CHAPTER 10

Women's Sleep Throughout the Lifespan

Jacqueline D. Kloss and Christina O. Nash

INTRODUCTION

The role of sleep in women's health and quality of life has garnered significant research and clinical attention over the past 10 to 15 years (Dzaja et al., 2005; Kravitz et al., 2008; Kryger, 2004; Lee & Kryger, 2008; National Sleep Foundation [NSF], 2007; Wolfson, 2001). Once considered a confound to sleep research, hormonal fluctuations of the menstrual cycle, pregnancy, and menopause are now of primary scientific inquiry. Gender disparities in sleep research further direct us toward the physiological, psychological, and sociocultural factors that contribute to sleep disturbance among women. It is well recognized that sleep is essential for health and well-being, and the consequences of acute and chronic sleep deprivation can be severe (see Bonnet, 2005; Dinges, Rogers, Baynard, 2005, for review), ranging from impaired daytime functioning to compromised physical and emotional health. Given that sleep disturbance among women is almost twice that of men (Soares, 2005; Zhang & Wing, 2006), a more comprehensive understanding of women's sleep is warranted.

Findings from the Sleep in America Poll (NSF, 2007; $n = 1{,}003$, American women ages 18–64) indicated that nearly half of the sample reported having sleep problems every night, and approximately 60% of women endorsed the item "I had a good night's sleep" only a few nights per week or less. Women's ratings of daytime sleepiness were correlated with high stress, less time with friends and family, work tardiness, complaints of "too tired for sex," and drowsy driving. Poor sleep also correlated with negative mood states, such as anxiety, worry, and sadness. Health-enhancing behaviors, such as sexual activity, nutrition, and exercise, were also compromised. All too often, women sacrifice sleep because of social, occupational, familial, and/or domestic demands, at the expense of such daytime sequelae; some may also find themselves, by choice or necessity, engaging in shift work, which may lead to serious health repercussions or vulnerability to disease (Shechter, James, & Boivin, 2008). Women are also predisposed to certain sleep disorders, including insomnia, restless legs syndrome (RLS), and obstructive sleep apnea (OSA), at various periods of their lives. Likewise, health conditions such as medical or psychiatric comorbidities can place women at greater risk for sleep disturbance.

After providing a cursory primer on sleep, women's sleep throughout the lifespan is explored, to include sleep variation during menstruation, pregnancy, peri- and postmenopause, and aging. Where available, literature is presented on sociocultural factors, such as race/ethnicity and socioeconomic status (SES), although these topics have

only recently gained attention in the area of sleep. Considering that the literature on sleep disorders is vast, primary insomnia and OSA were selected to illustrate their pertinence to women's health. Lastly, we include empirically based strategies that can be useful in ameliorating the symptoms that contribute to or stem from sleep complaints.

Sleep Primer

Homeostatic drive (the propensity to sleep contingent on the duration of wakefulness) and circadian rhythm (our roughly 24-hour biological clock) regulate sleep and wakefulness (see Carskadon & Dement, 2005, for review). These processes provide a backdrop by which reproductive hormones, mood, beliefs, cognition, and sociocultural influences interact to yield sleep. Technologies such as polysomnography (PSG) and actigraphy (a motion sensor worn as a wristwatch that measures gross motor movements and approximates sleep and wake states) have made it possible to measure sleep objectively, whereas questionnaires or daily sleep diaries of sleep/wakefulness patterns have enabled the subjective measurement of women's sleep quantity, continuity, or quality. Sleep architecture is characterized by both non–rapid eye movement (NREM) and rapid eye movement (REM) sleep, which alternate throughout the night, with the average sleep cycle lasting between 90 and 110 minutes. Stage 1 sleep initiates the transition from wakefulness to sleepiness and is followed by Stage 2 sleep (marked by sleep spindles on electroencephalogram [EEG]) and slow-wave sleep (SWS) or deep sleep. Approximately 80 minutes from sleep initiation, REM sleep begins (composing 20% to 25% of sleep). Notably, as the night progresses, periods of REM increase while SWS decreases. The average sleep need for adults is about eight hours per night (Van Dongen, Maislin, Mullington, & Dinges, 2003).

SLEEP AND THE MENSTRUAL CYCLE

The hormonal fluctuations of the menstrual cycle lead investigators to compare sleep during the follicular (pre-ovulatory) phase (FP) and the luteal (post-ovulatory) phase (LP) and to examine sleep in relation to estrogen, which peaks right before ovulation, and progesterone, which dominates after ovulation. The study of sleep throughout the menstrual cycle warrants attention with regard to: (a) variability in intra- and inter-individual timing of ovulation, peak levels of reproductive hormones, anovulation, cycle length, and hormonal intervals; (b) the interactive influences of mood, dysmenorrhea, pain, or other medical conditions; (c) expense of PSG, EEG studies; (d) maintaining constant environments; (e) impracticalities of enrolling women for long durations; and (f) subsequent small sample sizes (Manber & Armitage, 1999). Thus, data are limited and should be interpreted with caution.

Subjective and Objective Sleep Changes

According to the 2007 Sleep in America Poll, 33% of women reported sleep disruption coinciding with their menstrual cycle. The late LP (i.e., the 6 days leading up to menstruation) is marked by sleep complaints independent of other premenstrual symptoms. Manber & Bootzin (1997) found that among a sample of 32 healthy women, sleep-wake diaries recorded over two menstrual cycles revealed a self-reported increase

in sleep-onset latency (SOL) and decrease in sleep efficiency (SE) and sleep quality in the LP, compared to the mid-FP, independent of the severity of associated premenstrual complaints. In a similar study of 26 young, healthy women over the course of one menstrual cycle, sleep quality ratings were diminished in the 3 premenstrual and first 4 days of menstruation, but SOL, sleep continuity, sleep maintenance, and sleep duration did not vary across the menstrual cycle (Baker & Driver, 2004). Despite subjective complaints of sleep disturbance, sleep architecture among asymptomatic women (women without significant premenstrual symptoms) remains fairly stable during hormonal fluctuations. Although a complete review of the nuances of sleep architecture is beyond the scope of this chapter, recent reviews indicate that decreases in REM and increased Stage 2 sleep in the LP (Driver, Werth, Dijk, & Borbely, 2008; Shechter & Boivin, 2010) have been consistently documented. SWS does not typically vary in response to menstrual cycle changes (Driver, Dijk, Werth, Biedermann, & Borbely, 1996), signifying a resilient sleep homeostat.

Premenstrual Syndrome (PMS) or Premenstrual Dysphoric Disorder (PMDD), Dysmenorrhea, Sleepiness, and Sleep

PMS and PMDD Sleep disruption—characterized by hypersomnia, insomnia, fatigue, sleep disruption, frequent arousals, and disturbing dreams—is considered a defining symptom of PMS and PMDD (Mauri, 1990). The degree to which women with severe premenstrual symptoms experience more sleep disturbance compared to their asymptomatic counterparts is questionable, and the mechanism that contributes to premenstrual sleep disturbance has yet to be clearly defined. Similar to asymptomatic controls, increased Stage 2 (spindle activity) and reductions in REM are observed, and some differences in SWS are noted during the LP (Lamarche, Driver, Wiebe, Crawford, & DeKonnick, 2007), but robust differences in sleep architecture between PMS/PMDD sufferers have not been consistently documented. Some studies show no change in sleep-wake cycles or sleep architecture among women with premenstrual symptoms compared to controls (Chuong, Kim, Taskin, & Karacan, 1997; Parry et al., 1999). Lee, Shaver, Giblin, and Woods (1990) found that in a sample of young, healthy women, women who endorsed premenstrual symptoms marked by negative affect evidenced significantly less SWS across both the FP and LP, compared to asymptomatic individuals. Parry, Mendelson, Duncan, Sack, and Wehr (1989) found decreased REM and increased Stage 2 across the cycle among PMS sufferers compared to healthy controls.

More recently, Baker and Driver (2007) compared women with severe PMS symptoms and found that they perceived their sleep as more disturbed than asymptomatic women, although these differences were unrelated to menstrual phase. Baker, Lamarche, Iacovides, and Colrain (2008) characterized the sleep of individuals with PMS/PMDD as trait-like, with marginal differences compared to controls across the menstrual cycle. They also suggested that women with severe PMS may have a greater sensitivity to these subtle changes or exhibit a negative reporting bias. For example, women who suffered from menstrual pain or discomfort were more likely to evidence sleep disruption and report sleepiness, compared to asymptomatic women (Baker, Driver, Rogers, Paiker, & Mitchell 1999),

and EEG patterns of symptomatic women differed in a trait-like fashion compared to asymptomatic controls (Baker, Kahan, Trinder, & Colrain, 2007). Limited sample sizes, limited numbers of studies, and variation in measurement of symptom severity render conclusions about the nature of PMDD sleep disturbance tentative. Moreover, Baker and colleagues (2008) point to factors beyond sleep architecture that may account for symptom complaints, such as circadian processes in PMS/PMDD.

Circadian and Hormonal Influences

Both melatonin and core body temperature (cBT) may contribute to interactive effects between circadian and menstrual processes. Progesterone is associated with a rise of cBT but a decrease in temperature rhythm amplitude (see Baker & Driver, 2007; Driver et al., 2008 for review). Recently, Shechter, Varin, and Boivin (2010) showed that REM was sensitive to the changes of the menstrual cycle coinciding with rising progesterone and cBT changes. Among asymptomatic women, data on melatonin and the menstrual cycle have been equivocal (Shechter & Boivin, 2010), although several well-controlled studies suggest that melatonin appears unaltered (Berga & Yen, 1990; Shechter et al., 2010; Shibui et al., 2000; Wright & Badia, 1999). However, women with PMDD may have underlying circadian rhythm dysregulations (see Baker et al., 2008; Shechter & Boivin, 2010 for review) compared to controls.

Interestingly, both decreased melatonin secretion and altered circadian rhythms are also characteristic of sleep patterns among individuals with major depression (Germain & Kupfer, 2008; Srinivasan et al., 2006). An increased nocturnal cBT may account for premenstrual symptom reports (Parry, LeVeau, et al., 1997; Severino et al., 1991), as well as a trend for a phase-advanced temperature rhythm over the entire menstrual cycle (Parry et al., 1989). Hahn, Wong, and Reid (1998) also observed thermal dysregulation among reproductive women, particularly among women with more severe ratings of PMS, who indicated chills and sweats in the late LP (compared to the FP).

Dysmenorrhea and Sleepiness Women with dysmenorrhea (painful menstruation) reported significantly worse sleep quality, compared to women without primary dysmenorrhea (Baker et al., 1999) and during pain-free phases of their cycle (e.g., during the FP and earlier LP). PSG of dysmenorrheic women revealed reduced sleep efficiency, increased time awake, reduced Stage 1 sleep, and less REM compared to both controls and pain-free phases of their cycle.

Likewise, increased severity of PMS symptoms is associated with increased sleepiness ratings during the LP (Mauri, 1990; Manber & Bootzin, 1997). Perhaps women with greater severity of PMS may require more sleep, particularly during the LP (Manber & Bootzin, 1997). Lamarche et al. (2007) showed that women with severe premenstrual symptoms evidenced greater sleepiness and less alertness compared to their less-symptomatic counterparts in the late LP. Thus, sleepiness appears more marked among women with severe PMS symptoms, yet sleep architecture does not appear to account for sleepiness (Baker et al., 2007), warranting further study of the underlying link between sleepiness and menstrual changes.

Treatments to Alleviate Premenstrual Sleep Disturbance

Iacovides, Avidon, Bently, and Baker (2009) found not only relief, but also better sleep

quality and efficiency, among women with dysmenorrhea who were administered nonsteroidal anti-inflammatory drugs (NSAIDs) compared to a placebo. Napping (< 30 minutes) among women with and without premenstrual symptoms was beneficial in alleviating negative mood and sleepiness and increasing alertness and positive mood, as well as improving attention and cognitive processing (Lamarche, Driver, Forest, & DeKonnick, 2010). Women with premenstrual symptoms evidenced an even greater improvement in negative mood a half hour after napping compared to women with minimal or no symptoms, with no deleterious effects on the following night's sleep observed. Although antidepressants have been used as a gold standard to treat PMDD symptoms, several alternative, nonpharmacological treatments have been tried with some success. Evening bright light therapy has shown initial success in alleviating premenstrual symptoms (Lam et al., 1999). Similar to the use of sleep deprivation to treat depression, both partial and total nights of sleep deprivation have yielded encouraging results in improving mood among premenstrual syndrome sufferers (Parry et al., 1995; Parry & Wehr, 1987). Further study is needed to better understand the efficacy and practicality of these alternative treatments.

Oral Contraceptives (OCs) and Sleep

Women who take oral contraceptives, which suppress ovulation, have often been used as control subjects given their stable hormonal profile. However, the question arises about the degree to which the sleep of women is altered as a result of OC use. The sleep architecture of women taking OCs appears to differ from that of naturally ovulating women (see Armitage, Baker, & Parry, 2005; Baker & Driver, 2007; Baker et al., 2001; and Burdick, Hoffman, Armitage, 2002, for review). Women who take OCs may also evidence decreased deep sleep, increases in Stage 2, and changes in REM relative to women with naturally occurring cycles (Baker, Mitchell, & Driver, 2001; Burdick, Hoffmann, & Armitage, 2002). OCs containing estrogen and progestin may raise cBT, resulting in thermodysregulation, and are hypothesized to impact melatonin levels, yet prior investigations have yielded conflicting findings (Delfs et al., 1993; Wright & Badia, 1999). Sleep quality and sleep efficiency do not appear different among women taking OCs (Baker, Mitchell, & Driver, 2001; Burdick, Hoffman, & Armitage, 2002).

SLEEP DURING PREGNANCY AND POSTPARTUM

Sleep During Pregnancy

Estimates ranging from 15% to 80% of women experience altered sleep during pregnancy (Hedman, Pohjasvaara, & Tolonen, 2002). Physical, hormonal, and behavioral changes can threaten sleep quality during pregnancy (Soares & Murray, 2006). As levels of placental hormones rise, sleep becomes more vulnerable to disruption and fragmentation. Estrogen, progesterone, and prolactin are essential to the early stages of implantation and gestation (Sahota, Jain, & Dhand, 2003). In the early stages of pregnancy, progesterone has a sedative effect on women, and thus, a woman may experience fatigue and sleepiness as some of the first signs of pregnancy. During the first trimester, nausea, backaches, vomiting, and increased frequency to urinate are also

associated with sleep disruption (Lee, 1998). During the second and third trimesters, sleep continuity and quality are expected to decline (Lee, Zaffke, & McEnany, 2000; Signal, Gander, & Sangalli, 2007), with 60% to 97% of women reporting disrupted sleep patterns (Mindell & Jacobson, 2000). Fatigue, shortness of breath, discomfort from increasing fetal size, and leg cramps increase and likely contribute to more frequent nighttime awakenings (Soares & Murray, 2006).

The combination of increased fetus size, high levels of progesterone during the third trimester, and supine positioning at nighttime can also lead to increased complaints of breathing difficulty, including shortness of breath and snoring (Mindell & Jacobson, 2000). The prevalence of OSA among pregnant women is about 20% (Pien, Fife, & Pack, 2005), with obesity before pregnancy and additional weight gain during pregnancy increasing the risk for OSA. Although snoring is often overlooked during the third trimester, severe OSA may be related to a significant risk for fetal complications (Sahin et al., 2008), including increased fetal heart rate, gestational hypertension, smaller-sized infants relative to gestation time, preeclampsia (Okun, Roberts, Marsland, & Hall, 2009), gestational diabetes (Qui, Enquobahrie, Fredrick, Abetew, & Williams, 2010), and infants with lower Apgar scores and birth weights.

Pregnant women are also at increased risk for leg cramps and RLS, particularly during the third trimester. Leg cramps, classified as experiencing painful muscle contractions of the foot and/or calf area (American Sleep Disorders Association, 2006), affect up to 30% of pregnant women (Hensley, 2009). Women with RLS experience an urge to move the legs that worsens in the evening, throughout the night, or while at rest (Dzaja, Wehrie, Lancel, & Pollmacher, 2009). As many as 15% to 25% of women experience symptoms of RLS during the third trimester. Although women in general have higher reports of RLS relative to men, pregnant women experience RLS at two to three times the prevalence of the general population, which ranges from 5.5% to 10.6% (Allen et al., 2005). Lower levels of iron, folate, or ferritin and hemoglobin before pregnancy are hypothesized to contribute to increased incidence of RLS during pregnancy, and more recent evidence has suggested that estrogen may also trigger RLS symptoms (Dzaja et al., 2009). Although RLS is often a reversible syndrome during pregnancy (Wolfson & Lee, 2005) and often rescinds following delivery (Lee, Zaffke, & Baratte-Beebe, 2001), nearly 95% of RLS sufferers also experience difficulties falling asleep, fragmented sleep, and daytime sleepiness (Berger, Luedemann, Trenkwalder, John, & Kessler, 2004).

Disturbed sleep and sleep loss during the third trimester may also be linked to labor difficulties. Okun and colleagues (2009) also hypothesized that difficulties sleeping during pregnancy may be linked to preterm labor; however, this has yet to gain further empirical support. Lee and Gay (2004) found that among a sample of 131 women, those sleeping more than 6 hours had shorter labors and were less likely to give birth via cesarean section, relative to women who were sleeping less than 6 hours on average; women who slept less than 6 hours per night experienced 12-hour or longer labors and were 4.5 times more likely to require a cesarean section for delivery, relative to women sleeping more than 6 hours.

Sleep and Postpartum

Sleep problems prior to pregnancy have been associated with poor sleep quality postpartum

(Dorheim, Bondevik, Eberhard-Gran, & Bjorvatn, 2009). Empirical evidence has also linked poor sleep quality during pregnancy to an increase in depressive symptoms later in pregnancy and during the postpartum period (Skouteris, Germano, Wertheim, Paxton, & Milgrom, 2008; Wolfson, Crowley, Answer, & Bassett, 2003). Hormonal fluctuations postpartum may also explain the link between sleep and mood postpartum. Ross, Murray, and Steiner (2005) theorized that when estrogen and progesterone levels drop significantly after giving birth, a state of hyperarousal may ensue, thereby increasing insomnia and initiating a vicious cycle between disrupted sleep and mood. During the postpartum period, mothers experience poorer sleep efficiency (SE; total time asleep relative to total time in bed), less total sleep time (TST), and less REM, a key component of restorative sleep (Posmontier, 2008) and an important regulator of mood. Because the same neurotransmitters that mediate sleep quality also mediate mood, poor sleep quality may contribute to psychiatric symptomatology during the postpartum period (Ross, Murray & Steiner, 2005). However, several researchers have proposed that certain maternal behaviors may help moderate mood disturbances and be more conducive to improved sleep quality postpartum, including breastfeeding, co-sleeping, psychoeducational interventions (Stremler et al., 2006), and behavioral recommendations, such as initiating bedtime routines (Mindell, Telofski, Wiegand, & Kurtz, 2009).

Influence of Infant/Childcare on Sleep-Wake Patterns Infant care, especially during the first month postpartum, can lead to frequent nighttime awakenings (Lee et al., 2000), especially for novice mothers (Lee, 2006). Up to 35% of parents report difficulty settling their infants and coping with nighttime awakenings (Johnson, 1991; Ramchandani, Wiggs, Webb, & Stores, 2000), thus leading to random sleep-wake cycles when sharing in newborn care (Gay, Lee, & Lee, 2004). Physical changes, caring for other children, returning to work, and social demands may also contribute to sleep deprivation. Nighttime awakenings and, in many cases, sleep deprivation and daytime fatigue may precipitate insomnia symptoms (Bayer, Hiscock, Hampton, & Wake, 2007; Ross et al., 2005). These symptoms may also impact mood and are hypothesized to increase vulnerability to postpartum depression (Munk-Olsen, Laursen, Pederson, Mors, & Mortensen, 2006). Moline, Broch, and Zak (2004) suggested that women with a history of prior sleep disturbances are predisposed to having difficulties adjusting to the inconsistent sleep patterns resulting from newborn care.

Sleep and Breastfeeding Data on breastfeeding and sleep quality are sparse, which may be the result of the intrusiveness of objective sleep measures (e.g., actigraphy) or the burden of daily sleep logging while juggling responsibilities of caring for a newborn (Hunter, Rychnovsky, & Yount, 2009). Of the studies conducted to date, lactating women experience more SWS and fewer arousals, but no significant differences emerged for duration of REM sleep or total sleep time (Blyton, Sullivan, & Edwards, 2002). Doan, Gardiner, Gay, and Lee (2007) found that women who breastfed throughout the night averaged 40 to 45 more minutes of sleep and reported fewer sleep disturbances relative to bottle-feeding mothers. In contrast, Montgomery-Downs, Clawges, and Santy (2010) found no differences on objective, subjective, daytime sleepiness or fatigue among mothers

utilizing different feeding methods. Because of the many well-established benefits of breastfeeding, such as optimal health outcomes for the infant, the American Academy of Pediatrics (AAP) made a 2005 policy statement that reflects its position as a proponent of breastfeeding. Moreover, Montgomery-Downs and colleagues (2010) have suggested that pediatricians clarify to new parents that bottle-feeding has not been shown to improve maternal sleep, which is a common misconception.

Sleep and Bed-Sharing Families from diverse cultures have practiced bed-sharing or room-sharing between mothers and infants throughout history, but co-sleeping may be controversial as women weigh the value of breastfeeding and infant and mother bonding with the risk of Sudden Infant Death Syndrome (SIDS) (McKenna & McDade, 2005). In Western cultures, such as the United States, bed-sharing/room-sharing trends have doubled since 1993 (Willinger, Ko, Hoffman, Kessler, & Corwin, 2003), and it remains a common practice and societal norm among a majority of cultures worldwide (Owens, 2004). For example, the prevalence of children bed-sharing in China was up to 58.9% with 7-year-old children, and 73.5% of mothers in Korea agreed with bed-sharing for children ages 3 to 6 years old (Yang & Hahn, 2002).

In the United States, co-sleeping appears to be more prevalent among ethnically diverse families, families with lower SES, less parental education, and higher rates of familial stress (Lozoff, Wolf, & Davis, 1984). Proponents of co-sleeping believe that it promotes breastfeeding, maternal and infant bonding, and may reduce sleep disturbance among new mothers. However, opponents of bed-sharing argue that it may increase the risk for SIDS from smothering by heavy blankets. In a policy statement written by the American Academy of Pediatrics Task Force on Sudden Infant Death Syndrome (2005), pediatricians recommend that infants can sleep in the same room as their parents to increase bonding and breastfeeding, but should not co-sleep in the same bed before the age of 12 months.

Sleep Strategies for Expecting and New Mothers

Testing the efficacy of pharmacological and behavioral regimens, while protecting fetal and maternal safety during pregnancy, can be challenging. It is noteworthy that medications, including zolpidem and diphenhydramine, are considered Class B medications (i.e., possibly harmful to fetus), and evidence is mixed regarding their potential harmful side effects on the fetus (see Pien & Schwab, 2004, for review). Although research is limited, some obstetricians have suggested prolonging hospital stays after delivery and utilizing sedatives as strategies to promote protected sleep time and reduce sleep deprivation (Soares & Murray, 2006). Mindell (2005) recommends that new mothers nap when their babies nap and hire a babysitter or request a few hours of reprieve from a family member to obtain rest if they are feeling sleep deprived. With regard to psychotherapies, an 8-week mindfulness-based intervention was associated with significantly reduced reports of anxiety by women in their third trimester (Vieten & Astin, 2008). Additionally, in a pilot investigation, Beddoe, Lee, Weiss, Powell-Kennedy, and Yang (2010) showed that mindful yoga reduced nighttime awakenings and improved SEs among women during the second trimester compared to controls.

Dietary recommendations are the first line of prevention for RLS and include iron supplements and a diet rich in folic acid (e.g., leafy greens, folate-enriched products) to be initiated ideally before conception or at the earliest sign of pregnancy. With regard to sleep-disordered breathing, all pregnant women should be assessed for snoring and sleep apnea symptoms during prenatal checkups by asking questions about snoring—or if they "wake up gasping for air"—or experience morning headaches. Continuous positive airway pressure (CPAP) therapy for pregnant women with sleep apnea is safe (Roush & Bell, 2004) and also effective at controlling blood pressure among preeclamptic pregnant women (Edwards, Blyton, & Kiravainen, 2000).

SLEEP AND MENOPAUSE

It is important to place menopausal sleep disturbance in context. Insomnia is one of the foremost complaints of peri- and postmenopausal women (NIH, 2005; Ohayon, 2006; Owens & Matthews, 1998). However, the degree to which menopause per se causes sleep disruption is questionable. Subjective and objective indices of sleep disruption as a function of menopausal status are often discrepant. Widely held assumptions implicating hot flashes as causative in midlife sleep disruption are equivocal. Multiple factors may *coincide* with menopause (e.g., aging, distressed mood, physical compromise) and contribute to sleep disturbance, yet menopause per se may not *cause* such symptoms.

Bolge and colleagues (2010) found that sleep maintenance insomnia correlated with reduced health-related quality of life ratings, increased healthcare utilization, and decreased work productivity among *symptomatic* menopausal women. Shaver (2010) further acknowledges that distinctions between causal versus overlapping symptoms be made to best predict the healthcare utilization of those with insomnia symptoms during menopause. Menopause may be best understood through a biopsychosocial lens that captures the interplay between the changing hormonal milieu, concomitant physiological changes (e.g., hot flashes and night sweats, nocturnal micturition), coexisting medical or health conditions, psychological factors such as mood, perception, and beliefs about menopause, as well as sociocultural influences.

Reproductive Hormones and Sleep

In a large, multiethnic, community-based, prospective study (SWAN; Study of Women's Health Across the Nation) of midlife women, Kravitz and colleagues (2008) found that, controlling for age, hormone replacement therapies, and other health and related psychosocial variables, subjective sleep difficulties were pronounced during perimenopause, specifically noting associations with menopausal status, hormone levels (E2 and FSH), and vasomotor symptoms. Prior data from the Wisconsin Sleep Cohort Study, including primarily Caucasian women (Young, Rabago, Zgierska, Austin, & Finn, 2003), in which age and body mass index (BMI) were controlled, showed that peri- and postmenopausal women reported less satisfaction with their sleep, but PSG did not substantiate these complaints; likewise, Shaver, Giblin, and Paulsen (1991) indicated no marked objective changes in sleep. Pien, Sammel, Freeman, Lin, and DeBlasis (2008) found that among a stratified sample of Caucasians and African Americans, inhibin B—a reproductive hormone that falls in the early

menopausal transition—was an indicator of reported sleep disturbance; notably, sleep quality was not readily explained by menopausal status alone but in the context of other menopausal symptoms (depression, vasomotor symptoms).

Hot Flashes and Sleep Disturbance

Numerous studies of peri- and postmenopausal women have implicated hot flashes to account for menopausal sleep disturbance (Dennerstein, Dudley, Hopper, Guthrie, & Burger, 2000; Hollander et al., 2001; Kravitz et al., 2003; 2005; Owens & Matthews, 1998; Woodward & Freedman, 1994). Reports of hot flashes often coincide with reports of poor sleep quality and continuity, with approximately 44% of women who experience severe hot flashes meeting criteria for chronic insomnia (Ohayon, 2006). According to the NSF survey, approximately 20% of women attributed poor sleep to hot flashes on at least a few nights per week. Early work (Shaver, Giblin, Lentz, & Lee, 1988; Woodward & Freedman, 1994) demonstrated that hot flashes were associated with sleep alterations as measured by PSG, with a greater frequency of hot flashes seen earlier in the night (Woodward & Freedman, 1994). However, the widely held belief that hot flashes and changes in hormonal milieu *cause* disturbed sleep is not consistently substantiated by objective data (Freedman, 2005; Freedman & Roehrs, 2004; Moe, 2004; Regestein, 2006; Sharkey et al., 2003; Thurston, Blumenthal, Babyak, & Sherwood, 2006; Young et al., 2003). Methodological limitations, such as self-report and measurement error, make conclusions about hot flashes causing sleep disturbance tentative.

Likewise, PSG over a 1- to 3-night period may be insufficient to capture causative relationships. Perhaps sleep stage (REM vs. non-REM) may moderate the effect of hot flashes and awakenings (Freedman & Roehrs, 2006), and therefore more careful measurement and attention to stage may be indicated to determine to what extent hot flashes exert an adverse effect on sleep. Even asymptomatic women (who do not endorse having nocturnal flashes) objectively evidence hot flashes during the night (Freedman & Roehrs, 2004). In addition, hyperarousal and its link to thermoregulation may also be significant contributors to sleep disturbance in midlife women (see Minarik, 2009 for review). Thus, attention to cognitive and emotional arousal and perceived stress are also relevant in explaining sleep disturbance during the menopausal transition (Woods & Mitchell, 2010).

The Role of Cognitions Hunter and Mann (2010) offer a cognitive model for menopausal hot flashes and night sweats, and contend that the experience of hot flashes and night sweats are likely an interaction of physiological and psychological processes to include symptom perception (e.g., attention, negative affectivity, somatization), cognitive appraisals (beliefs and problem-rating), and behavioral responses (help-seeking). Likewise, dysfunctional beliefs and attitudes about sleep (DBAS) mediated the relationship between hot flashes and sleep quality among perimenopausal women (Kloss, Tweedy, & Gilrain, 2004). Similarly, "flashing" women may misattribute the cause of any poor sleep to hot flashes (Regestein, 2006), when it may be more accurately explained by events or behaviors other than hot flashes, such as alcohol or caffeine use. Interestingly, Krystal, Edinger, Wohlgemuth, and Marsh (1998) suggest that hot flashes serve as an initial trigger for

insomnia, but then a "behaviorally conditioned insomnia" may develop as a result of these events, and persist after the hot flashes have resolved. Given the importance of cognitive factors, Hunter and colleagues (Hunter & Liao, 1996; Hunter, Coventry, Hamed, Fentiman, & Grunfeld, 2009) describe initial support for the efficacy of cognitive and behavioral approaches toward the management of hot flashes.

Circadian Changes During Menopause

As women age, cBT and melatonin advances by about 1 hour, and consolidation and homeostatic pressure for sleep decreases (Dijk, Duffy, Czeisler, 2000). Age-related changes in melatonin depletion (Mahlberg, Tilmann, Salewski, & Kunz, 2006) may further influence the timing of the circadian rhythm in post- compared to premenopausal women, as well as the onset of sleepiness/offset of alertness (Walters, Hampton, Ferns, & Skene, 2005). Some have associated higher cBTs with poorer PSG ratings among postmenopausal women (Murphy & Campbell, 2007). Increases in cBT may also trigger hot flashes (Freedman, 2005; Freedman, Norton, Woodwar, & Cornelissen, 1995). A dysregulation in thermoregulation is hypothesized to play a role in sleep disruption among women as they age, perhaps requiring future study between thermoregulation, hot flashes, and sleep disruption (Joffee, Massler, & Sharkey, 2010).

Mood and Sleep

Depressed mood and psychological distress are significant predictors of sleep disturbance, in general, and their comorbidity may be pronounced among perimenopausal women (see Brown, Gallicchio, Flaws, & Tracy, 2009; Kravitz et al., 2003; Parry et al., 2006). Subjective ratings of sleep quality appear to be related to depressed or anxious mood, negative mood, or high perceived stress among midlife women (Dennerstein et al., 2000; Kravitz et al., 2003; Kravitz et al., 2005; Pien et al., 2008). Some suggest that depression is thought to accompany the menopausal transition (Freeman, Sammel, Lin, & Nelson, 2006; Rajewska & Rybakowski, 2003; Steiner, Dunn, & Born, 2003) and can, therefore, subsequently account for increased symptoms of insomnia. Brown et al. (2009) suggest that sleep disturbance may even mediate the relationship between menopausal symptoms and depressed mood. Interestingly, Woods and Mitchell (2010) suggest that severity of nighttime and early-morning awakenings was linked with depressed mood.

Perceptions of poor sleep may also account for psychological distress among midlife women, even when objective sleep measures show marginal differences (Shaver et al., 1991; Shaver & Paulsen, 1993). Burleson, Todd, and Travarthan (2010) conducted a prospective study on self-reported vasomotor symptoms, mood, and sleep disturbance. First, sleep problems accounted for more of the variance in mood ratings, compared to vasomotor symptoms. Second, their data only partially supported the model that hot flashes/night sweats resulted in sleep disruption, which then accounted for next-day mood disturbances. They also noted that, even while controlling for sleep problems, vasomotor symptoms continued to predict mood troubles the next day. In efforts to understand the relationships between sleep, hot flashes, and mood, Regestein (2010) purports a "domino hypothesis"—weakening in circadian timing that coincides with age may result in both hot flashes and decreased sleep quality, and,

contingent upon predisposition to negative affect, worsened depressed mood. Independent of the cause of these symptom clusters, the interplay between mood and sleep warrants attention during midlife. Furthermore, conditions such as sleep apnea, chronic pain, or other health conditions known to disrupt sleep (Ohayon, 2006), for example, nighttime voiding (Asplund & Aberg, 1996), that may become more prevalent in midlife may also account for insomnia that arises during the transition to menopause.

Cultural and Ethnic Factors

Hunter and Mann (2010) propose that culture and ethnicity may influence the experience of hot flashes/night sweats, which may have relevance for women's sleep perception. Gupta, Sturdee, and Hunter (2006) compared peri- and postmenopausal Asian women in the UK to Asian women in Delhi to Caucasian women in the UK. Although sleep problems did not differ by subgroup, *attribution* of sleep to menopause was greatest among the Caucasian women, followed by the Asian UK and Asian Delhi women, respectively. Lock (1994) also noted a striking difference in prevalence of hot flash and night sweat reporting among Japanese women compared to Western women. In Japan, night sweats were not even related to menopausal status. Moreover, reporting of sleep problems was significantly less, as was use of sleep medicines, compared to Canadian and American samples.

Likewise, in Hsu, Chen, Jou, An, and Tsa's (2009) study of Taiwanese perimenopausal women, very few attributed sleep problems to hot flashes. Thus, the menopasual symptom experience itself and/or the attribution of sleep problems to the symptom experience may differ by ethnicity or culture. While ethnic/racial differences are notable, other factors that may co-vary with sociocultural factors, such as climate, lifestyle, diet, and attitudes about menopause and aging, may also serve as key moderators of the hot flash/night sweat experience, thereby differentially influencing sleep (Gupta et al., 2006; Nagata, Takasuta, Inaba, Kawkami, & Shimizu, 1998). Furthermore, education or beliefs about menopause, such as positively embracing or pathologizing menopause, may vary, thereby ultimately affecting symptom reporting.

Based on SWAN study data, several multiracial and ethnic differences among menopause symptom reports were identified (see Avis et al., 2001; Gold et al., 2000). Caucasian women had significantly more difficulty staying asleep, and Hispanics had the fewest problems with sleep continuity and early-morning awakenings (Kravitz et al., 2008). Avis and colleagues (2001) found that African American women endorsed the greatest amount of vasomotor symptoms, with the least amount of vasomotor symptom reporting among Chinese and Japanese women. With regard to sleep-related variables, Caucasians reported sleeping worse than all other groups (Gold et al., 2000). In addition, lower education and self-reported inability to afford basic items were related to overall symptom reporting.

Of note, difficulty sleeping was also correlated to employment status. Hollander and colleagues (2001) conducted a longitudinal study of African American and White women in their late reproductive age over a 2-year period; African Americans and those with less employment and less education were at greater risk for worsened sleep complaints. In contrast, in Pien and colleagues (2008), racial identity (African American and Caucasian) was not related to sleep quality.

Clearly, menopause and ensuing sleep disturbance is not a universal experience. In addition to exploring women's beliefs and attributions about menopause, other factors such as SES, education, and employment could serve as potential stressors or context to poor sleep in women.

Sleep Strategies for Menopausal Women

Pharmacological Management of sleep disturbance involves multiple approaches and careful assessment to target the most beneficial regimen for each individual presentation. Empirically-based options include hormone therapies (HTs), hypnotics, behavioral strategies to address sleep (see following section), mood disturbance, and/or hot flashes, or other pharmacotherapies, such as antidepressants, that could help mood and/or hot flashes. Because of the fluctuating hormonal milieu and associated hot flashes presumed to disrupt sleep, HTs have traditionally been used to alleviate menopausal symptoms, including sleep disturbance. As reviewed by Joffee, Massler, and Sharkey (2010), use of HTs (e.g., estrogen replacement) for sleep improvement has been most successful for women whose sleep is associated with hot flashes, but they caution that because of small sample sizes, and at times, use of populations without significant sleep or symptom complaints, the degree to which HT exerts a meaningful effect on sleep is still questionable.

Results from the Women's Health Initiative associating the use of HT with increased risk for cancer, stroke, heart disease, and vascular dementia (Writing Group for the Women's Health Initiative Investigators, 2002) initiated a tempering of this practice, generally leading to recommendations to use HT for only a brief period to relieve hot flashes. More recently, Tranah and colleagues (2010) investigated long-term use of HT on objective sleep measures of postmenopausal women and found that HT users experienced significantly shorter wake after sleep onset (WASO). Another study suggested that HT may also be associated with less-severe OSA among postmenopausal women (Bixler & Kales, 2001). However, more studies are needed to determine whether HT should be recommended for postmenopausal women, particularly because of heightened risk of vascular side effects and limited research on the long-term impact of those side effects (Tranah et al., 2010). Further study on the mechanisms and formulations of HT (estrogens/progestins used alone or in combination) that exert their effect on sleep are needed, with attention to both objective as well as subjective measures of sleep quality and hot flashes. It seems to be the consensus that HT is indicated when the benefits are believed to outweigh the risks, especially when improving women's quality of life.

With regard to pharmacological, but nonhormonal treatments, several agents have been tried with some success. Eszopliclone and zolpidem have shown to improve sleep onset and sleep maintenance difficulties (see Joffee, Massler, & Sharkey, 2010, for review), perhaps in part by enabling women to sleep through nocturnal hot flashes. Initial support for ramelteon was found in one trial to treat insomnia among peri- and postmenopausal women with insomnia (Dobkin et al., 2009), without evidence of tolerance or withdrawal. Other alternatives include antidepressants, namely selective serotonin reuptake inhibitors (SSRIs), in alleviating menopausal symptoms to improve sleep (Stearns, Beebe, Iyengar, & Dube, 2003). Likewise, Yurchesen, Guttuso, McDermott,

Holloway, and Perlis (2009) found gabapentin to improve sleep quality among postmenopausal women with hot flashes. Similarly, Nelson and colleagues' (2006) meta-analysis showed some support for SSRIs and serotonin–norepinephrine reuptake inhibitors (SNRIs), clonidine, and gabapentin to alleviate the frequency and severity of hot flashes, but not for red clover isoflavone or soy. Although the findings for the former (SSRIs, clonidine, and gabapentin) were not judged to be as potent as estrogen, they may provide an alternative for women when HT is contraindicated.

Over-the-Counter Remedies In general, melatonin shows promise in an aging population, particularly when circadian rhythm sleep conditions, such as shift work and delayed sleep phase syndrome, are involved (Arendt & Skene, 2005; Gooneratne, 2008), yet its effect on primary insomnia needs to be better researched and established. Melatonin may even have the potential to lessen depressed mood and improve thyroid function among peri- and postmenopausal women (Bellipanni, Bianchi, Pierpaoli, Bulian, & Ilyia, 2001). The usefulness of soy, black cohosh, and/or other herbal remedies require further investigation. Newton et al. (2006) did not find strong substantiation for black cohosh to alleviate menopausal symptoms, whereas others found that black cohosh and other foods that contain phytoestrogens (Kronenberg & Fugh-Berman, 2002) show the potential to alleviate menopausal symptoms, such as hot flashes. Further investigation, particularly with regard to the long-term effects of these regimens, as well as the effects on sleep, is warranted.

Behavioral Strategies can be applied to reduce menopausal-associated symptoms, although no impact on sleep is ensured. Common recommendations for women to relieve hot flashes are based on reducing cBT and include wearing multiple layers or light layers to bed at night, keeping the ambient temperature cool, consuming cold drinks, and losing weight. Wijma, Melin, Nedstrand, and Hammar (1997) found that applied relaxation strategies may help reduce the number of hot flashes among postmenopausal women. Likewise, relaxation was also found to be helpful in reducing hot flash intensity and associated tension-anxiety and depression among a sample of midlife women with daily hot flashes (Irvin, Domar, Clark, Zuttermeister, & Friedman, 1996). Interestingly, relaxation strategies are among the empirically based treatments for insomnia (Morgenthaler et al., 2006). Investigations that study the concomitant effect on both daytime and nighttime hot flashes, anxiety, and sleep would be helpful. As discussed later, CBT-I is one of the most effective treatments for chronic insomnia (Morgenthaler et al., 2006). However, to our knowledge, the targeted use of CBT-I approaches specifically among symptomatic menopausal women has not been attempted.

SLEEP AND AGING

Nearly 50% of women over the age of 60 (without chronic health conditions) report difficulties sleeping (Foley et al., 1995; Schubert et al., 2002), and women are twice as likely as men to report sleep disturbances independent of mental health conditions (Brabbins et al., 1993). Hachul, Bittencourt, Soares, Tufik, and Baracat (2009) proposed that sleep complaints may be even *more* prevalent among postmenopausal women relative to women in early menopause. While

chronic medical conditions and/or medication side effects are often implicated in the increased sleep complaints, several factors may predispose an aging woman to sleep disorders, including, but not limited to, physiological, psychological and social factors.

Sleep Architecture and Circadian Rhythms

Changes in sleep architecture and circadian rhythmicity may account for some of the sleep changes that manifest as women age. They may be more likely to experience frequent nighttime awakenings, early-morning awakenings, and to a lesser extent, increased sleep-onset latencies. Perhaps the most salient change to sleep architecture as individuals age is the reduction of SWS (Lee & DeJoseph, 1992), although this is less pronounced in women than men. Stage 1 sleep *increases* with age for both men and women, thus making sleep more fragmented, with increases in nighttime awakenings and difficulties falling back to sleep (Carskadon, Brown, & Dement, 1982). Aging women also experience longer latencies to REM relative to men, but research suggests that REM sleep is more protected in women (as shown by increases in REM periods; Rediehs, Reis, & Creason, 1990). Circadian rhythm disruptions, particularly 1-hour phase-advances (Campbell, Christian, Kripke, Erikson, & Clopton, 1989; Dijk, Duffy, Czeisler, 2000), are also common among aging women and may also contribute to decreased TST.

Circadian rhythms and homeostatic sleep pressure work together to produce drive for sleep and are vulnerable to changes in both endogenous and exogenous factors (Dzaja et al., 2005). Endogenous factors include disruptions in neural connections of the suprachiasmatic nucleus (SCN), aging of the retina (i.e., where light enters the eye), and hormone production of the pineal gland (i.e., manufacturer of melatonin; Skene & Swaab, 2003). Several investigators have also found that changes in estrogen and gonadotropins and cBT likely contribute to phase-advanced changes seen in aging women relative to age-matched men (Moe, 1999; Murphy & Campbell, 2007). Nocturnal levels of estradiol and gonadotropin luteinizing hormone (LH) were associated with changes in objective measures of sleep, and higher LH levels were correlated with changes in the variation of sleep-related body temperature and greater sleep disruptions (Murphy & Campbell, 2007). Nocturnal melatonin secretion has also been observed in adults aged 65 to 80, who had nearly 43% lower levels of melatonin compared with individuals ages 20 to 35. Similarly, decreases in melatonin were observed among postmenopausal women (ages 48 to 60) (Kos-Kudla et al., 2002), and such decreases have been linked to complaints of sleep disturbance (Tuunainen et al., 2002). Taking naps during the day may also lead to changes in the homeostatic drive for sleep, decreasing pressure for sleep, and lack of a daytime routine after retirement may also contribute to shifts in sleep patterns (Dzaja et al., 2005).

Sleep Disorders and Their Correlates Among Aging Women

Comorbid medical illnesses and life changes (e.g., retirement, weight gain, more sedentary behaviors) may increase the vulnerability of elderly women to sleep complaints or disorders, such as insomnia, PLMS, sleep apnea, and circadian rhythm disorders (ASPD). Sleep disturbances may also be related to increased risk of cardiovascular disease (i.e., particularly from sleep apnea),

reduced immune functioning, psychopathology (i.e., depression), and a decline in cognitive functioning (Tworoger, Lee, Schernhammer, & Goldstein, 2006).

Apnea Postmenopausal women are also twice as likely (47% vs. 21%) to have sleep apnea, more likely to be obese, and to have larger neck circumferences (an anatomical feature of those with a higher risk for sleep apnea) compared to premenopausal women (Dancey et al., 2003). Even when controlling for obesity and neck circumference, postmenopausal women still reported significantly more sleep apnea relative to pre- and perimenopausal women (Dancey et al., 2003). Untreated sleep apnea is likely to result in compromised daytime functioning (e.g., excessive daytime sleepiness, cognitive and memory deficits; Décary, Rouleau, & Montplaisir, 2000), increased risk for motor vehicle accidents, heightened risk for other sleep disorders, such as insomnia (Teran-Santos, Jiminez-Gomez, & Cordero-Guevara, 1999), and cardiovascular disease (Yaggi et al., 2005). However, symptoms of sleep apnea among women are often overlooked in primary care, perhaps because snoring—a symptom suggestive of sleep apnea—is often less pronounced in women compared to men (Redline, Kump, Tishler, Browner, & Ferrette, 1994). Thus, a thorough assessment to include PSG for sleep apnea among postmenopausal women who complain of sleep disturbance and aforementioned daytime symptoms is critical (Young, Hutton, Finn, Badr, & Palta, 1996).

Insomnia Between 61% and 83% of women over the age of 60 indicate symptoms of insomnia, particularly difficulties maintaining sleep (Campos, Bittencourp, Haider, Tufik, & Baracat, 2005; Hachul et al., 2009). Webb and Campbell (1980) found that older women took nearly 4 times longer to return to sleep once awakened compared with their younger counterparts; this is corroborated with subjective ratings of increased number and length of nighttime awakenings (Morin & Grambling, 1989) and lower SEs (Baker, Simpson, & Dawson 1997). Daytime activities are hypothesized to impact insomnia prevalence during later life, with high levels of physical activity considered a protective factor against chronic insomnia (Morgan, 2003). However, older adults are more likely to lack an overall daytime routine and to spend more time napping or resting in bed during the day. These behaviors can result in reduced homeostatic pressure for sleep and sleep fragmentation at night (Bootzin, Engle-Friedman, & Hazelwood, 1983). In contrast, Morin and Grambling (1989) found that time napping appeared to be equivalent among both good and poor sleepers, suggesting that napping may not necessarily contribute to insomnia. Despite mixed evidence, daytime napping is typically not recommended to individuals if it is compromises the homeostatic drive for sleep at night.

Periodic Limb Movements During Sleep (PLMS) In addition, aging women are also at risk for PLMS. Among a community sample of elderly individuals, 25% to 58% experienced PLMS that impacted their sleep (Gehrman et al., 2002). Likewise, Claman and colleagues (2006) found that 73% of women with PLMS experienced poorer sleep quality and less restorative (i.e., SWS) sleep. PLMS are associated with sleep fragmentation, daytime sleepiness, and increased risk for insomnia. When PLMS events are greater than 15 events per hour and result in daytime sleepiness, a diagnosis of PLMD is warranted.

Advanced Sleep Phase Disorder (ASPD)

ASPD is characterized by involuntary sleep and wake times that are typically more than 3 hours earlier than what society considers to be the norm. Individuals with ASPD may also experience daytime sleepiness. Although ASPD is estimated to affect approximately 1% of middle-aged adults, no gender differences have emerged.

Treatments for Advanced Rhythms

The use of bright light therapy and melatonin has been hypothesized to shift phase-advanced circadian rhythms. Although this practice is indicated in the treatment of ASPD, the evidence is inconclusive (Sack et al., 2007). It is also of important note that melatonin is considered a homeopathic treatment and is not FDA-approved for the treatment of circadian rhythm disorders. Melatonin can typically be found at drugstores and health food stores, but it is often mixed with other additives, and it is recommended that individuals consult with their physician before taking melatonin.

Polypharmacy and Sleep Among Aging Women

Aging women are more likely to be on numerous medications to manage co-occurring conditions, such as chronic pain, cancer, diabetes, cardiovascular disease (Prinz, Vitiello, Raskind, & Thorpy, 1990), Alzheimer's disease and dementia (Brabbins et al., 1993), and even sleep disorders. Taken together, these conditions and the side effects of the medications used to treat them may impact sleep and daytime symptoms of sleepiness and fatigue (Monane, 1992). For example, some chemotherapies can produce fatigue, and some medications used to treat chronic pain can have a sedative effect. These symptoms may also contribute to sedentary lifestyles, excessive napping, or erratic sleep schedules, which can thereby affect sleep quality. The prevalence of the use of hypnotics is also heightened among the elderly, and this places elderly women (and men) at an increased risk for falls and hip fractures, as hypnotics create sedation and reduce overall vigilance.

SLEEP CHALLENGES

Shift Work

The adverse health and psychosocial consequences of shift work are quite extensive and range from daytime impairments in mood and cognitive performance to grave physical health consequences, such as increased risk for motor vehicle accidents, cardiovascular disease, and breast cancer (see Boivin, Trembly, & James, 2007; Shechter, James, & Boivin, 2008 for review). The SCN, the home of the circadian pacemaker, contains receptors for estrogen and progesterone (Kruijver & Swaab, 2002). The desynchrony between the circadian cycle and typical night/day (light and dark) rhythms places women who engage in shift work at significant risk for menstrual irregularities, reproduction difficulties, and breast cancer (see Baker & Driver, 2007; Shechter et al., 2008, for review). Examples of problems include menstrual dysregularity, dysmenorrhea, decreased alertness, and worsened mood. Shift work is also hypothesized to influence fertility, pregnancy, and fetal development (see Mahoney, 2010, for review). Shift work also appears to place women at an increased risk for breast cancer, perhaps because of the suppression of melatonin as a result of the

nighttime exposure to artificial light (Stevens et al., 1992).

Several strategies to counter the adverse effects of shift work (see Boivin et al., 2007; Shechter et al., 2008) have met with some success: strategic napping and/or the use of stimulants (i.e., caffeine or modafinil), or the monitored use of hypnotics or melatonin to help induce sleepiness to promote better sleep, and appropriately timed phototherapy, bright light at work, wearing sunglasses on the commute home, promoting bedroom darkness while sleeping during the day.

Chronic Insomnia in Women

Chronic primary insomnia (CPI) is a sleep disorder manifested by difficulty initiating or maintaining sleep, or waking too early without being able to fall back to sleep, and/or nonrestorative sleep despite allotting time to sleep, and daytime impairment (APA, 2000; American Sleep Disorders Association, 2005). Althoughsome individuals may experience transient (less than 1 week) or short-term (1 to 3 weeks) insomnia, CPI is recognized when the following criteria are met: (1) sleep onset 31 minutes or longer; (b) 3 or more nights per week; and (c) duration of 6 months or more (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). Although not all studies consistently operationally define *insomnia* as described, it is well documented that insomnia complaints and diagnoses are more common among women than men. Characterized as an "overlooked epidemic" (Soares, 2005), risk ratios for insomnia among women range from 1.28 among young adults (ages 15 to 30) to 1.78 in older adults (65 and older) (Zhang & Wing, 2006). Li, Wing, Ho, and Fong (2002) found similar patterns in Hong Kong, where women had 1.6 times greater risk than men.

Hormonal and psychosocial factors likely interact to produce these striking gender differences. For example, "empty nest" experiences, care for elderly or childcare and dealing with children's sleep disruptions, separation or divorce, and workforce demands (Meltzer & Mindell, 2007; Polo-Kantola & Erkola, 2004; Shaver, 2002) may place women at greater risk for sleep disruption compared to men. A Finnish study (Urponen, Vuori, Hasan, & Partinen, 1988) showed that worries, interpersonal difficulties, and regrets were more predominant risk factors among women, whereas work-related stress dominated as a risk factor among men. Li et al.'s (2002) study of Chinese women showed that insomnia risk heightened among women who were divorced or widowed, were exposed to nighttime noise, and had more frequent alcohol use.

Results from the NSF study (NSF, 2007) indicate noise (39%) childcare (20%) voiding (17%), pets (17%), "nothing" or no apparent reason (16%), pain (8%), spouse/bed partner (7%), nightmares (6%), and stress (5%) among the reasons for sleep disturbance (though notably unclear whether this is due to insomnia or other reasons for sleep disturbance). Psychiatric comorbidities, such as anxiety and depression, may also contribute to women's increased insomnia prevalence. Individuals with lower SES were also more likely to suffer from insomnia while controlling for gender, age, and ethnicity (Gellis et al., 2005). The incidence of other medical conditions, such as chronic pain, nocturnal micturition, or RLS, particularly as women (and men) age, may likely produce insomnia complaints. To illustrate, a vicious cycle between fibromyalgia, which is more common in women than men, and insomnia can

ensue with decreased sleep worsening pain reports among fibromyalgia patients (Lentz, Landis, Rothermel, & Shaver, 1999).

In addition to reduced quality of life, CPI is associated with significant daytime impairment (difficulty concentrating, worsened mood, decreased cognitive functioning, general malaise; Kloss, 2003). Moreover, elderly women (and men) with insomnia have significantly increased healthcare utilization (Sarsour, Kalsekar, Swindle, Foley, & Walsh, 2011). Both women and men with untreated CPI had a greater risk of an onset of major depression, anxiety disorder, and alcohol use disorder (Ford & Kamerow, 1989); women with CPI are more likely to develop depression (Mallon, Broman, & Hetta, 2000). Moderate to severe insomnia was associated with decreased productivity and increased healthcare utilization, compared to individuals without insomnia complaints (Sarsour et al., 2011).

Pharmacologic Treatment of Chronic Insomnia in Women Several both pharmacologic and cognitive-behavioral treatments are available for treating CPI. Benzodiazepines provide both sedative and soporific effects, but they carry risks of tolerance, dependence, changes in sleep architecture, insomnia rebound after stopping use, and cognitive impairment the following day (see Davidson, 2008, for review). Nonbenzodiazepines (zolpidem and zaleplon) are considered less "risky" than benzodiazepines, as they do not produce similar addictive potential, and they alleviate sleep-onset and maintenance insomnia symptoms. Of note, benzodiazepines are contraindicated during pregnancy and nursing. Moreover, use of any hypnotic is cautioned in elderly women (and men) because of cognitive and motor impairments (e.g., dizziness and risk of falls). Antidepressants may exert their effects on sleep by relieving other symptoms associated with menopause (vasomotor symptoms, pain, mood swings) and may provide a viable alternative to HRT (Soares & Murray, 2006). However, research on the hypnotic effects of antidepressants is limited among nondepressed individuals (Davidson, 2008).

According to the meta-analyses conducted by Brzezinski et al. (2005) and other reviews (Pandi-Perumal et al., 2005, 2007), melatonin shows promise in improving sleep quality and reducing insomnia symptoms, particularly where melatonin production may be decreased, and perhaps among individuals with comorbid insomnia and medical/psychiatric complaints. With carefully timed administration, melatonin is likely to improve sleep quality, particularly where circadian disruption occurs, such as shift work or jet lag (Arendt & Skene, 2005; Burgess, Sharkey, & Eastman, 2002). However, Gooneratne (2008) found the efficacy on melatonin for elderly populations for primary insomnia is less definitive. Ramelteon, a melatonin analogue with FDA approval for insomnia, has gained support in alleviating insomnia symptoms, with potential side effects judged to be minimal (headache, dizziness, fatigue at higher doses, stomach upset, sleepiness; Davidson, 2008, Pandi-Perumal et al., 2007). Fortunately, the use of melatonin is generally well tolerated, and the adverse effects of its short-term use are judged to be minimal, but research on its safety and long-term use is warranted (Arendt & Skene; 2005; Gooneratne, 2008; Pandi-Perumal, 2007). Because of unknown safety risk in pregnancy and potential influence on reproductive function, melatonin should only be used if the benefits are thought to outweigh the risks; also, its influence on cardiovascular and immune effects requires further study (Gooneratne, 2008).

Herbal therapies, such as lavender, valerian, chamomile, black cohosh, and kava kava, have gained popularity among women to alleviate sleep troubles, although the limited data available are inconclusive on herbal remedies for insomnia (Davidson, 2008; Soares & Murray, 2008). Although women are likely to experiment and tolerate herbal products and report beneficial effects, caution is heeded in the absence of well-constructed methodology and potential or unknown risks of some agents. Gooneratne (2008) reviewed the efficacy of valerian and showed that using valerian for at least 2 weeks yielded subjective sleep improvement, yet objective records were inconclusive. He also recommends larger sample sizes to obtain sufficient power, standardized compounds of the agent, and methodological rigor in conducting studies on herbal therapies.

Cognitive-Behavioral Therapy for Insomnia Empirically based, cognitive behavioral therapy for insomnia (CBT-I) is considered the treatment of choice for CPI (Morgenthaler et al., 2006; Morin, 2006; Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995). Treatment is comprised of regimens such as stimulus control, sleep restriction, relaxation, cognitive therapy (see Perlis, Aloia, & Kuhn's [2011] comprehensive handbook). In contrast to hypnotics, which generally work immediately in inducing their soporific effects, CBT-I requires patient education and training, and may take several sessions to derive benefit, yet the long-term efficacy far outweighs that of pharmacological approaches. It is noteworthy that sleep restriction is contra-indicated for patients with bipolar disorder or seizure disorders, and patients need to be cautioned about the side effects of sleep deprivation, such as drowsy driving or operating heavy machinery. For individuals with comorbid insomnia, research is encouraging and suggests that CBT-I is efficacious even in the presence of a comorbid medical or psychiatric diagnosis. CBT-I has shown promising efficacy for comorbid insomnia, with chronic pain, cancer, and other psychiatric comorbidities (Smith, Huang, & Manber, 2005). Although we would expect that CBT-I would also yield encouraging results in women who are experiencing menstrual-related or menopausal-related insomnia, CBT-I studies have yet to be conducted targeting women with these symptoms.

Obstructive Sleep Apnea

Manifestation of OSA in Women According to the *International Classification of Sleep Disorders* (ICSD-2), OSA is a result of the cessation of airflow while sleeping marked by apnea and hypopnea events on PSG, often accompanied by snoring, daytime sleepiness, morning headaches, and/or nocturia. Individuals with OSA often feel like they have "run a marathon" after sleeping, attesting to complaints of daytime fatigue and sleepiness. Patients may also report that they sleep better sitting up and experience the most difficulty lying supine, which places pressure on their airway. OSA may increase the risk for insomnia and heighten the chance of hypertension, cardiac arrhythmias, heart attack, stroke (Shepard, 1992), cognitive deficits, or pregnancy complications.

Commonly believed to affect mostly men, OSA is often overlooked in women (Lee & Kryger, 2008), which can have potentially harmful consequences. Although men may have higher reported incidence of sleep apnea relative to women (4% vs. 2%), some authors have suggested that women

may clinically present symptoms for OSA differently than men (e.g., Shepertycky, Banno, & Kryger, 2004). Alarmingly, Young, Evans, Finn, and Palta (1997) found that as many as 90% of women with moderate to severe apnea were undiagnosed and that women with sleep apnea may also have an increased 5-year mortality rate. According to Kapsimalis and Kryger's (2002) review, the reduction of female hormones during menopause may be a significant risk factor for the manifestation of OSA in older women. They also concluded that females with OSA were more likely to be diagnosed with depression and COPD, and PSG findings show that women were more likely to experience more hypopnea events rather than apnea events than men, thus making their apnea appear less severe. In addition to postmenopausal status, risk factors for sleep-disordered breathing also include elevated BMI (Godfrey, 2009), the FP (vs. the LP) (Driver et al., 2005), pregnancy, especially during the third trimester (Pien et al., 2005), and polycystic ovarian syndrome (Gopal, Duntley, Uhles, & Attarain, 2002). Older women with OSA show reduced cognitive functioning, including short-term memory difficulties, deficiencies in verbal recall tests, and decreased alertness, that have not been replicated in age-matched men with OSA (Décary et al., 2000).

OSA Treatments If an individual is experiencing mild OSA, the first line of treatment is typically weight loss and sleeping on one's side rather than supine or on the stomach. Weight loss of as little as 5 to 10 pounds has been shown to reduce OSA symptoms. The most efficacious treatment for obstructive sleep apnea is the use of a continuous positive airway pressure (CPAP) machine (El-Solh, Ayyar, Akinnusi, Relia, & Akinnusi, 2010). This machine works by placing air pressure on the airway to open the airway and make it easier for patients with OSA to breathe with less obstruction and resistance. However, certain individuals may experience difficulty adapting to their CPAP machines. Among these individuals are veterans with posttraumatic stress disorder (PTSD), who may be at a higher risk for sleep-disordered breathing in comparison with nonveterans (Maher, Rego, & Asnis, 2006). El-Solh and colleagues (2010) found that frequent nightmares were a strong predictor of nonadherence of CPAP use among veterans. To our knowledge, this has not specifically been examined among women, but with the growing number of female veterans, there is an impetus to give attention to this issue.

Several types of CPAP masks are available that may reduce their invasiveness. Psychotherapists can also conduct desensitization procedures to increase adherence. Overall, CPAP machines are only effective if they are worn consistently, every time an individual is going to sleep, even while napping. Some studies have also suggested that hormone replacement therapy (HRT) may be a protective factor for OSA in postmenopausal women (e.g., Bixler & Kales, 2001), as estrogen serves as a salient hormone in the regulation of fat distribution and progesterone helps stimulate respiratory function (Avidan, 2005). However, HRT may carry its own potential risks and is not suggested as a first line of treatment for OSA.

FUTURE DIRECTIONS

Advancing the knowledge of women's sleep in the context of hormonal changes, developmental periods, and sociocultural milieu

has yielded intriguing questions and findings, but not without methodological challenges. Investigations that use longitudinal methods, actigraphy or PSG, and other objective indices (e.g., hormones, circadian), coupled with psychological measures, could better qualify what underlies and maintains women's sleep disruption. Both the examination of the independent contribution of circadian, hormonal, and psychosocial factors *and* their interaction will help advance our understanding. For example, how do circadian, hormonal, and lifestyle factors affect cancer risk or reproductive capacity among shift workers? What is the role of cognition, attitudes, and beliefs about sleep during pregnancy or menopause, and how might ways of cognitive or behavioral coping during these periods enable better sleep?

Likewise, sleep problems comorbid with medical and psychiatric conditions are more often the norm, yet they are often understudied. Identification of moderating (and protective) factors is also increasingly recognized. For example, cognitive, emotional, and behavioral factors are acknowledged in the extant literature on insomnia, yet specific perceptions, attributions, and expectancies among women during their menstrual cycle, menopause, and postpartum have yet to be identified. For example, to what degree does sleep mediate mood during the postpartum period?

Critical moderators, such as SES, employment status, and racial and ethnic identity, have been relatively unexplored, yet these variables may differentially predict sleep (Mezick et al., 2008). Questions should be asked, such as: What are the protective factors in postpartum sleep? How does one's culture or beliefs about the premenstrual syndrome or menopause influence sleep-related complaints? How does the stress of un- or underemployment, juggling work and family, or being a new mom affect sleep? Moderating variables may also account for treatment receptivity and accessibility. Who has access to behavioral sleep medicine and how do we disseminate BSM to more diverse populations?

While a strong literature base for empirically validated treatments for behavioral sleep medicine (e.g., CBT-I) exists among general adult samples, these strategies still need to be applied and tested in the context of the menstrual cycle, pregnancy, or menopause. Furthermore, translational research is needed to determine efficient ways to disseminate sleep knowledge across diverse populations of women. For example, how can we help underserved women obtain proper sleep knowledge, empirically based strategies, and access sleep specialists? How could telehealth (Ritterband et al., 2009) foster the dissemination of BSM? Likewise, primary care or obstetrics and gynecology can serve as front-line resources for initial screening of sleep problems and providing referrals, yet future research and models about incorporating sleep education into these settings would be needed. Future endeavors may also look at the utility of third-wave therapies, such as acceptance and commitment therapy and mindfulness training (Ong & Sholtes, 2010).

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