Cognitive behavioral therapy for menopausal insomnia in perimenopausal and postmenopausal women with insomnia and nocturnal hot flashes: a randomized-controlled pilot trial

Emily J. Arentson-Lantz, PhD,[1,2] Alexandria Muench, PsyD,[3] Manasa Kokonda, MS,[4,5] Jessica M. Meers, PhD,[4,5] Maria Swartz, PhD,[1] Rachel Manber, PhD,[6] Rebecca C. Thurston, PhD,[7] and Sara Nowakowski, PhD[4,5]

Abstract

Objectives: The objective of this randomized-controlled pilot trial was to develop and test a cognitive behavioral therapy intervention for both insomnia and vasomotor symptoms (VMS) in perimenopausal and postmenopausal women with insomnia disorder experiencing nocturnal vasomotor symptoms.

Methods: Forty-three participants (mean age = 53.6 y) self-described as perimenopausal or postmenopausal who reported at minimum ≥ 1 nocturnal hot flash/night and met diagnostic criteria for insomnia disorder were randomized to cognitive behavioral therapy for menopausal insomnia (CBT-MI) or menopause education control (MEC). The CBT-I intervention was adapted for the study population by incorporating elements of CBT for menopausal symptoms. Primary outcomes measured at baseline, post-treatment measures, 1-month follow-up, and 3-month follow-up included: Insomnia Severity Index (ISI), Sleep Self-Efficacy Scale (SES), and Hot Flash Daily Interference Scale (HFDIS).

Results: CBT-MI compared with MEC significantly reduced ISI (CBT-MI vs. MEC: −10.2 ± 1.15 vs. −6.2 ± 0.99; $P = 0.007$), HFDIS (CBT-MI vs. MEC: −1.3 ± 0.29 vs. −0.5 ± 0.17; $P = 0.01$), and increased SES (10.2 ± 1.46 vs. 5.9 ± 1.24, $P = 0.03$) post-treatment and 1-month after the end of treatment (ISI: CBT-MI vs. MEC, −10.9 ± 1.19 vs. −6.5 ± 0.98; $P = 0.003$, HFRDIS: CBT-MI vs. MEC, −1.1 ± 0.28 vs. −0.4 ± 0.18, $P = 0.047$, SES: CBT-MI vs. MEC, 11.9 ± 2.06 vs. 5.6 ± 1.25; $P = 0.003$). Analysis of the ISI factors showed that CBT-MI significantly decreased night-time sleep symptoms and patient perception of insomnia at post-treatment and 1-month follow-up compared with MEC. Night-time sleep symptoms remained improved at 3-month follow-up compared with MEC.

Conclusions: In this pilot study, a CBT intervention targeting both insomnia and VMS showed promising improvements in sleep and hot flash interference in midlife women.

Key Words: Cognitive behavioral therapy (CBT), Insomnia, Menopause, Randomized clinical trial, Sleep, Vasomotor symptoms.

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Insomnia is defined as disturbed sleep associated with distress or impairment and is one of the most common complaint of perimenopause and postmenopause. As many as 20%-60% of perimenopausal and post-

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From the [1]Department of Nutrition Sciences and Health Behavior, University of Texas Medical Branch, Galveston, TX; [2]Center for Health Promotion, Performance, and Rehabilitation Research, University of Texas Medical Branch, Galveston, TX; [3]Department of Psychiatry, Penn Medicine Princeton Health, Plainsboro, NJ; [4]Department of Medicine, Baylor College of Medicine, Houston, TX; [5]Center for Innovation in Quality, Effectiveness, and Safety, Michael DeBakey VA Medical Center, Houston, TX; [6]Stanford University, Palo Alto, CA; and [7]University of Pittsburgh, Pittsburgh, PA.
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Address correspondence to: Sara Nowakowski, PhD, Department of Medicine, Baylor College of Medicine, 2450 Holcombe Blvd, Ste 01Y, Houston, TX 77021. E-mail: Sara.Nowakowski@bcm.edu.
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menopausal women in the United States have insomnia symptoms.[1,2] The health and functional consequences of insomnia include reduced quality of life, increased health care utilization and costs,[3] disability,[3] and incidence of depression and cardiovascular disease (CVD).[4,5] Closely inter-related psychological, social, and cultural factors shape a woman's experience of the menopause transition and contribute to sleep disturbances associated with the menopause transition.[6]

Vasomotor symptoms (VMS) occur in 60%-80% of women during the menopause transition[7] and persist for 4-5 years on average.[8,9] VMS that occur during the night are associated with awakenings from sleep.[10] Midlife women with vasomotor symptoms often report diminished sleep quality and are more likely to meet criteria for insomnia disorder.[11,12] Although it is important to note that not every awakening is associated with nocturnal VMS (nVMS). Women with nVMS may also experience nocturnal awakenings that are unrelated to a vasomotor event. Indeed, insomnia can occur during menopause independent of nVMS. Although self-reported nVMS correlate with subjective poor sleep quality, such association is less clear when objective sleep measures are used.[11-13] Regardless of etiology, once women are awake and alert, they may experience ruminating thoughts that prevent them from quickly retuning to sleep. Consequently, women may engage in compensatory behaviors, such as napping or extending time in bed, that can perpetuate insomnia.

Cognitive behavioral therapy for insomnia (CBT-I) is an effective treatment for insomnia that is based on structured, short-term, skill-focused psychotherapy aimed at changing maladaptive cognitions (ie, thoughts and beliefs) and behaviors contributing to insomnia. The weight of evidence supporting CBT-I, summarized in several meta-analyses,[14-16] led to its recognition as a first-line treatment for insomnia by the American College of Physicians[17] and the World Sleep Society.[18] Improvements after CBT-I are equivalent to those achieved during acute treatment with hypnotic medications[15,19-21] and its effects are more durable after treatment discontinuation.[22-24]

Insomnia in women during the menopause transition and beyond is complex and multifactorial, related to factors such as aging, hormone fluctuation, VMS, other sleep disorders, psychiatric and medical conditions, and psychosocial stressors.[6] As midlife women with acute and sustained insomnia experience greater negative health consequences, it is imperative to provide efficacious treatments for menopause-related insomnia. Although there is empirical support demonstrating CBT as an effective strategy for coping with VMS and other menopausal symptoms,[25-31] few trials of CBT have addressed both insomnia and nVMS. Therefore, the primary aim of this study was to demonstrate preliminary efficacy of a CBT intervention for both insomnia and menopausal symptoms in perimenopausal and postmenopausal women with insomnia disorder experiencing nVMS, titled cognitive behavioral therapy for menopausal insomnia (CBT-MI).

METHODS

This randomized pilot trial was conducted and reported in accordance with the CONSORT 2010 statement: extension to randomized pilot and feasibility trials.

Study design

This was a randomized-controlled pilot trial to assess the efficacy of a CBT-I intervention adapted for menopausal symptoms to reduce insomnia symptoms in perimenopausal and postmenopausal women with insomnia disorder and experiencing nVMS (> 1 per night). Enrolled participants were randomized 1:1 to either receive CBT-MI or a menopause education control (MEC) to supplement usual care. The CBT-MI intervention was delivered in four individual 50-minute sessions over 8 weeks by trained social workers, nurses, or psychologists in gynecology clinics. The MEC intervention was delivered in one individual 50-minute session by trained psychologists in gynecology clinics. Different therapists were used for the CBT-MI and MEC interventions, to ensure the integrity of each intervention and replicate methods used in prior trials.[32,33] Assessments of sleep, VMS, and mood were collected at baseline, within 7 days of completion of treatment (post-treatment) and 1 month after completion of treatment and 3 months after completion of treatment, unless otherwise indicated in the below text. The study was powered to detect differences in insomnia severity index (ISI) score at the post-treatment timepoint. One-month and 3-month follow-up timepoints were added to explore treatment durability. Although double-blind procedures were not possible due to the nature of the behavioral interventions, participants were not informed of which treatment was considered the active treatment or of the specific hypotheses and staff delivering the assessments were masked to the study intervention group.

Intervention

CBT-MI is a manualized treatment that combined CBTI with cognitive behavioral therapy for hot flashes (CBTH).[34] Before this pilot trial, a survey study and qualitative interviews were conducted of gynecology clinic patients aged 45-65 years old and providers. Midlife women reported VMS and sleep disturbance as top complaints during menopause. Providers reported lack of knowledge to treat or refer patients as barriers to inquiring about sleep complaints, which is in line with other published studies.[35-38] CBT-I consists of education about sleep; sleep restriction,[39] which aims to first improve sleep quality at the expense of total amount of sleep, that is, initially restricting time in bed, then expanding it slowly as long as a threshold sleep efficiency is preserved; stimulus control,[40] which aims to promote unlearning of conditioned insomnia by establishing the bed and bedroom as a cue for sleep by eliminating non-sleep activities from the bedroom; strategies for reducing hyperarousal (cognitive and somatic); cognitive restructuring of sleep interfering thoughts,[41] aimed at reducing cognitive arousal and removing obstacles to adherence with the behavioral strategies; and relapse prevention.

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TABLE 1. Description of content from each cognitive behavioral therapy adapted for insomnia in menopause (CBT-MI) session

Session	Content
1	**Sleep, menopause, and hot flash education** -Education about the normal sleep process, menopausal symptoms, the impact of the menopausal transition on sleep, the regulation of sleep, and conditioned arousal with a focus on sleep effort. -Supplemental handouts were provided to enhance education covered in session.
2	**Sleep restriction & stimulus control** -Introduced sleep restriction and stimulus control, which are adjusted in subsequent sessions.
3	**Sleep & menopausal cognitions and lifestyle factors** -Identifying and altering response to possible hot flash triggers thereby increasing the sense of control over reaction to hot flashes, identifying and restructuring negative beliefs about sleep and menopausal symptoms. -Participants were also encouraged to eliminate behaviors which might exacerbate sleep disturbance and/or hot flashes (eg, watching the clock, warm rooms, rushing, smoking, excessive alcohol and caffeine consumption).
4	**Relaxation techniques, acceptance, and maintenance plan** -Provided training on paced respiration (ie, slow, abdominal breathing) and exercises to promote calm observance of the hot flash and insomnia experience, without trying to escape; designed to change how participant related to her experiences of hot flashes and insomnia. -Additional training on adopting an objective non-judgmental awareness of hot flashes and insomnia from beginning to end to reduce catastrophic thoughts and improve self-image as well as overall sense of well-being. -Relapse prevention was also discussed.

Although CBT-I has established efficacy for treating insomnia complaints in midlife women, women have expressed interest in a more comprehensive intervention to improve whole health. CBTH consists of education about menopause, guided relaxation, and cognitive restructuring of negative beliefs about symptoms of menopause (including VMS). In adapting CBT-I, the investigative team interviewed midlife women to use a co-adaption approach, consulted with discipline experts (gynecologists, menopause research investigators), and reviewed empirically supported CBT intervention protocols and treatment manuals for menopausal symptoms to guide adaptation.[42,43] See Table 1 for more complete session descriptions.

During the MEC session participants were provided written educational materials from The Menopause Society (previously The North American Menopause Society),[44] National Institute on Aging,[45] and the National Heart Lung and Blood Institute[46] on menopause and sleep health that were discussed with participants. The MEC session was introduced as a self-guided intervention for menopausal insomnia. It was designed to be educational, supportive, and non-directive. Sleep hygiene instructions were provided to MEC participants. However, active CBT-I components (ie, sleep restriction and stimulus control therapy) were not discussed in the MEC session. Sleep was tracked using prospective sleep diaries in both conditions; however, feedback and recommendations from sleep diaries were not provided to MEC participants. The control intervention was designed to enhance usual care and did not control for therapist time or attention.

Treatment Fidelity and Refinement. Therapists (Bachelor's level nurses and social workers) were trained by the study principal investigator (PI; Nowakowski) using didactics, role-plays, and continuous monitoring. They were assessed for competency using the CBT-I competency rating scale and received weekly supervision by the study PI. To monitor the integrity of the treatment provided, therapy sessions were recorded and 20% were randomly selected to rate adherence to the treatment manual using a pre-determined checklist of topics that were covered during the session. All study therapists were rated as "good" or "excellent" competency and no therapist required retraining.

Participants

Adult females were recruited using flyers placed in primary-care gynecology clinics associated with the University Texas Medical Branch (UTMB) in greater Galveston, TX. All participants provided written informed consent and received monetary compensation for their time and effort. Eligible participants were perimenopausal (self-reported as variable cycle length 7 d different from their normal cycle or >2 skipped cycles and an interval of amenorrhea of 2-12 mo) or postmenopausal (self-reported >12 mo since last menstrual period), reported a minimum of one nVMS episode each night over a 7-day interval before the baseline assessment. Participants were also diagnosed with insomnia disorder using semi-structured interview of DSM-5 criteria. Participants were screened using the Insomnia Score Index (ISI) and Pittsburgh Sleep Quality Index (PSQI) to ensure that women presenting with clinically significant, moderate-to-severe sleep disturbance—whether characterized by insomnia severity (score >14 on the ISI)[47] or global poor sleep quality (score of >8 on the PSQI).[48]

Exclusion criteria included surgical or pharmacologically induced menopause (eg, hysterectomy or oophorectomy or chemically induced menopause), sleep-wake disorders other than insomnia, receiving hormone therapy, and taking medications/supplements known to impact sleep (eg, zolpidem, melatonin, etc.) or hot flashes (eg, black cohosh, or low-dose selective serotonin reuptake inhibitors such as Brisdelle or Effexor). Polysomnography (PSG) with sternal skin conductance monitoring was conducted to screen for sleep apnea. Participants were excluded if they exhibited > 5 apnea hypopnea index (AHI) on overnight PSG screening as part of the study. The study protocol was conducted in accordance

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with the Declaration of Helsinki and approved by the UTMB's Institutional Review Board. The CONSORT diagram is presented in Supplementary Figure 1, Supplemental Digital Content 1, http://links.lww.com/MENO/B503. This study was registered with ClinicalTrials.gov as NCT02092844.

Outcome measures

Indicators of insomnia severity, sleep quality, and sleep self-efficacy

The ISI is a seven-item validated self-report scale that assesses subjective symptoms of insomnia.[40] The instrument consists of seven items assessing difficulty falling asleep, difficulty staying asleep, problems with early awakening, satisfaction with current sleep pattern, interference of sleep problem with daily functioning, noticeability of impairment attributed to the sleep problem, and degree of distress caused by the sleep problem. Items are scored on a 0-4 scale to yield a score of 0-28. Higher scores indicate greater insomnia severity. A score below 7 indicates the absence of insomnia, a score of 8-14 indicates mild insomnia, a score 15-21 indicates moderate severity, clinical insomnia, and a score of 22-28 indicates severe clinical insomnia. We used three-factor analysis based on work by Ji et al[49] to look at changes in different dimensions of insomnia symptoms that are included in the ISI questionnaire. Sleep Symptoms (factor 1) considers disturbances in night-time sleep related to insomnia, daytime symptoms (factor 2) focuses on how insomnia disturbs daytime activities and Perception of Symptoms (factor 3) captures participant's perception of the severity of insomnia symptoms. Sleep symptoms are calculated by summing the scores for ISI Items 1, 2, and 3, daytime symptoms are calculated by summing the scores of ISI Items 5 and 6 and Perception of Symptoms is calculated by summing items 4 and 7.

The PSQI is a 19-item validated survey instrument designed to address general sleep quality and sleep disturbances over a 1-month time period.[48] Domains included in the instrument include self-reported sleep quality, sleep-onset latency, duration, and efficiency; sleep disturbances; use of sleeping medication; and daytime dysfunction. Seven component scores are summed to yield one global score (0-21), with a cutoff of five indicative of poor sleep.

The Sleep Self-Efficacy Scale (SES) is a nine-item instrument that uses a five-point Likert scale to assesses participant's perception of their ability to engage in productive sleep behaviors.[50] Lower scores indicate lower self-perceptions of ability to achieve good sleep and have been associated with the incidence of insomnia.[51]

Indicators of vasomotor symptoms

The hot flash daily interference scale (HFDIS) is designed to measure the impact of hot flashes on overall quality of life and nine specific activities (work, social activities, leisure activities, sleep, mood, concentration, relations with others, sexuality, enjoyment of life).[52,53] Higher scores indicate higher interference due to hot flashes and thus, greater impact on quality of life.

The Menopause Quality of Life Scale (MENQOL)[54,55] is a 29-item measure that assesses quality of life related to vasomotor symptoms, physical, psychosocial, and sexual domains. Items pertaining to a specific symptom are rated as present or not present, and if present, how bothersome on a zero (not bothersome) to six (extremely bothersome) scale. A lower score indicates that quality of life was not as impacted by menopausal symptoms. This assessment was only completed at baseline and the 3-month follow-up timepoints.

Indicators of mood

The Center for Epidemiological Studies Depression Scale (CES-D) assesses recent (within 1 wk) depressive symptoms using a 20-item Likert-scale form.[56] The score ranges from 0 to 60, with higher scores indicative a greater presence of depressive symptoms.

The State-Trait Anxiety Inventory (STAI) is a 40-item instrument used to measure of trait and state anxiety.[57] Items are rated on a four-point scale from "Almost Never" to "Almost Always". Higher scores indicate greater anxiety. This assessment was only completed at baseline and the 3-month follow-up timepoints.

Statistical analysis

Primary outcome variables were defined as ISI and SES for sleep and HFRDIS for hot flash. We used CES-D, MENQOL, and STAI as secondary measures. Demographics (age, education, race/ethnicity), insomnia history, and menstrual status were obtained from the patient's electronic medical record and verified with participants. Associations between potential covariates and primary study outcomes (sleep and hot flash) were evaluated using Pearson and Spearman correlations. Covariates were selected based on the potential clinical significance and associations with the outcomes were evaluated for inclusion in the regression with a $P < 0.05$ threshold.

Descriptive statistics (mean, SD for continuous variables and frequency and proportion for categorical variables) are computed for the demographic, clinical, and baseline variables by treatment groups. Baseline differences were compared between treatment groups using t test for continuous and χ^2 test for categorical variables. No significant baseline differences were found between treatment groups for age, education, race, ethnicity, insomnia history, menstrual status, or years past menopause. Thus, we did not include these variables in subsequent adjusted models. Missing data were examined for pattern using SAS's PROC MI and found that the data are missing at random. Repeatedly measured continuous outcomes are longitudinally modeled using a generalized mixed-effects model. Participants who had baseline and post-treatment or follow-up data were included in the model analysis. A mixed-effects linear model included treatment, time, and treatment by time interaction as fixed effects, and two random effects (random intercept and random slope).

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Before model analysis, outcome variables were assessed for normality and heterogeneity to check the validity of the distribution assumptions. Sleep variables and MENQOL outcomes followed residual normality and met the distribution assumptions. Depression (CES-D), hot flash (HFRDIS), and anxiety (STAI) outcomes were right skewed with over-dispersion (mean of outcome is much lower than variance), hence negative binomial distribution was used in model analysis for these outcomes. Treatment effect sizes (difference in mean outcome between groups, divided by the pooled SD) were computed for the sleep, VMS, and depression outcomes. Intent-to-treat analysis was conducted using last observation carried forward. All tests were two-sided with significance level of 0.05 and *p*-values are adjusted by Bonferroni procedure when conducting multiple comparisons. All analyses were performed using SAS software version 9.4 (SAS Institute, Inc., NC, USA).

This study had a power of $>97\%$ to detect a large effect with the total sample size of 43, for the primary outcomes ISI (d = 0.83), SES (d = 0.71), and HFRDIS (d = 0.8) at post-treatment with a significance level of 0.05, correlation among measures as 0.6, calculated using repeated measures, within-between interaction of F-test family in G-power software version 3.1.

TABLE 2. Participant demographics

Demographic variables	MEC (n = 25)	CBT-MI (n = 18)	P^a
Baseline age, mean (SD)	54.24 (6.75)	52.67 (5.17)	0.41
Age at menopause, mean (SD)	45.09 (9.61	47.11 (4.31)	0.39
Years past menopause, mean (SD)	9.14 (10.69)	5.56 (4.84)	0.31
Race, n (%)			0.96
White	19 (65.52)	11 (64.71)	
Black or African American	6 (20.69)	4 (23.53)	
Asian	1 (3.45)	1 (5.88)	
Native Hawaiian or Other Pacific Islander	0	0	
American Indian/Alaska native	0	0	
Other[b]	4 (13.79)	2 (9.88)	
Ethnicity, n (%)			0.37
Hispanic/Latino	3 (11.86)	6 (33.41)	
Not Hispanic/Latino	22 (88.14)	12 (66.59)	
Education, n (%)			0.17
Bachelor's degree	8 (31.48)	5 (27.77)	
High school diploma	13 (51.52)	3 (16.66)	
Master's degree	2 (8.5)	4 (22.22)	
Other	2 (8.5)	6 (33.33)	
Employment, n (%)			0.67
Working full-time	22 (87.28)	12 (66.67)	
Working part-time	2 (8.51)	2 (11.12)	
Other	1 (4.21)	4 (22.21)	
Menstrual status, n (%)			0.94
Perimenopause	6 (24.0)	6 (33.33)	
Postmenopause	19 (76.0)	12 (66.67)	

CBT-MI, cognitive behavior therapy for insomnia adapted for menopause; MEC, Menopause Education Condition.
[a]*p*-value is calculated using *t* test for continuous variables and χ^2 test for categorical variables.
[b]"Other" was the wording used in the original participant demographic survey for participants to select if they felt their race did not match the provided categories. We recognize this terminology does not well describe the diversity of our study sample.

RESULTS

Sample characteristics

There were 43 participants (mean age = 53.6 y) assigned to the two groups: 25 participants were randomized to receive MEC and 18 to CBT-MI. The groups did not differ significantly by age, race, ethnicity, education, employment, and menstrual status (Table 2).

Adherence, treatment discontinuation, and completeness of outcomes ascertainment

Participants in CBT-MI attended an average of 3.5 ± 0.8 sessions. MEC condition consisted of one 50-60-minute session. There were no between-group differences in the number of dropouts or reasons for treatment discontinuation (Supplementary Figure 1, Supplemental Digital Content 1, http://links.lww.com/MENO/B503) with the overall attrition rate of the study at 21%.

Indicators of insomnia severity and sleep self-efficacy

There was a significant between-group difference in the change of ISI score from baseline at the post-treatment ($P = 0.007$) and 1-month follow-up ($P = 0.003$) timepoints (Table 3). At baseline total ISI score was not significantly different between the women in the CBT-MI group and MEC groups (15.1 ± 3.5 vs. 15.0 ± 4.4; $P = 0.34$, Fig. 1A); 61% of women in the CBT-MI group and 48% in MEC group had ISI scores in the moderate (ISI = 15-21) to severe (ISI = 22-28) insomnia range (Fig. 1B, C). After the intervention period, ISI scores were significantly lower in the CBTI-MI group than the MEC group at the post-treatment (CBT-MI vs. MEC: 4.9 ± 1.1 vs. 8.8 ± 1.1; $P = 0.01$) and 1-month follow-up (CBT-MI vs. MEC: 4.1 ± 1.2 vs. 8.5 ± 1.1; $P = 0.01$), but not at the 3-month follow-up (CBT-MI vs. MEC: 5.1 ± 1.2 vs. 7.5 ± 1.1; $P = 0.08$) (Fig. 1A). Standardized mean differences between the groups (ie, effect sizes) for ISI at post-treatment and 1-month follow-up were 0.83 and 0.91 SD units, respectively, indicating large treatment effects for CBT-MI on insomnia severity. At the post-treatment timepoint, 69% of women in the CBT-MI had ISI scores in a "no clinically significant insomnia" range (ISI: 0-7) compared with only 30% of women randomized to the MEC group (Fig. 1C).

The ISI was also assessed for three factors including factor 1—ISI Sleep Symptoms, factor 2—ISI daytime symptoms related to insomnia, and factor 3—ISI perception of symptoms related to insomnia. The change from baseline of ISI sleep symptoms (factor 1) was greater in the CBT-MI group as compared with MEC at post-treatment (CBT-MI vs. MEC: −3.6 ± 0.5 vs. −1.8 ± 0.5; $P = 0.005$), 1-month follow-up (CBT-MI vs. MEC: −4.2 ± 0.5 vs. −1.8 ± 0.4; $P = 0.0003$), and 3-month follow-up (CBT-MI vs. MEC: −3.7 ± 0.5 vs. −2.3 ± 0.5; $P = 0.03$) (Fig. 1D). Similarly, the change from baseline of ISI Perception of Symptoms related to Insomnia (factor 3)

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TABLE 3. Change from baseline for study outcomes and between-group differences

	CBT-MI		MEC		Difference	
Outcome	n	β ± SE	n	β ± SE	β ± SE	P^a
ISI						
Baseline	18	15.1 ± 1.1	25	15.0 ± 0.91	0.1 ± 1.4	
Post-treatment—baseline	16	−10.2 ± 1.15	20	−6.2 ± 0.99	−4.0 ± 1.4	**0.007**
1-mo follow-up—baseline	14	−10.9 ± 1.19	20	−6.5 ± 0.98	−4.4 ± 1.5	**0.003**
3-mo follow-up—baseline	14	−9.9 ± 1.12	20	−7.5 ± 0.96	−2.5 ± 1.5	0.094
HFRDIS,[b]						
Baseline	17	1.09 ± 0.21	25	1.13 ± 0.16	−0.04 ± 0.27	
Post-treatment—baseline	16	−1.3 ± 0.29	20	−0.5 ± 0.17	−0.9 ± 0.4	**0.01**
1-mo follow-up—baseline	13	−1.1 ± 0.28	19	−0.4 ± 0.18	−0.7 ± 0.3	**0.047**
3-mo follow-up—baseline	13	−0.9 ± 0.26	19	−0.7 ± 0.2	−0.2 ± 0.3	0.5
SES						
Baseline	17	25.2 ± 1.56	25	24.9 ± 1.21	0.3 ± 1.97	0.9
Post-treatment—baseline	16	10.2 ± 1.46	20	5.9 ± 1.24	4.3 ± 1.92	**0.03**
1-mo follow-up—baseline	14	11.9 ± 2.06	20	5.6 ± 1.25	5.9 ± 1.97	**0.003**
3-mo follow-up—baseline	14	10.2 ± 1.49	20	6.7 ± 1.24	3.4 ± 1.95	0.08
CES-D,[b]						
Baseline	17	2.6 ± 0.15	25	2.8 ± 0.14	−0.04 ± 0.25	
Post-treatment—baseline	16	−0.8 ± 0.19	20	−0.3 ± 0.16	−0.4 ± 0.22	0.11
1-mo follow-up—baseline	14	−0.9 ± 0.21	20	−0.3 ± 0.20	−0.6 ± 0.25	**0.02**
3-mo follow-up—baseline	14	−0.8 ± 0.21	20	−0.4 ± 0.16	−0.5 ± 0.29	0.24
MENQOL						
Baseline	17	109.9 ± 9.99	25	113.0 ± 7.69	−3.0 ± 12.61	
3-mo follow-up—baseline	15	−33.2 ± 11.09	20	−25.7 ± 9.20	−7.5 ± 14.41	0.61
STAI,[b]						
Baseline	17	2.95 ± 0.12	25	2.99 ± 0.09	−0.04 ± 0.15	
3-mo follow-up—baseline	15	−0.14 ± 0.13	20	−0.002 ± 0.11	−0.14 ± 0.17	0.5

The p-values $p < .05$ are bolded.

CBT-MI, cognitive behavior therapy for insomnia adapted for menopause; CES-D, Center for Epidemiologic Studies Depression; HFRDIS, Hot Flash Daily Interference Scale; ISI, Insomnia Severity Index; MEC, Menopause Education Condition; MENQOL, Menopause Quality of Life Scale; SES, Self-Efficacy of Sleep Scale; STAI, State-Trait Anxiety Inventory

[a] *p*-values from contrasts of CBT-MI versus MEC in a repeated measures linear model of outcome as a function of intervention arm, post-treatment, 1-month follow-up, 3-month follow-up, and baseline outcome value.

[b] Negative binomial distribution.

was significantly greater in the CBT-MI group compared at post-treatment (CBT-MI vs. MEC: −3.9 ± 0.4 vs. −2.2 ± 0.4; $P = 0.002$) and 1-month follow-up (CBT-MI vs. MEC: −3.9 vs 0.4 vs. −2.5 ± 0.5; $P = 0.02$) (Fig. 1F). There were no significant differences in changes from baseline for ISI daytime symptoms related to insomnia (factor 2) between the CBT-MI and MEC groups (Fig. 1E). An alternative factor analysis of the ISI score based on the investigators of the Menopausal strategies: Finding Lasting Answers to Symptoms and Health (MsFLASH) study[58] is available in Supplementary Figure 2, Supplemental Digital Content 2, http://links.lww.com/MENO/B504.

From baseline to post-treatment, sleep self-efficacy increased 10.2 points in women receiving CBT-MI and 5.9 points in women receiving MEC, a mean between-group difference of 4.3 ± 1.92 points ($P = 0.03$) (Table 3). From baseline to 1-month follow-up, the mean between-group difference was 5.9 ± 1.97 points ($P = 0.003$). SES significantly differed between the CBT-MI and MEC groups in at 1-month follow-up (CBT-MI vs. MEC: 36.8 ± 1.7 vs. 30.5 ± 1.4; $P = 0.01$) but not at post-treatment ($P = 0.07$) or the 3-month follow-up ($P = 0.09$) (Fig. 2A). Standardized mean differences between the groups (ie, effect sizes) for SES at post-treatment and 1-month follow-up were 0.71 and 0.87 SD units, respectively, indicating large treatment effects for CBT-MI on sleep self-efficacy.

Indicators of vasomotor symptoms

The decline in hot flash interference from baseline to post-treatment ($P = 0.01$) and the 1-month follow-up ($P = 0.047$) decreased more in women who received CBT-MI (Table 3). There were significant differences between treatment group ratings of hot flash interference (HFDRIS score) at post-treatment (CBT-MI vs. MEC: 0.9 ± 0.5 vs. 2.2 ± 0.4; $P = 0.01$) and 1-month follow-up (CBT-MI vs. MEC: 1.2 ± 0.6 vs. 2.3 ± 0.5; $P = 0.04$) but not at 3-month follow-up ($P = 0.55$) (Fig. 2B). Standardized mean differences between the groups (ie, effect sizes) for HFRDIS at post-treatment and 1-month follow-up were 0.8 and 0.7 SD units, respectively, indicating large treatment effects for CBT-MI on hot flash interference. The HFDIS was scored with and without Item 4 sleep included and results did not change.

Quality of life

Quality of life related to menopause significantly increased for all participants from baseline to the 3-month follow-up timepoint (111 ± 43 vs. 82.5 ± 39; $P = 0.0025$),

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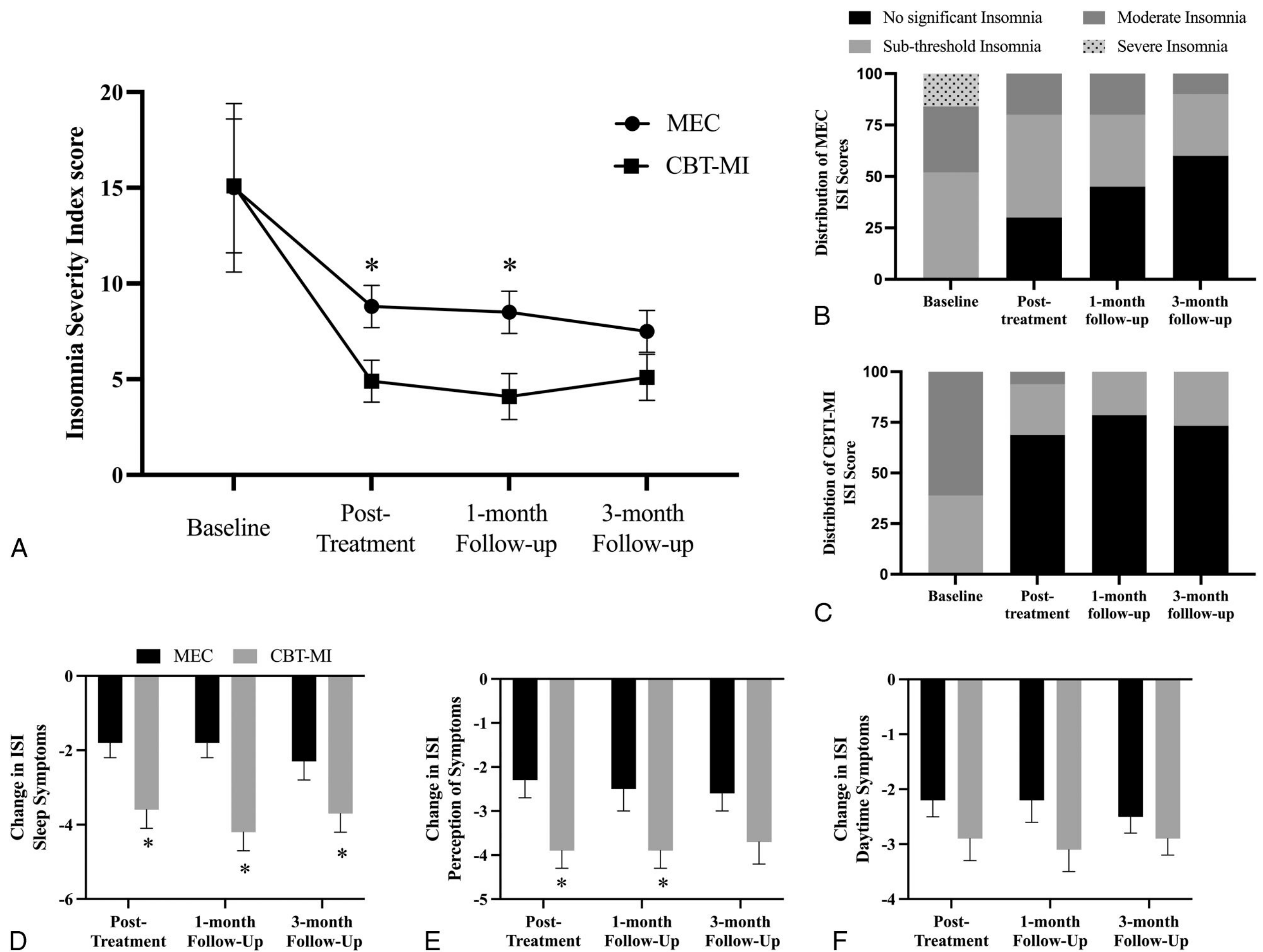

FIG. 1. Insomnia Severity Index, a validated scale to assesses subjective symptoms of insomnia, was administered baseline, post-treatment, 1 month after the completion of treatment, and 3 months after the completion of the treatment to perimenopausal and postmenopausal women randomized to receive cognitive behavioral therapy for insomnia adapted for menopause (CBT-MI, n = 18) or menopause education control (MEC; n = 25). ISI scores at each timepoint are shown in (**A**). The distribution of women reporting no insomnia (ISI score: 0-7), mild insomnia (8-14), moderate insomnia (15-21), and severe insomnia (22-28) across the study timepoints in the CBT-MI and MEC groups are shown in (**B**, **C**), respectively. The change from baseline in insomnia symptom types, sleep symptoms, daytime symptoms, and perception of symptoms, were calculated and presented in (**D-F**). Overall, the CBT-MI intervention significantly decreased symptoms of insomnia in perimenopausal and postmenopausal women, especially in domains related sleep symptoms and perception of symptoms. Data for ISI score and ISI symptom types are presented as mean ± SD. *$P < 0.05$, CBT-MI versus MEC. CBT-MI, cognitive behavior therapy for insomnia adapted for menopause; ISI, Insomnia Severity Index; MEC, Menopause Education Condition.

but did not significantly differ between groups (Table 3; $P = 0.61$).

Indicators of mood

The decline in the CES-D score from baseline to 1-month follow-up was significantly greater for the CBT-MI group compared with the MEC group ($P = 0.02$; Table 3); however, depressive symptoms did not significantly differ between the groups at any timepoint (Fig. 2C). Standardized mean differences (ie, effect sizes) for CES-D at 1-month follow-up were 0.75 SD units, indicating large treatment effect for CBT-MI on depression. There was no significant change in trait and state anxiety in response to the CBT-MI or MEC intervention (Table 3).

DISCUSSION

To address menopausal insomnia in midlife women, we developed a cognitive behavioral therapy that included components to address both insomnia and menopausal symptoms (CBT-MI). Women who met diagnostic criteria for insomnia disorder and reported nightly nVMS were randomized to receive CBT-MI or MEC. Women randomized to CBT-MI experienced a greater reduction in insomnia severity and hot flash interference, and improved self-efficacy related to sleep compared with

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women randomized to MEC. Our findings are consistent with previous studies that reported CBT-I can be used in this population to treat insomnia[59,60] and that it can improve non-sleep symptoms (eg, VMS interference, depression, and daytime effects).[61]

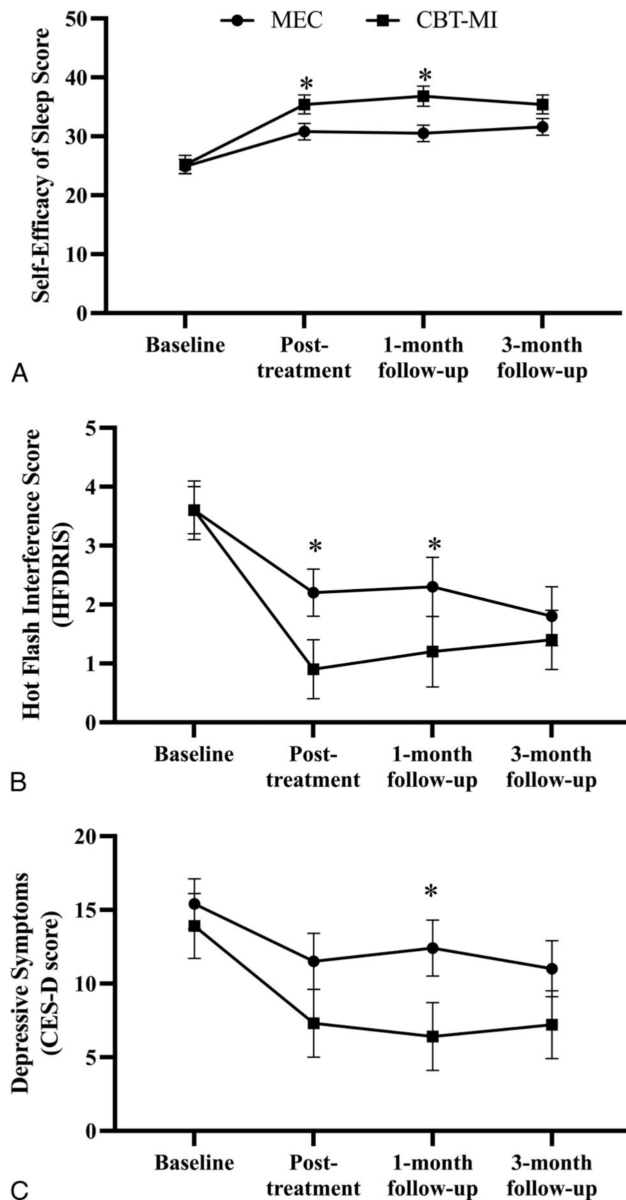

FIG. 2. Self-efficacy to engage in productive sleep behaviors (SES; **A**), ratings of hot flash interference (HFRDIS; **B**), and depressive symptoms (CES-D; **C**) measured at baseline, post-treatment, 1 month after the completion of treatment and 3 months after the completion of the treatment in perimenopausal and postmenopausal women randomized to receive cognitive behavioral therapy for insomnia adapted for menopause (CBT-MI, n = 18) or menopause education control (MEC; n = 25). $^*P < 0.05$, CBT-MI vs. MEC. CBT-MI, cognitive behavior therapy for insomnia adapted for menopause; CES-D, Center for Epidemiologic Studies Depression; HFRDIS, Hot Flash Daily Interference Scale; MEC, Menopause Education Condition; SES, Self-Efficacy of Sleep Scale.

Mechanisms and theoretical context

Given the high prevalence of insomnia in this population, there is a growing body of research that supports the use of CBT-I with this group. CBT-I is a brief, efficacious intervention and is now considered the first-line intervention for insomnia.[17] CBT-I is based on Spielman's three-factor model of insomnia, which posits that although insomnia usually begins with the combination of a predisposition towards insomnia (eg, high emotional reactivity) paired with a precipitating event (eg, menopause, nVMS), the transition to chronic insomnia is usually perpetuated by several factors (eg, increasing time in bed).[62] Some of these perpetuating factors may include an increased effort to induce sleep in response to distress about poor sleep, which leads to conditioned arousal (whereby the bed becomes a cue for arousal rather than sleep) and maintains the sleep problem often even after the causative factors are eliminated.[32] By using the principle of CBT to address distress after nocturnal VMS experience, CBT-I could lead to a reduction in suffering from VMS. Women who perceive greater control over their reactions to VMS tend to regard their VMS as less problematic and report fewer hot flashes.[60] This suggests that addressing reactions to VMS and providing strategies for coping with VMS could reduce distress associated with VMS and provide further benefit when treating insomnia during midlife.

The efficacy of CBT-I in a combined CBT for menopausal insomnia (CBT-MI) intervention in improving insomnia severity in peri and postmenopausal women in this present work is consistent with previously published work.[59,63] Drake and colleagues randomized 150 postmenopausal women with chronic DSM-5 insomnia disorder related to menopause to receive sleep hygiene education control, sleep restriction therapy only, or standard CBT-I.[63] However, it is important to note the participants in this trial did not report VMS. In line with our present work, CBT-I decreased ISI score more than the sleep restriction therapy or sleep hygiene control and the CBT-I group obtained 40-43 more minutes of nightly sleep. Moreover, both standard CBT-I and sleep restriction only therapy interventions improved daytime function, quality of life, and work performance;[61] although CBT-I produced superior results including the added benefit of improved emotional health. Indeed, we found that participants randomized to CBT-MI exhibited improved sleep self-efficacy and mood at follow-up.

Similarly, the MsFLASH network conducted a series of randomized clinical trials with healthy, menopausal women experiencing moderately bothersome hot flashes and found greater reductions in insomnia severity and improvements in self-reported sleep latency, wake time, and sleep efficiency in women randomized to telephone-delivered CBT-I compared with menopause education control.[61,64] Notably, none of the four MsFLASH trials enrolled menopausal women who met

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diagnostic criteria for insomnia disorder. Secondary analysis of the MsFLASH data set to examine the association of VMS to other menopausal symptoms, including insomnia, and found that VMS interference was moderately correlated with insomnia and fairly to moderately correlated with sleep quality/disturbance.[63] Greater improvement in VMS and sleep over time was associated with higher treatment satisfaction. Thus, addressing nVMS and improving VMS interference may have a synergistic effect on improving insomnia symptoms by improving reaction to and bother from nVMS at night and potential associated awakening.

Sustainability of effects

Although CBT-MI produced robust short-term improvements, treatment gains were not sustained at the 3-month follow-up. This attenuation is likely to reflect several factors. As a pilot study, the trial was powered to detect acute but not long-term effects. Attrition at follow-up reduced power further, and some regression toward the mean is expected in smaller behavioral trials. The four-session format may also have been insufficient for maintaining skills beyond the structured intervention period. Similar patterns have been observed in other behavioral sleep interventions. For example, McCurry and colleagues found that sleep improvements after telephone-delivered CBT-I among midlife women with VMS diminished somewhat by 6 months, suggesting that booster sessions or ongoing practice reinforcement may be necessary for durability.[59] Future work should evaluate maintenance strategies, such as brief follow-up sessions, digital tools, or integration into routine primary-care follow-up to sustain benefits.

Differential effects across insomnia dimensions

Exploratory analyses of ISI subscales clarified which aspects of insomnia responded to treatment. CBT-MI produced significant improvement in night-time sleep symptoms (factor 1) and perceived insomnia severity (factor 3), but not daytime symptoms (factor 2). The absence of change in daytime functioning is consistent with broader evidence that nocturnal gains from CBT-I do not uniformly translate into daytime recovery. A comprehensive systematic review and network meta-analysis[65] found that although CBT-I produces moderate improvements in sleep, its effects on daytime outcomes (eg, fatigue, mood, role/occupational functioning) are generally small-to-moderate, indicating that daytime recovery may require additional or tailored components. Complementing this synthesis, Morin et al[66] reported significant improvements in daytime fatigue, mood, and work/social functioning with behavioral therapy or zolpidem in adults with insomnia disorder, underscoring that daytime outcomes can improve when explicitly targeted. Taken together, these findings imply that menopausal fatigue, cognitive inefficiency, and psychosocial stressors may exert independent influences beyond sleep continuity. Future adaptations of CBT-MI could integrate behavioral activation, cognitive restructuring of daytime worry, and fatigue-management modules (eg, light therapy) to extend nocturnal gains to daytime well-being.

Strengths and limitations

The present results were consistent with prior findings by Drake et al[63] and McCurry et al[59] and has several notable strengths including the enrollment of a diverse sample (23% Hispanic/Latino and 23% non-Hispanic Black women) and both peri and postmenopausal women, which is often lacking in the literature. We also diagnosed insomnia disorder using DSM-5 diagnostic criteria and carefully evaluated history of insomnia and assessed if insomnia developed during the menopause transition or during postmenopause. All study participants reported that VMS interfered with their sleep. With respect to acceptability, adherence, and retention, participants rated the acceptability of the study as high and we had equivalent adherence and retention across study groups. Finally, the interventions were delivered by non-sleep experts in women's health clinics, demonstrating ability to deliver the intervention in a "primary care" model imbedded within women's care clinics delivered by nurses or social workers, which is especially important given that sleep interventions often are not accessible to patients

Several limitations of the present study should be noted, including the small and unbalanced sample. Although we were powered to detect significant differences, the relatively smaller sample size of this study may limit generalizability and our ability to conduct subgroup analysis, for example, examining treatment effects by race/ethnicity. Although our sample was relatively diverse, the CBT-MI intervention was not adapted with patient feedback to address health inequities found in sleep and menopausal symptoms. Black women have higher rates of insomnia and VMS and are at greater risk to experience CVD. They were under-represented in our sample. Thus, future studies are needed to further determine the need for intervention adaptation to address health inequities. Another limitation was the control condition did not control for time or attention with the study therapist. Participants randomized to CBT-MI met with a study therapist for four sessions across 8 weeks where participants randomized to MEC met with a study therapist for one session. Weighing pragmatics and goals of the pilot study, we selected a moderately stringent control condition (enhanced treatment as usual) from a continuum of no treatment/wait list to active treatment as control condition options. Future trials should test CBT-MI against a more stringent control condition that controls for time and attention of the study therapist. Another limitation of the study was the relative short duration of follow-up (3 mo). When we examined the three factors of the primary outcome measure (ISI), we found no significant group differences or daytime insomnia symptoms. This is likely due to the short follow-up period and improvements in night-time insomnia symptoms needing a longer period of time to see improvements in other downstream consequences of insomnia (eg, fatigue, performance, health). Also, given this was pilot study, we powered the study to post-

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treatment outcomes as the primary endpoint. Thus, we cannot draw conclusions about the longevity and sustainability of treatment effects. Indeed, we found there was not a statistically significant difference between groups at 3-month follow-up, but this is likely due to six participants being lost to follow-up and reduced power to examine group differences at follow-up. Future trials should power to follow-up assessment, account for attrition, and have a longer follow-up period to examine the long-term effects.

CONCLUSIONS

CBT-MI represents a feasible and promising approach to address menopausal insomnia and nocturnal VMS. The intervention produced meaningful short-term improvements in insomnia severity, hot flash interference, sleep self-efficacy, and depressive symptoms, although benefits attenuated by 3 months. These results were fairly equivalent to those found in other trials of standard CBT-I. Thus, both standard CBT-I and CBT-I adapted for menopausal insomnia to include CBT for menopausal symptoms are effective treatments for insomnia. Clinicians may opt to adapt standard CBT-I when women are presenting with bothersome VMS particularly at night that disrupts sleep. These findings underscore the importance of developing maintenance strategies and expanding behavioral targets to include daytime functioning.

REFERENCES

1. Kravitz HM, Ganz PA, Bromberger J, Powell LH, Sutton-Tyrrell K, Meyer PM. Sleep difficulty in women at midlife: a community survey of sleep and the menopausal transition. *Menopause* 2003;10:19-28. doi:10.1097/00042192-200310010-00005
2. National Institutes of Health. National Institutes of Health State of the Science Conference statement on Manifestations and Management of Chronic Insomnia in Adults, June 13-15, 2005. *Sleep* 2005;28:1049-1057. doi:10.1093/sleep/28.9.1049
3. Smith MT, Smith LJ, Nowakowski S, Perlis ML. *Primary insomnia: Diagnostic issues, treatment and future directions*, In ML Perlis & K Lichstein (Eds.). Hoboken, NJ: John Wiley & Sons; 2003.1:214-261
4. Nowakowski S, Meliska CJ, Martinez LF, Parry BL. Sleep and menopause. *Curr Neurol Neurosci Rep* 2009;9:165-172. doi:10.1007/s11910-009-0025-6
5. Thurston RC, Chang Y, Kline CE, et al. Trajectories of sleep over midlife and incident cardiovascular disease events in the study of women's health across the nation. *Circulation* 2024;149:545-555. doi:10.1161/circulationaha.123.066491
6. Bolge SC, Balkrishnan R, Kannan H, Seal B, Drake CL. Burden associated with chronic sleep maintenance insomnia characterized by nighttime awakenings among women with menopausal symptoms. *Menopause* 2010;17:80-86. doi:10.1097/gme.0b013e3181b4c286
7. Gold EB, Sternfeld B, Kelsey JL, et al. Relation of demographic and lifestyle factors to symptoms in a multi-racial/ethnic population of women 40–55 years of age. *Am J Epidemiol* 2000;152:463-473. doi:10.1093/aje/152.5.463
8. Col NF, Guthrie JR, Politi M, Dennerstein L. Duration of vasomotor symptoms in middle-aged women: a longitudinal study. *Menopause* 2009;16(3):453-457. doi:10.1097/gme.0b013e31818d414e
9. Politi MC, Schleinitz MD, Col NF. Revisiting the duration of vasomotor symptoms of menopause: a meta-analysis. *J Gen Intern Med* 2008;23:1507-1513. doi:10.1007/s11606-008-0655-4
10. Thurston RC, Chang Y, Buysse DJ, Hall MH, Matthews KA. Hot flashes and awakenings among midlife women. *Sleep* 2019;42(9):zsz131. doi:10.1093/sleep/zsz131
11. Joffe H, Massler A, Sharkey KM. Evaluation and management of sleep disturbance during the menopause transition. *Semin Reprod Med* 2010;28:404-421. doi:10.1055/s-0030-1262900
12. Ensrud KE, Stone KL, Blackwell TL, et al. Frequency and severity of hot flashes and sleep disturbance in postmenopausal women with hot flashes. *Menopause* 2009;16:286-292. doi:10.1097/gme.0b013e31818c0485
13. Thurston RC, Joffe H. Vasomotor symptoms and menopause: findings from the Study of Women's Health across the Nation. *Obstetr Gynecol Clin* 2011;38:489-501. doi:10.1016/j.ogc.2011.05.006
14. Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am J Psychiatry* 1994;151:1172-1180. doi:10.1176/ajp.151.8.1172
15. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *Am J Psychiatry* 2002;159:5-11. doi:10.1176/appi.ajp.159.1.5
16. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychol* 2006;25:3-14. doi:10.1037/0278-6133.25.1.3
17. Qaseem A, Kansagara D, Forciea MA, Cooke M, Denberg TD. Management of chronic insomnia disorder in adults: a Clinical Practice Guideline From the American College of Physicians. *Ann Intern Med* 2016;165:125-133. doi:10.7326/m15-2175
18. Morin CM, Bei B, Bjorvatn B, Poyares D, Spiegelhalder K, Wing YK. World sleep society international sleep medicine guidelines position statement endorsement of "behavioral and psychological treatments for chronic insomnia disorder in adults: An American Academy of sleep medicine clinical practice guidelines. *Sleep Med* 2023;109:164-169. doi:10.1016/j.sleep.2023.07.001
19. Mitchell MD, Gehrman P, Perlis M, Umscheid CA. Comparative effectiveness of cognitive behavioral therapy for insomnia: a systematic review. *BMC Fam Pract* 2012;13:40. doi:10.1186/1471-2296-13-40
20. Rios P, Cardoso R, Morra D, et al. Comparative effectiveness and safety of pharmacological and non-pharmacological interventions for insomnia: an overview of reviews. *Syst Rev* 2019;8:281. doi:10.1186/s13643-019-1163-9
21. Morin CM, Vallières A, Guay B, et al. Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. *JAMA* 2009;301:2005-2015. doi:10.1001/jama.2009.682
22. van der Zweerde T, Bisdounis L, Kyle SD, Lancee J, van Straten A. Cognitive behavioral therapy for insomnia: a meta-analysis of long-term effects in controlled studies. *Sleep Med Rev* 2019;48:101208. doi:10.1016/j.smrv.2019.08.002
23. Morin CM, Edinger JD, Krystal AD, et al. 0341 SEQUENTIAL THERAPIES FOR COMORBID AND PRIMARY INSOMNIA: A RANDOMIZED CONTROLLED TRIAL. *Sleep* 2017;40(suppl_1):A126. doi:10.1093/sleepj/zsx050.340
24. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. *JAMA* 1999;281:991-999. doi:10.1001/jama.281.11.991
25. The 2023 nonhormone therapy position statement of The North American Menopause Society. *Menopause* 2023;30(6):573-590. doi:10.1097/gme.0000000000002200
26. Samami E, Shahhosseini Z, Elyasi F. The effects of psychological interventions on menopausal hot flashes: a systematic review. *Int J Reprod Biomed* 2022;20:255-272. doi:10.18502/ijrm.v20i4.10898
27. Carmona NE, Starick E, Millett GE, Green SM, Carney CE. Sleep effects of psychological therapies for menopausal symptoms in women with hot flashes and night sweats: a systematic review. *Post Reprod Health* 2024;30:166-181. doi:10.1177/20533691241246365
28. Ye M, Shou M, Zhang J, et al. Efficacy of cognitive therapy and behavior therapy for menopausal symptoms: a systematic review and meta-analysis. *Psychol Med* 2022;52:433-445. doi:10.1017/s0033291721005407
29. Green SM, Donegan E, McCabe RE, Fedorkow DM, Streiner DL, Frey BN. Objective and subjective vasomotor symptom outcomes in

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the CBT-Meno randomized controlled trial. *Climacteric* 2020;23:482-488. doi:10.1080/13697137.2020.1737929
30. Sadeghijoola N, Afshari P, Jofreh MG, Haghighizadeh MH, Abedi P. Comparing the effects of face-to-face versus phone counseling based on cognitive-behavioral therapy for vasomotor symptoms in postmenopausal women: a randomized controlled trial. *Postep Psychiatr Neurol* 2022;31:114-120. doi:10.5114/ppn.2022.122499
31. Hunter MS. Cognitive behavioral therapy for menopausal symptoms. *Climacteric* 2021;24:51-56. doi:10.1080/13697137.2020.1777965
32. Edinger JD, Smith ED, Buysse DJ, et al. Objective sleep duration and response to combined pharmacotherapy and cognitive behavioral insomnia therapy among patients with comorbid depression and insomnia: a report from the TRIAD study. *J Clin Sleep Med* 2023;19:1111-1120. doi:10.5664/jcsm.10514
33. Bei B, Asarnow LD, Krystal A, Edinger JD, Buysse DJ, Manber R. Treating insomnia in depression: insomnia related factors predict long-term depression trajectories. *J Consult Clin Psychol* 2018;86:282-293. doi:10.1037/ccp0000282
34. Nowakowski S, Meers JM. Cognitive-behavior therapy for sleep disorders at midlife. *Menopause* 2021;28:833-835. doi:10.1097/gme.0000000000001771
35. Khademi K, Kaveh MH, Nazari M, Asadollahi A. Perceived lack of behavioral control is a barrier to a healthy lifestyle in post-menopause: a qualitative study. *J Health Popul Nutr* 2024;43:180. doi:10.1186/s41043-024-00674-5
36. Jiang Y, Tao M, Huang C, Lu Q. Menopausal transitional and postmenopausal women's voices: "what influences their adherence to self-management": a systematic review and meta-synthesis of qualitative studies. *Front Public Health* 2025;13:1653806. doi:10.3389/fpubh.2025.1653806
37. Anekwe CV, Cano A, Mulligan J, et al. The role of lifestyle medicine in menopausal health: a review of non-pharmacologic interventions. *Climacteric* 2025;28:478-496. doi:10.1080/13697137.2025.2548806
38. Yazdkhasti M, Simbar M, Abdi F. Empowerment and coping strategies in menopause women: a review. *Iran Red Crescent Med J* 2015;17:e18944. doi:10.5812/ircmj.18944
39. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. *Sleep* 1987;10(1):45-56. doi:10.1093/sleep/10.1.45
40. Bootzin RR, Epstein D, Wood JM. Stimulus control instructions. *Case studies in insomnia*. Springer; 1991:19-28.
41. Morin CM, Espie CA *Insomnia: A clinical guide to assessment and treatment*. Springer Science & Business Media; 2007.
42. Barber K, Charles A. Barriers to accessing effective treatment and support for menopausal symptoms: a qualitative study capturing the behaviours, beliefs and experiences of key stakeholders. *Patient Prefer Adherence* 2023;17:2971-2980. doi:10.2147/ppa.S430203
43. Meers JM, Dawson DB, Nowakowski S. CBT-I for perimenopause and postmenopause, In: Garland S, Gradner M, Cuddihy L. eds. editors *Adapting Cognitive Behavioral Therapy for Insomnia*. San Diego: Elsevier; 2022:333-346.
44. The North American Menopause Society *The Menopause Guidebook*, 8th ed.. United States of America; 2015.
45. National Institute on Aging *Menopause, Time for a Change*. National Institutes of Health USDoHHS. editor 2008.
46. National Heart Lung and Blood Institute. *Facts about Menopausal Hormone Therapy*. National Institutes of Health USDoHHS. editor. 2006.
47. Bastien CH, Vallières A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med* 2001;2:297-307. doi:10.1016/S1389-9457(00)00065-4
48. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28(2):193-213. doi:10.1016/0165-1781(89)90047-4
49. Ji X, Bastien CH, Ellis JG, Hale L, Grandner MA. Disassembling insomnia symptoms and their associations with depressive symptoms in a community sample: the differential role of sleep symptoms, daytime symptoms, and perception symptoms of insomnia. *Sleep Health* 2019;5:376-381. doi:10.1016/j.sleh.2018.12.009
50. Lacks P. *Behavioral treatment for persistent insomnia*. Pergamon Press; 1987.
51. Bluestein D, Rutledge CM, Healey AC. Psychosocial correlates of insomnia severity in primary care. *J Am Board Fam Med* 2010;23:204-211. doi:10.3122/jabfm.2010.02.090179
52. Carpenter JS. The Hot Flash Related Daily Interference Scale: a tool for assessing the impact of hot flashes on quality of life following breast cancer. *J Pain Symptom Manage* 2001;22:979-989. doi:10.1016/s0885-3924(01)00353-0
53. Carpenter JS, Larson JC, Hunter MS, Lensen S, Chen CX, Guthrie KA. Correlations among core outcomes in menopause-recommended vasomotor symptom outcomes in MsFLASH trials. *Menopause* 2024;31:3-9. doi:10.1097/gme.0000000000002280
54. Hilditch JR, Lewis J, Peter A, et al. A menopause-specific quality of life questionnaire: development and psychometric properties. *Maturitas* 1996;24:161-175. doi:10.1016/s0378-5122(96)82006-8
55. Lewis JE, Hilditch JR, Wong CJ. Further psychometric property development of the Menopause-Specific Quality of Life questionnaire and development of a modified version, MENQOL-Intervention questionnaire. *Maturitas* 2005;50:209-221. doi:10.1016/j.maturitas.2004.06.015
56. Lewinsohn PM, Seeley JR, Roberts RE, Allen NB. Center for Epidemiologic Studies Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. *Psychol Aging* 1997;12:277. doi:10.1037//0882-7974.12.2.277
57. Spielberger CD, Gonzalez-Reigosa F, Martinez-Urrutia A, Natalicio LF, Natalicio DS. The state-trait anxiety inventory. *Interamerican Journal of Psychology* 1971;5(3 & 4):145-158. doi:10.30849/rip/ijp.v5i3 & 4.620
58. Otte JL, Bakoyannis G, Rand KL, et al. Confirmatory factor analysis of the Insomnia Severity Index (ISI) and invariance across race: a pooled analysis of MsFLASH data. *Menopause* 2019;26:850-855. doi:10.1097/gme.0000000000001343
59. McCurry SM, Guthrie KA, Morin CM, et al. Telephone-based cognitive behavioral therapy for insomnia in perimenopausal and postmenopausal women with vasomotor symptoms: a MsFLASH randomized clinical trial. *JAMA Intern Med* 2016;176:913-920. doi:10.1001/jamainternmed.2016.1795
60. Kalmbach DA, Cheng P, Arnedt JT, et al. Treating insomnia improves depression, maladaptive thinking, and hyperarousal in postmenopausal women: comparing cognitive-behavioral therapy for insomnia (CBTI), sleep restriction therapy, and sleep hygiene education. *Sleep Med* 2019;55:124-134. doi:10.1016/j.sleep.2018.11.019
61. Kalmbach DA, Cheng P, Arnedt JT, et al. Improving daytime functioning, work performance, and quality of life in postmenopausal women with insomnia: comparing cognitive behavioral therapy for insomnia, sleep restriction therapy, and sleep hygiene education. *J Clin Sleep Med* 2019;15:999-1010. doi:10.5664/jcsm.7882
62. Ellis JG, Cushing T, Germain A. Treating acute insomnia: a randomized controlled trial of a "Single-Shot" of cognitive behavioral therapy for insomnia. *Sleep* 2015;38:971-978. doi:10.5665/sleep.4752
63. Drake CL, Kalmbach DA, Arnedt JT, et al. Treating chronic insomnia in postmenopausal women: a randomized clinical trial comparing cognitive-behavioral therapy for insomnia, sleep restriction therapy, and sleep hygiene education. *Sleep* 2019;42. doi:10.1093/sleep/zsy217
64. Ellis JG, Perlis ML, Espie CA, et al. The natural history of insomnia: predisposing, precipitating, coping, and perpetuating factors over the early developmental course of insomnia. *Sleep* 2021;44(9):zsab095. doi:10.1093/sleep/zsab095
65. Benz F, Knoop T, Ballesio A, et al. The efficacy of cognitive and behavior therapies for insomnia on daytime symptoms: A systematic review and network meta-analysis. *Clin Psychol Rev* 2020;80:101873. doi:10.1016/j.cpr.2020.101873
66. Morin CM, Chen SJ, Ivers H, et al. Effect of psychological and medication therapies for insomnia on daytime functions: a randomized clinical trial. *JAMA Netw Open* 2023;6:e2349638. doi:10.1001/jamanetworkopen.2023.49638

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