THE IMPACT OF DIET ON SLEEP QUALITY
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Sleep quality can impact a person’s food choices; for example, sleep-deprived people tend to snack more and eat energy-dense foods. To a lesser extent, researchers have examined the impact of diet on sleep quality. However, recent research suggests that the impact of diet on sleep may need to be considered as part of a treatment plan for patients with sleep disorders.

In people with eating disorders, scientists have long noted impaired sleep during the active disease phase and improved sleep as weight was restored. Which dietary constituent — carbohydrates, fat, fiber, protein, etc. — most contributes to the change in sleep patterns has been a more recent research focus. For example, Phillips and colleagues used electroencephalography to study sleep changes in men who ingested a normal balanced diet, a high-carbohydrate/low-fat diet, or a low-carbohydrate/high-fat isocaloric diet. (In an isocaloric diet, protein, carbohydrate, and fat each contribute the same percentage of calories.) They found that the men had significantly less slow-wave sleep (SWS) after consuming the high-carbohydrate/low-fat diet than after consuming the normal balanced diet or the low-carbohydrate/high-fat diet. The amount of SWS in the latter two diets was similar. Men consuming the high-carbohydrate/low-fat diet or the low-carbohydrate/high-fat isocaloric diet, but especially the former group, had significantly more rapid eye movement (REM) sleep, compared to men consuming the normal balanced diet. Because of these findings, Phillips suggested that an individual’s dietary habits and nutritional status should be considered in sleep research studies.

In a more recent study in 2011, Cibele Crispim and colleagues examined sex differences in the relationship between food intake and sleep patterns. In their study, men and women completed a 3-day food diary and underwent a polysomnographic study. Men had a greater percentage of stage N1 sleep; there was otherwise no sex differences in sleep patterns. In men, nocturnal fat intake was negatively correlated with sleep latency and REM sleep latency. In women, the percentage of nocturnal fat intake was correlated with sleep efficiency, sleep latency, REM latency, stage N2 sleep, REM sleep, and wake after sleep onset; and the percentage of nocturnal caloric intake was correlated with sleep latency and efficiency.

Based on these findings, Crispim concluded that nocturnal food intake (i.e., dinner and late night snacks) negatively affected sleep quality in both sexes.

Based on their findings in a recent review, Marie-Pierre St-Onge and colleagues concluded that changing a person’s carbohydrate and fat intake could be used to impact sleep, particularly SWS and REM sleep. They suggest that certain types of diets (e.g., high-carbohydrate/low-fat diet or low-carbohydrate diet) and certain foods (e.g., milk, fatty fish, tart cherry juice, and kiwi fruit) may promote high-quality sleep.

Most studies that have investigated the impact of food choice on sleep have been small, of short duration, with no clear focus on nighttime sleep, and have relied on self-reports of food intake or on information derived from a single meal. Therefore, St-Onge and colleagues in a more recent study directly observed the daily dietary intake (on a controlled or ad libitum diet) of normal weight adults to determine the effect of diet on nocturnal sleep and to determine whether the intake on the ad libitum feeding day had any correlation with nocturnal sleep patterns.

The controlled diet provided approximately 31 percent of calories from fat, 53 percent of calories from carbohydrates, and 17 percent of calories from protein. For 4 days, the study participants ate the controlled diet. On the fifth day, they ate the ad libitum diet. St-Onge found that an intake of low fiber and high saturated fat and sugar was associated with a lighter, less restorative sleep with more arousals. Sleep duration did not differ after 3 days of consuming the controlled diet, compared to the day of ad libitum diet. After consuming the ad libitum diet, the participants had less SWS and longer sleep onset latency. Greater fiber intake was associated with less stage N1 and more SWS. The percent of energy from saturated fat was associated with less SWS, and consuming a greater amount of sugar and nonsugar/nonfiber carbohydrates was associated with more arousals. St-Onge concluded that diet could be useful in the management of sleep disorders. For example, a fiber-rich diet with a reduced intake of sugars and other nonfiber carbohydrates could be useful in improving sleep depth and architecture in people with poor sleep.

Exactly how foods may be impacting sleep is unclear. One possibility is through the effect of certain constituents in food on...
tryptophan and serotonin utilization and/or production. Tryptophan, an essential amino acid (i.e., it must be consumed because the body cannot synthesize it), comes in two forms: albumin-bound (i.e., protein tryptophan) or not bound to albumin (i.e., free tryptophan). Fat makes tryptophan more available in the brain. After a person ingests fat, various liver and pancreatic enzymes degrade fat into its various constituents (fatty acids, cholesterol, monoglycerides, etc.). One type of fatty acid, called free fatty acid, can displace tryptophan from serum proteins such as albumin. This displacement allows free tryptophan to enter the bloodstream. Once in the bloodstream, free tryptophan can easily cross the blood-brain barrier. In the brain, the raphe nuclei convert tryptophan into serotonin, which is involved in various aspects of sleep. The pineal gland at the base of the brain converts serotonin into the sleep-promoting melatonin.

Carbohydrates (i.e., starch, sugar, fiber) can increase the free fatty acid concentration. The increased free fatty acid level consequently increases the brain levels of tryptophan, which indirectly increases the production of sleep-promoting melatonin in the brain.

Another possibility for how food impacts sleep may be through the effect of dietary constituents on brain structures involved in sleep and wake. For example, one animal study demonstrated that, compared to glucose-fed animals, fructose-fed rats had a greater decrease in serotonin synthesis in the raphe nuclei, brainstem and thalamus, but a greater increase in serotonin synthesis in the hypothalamus.

In animal studies, Murillo-Rodriguez and colleagues injected oleoylethanolamide or palmitoylethanolamide (both of which are fatty acids) into the lateral hypothalamus or dorsal raphe nuclei during the lights-off period (i.e., the animals’ normal sleep period). These areas of the brain are involved in maintaining wake. The compounds increased wake, decreased SWS, decreased REM sleep, and increased levels of the stimulatory neurotransmitter dopamine. Based on these findings, Murillo-Rodriguez suggests that the treatment of sleep disorders such as excessive somnolence may involve the use of oleoylethanolamide or palmitoylethanolamide (or drugs that prevent their enzymatic degradation) to enhance wakefulness.

Future studies are needed to determine to what extent particular types of diets may negatively impact sleep quality. For example, a ketogenic (i.e., low-carbohydrate) diet has been associated with reduced REM sleep, increased SWS, and reduced total sleep. A person with narcolepsy or electrical status epilepticus of sleep (i.e., epilepsy in which seizures are induced by profuse epileptiform activity during sleep) may benefit from the effect of a ketogenic diet on REM sleep or SWS, respectively, whereas a person with obstructive sleep apnea who decides to lose weight by consuming a ketogenic diet may experience lighter sleep and arousals, thereby contributing to sleepiness, despite the effective reduction of sleep apnea episodes with treatment.

Improving sleep quality in people with sleep disorders can be complicated. Drug therapy or other treatments (e.g., positive airway pressure treatment for obstructive sleep apnea) may offer substantial relief, but a person may have residual symptoms (e.g., sleepiness) of their sleep disorder. The impact of diet could be an overlooked factor that contributes to residual symptoms. If future studies clarify the impact of diet on certain aspects of sleep (e.g., sleep stages, sleep onset, wakefulness), then modification of an individual’s diet could become part of a treatment plan for patients with sleep disorders.
REFERENCES


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