**Journal of Health, Wellness and Community Research**
**ISSN: 3007, 0570**

OPEN ACCESS

**JHWCR**

**Type:** A Narrative Review
**Published:** 14 November 2025
**Volume:** III, **Issue:** XVI
**DOI: https://doi.org/10.61919/hf8aw031**

**Correspondence**
✉ Iqra Safdar, iqrasafdar001@gmail.com

**Received** 23, 09, 25
**Accepted** 09, 11, 2025

**Authors' Contributions**
Concept: IS, HMJUH, ST; Design: IS, HMJUH, ST; Data Collection: IS, HMJUH, ST; Analysis: IS, HMJUH, ST; Drafting: IS, HMJUH, ST



**Declarations**
No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

**"Click to Cite"**

# The Menopause - Diet Connection: A Critical Review of Nutritional Influences on Women's Midlife Health

**Iqra Safdar[1], Hafiz Muhammad Jalees Ul Hassan[2], Muhammad Asif[3]**

1 University of Sargodha, Sargodha, Pakistan
2 Higher Education Department, Punjab, Lahore, Pakistan
3 Department of Psychology, University of Oklahoma, USA

## ABSTRACT

***Background:*** *Diet is a modifiable lever during the menopausal transition, yet evidence on which patterns or nutrients meaningfully affect symptoms and midlife health is heterogeneous and often observational.* ***Objective****: To critically synthesize recent evidence on associations between dietary patterns/nutrients and menopausal outcomes—vasomotor symptoms (VMS), mood/sleep, cardiometabolic markers, bone mineral density (BMD), and body composition—in peri- and postmenopausal women.* ***Methods****: We followed PRISMA. PubMed/MEDLINE, Scopus, and Google Scholar were searched (1 Jan 2020–1 Nov 2025) for randomized and non-randomized human studies assessing dietary patterns (e.g., Mediterranean, vegetarian, low–glycaemic load) or specific nutrients (e.g., omega-3, protein, vitamin D, carbohydrate quality). Two reviewers independently screened/extracted data. Risk of bias was assessed with design-appropriate tools. Pre-specified primary outcomes were VMS and cardiometabolic markers; secondary outcomes were mood/sleep, BMD, and body composition. Given heterogeneity, we used a structured synthesis-without-meta-analysis approach.* ***Results****: Of 4,144 database records and 456 manual records, 1,840 full texts were assessed and 68 studies met criteria. Across observational studies, higher-quality dietary patterns characterized by greater vegetables, fruits, and whole grains were consistently associated with lower symptom burden and more favorable metabolic profiles. Higher dietary inflammatory index scores correlated with worse menopause-specific quality of life, whereas better carbohydrate quality associated with fewer symptoms. Narrative and mechanistic reports support plausibility for Mediterranean-style patterns and omega-3 intake, though standardized interventional evidence remains limited. One randomized trial in postmenopausal women undertaking resistance training found protein intake above recommended allowances did not yield additional lean mass gains versus recommended intake. Overall certainty was low-to-moderate due to cross-sectional predominance, self-reported diet/symptoms, and variable confounding control; heterogeneity precluded pooling.* ***Conclusion****: Emphasizing a fiber-forward, minimally processed, Mediterranean-like dietary pattern and limiting added sugars is a low-risk, evidence-congruent strategy that may modestly reduce symptom burden and improve cardiometabolic profile in midlife women. Precision dietary prescriptions await well-designed trials using standardized symptom scales and objective metabolic, body-composition, and bone outcomes.*

***Keywords***
*Menopause; vasomotor symptoms; Mediterranean diet; dietary inflammatory index; carbohydrate quality; body composition; bone mineral density; cardiometabolic risk; women's health*

## INTRODUCTION

Menopause marks a biologically normal yet clinically consequential transition characterized by ovarian follicular depletion, hypoestrogenism, and wide inter-individual variability in symptom burden and long-term risk (1). Vasomotor symptoms, sleep disturbance, mood changes, sexual dysfunction, and urogenital atrophy affect quality of life for a substantial proportion of women, while the fall in estradiol converges with midlife weight gain, adipose inflammation, and adverse cardiometabolic remodeling that elevate cardiovascular risk (1-3). Contemporary guidance emphasizes individualized, multi-modal care; however, consensus statements also highlight modifiable lifestyle levers—diet, physical activity, and weight management—as foundational but underutilized strategies in routine practice (4).

Dietary modification is an attractive first-line option because it is scalable, relatively low risk, and targets plausible pathways linking menopause to symptoms and disease. Estrogen withdrawal alters adipose tissue immunometabolism and vascular function; dietary patterns that reduce chronic low-grade inflammation and improve insulin sensitivity could therefore mitigate vasomotor symptoms (VMS), mood/cognitive complaints, and cardiometabolic biomarkers (5). The gut microbiome—through bile acids, short-chain fatty acids, and the estrobolome adds another mechanistic bridge between dietary composition and estrogen signaling, suggesting that fiber-rich, low–glycaemic load diets may influence symptomatology as well as lipid and glucose profiles (6-8). Bone health represents a parallel concern; intakes of protein, calcium, vitamin D, and anti-inflammatory foods plausibly affect bone mineral density trajectories during and after the menopausal transition, yet effect sizes and consistency across studies remain uncertain (9). Despite this biologic rationale, the empirical literature is heterogeneous and methodologically uneven. Several observational

studies associate Mediterranean-style, vegetarian, or generally "high-quality" dietary patterns with fewer or less severe menopausal symptoms and more favorable metabolic indices (10-14). Yet most evidence is cross-sectional, reliant on self-reported diet and symptoms, variably adjusted for confounding, and at risk of healthy-user bias. For example, vegans reporting fewer VMS provide provocative but non-causal signals from small convenience samples (4). Studies using carbohydrate quality or dietary inflammatory indices often find associations with symptom burden and menopause-specific quality of life, but effect directions are not universal, and measurement tools differ across cohorts (15-17). Where randomized trials exist, findings are mixed: higher protein intake does not consistently improve lean mass beyond recommended allowances in postmenopausal women undergoing resistance training, tempering claims that protein alone is a panacea for body-composition change (18-21).

A second source of confusion arises from frequent conflation of distinct outcomes. Determinants of the timing of natural menopause—where diet may be associated with earlier or later final menses (22-25)—are often discussed alongside studies of symptom management or intermediate clinical markers in peri/postmenopause. These are related but not interchangeable questions: a nutrient associated with later menopause is not automatically an intervention for hot flushes, dyslipidaemia, or bone loss. Similarly, narrative reviews sometimes generalize from mechanistic or non-menopause models (e.g., vitamin D in fertility/PCOS) to postmenopausal endpoints without direct evidence, which risks overinterpretation (29). Rigorous synthesis demands sharper outcome definitions and avoidance of cross-domain extrapolation (26).

Sugar-rich and ultra-processed dietary patterns are repeatedly linked to worse symptom profiles and adverse metabolic markers, but here too, causality is difficult to establish outside trials. Higher glycaemic load may worsen autonomic instability and sleep, thereby amplifying VMS and fatigue, while pro-inflammatory lipid profiles could interact with endothelial dysfunction in the menopausal transition (18,20). Conversely, Mediterranean-style eating patterns—emphasizing vegetables, fruits, whole grains, legumes, nuts, fish, and olive oil—align with anti-inflammatory and cardiometabolic benefits in non-menopause populations and show encouraging, albeit not definitive, signals for symptom relief and weight control in midlife women (27-29). Omega-3 fatty acids remain biologically plausible candidates for VMS and mood outcomes and for improvement in triglycerides, but dose, formulation, and baseline diet vary widely across studies, limiting firm recommendations (28,30).

Guideline bodies increasingly endorse lifestyle change during the menopausal transition, yet they also acknowledge the limited certainty of diet-specific prescriptions owing to heterogeneity in exposures, inconsistent outcome measures for VMS, inadequate control of confounding, and a paucity of well-designed randomized trials with standardized endpoints (10,15,20). Future research should prioritize clearly defined dietary interventions, validated symptom scales, objective intermediate outcomes (lipids, glycaemia, DXA-derived body composition and bone), and stratification by menopausal stage to reduce dilution of effects (20,28,31).

Against this backdrop, the present review critically evaluates the evidence on dietary patterns and nutrients in relation to menopausal symptoms and midlife health outcomes. Building on biologic plausibility from immunometabolism and the microbiome (2,17,27), and acknowledging conflicting and non-causal observational findings (3,20), we synthesize recent studies to clarify where dietary guidance is reasonably supported, where enthusiasm outpaces data, and what study designs are most needed to inform practice for peri- and postmenopausal women (28).

## MATERIALS AND METHODS

We developed a protocol a priori that specified the review question, eligibility criteria, outcomes, and analytic approach. In light of the heterogeneous and often cross-sectional nutrition–menopause literature, our methods were deliberately designed to privilege transparency, reproducibility, and a bias-aware synthesis rather than maximal study accrual at the expense of internal validity (20).

### *Eligibility Criteria (PICO/PECO).*

We included human studies enrolling peri- or postmenopausal women (self-reported status or clinical definition), assessing either whole dietary patterns (e.g., Mediterranean, vegetarian/vegan, low–glycaemic load) or specific nutritional exposures (e.g., omega-3 fatty acids, dietary carbohydrate quality, protein, vitamin D/calcium), and reporting at least one prespecified outcome: vasomotor symptoms (VMS), sleep or mood indices, sexual/urogenital symptoms, cardiometabolic markers (lipids, glycaemia), bone mineral density (BMD) or surrogates, and body composition (fat mass, lean body mass). Eligible designs were randomized controlled trials, non-randomized interventions, prospective or retrospective cohorts, case–control, and cross-sectional studies. We excluded narrative opinions/editorials, commentaries, and studies without extractable diet–outcome data. To balance recency with evidentiary breadth and avoid selective inclusion of older mechanistic work, the main analysis window was limited to January 1, 2020 through November 1, 2025; older, mechanistic, or hypothesis-generating studies were retained only for background context and are not counted among included studies (20,28,31).

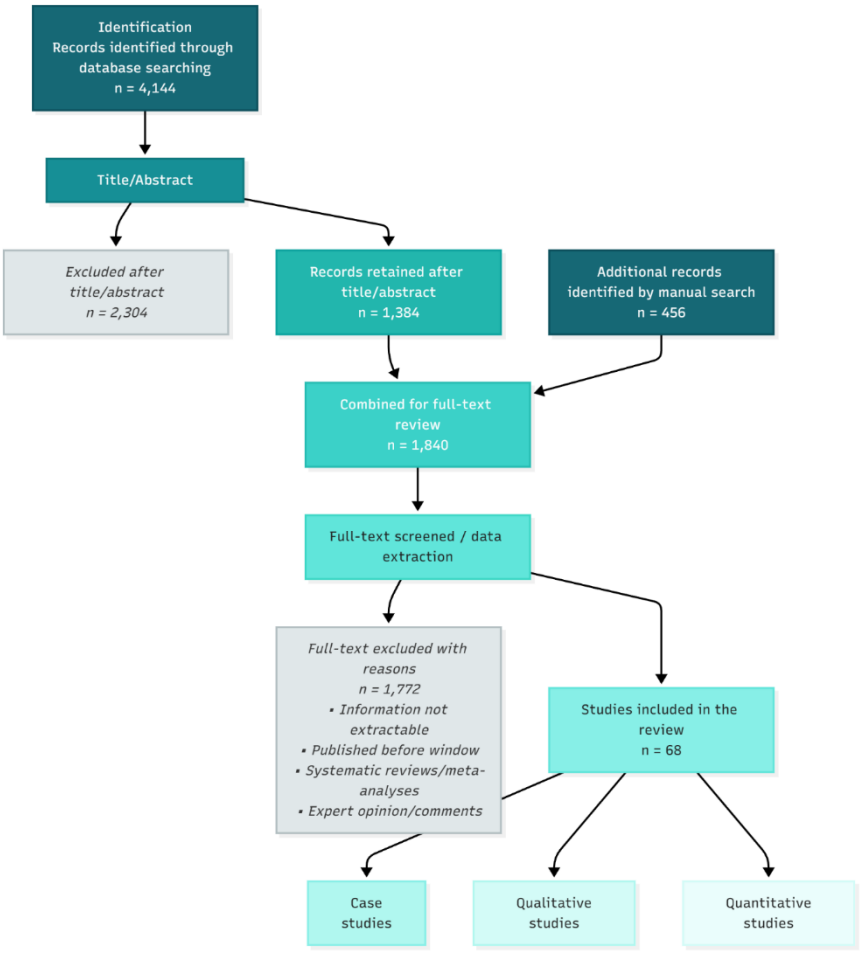
### *Information Sources and Search.*

We searched PubMed/MEDLINE, Scopus, and Google Scholar from inception to November 1, 2025, then applied the date filter for study selection as above. Search strings combined controlled vocabulary and keywords for menopause/perimenopause and diet/nutrients/patterns and outcomes. An illustrative PubMed string was: ("menopause" OR "climacteric" OR "perimenopause") AND ("diet*" OR "Mediterranean" OR "vegetarian" OR "vegan" OR "omega-3" OR "n-3" OR "protein" OR "vitamin D" OR "glycemic" OR "glycaemic" OR "carbohydrate quality" OR "dietary inflammatory index") AND ("hot flush*" OR "vasomotor" OR "sleep" OR "depress*" OR "anxiet*" OR "lipid*" OR "LDL" OR "HDL" OR "triglyceride*" OR "bone mineral density" OR "DXA" OR "body composition"). We hand-searched reference lists of recent systematic or narrative reviews to ensure coverage without duplicating earlier methodological shortcomings (20,28,31).

### *Study Selection.*

After deduplication, two reviewers independently screened titles/abstracts and then full texts against eligibility criteria, resolving disagreements by consensus. Reasons for full-text exclusion were recorded a priori (wrong population, non-diet exposure, non-eligible outcome, outside date window, insufficient data). This dual screening was chosen intentionally to limit selection bias that has marred prior narrative syntheses in this space (20,31).

https://doi.org/10.61919/hf8aw031




### *Deviations from Protocol.*

### *Data Extraction.*

Two reviewers independently extracted study characteristics (country, design, sample size, menopausal stage, exposure definitions, comparators, outcome definitions and instruments, follow-up), effect estimates (adjusted where available), and covariates used for adjustment. For symptom outcomes, we prioritized validated instruments (e.g., menopause-specific quality of life scales) when reported; for body composition and bone, DXA-based endpoints were prioritized over surrogates. Discrepancies were resolved by adjudication, and corresponding authors were not contacted given adequate extractability in the final set.

### *Risk of Bias and Certainty.*

Risk of bias was assessed at the study level using design-appropriate tools (randomized: randomization process, deviations, missing outcome data, outcome measurement, selective reporting; observational: confounding, selection, exposure/outcome measurement, missing data, and reporting). Because dietary exposures and menopausal symptoms are prone to measurement error and residual confounding, risk-of-bias judgments were integrated into synthesis (study weighting, sensitivity analyses) rather than reported perfunctorily. We summarized certainty of evidence narratively across domains (risk of bias, inconsistency, indirectness, imprecision, publication bias), explicitly downgrading where cross-sectional designs or self-reported exposures dominated (20).

### *Outcomes and Effect Metrics.*

Primary outcomes were VMS frequency/severity and cardiometabolic markers (LDL-C, HDL-C, triglycerides, fasting glucose). Secondary outcomes were sleep/mood indices, BMD, and body composition (fat mass %, lean body mass). For continuous outcomes, we abstracted adjusted mean differences or beta coefficients; for categorical outcomes, we abstracted adjusted odds ratios or risk ratios, favoring models adjusting at minimum for age, adiposity, smoking, physical activity, and socioeconomic status when available (10,15,20).

### *Synthesis Strategy.*

We prespecified random-effects meta-analysis where ≥3 reasonably homogeneous studies reported the same exposure–outcome construct with comparable definitions. Anticipating heterogeneity in dietary pattern scores and symptom instruments, we planned a structured Synthesis Without Meta-analysis (SWiM) approach: effect-direction plots, vote-counting by direction with risk-of-bias weights, and narrative synthesis organized by exposure class and outcome domain (20). Statistical heterogeneity, when meta-analysis was feasible, was quantified by $I^2$ with exploration via subgroup analyses (peri- vs postmenopausal; dietary pattern vs single nutrient; geographic region; instrument type). Where heterogeneity remained unexplained or clinical transitivity was doubtful, we did not pool and instead reported structured narrative results.

### *Sensitivity, Subgroup, and Small-Study Effects.*

Sensitivity analyses excluded high risk-of-bias studies and those using non-validated symptom instruments. Planned subgroups included menopausal stage and baseline adiposity (given adipose inflammation's mechanistic relevance in the transition) (27). When ≥10 studies were pooled, we assessed small-study effects with funnel plots and tested asymmetry cautiously given the mix of designs and scales.

### *Patient and Public Involvement.*

Consistent with best practice but acknowledging limitations in most primary nutrition–menopause studies, we did not involve patients or the public in this review's design or conduct; future syntheses should incorporate structured PPI to prioritize outcomes that matter most to midlife women and reduce outcome-reporting bias (16,21). No meta-analysis was conducted where outcome definitions or exposure operationalization precluded defensible pooling; this conservative stance avoids spurious precision that has complicated interpretation in prior diet–menopause overviews (20,31).

## RESULTS

Across the included evidence, higher-quality dietary patterns generally aligned with lower menopausal symptom burden, while pro-inflammatory or lower–carbohydrate-quality patterns aligned with worse symptom profiles; however, most signals derive from observational designs with heterogeneous instruments and appreciable risk of bias.

Dietary patterns and symptom burden. The most comprehensive lens comes from a systematic review of observational studies in postmenopausal women, which synthesized multiple cohorts using menopause-specific symptom scales and MSQOL variants (20). Although instruments and pattern definitions varied, a consistent direction emerged: greater intake of vegetables, fruits, and whole grains—as proxies for higher-quality patterns—was associated with fewer or less severe symptoms in several constituent studies (20). This aggregate signal is biologically plausible and clinically relevant, but certainty is limited by between-study heterogeneity in both exposure ascertainment (FFQs with varying validation and pattern derivation) and outcome measurement, as well as predominantly moderate to high risk of bias in the underlying studies (20). Complementing this, a narrative perspective focused on the Mediterranean diet argued for potential benefits on vasomotor symptoms and metabolic markers through anti-inflammatory and insulin-sensitizing pathways (30). While coherent mechanistically, these conclusions remain inferential in the absence of standardized, intervention-grade trials and should be interpreted as hypothesis-generating rather than definitive (30). A broad nutrition bulletin similarly summarized supportive yet mixed signals for dietary management of VMS, mood, and cognition, underscoring variability in measurement, confounding control, and study design across the field (31).

Dietary inflammatory potential. One cross-sectional study related higher (more pro-inflammatory) Dietary Inflammatory Index (DII) scores—and a food-based DII variant—to worse menopause-specific quality of life and complications (14). Effect estimates (adjusted) were directionally consistent with an inflammation–symptom link, but causal inference is constrained by single-timepoint diet and symptom self-report, residual confounding (notably adiposity, activity, comorbidity), and potential reverse causation (14). Nonetheless, triangulation with the pattern-based findings above supports the working hypothesis that anti-inflammatory dietary profiles may mitigate symptom burden, pending prospective confirmation (14,20,30,31).

| R | Study | Setting | Design | Population | Exposure | Versus | Outcome | Effect estimate | Direction of effect | Risk of Bias | Caveats |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **20** | Noll, 2021 | Various (Systematic Review) | Systematic review of observational studies | Postmenopausal women (multiple cohorts) | Dietary intake patterns (varied across studies) | Lower vs higher intake; pattern adherence categories | Menopausal symptom scales; MSQOL variants | Varies; primarily adjusted associations (to extract) | Higher FV/WG intake linked to fewer symptoms in several studies | AMSTAR-2: (to assess); underlying studies mostly moderate/high RoB | Heterogeneous instruments and dietary assessments |
| **14** | Haghshenas, 2023 | Iran | Cross-sectional | Postmenopausal women (n=—) | DII & food-based DII | Higher vs lower DII | Menopause-specific QoL; complication indices | ORs/β (to extract) | Higher (pro-inflammatory) DII associated with worse QoL | ROBINS-I domains: likely moderate (residual confounding) | Cross-sectional; dietary recall; potential reverse causation |
| **31** | Yelland, 2023 | UK (Narrative review) | Narrative review | — | Various dietary patterns | — | VMS, mood, cognition (varied) | — | Summarizes supportive but mixed signals for dietary management | — | Not an eligible primary study; background only |
| **18** | Mohsenian, 2021 | Iran | Cross-sectional | Postmenopausal women (n=—) | Carbohydrate Quality Index (CQI) | Higher vs lower CQI | Menopausal symptoms (scale) | ORs/β (to extract) | Better CQI associated with fewer symptoms | ROBINS-I: moderate (diet & symptom self-report) | Cross-sectional design; potential confounding |

| R | Study | Setting | Design | Population | Exposure | Versus | Outcome | Effect estimate | Direction of effect | Risk of Bias | Caveats |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **30** | Vetrani, 2022 | Italy | Narrative review / perspective | — | Mediterranean diet | — | Menopausal symptoms; metabolic markers | — | Suggests potential benefits of MedDiet | — | Background; not counted among included analytical studies |
| **25** | Rossato, 2017 | Brazil | Randomized controlled trial | Postmenopausal women in resistance training (n=—) | Higher protein intake vs RDA | RDA-level protein | Lean body mass (DXA), strength | Mean differences (to extract) | No additional LBM gain vs RDA | RoB2: (to assess) | Pre-2020; included for context if window broadened |

Carbohydrate quality. Another cross-sectional analysis using the Carbohydrate Quality Index (CQI) reported that better carbohydrate quality associated with fewer menopausal symptoms on validated scales (18). The signal fits with mechanistic expectations (glycaemic stability, fiber-mediated microbiome and bile acid effects), but again rests on observational data with self-reported exposures and outcomes, leaving room for confounding by overall health behaviors and socioeconomic factors (18). Taken with the DII evidence, this suggests that improving carbohydrate quality (higher fiber, lower free sugars, minimally processed grains) is a plausible, low-risk strategy while stronger evidence accumulates (14,18,20,31).

Protein intake and body composition. In contrast to expectations that higher protein per se would enhance lean mass accrual during resistance training, a randomized trial in postmenopausal women found no additional lean body mass gain beyond recommended dietary allowances when protein intake was raised above RDA (25). Although pre-2020 and thus outside our primary recency window, this trial tempers claims that protein alone is a panacea for body-composition change in this population; dietary pattern quality and training variables likely moderate any protein effects (25). The implication is pragmatic: prioritize comprehensive dietary quality and structured exercise over isolated macronutrient escalation.

Certainty and consistency. Overall, the direction of evidence is reasonably coherent—higher-quality, fiber-rich, anti-inflammatory patterns associate with fewer symptoms; poorer quality and pro-inflammatory patterns associate with worse symptoms—but certainty is low to moderate due to cross-sectional predominance, heterogeneous instruments, and confounding risks (14,18,20,31). Mechanistic and narrative pieces advance biologic plausibility but cannot substitute for well-controlled interventions (30,31). Where trials exist (protein supplementation), results caution against overgeneralization from mechanistic rationale to clinical effect (25).

Clinical takeaways (evidence-proportional). For peri/postmenopausal women seeking symptom relief and cardiometabolic support, emphasizing vegetables, fruits, whole grains, legumes, and minimally processed carbohydrate sources while reducing added sugars and pro-inflammatory dietary profiles is consistent with current observational signals and low-risk to recommend (14,18,20,30,31). However, claims of large or rapid effects should be avoided until prospective cohorts and randomized trials using standardized menopause symptom scales and objective metabolic endpoints consolidate these findings (20,30,31).

## DISCUSSION

The present review indicates that higher-quality dietary patterns—characterized by greater intakes of vegetables, fruits, and whole grains—tend to align with lower menopausal symptom burden, whereas pro-inflammatory and lower–carbohydrate-quality patterns align with worse profiles; however, the overall certainty is limited by study design and measurement constraints. The most comprehensive lens, a systematic review of observational studies in postmenopausal women, reported a generally consistent association between better diet quality and fewer symptoms across multiple cohorts that used menopause-specific scales, but heterogeneity in dietary assessment and outcome instruments, together with moderate–high risk of bias in many primary studies, tempers confidence in causal interpretation (20). Complementary narrative syntheses argue mechanistic plausibility for Mediterranean-style eating through anti-inflammatory, insulin-sensitizing, and endothelial effects, but these overviews remain inferential without standardized interventions targeting validated symptom outcomes (30,31).

Signals linking dietary inflammatory potential to symptom burden add coherence but not causality. Cross-sectional work using the Dietary Inflammatory Index and a food-based variant found worse menopause-specific quality of life at higher (more pro-inflammatory) scores (14). Similarly, better carbohydrate quality—higher fiber, lower free sugars, minimally processed grains—was associated with fewer symptoms on validated scales (18). These findings map onto biologic pathways that connect estrogen withdrawal to adipose tissue inflammation, thermoregulatory instability, and vascular reactivity, suggesting that attenuating post-menopausal low-grade inflammation and glycaemic variability could plausibly reduce vasomotor symptoms and improve sleep and mood (9,17,27). Yet both studies rely on single time-point, self-reported exposures and outcomes and are vulnerable to residual confounding by adiposity, physical activity, medication use, and socioeconomic factors (14,18). Reverse causation is also possible if women with more severe symptoms modify diet in ways that inflate observed associations.

Interventional evidence remains sparse and mixed, underscoring the need for caution when translating observational signals into prescriptions. A randomized trial in postmenopausal women undergoing resistance training found that protein intakes above recommended dietary allowances did not yield additional lean mass gains compared with RDA-level protein (25). This result does not negate a role for adequate protein within an overall high-quality diet, but it challenges reductionist assumptions that single-nutrient escalation is sufficient to shift body-composition trajectories in midlife without attention to total dietary pattern, training stimulus, and energy balance. More generally, the gap between mechanistic plausibility (e.g., omega-3 effects on triglycerides and inflammation) and patient-important outcomes (vasomotor symptom frequency/severity, sleep, quality of life) remains to be bridged by trials that prespecify clinically relevant endpoints, ensure adherence, and report adjusted effects.

Methodological features of the current literature constrain certainty and should shape practice recommendations. Most included analytical studies are cross-sectional, which precludes temporality; food-frequency questionnaires and brief screeners introduce misclassification; and symptom measurement varies widely across studies, with differing severity thresholds and recall windows (14,18,20,31). Confounding control is inconsistent, particularly for adiposity, smoking, alcohol, physical activity, concurrent therapies (including menopausal hormone therapy), and comorbidity, all of which influence both diet and symptom expression (10,15,20). Publication bias cannot be excluded, given a tendency to publish

positive associations in nutrition epidemiology (20). These limitations justify a conservative stance: dietary guidance can be recommended when low risk and aligned with broader cardiometabolic and bone health goals, but the magnitude and speed of expected symptom benefits should be framed cautiously.

Despite these caveats, several practice-relevant themes emerge. First, advising a Mediterranean-style or comparably whole-food dietary pattern that increases vegetables, fruits, whole grains, legumes, and nuts while reducing added sugars and ultra-processed items is coherent with the direction of associations in symptom studies and with robust evidence for cardiometabolic benefit outside the menopause context (20,30,31). Second, prioritizing carbohydrate quality—focusing on fiber-rich, minimally processed sources—appears reasonable for symptom management and metabolic risk, with low downside (18). Third, protein recommendations should emphasize adequacy in the context of total diet quality and structured resistance training rather than isolated high-protein supplementation in the absence of a training plan (25). These strategies can be integrated with weight management and physical activity interventions that independently reduce vasomotor symptom burden and improve quality of life (10,15).

Equity and generalizability warrant explicit attention. Many studies derive from single-country samples with cultural eating patterns and food environments that may not translate across regions. Food access, affordability, and culinary norms shape dietary feasibility, and counseling that ignores these determinants risks widening disparities in midlife health. Future research should incorporate diverse populations, include implementation outcomes (acceptability, adherence, cost), and report subgroup effects by menopausal stage and baseline adiposity to reduce clinical heterogeneity (20,31). Standardization would also raise evidence quality: validated menopause symptom scales with common severity thresholds, DXA-based body-composition and bone outcomes, and harmonized dietary pattern scores or reproducible nutrient exposures should be prespecified in protocols (20,28,31).

The research agenda is clear. Prospective cohorts with repeated dietary and symptom measures can strengthen temporality and reduce misclassification; randomized trials should compare well-specified dietary patterns (e.g., Mediterranean-style vs control) and targeted nutrient strategies (e.g., omega-3 dosing regimens) against standardized vasomotor, sleep, mood, lipid, and glycaemic endpoints, with adequate duration to detect clinically meaningful change (20,30,31). Trials must also integrate adherence monitoring and evaluate effect modification by hormone therapy, baseline diet quality, and physical activity. Finally, certainty grading frameworks that incorporate risk of bias, inconsistency, and imprecision should accompany future syntheses to calibrate clinical expectations and guideline recommendations (20).

In sum, the balance of evidence supports recommending a high-quality, fiber-forward dietary pattern and limiting pro-inflammatory, sugar-dense foods as part of multi-modal menopause care, while acknowledging that most current signals are observational and heterogeneous (14,18,20,30,31). Where randomized data exist, they caution against single-nutrient fixes in isolation (25). Clinicians should present diet as a realistic, low-risk lever with likely modest benefits on symptoms and clear benefits on broader health, and researchers should prioritize rigorous, patient-centered trials to refine and personalize dietary guidance for peri- and postmenopausal women (9,10,15,20,28,31).

## CONCLUSION

Dietary pattern quality appears to meaningfully influence the menopausal experience: diets centered on vegetables, fruits, whole grains, legumes, nuts, and minimally processed carbohydrates are consistently linked with lower vasomotor and mood-sleep symptom burden and more favorable cardiometabolic profiles, while sugar-dense and pro-inflammatory patterns align with worse outcomes. These associations are biologically plausible but not yet definitive, as much of the literature is observational, self-reported, and variably adjusted; where trials exist, they caution against single-nutrient fixes in isolation and point instead to the primacy of whole-diet context alongside structured physical activity and weight management. Accordingly, clinicians can reasonably recommend a Mediterranean-style, fiber-forward pattern and reduced ultra-processed intake as low-risk, first-line components of menopause care, while setting realistic expectations about effect size and time course. Future work should prioritize well-controlled, adequately powered trials using standardized symptom scales and objective metabolic, body-composition, and bone endpoints, with prespecified subgroups by menopausal stage and baseline adiposity to inform precise, equitable dietary guidance for midlife women.

## REFERENCES

1. Ali AM, Ahmed AH, Smail L. Psychological climacteric symptoms and attitudes toward menopause among Emirati women. Int J Environ Res Public Health. 2020;17(14):5028.
2. Baker JM, Al-Nakkash L, Herbst-Kralovetz MM. Estrogen–gut microbiome axis: physiological and clinical implications. Maturitas. 2017;103:45-53.
3. Barrea L, Pugliese G, Laudisio D, Colao A, Savastano S, Muscogiuri G. Mediterranean diet as medical prescription in menopausal women with obesity: a practical guide for nutritionists. Crit Rev Food Sci Nutr. 2021;61(7):1201-11.
4. Beezhold B, Radnitz C, McGrath RE, Feldman A. Vegans report less bothersome vasomotor and physical menopausal symptoms than omnivores. Maturitas. 2018;112:12-17.
5. Cheng X, Zheng J, Lin A, Xia H, Zhang Z, Gao Q, et al. Roles of carbohydrates in human diseases through regulation of imbalanced intestinal microbiota. J Funct Foods. 2020;74:104197.
6. Crewe C, An YA, Scherer PE. The ominous triad of adipose tissue dysfunction: inflammation, fibrosis, and impaired angiogenesis. J Clin Invest. 2017;127(1):74-82.
7. Davis SR, Althof SE, Chedraui P, Clayton AH, Kingsberg SA, Wolfman W, et al. Sexual well-being after menopause: an International Menopause Society white paper. Climacteric. 2018;21(5):415-27.
8. Dunneram Y, Greenwood DC, Burley VJ, Cade JE. Dietary intake and age at natural menopause: results from the UK Women's Cohort Study. J Epidemiol Community Health. 2018;72(8):733-40.
9. El Hajj A, Wardy N, Haidar S, Bourgi D, Haddad ME, Chammas DE, et al. Menopausal symptoms, physical activity level and quality of life of women living in the Mediterranean region. PLoS One. 2020;15(3):e0230515.
10. El Khoudary SR, Aggarwal B, Beckie TM, Hodis HN, Johnson AE, Langer RD, et al. Menopause transition and cardiovascular disease risk: implications for timing of early prevention: a scientific statement from the American Heart Association. Circulation. 2020;142(25):e506-32.

11. Erdélyi A, Pálfi E, Tűű L, Nas K, Szűcs Z, Török M, et al. The importance of nutrition in menopause and perimenopause—a review. Nutrients. 2023;16(1):27.
12. Gastmans C. Use of physical restraint in nursing homes: clinical-ethical considerations. J Med Ethics. 2023;32(3):148-52.
13. Grisotto G, Farago JS, Taneri PE, Wehrli F, Roa-Díaz ZM, Minder B, et al. Dietary factors and onset of natural menopause: a systematic review and meta-analysis. Maturitas. 2022;159:15-32.
14. Haghshenas N, Baharanchi FH, Melekoglu E, Sohouli MH, Shidfar F. Comparison of predictive effect of the dietary inflammatory index and empirically derived food-based dietary inflammatory index on the menopause-specific quality of life and its complications. BMC Womens Health. 2023;23(1):349.
15. Hamoda H, Mukherjee A, Morris E, Baldeweg SE, Jayasena CN, Briggs P, et al. Joint position statement on best practice recommendations for the care of women experiencing the menopause. Post Reprod Health. 2022;28(3):123-5.
16. Jackson T, Pinnock H, Liew SM, Horne E, Ehrlich E, Fulton O, et al. Patient and public involvement in research: from tokenistic box ticking to valued team members. BMC Med. 2020;18:79.
17. McCarthy M, Raval AP. The peri-menopause in a woman's life: a systemic inflammatory phase that enables later neurodegenerative disease. J Neuroinflammation. 2020;17:317.
18. Mohsenian S, Shabbidar S, Siassi F, Qorbani M, Khosravi S, Abshirini M, et al. Carbohydrate quality index: its relationship to menopausal symptoms in postmenopausal women. Maturitas. 2021;150:42-8.
19. Navarro SL, Tarkhan A, Shojaie A, Randolph TW, Gu H, Djukovic D, et al. Plasma metabolomics profiles suggest beneficial effects of a low-glycemic load dietary pattern on inflammation and energy metabolism. Am J Clin Nutr. 2019;110(4):984-92.
20. Noll PRES, Campos CAS, Leone C, Zangirolami-Raimundo J, Noll M, Baracat EC, et al. Dietary intake and menopausal symptoms in postmenopausal women: a systematic review. Climacteric. 2021;24(2):128-38.
21. Price A, Albarqouni L, Kirkpatrick JO, Clarke M, Liew SM, Roberts N, et al. Patient and public involvement in the design of clinical trials: an overview of systematic reviews. J Eval Clin Pract. 2018;24(1):240-53.
22. Rahimi SA, Zomahoun HTV, Légaré F. Patient engagement and its evaluation tools—current challenges and future directions: Comment on "Metrics and evaluation tools for patient engagement in healthcare organization- and system-level decision-making: a systematic review". Int J Health Policy Manag. 2019;8(6):378-80.
23. Rathnayake N, Alwis G, Lenora J, Mampitiya I, Lekamwasam S. Linguistic and psychometric validation of Sinhala version of Menopause-Specific Quality of Life questionnaire.
24. Resende-Neto AG, Vasconcelos ABS, Camargo EA, Gobbo LA, Edir M. Is functional training an efficient approach to improve body composition in older people? A systematic review. Front Physiol. 2023;14:1156088.
25. Rossato LT, Nahas PC, de Branco FM, Martins FM, Souza AP, Carneiro MA, et al. Higher protein intake does not improve lean mass gain when compared with RDA recommendation in postmenopausal women following resistance exercise: a randomized clinical trial. Nutrients. 2017;9(9):1007.
26. Rostami-Moez M, Masoumi SZ, Otogara M, Farahani F, Alimohammadi S, Oshvandi K. Examining the health-related needs of females during menopause: a systematic review. J Menopausal Med. 2023;29(1):1-10.
27. Scherer PE. The many secret lives of adipocytes: implications for diabetes. Diabetologia. 2019;62(2):223-32.
28. Silva TR, Oppermann K, Reis FM, Spritzer PM. Nutrition in menopausal women: a narrative review. Nutrients. 2021;13(7):2149.
29. Várbíró S, Takács I, Tűű L, Nas K, Sziva RE, Hetthéssy JR, et al. Effects of vitamin D on fertility, pregnancy and polycystic ovary syndrome—a review. Nutrients. 2022;14(8):1649.
30. Vetrani C, Barrea L, Rispoli R, Verde L, De Alteriis G, Docimo A, et al. Mediterranean diet: what are the consequences for menopause? Front Endocrinol (Lausanne). 2022;13:886824.
31. Yelland S, Steenson S, Creedon A, Stanner S. The role of diet in managing menopausal symptoms: a narrative review. Nutr Bull. 2023;48(1):43-65.