REVIEW

Effects of Diet on Sleep Quality[1,2]

Marie-Pierre St-Onge,* Anja Mikic, and Cara E Pietrolungo

Institute of Human Nutrition and Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, NY

ABSTRACT

There is much emerging information surrounding the impact of sleep duration and quality on food choice and consumption in both children and adults. However, less attention has been paid to the effects of dietary patterns and specific foods on nighttime sleep. Early studies have shown that certain dietary patterns may affect not only daytime alertness but also nighttime sleep. In this review, we surveyed the literature to describe the role of food consumption on sleep. Research has focused on the effects of mixed meal patterns, such as high-carbohydrate plus low-fat or low-carbohydrate diets, over the short term on sleep. Such studies highlight a potential effect of macronutrient intakes on sleep variables, particularly alterations in slow wave sleep and rapid eye movement sleep with changes in carbohydrate and fat intakes. Other studies instead examined the intake of specific foods, consumed at a fixed time relative to sleep, on sleep architecture and quality. Those foods, specifically milk, fatty fish, tart cherry juice, and kiwifruit, are reviewed here. Studies provide some evidence for a role of certain dietary patterns and foods in the promotion of high-quality sleep, but more studies are necessary to confirm those preliminary findings. *Adv Nutr* 2016;7:938–49.

Keywords: diet, cherry, kiwi, dairy, carbohydrate, glycemic index, sleep, REM

Introduction

Because studies have proposed a relation between sleep duration and obesity (1–3), there has been much interest in assessing the impact of sleep on energy intakes. Studies have shown that short sleepers have higher energy intakes, notably from fat (4, 5) and snacks (6), than do normal sleepers. NHANES data in the United States showed that short sleepers, generally defined as those who sleep <7 h/night, consume a smaller variety of foods, with lower protein, carbohydrate, fiber, and fat intakes relative to normal sleepers who report 7–8 h of sleep/night (7). These data are corroborated by clinical intervention studies that also showed greater snack intakes during periods of sleep restriction relative to habitual sleep in normal sleepers (8). Fat was also highlighted as a macronutrient of choice during periods of sleep restriction relative to habitual sleep (9, 10).

Of note, however, is that epidemiologic studies cannot address causality or the direction of the relation between variables. Therefore, although those studies reported a link between sleep and diet, it is unknown whether it is sleep that affects dietary intakes or dietary intakes that affect sleep. In this review, we sought to determine from clinical intervention studies whether, and how, dietary intakes could influence sleep variables, specifically duration, efficiency, and architecture. We mainly focus on intervention studies that examined the effects of diets, meals, or foods on sleep at night, not daytime napping, but we also report on more general epidemiologic findings of associations between diet and sleep quality. We did not include studies of single micronutrients or dietary supplements.

Unlike sleep duration, which is clearly defined by the amount of sleep one gets at night, sleep quality can be defined in different ways. By using objective measures of sleep, such polysomnography, sleep quality can be characterized by the amount of slow wave sleep (SWS)[3] and rapid eye movement (REM) sleep one gets at night. These 2 stages of sleep occur with greater duration as the night progresses (11). SWS is deep sleep and has a restorative function (12), whereas both REM and SWS function toward memory consolidation (11, 13). Of relevance to this review, we have shown that these stages of sleep were inversely associated with fat and carbohydrate intakes (14). By using polysomnography and actigraphy, sleep quality can be defined by sleep efficiency (SE), or the amount of time in bed spent asleep, as well as sleep-onset latency (SOL), the amount of time one takes to fall asleep at night. Low SE (generally <85%) and long

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*To whom correspondence should be addressed. E-mail: ms2554@cumc.columbia.edu.

[3] Abbreviations used: GI, glycemic index; HC, high carbohydrate; HF, high fat; LC, low carbohydrate; LCNAA, large-chain neutral amino acid; LF, low fat; NREM, nonrapid eye movement; REM, rapid eye movement; SE, sleep efficiency; SOL, sleep-onset latency; SWS, slow wave sleep; TST, total sleep time; WASO, wake after sleep onset.

SOL (>20–30 min depending on age) typically characterize poor sleep. Finally, subjective measures of sleep quality can be obtained by questionnaire. Typically, the Pittsburgh Sleep Quality Index questionnaire is used.

Sleep duration and quality have been associated with obesity, diabetes, hypertension, and cardiovascular disease risk in cross-sectional and longitudinal studies (11). An excellent review in this Journal covered some mechanistic explanations for this association and provides recommendations for nutrition professionals with regard to sleep hygiene and its importance in nutrition counseling (11).

Dietary Patterns and Sleep Quality

Epidemiologic findings

Associations between sleep quality and dietary patterns were recently reported in a cross-sectional study (15) in female Japanese workers who responded to lifestyle questionnaires. A high intake of confectionary and noodles was associated with poor sleep quality, as evidenced by a high global Pittsburg Sleep Quality Index score, whereas a high intake of fish and vegetables was associated with good sleep quality. A significant trend toward worse sleep quality with increasing carbohydrate intake was found. The quality of carbohydrate seemed to be more important than its quantity in mediating this association. Poor sleepers with the highest carbohydrate intake consumed more confectionary and noodles than rice than did good sleepers with a similarly high carbohydrate intake. Moreover, frequent consumption (≥1 time/mo) of energy drinks and sugar-sweetened beverages was associated with poor sleep quality. Other eating patterns indicative of poor dietary habits were also related to sleep quality. For example, skipping breakfast and eating irregularly were strongly associated with poor sleep quality. Although relations between sleep quality and dietary patterns were observed, the directionality of the findings cannot be established from this study. Furthermore, poor-quality sleepers were also short sleepers, thus making sleep duration a likely confounding variable that was not taken into account.

Other epidemiologic studies have found associations between disordered sleep and diet (16–18). Tanaka et al. (16) reported a relation between macronutrient intakes and insomnia symptoms in a cross-sectional analysis of non–shift workers who responded to a brief diet history questionnaire. Low protein intake (<16% of energy from protein) was associated with poor quality of sleep and marginally associated with difficulty initiating sleep, whereas high protein intake (>19% of energy from protein) was associated with difficulty maintaining sleep. Low carbohydrate intake (<50% of energy from carbohydrate) was marginally associated with difficulty maintaining sleep. When stratified by sex, these associations were significant in men but not in women.

Similar results were found with respect to the association between carbohydrate intake and sleep quality in men (17). Individuals with disordered sleep (insomnia, obstructive sleep apnea, or a combination of the 2) assessed by questionnaires reported lower total carbohydrate intakes than did normal-weight individuals who were free from sleep disorders. Overweight individuals with insomnia also had lower carbohydrate intakes than did healthy overweight counterparts. In addition, they had higher fat intakes than individuals who were free from sleep disorders.

The Mediterranean diet was associated with sleep quality in older adults (18). On the basis of self-reported questionnaires evaluating sleep quality, lifestyle factors, and dietary intake, the Mediterranean diet was inversely associated with insomnia symptoms (difficulty initiating sleep, difficulty maintaining sleep, early morning awakening) in women but not in men. Data from the 2007–2008 NHANES showed that difficulty maintaining sleep was associated with lower food variety and adhering to a special diet; however, this was no longer significant after adjusting for covariates (19). Increased caloric intake was associated with daytime sleepiness.

The epidemiologic studies that reported associations between dietary patterns and sleep quality are informative. In general, those studies indicate higher fat intakes with sleep disorders (17) and that following a Mediterranean dietary profile is associated with fewer insomnia symptoms in women (18). Information on the association between carbohydrate intakes and sleep quality is conflicted (15–17), with studies reporting low intakes in those with insomnia symptoms (16, 17) but high intakes of sweets (15). This would suggest that carbohydrate quality may be important to consider when examining the association between diet and sleep quality. However, epidemiologic studies are limited by an unclear direction of the associations and self-reported data. Clinical trials that investigated the effect of individual macronutrients on sleep architecture are more elucidative.

Experimental findings

High-carbohydrate diet. There is a substantial body of evidence to indicate a role of carbohydrate intake on sleep indexes (**Table 1**). Both high-carbohydrate (HC) and low-carbohydrate (LC) diets are associated with changes in sleep architecture (20–25). Carbohydrate manipulation has primarily been shown to affect REM sleep and SWS; however, non-REM (NREM) sleep, SOL, and REM-onset latency have also been affected.

Phillips et al. (20) showed that HC and LC diets have opposite effects on SWS. In this study, healthy men were randomly assigned to consume a controlled diet, either LC plus high fat [(HF) LC/HF] or HC plus low fat [(LF) HC/LF] for a period of 2 d after 2 d of a lead-in balanced diet. The LC/HF and HC/LF diets provided 100 and 600 g carbohydrates and 255 and 33 g fat, respectively. The lead-in diet contained 350 g carbohydrates and 140 g fat. Diets were designed to maintain weight and meals were administered at fixed times. SWS significantly decreased with the HC/LF diet relative to the LC/HF diet and the lead-in diets. REM sleep significantly increased with both intervention diets relative to the lead-in diet, with a significantly greater increase after consumption of the HC/LF diet. Similarly, stage 1 sleep was reduced with both diets compared with the lead-in diet.

Yajima et al. (21) found similar changes in SWS after the consumption of an HC test meal. In a similar fashion, healthy men underwent a 1-d randomized crossover intervention with

TABLE 1 Summary of clinical studies that investigated the effect of dietary patterns on sleep architecture[1]

Study (ref)	Diet pattern	Subjects	Duration	Methods	Treatment group results[2]
Phillips et al. (20)	HC/LF diet vs. LC/HF diet	8 healthy men	4 d	Days 1–2: control diet (350 g carbohydrate, 140 g fat, 75 g protein) Days 3–4: HC/LF diet (600 g carbohydrate, 33 g fat, 75 g protein) or LC/HF diet (100 g carbohydrate, 225 g fat, 75 g protein)	SWS: lower with the HC/LF diet (97.8 min) and higher with the LC/HF diet (117.2 min) vs. the control diet (115.5 min) REM: higher with the HC/LF diet (136.9 min) vs. the LC/HF (122.1 min) and control (103.6 min) diets NREM 1: lower with both the HC/LF (319.5 min) and LC/HF (331.5 min) diets vs. the control diet (342.2 min)
Yajima et al. (21)[3]	HC vs. HF meals	10 healthy men	1 d	HC test meal: dinner consumed at 2000 (10% protein, 10% fat, 80% carbohydrate) HF test meal: dinner consumed at 2000 (78% fat, 10% protein, 12% carbohydrate)	SWS: decreased during sleep cycle 1 with the HC diet vs. the HF diet
Lindseth et al. (22)	High-protein vs. HF vs. HC diets	44 healthy young adults (19–22 y old)	4 d	High-protein diet (56% protein, 22% carbohydrate, and 22% fat) HC diet (56% carbohydrate, 22% protein, 22% fat) HF diet (56% fat, 22% carbohydrate, 22% protein) Control diet (50% carbohydrate, 35% fat, 15% protein)	Wake episodes: decreased with the high-protein diet (13.5 times) vs. the control diet (16.7 times) (between-group) SOL: lower with the HC diet (9.1 min) vs. the control diet (13.9 min)
Afaghi et al. (23)	High- vs. low-GI	12 healthy men (18–35 y old)	1 d	767 kcal/meal (8% protein, 1.6% fat, 90.4% carbohydrate) Low-GI diet [Mahatma rice (GI = 50) with meal 4 h before bedtime] High-GI diet 1 [jasmine rice (GI = 109) with meal 4 h before bedtime] High-GI diet 2 [jasmine rice (GI = 109) with meal 1 h before bedtime]	SOL: lower with the high-GI diet at 4 h before bedtime (9.0 ± 6.2 min) vs. both low-GI diet at 4 h before bedtime (17.5 ± 6.2 min) and high-GI diet at 1 h before bedtime (14.6 ± 9.9 min)
Afaghi et al. (24)	Very LC	14 healthy men (18–35 y old)	5 d	Control phase [3 d of mixed meals (15.5% protein, 12.5% fat, 72% carbohydrate) with 1 evening mixed test meal[4]] Acute phase [night 3: very LC test meal[4] (2400 kcal; 38% protein, 61% fat, <1% carbohydrate)] Ketosis phase (2 d of very LC diet)	REM: percentage of TST lower during very LC acute (17.6% ± 5.3%) and very LC ketosis (17.7% ± 5.4%) phases vs. control (21.4% ± 6.3%) SWS: higher during very LC acute (83.3 ± 33.8 min) and very LC ketosis (80.4 ± 628.0 min) phases vs. control (66.2 ± 30.1 min)
Kwan et al. (25)	LC	6 healthy young women (20–23 y old)	2 wk	Week 1: weighing and recording habitual diet Week 2: isoenergetic diet of 50-g/d carbohydrate restriction	REM: onset latency increased from 66 ± 8 min to 111 ± 38 min
St-Onge et al. (9)	Controlled vs. ad libitum food intake	26 healthy adults (30–45 y old)	1 d	Habitual sleep phase: 9 h/night in bed (2200–0700) Test day: ad libitum food intake	SWS: lower during the ad libitum food intake period (24.6 ± 12.8 min) than during controlled intake period (29.3 ± 13.9 min) SOL: higher during the ad libitum food intake period (29.2 ± 23.1 min) than during controlled intake period (16.9 ± 11.1 min)

(Continued)

TABLE 1 (*Continued*)

Study (ref)	Diet pattern	Subjects	Duration	Methods	Treatment group results[2]
Crispim et al. (26)[3]	Ad libitum food intake	52 healthy adults (19–45 y old)	3 d	Test days: ad libitum food intake recorded by using food diary	Men: NREM 2: negatively correlated with nocturnal fat intake SE: negatively correlated with nocturnal fat intake REM: negatively correlated with nocturnal fat intake SOL: negatively correlated with nocturnal fat intake WASO: negatively correlated with nocturnal fat intake Women: SOL: positively correlated with nocturnal caloric, protein, carbohydrate, and fat intake SE: negatively correlated with nocturnal caloric, carbohydrate, and fat intake REM: negatively correlated with nocturnal fat intake
Driver et al. (27)	High-energy meal vs. evening fast vs. control meal	7 healthy men (20–24 y old)	1 d	Fast: evening fast beginning at 1300; maximum energy intake of 38 kcal consumed as fruit juice and water Control meal: administered at 2100 with a macronutrient ratio of 12:26:61 for fat, protein, and carbohydrate High-energy meal: administered at 2100 with a macronutrient ratio of 37:21:42 for fat, protein, and carbohydrate, with double the energy content of the control meal	No effect of evening fast (10 h) or high-energy evening meal on sleep architecture
Lieberman et al. (28)	Calorie deprivation	27 healthy young adults	2 d	All diets composed of hydrocolloid gels Carbohydrate diet: starch and maltodextrin gel Carbohydrate+fat diet: starch, maltodextrin, and polyunsaturated lipid gel Calorie deprivation: hydrocolloid-based gel with artificial sweeteners and flavors	No effects of 2-d calorie deprivation on sleep
Karacan et al. (29)	Calorie deprivation	11 healthy men (22–25 y old)	3 d	Day 1: normal food intake with dinner meal as the last meal before fast Days 2–3: fasting days (no food intake)	REM: lower number of REM episodes (3.49 ± 0.9 vs. 4.4 ± 0.5 episodes) and higher percentage of stage 4 REM sleep (15% ± 7% vs. 11% ± 6%) on day 3 vs. day 1; higher percentage of stage 4 REM sleep (15% ± 7% vs. 10% ± 7%) and lower percentage of stage 2 REM sleep (49% ± 9% vs. 53% ± 7%) on day 3 vs. day 2

[1] GI, glycemic index; HC, high carbohydrate; HF, high fat; LC, low carbohydrate; LF, low fat; NREM, nonrapid eye movement; NREM 1, nonrapid eye movement stage 1; NREM 2, nonrapid eye movement stage 2; ref, reference; REM, rapid eye movement; SE, sleep efficiency; SOL, sleep onset latency; SWS, slow wave sleep; WASO, wake after sleep onset.
[2] Only significant results are reported, $P < 0.05$. Results are shown relative to the control group unless otherwise noted.
[3] Numerical data not provided.
[4] Test meals 4 h before bedtime.

either an HC or an HF evening test meal to investigate their effects on sleep architecture. Participants were prescribed either an HF (78% fat, 10% protein, 12% carbohydrate) or an HC (10% protein, 10% fat, 80% carbohydrate) test meal to be eaten at 2000. Breakfast and lunch on the test day were the same for each intervention and provided 13–15% protein, 19–24% fat, and 60–63% carbohydrate. SWS decreased within the first sleep cycle after the HC meal compared with the HF meal. However, there was no difference in sleep architecture between interventions over the entire sleep period. The authors suggested that the reduction in SWS observed during the first sleep cycle was related to the degree of carbohydrate oxidation. Carbohydrate oxidation was higher after the HC meal than after the HF meal, especially during the first half of the sleep episode when SWS was markedly reduced with the HC meal. In addition, they showed that carbohydrate oxidation was highest during REM sleep and lowest during SWS (21), possibly indicating a greater reliance on carbohydrate for energy during REM sleep. However, this study was limited by the absence of a control (balanced) meal. Because of this, it is not possible to determine whether SWS was, in fact, increased with the HF meal or whether it was reduced with the HC meal. Moreover, the clinical significance of a change in sleep architecture in one sleep cycle, but not over the entire night, is unknown.

Another study (30) investigated the effects of a pre-bedtime test meal on sleep architecture in men. Participants underwent a 3-night intervention consisting of an HC, an LC, or a zero-carbohydrate snack administered 45 min before bedtime. The HC snack consisted of a glucose drink and fried potatoes (521 kcal or 130 g carbohydrate) and the LC snack consisted of crispbread, salad, and butter (188 kcal or 47 g carbohydrate). Both the HC and LC snacks were similar in protein and fat content but differed in carbohydrate content. The carbohydrate-free snack consisted of a methyl-cellulose supplement and contained no energy. Participants were allowed 8.5 h of sleep/night. NREM sleep decreased and the number of REM periods increased during the HC relative to the no-carbohydrate treatment over the entire sleep period. SWS decreased during the carbohydrate-free treatment relative to the LC treatment over the entire sleep period. During only the first half of the sleep period, REM sleep and stage 3 sleep increased during the HC treatment. Persistent effects were observed on recovery nights after the 3-d intervention period. During the post-HC conditions, stage 4 sleep decreased and the number of REM periods increased relative to the other 2 treatments, whereas stage 3 sleep increased relative to the no-carbohydrate condition over the entire sleep period. REM latency increased after the LC relative to the HC conditions over the entire sleep period. Consistent with the findings of Phillips et al. (20), most of the changes in sleep architecture occurred during the HC condition, with a trend toward higher REM sleep and lower NREM sleep, with the exception of stage 3 sleep. Although the direct mechanisms mediating these changes are unclear, the authors proposed that the effect of the HC condition on changes in sleep stages is related to increased serotonin synthesis.

Lindseth et al. (22) investigated the effect of individual macronutrients on sleep indexes in adults with the use of a controlled-feeding crossover study design. The dietary interventions included a high-protein diet (56% of energy from protein, 22% from carbohydrate, and 22% from fat), an HC diet (56% of energy from carbohydrate, 22% from protein, and 22% from fat), an HF diet (56% of energy from fat, 22% from carbohydrate, and 22% from protein), and a control diet (50% of energy from carbohydrate, 35% from fat, and 15% from protein) that were each consumed for 4 d. Sleep was monitored continuously throughout each 4-d intervention period by using actigraphy. A significant effect of diet on the number of wake episodes and SOL was observed. The consumption of the high-protein diet decreased the number of wake episodes compared with the control diet, and SOL was significantly lower after the HC diet than after the control diet.

Glycemic index. The glycemic index (GI) of carbohydrates has also been studied as a dietary factor related to sleep architecture. Afaghi et al. (23) investigated the effects of both GI and meal timing on sleep architecture in men. Sleep was measured on 3 separate test nights, which differed in the glycemic index of the pre-bedtime meal: either a low-GI (GI = 50) or a high-GI (GI = 109) meal was consumed 4 h before bedtime or a high-GI meal was consumed 1 h before bedtime. SOL was significantly lower after the high-GI meal consumed 4 h before bedtime than after both the low-GI meal and the high-GI meal consumed 1 h before bedtime. Consistent with these findings, subjective ratings of sleepiness were significantly higher after the high-GI meal ingested 4 h before bedtime.

LC diet. Afaghi et al. (24) also investigated the effects of a very LC diet on sleep architecture in men. The intervention consisted of a familiarization phase with 1 evening control test meal for 3 d, an acute intervention phase (1 meal), and a longer-term ketosis phase of 2 d. The familiarization phase consisted of mixed, balanced meals (15% of energy from protein, 25% from fat, and 60% from carbohydrate) provided on day 1 and for breakfast and lunch on day 2. The control test meal, altered to resemble an HC/LF diet (15.5% of energy from protein, 12.5% from fat, 72% from carbohydrate), was administered on the evening of day 2. The acute phase took place on the third night, after consuming the familiarization diet for breakfast and lunch. The acute phase consisted of an evening test meal (38% of energy from protein, 61% from fat, <1% from carbohydrate). The ketosis phase was followed over the next 2 d with maintenance of the very LC diet. Each participant was maintained on 2400 kcal/d for the 5-d intervention period, and test meals were administered 4 h before bedtime. Participants were permitted to sleep at their discretion, and sleep was recorded by using polysomnography. REM sleep was reduced and SWS was increased during both the acute and ketosis phases relative to the control phase. The arousal index was significantly increased during sleep stages 1 and 2 after the very LC acute and ketosis phases compared with after the control phase. In addition, there was a trend toward improved SE after the acute phase ($P = 0.08$) but not after the ketosis phase.

In another study, Kwan et al. (25) investigated the effect of an LC diet, 50 g/d for 1 wk, on sleep architecture in women. REM-onset latency increased after the LC diet relative to the prestudy habitual diet.

Mixed meals. The effect of various macronutrient intakes on sleep architecture was assessed by St-Onge et al. (9). Participants consumed a fixed diet that provided 31% of energy from fat, 53% from carbohydrate, and 17% from protein for 4 d. On day 5, participants self-selected their food intake. On that night, SWS was lower and SOL was longer than sleep measured after the fixed diet. Higher fiber intakes on the ad libitum day were associated with more SWS and less time spent in stage 1 sleep. A higher percentage of energy consumed from saturated fat was associated with less time spent in SWS. In addition, greater sugar and nonsugar, nonfiber carbohydrate intakes were associated with more wake bouts during the sleep episode. These associations indicate that higher saturated fat and lower fiber intakes may produce less SWS, more nighttime arousals, and a reduction in overall sleep quality.

Crispim et al. (26) similarly investigated the effects of ad libitum food intake on sleep architecture. Over 3 nonconsecutive days, subjects recorded their food intake in a food diary and reported to the sleep laboratory for polysomnography. Nocturnal food intake (30–60 min before bedtime), but not total daily intake, was correlated with several sleep variables and differed by sex. In men, stage 2 sleep, REM sleep latency, SOL, and wake after sleep onset (WASO) were positively correlated with fat intake at night. In addition, fat intake at night was negatively correlated with SE and REM in men. In women, positive associations for evening intake included the following: SOL and energy, protein, carbohydrate, and fat intakes; REM sleep latency and energy, carbohydrate, and fat intakes; stage 2 sleep and energy, carbohydrate, and fat intakes; and WASO and energy and fat intakes. Negative associations for evening intake included REM sleep and fat intake and SE and energy, carbohydrate, and fat intakes in women. Overall, the results of this study confirmed that diet quality, particularly closer to bedtime, influences sleep architecture. Nocturnal eating, considered in this study to be any food intake 30–60 min before bedtime, was shown to negatively influence sleep quality, with a greater effect in women than in men (26). This effect was proposed by the authors to be mediated by postprandial physical discomfort and reduced digestive activity; however, this was not confirmed.

The studies to date point to an effect of carbohydrate intake on sleep, albeit with mixed results. Some found reduced SOL with the consumption of a higher carbohydrate diet (22, 23) but others reported a trend for greater SE after an acute intake of a very LC meal (24). The findings from these studies support the idea that dietary carbohydrate intake or pre-bedtime meal also influence sleep architecture, particularly REM and SWS. The consumption of an LC diet appears to reduce REM sleep while increasing SWS (20, 24), with the consumption of an HC diet having the opposite effect. The effects of an HC diet on REM and SWS have been linked to fuel utilization during the different sleep stages (21). Nocturnal energy metabolism has been shown to differ between the different sleep stages (31). Energy expenditure and fat oxidation decline in the first 4 h of the sleep period and subsequently remain stable for the following 4 h of the night in healthy men. Carbohydrate oxidation was higher during REM sleep than in NREM sleep and highest in the last hour of the sleep episode before waking. The difference in carbohydrate oxidation between sleep stages was greatest between sleep stages 3 and 4 and REM sleep, indicating a higher energy demand for REM sleep. Therefore, an HC meal or diet could enhance nocturnal carbohydrate utilization and promote REM sleep.

Although it cannot be confirmed, it has been hypothesized that carbohydrate oxidation also suppresses SWS (21, 31), which would support the findings of reciprocal changes in REM and SWS due to carbohydrate manipulation (20, 24). It was previously reported that an HC diet reduces growth hormone secretion in men, but not in women, after a 10-d dietary intervention (32). Because growth hormone has been linked to SWS (20, 24), it is possible that a reduction in SWS with an HC diet may be mediated by a diet-induced reduction in growth hormone secretion. However, additional research is warranted to confirm this hypothesis.

The effect of an HC diet on SOL has been linked to elevated postprandial insulin and Trp response (23). Trp, a precursor of serotonin (23), enters the brain in a competitive manner with large-chain neutral amino acids (LCNAAs) (33). An HC diet, low in protein, has been shown in animal models to elevate brain Trp concentrations relative to higher-protein diets (33). When the concentration of Trp is higher than that of LCNAAs, its entry into the brain is favored and serotonin production is upregulated (34, 35), which would promote sleep (24). Because the Trp-to-LCNAA ratio is affected by dietary carbohydrate intake (33, 36), it is possible that changes in sleep architecture as a result of carbohydrate manipulation are mediated by the Trp-to-LCNAA ratio. An HC diet or meal would increase the Trp-to-LCNAA ratio and promote sleep through increased serotonin production (33, 34). Conversely, an LC diet would result in a low ratio of Trp to LCNAA, thus limiting serotonin production and prolonging SOL (25). With the consumption of an HC diet, the resulting higher postprandial insulin enhances the Trp-to-LCNAA ratio by facilitating uptake of LCNAA by muscle, further promoting Trp entry into the brain and enabling serotonin production (23). In support of this, a correlation between plasma glucose and insulin, and peak Trp:LCNAA has been reported (36). Therefore, an HC diet, especially one with a high GI, would promote a higher Trp-to-LCNAA ratio and have a greater serotonergic effect (36). Moreover, it has been reported that the Trp-to-LCNAA ratio peaks between 2 and 4 h after the ingestion of an HC meal (36), which likely accounts for shorter SOL after the consumption of an HC or high-GI meal 4 h compared with 1 h before bedtime (23). This proposed mechanism must be explored further, however, because the Trp-to-LCNAA ratio was not measured

in the studies reported in the current review. However, urine 6-sulfatoxymelatonin, a metabolite of melatonin, was highest after the consumption of the high-GI meals (23). This finding may also be related to the plasma concentration of Trp (23). Therefore, an increase in plasma Trp after a high-GI meal may influence both melatonin and serotonin, thus promoting sleep onset. However, subsequent sleep quality, such as duration of SWS and arousals during sleep, may be adversely affected by HC intakes, particularly of simple sugars.

The fat content of the meal has been suggested to mediate the observed changes in REM and SWS (24) due to an LC diet. This effect may be mediated through the postprandial release of cholecystokinin, a satiety hormone released by the duodenum after an HF meal (24). The role of cholecystokinin in mediating changes in sleep architecture in humans has not yet been defined; however, an animal study showed that injection of cholecystokinin into rats promoted SWS and NREM sleep (37). In humans, both subjective ratings of fatigue and cholecystokinin concentrations were significantly higher after an HF/LC meal than after an LF/HC meal in 18 healthy adults (25). Furthermore, cholecystokinin was reported to be predictive of and positively correlated with fatigue (25). Although clinical trials directly investigating the effect of cholecystokinin concentrations on sleep architecture are lacking, the association between cholecystokinin and fatigue in response to an HF/LC meal suggests that sleep architecture may be mediated by this satiety hormone.

Calorie Restriction and Sleep Quality: Experimental Findings

In a study by Driver et al. (27), the effect of short-term (10 h) calorie restriction on sleep indexes was investigated. Participants consumed food ad libitum until 1300 when they reported to the laboratory. A test meal was provided at 2100. The test meals consisted of no meal, a control meal, or a high-energy meal. The fast (no meal) allowed only the consumption of fruit juice and water, with a maximum energy intake of ~38 kcal. The control meal had a macronutrient ratio of 13:26:61 for fat:protein:carbohydrate and provided ~1370 kcal, whereas the high-energy meal had a ratio of 37:21:42 with double the energy content, resembling a higher-fat meal. Bedtime was set between 2250 and 2306, and sleep was monitored by polysomnography. The authors found no effect of diet on sleep variables.

Another study investigated the effects of 2.5 d of calorie deprivation on sleep, cognition, activity, and blood glucose concentrations (28). Participants underwent 3 test periods: near-complete fasting, carbohydrate only, and carbohydrate+fat diets. Diets were composed exclusively of hydrocolloid gels to maintain blinding. The carbohydrate diet (940 kcal/meal) consisted of a starch and maltodextrin gel; the carbohydrate+fat diet (940 kcal/meal) consisted of starch, maltodextrin, and polyunsaturated lipid gel; and the calorie-deprivation diet (61 kcal/meal) consisted of an artificial sweetener and artificial flavor gel. There was no effect of dietary intervention on sleep variables or other outcome measures.

The short duration of the above studies suggests that prolonged calorie restriction may be required to influence sleep architecture. In fact, prolonged fasting (60–67 h) decreased the number of REM episodes but increased the percentage of REM sleep compared with baseline (29). A comparison of fasting periods of 30–37 h and 60–67 h showed an increase in the percentage of stage 4 REM sleep with a compensatory decrease in stage 2 REM sleep.

Although Karacan et al. (29) were the only authors, to our knowledge, to report an effect of calorie restriction on sleep variables, it is surprising that there was no effect of the other dietary interventions (27, 28). In the study performed by Driver et al. (27), neither the control meal nor the high-energy meal resulted in the acute changes in sleep architecture that had been previously shown (23). Similarly, the study by Lieberman et al. (28) failed to show an effect of the carbohydrate or carbohydrate+fat gel preparations on sleep. It is possible that a longer adaptation period to the dietary interventions is required for changes in sleep variables to be observed, although acute changes in sleep have been reported after one evening test meal (23). Overall, it is reasonable to suggest that calorie restriction influences sleep architecture over longer durations; nevertheless, research is limited on this topic and further investigation is warranted.

Sleep-Promoting Foods and Sleep Quality: Experimental Findings

Despite the availability of anecdotal evidence, scientific research on the effects of various foods on sleep enhancement is limited (**Table 2**). Daily incorporation of sleep-promoting foods, such as milk, fatty fish, cherries, and kiwifruit, has been studied for their potential benefits for immediate and acute sleep improvement without large changes in dietary patterns.

Milk. The first studies to examine the sleep-inducing effects of a specific food date to the 1970s, when Horlicks, a malted milk drink, was tested. Southwell et al. (38) used time-lapse cinematography to record sleep movements after the consumption of 350 mL warm water, 350 mL warm milk with 5 teaspoons Horlicks powder, or no beverage (control). Participants with no history of sleep disorders consumed the drink ~30 min before bedtime, which was fixed at midnight. The authors reported fewer small movements during sleep after consumption of the Horlicks drink, particularly from 0400 to 0700, than after consumption of water and the control. In support of these findings, another study (39) also found that young adults experienced fewer movements during sleep in the latter half of the night after the consumption of a Horlicks drink 30 min before bedtime. The study used polysomnography recordings to assess the sleep quality of healthy young and middle-aged adults after the consumption of Horlicks relative to an inert capsule. Compared with the younger participants, the older adults experienced increased total sleep time (TST) and greater sleep continuity after the consumption of Horlicks.

The effect of Horlicks on sleep quality and duration appears to be partially mediated by age. Aging is associated with a decline in nighttime sleep quality (47) as well as with

TABLE 2 Summary of clinical studies that assessed effects of food on sleep quality[1]

Study (ref)	Food	Subjects	Methods	Treatment group results[2]
Southwell et al. (38)	MM, Horlicks	4 healthy men	No drink (control), 350 mL warm water, or 350 mL MM drink 30 min before bedtime	Less movement after MM (412 frames of movements) vs. control (500 frames of movements)
Brezinová et al. (39)	MM, Horlicks	Group 1: 10 subjects (4 women), aged 20–30 y Group 2: 8 subjects (5 women), aged 42–66 y	MM drink (32 g Horlicks powder + 250 mL hot milk) or inert capsule (control) at night for 10 d, 30 min before bedtime	Group 1: Wake episodes: decreased (11.6 times) vs. control (14.5 times) in the seventh hour of sleep Group 2: TST: higher (450.5 min) vs. control (439.6 min) WASO: lower (3.6 min) vs. control (15.5 min) in the second 3 h of sleep
Adam (40)	MM, Horlicks	16 subjects	Inert capsule (control), MM drink (32 g Horlicks powder + hot milk), flavored drink (devoid of milk and cereal), or milk alone at night for 5 d	TST: higher in those who habitually eat before bedtime after MM (463.8 min) and milk alone (471.2 min) vs. control (452.0 min)
Valtonen et al. (41)	Melatonin-enriched milk	70 elderly subjects with a chronic illness	17 oz melatonin-enriched milk for 8 wk	Increased morning and evening physical activity (within groups)
Yamamura et al. (42)	Fermented milk, *Lactobacillus helveticus*	29 subjects, aged 60–81 y	100 g fermented milk or artificially acidified milk (control) 1 time/d at any time for 3 wk	SE: higher after intervention (91.18% ± 1.08%) vs. control (91.37% ± 0.98%) Wake episodes: decreased after intervention (8.31 ± 0.62 times) vs. control (8.85 ± 0.75 times)
Pigeon et al. (43)	TCJ, Montmorency	15 subjects (7 women), aged >65 y, with insomnia	8 oz TCJ or cherry-flavored drink (control) for 2 wk in morning and evening	ISI: lower after TCJ (13.2 ± 2.8) vs. control (14.9 ± 3.6) WASO: lower after TCJ (62.1 ± 37.4 min) vs. control (79.1 ± 38.6 min)
Garrido et al. (44)	Jerte Valley cherries (7 cultivars)	M group: 6 subjects, aged 35–55 y; E group: 6 subjects, aged 65–85 y	200 g cherries for 3 d as lunch and dinner desserts (no control)	TST: increased after consumption of 6 of the 7 cultivars in M group (1.15- to 1.45-fold increase vs. control) and after all 7 cultivars in E group (1.14- to 1.33-fold increase vs. control) SE: increased 1.12- ± 0.02-fold in Van cultivar in M group SOL: decreased 0.54- ± 0.10-fold with consumption of Navalinda cultivar in M group and 0.51- ± 0.07-fold with consumption of Pico Negro cultivar in E group
Howatson et al. (45)	TCJ, Montmorency	20 subjects, aged 18–40 y	8 oz TCJ or cherry-flavored drink (control) for 1 wk within 30 min of awakening and 30 min before the evening meal	TIB: higher after TCJ (514.7 ± 17.0 min) vs. control (492.2 ± 40.6 min) TST: higher after TCJ (419 ± 22 min) vs. control (380 ± 49 min) SE: higher after TCJ (86.8 ± 3.6%) vs. control (84.1 ± 5.8%)
Lin et al. (46)	Kiwifruit	24 subjects (2 men), aged 20–55 y	2 kiwifruits 1 h before bedtime for 4 wk (no control)	TST: higher with kiwifruit intake (395.3 ± 17.4 min) vs. control (354.5 ± 17.1 min) SE: higher with kiwifruit intake (91.2 ± 1.53%) vs. control (86.9 ± 1.94%) WASO: lower with kiwifruit intake (12.8 ± 3.49 min) vs. control (18.9 ± 4.31 min) SOL: lower with kiwifruit intake (20.4 ± 3.53 min) vs. control (34.3 ± 3.86 min)

[1] E, elderly; ISI, Insomnia Severity Index; M, middle-aged; MM, malted milk; oz, ounce; ref, reference; SE, sleep efficiency; SOL, sleep onset latency; TCJ, tart cherry juice; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset.
[2] Only significant results are reported, $P < 0.05$. Results are shown relative to the control group unless otherwise noted.

changes in the circadian regulation of the sleep-wake cycle (48). It has been suggested that the age-related changes in sleep are partly due to a decrease in circadian amplitude (47). It has also long been known that endogenous melatonin production declines with increasing age (48). This may offer an explanation as to why the effects of Horlicks are more effective after serial administration in older adults but not in younger adults.

In an effort to distinguish the sleep-enhancing factors of the Horlicks drink, one study (40) compared the consumption of Horlicks to inert capsules, milk alone, and a flavored drink with energy and macronutrient contents similar to Horlicks. Of note, the participants in this study (40) were older than those in the other Horlicks studies: 52–67 y old compared with 20–66 y old for the other studies (38, 39). TST was not different between the 4 treatments (40); however, as in previous studies (38, 39), the authors noted fewer sleep disturbances after the consumption of Horlicks. The authors also examined habitual dietary habits and divided participants into those who usually ate within 1 h of bedtime (eaters) and those who did not (noneaters) (40). The noneaters slept best after consuming the inert capsules, whereas the eaters slept best after consuming the Horlicks drink, leading the authors to conclude that an individual's dietary habits primarily influence their sleep response to bedtime foods. This is supported by others (26), who showed that nocturnal food intake negatively influences sleep quality, which may be mediated by postprandial discomfort due to reduced digestive activity. It is possible that pre-bedtime food consumption, of any kind, in those who typically do not eat before bedtime negatively influences sleep. However, in those who eat before bedtime, choosing the right nighttime snack may be important in modifying their sleep quality.

In addition to malted milk, natural melatonin-enriched milk, obtained by milking cows at nighttime (nighttime milk) as opposed to daytime (daytime milk), is of scientific interest. A long-term crossover study in 70 elderly patients with dementia examined the effect of daily nighttime milk consumption on sleep quality and circadian activity. The study found no effect of nighttime milk over 8 wk on sleep quality in patients when compared with the consumption of normal milk from cows milked during the day (41). However, in this study, the elderly participants experienced greater morning and evening physical activity after the consumption of nighttime milk, which was seen as beneficial. To further corroborate the potential sleep-inducing effects of nighttime milk, another study showed that melatonin-enriched milk improved sleep efficiency and reduced the number of awakenings in middle-aged adults diagnosed with insomnia (49). Nighttime milk, which is abundant in Trp and melatonin, shortens the onset and prolongs the duration of sleep in mice (50) and has a sedating effect. In mice, motor balance and coordination are reduced to a level comparable to known sedatives with the administration of nighttime milk.

Clinical trials that examined the influence of malted milk and related nutrients on sleep are limited by small study populations and short interventions. The current available evidence suggests that malted milk promotes less restless sleep in both young and old populations, although the mechanisms remain unclear. However, studies indicate that the timing of consumption may play an additional role as to whether the consumption of a malted milk beverage before bedtime enhances sleep. More research with the use of objective measurements is necessary to confirm these findings.

There are several mechanisms by which malted milk may affect sleep quality. Horlicks is composed of wheat, malt barley, sugar, milk, and 14 vitamins and minerals, including vitamin D and several B-group vitamins. Emerging clinical evidence supports the association between vitamin and mineral deficiencies and disrupted sleep. In individuals with low serum concentrations, 3 mo of vitamin D supplementation of either 1200 IU/d or 50,000 IU/wk improved SOL and increased sleep duration (51). However, the mechanisms by which vitamin D may affect sleep are not yet clear.

There is also substantial evidence with regard to the influence of B vitamins on sleep. A small clinical crossover study (52) showed that vitamin B-12 affects plasma melatonin concentrations and contributes to the entrainment of the light-dark cycle. Vitamin B-12 was also associated with improvements in sleep quality and alertness assessed by using visual analog scales (53). Furthermore, vitamin B-6 serves as a cofactor in the synthesis of serotonin from 5-hydroxytryptophan and thus indirectly affects the synthesis of melatonin. However, supplementation of 100 mg vitamin B-6 had no effect on melatonin secretion or sleep duration and architecture in a study in 12 healthy men (54).

Higher Trp and melatonin concentrations appear to be mainly responsible for the sleep-promoting effect of nighttime milk. An analysis of milk content in one study revealed that nighttime milk contained higher amounts of Trp (4.66 mg/g) and melatonin (85.5 pg/g) than daytime milk (3.75 mg/g and 8.8 pg/g, respectively) (50). Another study used nighttime milk with a melatonin concentration of 10.2–18.3 pg/mL, with subjects consuming 0.5 L milk/d (41). This amount did not increase the subjects' blood melatonin concentrations. However, the consumption of nighttime milk with a melatonin concentration of 39.43 pg/mL, ~10 times the concentration found in daytime milk, was associated with increased circulating melatonin concentrations in rats (55). Thus, it appears that high milk melatonin concentrations are necessary to affect blood concentrations.

Fatty fish. Fatty fish (>5% fat) is a good source of vitamin D and omega-3 FAs, nutrients important for the regulation of serotonin and therefore sleep regulation. Hansen et al. (56) investigated the effects of fatty fish consumption on sleep variables in inmates with limited daylight exposure. The fish group consumed 300 g Atlantic salmon 3 times/wk for 6 mo, whereas the control group consumed an equivalent amount of meat (chicken, pork, or beef); however, the portions were reduced to 150 g during the last 4 wk of the study. Participants wore wrist actigraphy monitors and kept sleep diaries for 1 wk before and during the last week of the intervention. From pre- to post-test, SOL and actual wake time increased in the control group and SE decreased in both the control and fish groups. By

the end of the intervention, the men consuming fatty fish during the study had higher concentrations of vitamin D and n–3 fatty acids (EPA and DHA) than the control group, which may partially mediate the reported differences in sleep quality between the groups. Consistent with previous studies (51, 57), vitamin D status was positively correlated with sleep efficiency and sleep quality. Given that SOL and wake time did not change in the fish group but rather worsened in the control group, the conclusion that fatty fish is beneficial for sleep quality is not appropriate. It would be more adequate to state that meat consumption may worsen sleep quality. This, however, deserves further exploration.

Fruit. Other studies have looked at the consumption of fruit on sleep promotion. The consumption of 2 kiwifruits/d, 1 h before bedtime for 4 wk, significantly increased TST and SE as measured by sleep actigraphy in adults with self-reported sleep disorders (46). In addition, sleep diary data showed a significant reduction in WASO and SOL compared with baseline values. Daily consumption of kiwifruit before bedtime thus appears to be beneficial in increasing TST and SE in adults with sleep disturbances but warrants additional research, particularly with studies that include a control food.

More recent studies have examined the effect of tart cherries on sleep regulation. The consumption of 8 ounces of tart cherry juice in the morning and nighttime for 2 wk was associated with a significant reduction in insomnia severity and WASO in adults with chronic insomnia (43). Howatson et al. (45) later replicated the study in a population of young, healthy adults. One week of tart cherry juice supplementation increased urinary melatonin concentrations, TST, and SE compared with a placebo juice.

Other varieties of cherries were also assessed for their effects on sleep variables (44). Participants consumed 200 g of 7 different Jerte Valley cherry cultivars (not including the Montmorency cherry) as lunch and dinner desserts for 3 d each with a 1-wk washout period between cultivars. Compared with baseline values, there was an increase in urinary melatonin, antioxidant capacity, and TST after the consumption of each of the 7 cherry cultivars in both middle-aged and elderly individuals. However, other sleep variables varied depending on the age group (middle-aged compared with elderly) and cherry cultivar consumed. The number of nighttime awakenings decreased significantly after the consumption of the Pico Limón cultivar in the middle-aged group, whereas the elderly group saw a similar decrease after the consumption of the Pico Colorado cultivar. In addition, SOL decreased in both age groups after the consumption of Navalinda cherries and after intake of the Pico Negro cultivar in the elderly group. Although Jerte Valley cherries naturally have higher concentrations of melatonin and Trp (46), it is possible that the melatonin concentrations vary between the different cultivars. Differences in melatonin concentrations may explain why the consumption of specific cherry cultivars resulted in sleep improvements in certain age groups and others did not. However, the study did not include a control group, and additional studies on Jerte Valley cherries are necessary.

In summary, clinical evidence supports the sleep-promoting effects of tart cherries and kiwifruit. The consumption of 2 kiwifruits 1 h before bedtime appears to enhance the sleep of individuals with self-reported sleep disorders and may also promote sleep in healthy individuals, although this has not been confirmed. It is also uncertain if the timing of consumption plays an important role in determining whether kiwifruit consumption will enhance sleep. Tart cherries are an additional fruit that has been shown to improve sleep quality and increase urinary melatonin concentrations. However, the effects of cherries on sleep variables appear to be partially mediated by age as well as the cherry cultivar consumed. Clinical evidence for both cherries and kiwifruit is based on individual studies and the mentioned observations have yet to be confirmed.

The melatonin and phytonutrient profile of tart cherries is often associated with their health and sleep benefits. Tart cherries have a high dietary melatonin concentration, and the consumption of tart cherry juice has been shown to increase urinary melatonin concentrations (43). However, this remains to be confirmed. Tart cherries have also been shown to exhibit anti-inflammatory characteristics that may be beneficial in improving sleep quality. In studies that examined the impact of Montmorency tart cherry juice supplementation on exercise-induced inflammation, tart cherry juice attenuated circulating inflammatory markers and increased the antioxidant capacity of cyclists and marathon runners (58, 59). Because patients with sleep and psychiatric disorders exhibit increased levels of oxidative stress (60), the abundance of antioxidants in cherries may mediate improvements in sleep quality by minimizing oxidative damage.

Although further research into the sleep-promoting mechanisms of kiwifruit is needed, several explanations for the effects of kiwifruit on sleep exist. Lin et al. (46) hypothesized that the high antioxidant capacity and serotonin and folate content of kiwifruit may contribute to the observed sleep benefits of kiwifruit consumption. Kiwifruit is a good source of vitamins C and E (46), both of which protect against the damaging effects of free radicals, and is a source of folate. Previous studies reported an association between disordered sleep and oxidative stress (60), and folate deficiency has been linked to insomnia and restless leg syndrome (61). Folic acid supplementation has been shown to alleviate these symptoms (62). The high antioxidant capacity of kiwifruit may also reduce oxidative damage and consequently improve sleep quality. In addition, kiwifruit is one of the few fruits that has a high serotonin concentration (63), which may be another possible sleep-promoting mechanism of kiwifruit. However, the authors did not measure any of these biological compounds and therefore the mechanism of action remains unclear. Although the study did not have a control group and participants could not be blinded to the intervention, the objective nature of the sleep measurements helps to moderate such biases.

Conclusions

In conclusion, there is evidence to suggest that dietary patterns that favor HC intakes are associated with reduced SOL and SWS and increased REM, whereas HF intakes promote

lower SE and REM and higher SWS and arousals. However, longer-term effects have not been examined in randomized controlled studies. Some foods, such as milk products, fish, fruit, and vegetables, also show sleep-promoting effects, but studies have been too diverse, short, and small to lead to firm conclusions. This review thus finds that some dietary patterns and foods show promise as sleep modulators, but more research is necessary to draw definitive conclusions. Future studies should include a larger sample size, including both men and women, and focus on individuals with sleep disorders. In addition, studies should test whether the timing of the intake of specific foods is important in modulating sleep at night and in determining the most appropriate dose. Finally, it is unknown at this time if an overall diet approach, rather than inclusion or exclusion of specific foods, can improve sleep and, if it does, within what time frame benefits should be observed. Nevertheless, as nutrition professionals, it is important to educate patients on the role of sleep on dietary intakes and health but also to initiate discussions about how diet could be modified to improve sleep quality. It is comforting to note that the findings reported herein are in line with other dietary recommendations for health in the general population: increasing fruit and vegetable intakes, choosing whole grains (higher in fiber), and favoring vegetable oils (low in saturated fat) (64).

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