Sleep disturbance and menopause

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Purpose of review
Sleep problems are among the most prevalent and bothersome symptoms of menopause. This review characterizes menopausal sleep disturbances, describes biopsychosocial predictors, and summarizes the evidence supporting pharmacological and nonpharmacological treatment options.

Recent findings
Recent studies found that sleep changes are early indicators of perimenopause and sought to disentangle the respective impacts of menopausal status, hot flashes (HFs), and changes in reproductive hormones on peri-/postmenopausal sleep problems. Both HFs and reproductive hormones predicted sleep problems, but neither solely accounted for the myriad changes in sleep, thus highlighting the contribution of additional biopsychosocial risk factors. Inconsistencies across studies were likely due to differences in study design and methodology, participants' menopausal stage, and the presence of sleep complaints. Recent studies support the use of psychological (cognitive-behavioral therapy for insomnia) and pharmacological (e.g., neurokinin B antagonists) treatments in addition to hormone therapy.

Summary
Sleep problems are common and of critical import to women during the menopausal transition, significantly influencing treatment preferences and satisfaction. Thus, sleep problems should be routinely assessed from a biopsychosocial perspective and treated with evidence-based interventions throughout menopause. Treatment selection should be based on diagnosis and careful assessment.

Keywords
menopause, sleep, sleep disorders

INTRODUCTION

Menopause represents a period of significant biopsychosocial changes as women transition from reproductive to nonreproductive status. According to the Stages of Reproductive Aging Workshop (STRAW), perimenopause begins when there are persistent changes in menstrual cycle length ($\geq$7 days) between consecutive cycles and lasts until 12 months after the final menstrual period, after which a woman enters postmenopause [1]. Sleep problems are among the most prevalent and bothersome symptoms of menopause, and may even signal the onset of the menopausal transition [2,3,4▪,5▪▪]. Between 40% and 60% of peri- and postmenopausal women (PPMW) report sleep problems that are distressing and substantially impact quality of life, work productivity, healthcare utilization, and physical and mental health [6–10]. In weighing different treatment options for menopausal symptoms, women cite improvement in sleep as the most important treatment attribute [11▪▪]. This article provides an overview of the sleep changes during menopause, the biopsychosocial factors that contribute to these sleep changes, and evidence-based treatments.

SLEEP CHANGES DURING MENOPAUSE

During the menopausal transition, women are at increased risk for emergent or worsening sleep problems. Changes to sleep can be assessed subjectively (e.g., using self-report or sleep diaries) or objectively [e.g., using polysomnography (PSG) or actigraphy], which reveal different results [12].

Compared to premenopausal women, PPMW report poorer sleep quality, nonrestorative sleep, increased sleep fragmentation/awakenings, and greater insomnia symptoms (difficulty falling and staying sleep), and these differences remain after accounting for age [13–16,17▪]. The odds of sleep

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KEY POINTS

- Women cite improvement in sleep as their highest priority for menopausal symptom treatment, higher even than treatment of vasomotor symptoms.
- The people at highest risk for sleep disturbance during menopause are those with preexisting moderate-to-severe sleep problems prior to menopause.
- In-office assessment of sleep disturbance can be performed using the Insomnia Severity Index and STOP BANG questionnaires as part of a menopause symptom evaluation.
- Safe and effective pharmacologic and nonpharmacologic treatment options are available to treat sleep disturbance in the menopause transition, thereby improving quality of life as well as overall mental and physical health.

disturbance increase throughout the menopausal transition [16,18].

Whereas subjective assessments clearly support decreases in sleep duration, maintenance, and quality, evidence from objective measures is inconsistent. In a cohort study, PPMW demonstrated improved sleep quality compared to premenopausal women, indicated by more slow wave sleep (SWS) [19]. Another cohort study found no objective sleep changes but an increase in cortical arousal during sleep in late PPMW, which may be related to reports of less satisfactory sleep and underestimation of sleep duration [20,21]. Longitudinal studies also support objective sleep improvement along with increases in cortical arousal during menopause [22,23,24▪▪]. Studies have reported a significant deterioration in sleep continuity from age 46 to 52, followed by increased periods of SWS as women transition from pre to postmenopause [22,25]. Subjective-objective discrepancies may reflect the limitations of PSG (i.e., single-night studies do not capture within-person variability in sleep patterns, which is influenced by menopausal symptoms [26]), differences in study design, between-person sleep variability, or that PPMW consider additional "inputs" when judging their sleep quality (e.g., mood, fatigue). More research to understand these discrepancies is needed.

RISK AND TRAJECTORY OF SLEEP PROBLEMS IN MENOPAUSE

Longitudinal studies support different risk trajectories based on premenopausal sleep disturbances. Women with moderate-to-severe premenopausal sleep problems are at high risk of persistent/worsening sleep difficulties during perimenopause whereas those with mild premenopausal sleep difficulties are at lower risk of peri/postmenopausal sleep problems [14,27,28]. Nevertheless, about one third of PPMW with insomnia attribute its onset to menopause [15].

Surgical menopause resulting from hysterectomy with or without bilateral oophorectomy also increases the risk of sleep problems and changes to sleep architecture [29,30]. Relative to natural menopause, women who experienced surgical menopause were over twice as likely to experience insomnia symptoms and report poorer sleep quality [29,31][31;cf.29]. Greater risk may be due to more severe hot flashes (HFs), ovarian hormone loss, and possibly poorer psychological health postsurgery [27,30,32–34]. Those with presurgical sleep maintenance problems are at the greatest risk of worsening sleep after surgical menopause [35]. Women undergoing surgical menopause are also at greater risk of developing obstructive sleep apnea (OSA) [36,37]. Bilateral oophorectomy *after* natural menopause also increased OSA risk [36].

ETIOLOGY

Menopausal sleep problems are varied and have multiple interacting etiological factors. Understanding these factors is the bedrock of successful biopsychosocial assessment.

Biological contributions

HFs fragment sleep and increase wakefulness using subjective [7,15,18,38▪▪,39,40,41▪] and objective sleep assessments [42,43], particularly when they are more severe [40] and perceived as more bothersome/interfering [38▪▪,44]. Subjective HFs are more strongly related to awakenings than objective HFs because not all objective HFs induce an awakening [45]. Some have questioned whether HFs and awakenings truly occur simultaneously, finding that 94% of awakenings occurred 2 min before/after the HF and 6% occurred concurrently [46]. While recent studies suggest that nearly 80% of objective HFs are associated with awakenings (even without a subjective HF report), two-thirds of awakenings are not accompanied by a HF suggesting that they are not the sole contributor [47,48]. Greater HF improvements are associated with improvements in sleep and treatment satisfaction [38▪▪,49].

Increases in follicle stimulating hormone (FSH) and decreases in estrogen and progesterone are associated with poorer sleep quality and difficulties falling and staying asleep [18,40,50▪]. A steeper rate of change in FSH correlates with poorer subjective but greater objective sleep quality (increased sleep

duration and SWS) [51]. However, the relationship between FSH and objective sleep is weaker in PPMW with insomnia ($n = 16$) compared to PPMW without insomnia ($n = 17$) [52]. A longitudinal study found that steeper declines in estrogen are associated with more severe sleep problems [53]. A lower ratio of E2 to testosterone is associated with shorter nighttime awakenings, suggesting that a hormonal "levelling out" as menopause ends may improve sleep continuity [51]. Progesterone is hypothesized to have hypnotic effects via its metabolites pregnanolone and allopregnanolone, which act on the GABAergic system; thus, some posit that menopausal reductions in progesterone can disturb sleep via less GABAergic activation [54–56]. Reproductive hormones are involved in maintaining respiratory muscle tone and respiratory stimulation, potentially implicating hormonal changes in the development of OSA. Evidence from a population-based study supports a relationship between decreased reproductive hormones and increased risk of OSA and snoring [57▪].

Circadian rhythm alterations in menopause are less studied. Some studies support a shift toward morningness and less robust rhythms that may underlie sleep disturbances [58–61]. Hormonal changes alter the circadian rhythm during the menstrual cycle, suggesting that further assessment of circadian rhythm changes during menopause is warranted [62].

Chronic health conditions in midlife can also negatively impact sleep, including cancer, thyroid problems, pain conditions, obesity, and gastroesophageal reflux [32]. Among PPMW, poor perceived health is associated with difficulties falling and staying asleep [63,64]. Weight gain and shifts in the distribution of adipose tissue with aging increase risk of OSA [65,66]. Medication adverse effects can also contribute to sleep problems [64].

Psychosocial contributions

Midlife is characterized by role changes (e.g., caregiving for elderly parents, empty nesting, marital changes) and stressors that can impact sleep [67,68]. The menopausal transition itself may be perceived as a stressor [67]. Among PPMW, stress and psychological distress predict poor sleep quality [27,63,64].

Symptoms of depression and anxiety are also associated with poor sleep and insomnia among PPMW [29,52,63,64,69▪▪]. Depressive symptoms worsen during menopause and those with a history of depressive episodes are at risk of recurrence [70,71]. The relationship between sleep and depression in PPMW is bidirectional [72–76]. Sleep disturbance may mediate the relationship between HFs and mood symptoms [41▪,77].

Psychological and behavioral responses to sleep loss can increase the risk of chronic insomnia. Predisposing, precipitating, and perpetuating factors contribute to the development and maintenance of chronic insomnia [78]. Using this framework, HFs precipitate acute insomnia. For some, sleep problems resolve in the absence of HFs whereas others continue to suffer, indicating that additional perpetuating factors are responsible for maintaining insomnia. Responses to sleep loss can undermine the homeostatic and circadian systems responsible for regulating sleep. Examples include increased time and effort spent trying to sleep (e.g., earlier bedtimes, sleeping in), napping, and increased caffeine intake. Over time, the bed can become a conditioned signal for wakefulness. This explains why PPMW may experience a worsening of their sleep over time, such as progressing from brief, HF-induced awakenings to the development of difficulties with falling and staying asleep.

SLEEP DISORDERS IN MENOPAUSE

We have described sleep changes that can emerge/worsen during the menopausal transition. Next, we discuss sleep disorders and their assessment.

Insomnia disorder

Insomnia is difficulty initiating or maintaining sleep. Insomnia *disorder* is when symptoms occur at least three times per week, for at least 3 months, and cause distress or impairment [79]. Not all sleep disturbance is insomnia; for example, awakenings due to HFs *without difficulty returning to sleep* would not be considered insomnia. Frequent difficulty returning to sleep even after HF symptoms have subsided is suggestive of insomnia.

Many women experience greater difficulty initiating or maintaining sleep during the menopausal transition. These symptoms are persistent and affect daytime functioning in about 26% of women [15]. As detailed above, insomnia symptoms are not simply caused by menopause or HFs, and behavioral responses to sleep loss play a significant role in the transition of acute to chronic insomnia.

The Insomnia Severity Index (ISI) [80] is a validated measure of insomnia symptom severity with established cut-off scores to identify those experiencing clinically significant insomnia symptoms. Insomnia disorder is diagnosed based on a clinical interview of self-reported symptoms or use of a structured diagnostic interview [81]. Obstetrician-gynecologists (OB-GYNs) can utilize the ISI to assess patients' insomnia symptoms and consider referral

to a sleep specialist for additional assessment if indicated. Discussing with patients the severity, duration, and timing of onset of sleep disturbances (before/after menopause) can point to most effective treatment options.

Obstructive sleep apnea

OSA is the frequent obstruction of the upper airway during sleep. Repeated cessations of airflow result in micro-arousals that fragment sleep, with negative effects on sleep quality, cognition, and cardiometabolic health. OSA is more prevalent in men compared to premenopausal women; however, women's rates equal that of men's postmenopause [82]. In addition to aging, increased rates of OSA are due to changes in reproductive hormones during menopause and increased adipose tissue. Whereas men display more snoring and witnessed apneas, women with OSA often present with more nonspecific symptoms including insomnia, nonrestorative sleep, daytime sleepiness, and depressed mood [77,83–85]. Reports of daytime sleepiness or sleep complaints in PPMW should prompt consideration and assessment of snoring and OSA. In a sample of 6179 women, postmenopausal women had 48% higher odds of screening positive for obstructive sleep apnea, compared to pre/perimenopausal women [86]. The STOP-BANG questionnaire [87] is a screening tool for OSA that providers can use to determine if a referral to a sleep center is indicated for further evaluation using an overnight sleep study.

Restless legs syndrome and periodic limb movement disorder

Restless legs syndrome (RLS) involves an uncomfortable sensation in the legs, typically emerging in the evening, that is associated with a strong urge to move the legs. Periodic limb movement disorder (PLMD) involves repetitive leg and/or arm movements or cramping during sleep. The prevalence of both RLS and PLMD increases with age, and women are 37% more likely than men to report RLS symptoms [88]. There is a relationship between RLS and female reproductive hormones but it is difficult to disentangle hormonal from age-related contributions. Studies examining estradiol, FSH, and short-term estrogen therapy indicate that the increased prevalence of RLS and PLMD after menopause may be related more to aging than to menopause [89].

TREATMENT

Effective management of sleep problems during the menopausal transition relies on careful assessment of symptoms, their onset/duration, etiological contributions, and patient preferences to provide effective, sometimes multicomponent treatment. OSA, RLS, and PLMD are best managed by a sleep medicine specialist.

Hormone therapy

The evidence supporting the use of HT for insomnia in midlife women is mixed. Some studies show improvement while others show no effect on PSG measures and perceived sleep quality [90–93]. Women with co-occurring nocturnal HFs benefit the most from HT, indicating that HT may improve sleep as an indirect consequence of its beneficial effect on HFs [94]. A meta-analysis of 10 randomized placebo-controlled trials ($N = 388$) showed that, compared to placebo, micronized progesterone 200–300 mg daily (but not 100 mg) improved PSG-measured sleep parameters, including sleep duration and sleep onset latency [95]. Subjective sleep outcomes improved in most trials. The authors acknowledged that the concomitant administration of estradiol in some studies prohibited conclusions regarding the magnitude of the direct effect of progesterone. Evidence supporting the use of HT to treat OSA is inconclusive.

Neurokinin B antagonists

The thermoregulatory center of the hypothalamus is innervated by kisspeptin-neurokinin B-dynorphin (KNDy) neurons, which are stimulated by neurokinin B (NKB) via neurokinin 3 receptors (NK3Rs) and inhibited by estrogen. Fezolinetant is a nonhormonal, selective NK3R antagonist that blocks NKB binding on the KNDy neuron to reduce the frequency and severity of HFs [96]. Fezolinetant had a beneficial effect on four patient-reported assessments of sleep disturbance and impairment [97▪]. Elinzanetant is similar but targets both NK1 and NK3 receptors. In initial trials, elinzanetant led to improvements in sleep disturbance in women not selected for sleep problems. Studies are ongoing to assess its use in populations with sleep disturbance [98▪].

Cognitive-behavioral therapy for insomnia

Cognitive-behavioral therapy for insomnia (CBT-I) is a short-term treatment for insomnia disorder. With moderate to large effects on subjective and objective insomnia symptoms, and more durable effects than pharmacological interventions [99–101], CBT-I is recommended as the *first-line* treatment for chronic insomnia [102–105].

Randomized controlled trials have shown that CBT-I effectively improves insomnia in PPMW with HFs [106,107,108▪]. A pooled analysis from several trials revealed a significantly larger benefit of CBT-I on insomnia symptoms in women with HFs than HT, SSRI, SNRI, yoga, or exercise [109]. Evidence-based apps (e.g., Sleepio, Insomnia Coach) can facilitate access to CBT-I in the absence of a trained provider.

Hypnotics

Selective GABAergic agents improve sleep onset and maintenance in women with insomnia and HFs [110,111]. Eszopiclone also reduces the number of hot flashes reported at night, but not during the day [112]. Consistent with treatment guidelines, hypnotic medication may be considered for short-term use when distress is great, but is not recommended for long-term use [103,104,113].

Selective serotonin reuptake inhibitors, serotonin norepinephrine reuptake inhibitors, and gabapentin

Nonhormonal neuroactive pharmacotherapies are frequently prescribed for HFs and can produce corollary improvements in sleep. Serotonergic antidepressants have been shown to be more effective than placebo in reducing insomnia symptoms and improving sleep quality [114–116]. Similarly, gabapentin and pregabalin are effective at treating hot flashes and show some benefit for sleep complaints [117].

Alternative approaches

Cognitive-behavioral treatments for menopausal symptoms are associated with sleep improvements in PPMW with HFs [118]. There is modest evidence that exercise, mindfulness, hypnosis, and relaxation can improve sleep quality in menopausal women [108▪,109,118–122]. However, improvements in sleep quality are distinct from insomnia symptoms. Sleep hygiene is not a treatment for insomnia, and insomnia in midlife women is not associated with poor sleep hygiene [123]. Providing sleep hygiene counseling as a standalone treatment to PPMW struggling with sleep can increase frustration and negatively impact the therapeutic alliance.

CONCLUSION

Biopsychosocial changes that occur during menopause leave women vulnerable to worsening sleep and its disorders. Hormonal changes and HFs uniquely impact sleep during menopause, yet those most at risk for menopausal sleep disturbance have preexisting sleep problems. How these changes interact to moderate vulnerability to sleep disturbance merits additional investigation. Effective management requires a thorough assessment and selection of treatments that target maintaining factors and consider patient preferences. To develop safe and effective alternatives to HT, more research is needed on the mechanisms of HF-induced sleep disturbance (e.g., the hypothalamic estrogen-sensitive KNDY neuron pathways). Improving sleep during menopause improves patients' quality of life and treatment satisfaction.

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Conflicts of interest

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- ▪ of special interest
- ▪▪ of outstanding interest

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This article reports on the results of three studies that assessed symptom prevalence, attitudes and beliefs about the menopausal transition, and use of digital health technology for menopausal symptom management. In a total sample of 1182 women, sleep problems were among the most frequently reported symptoms of menopause; this was consistent across each individual survey. Although most people associate the menopausal transition with VMS, it is well established that sleep problems are frequent, distressing, and impairing for midlife women.

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In this longitudinal study, data were collected every 2-6 months over a period of 2 years to overcome the limitation of infrequent assessments in previous longitudinal studies, which may threaten validity. They included women with 4 consecutive FSH assessments; for those in the "transition" group, assessments consisted of two premenopausal assessments (T1, T2), the first assessment at which FSH was elevated (onset of perimenopause, T3), and the next assessment 6 months later (T4). Trouble sleeping *because of being too hot* increased significantly from T3 to T4 in the "transition" group despite no within- or between-group changes in VMS over time. The authors conclude that trouble sleeping because of being too hot is an early symptom of the menopausal transition.

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This high-quality study used a sophisticated discrete choice experiment (N = 467) to elucidate treatment preferences for vasomotor symptoms and determine contributions to willingness-to-pay for various treatment attributes. Substantial improvement in sleep problems was identified the most important attribute of treatments for VMS, outperforming improvement in VMS frequence and severity, and was associated with willingness to pay an additional US $46/month to obtain this treatment benefit. Taking sleep problems seriously is of critical import to peri-/postmenopausal women.

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This representative, population-based observational study using the Northern Finland Birth Cohort analyzed sleep disturbances in women with early-onset menopause. They selected women who were 46 years old and compared those who were late peri-/postmenopausal ($n = 359$) and premenopausal ($n = 2302$). Findings among women with early-onset menopause largely replicated those with later onset menopause. Using a validated measure of insomnia symptoms, the authors found that menopausal women reported more difficulties with sleep onset, maintenance, and early morning awakenings, and more dissatisfaction with sleep quality. Menopausal status increased the odds of each of these sleep complaints (ORs 1.31–1.79), and this was accounted for by the presence of HFs.

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Women ($N = 159$) participating in the Study of Women's Health Across the Nation (SWAN) ancillary sleep study were followed up after 3.5 years and divided into 3 transition groups: (1) remained pre/early perimenopausal, (2) transitioned to late perimenopause, or (3) transitioned to postmenopause. This longitudinal study was the first to compare polysomnographic sleep characteristics between those who remained pre/early perimenopausal and those whose menopausal status changed. They replicated a EEG power postmenopause in the absence of changes in sleep duration, continuity, or finding from a cross-sectional study of an increase in beta delta power. This was independent of demographic variables, BMI, presence of HFs during the PSG, and time between assessments, and held after controlling for beta EEG power at time 1. This indicates that postmenopause is associated with an increase in cortical arousal, which has implications for self-reported estimates of sleep quality and duration.

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This study uses pooled data from three clinical trials of menopausal women (Menopause Strategies Finding Lasting Answers to Symptoms and Health, or MsFLASH) to analyze the relationships between various vasomotor outcome measures recently proposed by COMMA, which were put forth to standardize the conceptualization and measurement of vasomotor symptoms across clinical trials. Given the replicated finding that VMS are associated with poorer sleep, this study does an excellent job at disentangling the various dimensions of VMS and their associations with sleep variables (insomnia and sleep quality) using standardized, reliable, and well validated measures. This helps advance research design (outcome selection) and adds to our understanding of how and why VMS negatively impact sleep. Women's relationships to their VMS (i.e., interference) were more strongly related to insomnia and sleep quality than the frequency or severity of their VMS. Further, the data showed that greater improvements in sleep were associated with greater treatment satisfaction, underscoring the importance of treating sleep disturbance during the menopausal transition.

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This study used Bayesian network analysis, which identifies the most likely relationship between variables while considering all factors included in the model. Using data from a longitudinal cohort study (Midlife Women's Health Study, $n = 742$ at year 1 and $n = 389$ at year 4), the found that HFs were the only predictor of insomnia (at the same timepoint). Reproductive hormone concentrations did not predict insomnia at any timepoint.

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