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Sleep disturbances, body fat distribution, food intake and/or energy expenditure: pathophysiological aspects

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Abstract

Data from cross-sectional and longitudinal studies have illustrated a relationship between short sleep duration (SSD) and weight gain. Individuals with SSD are heavier and gain more weight over time than normal-duration sleepers. This sleep-obesity relationship may have consequences for obesity treatments, as it appears that short sleepers have reduced ability to lose weight. Laboratory-based clinical studies found that experimental sleep restriction affects energy expenditure and intake, possibly providing a mechanistic explanation for the weight gain observed in chronic short sleepers. Specifically, compared to normal sleep duration, sleep restriction increases food intake beyond the energetic costs of increased time spent awake. Reasons for this increased energy intake after sleep restriction are unclear but may include disrupted appetite-regulating hormones, altered brain mechanisms involved in the hedonic aspects of appetite, and/or changes in sleep quality and architecture. Obstructive sleep apnea (OSA) is a disorder at the intersection of sleep and obesity, and the characteristics of the disorder illustrate many of the effects of sleep disturbances on body weight and vice versa. Specifically, while obesity is among the main risk factors for OSA, the disorder itself and its associated disturbances in sleep quality and architecture seem to alter energy balance parameters and may induce further weight gain. Several intervention trials have shown that weight loss is associated with reduced OSA severity. Thus, weight loss may improve sleep, and these improvements may promote further weight loss. Future studies should establish whether increasing sleep duration/improving sleep quality can induce weight loss.

Keywords

body weight; energy balance; sleep

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Introduction

Sleep has traditionally been overlooked in the search for modifiable risk factors for obesity. However, since the mid-2000s, there has been increasing evidence relating short sleep duration (SSD) to obesity. In fact, many cross-sectional studies in both adults and children have reported that the risk of obesity is greater in short sleepers (generally those reporting sleeping <7 h/night) than normal sleepers (those reporting sleeping 7–8 h/night) and that there is a greater prevalence of obesity among short than normal sleepers. This has been the topic of several meta-analyses and systematic reviews [1–4] and will not be elaborated on in this report.

Providing further clues that SSD could be a trigger for weight gain and the development of obesity, longitudinal studies have also shown that SSD is associated with greater weight gain than normal sleep. Data from the Zurich Cohort Study showed that sleep duration was a strong predictor of obesity in longitudinal models and there was a trend for the change in sleep duration over time to be negatively associated with the change in body mass index (BMI) [5]. The Nurses' Health Study also revealed that women who reported sleeping ≤5 h/night had the greatest weight gain over the 16-year follow-up period, followed by those reporting sleeping 6 and 9 h/night. Women who reported sleeping 7 and 8 h/night had the lowest weight gain [6]. In that cohort, short sleepers had an increased risk of developing obesity and having a large weight gain (≥15 kg) compared to normal sleepers. Similar results were obtained in a cohort of older adults followed over 2 years: women who reported sleeping ≤5 h/night had higher odds of gaining ≥5 kg than normal sleepers [7]. Interestingly, this association was not observed in men. A similar increased risk of a 5-kg weight gain was also observed over a 6-year period in short sleepers in the Quebec Family Study, relative to normal sleepers [8]. Conversely, Watanabe et al. [9] reported that SSD increased the odds of developing obesity over a 1-year follow-up period in men but not women, in a large Japanese cohort of working adults.

It is interesting to note that two longitudinal studies that have failed to find a relationship between sleep duration and obesity risk had measured sleep duration with actigraphy [10, 11]. These studies illustrate the inherent flaws associated with self-report of sleep duration. Moreover, longitudinal studies, although providing a better insight into the relationship between sleep and obesity than cross-sectional studies, still do not show causality. This report will focus on intervention studies illustrating the impact of sleep duration and quality on obesity risk. The primary aim will be to describe potential mechanisms by which sleep could play a causal role in the etiology of obesity and studies that have intervened to induce weight loss.

Impact of sleep on weight loss

Given the epidemiological findings of a strong cross-sectional relationship between SSD and obesity, as well as longitudinal associations between SSD and weight gain, it is expected that short sleepers would also have reduced ability to lose weight when embarking on a weight-loss program or that normal sleepers would gain weight if asked to restrict their sleep. Surprisingly, very few studies to date have examined these questions. What's more,

most studies that have assessed the role of sleep duration on weight-loss success or weight gain have not been designed to test whether short sleepers lose less weight than normal sleepers or are less likely to achieve 5% or 10% weight loss over the course of a structured weight-loss program.

A small pilot study by Nedeltcheva et al. [12] was the first published to specifically test the hypothesis that recurrent SSD could impede weight loss during a reduced-calorie diet. Ten overweight and obese men and women were fed at 90% of their resting metabolic rate (RMR) for two 14-day periods, with either 8.5 h time in bed (TIB; 7 h 25 min actual sleep) or 5.5 h TIB (5 h 14 min actual sleep). As food intake was controlled, participants lost a similar amount of weight in both periods. However, the composition of the lost weight differed: 25% of lost weight was fat mass during restricted sleep compared to 55% in the normal sleep condition. Participants also had higher fasting and postprandial respiratory quotient and lower RMR at the end of the sleep restriction weight-loss period compared to normal sleep. The authors concluded that sleep restriction compromised the efficacy of energy-restriction practices for weight loss.

Robertson et al. [13] also performed a short-term intervention study to assess the effects of a 1.5-h sleep restriction on insulin sensitivity and metabolic parameters. Young, normal-weight men either maintained their habitual sleep schedules for 3 weeks or were asked to set their alarm clocks 1.5 h earlier. The authors found a significant week by sleep duration interaction, such that in men who restricted their sleep, body weight was reduced after the first week but returned to baseline during the last week of the intervention. Those who maintained their habitual sleep routine did not have any change in body weight. It is unknown whether the rise back to baseline in the sleep restricted group would have continued to lead to overall weight gain if the study had been prolonged. Nevertheless, this study brings forth interesting questions concerning the time course of metabolic changes during extended mild sleep restriction. However, this was a very small, parallel-arm study with nine and ten participants per group, increasing the chance of bias.

Other studies have examined charts from participants of various weight-loss studies to assess whether sleep duration at baseline plays a role on the achievement of weight loss. Data from three weight-loss studies conducted at Laval University in Quebec City showed that sleep duration was positively associated with loss of body fat, after adjusting for age, sex, baseline BMI, length of the intervention, and change in energy intakes [14]. In that study, sleep quality was inversely associated with loss of body fat, such that those with poor sleep quality had lower body fat loss. Thomson et al. [15] also found that both sleep quality and duration were independent markers of weight-loss success in participants followed over 2 years in a commercial weight-loss program. Significant weight loss was defined as achieving 10% weight loss at 6 months; weight maintenance success was assessed at 12, 18, and 24 months. The Pittsburgh Sleep Quality Index at baseline was used to determine its association with weight-loss success, and at 6 months, to determine whether it predicted weight maintenance. Women who reported SSD and those with fair/poor sleep quality were less likely to achieve significant weight loss. At 6 months, women who reported very good sleep quality or duration >7 h/night had an increased likelihood of successfully maintaining weight loss at 12 and 18 months; those associations were attenuated and no longer

significant at 24 months. In addition, poor habitual sleep efficiency (≤85%) was associated with a 38% lower likelihood of successful weight-loss maintenance at 18 months. These results suggest that weight-loss success is more likely if women enter a weight-loss program with good sleep quality and adequate sleep duration and that they have a better chance of maintaining that weight loss than if they have poor sleep quality and SSD.

Data from the LIFE Study, a two-phase trial examining two alternative strategies for weight loss, also showed greater weight-loss success with adequate sleep duration [16]. In phase 1, participants underwent a weight-loss diet based on the Dietary Approaches to Stop Hypertension. Participants who lost at least 4.5 kg in phase 1 moved on to phase 2, which evaluated weight maintenance strategies over 18 months. Participants who reported sleeping 6–8 h/night at baseline had higher rates of eligibility for phase 2 than those reporting sleeping ≤6 h or >8 h/night. The authors proposed that assessing sleep duration at the onset of a weight-loss program could identify individuals who may require additional counseling and resources for weight-loss success. This, however, would need to be tested.

Only one study of this kind has been conducted using data from children. Sallinen et al. [17] performed a retrospective chart review of obese adolescents who participated in a clinical, family-based, weight-loss program to assess whether self-reported sleep duration at entry predicted weight loss at 3 months. More baseline sleep was predictive of greater BMI reductions: a 30-min increase in baseline sleep duration predicted an additional reduction of 0.2 kg/m^2 in BMI. Adolescents who had a loss of at least one BMI unit also reported significantly more baseline sleep than those who did not achieve this BMI reduction.

Mechanistic insights: impact of sleep on energy balance

Sleep duration

If SSD plays a causal role in the development of obesity, then it must affect regulators of energy balance. Spiegel and colleagues [18] were among the first to report a SSD-associated decrease in leptin, an adipose tissue-derived hormone signaling satiety and rise in ghrelin, a gut-derived hormone signaling hunger. While this finding was promising on a mechanistic level, subsequent studies have yielded conflicting results, with some reporting no change [19–21] and or an increase [22–25] in leptin after partial sleep restriction. Ghrelin findings are also inconsistent [19–21]. The reasons for the inconsistency in SSD-induced alterations in leptin and ghrelin are unclear but are possibly related to differences in feeding protocol and energy balance state between experiments, both of which are factors that can influence appetite hormone secretion [26]. The timing of the sleep-wake schedule and alterations in sleep architecture, and a role of sex on modulating the effects of sleep curtailment on hormone secretion, may also play a role in the observed discrepancies. Recently, increased leptin and decreased ghrelin in restricted vs. habitual sleep conditions were observed in a study in which participants had ad libitum access to food. Somewhat paradoxically, this observed hormone profile was associated with increased food intake under SSD vs. habitual sleep [27].

While the ad libitum access to food and increased food intake during SSD are likely to have influenced the above observation, the results also importantly suggest that brain mechanisms

involved in the reward-driven or hedonic aspects of appetite, as opposed to homeostatic or peripheral hormonal controls of hunger, may influence food intake under restricted sleep conditions. Indeed, using functional magnetic resonance imaging (fMRI), recent work from our laboratory has implicated brain regions involved in motivation, reward, and cognitive processing in the response to experimental sleep restriction [28, 29]. Specifically, relative increases in activity within the orbitofrontal cortex, putamen, nucleus accumbens, thalamus, and insula in response to food stimuli were seen after 5 nights of restricted sleep (4 h TIB/night) compared to habitual sleep (9 h TIB/night) [28]. Moreover, the same partial sleep curtailment manipulation resulted in enhanced activation in the insular cortex, orbitofrontal cortex, and dorsolateral prefrontal cortex in response to unhealthy compared to healthy food stimuli [29].

Regardless of the mechanism, experimental sleep curtailment is shown to induce significant increases in food intake [19, 27, 30–32]. Interestingly, the increased total energy intake after sleep restriction is often characterized by a disproportionate increase in food consumption late in the day [19, 27, 31]. Research should focus not only on the overall increase in energy intake under short sleep but also on the distribution of caloric consumption across the day and the reasons underlying this delay in food intake, as nighttime eating in particular may be associated with increased weight gain [33].

Increased energy intake in response to sleep curtailment beyond the total daily energy that is expended by the combination of metabolic processes and physical activity is a possible means by which SSD can lead to weight gain. It is therefore important to consider how sleep restriction affects energy expenditure (EE). Initial studies [19, 32] reported no effect of sleep duration on total EE when assessed with doubly-labeled water. More recently, whole-room indirect calorimetry has been used to more accurately measure 24-h EE in response to experimental sleep restriction [27, 34, 35]. These studies consistently demonstrated an increase in 24-h EE of ~4–5% after short vs. habitual sleep, which corresponds to an increase of ~90–110 kcal/day. In looking at the distribution of EE across the 24-h day, it is apparent that these increases are mainly the result of the additional hours spent awake during the night [27, 34, 35], suggesting that the increased risk of obesity observed in short sleepers in many epidemiological studies [4] may be caused by a substantially large increase in energy intake that is not offset by the more modest increases in EE associated with reduced sleep duration.

Sleep quality and architecture

In addition to sleep duration, characteristics of sleep, like quality and architecture, can impact energy balance. Rapid eye movement (REM) sleep was found to be negatively associated with overweight in children and adolescents [36]. Slow wave sleep (SWS) was also found to be inversely related to BMI and waist circumference in older adult males [37]. Rutters and colleagues [38] investigated the inter-individual relationships between sleep architecture and energy balance during a 2-day in-lab investigation that included measures of polysomnographic sleep, hunger levels, and a calculated assessment of energy balance. REM sleep duration and energy balance were positively correlated, whereas SWS duration was inversely related to energy balance. This effect may have been mediated by the

observed inverse relationship between SWS and hunger [38]. Recent work from our group [39] explored a similar question in the context of experimental sleep curtailment. Negative associations were found between REM sleep duration and hunger and between stage 2 sleep (duration and percent total sleep time) and desire to eat something sweet. Moreover, the proportion of total sleep time spent in stage 2 sleep was negatively associated with total energy intake and that spent in SWS and REM sleep negatively associated with intake of fat and carbohydrate under ad libitum eating conditions [39].

An experimental sleep fragmentation study has further demonstrated a role of sleep quality and architecture in influencing hunger and appetite-regulating hormones [40]. When participants were exposed to sleep fragmentation, via repeated wake-up calls, levels of glucagon-like peptide 1 (a gut-derived hormone signaling satiety) and fullness scores were reduced relative to non-fragmented sleep. Interestingly, experimental sleep fragmentation in this study caused significant reductions in duration of stage 2 and REM sleep compared to the non-fragmented night.

Together, these findings imply that sleep-related alterations in energy balance parameters may not be solely the result of reduced sleep duration alone. Rather, altered sleep architecture – specifically decreases in SWS and REM sleep – likely plays a large role in the development of adverse body weight outcomes by influencing hormone pathways, appetite and hunger.

Obstructive sleep apnea

Obstructive sleep apnea (OSA) is characterized by frequent arousals from sleep and disruptions in sleep quality and architecture. Given the relationships between sleep disturbances, architecture, and energy balance highlighted above, it would be expected that OSA is accompanied by an increased risk of obesity. Indeed, both body fat percentage and BMI are found to be significantly related to the apnea-hypopnea index (AHI), an index of OSA severity [41–43]. Moreover, visceral adiposity [41, 42, 44] is significantly and positively correlated with AHI. As might be expected, increased fat accumulation around the neck and pharyngeal region could lead to narrowing or compression of the upper airway and has been proposed as a cause of OSA [45] and predictor of its severity [46]. OSA patients have a larger parapharyngeal fat pad volume [47], a significant predictor of AHI [45].

While obesity is well-established as a leading risk factor for the development of OSA, recent findings suggest that OSA itself plays a role in promoting further weight gain [48]. Indeed, newly diagnosed OSA patients were demonstrated to have a history of significant weight gain compared to sex, age, and BMI-matched controls over the year prior to diagnosis [49].

Reasons for this reciprocal relationship are uncertain. However, OSA affects various factors influencing the regulation of energy balance, including a disruption of sleep quality and continuity. Specifically, the intermittent hypoxia that occurs in the disorder causes repeated nocturnal arousals from sleep, resulting in sleep fragmentation and a disruption of sleep architecture. Obese individuals with OSA have increased expression of stage 1 sleep and decreased expression of stage 2, REM, and SWS compared to non-OSA obese individuals [50]. As REM sleep is inversely related to hunger levels, and both REM sleep and SWS are

inversely related to ad libitum intakes of fat and carbohydrate [39], the particular profile of sleep associated with OSA would indicate propensity towards positive energy balance. Other behavioral, metabolic, or hormonal effects of OSA may further enhance this state, favoring weight gain and/or difficulty losing weight.

Excessive daytime sleepiness is an important consequence of OSA, and is present in 20–40% of patients [51, 52]. A relationship between lack of regular exercise and excessive daytime sleepiness was observed in obese OSA patients [53]. OSA may also affect energy metabolism. RMR [54], sleeping EE [55] and 24-h EE are increased in OSA patients compared to snoring controls [56]. In another study, resting EE was found to be independently and positively associated with AHI even after adjusting for BMI [57] or fat-free mass [58].

These increases in EE in response to OSA may seem paradoxical, as it can be assumed that increased EE could preclude the development of a positive energy balance in these patients. However, we have recently shown that the added thermogenesis associated with increased wake time during SSD is much smaller in magnitude than the increased energy intake observed in response to sleep restriction [35]. This is likely to be the case with OSA [59], although this has not been directly tested. There is some evidence for a dysregulation of appetite-regulating hormones in OSA patients, with increased leptin levels, suggestive of leptin resistance [60–63], and increased ghrelin [62] relative to controls. This aspect of energy balance has not been extensively studied in OSA, although AHI was found to be positively associated with the caloric as well as fat and carbohydrate content of a self-selected meal from a standard hospital menu in adolescents [64]. Food choice and energy intake should be systematically studied in OSA patients to determine how the disorder may influence body weight management.

There is therefore reason to suggest OSA as a risk factor for obesity. This was investigated in a population-based, prospective cohort [65]. A 10% increase in weight predicted a 32% increase in AHI and similar weight loss predicted a 26% decrease in AHI over 4 years of follow-up, after adjusting for sex, smoking, baseline BMI, and age [65].

Somewhat similar results were subsequently reported from the Sleep Heart Health Study [66]. While OSA severity increased with weight gain and decreased with weight loss over the 5 years follow-up, the observed change was greater for weight gain than weight loss. These effects were modulated by sex such that men gaining ≥10 kg had 5.2 times the odds of having a large (>15 events/h) increase in respiratory disturbance index (RDI) compared to weight-stable men whereas the odds in women was 2.6. The impact of weight loss also differed by sex: men losing ≥10 kg had 5.4 times the odds of a large reduction in RDI, whereas this relationship was not significant for women. A similar effect of sex was reported from data from the Cleveland Family Study [67].

Together, these longitudinal studies clearly indicate that increasing prevalence of obesity in the population and within individuals over time can increase the incidence and severity of OSA. Conversely, the findings of these studies also imply that lifestyle interventions, which

can successfully reduce body weight can be useful therapeutic options for the management and treatment of OSA.

Relationship between weight change and OSA

Despite the information obtained thus far, it still remains unclear whether SSD and/or sleep disruption as is seen in OSA causes obesity or hinders weight loss. Based on the studies described above, sleep duration and quality at baseline seem to be important factors in an individual's response to a weight-loss diet. However, because obesity is accompanied by sleep disorders, including a high prevalence of OSA, it is reasonable to hypothesize that obesity leads to sleep disorders, which can then reduce sleep quality and duration. This could lead to further weight gain or difficulty achieving weight loss. Borel et al. [68] showed that patients with visceral obesity and OSA had a smaller decrease in BMI, waist circumference, and fat mass in response to a lifestyle weight-loss intervention compared to those without OSA. The authors suggested that this decreased response to the weight-loss intervention program in OSA patients was due to alterations in sleep quality and/or duration.

Conversely, weight loss could improve sleep disorders. In such a case then, a reduction in weight could reduce sleep disturbances and sleep-disordered breathing and increase sleep quality and duration. Perhaps this could assist with weight-loss efforts and help individuals achieve further weight loss, propelling a cycle of improved weight and sleep quality/duration (Figure 1).

Verhoef et al. [69] attempted to untangle the "chicken-or-the-egg" question of the sleep-obesity paradox and reveal the temporal sequence of changes in sleep and body weight during a weight-loss intervention. Overweight and obese men and women underwent a very-low-calorie diet for 8 weeks followed by 10 months of weight maintenance surveillance. Sleep was assessed by questionnaire at all study visits: before weight loss, after weight loss (8 weeks), and at 3 and 10 months of follow-up. In short and normal sleepers, sleep duration increased with weight-loss: change in sleep duration was negatively correlated with change in BMI. This was also noted at the 3-months follow-up visit but not at 10 months. However, the change in sleep duration during weight loss was not associated with the change in BMI during follow-up. This latter finding suggests that changes in sleep, BMI and fat mass occurred in parallel during weight loss. The authors concluded that sleep duration benefits from weight loss and vice versa, therefore, no conclusions could be drawn on the temporal sequence of the association.

Illustrative of the impact of body weight on sleep disorders, several randomized controlled trials consisting of either diet [70, 71] or diet and exercise [72] have demonstrated the effectiveness of lifestyle interventions in inducing weight loss to improve OSA severity. In a study by Johansson and colleagues [71], obese individuals with at least moderate (AHI>15) OSA at baseline were randomized to a control group or lifestyle intervention consisting of a very-low-energy diet (2.3 MJ/day liquid meals) for 7 weeks, followed by 2 weeks of gradually re-introducing normal food to reach 6.3 MJ/day at week 9. The intervention group had greater reductions in body weight and adiposity measures at week 9 than the control group. The treatment group also had reduced AHI at week 9 compared to the control. Longer-term effects of the intervention were studied in the treatment group participants, who

underwent a weight-loss maintenance program to week 52 [73]. Participants in the control group were crossed over to the low-energy intervention phase for 9 weeks and ultimately the weight-loss maintenance program until week 52. At week 52, AHI and body weight were significantly reduced compared to baseline and a 10 kg decrease in weight was associated with a decrease in AHI of 5 events/h.

Tuomilehto and colleagues [70] conducted a prospective randomized controlled trial on obese adults, BMI 28–40 kg/m^2, with mild-to-moderate (AHI 5–15 events/h) OSA at baseline. The 1-year lifestyle intervention consisted of a very low calorie diet (600–800 kcal/day) for 12 weeks, followed by a recommendation to reduce fat intake to <30% of total energy for the remainder of the year, combined with counseling sessions. Participants in the control group were provided with general information regarding diet and exercise. The intervention produced greater weight loss than control and differences in AHI between groups. A loss of 5–15 kg body weight was associated with a reduction in AHI of 4 events/h; losing >15 kg was associated with a reduction in AHI of 7 events/h. Any gain in weight was associated with a rise in AHI of 3 events/h. The authors subsequently described the findings of a 4-year post-intervention follow-up, in which no active counseling was provided to either group [74]. The mean change in body weight across the 5-year period was significantly different for participants in the treatment (−5.5 kg) vs. control (+0.6 kg) group as was the change in AHI (−0.8 events/h vs. + 5.0 events/h).

Foster and colleagues [72] conducted a randomized controlled trial on middle-aged to older adults with type 2 diabetes, with BMI>25 kg/m^2 (or ≥27 kg/m^2 if taking insulin), and AHI≥5 events/h. The intensive lifestyle intervention of reduced-calorie diet and an exercise regimen resulted in significantly larger weight loss and reductions in waist and neck circumferences compared to control, diabetes education group after 1 year. Also, the intensive lifestyle intervention group had a reduction in AHI of 5.4 events/h whereas the control group had an increase in AHI of 4.2 events/h. The change in AHI was significantly related to the change in body weight. Participants were followed for an additional 3 years [75]. During follow-up, weight loss remained significantly greater in the treatment vs. the control group and reductions in AHI persisted. Taken together, these three randomized controlled trials strongly suggest that weight loss induced by lifestyle modifications consisting of diet and exercise can be an effective means of reducing OSA severity.

Expert opinion

It is currently unclear how sleep duration and quality impact body weight. It is possible that poor sleep characteristics cause weight gain/obesity, or hinder weight loss. Conversely, weight gain/obesity could cause alterations in sleep characteristics, which may further influence body weight. Despite strong epidemiological evidence for associations between SSD/poor sleep quality and obesity, and increasing clinical evidence for a causal effect of sleep on weight status, studies are lacking to clearly show that SSD/poor sleep quality leads to actual changes in body weight. A similar pattern of evidence exists for sleep-disordered breathing and obesity. Once it is clearly shown that SSD/poor sleep quality can cause weight gain, then addressing the reverse question of whether increasing sleep duration/improving

sleep quality can improve weight loss will be warranted. Information from such studies would then lead to public health messages about appropriate sleep for weight management.

Outlook

We are aware that clinical trials are under way to assess whether extending sleep duration improves weight status in obese short sleepers. Results from such studies will prompt additional questions on the mechanism by which extending sleep can improve body weight control. To date, data are mixed regarding the impact of sleep duration on hormonal regulation of food intake. We hope that future studies will have clarified these questions.

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Highlights

- Short sleepers are heavier and gain more weight over time than normal sleepers.
- Short sleep hinders weight loss efforts compared to normal sleep duration.
- Sleep restriction increases food intake beyond the additional energetic costs of added wake time.
- Sleep architecture, particularly time spent in stage 2 and REM sleep, is related to energy balance parameters.
- Weight loss improves OSA.
- Future studies are needed to establish whether restricting sleep or inducing poor sleep quality hinder weight-loss efforts or cause weight gain.

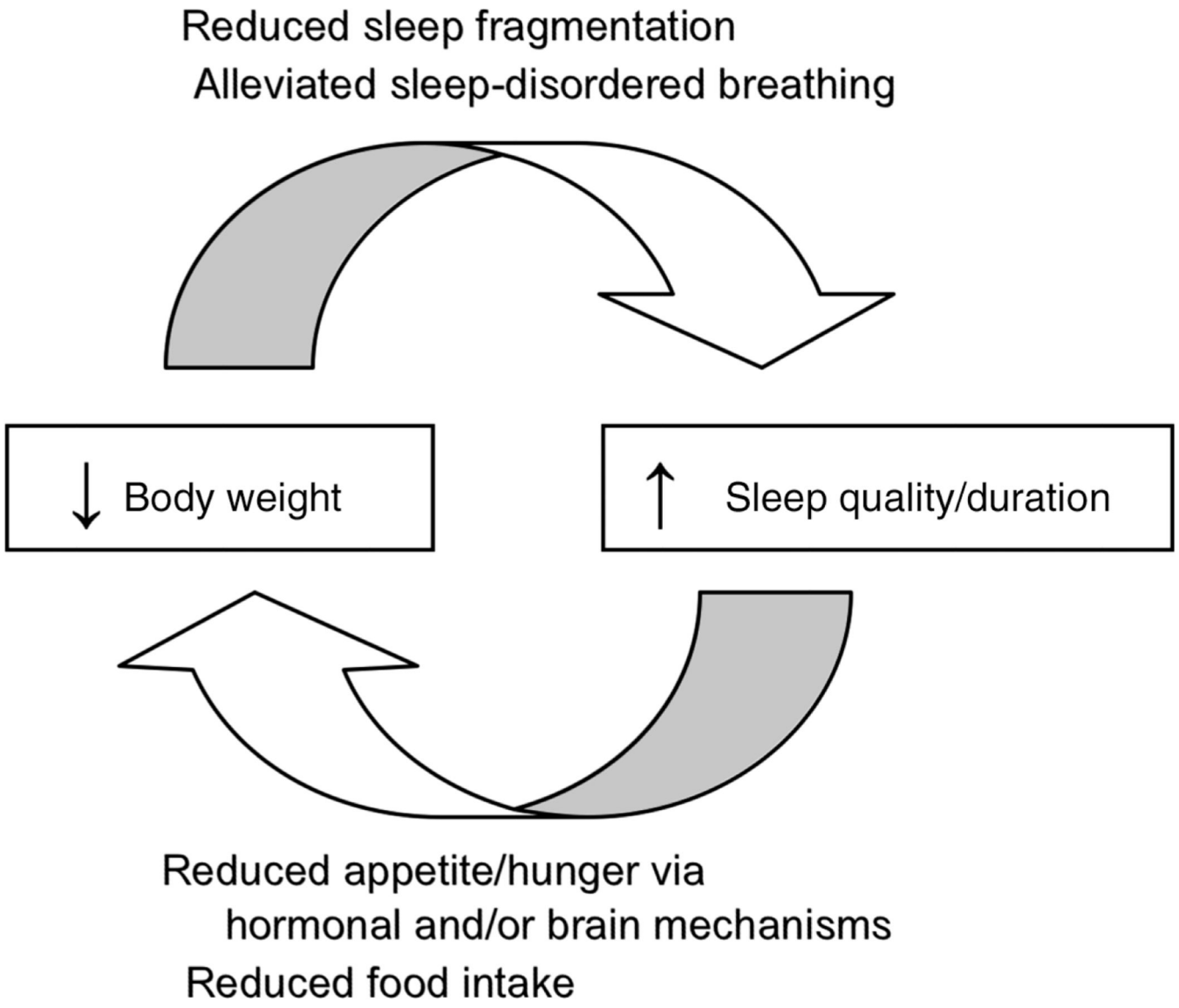

Figure 1.

Schematic diagram of the proposed reciprocal relationship between weight loss and improved sleep quality/duration. Putative mechanisms by which body weight and sleep quality/duration affect each other are listed in italics, and include (top) reduced sleep fragmentation and alleviation of sleep-disordered breathing in response to weight loss; and (bottom) reduced hunger and food intake via hormonal or brain mechanisms in response to improved sleep quality and duration.