

## INVITED CONTRIBUTION

### Sleep in Women: A Review

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Sleep complaints are common in women, and women are more likely to suffer from insomnia than men. Multiple factors across a woman's lifespan, including hormonal changes, age-related physiological changes, psychosocial factors, the presence of sleep disorders, and physical and mental health conditions, can contribute to complaints of poor sleep in women. This article reviews the literature on the characteristics of, and contributing factors to, subjectively and objectively measured sleep during the menstrual cycle, pregnancy, and post-partum period, as well as the menopausal transition and postmenopause. Evidence from both subjective and objective measurements supports the presence of chronic sleep fragmentation associated with pregnancy, acute sleep deprivation during labour and the immediate post-partum periods, as well as disrupted sleep during the first few months after childbirth. While there is evidence for menstrual cycle and menopause related sleep disturbance based on women's self report, findings from objectively measured sleep have been mixed. Observational and intervention studies on the relationship between sleep and women's psychological well-being suggest that underlying causes of sleep disturbance across a woman's lifespan are often multi-factorial. Comprehensive assessments and targeted interventions are needed in managing sleep problems in women. Cognitive behavioural interventions have been shown to reduce sleep complaints during the perinatal and menopausal periods, and improvements in sleep are likely to lead to improvements in women's overall well-being.

**Key words:** menopause; menstrual cycle; mental health; pregnancy; sleep; women.

#### What is already known on this topic

- 1 Sleep disturbances are common in women across the lifespan.
- 2 Multiple physiological and psychosocial factors contribute to sleep problems in women.
- 3 There is a bidirectional relationship between sleep disturbance and psychological well-being.

#### What this paper adds

- 1 A comprehensive review of the characteristics of, and contributing factors to, subjectively and objectively measured sleep during the menstrual cycle, the perinatal period, and the menopausal transition.
- 2 Implications of sleep disturbance for women's psychological well-being.
- 3 An argument for comprehensive assessments and targeted interventions for managing sleep problems in women.

Sleep is closely associated with psychological well-being. Insufficient sleep and/or insomnia symptoms lead to adverse medical and psychological consequences and a poorer quality of life. Women have a higher risk of insomnia (ratio women : men 1.41:1), which emerges after menarche (Johnson, Roth, Schultz, & Breslau, 2006) and persists across a woman's lifespan (Zhang & Wing, 2006). The sex difference in insomnia remains even after taking into account underlying psychiatric disorders (Zhang & Wing, 2006). Women also report needing more sleep, spending more time in bed, and having more sleep difficulties than men across adulthood (Groeger, Zijlstra, & Dijk, 2004;

Middelkoop, Smilde-van den Doel, Neven, Kamphuisen, & Springer, 1996; Vitiello, Larsen, & Moe, 2004). These differences may be driven by several factors, including sex hormones acting on the brain, sex-linked genetic mechanisms, and sex differences in psychosocial factors (Mong et al., 2011). Since sleep regulatory systems are sensitive to the impact of reproductive hormones, sleep in women is also impacted by the changing hormone environment during the menstrual cycle, pregnancy, and post-partum, and in the approach to menopause. This review discusses what is known about sleep behaviour and physiology particular to women and how sleep impacts their psychological well-being.

Sleep is commonly assessed through subjective measurements such as self-report questionnaires and sleep diaries, and objective assessments such as polysomnography (PSG) and actigraphy. PSG is a comprehensive recording of the biophysiological changes that occur during sleep, including brain (electroencephalography [EEG]), muscle (electromyography),

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and heart (cardiography) activities. An actigraph is a watch-like device that estimates sleep duration and quality based on wrist movement. Although actigraphy does not provide information regarding sleep structure (architecture), it is capable of continuous measurement of sleep over multiple days or even weeks in participants' natural environment with minimum intrusion.

## Menstrual Cycle

### The Menstrual Cycle

A woman's menstrual cycle is exquisitely regulated by the hypothalamic-pituitary-ovarian axis to coordinate ovarian follicular development, ovulation, luteinisation, and luteolysis (Mihm, Gangooly, & Muttukrishna, 2011). Typically, the menstrual cycle is about 28 days (Mihm *et al.*, 2011), and is broadly divided into two phases: the follicular or proliferative phase, starting at the first day of menses and continuing until ovulation, and the luteal or secretory phase, from ovulation until the onset of menstruation. Each phase is characterised by a distinct hormone profile that has the potential to interact with sleep/wake cycles. During the follicular phase, follicle-stimulating hormone and estrogen levels rise and ovarian follicles develop and mature. Luteinising hormone peaks shortly before ovulation, when an oocyte is released from the follicle. In the luteal phase, progesterone dominates, being released from the corpus luteum, together with estradiol. Approximately 14 days after ovulation, if there is no implantation of a fertilised ovum, hormone levels rapidly drop and menses begin.

There are receptors for estrogen and progesterone in many sleep/wake-regulatory nuclei in the central nervous system (Mong *et al.*, 2011) and estradiol induces increased arousal in rodents and also consolidates sleep-wake rhythms, possibly via its actions on the suprachiasmatic nucleus (Schwartz & Mong, 2013). The fluctuations in reproductive hormones that characterises the menstrual cycle, therefore, have the potential to influence sleep.

### Sleep during the Menstrual Cycle

A seminal study that addressed many methodological limitations of earlier studies of changes in sleep across the menstrual cycle reported that in young healthy women, aspects of PSG measured sleep were remarkably stable across the menstrual cycle, with no change in slow wave sleep (SWS) or slow wave activity, sleep onset latency (SOL), total sleep time (TST), or sleep efficiency (SE) (Driver, Dijk, Werth, Biedermann, & Borbély, 1996). Rapid eye movement (REM) sleep was found to be more sensitive to the menstrual cycle, with a tendency to decrease in the luteal phase when body temperature was raised. A subsequent analysis showed that the trend for decreased REM sleep was due to a significant shortening of REM sleep episodes in the luteal phase, while non-REM episode duration was unchanged (Driver, Werth, Dijk, & Borbély, 2008). A reduction in REM sleep in the luteal phase has been confirmed by others (F. C. Baker, Kahan, Trinder, & Colrain, 2007; F. C. Baker *et al.*, 2012; Shechter, Varin, & Boivin, 2010). The amount of REM sleep correlates negatively with both estradiol and progesterone levels in the luteal phase (F. C. Baker *et al.*, 2012) and may be

mediated directly by the changing sex hormone profile or may be secondary to the increase in body temperature (Driver *et al.*, 1996).

The most striking effect of the menstrual cycle on physiological sleep is increased EEG activity within the upper spindle frequency range (14.25–15 Hz) during the luteal phase when progesterone typically rises, an effect that is often associated with increased Stage 2 sleep (Driver *et al.*, 1996). The increase in spindle activity is hypothesised to reflect an interaction between endogenous progesterone metabolites and GABA<sub>A</sub> membrane receptors (Driver *et al.*, 1996), the latter playing a critical role in sleep spindle generation (Belelli & Lambert, 2005). In support of this hypothesis, use of a synthetic progestin (medroxyprogesterone) is associated with a specific increase in upper spindle frequency activity (Plante & Goldstein, 2013) and use of combined progestin-estradiol contraceptive pills is associated with increased Stage 2 sleep in women (F. C. Baker, Mitchell, & Driver, 2001). However, progesterone levels and spindle frequency activity are not correlated, at least in the late-luteal phase of women with ovulatory menstrual cycles (F. C. Baker *et al.*, 2012). Alternatively, the increased spindle frequency activity may be explained by increased body temperature in the luteal phase (Driver *et al.*, 2008), or may help maintain sleep quality in the presence of substantial physiological and hormonal changes (Shechter & Boivin, 2010).

Contrary to most laboratory-based PSG studies that have not observed menstrual cycle related changes in overall sleep duration or quality, a recent in-home actigraphic study of 163 late-reproductive age women reported a moderate decline in SE (5%) and a decrease in TST (25 min) in the premenstrual week (Zheng *et al.*, 2014). This discrepancy might be due to a larger sample size (*i.e.*, greater power in detecting small changes) compared with PSG studies, or that sleep may be impacted by the menstrual cycle to a greater extent with advancing age.

Given these effects of the menstrual cycle on objectively measured sleep, it is pertinent to ask whether the subjective experience of sleep is also affected. Approximately 70% of women report that their sleep is negatively impacted by menstrual symptoms, on average, 2.5 days every month (National Sleep Foundation [NSF], 1998). The premenstrual week and first few days of menstruation, when estradiol and progesterone levels are low or declining, are the times when women across a wide age range are most likely to report poor sleep (F. C. Baker & Driver, 2004; Kravitz *et al.*, 2005; Manber & Bootzin, 1997). As described in the sections below, women who experience severe menstrual-related symptoms, such as premenstrual mood changes or menstrual pain, are more likely to experience poor sleep quality.

### Premenstrual Syndrome (PMS)

Premenstrual syndrome (PMS) is characterised by emotional, behavioural, and physical symptoms that occur in the premenstrual phase of the menstrual cycle, with resolution soon after the onset of menses. Up to 18% of women have clinically relevant premenstrual symptoms that they perceive as distressing and that impact daily function (Halbreich, 2004).

Premenstrual dysphoric disorder (PMDD), a severe form of PMS that occurs in 3–8% of women (Halbreich, 2004), is classified as a depressive disorder (American Psychiatric Association, 2013). Women with severe PMS/PMDD typically report sleep-related complaints as well as daytime sleepiness, fatigue, and an inability to concentrate during the late-luteal phase (reviewed in F. C. Baker, Lamarche, Iacovides, & Colrain, 2008).

Laboratory-based PSG studies have not identified clear sleep-related causes of the sleep complaints reported by women with PMS/PMDD (F. C. Baker et al., 2008; Shechter & Boivin, 2010). A recent study found that 18 women with either severe PMS or PMDD reported poorer subjective sleep quality in their symptomatic late-luteal phase compared with the follicular phase (F. C. Baker et al., 2012). However, like most earlier studies (F. C. Baker et al., 2008), no corresponding worsening of PSG-measure of sleep quality (e.g., SE, arousals, SOL) was found. The perception of poorer sleep quality correlated with anxiety levels, suggesting that the mood state of women with severe PMS impacts their sleep assessments in the late-luteal phase (F. C. Baker et al., 2012). While objective measures of sleep disturbance such as number and duration of awakenings could affect perceived sleep quality (Keklund & Akerstedt, 1997) other physiological (e.g., autonomic nervous system activity) and psychological (e.g., mood state) factors could also influence subjective sleep quality, leading to discrepancies between objective and subjective measurements of sleep.

Despite there being little evidence from PSG studies of a *change* in sleep architecture from the follicular to the symptomatic late-luteal phase, PSG studies have shown *trait-like* differences (evident in both the follicular phase and late-luteal phase) in sleep architecture in women with PMS or PMDD compared with controls (F. C. Baker et al., 2008). In a comprehensive assessment of sleep every third night across an entire menstrual cycle, women with PMDD and insomnia symptoms had more SWS than controls regardless of menstrual cycle phase (Shechter, Lespérance, Ng Ying Kin, & Boivin, 2012a). Similarly, F. C. Baker et al. (2012) found increased SWS in both the follicular and late-luteal phases of the menstrual cycle in women with severe PMS/PMDD compared with controls. The increased SWS was hypothesised to be functionally linked to decreased melatonin secretion evident in the women with PMDD (Shechter et al., 2012a; Shechter, Lespérance, Ng Ying Kin, & Boivin, 2012b). Further evidence of a possible involvement of the sleep regulatory system with PMDD comes from studies of the effect of sleep deprivation (Parry et al., 1999). Partial sleep deprivation has a positive effect on mood in two thirds of patients with PMDD, although the effect may only be evident after recovery sleep (Parry et al., 1999). Although not well integrated, these findings suggest evidence of alterations in both homeostatic and circadian processes in women with PMDD throughout the menstrual cycle. Indeed, others have also reported differences in psychophysiological parameters that are evident outside of the symptomatic late-luteal phase in women with PMDD compared with controls (Poromaa, 2014). These underlying “trait-like” differences may contribute to susceptibility to symptom expression in the late-luteal phase in women with PMDD. PMDD management includes selective serotonin reuptake inhibitors (the drug of choice recommended by the American

College of Obstetricians and Gynecologists), which are effective even if given only during the luteal phase. Other pharmacological options include anxiolytics and agents that suppress ovulation. Non-pharmacological agents such as calcium supplements, L-tryptophan administration, and cognitive-behavioral therapy have also been shown to be effective in some women (Rapkin, 2003).

## Dysmenorrhea

Dysmenorrhea, defined as painful menstrual cramps of uterine origin, is the most common gynaecological condition among women of reproductive age, and is very severe in approximately 10–25% of women (Dawood, 1990). Primary dysmenorrhea is menstrual pain without organic disease and secondary dysmenorrhea is associated with conditions such as endometriosis and pelvic inflammatory disease. Menstrual cramps impact quality of life (Iacovides, Avidon, Bentley, & Baker, 2014), mood, and sleep quality. Women with primary dysmenorrhea have more disturbed sleep (poorer subjective sleep quality; lower sleep efficiency; increased time spent awake, moving, and Stage 1 light sleep; and less REM sleep) when they are experiencing pain during menstruation compared with pain-free phases of the menstrual cycle, and compared with women who do not suffer menstrual pain (F. C. Baker, Driver, Rogers, Paiker, & Mitchell, 1999). The relationship between sleep and pain is bidirectional (Lautenbacher, Kundermann, & Krieg, 2005) such that menstrual pain disrupts sleep and disturbed sleep may exacerbate pain. Treatment of nocturnal pain with non-steroidal anti-inflammatory drugs alleviates painful cramps and is associated with an improved sleep quality in women with primary dysmenorrhea (Iacovides et al., 2014).

## Perinatal Period

Sleep and wake patterns are often disrupted during the perinatal period, with evidence from subjective and objective measurements of sleep suggesting that sleep quality and quantity decrease as pregnancy progresses, and are still compromised 3 months after childbirth compared with pre-pregnancy levels. The following sections will discuss specific findings on objectively and subjectively measured sleep during pregnancy and the post-partum period.

## Subjective Sleep

Compared with non-pregnant controls, pregnant women self-report significantly poorer sleep (Ko, Chang, & Chen, 2010). Longitudinal studies have described changes in sleep over the perinatal period. A study that measured subjective sleep in 325 women during each trimester of pregnancy and at 3 months after delivery, found night-time awakenings increased from the first trimester, and SE decreased throughout pregnancy (Hedman, Pohjasvaara, Tolonen, Suhonen-Malm, & Myllylä, 2002). Self-report TST was elevated during the first trimester, slightly decreased during the second trimester, and reduced substantially during the third trimester of pregnancy (Hedman et al., 2002). A small study ( $N = 7$ ) using continuous sleep diaries from 5 until 12 weeks post-partum found a progressive

decrease in wake after sleep onset across the assessment period, with sleep patterns from 9 to 12 weeks being consistent with the mothers' reports of infant sleep-wake patterns and feeding practices (Horiuchi & Nishihara, 1999). These findings suggest that women perceive their sleep to be disrupted from as early as the first trimester throughout the pregnancy, and improve gradually during the post-partum period.

### Objective Sleep

Cross-sectional PSG studies have found significant differences in sleep architecture in pregnant women compared with non-pregnant controls. An early study (Karacan, 1968) that compared PSG measures of 7 women in the last month of pregnancy with 9 age-matched non-pregnant controls, found that pregnant women had longer SOL, more night-time awakenings, less TST, and less SWS. Similarly, a study that compared 12 women in the third trimester to 10 non-pregnant controls (Hertz *et al.*, 1992), found that the pregnant group had lower SE due to night-time awakenings, spent more time in Stage 1 sleep, and had a lower percentage of REM sleep; however, no differences in TST or SWS between groups were found. Using a longitudinal design in 31 women, Lee, McEnany, and Zaffke (K. A. Lee, McEnany, & Zaffke, 2000a) explored pregnancy-changes in sleep architecture by measuring sleep prior to pregnancy, at each trimester of pregnancy, and at 1 and 3 months post-partum. TST was the lowest at 1 month post-partum (*i.e.*, 6.2 hr), followed by the third trimester and pre-pregnancy baseline, and was highest during the first trimester (*i.e.*, 7.4 hr). SE decreased progressively across pregnancy, from 93% before pregnancy to 81% at 1 month post-partum. In addition, SWS progressively decreased throughout pregnancy, but there were no significant changes in REM sleep over time. An improving trend in all aspects of sleep was observed at 3 months post-partum, although neither sleep quality nor quantity returned to pre-pregnancy levels (K. A. Lee *et al.*, 2000a).

Studies using actigraphy support the PSG finding that night-time sleep deteriorates progressively throughout pregnancy, particularly during the last weeks of gestation, and is the worst immediately before childbirth (Beebe & Lee, 2007; Matsumoto, Shinkoda, Kang, & Seo, 2003). A recent study of actigraphy measured sleep during the weeks immediately before and after delivery (Bei, Calcagni, Milgrom, & Trinder, 2012) reported that, although TST at night was significantly disrupted after giving birth, napping increased significantly, resulting in TST across 24 hr remaining relatively stable. Compared with the antenatal period when naps were most likely to occur during early afternoon, naps were evenly distributed across late morning to early evening during the first post-partum week. These findings suggest that childbirth is followed by redistribution of sleep across 24-hr periods, raising questions as to the restorative value of sleep new mothers obtain. Similar to findings from PSG studies, actigraphy-measured sleep showed an improving trend during the post-partum period, with reports that at 10 weeks post-partum, sleep patterns were similar to that in the third trimester, although sleep quality was still worse than matched non-pregnant controls (Matsumoto *et al.*, 2003).

## Factors That Affect Sleep during the Perinatal Period

### Physiological alterations

Many steroid hormones rise dramatically during pregnancy, including estrogen, progesterone, and prolactin. Elevated levels of progesterone have been associated with increased daytime sleepiness and earlier sleep onset (Herrmann & Beach, 1978). The inhibitory effect of progesterone on smooth muscles might also lead to increased urinary frequency during early pregnancy that might disrupt sleep (K. A. Lee, 1998). Other pregnancy related physical discomforts, such as nausea, tender breasts, headache, vaginal discharge, flatulence, constipation, shortness of breath, backache, and heartburn (K. A. Lee, 1998) have also been recognised as sleep disrupting (Kamysheva, Skouteris, Wertheim, Paxton, & Milgrom, 2010).

### Sleep disorders

The prevalence of some sleep disorders increases during pregnancy, an effect that has been linked to sleep fragmentation and increased daytime sleepiness among affected women. In a study of 502 pregnancies (Franklin *et al.*, 2000), 23% of the sample reported regular snoring, while the rate for before pregnancy was only 4%. Potential contributing factors include changes in the respiratory system during pregnancy, such as reduced pharyngeal dimensions (Pilkington *et al.*, 1995), decreased nasal patency, and increased congestion and rhinitis (Bende & Gredmark, 1999). The prevalence of restless legs syndrome (RLS) has also been shown to increase during pregnancy (K. A. Lee, Zaffke, & Baratte-Beebe, 2001) and to resolve rapidly post-partum (Goodman, Brodie, & Ayida, 1988). A large cross-sectional study involving approximately 16,000 pregnant women (Suzuki *et al.*, 2003) found that at 3 to 4 months of gestation, 15% reported RLS symptoms, and this increased to 23% at term. Potential contributing factors include iron deficiency and serum folate levels (K. A. Lee *et al.*, 2001).

### Labour

Beyond acute sleep deprivation caused by birth-giving, especially nighttime labour, there is nighttime sleep deprivation the week immediately following labour (Bei, Milgrom, Ericksen, & Trinder, 2010). Some studies have reported that caesarean sections were associated with shorter TST and more frequent nighttime awakenings than vaginal deliveries, probably due to factors related to surgical recovery (S.-Y. Lee & Lee, 2007).

### Infant behaviours

During the post-partum period, various needs of the newborn are the main contributing factors to sleep disruptions. Actigraphy studies have shown that maternal sleep was closely associated with infant sleep/wake behaviours during the first 3 months post-partum (Goyal, Gay, & Lee, 2007; Horiuchi & Nishihara, 1999). Nighttime feeding and care-taking often lead to multiple awakenings in new parents, and more so when the temperament of the infant is difficult (Dennis & Ross, 2005; Hiscock & Wake, 2001). The number of self-report infant-related nighttime awakenings, but not the self-estimated total

wake time, was found to be associated with perceived sleep quality (Gress et al., 2010), highlighting the role of sleep disruption over reduced TST in perceived sleep quality.

### Parity

Findings on the effects of parity on sleep have been mixed. Some authors reported multiparas mothers to have less efficient sleep than nulliparas from before pregnancy until 3 months post-partum (K. A. Lee, Zaffke, & McEnany, 2000b) whereas others (Coo Calcagni, Bei, Milgrom, & Trinder, 2012) observed a greater deterioration in objective sleep and increased number of daytime naps in nulliparas during the third trimester and 1 week post-partum. Multiparas have also been reported to initiate SWS quicker than nulliparas, although no differences have been reported in objective TST, %REM sleep, and %SWS (K. A. Lee et al., 2000a).

These findings suggest that sleep disruption is common, and the cause is multi-factorial during the perinatal periods. Child-bearing women might experience chronic sleep fragmentation during pregnancy, acute sleep deprivation during labour and immediate post-partum periods, as well as chronic partial sleep deprivation and disruption during the first few months after childbirth.

### Menopausal Transition and Postmenopause

The natural menopausal transition typically starts in women's in their late 40s, the median age at the final menstrual period being 51.4 years (Santoro, 2005). There are substantial endocrine changes during this period, for example, a decrease in estrogen, and increases in follicle-stimulating hormone and testosterone. The majority of women also experience marked physiological changes, including changes in menstrual cycles, skin conditions, and body temperature. Vasomotor symptoms comprise night sweats and hot flushes, are experienced by up to 74% of women (Guthrie, Dennerstein, Taffe, & Donnelly, 2003), and are considered among the core symptoms of menopause.

Complaints regarding sleep quality and continuity are not only common (reported by 33–51% of women [Joffe, Massler, & Sharkey, 2010; Polo-Kantola, 2008; Shaver & Zenk, 2000]), but are also among the most bothersome symptoms (Ford, Sowers, Crutchfield, Wilson, & Jannausch, 2005) during the menopausal transition. Epidemiological studies, for example, the Survey of Women's Health Across the Nation (SWAN) study (Kravitz et al., 2003) and the Seattle Midlife Women's Health Study (SMWHS; Woods & Mitchell, 2010), have consistently shown that sleep complaints increase across the menopausal transition and persist into postmenopause. Compared with premenopausal women, odds ratio for sleep complaints were 1.3 for perimenopausal women (Kravitz et al., 2003) and 1.3–3.4 for postmenopausal women (Kravitz et al., 2003; Kuh, Wadsworth, & Hardy, 1997; Young, Rabago, Zgierska, Austin, & Laurel, 2003). In contrast to the association between menopausal status and subjective sleep complaints, PSG studies have shown either no significant differences in sleep architecture among pre-, peri-, and postmenopausal women (Shaver, Giblin, Lentz, & Lee, 1988), or that sleep was better after menopause (Young

et al., 2003). Quantitative analysis of sleep EEGs from the SWAN study revealed that although no menopause status related differences were found in PSG derived sleep variables, beta activity during sleep was elevated in peri- and post-, compared with premenopausal women, an effect partially accounted for by the occurrence of vasomotor symptoms (I. G. Campbell et al., 2011).

Indeed, poor sleep has often been attributed to nighttime vasomotor symptoms (i.e., night sweats) (Ameratunga, Goldin, & Hickey, 2012; Moe, 2004). However, the extent to which vasomotor symptoms disturb sleep is not clear in the current literature. Although it is well established that self-reported vasomotor symptoms are associated with symptoms of insomnia and subjective sleep complaints (Ameratunga et al., 2012), findings have been mixed and inconclusive when sleep was measured objectively using PSG or actigraphy (Ensrud et al., 2009; Polo-Kantola et al., 1999; Sharkey, Bearpark, & Acebo, 2003; Shaver et al., 1988; Thurston, Santoro, & Matthews, 2012; Young et al., 2003). Further, when vasomotor symptoms were also measured objectively using skin conductance or skin temperature measures, reports of their relationship with objectively measured sleep have also been somewhat mixed. While most studies reported objective vasomotor symptoms were associated with poorer objective sleep (I. G. Campbell et al., 2011; Erlik et al., 1981; R. R. Freedman & Roehrs, 2006; Gonen, Sharf, & Lavie, 2009; Woodward & Freedman, 1994), others showed no significant relationship between the two (R. R. Freedman & Roehrs, 2004; Thurston et al., 2012). Among studies that showed an association between objective vasomotor symptoms and PSG sleep, between 46% (Gonen et al., 2009) to 59.7% (Erlik et al., 1981) of waking episodes occurred within 5 min before and after a hot flush. More recently, Joffe et al. (2013) experimentally induced hot flushes using gonadotropin-releasing hormone agonist leuprolide in healthy premenopausal women without hot flashes or sleep disturbances. This study demonstrated a causal relationship between objectively recorded hot flushes and worse sleep quality on both actigraphy and self-report.

These findings suggest that while vasomotor symptoms probably play a role in menopause related sleep disturbance, other factors are also likely to be relevant. Data from the SWAN study showed that the increase in sleep complaints during the menopausal transition was partially related to a change in hormone levels (Kravitz et al., 2008), while the SWMHS study showed that other factors such as depressed mood, perceived health, and stress contributed significantly to a menopause-related increase in sleep complaints (Woods & Mitchell, 2010). Personality factors such as neuroticism have also been linked to menopause-associated insomnia, suggesting that some women may be more susceptible to developing insomnia in association with menopausal hormone changes (Sassoon, de Zambotti, Colrain, & Baker, 2014). Indeed, it is hypothesised that some women are more sensitive to reproductive hormone transitions. Women who suffer from PMDD are at a greater risk for post-partum depression and menopausal depression (Freeman, Sammel, Lin, & Nelson, 2006; Yonkers, 1997). Aging related changes in sleep might also contribute to poor sleep in menopausal women. Compared with women in their 20s, late reproductive stage women (aged 45–51 years) were found to have

poorer PSG SE that was similar to that of postmenopausal women (Kalleinen *et al.*, 2008). Primary sleep disorders such as sleep-disordered breathing, RLS, periodic limb movement syndrome, which are under-recognised and under-diagnosed in women, may contribute to the age-related deterioration in women's sleep. A study of over 100 women aged 44–56 who reported disturbed sleep found that 53% had primary sleep disorders, mainly sleep disordered breathing, RLS, or both (R. R. Freedman & Roehrs, 2007). In addition, comorbid physical and psychiatric conditions, as well as pain disorders are also commonly associated with sleep complaints during the menopausal transition (Ameratunga *et al.*, 2012).

In summary, women have increased sleep complaints during the menopausal transition and postmenopause. Although such sleep complaints have not been consistently confirmed when sleep was measured objectively, there is evidence supporting a probable association between vasomotor symptoms and sleep disturbance. Sleep disturbance during the menopausal transition is likely to be multi-factorial, with changes in reproductive hormones, aging, and comorbid sleep, physical, and psychiatric conditions all playing roles.

## Sleep and Psychological Well-Being

Sleep disturbances over a woman's lifespan are closely associated with their psychological well-being. The bidirectional relationship between sleep and mental health, particularly mood problems, is well established in the general population (Alvaro, Roberts, & Harris, 2013). On one hand, psychiatric disorders such as major depression are associated with high rates (50–85%) of sleep complaints (Morin & Ware, 1996). On the other hand, both acute and chronic sleep disruption have been linked to worsened mood, irritability, and decreased motivation in subclinical populations (Rosen, Gimotty, Shea, & Bellini, 2006; Zohar, Tzischinsky, Epstein, & Lavie, 2005), leading to the notion that sleep disturbances might play mechanistic roles in the development and maintenance of mood disorders (Harvey, 2011).

A significant association between sleep disturbance and mood problems in women has been consistently shown by studies that measured sleep subjectively. For example, in subclinical samples, poor subjective sleep quality during early pregnancy significantly predicted depressive symptoms in late pregnancy (Skouteris, Germano, Wertheim, Paxton, & Milgrom, 2008), while higher levels of depressive symptoms were associated with lower TST, longer SOL, and greater daytime sleepiness during the first 3 months post-partum (Goyal *et al.*, 2007). Similarly, among community dwelling middle-aged women, poorer perceived sleep quality has been associated with higher levels of anxiety (Cheng *et al.*, 2008) and depression (Brown, Gallicchio, Flaws, & Tracy, 2009). While an association between premenstrual negative mood symptoms, such as anxiety, and poor sleep quality has been shown (F. C. Baker *et al.*, 2012), it is unknown whether the poor mood contributes to poor perceived sleep quality or vice versa. It is also unclear whether women who do not suffer from PMDD but who have chronic insomnia experience a worsening of their symptoms in association with the premenstrual phase of the menstrual cycle. Interestingly, in women with prior depressive disorder, who were not

depressed during pregnancy, sleep complaints in the first 4 months post-partum were associated with a higher risk for recurrent depression (Okun *et al.*, 2011). These findings suggest that sleep complaints are not only relevant to concurrent mood, but might also be a risk factor for future mood problems, especially among vulnerable women.

In studies in which sleep was measured objectively, the sleep–mood relationship has been shown to be weaker compared with when sleep was assessed through subjective reports. In the perinatal population, the absence of significant associations between objective nighttime sleep and mood has been reported by several studies (Bei *et al.*, 2010; Dørheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009; Okun *et al.*, 2013). However, some objective sleep parameters, for example higher sleep fragmentation and WASO, lower SE (Park, Meltzer-Brody, & Stickgold, 2013), more variable sleep duration (Tsai & Thomas, 2012), and more frequent napping behaviours (Bei *et al.*, 2010) have been associated with worse mood in post-partum women. In perimenopausal (but not premenopausal) women, a significant association has been reported between worse mood and greater actigraphy-measured arousals from sleep (A. Baker, Simpson, & Dawson, 1997), alluding to the notion that sleep disturbance associated with the menopausal transition might have negative effects on mood. The “domino hypothesis” (S. Campbell & Whitehead, 1977), in which vasomotor symptoms lead to sleep disturbance and poor sleep leads to poor mood, was recently tested in a study using daily ratings of vasomotor symptoms, sleep, and mood (Burlison, Todd, & Trevathan, 2010). The study reported that sleep had significant effects on mood the next day, but did not mediate the relationship between vasomotor symptoms and mood (Burlison *et al.*, 2010). In addition, a PSG study suggested that the presence of sleep disorders such as sleep disordered breathing and RLS were the best predictors of objective sleep quality, while anxiety and hot flushes were the best predictors of subjective sleep (R. R. Freedman & Roehrs, 2007), suggesting multiple factors affect the relationship between sleep and mood in this population.

The strong association between subjective sleep and mood suggests that individual differences in psychological factors might contribute to the underlying mechanisms in the sleep–mood relationship. Indeed, a recent study on 122 new mothers found that poor perceived sleep quality and high sleep-related daytime dysfunction, were associated with aspects of cognitive appraisal that were closely linked to maternal distress such as negative expectations about the future and low perceived ability to cope practically and emotionally with motherhood (Coo, Milgrom, Kuppens, Cox, & Trinder, 2014). During the menopausal transition, the role of cognitive appraisal in psychological consequences of vasomotor symptoms has been well established (Hunter & Mann, 2010). Although not directly tested in the context of sleep, this cognitive model highlights the importance of women's thoughts, beliefs, and subjective appraisal in how they psychologically respond to physiological changes, which like many types of sleep disturbance discussed earlier, are relatively external to their personal control. This also suggests that psychological interventions that help improve sleep and address unhelpful cognitive processes might be beneficial to women's psychological well-being.

Psychological interventions that aim to improve women's sleep are currently limited but growing. In new mothers, sleep hygiene (K. A. Lee & Gay, 2011) and behavioural/education (Stremler et al., 2006) based interventions have led to longer and better sleep. A recent open pilot trial of Cognitive Behavioural Therapy for Insomnia (CBT-I) in women with coexisting post-partum depression and insomnia (Swanson, Flynn, Adams-Mundy, Armitage, & Arnedt, 2013) found that after a 5-week individual treatment program, there were significant increases in sleep duration and quality, as well as reduction in symptoms of depression and fatigue. This suggests that a brief course of CBT-I has the potential of improving both sleep and mood in new mothers with insomnia and depression. Although non-pharmacological interventions specifically targeting menopause-related sleep problems are still under development, interventions for menopausal symptoms (e.g., vasomotor symptoms) utilising CBT (Norton, Chilcot, & Hunter, 2013), mindfulness (Carmody et al., 2011), and paced respiration (Carpenter et al., 2012) have been shown to have sleep-improving effects.

## Implications

Research findings reviewed in this article have a number of practical implications in the assessment and treatment of sleep complaints in women. Sleep disturbance is common among women of all ages seeking psychological treatment, even though it might not always present as a chief complaint. The bidirectional relationship between sleep and mental health suggests that sleep complaints might be a symptom of, and/or risk factor for psychopathology (e.g., mood disorders) that might require clinical attention and therefore should not be dismissed when presented in clinical settings. In addition, addressing sleep problems might be beneficial for women's overall health and well-being. In assessing sleep complaints in women, health professionals need to be mindful that multiple factors, such as hormonal changes, age-related physiological changes, psychosocial factors, the presence of sleep disorders, as well as physical and mental health conditions, can all contribute to complaints of poor sleep. Therefore, an individualised assessment and treatment approach could help address the heterogeneity in sleep problems presented across women of different ages.

A careful evaluation of a woman's sleep problems will take into consideration her sleep and mental/physical health history, both nighttime symptoms and daytime consequences, as well as potential contributing factors to poor sleep. Given many sleep problems in women involve sleep restriction and/or disturbance that are associated with health or reproductive events (e.g., menstrual cycles, pregnancy, infant care), a distinction between such sleep disturbance and insomnia disorder is of particular relevance to effective management of sleep problems in women. While compromised sleep duration and quality might be present in both externally caused sleep disturbance/sleep restriction and insomnia, the presence of sleep difficulty despite adequate sleep opportunity, as well as clinically significant distress (e.g., worries and preoccupation about not getting enough sleep, distress associated with bed and sleep) associated with sleep disturbance are

needed for the diagnoses of insomnia (American Psychiatric Association, 2013).

To address externally caused sleep disruption/restriction, reducing or eliminating factors contributing to disrupted/restricted sleep opportunity have been shown to be effective, for example, addressing infant sleep problems (Stremler et al., 2006), or reducing vasomotor symptoms (Veerus, Hovi, Sevón, Hunter, & Hemminki, 2012). In cases of insomnia disorder, there is strong evidence for CBT-I being an effectiveness intervention for improving sleep (Morin et al., 2006), and clinicians should consider incorporating CBT-I, especially for those women who might have concerns regarding the use of medication (e.g., lactating women). In light of high prevalence of other mental/physical conditions with insomnia, especially among women, the current guidelines suggests concurrently treating and managing insomnia as a comorbid condition, rather than a condition secondary to other mental/physical conditions (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). In addition, when the presence of other sleep disorder (e.g., sleep-disordered breathing, RLS) is suspected, a referral for comprehensive assessments and specialised treatments by a sleep physician should be made.

## Conclusion

Sleep disturbances are common, and the causes are heterogeneous in women across the lifespan. The collective literature points to a bidirectional relationship between sleep disturbance and psychological well-being. Comprehensive assessment and targeted intervention with an individualised approach are required for effective treatment and management of sleep problems in women. Future studies are needed to better understand the mechanisms underlying the links between sleep and mental health in women, which will help identify those who might be particularly vulnerable to sleep-related psychological problems for targeted intervention. Further, assessment tools for better distinguishing sleep disturbance/restriction caused by health and reproductive events from insomnia disorder are particularly needed for women presenting with sleep complaints.

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