimulus control has received the most empirical support and is identified as an “effective and recommended therapy in the treatment of insomnia” [17, 18]. The American Academy of Sleep Medicine currently recommends that stimulus therapy, relaxation training, and cognitive behavioral therapy are individually effective therapies in the treatment of insomnia as a standard [18]. Stimulus control and sleep restriction have been considered first-line interventions and sleep hygiene, cognitive therapy, and relaxation training have been considered adjunctive interventions [16].

Sleep Restriction

Sleep restriction involves limiting the amount of time spent in bed to the amount of actual total sleep time, which is typically derived from 1 to 2 weeks of sleep diary data [19]. It is usually indicated in patients whose sleep efficiency (total sleep time/time in bed $\times$ 100) is less than 85 % [20]. Sleep restriction systematically reduces time in bed to a degree that is less than to what the patient is accustomed, and uses the homeostatic drive of sleep to increase sleep consolidation. Regardless of total reported sleep time, total time in bed is not typically recommended to be less than 5 or 5.5 h, and under certain circumstances that are not discussed here, the time in bed may need to be modified to address safety concerns. The patient’s sleep efficiency is monitored in subsequent follow-up sessions and modified throughout treatment based on sleep diary information and patient report. Sleep restriction therapy is effective because it strengthens the sleep and wake system that is controlled by the endogenous circadian pacemaker, by reducing variability in sleep schedules [20]. Finally, initial time in bed decreases in the earlier stages of sleep restriction and increases the homeostatic drive for sleep, which subsequently leads to faster sleep onset latency, decreased wakefulness after sleep onset, and higher sleep efficiency. Modifications of the protocol can include gradual sleep compression instead of abruptly curtailing prescribed time in bed, or planning for a mandatory or optional daytime nap [21–24].

Stimulus Control

The main objective of stimulus control is to have the patient limit the amount of time spent awake in bed and re-associate the bed and bedroom with sleep to regulate sleep–wake

schedules [25, 26]. The guidelines that are discussed with the patient include the following: 1) only going to bed when sleepy; 2) using the bed and bedroom only for sleep and sexual activity; 3) leaving the bed and bedroom if unable to fall asleep for longer than 15 to 20 minutes, and return only when sleepy; and 4) keeping a fixed wake time in the morning every day, which will help the patient acquire a consistent sleep and wake rhythm. These instructions are designed to help re-establish the bed and bedroom as strong cues for sleep.

Cognitive Therapy

Cognitive therapy is designed to challenge maladaptive beliefs and attitudes that serve to maintain insomnia. Worrying, faulty attributions, or unrealistic expectations of sleep may lead to increased emotional distress, and thus lead to additional sleep disturbance, causing a vicious cycle. Challenging dysfunctional thoughts associated with sleep is believed to decrease the anxiety and arousal associated with insomnia [26]. The first step is to make the patient aware of his/her dysfunctional thoughts of sleep, which is usually done through self-monitoring or questionnaires [27]. Once these sleep cognitions are identified, the main task is to help the patient challenge dysfunctional thoughts through guided discovery. Instead of regarding cognitions as an absolute truth, the patient is encouraged to view his/her thoughts as one of the many possible interpretations. The next step is to replace dysfunctional cognitions with more adaptive, realistic, and alternative interpretations based on past evidence.

Sleep Hygiene

Although there is insufficient evidence for sleep hygiene to be an option for single therapy, it is usually provided in conjunction with other modalities [18, 28]. It is likely that patients with insomnia presenting to a sleep clinic will have some knowledge of sleep hygiene, so that providing sleep hygiene instructions may contribute to optimizing clinical outcomes and relapse prevention [28]. Sleep hygiene consists of recommending a variety of behaviors and tending to environmental factors (e.g., light, bedroom temperature) that are conducive to sleep. Examples of sleep hygiene instructions include avoiding heavy meals close to bedtime, limiting caffeine products throughout the day, avoiding alcohol to aid sleeping, avoiding smoking close to bedtime, avoiding naps during the day time, and avoiding vigorous exercise close to bed time. Two approaches are recommended when delivering sleep hygiene instructions. One involves reviewing all sleep hygiene instructions with the patient using a didactic or Socratic approach [28]. Another

approach that is used more frequently involves assessing current sleep hygiene practices that the patient is already implementing, and tailoring intervention only to relevant behaviors that present as problems for the patient.

Relaxation Training

Learning relaxation techniques can be effective in reducing physiological hyperarousal in the patient. Research suggests that relaxation is especially effective in helping with sleep initiation [29]. Relaxation practice involves practicing relaxation techniques during the day, prior to bedtime, and also in the middle of the night, if the patient is unable to fall back asleep. There is little evidence to suggest differential effectiveness for various relaxation techniques. Common relaxation techniques include progressive muscle relaxation, which involves alternately tensing and relaxing different muscle groups in the body; deep breathing techniques, which involve diaphragmatic breathing; body scanning, which involves focusing on a body-part sequence that covers the whole body; and autogenic training, which involves visualizing a peaceful scene and repeating autogenic phrases to deepen the relaxation response [30]. Use of mindfulness-based therapy for insomnia, which is based on mindfulness mediation, guided imagery, and biofeedback, can also be incorporated into the treatment.

Cognitive Behavioral Therapy for Insomnia: Evidence of Effectiveness

The National Institutes of Health consensus and the American Academy of Sleep Medicine Practice parameters recommend that CBTI be considered standard treatment for insomnia based on strong empirical support of effectiveness. The American Academy of Sleep Medicine publishes practice parameter guidelines based on systematic reviews by a commissioned task force. The first systematic review of 48 clinical trials and 2 meta-analyses in 1999 revealed 70 to 80 % of those treated with CBTI experienced treatment benefit. For the average participant, the benefit was reduction in main insomnia symptom (i.e., time to get to sleep or time awake in the middle of the night) and a total sleep time increase of approximately 30 minutes, along with significant improvement in the patient's perceived sleep quality and satisfaction with sleep [31]. In 2006, the task force completed another systematic review of 37 treatment studies subsequent to the previous review (1998-2004) [32]. In addition to revealing sleep improvements sustained in time in primary insomnia and in the presence of medical/psychiatric comorbidities, 9 treatment studies in older adults also indicated facilitation of discontinuation of hypnotic medication after chronic use [32].

Randomized control trials comparing CBTI with hypnotic medications reveal comparable efficacy immediately after treatment, but longer lasting effects at follow-up were compared to medication [33–35]. Morin et al. [33] conducted a randomized placebo-controlled clinical trial examining insomnia in late life adults (mean age, 65 years old) comparing CBTI, Temazepam, combined (CBTI and Temazepam), and placebo groups. They found that at post-treatment, the 3 active treatment groups were statistically improved ($p < 0.01$ for all 3 groups) in time awake after sleep onset (middle of the night insomnia), but at 3, 12, and 24 months follow-up post-treatment, the CBTI group sustained the sleep improvements, indicating maintenance of treatment gains in comparison to the combined treatment group (total wake time and wakefulness after sleep onset were significantly changed indicative of worsening in time), and the medication group (significant worsening in time awake after sleep onset was noted at 24 months follow-up). Another randomized controlled trial compared CBTI, combined CBTI and medication (Zolpidem), medication only (Zolpidem), and a placebo [34]. Sleep latency percentage change based on sleep diary revealed pre- to post-treatment effect sizes for the CBTI group (Cohen's $d = 1.17$), combined CBTI and medication group ($d = 1.08$), and medication group ($d = 0.51$). At follow-up, increased sleep latency was seen in the medication group and the combined CBTI and medication group, as compared with the CBTI only group. The supposition is that CBTI teaches skills that can be used in the future if needed, whereas medication has to be administered to be effective. Jacobs et al. [34] suggests that the reason why the combined CBTI and medication group did not maintain long-term benefits may have been due to the co-administration of sleep medication with CBTI at the time of treatment, leading to less investment in the CBTI treatment, which may have caused the participants in this group to be more susceptible to relapse after treatment. CBTI has also been found to be effective in younger and older individuals. A meta-analysis of 23 studies by Irwin et al. [36] examined efficacy of CBTI components and the effect of age on sleep treatment outcome. The studies were divided into 2 age groups: mean age less than 55 years old and another group for 55 years and older. Consistent with previous findings [29, 37, 38], significant effect sizes were found for the behavioral treatment for sleep latency (-0.52 adult group vs -0.051 older adult group), improvement in wakefulness after sleep onset (-0.57 adult group vs -0.73 older adult group), and sleep quality (0.89 adult group vs 0.60 older adult group). The 23 studies were also divided into 3 categories based on treatment type: 1) CBT, 2) relaxation, or 3) behavioral therapy (BT) only. The treatment components revealed robust effect sizes: sleep latency (-0.38 for CBT vs -0.60 for relaxation, 0.59 for BT only), wakefulness after sleep onset (-0.75 for CBT, -0.35 for relaxation, and -0.82 for BT), and sleep quality (0.53 for

relaxation, 0.91 for BT only). The meta-analysis reveals moderate-to-large effect sizes for treatment modalities, and for both adult and older age groups in subjective sleep outcomes of latency to sleep, wakefulness after sleep onset and sleep quality.

Psychiatric and Medical Comorbidities

Prevalence estimates of insomnia experienced in the presence of a comorbid psychiatric condition is approximately 28 % [39]. Longitudinal studies suggest that insomnia increases the risk of developing a psychiatric condition [6, 40, 41]; these studies also suggest that the treatment of the psychiatric condition does not necessarily resolve disturbed sleep [42, 43]. Ohayon [44] examined patients with chronic pain conditions and found 3 times more frequency of insomnia symptoms and more reported daytime impairments due to sleep issues in those with chronic pain in comparison to those with just reported insomnia symptoms. Insomnia rates range from 16 to 33 % for those with comorbid medical conditions, such as diabetes mellitus, heart disease, and chronic back pain [14, 45], and the rates range as high as 88 % for those with insomnia symptoms and chronic pain [46].

The assumption that insomnia is caused by and resolved once the parent disorder is treated is untenable, considering insomnia is highly refractory after treatment of the comorbid condition [45]. This is suggestive of insomnia having a reciprocal feedback on the comorbid condition that speaks to a more complex relationship than cause (medical/psychiatric condition) and effect (insomnia), but rather a possible shared pathway of development, although the etiology of insomnia remains under investigation.

Chronic illness or presence of a psychiatric condition may directly impact adherence to CBTI treatment. For example, chronic pain patients may spend excessive time in bed due to physical pain or fatigue, and depressed patients may use the bed as an escape from emotional pain, and both groups may have increased napping during the day. In addition, use of substances that may interfere with sleep (such as caffeine or alcohol) [47] and medications used to treat a coexisting disorder may impact sleep (such as stimulating medication used in the treatment of the human immunodeficiency virus or medication that is sedating, which make it challenging to wake up at a particular time). Also there are safety concerns with sedating medications for patients when getting out of bed during the night if unable to sleep, which may require modification of stimulus control guidelines. The previously mentioned issues highlight the importance of CBTI as not being a “one size fits all” treatment and incorporating modifications for specific individuals. Nevertheless, research demonstrates that in those with insomnia disorder and presence of a comorbid medical or psychiatric condition, CBTI is effective.

A case series study that was applied in a clinical setting capturing participants with primary insomnia, and in the presence of medical (most commonly headaches, musculoskeletal, and gastrointestinal disorders) and psychiatric conditions (most commonly mood and anxiety disorders), have found large effect sizes for reduction in sleep latency (0.85), decrease in frequency of awakenings after sleep onset (0.54), decrease in time awake after sleep onset (1.14), and an increase in total sleep time of approximately 50 minutes more a night (0.55). These findings are consistent with a previous study that revealed those with medical or psychiatric comorbidities responding to CBTI by Lichstein et al. [48], which produced significant post-treatment gains as follows: reduction in sleep latency by 35 %, reduction in frequency of awakenings by 14 %, a 30 % reduction in the amount of time awake after sleep onset, and a 14 % increase in total sleep duration. Dashevsky and Kramer [49] examined CBTI (6 sessions for a span of 2 months) in those with a psychiatric disorder of which all participants had not responded to pharmacological treatment for insomnia, and also found significant changes in sleep patterns after treatment.

The greatest attention in outcome research thus far involving psychiatric conditions has been on depression. A study by Morawetz [50] found that more than 80 % of those with and without depression significantly improved post-treatment, and interestingly those with depression also experienced improvement in their depressive symptoms as measured by the Beck Depression Inventory, despite no specific depression treatment. Of those who were depressed and had significant improvement in sleep post-treatment, 57 % were no longer depressed and 13 % of participants had a 40 % reduction in depressive symptoms post-treatment compared to those who were depressed and did not have a significant improvement in sleep, none of these participants had elimination or significant reduction in depression at post-treatment [50]. This study highlights the complex relationship between depression and insomnia, and challenges the previously held assumption that treating depression will resolve insomnia. In addition, it also demonstrates that insomnia can be a risk factor for the development of depression [51].

Cognitive Behavioral Therapy for Insomnia: Postpartum and Menopause

Research has shown that women experience greater subjective complaints of insufficient or nonrestorative sleep, as well as increased need for sleep compared to men. Although insomnia disorder is widespread in the general population, it tends to occur more frequently in women, particularly during times of hormonal fluctuation (with the onset of menses, during pregnancy, postpartum period, and with peri- and post-menopause). Female reproductive hormones,

specifically estrogen and progesterone, not only regulate reproductive tissue function during the menstrual cycle, but also, through their secondary actions in the central nervous system, influence sleep and circadian rhythms.

Pregnancy

Pregnancy causes significant changes in the neuroendocrine system, including gonadal steroids (estrogen and progesterone), pituitary hormones (gonadotrophins, prolactin, and growth hormone), melatonin, and cortisol [52, 53]. Hormonal changes not only affect the sleep-wake cycle and sleep structure, but also cause physiological changes that lead to sleep disturbance. In addition to the hormonal changes, pregnancy causes a multitude of anatomic and physiological changes that are essential to maintain the pregnancy, but can also contribute to sleep problems during this time. Common causes of sleep disturbance during pregnancy include anxiety, urinary frequency, backache, fetal movement, general abdominal discomfort, breast tenderness, leg cramps, heartburn, and reflux.

Complaints of sleep disturbance are common during pregnancy, generally commencing with the onset of pregnancy, and increasing in frequency and duration as the pregnancy progresses due to pregnancy-related anatomic, physiologic, and hormonal changes [54–56]. Poor and insufficient sleep during pregnancy are also associated with increased circulating levels of inflammatory markers involved in poor health [57–61] and adverse pregnancy outcomes, including intrauterine growth restriction and preterm delivery [62–68]. During the third trimester of pregnancy, insufficient sleep and generally poor sleep may place women at increased risk for prolonged labor and Cesarean deliveries (OR, 4.5 for insufficient sleep and 5.2 for disturbed sleep) [69], and for having an infant small for gestational age (OR, 1.75) [70].

For most women, the disruptions to sleep continuity are caused by factors related to pregnancy, such as frequent need for urination during pregnancy [56]. Some women also have difficulties initiating sleep, which are unrelated to perinatal factors, and/or experience difficulties returning to sleep after a trip to the bathroom. When the nocturnal sleep disturbances are substantial and associated with clinically significant distress or impairment of performance and other aspects of functioning, an insomnia disorder diagnosis is warranted. The prevalence of a probable diagnosis of perinatal insomnia range from 41 to 58 % of perinatal women [54, 71, 72]. Daytime coping strategies, such as napping, spending more time in bed, or increasing caffeine intake can perpetuate sleep difficulties. The presence of insomnia has a significant impact on quality of life and daytime functioning, and its management is imperative. Many women, however, do not seek help because they wish to avoid taking

medications. Concerns regarding use of sleep medication during pregnancy and lactation make non-pharmacological treatment options for insomnia particularly attractive. However, to our knowledge, no randomized controlled trials have tested CBTI during pregnancy.

Postpartum

Sleep disturbance during the postpartum period and its effects on maternal role functioning and mother–infant interactions are not well understood. Both self-report and actigraphy studies have demonstrated that nearly 30 % of mothers have disturbed sleep after the birth of their baby. The abrupt drop in hormone levels after the birth of the placenta can affect a new mother's sleep as much as the 24-h care she provides for her newborn. Longitudinal studies have documented that the first 6 months postpartum are associated with a substantial increase in time awake after sleep onset and a decrease in sleep efficiency compared to the last trimester of pregnancy [54, 73–75]. Fatigue and lack of energy remain high from pregnancy into the postpartum period through the first year after delivery. Sleep begins to normalize at 3 to 6 months postpartum, at approximately the time when infants begin distinguishing between day and night, and sleep for longer intervals of time at night. Confounding factors, such as the mother's age, type of delivery, type of infant feeding, infant temperament, return-to-work issues, prior birth experience, number of other children at home, and availability of nighttime support from the partner or other family member can have an impact on quality and quantity of sleep in new mothers, especially until the infant begins to sleep through the night. Women seem to compensate for their sleep disruptions by spending more time napping and sleeping later in the morning during the first postpartum month [76–80].

Several prospective studies document the relationship between sleep disturbance during pregnancy and subsequent reports of depressive symptoms at a later time among perinatal women (later in pregnancy [81] or in the early postpartum period [79, 82, 83]). The association between poor sleep and subsequent depressive symptoms also holds true when sleep disturbance is experienced during the early postpartum period and postpartum depression develops at a later postpartum time [84, 85].

Interventions to improve maternal sleep and fatigue in the postpartum are limited, perhaps because of the universal nature of the experience and the belief that disturbed sleep is an unavoidable part of motherhood. Two recent pilot studies provide evidence for the efficacy of CBTI during the postpartum period and both demonstrate that the benefits of CBTI extend beyond improvement in sleep to other domains. One study provided 5 CBTI sessions, between the second and

seventh postpartum weeks, to women who stopped smoking during pregnancy and found a significant decrease in the time awake in the middle of the night and a significant increase in nocturnal (as well as per 24-h) sleep time. Importantly, compared to women who did not receive the sleep intervention, those who did undergo CBTI had lower average daily cigarettes smoked and a higher percentage of cigarette-free days [86]. The second study provided CBTI to women with postpartum depression who also had disturbed sleep and reported pre- to post-treatment improvement in insomnia severity, sleep quality, and sleep efficiency, and mood and daytime fatigue [87]. These 2 studies provide further emerging evidence supporting the efficacy of CBT for maternal insomnia.

Menopause

Menopause is a natural process that occurs in the lives of women as part of normal aging. Menopause is defined as the cessation of menstruation due to degeneration of ovaries and follicles, and changing ovarian hormone levels (estrogen and progesterone). The World Health Organization [88] characterizes menopause as the permanent cessation of menstrual periods that occurs naturally or is induced by surgery, chemotherapy, or radiation. More recently menopause has been categorized in stages such as menopausal transition (defined by standardized criteria [89] as variable cycle length 7 days different from their normal cycle or >2 skipped cycles and an interval of amenorrhea of 2 to 12 months) or postmenopausal (defined as >12 months from the last menstrual period). Menopause occurs at a mean age of 51.4 years for Western women, but the range can be as wide as 40 to 58 years of age. The worldwide population of 470 million postmenopausal women is expected to increase, as 1.5 million women enter menopause each year, reaching a total of 1.2 billion by the year 2030 [90]. Most women now live long enough to become menopausal and can expect to live at least another 30 years beyond their final menstrual period.

Many women go through the menopausal transition with few or no symptoms, whereas a small percentage of women suffer from symptoms severe enough to interfere with their ability to function effectively at home, work, or school. Common complaints include hot flashes, night sweats, insomnia, mood changes, fatigue, and excessive daytime sleepiness. In the 2005 National Institutes of Health State-of-the-Science Conference panel report on menopause-related symptoms, sleep disturbance was identified as a core symptom of menopause [91]. The prevalence of insomnia, defined as disturbed sleep associated with distress or impairment, is estimated at 38 to 60 % in peri- and postmenopausal women [92–94]. Troubled sleep was reported by 54 to 58 % of women between 40 and 60 years of age in the Ohio Midlife Women's study [95]. The Wisconsin Sleep

Cohort study found that peri-menopausal women and post-menopausal women were twice as likely to be dissatisfied with their sleep as pre-menopausal women [96]. The Study of Women's Health Across the Nation (SWAN) has shown that difficulty sleeping is reported by 38 % of women between 40 and 55 years of age, with higher levels among late peri-menopausal (45.4 %) and surgical post-menopausal (47.6 %) women [92]. The most common menopausal sleep complaint is difficulty falling asleep, but significant increases in self-reported night-time awakenings and daytime drowsiness have also been described, even after controlling for confounding variables, such as age and depression [92, 95–101].

We were unable to find an estimate of prevalence on nocturnal hot flashes/night sweats; however, it is generally believed that hot flashes occur in 60 to 80 % of women during the menopausal transition [102] and persist for 4 to 5 years on average [103, 104]. When hot flashes occur during the night, they frequently awaken women from sleep, although not every nocturnal flash is associated with an awakening. Women with nocturnal flashes may also experience awakenings that are unrelated to a vasomotor event. Indeed, insomnia can occur during menopause independent of nocturnal flashes. Although self-reported nocturnal flashes correlate with subjective poor sleep quality, such association is less clear when objective sleep measures are used [96, 105, 106]. There is only limited and contradictory evidence supporting an association between nocturnal flashes and sleep disturbance when both variables were measured objectively [96, 105–111].

Sleep disruption associated with hot flashes may be treated in several ways. The effect of hormone therapy (HT) on sleep has been studied widely in post-menopausal women, although results are equivocal. Although several studies comparing HT to a placebo have found that HT improves perceived sleep quality and self-reported sleeping problems more than a placebo [112–123], some did not find an advantage of HT in comparison to a placebo [124, 125]. Among 11 studies that examined the effects of HT on objective measures of sleep, few found significant benefits [126]. In addition, although zolpidem and eszopiclone have been shown to be more effective than a placebo in the treatment of insomnia in the early stages of menopause [127–129], long-term use is associated with concerns of tolerance, abuse, dependence, and rebound insomnia after discontinuation. CBTI has also been shown to be efficacious for the treatment of chronic insomnia in older adults [33, 36]. It would appear that CBTI could be readily adapted to the sleep disturbances often reported by symptomatic peri- and post-menopausal women [130]. To preliminarily examine the efficacy of CBTI for menopausal sleep disturbance, our group [131] identified 44 women (age range, 42–68 years; $m=55\pm5.9$) self-described as peri- or post-menopausal from a pool of 455 patients who received group CBTI in a sleep medicine clinic. Twenty-nine (66 %) of those

women endorsed “my sleep at night is affected by my menopause” on a baseline sleep questionnaire, which constituted the “peri-menopausal” insomnia group. Sixty-three women (age range, 24–49 years; mean age= 35 ± 5.0) self-described as pre-menopausal constituted the control group. Repeated measures of analysis of variance revealed a significant reduction from pre- to post-CBTI in Insomnia Severity Index (from 20.2 ± 4.4 to 10.3 ± 4.2 ; $p<0.001$) and Beck Depression Inventory (from 11.6 ± 7.8 to 6.6 ± 5.4 ; $p<0.001$), with no significant main effect for group or interaction between time and group. This is promising data and requires further examination of CBTI in women during menopause when insomnia disorder is present.

Conclusion

CBTI is an effective non-pharmacological treatment for primary insomnia and insomnia comorbid with a medical and/or psychiatric condition. CBTI demonstrates comparable efficacy and long-term benefits in randomized controlled trials comparing CBTI with sleep medications. Strong empirical support has led the National Institutes of Health Consensus and the American Academy of Sleep Medicine parameters to recommend CBTI as a standard treatment for insomnia. Promising data is emerging in the area of application of CBTI in women during the post-partum and menopause stages when insomnia is present. Research is warranted in the area of CBTI during pregnancy when insomnia is experienced. Based on the economic and societal impact of insomnia, increased awareness is needed regarding the importance of treatment, and an effective non-pharmacological treatment is available to patients. The American Board of Sleep Medicine offers certification for psychologists in behavioral sleep medicine including CBTI treatment and a list of certified providers can be found at: <http://www.absm.org/bsmspecialists.aspx>.

Required Author Forms Disclosure forms provided by the authors are available with the online version of this article.

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