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Nutritional Determinants of Sleep Quality: A Narrative Review

Review Article

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Abstract

Background: Sleep is a fundamental physiological process, and chronic sleep disturbances are common worldwide. Recent estimates suggest that clinically relevant insomnia affects approximately one in six adults globally. Nutrition is increasingly recognized as a modifiable component of sleep health because dietary composition and timing influence neurotransmitter availability, circadian signaling, metabolic homeostasis, inflammation, and the gut–brain axis. The aim of this review is to synthesize current evidence on how macronutrients, micronutrients, dietary patterns, meal timing, specific foods and bioactive compounds, and gut microbiome-related pathways are associated with sleep quality, duration, continuity, and architecture.

Methods: This narrative review synthesizes randomized controlled trials, systematic reviews, meta-analyses, and relevant mechanistic and epidemiological studies identified in PubMed/MEDLINE, Scopus, and Web of Science. The final database search was completed on 1 September 2026, with no lower publication-year restriction. After duplicate references were removed, 77 unique sources were retained in the final narrative synthesis. Evidence strength was appraised using structured narrative criteria based on study design, consistency, precision, directness, sleep-outcome measurement, and risk of confounding or bias. Findings were integrated thematically. No, de novo meta-analysis was performed, and effect estimates with 95% confidence intervals were reported when available.

Results: The most consistent evidence favors overall diet quality rather than isolated nutrients. Mediterranean-style and other high-quality dietary patterns are associated with fewer insomnia symptoms and better sleep characteristics, whereas Western and ultra-processed dietary patterns are associated with poorer sleep outcomes. Caffeine and alcohol have reproducible adverse effects on objective sleep measures, including total sleep time, sleep efficiency, and sleep architecture. Tryptophan, omega-3 fatty acids, vitamin D, magnesium, and selected functional foods such as tart cherries show potential benefit, but effects are generally modest, heterogeneous, or strongest in deficient or symptomatic subgroups. Evidence for time-restricted eating and microbiome-targeted interventions remains promising but insufficient for universal clinical recommendations.

Conclusions: Nutrition is a clinically relevant component of sleep health, but the evidence supports a food-first and pattern-based approach rather than routine use of individual supplements. The most defensible strategy is a nutrient-dense, minimally processed dietary pattern with adequate fiber and protein, regular meal timing aligned with the daytime active phase, limited evening caffeine and alcohol, and correction of documented nutrient deficiencies. Larger randomized trials using objective sleep measures and standardized dietary interventions are needed to establish causality and define personalized recommendations.

Keywords: Sleep Quality; Nutrition, Dietary Patterns; Mediterranean Diet; Tryptophan, Caffeine; Alcohol, Micronutrients; Gut–Brain Axis;Circadian Rhythm

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Introduction

Sleep is an essential biological process that supports neurocognitive function, emotional regulation, metabolic homeostasis, cardiovascular health, endocrine signaling, and immune competence [1]. Sleep disorders and persistent sleep insufficiency are therefore not simply symptoms of fatigue but clinically relevant exposures associated with a broad range of adverse outcomes [2]. Recent global estimates suggest that approximately 16.2% of adults meet criteria for clinically relevant insomnia and around 7.9% may have severe insomnia, although prevalence varies according to the diagnostic method, country, age, and sex [3]. Poor sleep quality and short sleep duration are also associated with obesity, type 2 diabetes, cardiovascular disease, depression, anxiety, impaired cognition, accidents, and reduced productivity [2].

Sleep health is multidimensional. Relevant outcomes include sleep duration, sleep onset latency, wake after sleep onset (WASO), sleep efficiency, sleep architecture, circadian timing, daytime sleepiness, and subjective sleep quality. These outcomes are not interchangeable [4]. A nutritional exposure may improve one dimension without affecting another - for example, a supplement may change subjective sleep quality without materially altering objective total sleep time. This distinction is important when interpreting the literature and helps explain why findings across nutrition–sleep studies are often heterogeneous [5].

Pharmacological treatments can be appropriate for selected sleep disorders, but medication is not the only modifiable component of sleep health [6]. Behavioral interventions, circadian alignment, physical activity, and diet all contribute to the physiological environment in which sleep occurs [7]. Nutrition is particularly attractive as a preventive and adjunctive target because food intake is repeated daily and simultaneously influences substrate availability, glucose and insulin dynamics, inflammatory tone, autonomic signaling, gut microbial metabolism, and peripheral circadian clocks [8,9].

The relationship is bidirectional. Diet can influence sleep, but insufficient or mistimed sleep can also change appetite, food choice, and energy intake [10]. Experimental sleep restriction has been associated with lower leptin, higher ghrelin, increased hunger, and greater preference for energy-dense foods [11,12]. Consequently, cross-sectional associations between poor diet and poor sleep may reflect a feedback loop rather than a one-way causal pathway [13]. A contemporary review therefore needs to distinguish biological plausibility from clinical efficacy and observational association from interventional evidence.

Human sleep is organized into non-rapid eye movement (NREM) sleep, comprising stages N1, N2, and N3, and rapid eye movement (REM) sleep. N3, or slow-wave sleep, is concentrated predominantly in the first part of the night and is associated with high-amplitude slow-wave activity and restorative physiological processes. REM sleep becomes more prominent later in the night and is involved in emotional and memory-related processing. Sleep is regulated by the interaction of homeostatic sleep pressure (Process S) and circadian timing (Process C), with the suprachiasmatic nucleus coordinating endogenous rhythms across the 24-hour cycle (14). Diet can interact with these systems through several mechanisms. First, amino acids and micronutrients provide substrates and cofactors for neurotransmitter synthesis. Tryptophan is a precursor of serotonin and melatonin, while vitamin B6 supports enzymatic reactions in tryptophan metabolism [5,15]. Second, meal composition alters insulin and the plasma ratio of tryptophan to competing large neutral amino acids, thereby influencing tryptophan transport across the blood–brain barrier [15,16]. Third, meal timing acts as a zeitgeber for peripheral clocks. Eating late in the biological night may contribute to circadian misalignment even when the central light-entrained clock remains unchanged [17]. Fourth, dietary patterns modify systemic inflammation, oxidative stress, and metabolic health, all of which can influence arousal and sleep continuity [5] Fifth, diet is a major determinant of gut microbial composition and microbial metabolites, including short-chain fatty acids (SCFAs), that participate in gut–brain communication [18].

Growing evidence indicates that nutrition may influence multiple dimensions of sleep through interacting metabolic, neurochemical, circadian, inflammatory, and gut microbiome-related pathways. However, the available literature remains heterogeneous with respect to study design, population characteristics, dietary exposures, and sleep assessment methods, making it difficult to draw uniform conclusions across individual nutrients, foods, and dietary patterns. This review therefore aims to provide an integrated overview of current evidence on the relationship between nutrition and sleep. Specifically, it examines the roles of macronutrients, micronutrients, dietary patterns, meal timing, selected foods and bioactive compounds, and gut microbiome-related mechanisms in relation to sleep quality, duration, continuity, and architecture. Particular attention is given to the biological mechanisms that may explain these associations, the consistency and strength of the available evidence, and the methodological limitations that should be considered when interpreting current findings.

Materials and Methods

This narrative review was based on a focused literature search of PubMed/MEDLINE, Scopus, and Web of Science. The final database search was completed on 1 September 2026. No lower publication-year restriction was applied. Literature available from database inception through 1 September 2026 was eligible, and the sources

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retained in the final manuscript ranged from 1986 to 2026. The search strategy combined terms related to sleep, including sleep quality, sleep duration, sleep efficiency, sleep architecture, insomnia, circadian rhythm, and sleep disturbance, with nutrition-related terms covering macronutrients, micronutrients, dietary patterns, Mediterranean diet, meal timing, time-restricted eating, tryptophan, omega-3 fatty acids, magnesium, zinc, vitamin D, caffeine, alcohol, ultra-processed foods, functional foods, probiotics, prebiotics, and gut microbiome. Reference lists of relevant reviews and key articles were also examined to identify additional sources of importance.

Priority was given to systematic reviews, meta-analyses, randomized controlled trials (RCTs), and recent observational studies with direct relevance to the relationship between nutritional exposures and sleep outcomes. Representative mechanistic studies were additionally included when necessary to explain biological pathways involving neurotransmitter synthesis, circadian regulation, inflammatory signaling, and gut–brain communication. Titles and abstracts were screened for direct relevance, followed by full-text assessment of potentially eligible sources. Eligible evidence addressed at least one relevant sleep outcome, including subjective sleep quality, sleep duration, sleep onset latency, wake after sleep onset, sleep efficiency, daytime sleepiness, or sleep architecture assessed using questionnaires, actigraphy, or polysomnography. Sources were excluded when they lacked a direct nutrition–sleep relationship, did not report a relevant sleep outcome, duplicated another source, or did not materially inform the focused synthesis. Because the review was conceived as a narrative rather than a systematic review, a prospective screening log was not maintained. After removal of duplicate references, the final narrative synthesis comprised 77 unique sources.

Methodological quality and evidence strength were appraised using a structured narrative framework. For each exposure, interpretative weight was based on study-design hierarchy (systematic reviews/meta-analyses and RCTs given greater weight than observational or mechanistic studies), sample size and precision, consistency across studies, directness to the nutrition–sleep question, use of objective versus subjective sleep measures, baseline nutritional status, and susceptibility to confounding or other bias. Major exposures were graded as strong, moderate, limited, or emerging evidence. These author-defined categories are intended as a transparent narrative appraisal and are not equivalent to a formal GRADE assessment. Where pooled or primary effect estimates were available, effect sizes were extracted with 95% confidence intervals, units, and comparison groups. Findings were integrated using a thematic narrative synthesis organized a priori into macronutrients, micronutrients, dietary patterns, and meal timing/bioactive foods/gut microbiome. No, de novo meta-analysis or formal qualitative meta-synthesis was performed because the included evidence was heterogeneous in design, exposure definition, population, and sleep outcome. Existing pooled estimates from systematic reviews and meta-analyses were preferentially reported rather than recomputed.

Results

Macronutrients and Sleep

Carbohydrate intake can influence sleep through both acute and chronic pathways. Acutely, carbohydrate-induced insulin release lowers circulating concentrations of several large neutral amino acids relative to tryptophan, potentially increasing the tryptophan-to-LNAA ratio and facilitating central serotonin and melatonin synthesis [19]. In a crossover experimental study of 12 healthy men, a high-glycemic-index jasmine rice meal consumed four hours before bedtime shortened mean sleep onset latency to 9.0 ± 6.2 minutes compared with 17.5 ± 6.2 minutes after a low-GI meal ($P = 0.009$). A 95% confidence interval for this comparison was not reported [20]. This finding supports a plausible acute mechanism but should not be extrapolated to recommend chronically high-glycemic diets. Evidence strength for this acute effect is limited because it is based on a small experimental sample.

Long-term evidence increasingly distinguishes isolated pre-sleep carbohydrate effects from overall diet quality. A 2024 systematic review and meta-analysis of 37 observational studies involving 591,223 participants reported that higher dietary glycemic index was associated with greater odds of insomnia symptoms (OR 1.16, 95% CI 1.08–1.25), as was higher glycemic load (OR 1.10, 95% CI 1.01–1.20), whereas healthier dietary patterns were associated with lower risk [21]. These findings suggest that carbohydrate quality and habitual dietary context are more important for chronic sleep health than the short-term sleep-latency effects observed after selected high-GI meals. Overall evidence strength for habitual carbohydrate quality is moderate, with causal inference limited by the predominance of observational data.

Dietary fiber is one of the more consistent macronutrient-related correlates of sleep architecture. In a controlled feeding study, higher fiber intake was associated with more slow-wave sleep, whereas higher saturated fat and sugar intake were associated with lighter, more fragmented sleep [22]. Fiber may also influence sleep indirectly by improving glycemic stability, supporting microbial diversity, and increasing SCFA production [23]. These mechanisms are biologically plausible, but long-term randomized trials designed specifically around fiber and objective sleep endpoints remain limited. Evidence strength is therefore limited for a causal fiber effect on sleep architecture.

Protein provides tryptophan, the essential amino acid required for serotonin and melatonin synthesis. However, absolute tryptophan intake is not the sole determinant of central availability because

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tryptophan competes with other LNAAs for transport across the blood–brain barrier [24]. Dietary strategies that increase the tryptophan-to-LNAA ratio, including selected carbohydrate–protein combinations or proteins naturally rich in tryptophan relative to other amino acids, may therefore be more relevant than total protein alone [19].

The literature supports a modest sleep-promoting effect of tryptophan but not a universal hypnotic effect. A systematic review, meta-analysis, and meta-regression found that tryptophan supplementation reduced wake after sleep onset (SMD −1.08, 95% CI −1.89 to −0.28), with shorter wake after sleep onset in studies using doses of at least 1 g/day. Other sleep outcomes were not consistently improved [25]. Tryptophan is biologically relevant and may improve selected sleep-continuity measures, but the evidence does not justify indiscriminate supplementation in the general population. Overall evidence strength is moderate but constrained by small, heterogeneous intervention studies.

Protein timing is less established. Evening intake of alpha-lactalbumin, a relatively tryptophan-rich protein, has been shown to increase plasma tryptophan availability and improve morning alertness in selected participants [26]. Claims that pre-sleep casein or other proteins independently improve sleep remain preliminary and are confounded by training status, total daily protein intake, energy balance, and the composition of the accompanying meal [27]. Adequate protein distribution should therefore be considered part of overall dietary adequacy rather than a stand-alone sleep treatment. Evidence strength for protein timing as an independent sleep intervention is emerging.

Long-chain omega-3 polyunsaturated fatty acids (LC-PUFAs), especially eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may influence sleep through membrane composition, inflammatory signaling, and serotonergic pathways [28]. Earlier pediatric and adult trials suggested potential benefits [29,30]. In a 2024 systematic review and meta-analysis of eight controlled trials, omega-3 LC-PUFAs improved sleep efficiency by 1.88 percentage points versus control (MD 1.88, 95% CI 1.00–2.77) and improved subjective sleep measures (MD −0.41, 95% CI −0.76 to −0.06), but did not significantly change sleep latency (MD −0.44 minutes, 95% CI −1.27 to 0.39) or total sleep duration (MD 5.45 minutes, 95% CI −10.33 to 21.23). Heterogeneity was substantial for subjective outcomes [31] Overall evidence strength is moderate.

Accordingly, omega-3 intake should be viewed as potentially supportive rather than a primary treatment for insomnia. Food sources such as oily fish also contribute protein, vitamin D, selenium, and other nutrients, making it difficult to isolate the sleep effect of EPA and DHA from the overall dietary pattern [32]. In contrast, high saturated-fat intake has been associated with reduced slow-wave sleep and greater arousal in controlled and observational studies. Chronic diets high in saturated fat may also promote inflammatory and metabolic disturbances that indirectly impair sleep, although direct causal evidence on sleep architecture remains limited [22]. Evidence strength for saturated fat as an independent determinant of sleep architecture is limited.

Micronutrients and Sleep

Magnesium participates in hundreds of enzymatic reactions and contributes to neuronal excitability, muscle function, and regulation of glutamatergic and GABAergic signaling. These physiological roles make magnesium a plausible sleep-related nutrient, particularly in individuals with low intake or deficiency [33,34]. Earlier placebo-controlled trials in older adults reported improvements in subjective insomnia measures. However, the evidence base is much smaller than popular claims imply [35].

A 2021 systematic review and meta-analysis identified only three RCTs involving 151 older adults. Compared with placebo, magnesium supplementation reduced sleep onset latency by 17.36 minutes (MD −17.36 minutes, 95% CI −27.27 to −7.44), whereas the increase in total sleep time was not statistically significant (MD 16.06 minutes, 95% CI −5.99 to 38.12). The certainty of evidence was low to very low because of small samples and risk of bias [36]. Thus, correcting inadequate magnesium intake is reasonable, but routine supplementation for insomnia in magnesium-replete adults is not strongly supported. Overall evidence strength is limited.

Zinc participates in neurotransmission and may modulate sleep–wake regulation [37]. Experimental and observational evidence has linked zinc status with sleep, and zinc is involved in enzymes and signaling pathways relevant to melatonin biology. In an older trial of institutionalized adults with primary insomnia, a combined preparation containing melatonin, magnesium, and zinc improved sleep quality, but the multicomponent design prevents attribution of the effect to zinc alone. Food-first correction of inadequate zinc intake is therefore more defensible than using zinc as an isolated hypnotic intervention [38].

Iron has a clinically important relationship with sleep because brain iron and dopaminergic function are central to the pathophysiology of restless legs syndrome (RLS) [39]. Low ferritin or iron deficiency can exacerbate RLS symptoms and thereby disrupt sleep initiation and maintenance. This relationship is more clinically established than many other nutrient–sleep associations. In patients with RLS, assessment and correction of iron deficiency should follow disorder-specific clinical guidance rather than generic sleep supplementation [40]. Iron should not be taken empirically in individuals without evidence of deficiency because excess iron can be harmful [41]. Evidence strength is strong for correction of

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iron deficiency in the specific context of RLS, but this should not be generalized to insomnia without iron deficiency.

Vitamin B6 acts as a cofactor in amino-acid metabolism and neurotransmitter synthesis, including pathways involving tryptophan and serotonin [42]. Vitamin B12 participates in one-carbon metabolism and methylation reactions and has been investigated in relation to circadian timing [43]. These biochemical roles support plausibility, but direct intervention evidence showing that B6 or B12 supplementation improves sleep in nutritionally sufficient individuals is limited. Deficiency should be corrected for general neurological and hematological health, while sleep-specific benefits should not be overstated [44]. Evidence strength for direct sleep improvement is limited.

Vitamin D receptors are expressed in brain regions involved in sleep regulation, and low circulating 25-hydroxyvitamin D has been associated with poor sleep quality and sleep disorders in observational studies [45]. Intervention evidence is more encouraging than for several other micronutrients but remains heterogeneous. A 2022 systematic review and meta-analysis of intervention studies found that vitamin D supplementation reduced Pittsburgh Sleep Quality Index scores by 2.33 points versus placebo (MD −2.33, 95% CI −3.09 to −1.57), while effects on sleep duration, specific sleep disorders, and daytime sleepiness were inconsistent [46]. Vitamin D supplementation may improve subjective sleep quality in deficient or selected populations, but vitamin D should not be considered a universal sleep aid. Baseline vitamin D status, dose, treatment duration, comorbidities, seasonal light exposure, and the use of subjective versus objective sleep outcomes are important sources of heterogeneity [47]. Overall evidence strength is moderate for subjective sleep quality and limited for other sleep outcomes.

Calcium is involved in neuronal signaling and may interact with tryptophan–melatonin pathways. Dairy foods also provide protein and tryptophan [48]. Observational studies have explored associations between dairy intake and sleep, but the independent sleep effect of calcium is difficult to isolate from other nutrients and food-matrix effects [49]. Similarly, folate, vitamin C, copper, and other micronutrients participate in metabolic pathways relevant to neurotransmission or oxidative balance, yet direct sleep-specific supplementation evidence is insufficient for routine recommendations [50]. The broader principle is that micronutrient adequacy matters most when a deficiency is present. Evidence strength for calcium and these additional isolated micronutrients as sleep interventions is limited.

Dietary Patterns and Sleep Quality

Among nutritional exposures, overall dietary pattern now has some of the most consistent epidemiological support. Mediterranean-style diets emphasize vegetables, fruits, legumes, whole grains, nuts, olive oil, fish, and relatively low consumption of processed meats and highly refined foods. This pattern provides fiber, unsaturated fats, polyphenols, tryptophan-containing foods, magnesium, and other micronutrients while generally producing a lower inflammatory dietary profile [51]

A 2024 systematic review and meta-analysis found that Mediterranean-diet adherence was associated with lower odds of insomnia symptoms (OR 0.86, 95% CI 0.79–0.93), and broader high-quality dietary patterns were also associated with lower odds (OR 0.66, 95% CI 0.48–0.90) [21]. A 2025 meta-analysis of 62 studies involving 328,493 participants found that sufficient sleep duration, good sleep quality, and earlier chronotype were each associated with greater Mediterranean-diet adherence, although heterogeneity was substantial [52]. These data do not prove that the Mediterranean diet directly treats insomnia because healthier sleep can also facilitate healthier eating and because physical activity, socioeconomic status, body weight, and other lifestyle factors cluster with diet quality. Nevertheless, convergence across large observational datasets and plausible mechanisms makes a Mediterranean-style pattern a reasonable food-first recommendation [53]. Overall evidence strength is moderate, primarily because most data are observational.

Western dietary patterns typically contain high amounts of refined carbohydrates, added sugars, sodium, saturated fat, and ultra-processed foods (UPFs), while providing less fiber and micronutrient density. Such patterns may influence sleep through postprandial metabolic instability, obesity and sleep-disordered breathing risk, inflammation, appetite signaling, and unfavorable changes in the gut microbiome. The relationship is likely bidirectional because insufficient sleep increases preference for energy-dense foods [54]. A 2024 meta-analysis of seven observational studies found that higher UPF intake was associated with greater odds of insomnia (OR 1.53, 95% CI 1.20–1.95) [55]. Another systematic review concluded that evidence for a bidirectional UPF–sleep association is accumulating, but most studies relied on cross-sectional designs and self-reported sleep [56]. Thus, minimizing UPFs is consistent with sleep-health goals, while the causal contribution of processing level remains under investigation. Overall evidence strength is moderate but observational.

Vegetarian and vegan diets can be compatible with healthy sleep when they are well planned, because they are often rich in fiber, phytochemicals, complex carbohydrates, and unsaturated fats [57]. However, restrictive plant-based diets may increase the risk of inadequate vitamin B12, iron, zinc, calcium, or long-chain omega-3 intake if fortified foods or supplementation are not used appropriately [58]. Diet quality, nutrient adequacy, degree of processing, energy intake, and meal timing are likely more important than the presence

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or absence of animal products alone [59]. Evidence strength for plant-based diet status itself as an independent determinant of sleep is limited.

Meal Timing, Bioactive Foods, and Gut Microbiome

Meal timing is increasingly recognized as an important component of circadian health. Peripheral metabolic clocks respond to feeding–fasting cycles, and late-night eating may contribute to circadian misalignment, adverse metabolic effects, and poorer sleep. Time-restricted eating (TRE) has therefore been investigated as a strategy to consolidate food intake within a consistent daytime window [17]. A 2026 meta-analysis of 13 studies involving 638 participants found a small improvement in PSQI in single-arm/TRE-arm analyses (mean change −0.47 points, 95% CI −0.80 to −0.15), with a larger improvement for early TRE schedules (MD −0.77 PSQI points, 95% CI −1.29 to −0.24). However, controlled-trial meta-analyses showed no significant differences in sleep duration or PSQI [60]. Thus, regular daytime-aligned eating may support circadian health, but TRE cannot currently be recommended as a primary treatment for sleep disorders. Overall evidence strength is limited because controlled comparisons are inconsistent.

Specific foods, beverages, and bioactive compounds may also influence sleep. Tryptophan-rich foods, including dairy products, eggs, poultry, fish, soy, nuts, seeds, and legumes, may support serotonin and melatonin synthesis, although central tryptophan availability also depends on competition with other large neutral amino acids (LNAAs) [5]. Consequently, the overall composition of a meal may be more relevant than its absolute tryptophan content, supporting a food-based rather than high-dose supplementation approach [61].

Among commonly consumed beverages, caffeine and alcohol have some of the strongest evidence for adverse effects on sleep. Caffeine antagonizes adenosine receptors and can reduce sleep pressure [62]. In a 2023 meta-analysis of 24 studies, caffeine reduced total sleep time by 45.3 minutes versus control (95% CI 29.0–61.5 minutes reduction) and sleep efficiency by 7.0 percentage points (95% CI 4.0–10.0 reduction), while increasing sleep onset latency by 9.1 minutes (95% CI 3.8–14.4) and wake after sleep onset by 11.8 minutes (95% CI 2.5–21.0) [63]. Individual responses vary according to dose, habitual intake, metabolism, and timing. Overall evidence strength is strong because the effects are reproduced in controlled studies using objective sleep outcomes.

Although alcohol may initially shorten sleep onset latency, it disrupts subsequent sleep architecture (64). A 2025 systematic review and meta-analysis found that, versus control, alcohol delayed REM sleep onset by 18.0 minutes (95% CI 8.8–27.1), reduced REM sleep duration by 11.3 minutes (95% CI −20.8 to −1.8), and reduced the proportion of REM sleep by 2.8 percentage points (95% CI −3.9 to −1.7) [65]. These findings do not support the use of alcohol as a sleep aid and show measurable REM disruption even at relatively low doses. Overall evidence strength is strong for adverse effects on REM sleep architecture.

Several functional foods have shown potential sleep-promoting effects, although the evidence remains limited. Tart cherries contain melatonin and polyphenols, and small intervention studies have reported modest improvements in sleep duration and efficiency [66]. The available systematic review did not provide a single robust pooled effect estimate because studies differed in product formulation, dose, population, and sleep outcomes. Similarly, a small study of adults with sleep problems found that consuming two kiwifruits one hour before bedtime for four weeks was associated with improvements in sleep onset latency, sleep efficiency, and total sleep time [67]. These findings are promising but require confirmation in larger controlled trials. Overall evidence strength is emerging.

Herbal preparations have also been investigated. For example, chamomile contains apigenin, which can interact with GABA-A receptor binding sites and may contribute to mild sedative effects [68]. Small randomized trials have reported improvements in subjective sleep quality, while evidence for valerian, passionflower, lemon balm, and lavender remains inconsistent; because preparations, doses, and outcome scales differ substantially, a single comparable pooled effect estimate is not available for this heterogeneous group [69]. Potential drug interactions further limit firm clinical recommendations. Overall evidence strength is emerging.

The gut microbiome represents another potential link between nutrition and sleep. The gut–brain axis involves neural, endocrine, immune, and metabolic pathways, while microbial metabolites such as short-chain fatty acids (SCFAs) may influence inflammatory signaling, tryptophan metabolism, and neurotransmitter-related processes [70]. Diet is a major determinant of microbial composition: fiber, polyphenols, and unsaturated fats generally support microbial diversity and SCFA production, whereas highly processed, low-fiber diets may have less favorable effects [9]. Evidence strength for the microbiome as a causal mediator of diet-related sleep effects remains emerging because human mediation studies are limited.

Experimental studies suggest that selected Lactobacillus and Bifidobacterium strains can influence GABAergic signaling and stress-related responses [71]. In a 2025 meta-analysis of six RCTs including 424 patients with insomnia, probiotics reduced PSQI scores by 2.10 points versus control (MD −2.10, 95% CI −3.86 to −0.34), but did not significantly improve total sleep time (MD 43.70 minutes, 95% CI −18.07 to 105.46), sleep efficiency (MD 0.41 percentage points, 95% CI −1.67 to 2.48), or sleep latency according to the authors' significance criterion (MD −4.74 minutes, 95% CI

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−9.42 to −0.05; P = 0.05) (72). Probiotic effects remain strain-, dose-, and population-specific. Evidence strength is moderate for subjective sleep quality in insomnia but limited for objective sleep outcomes; the microbiome is therefore better regarded as a plausible mediator than an established universal therapeutic target [73].

Methodological Considerations in Nutrition–Sleep Research

A large proportion of nutrition–sleep research relies on food-frequency questionnaires, diet histories, or 24-hour recalls. These methods are vulnerable to recall error, social desirability, portion-size misestimation, and systematic underreporting of energy-dense foods. Sleep is also frequently measured using one or two self-report questions or questionnaires such as the PSQI and Epworth Sleepiness Scale. These instruments are valuable for symptoms and patient experience but do not directly measure sleep stages or respiratory events.

Measurement error can attenuate true associations, generate spurious associations, and make studies difficult to compare. Future studies should combine validated dietary methods with repeated assessment and, where feasible, objective biomarkers of nutritional exposure. Similarly, sleep outcomes should be matched to the research question: questionnaires for symptoms, actigraphy for multi-night sleep–wake patterns, and PSG for architecture and sleep-disordered breathing.

Diet and sleep share many determinants, including body mass index, socioeconomic status, stress, depression, anxiety, physical activity, shift work, alcohol use, smoking, medication, chronic disease, and chronotype [74]. Inadequate statistical adjustment can therefore produce apparent diet–sleep relationships that partly reflect other lifestyle or clinical factors. Reverse causality is especially important because poor sleep alters appetite and food choice, while better sleepers may have more regular schedules and greater capacity to prepare healthy meals [75].

Heterogeneity also arises from age, sex, metabolic health, baseline nutrient status, cultural diet, habitual caffeine intake, chronotype, and sleep disorder phenotype. An intervention that benefits an older adult with a nutrient deficiency may not benefit a young, healthy adult with adequate intake. Future trials should therefore characterize baseline nutritional and sleep phenotypes rather than treating participants as a homogeneous group.

Polysomnography remains the reference method for detailed sleep architecture and the diagnosis of disorders such as obstructive sleep apnea. It simultaneously records electroencephalography, electrooculography, electromyography, cardiorespiratory signals, and oxygen saturation. Its limitations include cost, participant burden, limited scalability, and the first-night effect, in which unfamiliar laboratory conditions alter sleep [76].

Actigraphy provides a practical alternative for estimating sleep–wake patterns across multiple nights in naturalistic conditions and is well suited to dietary intervention studies [77]. Consumer devices are increasingly used, but performance differs across brands, firmware versions, and populations. Validation against PSG or research-grade actigraphy remains essential before consumer-derived sleep-stage estimates are interpreted as physiological endpoints.

Discussion

Taken together, the findings are best interpreted as a hierarchy of evidence rather than as a list of individuals "sleep-promoting" foods or nutrients. Controlled evidence is most persuasive when a nutritional exposure produces reproducible changes in objective sleep physiology, whereas associations involving overall dietary patterns, micronutrients, and functional foods require greater caution because confounding, reverse causality, baseline nutritional status, and heterogeneous outcome measurement can materially alter the apparent effect [21,36,46,52,63,65]. This distinction should guide both clinical interpretation and the wording of public-health recommendations.

A second cross-cutting issue is context dependence. The same exposure may have different implications according to deficiency status, age, metabolic health, chronotype, habitual intake, and the presence of a specific sleep disorder. Consequently, evidence of benefit in a deficient or symptomatic subgroup should not be generalized to nutritionally replete adults, and statistically significant changes in subjective sleep scores should not automatically be assumed to represent equivalent changes in objective sleep architecture. The evidence-strength ratings in Table 1 were therefore used to separate relatively reproducible effects from signals that remain supportive, limited, or hypothesis-generating.

At the dietary-pattern level, the principal interpretative advantage is that whole diets capture interactions among fiber, unsaturated fats, polyphenols, micronutrients, protein quality, food processing, and meal timing rather than isolating one component from its usual food matrix [2,53]. This systems perspective may explain why pattern-level associations are often more internally consistent than single-supplement findings, while still falling short of proving causality when the underlying studies are predominantly observational. Deficiency correction remains clinically justified, but routine supplementation in nutritionally replete individuals requires stronger and more reproducible trial evidence [36,46].

The gut microbiome provides a useful mechanistic framework that links these observations. A high-fiber, plant-rich, minimally processed diet can simultaneously increase SCFA substrate availability, reduce inflammatory burden, improve metabolic stability, and support microbial diversity [9,73]. These pathways

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Table 1: Summary of major nutritional exposures and current sleep evidence.

Exposure	Evidence strength and basis	Key sleep findings / effect size	Interpretation
Carbohydrate quality / glycemic index	Moderate — meta-analytic observational evidence; acute trial evidence is small.	Higher GI: OR 1.16 (95% CI 1.08–1.25); higher GL: OR 1.10 (1.01–1.20) for insomnia symptoms [21].	Favor carbohydrate quality and overall dietary context; do not use acute high-GI findings as a chronic recommendation.
Fiber / saturated fat	Limited — controlled feeding signal but few long-term trials.	Higher fiber associated with more slow-wave sleep; higher saturated fat/sugar with lighter, more fragmented sleep [22]; no robust pooled estimate.	Encourage fiber-rich foods within an overall high-quality diet; causal sleep effects need stronger trials.
Tryptophan	Moderate — pooled intervention evidence, but heterogeneous and relatively small.	WASO: SMD −1.08 (95% CI −1.89 to −0.28); other sleep outcomes inconsistent [25].	Food-first intake preferred; supplementation should not be presented as a universal hypnotic.
Protein timing	Emerging — small mechanistic/ intervention studies.	Alpha-lactalbumin can increase tryptophan availability; no robust pooled sleep effect estimate [26,27].	Treat protein distribution as part of dietary adequacy rather than a stand-alone sleep treatment.
Omega-3 LC-PUFAs	Moderate — meta-analysis of controlled trials.	Sleep efficiency: MD +1.88 percentage points (95% CI 1.00–2.77); sleep latency and total sleep duration not significant [31].	Potential supportive role, especially through fish-rich diets; effects are modest.
Magnesium	Limited — only three small RCTs; low/very-low certainty.	Sleep onset latency: MD −17.36 min (95% CI −27.27 to −7.44); total sleep time not significant [36].	Correct inadequate intake/deficiency; avoid routine insomnia claims in magnesium-replete adults.
Zinc	Limited — biological plausibility and multicomponent trials; isolated effect uncertain.	No reliable isolated pooled effect; combination trials cannot attribute benefit to zinc alone [37,38].	Food-first correction of inadequacy is more defensible than zinc as a hypnotic.
Iron / RLS	Strong in iron-deficient RLS; not generalizable to nonspecific insomnia.	Low iron status can worsen RLS-related sleep disruption; effect depends on deficiency and disorder-specific treatment context [39,40].	Assess and correct iron deficiency according to RLS guidance; avoid empirical iron in replete individuals.
Vitamin B6/B12 and other micronutrients	Limited — mechanistic plausibility exceeds direct sleep-trial evidence.	No consistent pooled evidence of sleep benefit in nutritionally sufficient adults [42–44,48–50].	Correct deficiency for general health; do not overstate sleep-specific supplementation benefits.
Vitamin D	Moderate for subjective sleep quality; limited for other outcomes.	PSQI: MD −2.33 points vs placebo (95% CI −3.09 to −1.57) [46]; duration/disorder outcomes inconsistent.	Consider baseline status and clinical context; not a universal sleep aid.
Mediterranean / high-quality patterns	Moderate — consistent meta-analytic observational evidence.	Mediterranean adherence: OR 0.86 (95% CI 0.79–0.93); high-quality diet: OR 0.66 (0.48–0.90) for insomnia symptoms [21].	Most defensible food-first pattern, while acknowledging residual confounding and reverse causality.
Ultra-processed / Western patterns	Moderate — observational meta-analytic evidence.	Higher UPF intake: OR 1.53 (95% CI 1.20–1.95) for insomnia [55].	Reduce UPFs as part of overall diet quality; causality is not fully established.
Plant-based diets	Limited — associations depend strongly on diet quality and nutrient adequacy.	No robust pooled independent effect of vegetarian/vegan status on sleep [57–59].	Focus on nutrient adequacy, processing level, and meal timing rather than dietary label alone.
Meal timing / time-restricted eating	Limited — small improvements in uncontrolled analyses; controlled analyses often null.	PSQI mean change −0.47 points (95% CI −0.80 to −0.15) in TRE-arm analyses; controlled trials showed no significant PSQI or duration benefit [60].	Daytime-aligned regularity may support circadian health; TRE is not an established insomnia therapy.
Caffeine	Strong — controlled experimental/ meta-analytic evidence using objective sleep outcomes.	TST −45.3 min (95% CI 29.0–61.5 reduction); sleep efficiency −7.0 points (4.0–10.0); SOL +9.1 min (3.8–14.4) [63].	Dose and timing matter; caffeine restriction may need to begin substantially earlier than bedtime.
Alcohol	Strong for REM disruption — controlled meta-analytic evidence.	REM onset +18.0 min (95% CI 8.8–27.1); REM duration −11.3 min (−20.8 to −1.8) [65].	Do not use alcohol as a sleep aid; REM disruption occurs even at relatively low doses.
Tart cherry / kiwifruit	Emerging — small heterogeneous trials and limited replication.	Potential improvements in sleep duration/efficiency, but no single robust pooled estimate across products/populations [66,67].	Optional food-based adjuncts with modest expectations; larger controlled trials are needed.
Herbal preparations	Emerging — small trials, heterogeneous preparations and outcome scales.	No comparable pooled estimate across chamomile/ valerian/passionflower/lemon balm/lavender evidence [68,69].	Avoid firm treatment claims; consider dose variability and drug interactions.
Probiotics / microbiome	Moderate for subjective PSQI in insomnia; limited for objective sleep and causal mediation.	Probiotics: PSQI MD −2.10 points (95% CI −3.86 to −0.34); TST and sleep efficiency not significant [72].	Potential adjunct in selected insomnia populations; strain specificity and high heterogeneity limit generalization.

may converge on sleep regulation without requiring that any single food exert a direct hypnotic effect [18,73] This systems-level view is consistent with the broader literature showing that dietary patterns often produce more reproducible health associations than isolated nutrients [21,53].

For clinical translation, dietary advice should complement rather than replace evidence-based evaluation and treatment of sleep disorders. Persistent insomnia may require cognitive behavioral

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therapy for insomnia (CBT-I), while symptoms of sleep apnea, RLS, circadian rhythm disorders, or parasomnias require disorder-specific assessment. Nutrition is most appropriately integrated as part of a broader sleep-health plan [5,39,40].

The bidirectional nature of the nutrition–sleep relationship also deserves greater emphasis. Short or fragmented sleep can alter leptin and ghrelin signaling, increase hunger, and favor energy-dense food choices, while irregular schedules may promote late eating and greater caffeine use [11,12,75]. Consequently, an observed association between a poor diet and poor sleep may represent a reinforcing cycle rather than a one-directional effect [13,22]. This has practical implications: dietary counseling may be less effective if chronic insomnia, shift-work-related circadian misalignment, or untreated sleep-disordered breathing continues to disrupt appetite regulation and daily routines. Conversely, sleep treatment may indirectly improve adherence to healthier dietary behaviors. Future intervention studies should therefore consider combined sleep-and-nutrition strategies instead of treating the two domains as independent lifestyle factors [13].

A practical hierarchy can be derived from the evidence (Figure.1). First, optimize overall diet quality using a Mediterranean-style or similarly minimally processed pattern [21,52,53]. Second, ensure adequate intake of protein, fiber, omega-3-rich foods, and micronutrients, with laboratory assessment and targeted supplementation when deficiency is clinically plausible [31,36,46]. Third, modify timing: avoid large late-night meals, maintain reasonably consistent eating times, and consider an earlier daytime eating window if compatible with the individual's schedule [17,60]. Fourth, address the exposures with the clearest adverse effects—particularly caffeine timing and alcohol near bedtime [63,65]. Finally, consider functional foods or supplements only as adjuncts, with realistic expectations about effect size and attention to interactions, dose, and comorbid disease [66,67].

A further challenge is the substantial interindividual variability in nutritional responses. Chronotype, habitual caffeine exposure, sex, age, genetic variation in metabolism, comorbid disease, medication use, and baseline nutrient status can all modify the effect of a dietary exposure on sleep [17,62]. The same caffeine dose, for example, may have little effect in one habitual user but meaningfully delay sleep in a slow metabolizer or a person with high caffeine sensitivity [62,63]. Similarly, magnesium or vitamin D supplementation is more biologically plausible in individuals with low baseline status than in those with adequate stores [36,46,47]. These observations support a move toward individualized recommendations based on clinical context and exposure timing rather than universal lists of foods or supplements that are presumed to improve sleep.

The principal strength of the current literature is the convergence of mechanistic, observational, and interventional data across several domains. Recent systematic reviews have also improved quantitative estimates for dietary patterns, caffeine, alcohol, omega-3 fatty acids, TRE, UPFs, vitamin D, magnesium, and tart cherries [21,31,36,46,52,55,60,63,65,66]. However, several limitations remain. Many associations are cross-sectional; intervention trials are often small and short; dietary and sleep assessments vary widely; baseline nutritional



Figure 1: Practical hierarchy of nutritional strategies for supporting sleep health.

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Table 2: Evidence-based clinical interpretation of nutrition–sleep findings.

Domain	What the evidence supports	Main limitations	Practical implication
Overall diet quality	Mediterranean-style and other high-quality patterns are consistently associated with better sleep and fewer insomnia symptoms.	Most evidence is observational; lifestyle clustering and reverse causality remain.	Prioritize a minimally processed, plant-rich, nutrient-dense pattern rather than isolated "sleep foods."
Deficiency correction	Iron in RLS, vitamin D in deficiency, and possibly magnesium in selected older adults have the clearest rationale when low status is present.	Baseline status is often unmeasured; supplement trials are small.	Test and correct clinically relevant deficiencies; avoid routine megadosing.
Stimulants/sedatives	Caffeine and alcohol alter objective sleep in controlled studies.	Individual metabolism and tolerance vary.	Move caffeine earlier; do not use alcohol as a sleep aid.
Meal timing	Regular daytime-aligned intake is circadianly plausible; early TRE may be modestly favorable.	Controlled sleep benefits are inconsistent.	Avoid large late meals; use TRE for metabolic/circadian goals rather than as a primary insomnia treatment.
Microbiome	Dietary fiber, polyphenols, and unsaturated fats support microbial metabolites plausibly linked to sleep.	Human mediation evidence and strain-specific intervention data remain limited.	Support microbiome health through diet; do not overstate probiotic sleep claims.
Functional foods	Tart cherries and kiwifruit have small positive trials.	Small samples, variable products, limited replication.	Optional adjuncts with modest expectations, not substitutes for sleep-disorder treatment.

status is not consistently measured; and sleep disorders are frequently not phenotyped using objective methods [21,36,56].

Another limitation is publication and product heterogeneity. Supplements labeled with the same nutrient can differ in dose, formulation, co-ingredients, and bioavailability. Probiotics are strain specific [72]. Tart-cherry products vary in concentration [66]. Caffeine exposure differs by beverage size and preparation [63]. These sources of heterogeneity make simple public-facing claims difficult to defend. Future evidence synthesis should therefore emphasize standardized exposure reporting and clinically interpretable effect sizes.

From a public-health perspective, the current evidence also supports greater integration between sleep medicine and nutritional assessment [5,22]. Routine questions about caffeine timing, alcohol use, late-night eating, overall dietary quality, and possible micronutrient deficiency can be incorporated into the clinical evaluation of patients reporting poor sleep at relatively low cost [17,63,65]. Likewise, nutrition studies should treat sleep duration, quality, timing, and sleep-disorder symptoms as relevant covariates rather than background characteristics. Such cross-disciplinary assessment may improve both clinical care and research validity because it recognizes that sleep and diet share common behavioral, metabolic, and circadian determinants [13,22]. However, nutrition-based recommendations should remain proportionate to the evidence: they are best positioned as modifiable components of comprehensive sleep health, not substitutes for established therapies such as CBT-I or the diagnosis and treatment of obstructive sleep apnea and RLS [39,40].

Future studies should prioritize: (1) adequately powered RCTs of whole dietary patterns with objective multi-night sleep measurement; (2) prospective cohorts with repeated diet and sleep assessments to clarify directionality; (3) trials stratified by baseline nutrient status, chronotype, sex, age, and sleep-disorder phenotype; (4) studies combining actigraphy or PSG with metabolomics and microbiome profiling to test mediation pathways; (5) standardized reporting of meal timing, caffeine dose and timing, alcohol dose, and supplement formulation; and (6) pragmatic studies evaluating whether nutrition-focused interventions add clinically meaningful benefit when combined with established sleep therapies [73,76,77].

Conclusions

Nutrition is a modifiable component of sleep health, but the relationship is complex, bidirectional, and strongly influenced by overall lifestyle and clinical context. Current evidence most strongly supports a pattern-based approach: Mediterranean-style or similarly high-quality diets are associated with better sleep, whereas ultra-processed dietary patterns are associated with poorer outcomes. Caffeine and alcohol have the most reproducible adverse effects on objective sleep and should be addressed explicitly when sleep complaints are present.

Specific nutrients—including tryptophan, omega-3 fatty acids, vitamin D, magnesium, zinc, and iron—participate in pathways relevant to sleep physiology, but supplementation is not uniformly beneficial. The clearest clinical rationale is to correct documented or likely nutritional deficiencies and to use food-first strategies whenever possible. Meal timing and the gut microbiome are important emerging domains, although controlled evidence is not yet sufficient for universal prescriptions. Overall, dietary optimization should be viewed as a foundation that supports sleep health and complements, rather than replaces, established diagnostic and therapeutic approaches for sleep disorders.

Author contributions (CRediT)

Gabriela Georgieva Panayotova: Conceptualization,

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Methodology, Investigation, Writing – review & editing, Visualization, Project administration.

Vaisakhkrishna Harikumar: Investigation, Writing – original draft.

Anjali Harikumar: Investigation, Writing – original draft.

Antoniya Hachmeriyan: Conceptualization, Methodology, Supervision, Writing – review & editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the work.

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