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Low-Carbohydrate Diets, Carbohydrate Quality, and Sleep Regulation after Menopause: A Narrative Review

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Abstract. Driven by hormonal fluctuations, vasomotor symptoms, metabolic disturbances, and systemic inflammation, sleep problems often arise during the menopausal transition and postmenopause. Nevertheless, dietary modification may help improve sleep, but the roles of carbohydrate quantity and quality in postmenopausal women remain unclear. This narrative review examines how low-carbohydrate diets (LCDs) and carbohydrate quality relate to sleep after menopause, drawing on observational studies, clinical trials, systematic reviews, and mechanistic research. Existing studies shows that greater adherence to LCDs and very low-carbohydrate ketogenic diets may improve subjective sleep quality, potentially through changes in adiposity, inflammation, and neurochemical pathways. Beyond carbohydrate quantity, carbohydrate quality also seems to affect sleep. In postmenopausal women, higher dietary glycemic index has been linked to higher odds of prevalent and incident insomnia, while higher intake of fiber-rich foods is linked to better sleep outcomes. However, findings vary by diet, study design, and population. Few studies have examined postmenopausal women, as most have focused on younger or different groups. As a result, long-term trials using objective sleep measures are needed to assess whether reducing carbohydrate intake improves sleep after menopause.

Keywords: Postmenopause, sleep quality, low-carbohydrate diet, carbohydrate quality, glycemic index

1. Introduction

During the menopausal transition and postmenopause, fluctuations and declines in ovarian hormone levels are associated with vasomotor symptoms and an increased prevalence of sleep disturbances. In particular, sleep problems have been reported in around 16–47% of perimenopausal women and 35–60% of women during and after the menopausal transition, with frequent nocturnal awakenings, difficulty initiating sleep, and fragmented sleep being among the most common complaints [1]. Poor sleep is also linked to a pro-inflammatory state in this population, with elevated concentrations of high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6) reported in women with poorer sleep quality [2]. Despite these associations, the role of dietary carbohydrate modification in sleep regulation remains unclear.

Growing evidence suggests that carbohydrate quantity and quality may influence sleep through glycemic regulation, neurotransmitter synthesis, inflammatory pathways, and alterations in sleep

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architecture. Low-carbohydrate and ketogenic diets have been linked to changes in subjective sleep quality and sleep architecture, while carbohydrate quality indicators, including glycemic index (GI), glycemic load (GL), and dietary fiber intake, may affect sleep initiation, continuity, and insomnia risk. Yet, much of the available research on carbohydrate restriction has focused on overweight, obese, or metabolically compromised groups rather than postmenopausal women. As a result, direct dietary intervention data in postmenopausal women remain limited, and findings from other groups should be viewed as indirect rather than directly applicable to this population. This review examines the relationship between dietary carbohydrate intake and sleep outcomes in postmenopausal women, focusing on two key dimensions: the quantity of carbohydrates, particularly low-carbohydrate and ketogenic dietary patterns, as well as the quality of carbohydrates, including GI, GL, and dietary fiber. It further considers the metabolic and neurochemical pathways that may underlie these relationships, and evaluates the methodological limitations of the current evidence. By separating direct evidence in postmenopausal women from indirect findings in other populations, it aims to identify key knowledge gaps and priorities for future dietary intervention research.

2. Clinical features and correlates of poor sleep quality in postmenopausal women

2.1. Prevalence and clinical presentation of postmenopausal sleep quality decline

Across the menopausal transition and postmenopause, subjective sleep disturbances occur more frequently than during the premenopausal stage. Community-based epidemiological data reveal that 40% to 56% of perimenopausal and postmenopausal women experience sleep difficulties, which is substantially higher than the 31% reported by premenopausal women in the late reproductive stage [3]. In the Study of Women's Health Across the Nation (SWAN) cross-sectional survey of over 16,000 midlife women aged 40-55, 38% reported difficulty sleeping within the preceding two weeks [4]. Furthermore, about 26% of transitioning women experience symptoms severe enough to meet the diagnostic criteria for clinical insomnia, with marked daytime functional impairment [3].

Regarding specific sleep complaints, sleep maintenance difficulty, marked by frequent nocturnal awakenings, is the most common complaint. In the baseline SWAN cohort of premenopausal and early perimenopausal women, difficulty staying asleep was the most prevalent complaint, reported by 25.9% of participants, followed by early morning awakening (12.4%) and trouble falling asleep (10.1%) [4]. All three types of sleep difficulty vary by menopausal stage, with greater disturbance during the menopausal transition; surgically postmenopausal women not using hormone therapy (HT) show particularly elevated levels of sleep difficulty [4]. However, subjective sleep quality does not always correspond to the objective sleep parameters measured by polysomnography (PSG) [3, 4]. PSG studies show that the impact of the menopausal stage on sleep structure is relatively limited. For instance, the Wisconsin Sleep Cohort Study found that women in the perimenopausal and postmenopausal stages had longer slow-wave sleep duration and slightly higher sleep efficiency than premenopausal women. This difference may be partly explained by quantitative EEG results, as in post-menopausal and post-menopausal women, the power of beta and gamma waves during non-rapid eye movement sleep is higher, suggesting cortical hyperarousal [3]. Besides, longitudinal endocrine tracking further links declining estradiol and rising follicle-stimulating hormone to these subjective complaints [3, 4].

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2.2. Influencing factors of postmenopausal sleep quality decline

During the menopausal transition and postmenopause, sleep disturbances arise from multiple factors. Vasomotor symptoms (VMS), sex steroid changes, body composition, and systemic inflammation are most relevant to the dietary mechanisms discussed below and are examined in more detail here.

VMS, particularly hot flashes and night sweats, are major contributors to sleep-maintenance difficulty. Hot flashes are reported by up to 80% of women during the menopausal transition, with a median duration of approximately 7.4 years, and women reporting VMS on ≥6 of the past 14 days had approximately two- to three-fold higher odds of sleep difficulties (OR = 2.16–2.93) [3, 4]. Specifically, objective sternal skin conductance monitoring indicates that 69.4% of nocturnal hot flashes coincide with awakenings, and hot flash-associated wake time accounts for an average of 27.2% of objective wakefulness after sleep onset (WASO) [3, 5].

Declining sex steroid hormones affect sleep via both direct and indirect pathways. In particular, estradiol acts as a neuroactive steroid on sleep-regulatory regions, including the preoptic area of the hypothalamus and the suprachiasmatic nucleus, and its depletion disrupts these circuits, triggering body temperature fluctuations and hyperarousal [1, 6]. Progesterone and its neuroactive metabolites, such as allopregnanolone, exert sedative effects via GABAergic activity, so declining progesterone may reduce this support [1]. Besides, mood disturbance is also bidirectionally associated with sleep difficulty, and limited evidence suggests that an advanced circadian phase and reduced nocturnal melatonin production may contribute to fragmented sleep and early-morning awakening [1, 3].

After menopause, changes in body composition become particularly relevant to diet. Increased visceral and upper-body adiposity, together with reduced progesterone-dependent ventilatory drive and upper-airway muscle tone, predisposes women to sleep-disordered breathing and increases the risk of obstructive sleep apnea [1, 3]. Poor sleep is further associated with a pro-inflammatory state: higher global Pittsburgh Sleep Quality Index (PSQI) scores, indicating poorer sleep quality, and lower sleep efficiency are associated with higher hs-CRP, IL-6, and interferon gamma-inducible protein-10 levels, while shorter sleep duration is associated with higher hs-CRP [2]. As adiposity and low-grade inflammation are both diet-responsive, they provide the most plausible point of contact between carbohydrate intake and sleep in this population.

3. Low-carbohydrate dietary patterns and sleep quality

3.1. Low-carbohydrate diets and subjective sleep quality

In a cross-sectional study conducted on overweight or obese women, higher LCD compliance was associated with better subjective sleep quality, as indicated by lower PSQI scores. Besides, after adjustment for age, occupational status, psychological stress, and body weight, high LCD adherence was associated with 57% lower odds of poor sleep quality than low adherence (OR = 0.43, 95% CI: 0.19–0.94; p = 0.03) [7]. Mechanistically, carbohydrate restriction may influence sleep partly through systemic inflammation. Reduced carbohydrate intake has been associated with lower concentrations of IL-6, hs-CRP, and IL-8, and with higher concentrations of the anti-inflammatory adipokine adiponectin [7]. Consistent with a mediating role, the association between LCD adherence and sleep quality was attenuated when hs-CRP (OR = 0.61, 95% CI: 0.30–1.21; p = 0.16) or total antioxidant capacity (OR = 0.60, 95% CI: 0.25–1.40; p = 0.24) was entered into the model, whereas transforming growth factor-β and interleukin-1β did not attenuate it [7]. Another potential pathway is neurochemical: when carbohydrate restriction is accompanied by higher protein intake, greater

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dietary tryptophan availability may support serotonin and subsequently melatonin synthesis [7]. Table 1 summarizes the study designs, populations, and sleep-related findings, along with the relevance of each study to postmenopausal women.

Table 1. Summary of evidence on low-carbohydrate dietary patterns and sleep outcomes

Study	Study design and population	Dietary exposure/intervention	Sleep assessment	Main sleep-related findings	Relevance to postmenopausal women
[7]	Cross-sectional; 304 women with overweight or obesity, aged 19–50 years	LCD adherence assessed using an LCD score	PSQI	Higher LCD adherence was associated with lower odds of poor sleep quality after adjustment (OR = 0.43, 95% CI: 0.19–0.94; p = 0.03)	Indirect: women were not selected by menopausal status; findings may be relevant to shared adiposity and inflammatory pathways but do not reflect menopausal hormonal changes
[8]	Uncontrolled, single-center, open-label pilot study; 324 women with overweight or obesity, aged 18–69 years	31-day active-phase VLCKD	PSQI	Significant improvements in all PSQI components and global PSQI score (p < 0.001); 22.2% improved their sleep-quality category	Indirect: the age range included postmenopausal women, but results were not reported by menopausal status
[9]	Retrospective study; 220 patients with PCOS aged 18–40 years	Ketogenic diet	PSQI	Sleep disorders after KD were associated with anxiety, depression, impaired glucose regulation, and elevated LDL-C	Indirect: reproductive-age women with PCOS have distinct metabolic and hormonal characteristics that limit direct applicability to postmenopausal women

Note. KD, ketogenic diet; LCD, low-carbohydrate diet; PCOS, polycystic ovary syndrome; PSQI, Pittsburgh Sleep Quality Index; VLCKD, very low-calorie ketogenic diet. None of the studies summarized here enrolled postmenopausal women specifically; all evidence on low-carbohydrate and ketogenic diets discussed in this review is therefore indirect.

3.2. Potential impacts of very low-carbohydrate / ketogenic diets on sleep quality

Very low-calorie ketogenic diets (VLCKDs) and ketogenic diets (KDs) are characterized by marked carbohydrate restriction aimed at inducing nutritional ketosis. In an uncontrolled single-center pilot study, a total of 324 overweight or obese women participated. The 31-day intensive VLCKD intervention was significantly associated with improved sleep quality. The average PSQI score decreased from 7.74 ± 4.89 at baseline to 4.97 ± 3.57 after the intervention ($p < 0.001$) [8]. All components of PSQI also showed significant improvement ($p < 0.001$) [8]. In addition, short-term experimental studies have shown that an extremely low-carbohydrate diet can alter sleep structure, increase slow-wave sleep (SWS), and reduce rapid eye movement sleep (REM) [10].

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Of the factors examined, fat loss showed the strongest link with improved sleep. The percentage change in fat mass (Δ% FM) was the only independent predictor of changes in sleep quality after VLCKD ($R^2 = 0.201$, $\beta = -0.452$; $p < 0.001$)). Moreover, receiver operating characteristic analysis identified a Δ% FM threshold of > −8.4% as predictive of improved sleep quality (AUC = 0.686; $p < 0.001$) [8]. Additionally, fat loss may act by reducing obesity-related low-grade inflammation and fat deposition around the upper airway [8]. Besides, another mechanism has been proposed but not demonstrated in humans: ketone bodies, particularly β-hydroxybutyrate, inhibit histone deacetylases including HDAC4 in cell-culture studies, and HDAC4 participates in signaling pathways regulating sleep and wakefulness [8].

Ketogenic diets may also yield mixed effects. For example, early in ketosis, transient drowsiness and fatigue have been reported in women with polycystic ovary syndrome (PCOS), possibly due to metabolic adaptation or electrolyte loss [9]. In the same population, anxiety (OR = 1.768, $p < 0.001$), depression (OR = 1.494, $p < 0.001$), impaired fasting glucose or tolerance (OR = 5.278, $p = 0.008$), and LDL-cholesterol (OR = 1.619, $p = 0.002$) were independently associated with the occurrence of sleep disorders following KD intervention [9]. Responses to carbohydrate restriction may therefore vary with metabolic and psychological status. It should be emphasized that no trial to date has examined low-carbohydrate or ketogenic diets specifically in postmenopausal women; the evidence discussed in this section is extrapolated from younger or metabolically distinct populations, and its applicability to postmenopausal women remains to be established.

4. Carbohydrate quality and sleep regulation: associations and potential pathways

4.1. Glycemic index and glycemic load effects on sleep quality

Recent meta-analytic and prospective evidence suggests that carbohydrate quality, particularly GI and GL (GI × available carbohydrate [g] / 100), may affect specific aspects of sleep continuity and insomnia risk. In a systematic review and meta-analysis of clinical trials, meta-regression analyses showed that differences in dietary GL explained a substantial proportion of the variance in the effects of carbohydrate quality on sleep efficiency (SE; adjusted $R^2 = 89.2\%$, $p < 0.001$), wake after sleep onset (WASO; adjusted $R^2 = 64.9\%$, $p = 0.018$), and sleep onset latency (SOL; adjusted $R^2 = 50.8\%$, $p = 0.048$) [11]. Unlike these acute trials, long-term observational data from the Women's Health Initiative (WHI) indicate that at baseline, participants in the highest quintile group of dietary GI had a significantly higher prevalence of insomnia compared to those in the lowest quintile group (OR = 1.11, 95% CI: 1.05–1.16; P-trend = 0.0014), and after a 3-year longitudinal analysis, the risk of new-onset insomnia was also significantly higher (OR = 1.16, 95% CI: 1.08–1.25; P-trend < 0.0001). This result was confirmed in the fully adjusted model [12]. Moreover, the higher the intake of added sugars, starch, and non-whole grains or refined grains, the slightly higher the risk of new-onset insomnia (OR for the highest quintile group compared to the lowest quintile group was 1.13–1.16, all P-trend ≤ 0.0043) [12].

These two sets of evidence concern different exposures and are not contradictory. Acute feeding trials test how a single high- or low-GI meal influences the following sleep episode, while cohort studies such as the WHI assess habitual dietary patterns over years. Accordingly, a pre-bed high-GI meal may shorten sleep onset latency via a transient rise in tryptophan availability and subsequent melatonin synthesis, whereas repeated postprandial glycemic excursions over the long term may promote nocturnal arousal and fragmented sleep [11, 12]. Even within the acute literature, findings are heterogeneous: some trials report improvements in sleep onset latency, sleep efficiency, or WASO after high-GI meals, whereas others report weak or no effects [11].

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Observational findings are also mixed. In a cross-sectional study of an elderly population, higher GL was linked to significantly *lower* odds of poor sleep quality after multivariable adjustment (OR = 0.159, 95% CI: 0.054–0.468; P-trend < 0.001), a direction opposite to that observed in the WHI [13]. Differences in age, dietary context, and sleep measurement may contribute to this discrepancy, which remains unresolved.

4.2. Dietary fiber, carbohydrate quality index, and sleep-related outcomes

The most direct evidence linking carbohydrate quality to sleep in postmenopausal women comes from the WHI. At baseline, women in the highest versus the lowest quintile of dietary fiber intake had lower odds of prevalent insomnia (OR = 0.87, 95% CI: 0.82–0.92; P-trend < 0.0001) in fully adjusted models, and higher intakes of whole grains, nonjuice fruit, and vegetables (ORs 0.85–0.93; all P-trend ≤ 0.0010) were likewise associated with lower odds of prevalent insomnia (Gangwisch et al., 2020, pp. 434–435). In the 3-year longitudinal analysis, only nonjuice fruit intake remained associated with lower odds of incident insomnia (OR = 0.90, 95% CI: 0.83–0.97; P-trend = 0.0199), whereas the association for vegetables was attenuated and its linear trend was no longer statistically significant [12].

Beyond insomnia itself, two cross-sectional studies suggest that carbohydrate quality may also relate to symptoms that commonly accompany poor sleep after menopause. Higher Carbohydrate Quality Index (CQI) scores, which is derived from dietary fiber intake, GI, the whole grain-to-total grain ratio, and the solid carbohydrate-to-total carbohydrate ratio, were inversely associated with menopausal symptom burden; women in the highest versus the lowest CQI quartile had lower odds of a higher total Menopause Rating Scale score (OR = 0.36, 95% CI: 0.19–0.68) [14]. In an elderly population, higher fiber intake was associated with lower odds of anxiety and stress [13]. Neither study measured sleep as the primary outcome, so both provide indirect support only.

4.3. Carbohydrate quality-related metabolic changes and sleep regulation pathways

Three pathways may link carbohydrate quality to sleep. At present, only the glycemic pathway has human sleep data; the gastrointestinal peptide and gut-brain pathways remain hypothetical.

First, high-GI and high-GL carbohydrate intake may affect sleep through rapid postprandial changes in glucose and insulin. Postprandial hyperglycemia may cause a rise in insulin that lowers plasma glucose to levels that limit brain glucose supply (~70 mg/dL), triggering counterregulatory hormones such as adrenaline, cortisol, glucagon, and growth hormone. The resulting hypoglycemia may cause nocturnal arousal and reduce sleep efficiency [12, 15]. Although higher-fiber, lower-GI carbohydrate sources may support more stable postprandial glucose responses, whether these effects improve sleep remains unclear.

Second, carbohydrate intake may affect sleep by increasing tryptophan availability. This occurs because insulin release after carbohydrate intake promotes peripheral uptake of large neutral amino acids, increasing the plasma tryptophan-to-large neutral amino acid ratio and facilitating tryptophan transport across the blood-brain barrier for serotonin and melatonin synthesis [12, 11]. Because it depends on meal composition and is attenuated when protein is consumed concurrently, it is not necessarily at odds with findings from low-carbohydrate, higher-protein diets [12]. Meta-analytic evidence indicates that higher carbohydrate intake prolongs REM sleep, while lower carbohydrate intake increases N3 sleep; greater postprandial secretion of cholecystokinin and peptide YY after lower-carbohydrate meals has been proposed as an explanation, but the supporting evidence comes from experimental and animal studies rather than human sleep trials [11].

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Third, dietary fiber may act through the gut–brain axis. Fiber serves as a prebiotic substrate for colonic bacteria, promoting the production of short-chain fatty acids with anti-inflammatory and immunomodulatory effects that have been proposed to influence neurotransmitter regulation and mental health [13]. An estrobolome-mediated pathway linking dietary fiber, estrogen recycling, vasomotor symptoms, and sleep has also been suggested, but it has not been demonstrated and is best regarded as a hypothesis.

5. Limitations and future directions in carbohydrate-sleep research

5.1. Uncertainties and methodological limitations in current evidence

Five considerations limit the interpretation of this literature: measurement error in dietary exposure, the assessment of sleep, residual confounding, differences between the populations studied, and inconsistent definitions of carbohydrate quality.

Dietary exposure is difficult to measure accurately because food frequency questionnaires (FFQs) and 24-hour dietary recalls are prone to recall bias, misreporting, and errors in food-composition databases [16]. In particular, FFQs are particularly prone to systematic error when quantitative estimates of habitual intake are needed [17]. A recent methodological review found that 53% of studies using substitution modelling relied on unvalidated FFQ-derived variables, which suggests that dietary exposures in this field are often less precisely characterized than reported estimates imply [17]. Similarly, sleep studies also often rely on subjective diaries or questionnaires rather than polysomnography (PSG) or actigraphy, limiting the assessment of sleep architecture [18].

Most evidence is observational, limiting causal inference. Diet, adiposity, mood, and sleep are closely related, making residual confounding difficult to exclude. This is particularly relevant when dietary components interact with one another and with lifestyle factors, thua making it difficult to isolate the effects of carbohydrate intake. Besides, the study populations also limit generalizability. Research on carbohydrate restriction has largely focused on premenopausal or reproductive-age women or samples without reported menopausal status [7-9]. Meta-analytic evidence on sleep architecture is also based on 179 participants aged 8–45 years, only six of whom were identified as female [11]. These findings should therefore be applied to postmenopausal women with caution, given differences in hormonal status, body composition, and inflammatory profile.

Carbohydrate quality is another challenge because it is defined inconsistently across studies. This reflects differences in fiber content, degree of processing, food-matrix structure, intrinsic versus added sugars, and glycemic response [19]. No single indicator captures all these features, making comparisons across studies difficult and potentially contributing to inconsistent findings.

5.2. Future directions for dietary intervention research in postmenopausal women

Future research should prioritize randomized controlled trials in postmenopausal women. Existing evidence on carbohydrate restriction is largely based on uncontrolled or cross-sectional studies in other populations. Well-powered trials that specifically recruit postmenopausal women and report outcomes by menopausal status are needed before dietary recommendations for sleep can be made.

Such trials should include objective sleep measures. Specifically, combining validated subjective tools with PSG or actigraphy would provide a more complete assessment of sleep architecture and continuity and reduce reliance on self-reported sleep quality [18].

Standardized assessment of carbohydrate quality is a third requirement. This could be achieved by using composite metrics incorporating total fiber, nutrient density, food-group classification,

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food-matrix characteristics, processing, and bioactive components to improve comparability across studies relative to single indicators like GI or GL. Repeated dietary records combined with recovery biomarkers, where available, would also reduce exposure misclassification [16, 17, 19]. Emerging tools like image-based dietary assessment, wearable sleep monitoring, metabolomic profiling, and chronotype-based personalization may further refine these designs, but they are secondary to the need for adequately powered trials in this population [18].

6. Conclusion

This study shows that sleep problems after menopause have multiple causes and are closely linked to hormonal changes, vasomotor symptoms, metabolic changes, and inflammation. In particular, both carbohydrate quantity and quality may play a role in sleep regulation. Low-carbohydrate and ketogenic dietary approaches have been associated with improved sleep quality, potentially through changes in adiposity, inflammation, glycemic regulation, and neurochemical pathways. However, no trial has yet examined these dietary patterns specifically in postmenopausal women, and findings from younger or metabolically distinct populations should be interpreted cautiously. Carbohydrate quality may be particularly relevant to sleep health after menopause, with more direct evidence from postmenopausal women: in the WHI, higher dietary GI was associated with greater odds of insomnia, whereas higher intake of dietary fiber and fiber-rich foods was associated with more favorable sleep outcomes. Nevertheless, inconsistencies across studies, reliance on self-reported dietary and sleep assessments, and heterogeneity in carbohydrate-quality definitions limit causal interpretation. Long-term randomized controlled trials in postmenopausal women are needed to determine whether targeted changes in carbohydrate quantity and quality can improve sleep, with comprehensive dietary and objective sleep measures.

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