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REVIEW

Impact of Body Composition on Sleep and Its Relationship with Sleep Disorders: Current Insights

Yu Kawasaki[1], Eri Kitamura[1], Takatoshi Kasai[2–5]

[1]Department of Obstetrics and Gynecology, Juntendo University Graduate School of Medicine, Tokyo, Japan; [2]Department of Cardiovascular Medicine, Juntendo University Graduate School of Medicine, Tokyo, Japan; [3]Sleep and Sleep-Disordered Breathing Center, Juntendo University Hospital, Tokyo, Japan; [4]Cardiovascular Respiratory Sleep Medicine, Juntendo University Graduate School of Medicine, Tokyo, Japan; [5]Department of Cardiovascular Management and Remote Monitoring, Juntendo University Graduate School of Medicine, Tokyo, Japan

Correspondence: Takatoshi Kasai, Department of Cardiovascular Medicine, Juntendo University Graduate School of Medicine, 3-1-3 Hongo, Bunkyo-ku, Tokyo, Japan, Tel +81-3-6873-2869, Fax +81-3-6873-2869, Email kasai-t@mx6.nisiq.net

Abstract: Sleep is involved in many physiological processes and is essential for both physical and mental health. Obesity and sleep deprivation due to sleep disorders are major public health issues. Their incidence is increasing, and they have a wide range of adverse health-related consequences, including life-threatening cardiovascular disease. The impact of sleep on obesity and body composition is well-known, and many studies have shown an association between insufficient or excessive sleep duration and obesity, body fat percentage, and weight gain. However, there is growing evidence of the effects of body composition on sleep and sleep disorders (particularly sleep disordered breathing) through anatomical and physiological mechanisms (nocturnal fluid shift, core body temperature, or diet). Although some research has been conducted on the bidirectional effects of sleep-disordered breathing and body composition, the specific effects of obesity and body composition on sleep and the underlying mechanisms that explain these effects remain unclear. Therefore, this review summarizes the findings on the effects of body composition on sleep and draws conclusions and proposals for future research in this field.

Keywords: body composition, sleep, sleep disorder, weight gain

Introduction

Sleep is involved in many physiological systems and is known to be an essential factor in physical and mental health.[1,2] However, sleep disorders are one of the most commonly encountered clinical issues in practice.[3] Sleep disorders include several types such as insomnia and sleep-disordered breathing (SDB). For example, SDB is reported to be a problem in approximately 50% of the elderly[4] and is twice as common in men than in premenopausal women.[5] In the Wisconsin Sleep Cohort Study, the prevalence of SDB was increasing in both men and women, and one possible cause for this is the increase in obesity in the general population.[6]

The interaction between sleep disturbance and obesity is well established. Cross-sectional studies[7,8] and prospective studies[9–12] have reported an association between sleep deprivation and risk of obesity, body fat percentage, and weight gain. Short and long sleep also increases the risk of obesity, with 27% and 21% increased risk, respectively, compared to sleepers who sleep for an average amount of time.[13]

In addition to Body mass index (BMI), cross-sectional studies have shown that several body composition parameters are tightly associated with sleep duration and quality. Body composition includes various parts of the body, especially fat mass (consisting primarily of subcutaneous fat and visceral fat tissue) and lean mass (muscle, bone, viscera, ligaments, and tendons).[14] Specifically, lean body mass is positively correlated with sleep duration and sleep quality, while high-fat mass appears to be related to reduced sleep duration and poorer sleep quality.[15–17]

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Numerous researches have explored the impact of sleep on obesity and body composition, with many recommendations proposed for consideration. However, the specific effects of obesity and body composition on sleep and the underlying mechanisms that explain them remain unclear.[18,19] This review summarizes the current data on the impact of body composition on sleep and attempts to draw conclusions and recommendations for future research in this area.

This review presents the current reports on the effects of body composition on sleep, focusing on 1) physical and biological aspects, 2) epidemiological aspects, and 3) interventions. There are many reports on the bidirectional influence between body composition and SDB. However, reports on the impact of body composition on sleep disorders/poor sleep quality other than SDB have been scattered through epidemiological studies, and no systematic research has explained the underlying mechanisms. Therefore, studies related to SDB were considered as a standalone item.

Literature Search Methods

This narrative review searched for studies that discussed the specific effects of obesity and body composition on sleep and its underlying mechanisms. Original articles were identified through a systematic search strategy. MEDLINE (PubMed) was searched for eligible studies without specifying the study period. The review covers a wide range of areas, and the key search terms for each item are as follows: "body composition", "body shape", "body mass index", "obesity", "skinfold measurements", "waist size", "waist-to-hip ratio", "abdominal visceral adiposity", "body fat mass", "percentage of body fat", "lean body mass", "muscle mass", "obstructive sleep apnea syndrome", "sleep-disordered breathing", "apnea-hypopnea index", "pharyngeal obstruction", "nocturnal fluid shift", "core body temperature", "sleep parameter", "total sleep time", "sleep onset latency", "wake after sleep onset", "sleep stage", "slow-wave sleep", "rapid eye movement sleep", "sleep quality", "appetite", and "appetite-related hormones".

This was a narrative review, and the reviewed studies were not evaluated. However, preliminary screening of studies was undertaken by reviewing titles and abstracts to evaluate eligibility, and full-text articles were reviewed to determine eligibility for studies deemed accurate.

Influence of Body Composition on Sleep (Physical and Biological Aspects)

Impact of Body Composition on Obstructive Sleep Apnea Syndrome

Obstructive sleep apnea syndrome (OSAS) is the most frequent SDB disorder with obesity as its most critical pathogenic factor.[20–23]

Obstructive sleep apnea (OSA) is a sleep-related condition in which the pharyngeal airway is periodically obstructed and reopened.[20,23] In terms of body composition and shape, the increase in pharyngeal obstruction may be due to a decreased lung capacity[24] and an anatomical imbalance around the pharynx due to increased body size.[25]

The lungs, although distant from the pharyngeal airway, affect the obstructive potential of the pharyngeal airway. It is thought that the tension that traps pharyngeal air in the caudal longitudinal direction during increased lung volume reduces pharyngeal wall compliance and makes hinders pharyngeal collapse.[26] The pharyngeal airway is more likely to be obstructed when the lung capacity is reduced by central obesity. Increased lung capacity in obese OSA patients during sleep improves the apnea-hypopnea index (AHI).

The pharyngeal airway is encircled by soft tissue (the tongue, soft palate, and palatine tonsils) and is surrounded by the maxilla and mandible.[27] The pharyngeal airway is the space left over when the soft tissue is stored in the bony structure, and the size of this space is determined by the balance between the capacity of the bony structure and the volume of the soft tissue.[28] When soft tissue volume is increased by obesity, the peripharyngeal anatomy is unbalanced, resulting in pharyngeal obstruction.[29]

OSA is marked by recurrent nocturnal episodes of apnea, the severity of which is expressed as AHI. Although AHI is higher in OSA patients with severe pharyngeal obstruction, the reason for pharyngeal obstruction alone cannot explain the change in AHI in OSA patients whose pharyngeal obstruction is near atmospheric pressure. Instability in respiratory coordination increases the periodicity of OSA.[30,31] Leptin secreted from internal fat increases the hypercapnic ventilatory response, resulting in overreaction to abnormal breathing and unstable breathing with repeated hyperventilation and hypopnea. Consequently, obesity acts to increase AHI.[32] Furthermore, increased ventilation efficiency due to decreased

functional residual capacity associated with obesity, hypometabolism, decreased cardiac output, and hypercarbonatemia can also cause increased AHI.[32,33] In addition to obesity, as defined in terms of BMI, the deposition of fatty tissue, especially in the abdomen and chest, leads to decreased compliance of the chest wall and lungs and increased lung elastic recoil pressure, resulting in a high incidence of SDB.[34–36] Furthermore, increased visceral adipose tissue may be involved in the secretion of inflammatory cytokines disturb sleep-wake rhythms.[35]

Impact of the Nocturnal Fluid Shift on OSAS

Fluid accumulates in the lower extremities during the day owing to gravity and is redistributed at night in the supine position.[37] Some of the fluid that migrates accumulates in the neck, narrowing the upper airway and causing OSA.[38,39] The degree of fluid transfer is closely related to the severity of sleep apnea.[40,41] Patients with OSA had a higher fluid index, which indicates "water content" in various parts of the body including both legs. They also had more fluid transfer from the leg to the neck than non-OSA patients (increased neck circumference, $P<0.05$).[42,43] Regarding the correlation between overnight calf circumference and OSA severity, there was a significant association in men but no association in women.[44,45] Sex differences in the response to fluid transfer may be related to anatomical differences in fat distribution.[45] Furthermore, a study examining body composition using bioelectrical impedance analysis in OSA patients with Type 2 Diabetes (DM) showed that the muscle-to-fat ratio could be helpful in screening for OSA in patients with poorly controlled diabetes.[46] This suggests that the muscle-to-fat ratio is likely related to the OSA severity in this population. Although an increase in head and neck fluid volume leads to an increase in neck circumference, and AHI is significantly correlated with changes in neck circumference, it is not directly associated with changes in head and neck fluid volume, and fluid transfer accumulation in the head and neck is not related to OSA.[45] Fluid tends to accumulate in the peripharyngeal tissues rather than in the craniofacial soft tissues, leading to upper airway collapse and OSA. The incidence of OSA also varies depending on the location of the fluid accumulation.

Impact of Body Composition on Core Body Temperature

Body composition and shape may affect core body temperature. The relationship between body composition and sleep is mediated by thermoregulation. In general, sleep is triggered with the maximal rate of decrease in core body temperature,[47,48] and after sleep onset, integrated sleep with increased slow-wave sleep results from low minimum core body temperature.[49,50] Peripheral muscle tissue can act as an insulator, and increased peripheral muscle mass can prevent a decrease in deep-body temperature.[51,52] Conversely, heat production by muscles causes an increase in the basal metabolic rate.[53–56] Generally, the greater the muscle mass, the greater the heat production.[57] Thus, increased muscle mass increased heat production and decreased heat dissipation. Ultimately, an increased muscle mass may decrease sleep quality. It has been reported that the greater the muscle mass, as measured by lean body mass, the shorter the slow-wave sleep proportion.[58] Interestingly, the inverse association between muscle mass and sleep quality has been reported to be more pronounced in athletes than in non-athletes. Furthermore, a report that evaluated the relationship between body composition and sleep quality in athletes[59] identified a link between body composition parameters and the proportion of N3 sleep in early non-REM sleep, with higher muscle mass indicating lower N3 sleep. This suggests that the opposite may be also true, ie the percentage of N3 sleep in early non-REM sleep is also higher with a higher percentage of fat mass. These findings indicate that muscle mass and fat mass may be the main determinants of objective sleep quality.

Impact of Body Composition on Appetite-Related Hormones (Figure 1)

One mechanism leading to obesity is assumed to be the influence of hormones related to appetite and energy balance, such as leptin and ghrelin.[60] Leptin, a metabolic hormone, plays a pivotal role in balancing appetite and satiety by regulating food intake and energy homeostasis.[61] Leptin acts synergistically with the metabolic hormone ghrelin. Leptin inhibits food intake and leads weight loss whereas ghrelin increases hunger and food intake.[61] Lack of sleep causes a decrease in leptin, an appetite inhibitor secreted by fat cells, and an increase in ghrelin, an appetite stimulant secreted primarily by gastric wall cells.[62,63] Additionally, a 30% increase in carbohydrate-rich snack intake was reported in subjects with restricted sleep duration.[63] Obesity is not only a consequence of sleep deprivation via appetite and energy balance, but can also cause sleep deprivation. Obesity increases leptin production and secretion and decreases ghrelin

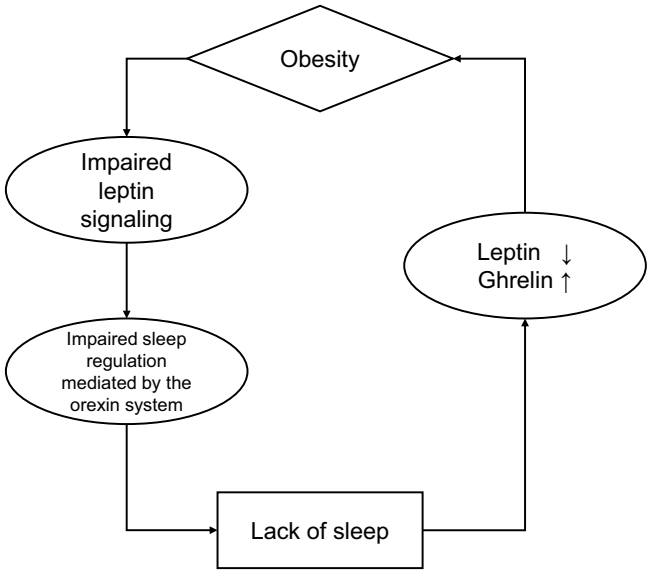

Figure 1 The circle between obesity and sleep.

production and secretion.[64,65] Serum leptin concentration is directly related to fat mass, and increased circulating leptin levels increase leptin resistance and is associated with obesity.[66] Leptin has central nervous system-specific effects beyond appetite regulation.[67] Leptin levels show a circadian pattern, increasing during the night's first half and decreasing during the second half.[68] Furthermore, leptin maintains deep sleep by opposing the function of orexin neurons in the hypothalamus.[69] In animal studies, the orexin (hypocretin) system has been shown to monitor sleep and awakening via interactions with systems that control energy homeostasis. The action of orexin neurons is modulated by leptin, blood glucose, and food intake.[69–71] In animal studies using leptin-resistant genetically obese and diabetic mice, disrupted leptin signaling in obesity has been shown to have detrimental effects on sleep regulation.[72] Although obesity may be caused by poor sleep quality, as mentioned above, few reports have used clinical data to prove that obesity (increased fat mass), in turn, affects sleep via leptin, and future reports are awaited. In support of this claim, we propose that body composition may influence sleep via appetite-related hormones, as leptin plays an important role in regulating sleep-wake states and metabolism.

Influence of Body Composition on Sleep (Epidemiological Aspects)

Relationship Between Body Composition and SDB

As mentioned above, the hypothesis that body composition, particularly obesity, alters breathing during sleep is based on multiple mechanisms.[73] Cross-sectional clinic studies[74–82] and population studies[83–91] have typically found significant correlations in the relationship between overweight and SDB. In a large-scale population-based, prospective cohort study carried out between July 1989 and January 2000,[92] the incidence of SDB was reported to change with increasing or decreasing body weight. A 10% increase in body weight increased AHI by 32% (95% confidence interval [CI], 20%-45), and a 10% decrease in body weight decreased AHI by 26% (95% CI, 18–34%). This trend was even more pronounced when the weight was increased or decreased by 20%. A 20% increase in body weight increased AHI by 70% (95% confidence interval [CI], 42–104%), and a 20% decrease in body weight decreased AHI by 48% (95% CI, 35–58%).[92] Furthermore, a 10% increase in body weight was associated with a 6-fold (95% CI, 2.2–17.0) increase in the odds of developing moderate to severe SDB (AHI>15), and a 20% increase in body weight was associated with a 36.6-fold (95% CI, 4.6->50) increase in the odds.[92] This shows that even in prospective studies, the incidence of SDB varies with simple weight change. Various other anthropometric parameters have been associated with SDB in cross-sectional studies, including neck morphology[22,74–77,80,83,85,86,90] (neck circumference or fat distribution), and obesity[78,79,81,82,90] (BMI, skinfold measurements, waist size, waist-to-hip ratio and abdominal visceral adiposity). BMI is an estimate of body fat

mass (BFM), an indicator of obesity. However, BMI is limited in its ability to assess actual body composition because it does not precisely reflect true body fat composition and may therefore underestimate health risks of obesity in populations with normal BMI.[93] Body composition, such as fat distribution, differs between men and women, with women generally having a higher percentage of body fat (PBF).[94] Fat and muscle parameters were correlated in all patients with OSA, with a similar pattern observed in men. However, it is reported that fat parameters were associated with OSA severity in women, but not with muscle parameters.[95,96] Furthermore, the type of fat associated with OSA is sex-differentiated, with visceral fat being more implicated in men and total-body fat in women.[97] Computed tomography visceral fat area (VFA) assessment shows that men have larger VFA and more severe OSA than women, despite similar BMI and waist circumference, indicating an independent association.[98] In a bioelectrical impedance analysis study, AHI was positively correlated with BFM, PBF, and VFA in both women and men, and the correlation was more robust in men.[94] Moreover, patients with OSA showed significant correlations with muscle mass (MM), skeletal muscle mass (SMM), and segmental muscle mass. Conversely, female OSA patients only showed correlations with the MM of the trunk, right arm, and left arm, but not with other muscle groups. The authors also reported that the SMM/VFA ratio adjusted for age and BMI showed a significant negative correlation ($p=0.015$) in men with OSA, but not in women ($p=0.354$). These reports suggest that the relationship between body composition and OSA is related to both MM and fat mass in men and only to fat mass in women. Muscle and fat distributions may also influence the development of OSA. Furthermore, as mentioned above, OSA is affected by fluid shifts at night. Several epidemiological studies support this hypothesis. In the general population, the prevalence of OSA ranges from 3–14% in men and 4–9% in women.[84,99–101] In contrast, the prevalence of OSA is 23–30%[102,103] in hypertensive patients and 65–83%[104,105] in drug-resistant hypertensive patients. The prevalence of OSA tends to be higher in patients with fluid retention,[37] ranging from 31–44%[106,107] in patients with end-stage renal disease and 12–26%[108,109] in patients with heart failure. In contrast to the general population, the severity of OSA is not associated with a higher BMI in patients with heart failure, suggesting that factors other than obesity are more influential in the development of OSA in this population.[110] Moreover, heart failure patients with sleep apnea have higher sodium intake, which enhances fluid retention, than those without sleep apnea, and that AHI correlates with sodium intake.[111]

Relationship Between Body Composition and Sleep Parameters

Besides BMI, cross-sectional studies have shown that certain body composition parameters are inextricably related to sleep duration and sleep quality.[15–17] The combination of reduced SMM and increased fat mass, or sarcopenia-obesity[112] is strongly associated with chronic disease,[113] especially with short sleep duration.[114]

Few studies have explicitly investigated whether body composition affects sleep, but a significant correlation between lean body mass and non-REM sleep has been reported. Shapiro et al found a significant positive correlation between lean body mass and the first 7 h of non-REM sleep in men and women ($r=0.49$, $p<0.02$) and between lean body mass and slow-wave sleep ($r=0.37$, $p<0.04$).[115]

Although BMI > 25 is considered overweight and the risk of lifestyle-related diseases is a concern, those defined as heavy may include people with shallow fat content, such as bodybuilders. However, people with a high percentage of fat in their body weight may have a BMI that is within the normal range.[116] Although it is rare to have difficulty classifying a population's body size based on BMI alone,[117] police officers, firefighters, and other people who require a high level of fitness need to be correctly assessed not only by BMI but also by body fat percentage.[118] The relationship between body composition and sleep has been a focus of attention, especially in athletes, who may have a body composition different from the general population, and several reports on the effects of body composition on sleep are acknowledged. In men, lean body mass was negatively correlated with slow-wave sleep ($r=-0.41$, $p<0.05$), and these correlations were more pronounced in athletes than in non-athletes ($r=-0.54$).[58] In male and female athletes, the MM percentage was negatively correlated with the rate of slow-wave sleep in the initial non-REM sleep. In contrast, fat mass was reported to be positively correlated with the proportion of slow-wave sleep in initial non-REM sleep.[59] Furthermore, this report showed that, while these did not differ significantly between sexes, they did show significant correlations within each sex. Objective sleep quality was poorer in male athletes than in female athletes, indicating that sleep structure may be related

to MM.[59] Thus, not only obesity but also MM and fat mass may affect sleep structure, indicating that body composition assessment may be meaningful in terms of sleep quality.

Fat distribution may be another factor that defines sleep quality. Based on data from the Korean National Health and Nutrition Examination Survey (2010), a report evaluating the relationship between sleep duration and body composition by dual-energy X-ray absorptiometry in 303 girls revealed that sleep duration was associated with fat distribution and correlated more with fat mass in the trunk than in the head, arms and legs.[119] In a report evaluating the association between sleep duration and site-specific fat mass in US adults,[120] the authors reported that short sleepers (<7 hours) had a higher trunk fat mass index (dividing the fat mass (kg) by the square of body height (m^2)) and higher arm fat mass, arm fat mass index, and leg fat mass than normal sleepers (7–9 hours), but there were no significant differences among those sleeping longer than 9 hours. Similar results were obtained for men, but higher arm fat mass in women was observed only in short-time sleepers. Specifically, short-time sleepers in women had higher arm fat mass and leg fat mass index in the obese group than in normal sleepers, but no association was found in the non-obese group. In US adults, short-time sleep differed from long-time sleep as it was independently related to body fat distribution in different regions, especially in men and obese individuals. In a study whose main objective was to determine the relationship between body composition, including BMI, waist-to-hip ratio, waist-to-height ratio, fat mass index, visceral adipose tissue, and lean BMI (dividing the lean mass [kg] by the square of body height [m^2]), and sleep duration and quality, a negative correlation was found between BMI and total sleep time, and the waist-hip ratio was negatively associated with total sleep time and sleep efficiency.[121] It also shows that participants with a higher lean mass index have shorter sleep duration and lower sleep efficiency.[121] Conversely, no correlation has been reported in objective sleep quality, as indicated by accelerometers, concerning lean BMI.[15] Although the difference between these results cannot be definitively stated, Carneiro-Barrera et al argued that their interpretation of the results may be interpreted by the possibility that a lower lean mass index is associated with longer or excessive sleep duration and increased cortisol levels caused by sleep disturbance, which in turn may be associated with muscle deterioration.[121]

Some studies found that body composition parameters were not associated with subjective sleep quality assessed by the Pittsburgh sleep quality index questionnaire.[121] In contrast, others found a negative correlation between lean BMI[15] and subjective sleep quality. Subjective sleep quality is highly related to psychological and cognitive functioning.[122–124] Body composition parameters are potentially highly relevant to an individual's intake behavior and daily life and may be due to uncontrollable factors in each study population.

Thus, the effects of the amount and distribution of lean and fat mass on sleep have been reported. Many studies have shown a correlation between sleep parameters and body composition over time. When interpreting the results, it should be considered that it is not possible to clearly show whether sleep affects body composition or whether body composition affects sleep as multiple factors may influence both.

Impact of Interventions on Body Composition on Sleep

Intervention on Nocturnal Fluid Shift

The magnitude and distribution of fluid shift during the night are directly related to the severity of sleep apnea.[38–41] A study evaluating the association between fluid shift measurements and respiratory event index in patients hospitalized for ischemic stroke showed a significant association between sitting time and reductions in calf circumference and fluid volume at night.[43] The results of this study suggest that activity during the daytime and physical therapy can reduce fluid displacement from the legs overnight. Furthermore, diuretic therapy reportedly decreases AHI, reduces fluid retention in the leg, and increases the neck circumference.[125,126] Compression stockings, which prevent lower extremity fluid accumulation during the day and decrease lower extremity fluid volume transfer at night, also decrease AHI in patients with chronic venous insufficiency and OSA.[127] Although these reports only involved populations with ischemic stroke[43] and cardiovascular disease,[125,126] such treatment may be effective in patients with OSA with high baseline fluid retention after body composition analysis.

Intervention on Core Body Temperature

Body composition and body size affect core body temperature through heat production and heat release in the muscle and fat.[51–56] Sleep is regulated by core body temperature;[47–50] in fact, it has been reported that a higher MM is associated with lower sleep quality.[58,59] However, interventions to reduce MM and improve sleep quality are futile. A low core body temperature during the first half of the sleep period is important for sleep quality.[128] Furthermore, as the blood flow to the skin increases and body heat is lost, the core body temperature decreases.[129] This suggests that manipulation of body temperature may improve sleep quality. Increased sleep quality has been reported with the use of adjusting the temperature of the sleep environment,[130] wearing thermosuits that alter body temperature,[131,132] wearing socks,[133] sleeping on an electric blanket[134] and high-heat-capacity mattresses.[128] Furthermore, the quality of sleep could be improved not only by manipulating body temperature during sleep but also by adjusting bathing time.[135] It has been reported that bathing before bedtime increases the proportion of slow-wave sleep in the first half of sleep[136] and decreases the frequency of body movements during sleep,[137] thus improving sleep quality. A systematic review and meta-analysis of "water-based passive body heating"[138] such as a warm shower or bath before sleep concluded that a nightly shower, foot bath, or body bath for as little as 10 minutes to one-two hours before bedtime can promote a decrease in bedtime core body temperature and optimize sleep onset latency. Based on these reports, it may be possible to improve sleep quality by controlling body temperature before and after bedtime in populations with high MM, such as athletes.

Intervention from the Dietary Aspect

As mentioned above, studies have showed a relationship between sleep quality and obesity.[139–141] Several reports exist that, more specifically, evaluate the relationship between sleep and energy intake. People who sleep less have a higher energy intake from fat[142,143] and snacking.[144] In the National Health and Nutrition Examination Survey data from the United States, populations that sleep less than seven hours per night consume less protein, carbohydrates, fiber, and fat, and to inoculate fewer types of foods, compared to those consumed by normal populations that report sleeping seven to eight hours per night.[145] These data are supported by clinical intervention studies, which also showed that restricting sleep increases snacking intake.[146] Moreover, participants with sleep restriction tend to prefer foods with high fat.[147,148] However, while these studies show a connection between sleep and diet, it is not clear whether sleep influences dietary intake or whether dietary intake affects sleep. Although few studies have evaluated sleep architecture through dietary interventions, Karklin et al reported an increase in sleep onset latency and a decrease in slow-wave sleep duration in nine overweight women following an 800-kcal diet for four weeks.[149] One study also reported a decrease in slow-wave sleep and an increase in REM sleep in eight healthy-weight men on a high-carbohydrate, low-fat diet for two days compared to a low-carbohydrate, high-fat diet and a balanced diet for two days.[150] Moreover, a study of 26 healthy adults who evaluated sleep architecture through dietary interventions showed that higher fiber intake was associated with less stage N1 sleep ($P = 0.0198$) and more slow-wave sleep ($P = 0.0286$), a higher percentage of energy from saturated fatty acids was associated with less slow-wave sleep ($P = 0.0422$), and a higher rate of energy from sugar and other carbohydrates not considered sugar or fiber was associated with arousal ($P = 0.0320$ and 0.0481).[151] In a review summarizing the effects of food intake on sleep, the authors concluded that a high-carbohydrate diet is associated with decreased sleep onset latency, slow-wave sleep, and increased REM sleep, while a high-fat diet promotes decreased sleep efficiency and REM sleep and increased slow-wave sleep and arousal.[152] A similar dietary content for general health (increasing fruit and vegetable intake and choosing whole grains with "high fiber" and vegetable oils with "low saturated fat") may improve sleep.[152]

Furthermore, diet may affect body composition and sleep.[121] Obesity and central obesity are the most critical pathogenetic factors.[20–23] Moreover, obesity may reduce sleep quality through leptin, as shown in Figure 1. Regarding body composition, lean body mass is significantly positively correlated with non-REM and slow-wave sleep[115] and sarcopenia-obesity,[112] and a combination of reduced skeletal muscle and increased fat mass is strongly associated with reduced sleep duration.[114] These findings suggest that a diet that prevents obesity and maintains adequate MM should be implemented. The critical point is that the dietary approach to obesity prevention must also focus on changes in body

composition, as the loss of lean body mass can be problematic.[153] The Harris-Benedict[154] or WHO[155] equation should be used to determine the daily energy requirement. Furthermore, the manipulation of diet composition is important.[156] Dietary carbohydrates cause excessive insulin secretion[157] and promotes body fat accumulation.[158,159] Therefore, one way to control obesity is to reduce the carbohydrate ratio in the diet.[160] Low-carbohydrate diets for weight loss are long been known.[161] Low-carbohydrate diets decrease insulin secretion, promote fat mobilization from adipose tissues, and stimulate the oxidation of free fatty acids.[157,162] This results in a decrease in body fat and an increase in energy expenditure.[157] Ketogenic diets, a low-carbohydrate diet that maintains a moderate protein intake and consumes at least 70% of its energy from fat while severely limiting carbohydrate intake,[163] are considered a strategy for weight loss and improved metabolic control.[164] Ketogenic diets are expected to reduce carbohydrate metabolism, increase lipid oxidation, and improve the conversion of free fatty acids to ketone bodies.[165–167] In a brief review[168] of weight loss methods and lean body mass reduction, 76% of the total weight loss with low-carbohydrate diets was attributed to fat mass and the remaining 24% to lean mass. Conversely, low-fat diets that reduce fat intake similarly result in weight loss.[157,169,170] Some meta-analyses have concluded that low-fat diets produce more significant energy expenditure and fat loss than low-carbohydrate diets at the same calories.[160] In a brief review[168] of weight loss methods and lean body mass reduction, such as low-fat or low-carbohydrate diets, 76% of the total weight loss was attributed to fat mass, and the remaining 24% to lean mass. High-protein diets, with high-fat content and more than 20% of energy derived from protein, prevent obesity by sustaining satiety while maintaining energy expenditure.[171] Additionally, proteins, especially the amino acid leucine, can induce muscle protein synthesis and prevent the reduction in lean body mass caused by caloric restriction.[172] In a brief review[168] of weight loss methods and lean body mass reduction, high-protein diets attributed 89% of the total weight loss to fat mass and the remaining 11% to lean mass, which may be a more appropriate diet from a sleep perspective. Plant-based diets containing plant-based foods (vegetables, fruits, grains, and legumes) also have positive effects on blood lipid levels and body fat percentage.[173] These results are generally consistent with the results of epidemiological studies that have evaluated the effects of diet on sleep, and strongly support the possibility that diet mediates body composition and sleep.[121] Interventions in body composition based on dietary changes may promote increased sleep quality.

Future Perspectives

The bidirectional effects between SDB, represented by OSAS and obesity, and body composition, have been discussed in terms of epidemiological facts and detailed mechanisms. SDB can demonstrate a bidirectional association between body composition and breathing disorders/nocturnal fluid shifts through an anatomical mechanism. Although there have been epidemiological reports on the effects of obesity and body composition on sleep disturbances and poor sleep quality that differ from those of SDB, few studies have examined the underlying mechanisms. This may be difficult to explain by a single mechanism, as there are other factors that bridge body composition and sleep besides core body temperature and diet, which have been presented in this study. Furthermore, when discussing the impact of body composition on sleep, it is necessary to consider that it is not always possible to indicate whether sleep affects body composition or body composition affects sleep because multiple factors can affect both body composition and sleep.

Further research is needed to demonstrate, with clinical data, that body composition influences sleep via the hypothesized mechanisms (core body temperature and appetite-related hormones). Therefore, it is essential to conduct intervention studies that simultaneously monitor body composition, hypothesized mechanisms (eg, core body temperature and appetite-related hormones), and sleep parameters.

Conclusions

While the epidemiological and mechanistic aspects of the effects of body composition on SDB are being established, there is ambiguity as to whether body composition causes sleep disturbances and poor sleep quality distinct from SDB. Intervention studies to clarify the hypotheses of the mechanisms (core body temperature, diet, something not yet presented) are needed.

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