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Sleep patterns, diet quality and energy balance

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HIGHLIGHTS

- Insufficient sleep is associated with increased caloric consumption, poor dietary habits, and obesity.
- Insufficient sleep increases snacking and the number of meals consumed per day.
- Excess energy intake associated with insufficient sleep is more driven by hedonic rather than homeostatic factors.
- Although certain foods may promote sleep or improve sleep quality, scientific data are lacking.

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ABSTRACT

There is increasing evidence showing that sleep has an influence on eating behaviors. Short sleep duration, poor sleep quality, and later bedtimes are all associated with increased food intake, poor diet quality, and excess body weight. Insufficient sleep seems to facilitate the ingestion of calories when exposed to the modern obesogenic environment of readily accessible food. Lack of sleep has been shown to increase snacking, the number of meals consumed per day, and the preference for energy-rich foods. Proposed mechanisms by which insufficient sleep may increase caloric consumption include: (1) more time and opportunities for eating, (2) psychological distress, (3) greater sensitivity to food reward, (4) disinhibited eating, (5) more energy needed to sustain extended wakefulness, and (6) changes in appetite hormones. Globally, excess energy intake associated with not getting adequate sleep seems to be preferentially driven by hedonic rather than homeostatic factors. Moreover, the consumption of certain types of foods which impact the availability of tryptophan as well as the synthesis of serotonin and melatonin may aid in promoting sleep. In summary, multiple connections exist between sleep patterns, eating behavior and energy balance. Sleep should not be overlooked in obesity research and should be included as part of the lifestyle package that traditionally has focused on diet and physical activity.

1. Introduction

Insufficient sleep (short sleep duration and/or poor sleep quality) has become pervasive in modern societies with 24/7 availability of commodities [1,2]. Factors responsible for this secular decline in sleep duration are numerous and generally ascribed to the modern way of living (e.g. artificial light, caffeine use, late-night screen time, parental attitudes) [3]. Although reducing sleep time is thought to be a good strategy to cope with time constraints, an accumulating body of evidence shows that lack of sleep exerts deleterious effects on a variety of systems, including detectable hormonal perturbations that may adversely impact health [4,5]. Epidemiologic studies have shown that both short and long sleep durations are associated with poor health outcomes including obesity [6,7], type 2 diabetes [8,9], coronary heart disease [10,11], hypertension [12,13], and premature death [14,15]. Although the association between sleep duration and health indicators has been reported to be U-shaped in many studies (especially in studies using self-reported sleep duration), the adverse effects of inadequate sleep appear far more important in today's environment [16].

Insufficient sleep has been reported to increase our vulnerability to overeat in the current obesogenic environment [17]. Overeating as a result of insufficient sleep is in part due to changes in cognitive functions involved in reward saliency and inhibitory control [18]. Furthermore, we have more time and opportunities for eating when spending more time awake, which allows for easy access to palatable foods. Although there is increasing evidence showing that sleep has an influence on dietary choices (e.g. short sleepers are more likely to consume energy-dense foods), the consumption of certain types of foods has also been shown to improve sleep. The objective of this narrative review is to briefly discuss the evidence linking sleep patterns (especially insufficient sleep and sleep timing) to appetite control, feeding behavior and energy balance.

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2. Is insufficient sleep a contributor to obesity?

2.1. Observational evidence

A growing body of epidemiologic evidence associates insufficient sleep with weight gain and obesity [19–21]. A recent systematic review and meta-analysis including 634,511 participants from around the world reported a pooled odds ratio of 1.89 (95% confidence interval: 1.46–2.43) in children and 1.55 (95% confidence interval: 1.43–1.68) in adults for short sleep duration and its relationship with obesity [22]. Although the majority of studies have found a significant association between inadequate sleep and obesity, the association appears stronger at younger ages, suggesting that children and adolescents may be more vulnerable to the effects of inadequate sleep.

It is however difficult to draw causal inferences with epidemiological studies due to the lack of control for important confounders in many studies (e.g. depression, psychosocial problems, chronic illness, use of hypnotics) and inconsistent evidence of temporal sequence in prospective studies. Furthermore, certain findings suggest that there may be bidirectional effects, such as insufficient sleep causing weight gain and obesity causing insufficient sleep, hence creating a setting for a vicious circle [23]. Another concern is that most studies rely on self-reported measures of sleep, with only a few studies that have assessed sleep using actigraphy. Finally, other aspects have the potential to hamper the interpretation of the evidence in terms of causality, such as the body weight history and the possibility of common or upstream underlying causes. Despite the limitations of epidemiologic studies examining the association between sleep and adiposity, the preponderance of the evidence taken as a whole points towards an effect of insufficient sleep on the vulnerability to develop obesity. Future epidemiologic research from large prospective cohort studies with objective assessment of sleep habits and repeated measures of both sleep and adiposity is needed to more adequately establish a causal link, and to better define the magnitude of any causal effect.

2.2. Experimental evidence

Many short-term intervention studies investigating the effects of sleep restriction on energy balance have been published over the last 10 years and have significantly contributed to our understanding of the potential mechanisms by which insufficient sleep may predispose to weight gain. The seminal study by Spiegel et al. [24] published in 2004 certainly helped to fuel this field of research by experimentally testing the acute effects of sleep restriction on feeding behavior and key appetite hormones. They observed that adults undergoing 2 nights of sleep restriction (4 h time in bed per night) with controlled energy intake via an intravenous glucose infusion exhibited increased levels of ghrelin (an orexigenic hormone) and decreased levels of leptin (an anorexigenic hormone) in conjunction with increased sensations of hunger and appetite, and this specifically for foods with high carbohydrate content. Other experiments have also shown that sleep restriction increased ghrelin levels and decreased leptin levels in caloricallyrestricted adults [25,26]. By contrast, in laboratory studies using ad libitum food access, which mimics a more natural environment, sleep restriction was associated with either no change in ghrelin or leptin or an increase in leptin levels [27–32]. However, ad libitum experiments are generally consistent in showing that sleep restriction is associated with increased caloric intake [27,33–35]. Differences in methodology (e.g. sleep timing) and the energy balance of study participants may account for the conflicting results obtained with regard to ghrelin and leptin levels. For example, delaying bedtime might not be the same as getting up earlier and future studies should better consider the implications of sleep timing on sleep architecture, food intake and metabolic and hormonal variables.

Interestingly, Spaeth et al. [36] recently examined the effect of experimental sleep restriction (5 consecutive nights of 4 h time in bed per night) on weight gain in 225 healthy adults under controlled laboratory conditions. They observed that sleep-restricted participants gained 1 kg more than control participants ($P = 0.007$), consumed extra calories (130% of daily caloric requirements), and this increase in daily caloric intake was due to an increase in meal consumption frequency, as well as the consumption of about 550 additional calories between 22:00 and 03:59. This suggests that sleep-restricted adults with later bedtimes may be more susceptible to weight gain due to greater daily caloric intake and the consumption of excess calories during late-night hours. Similarly, Markwald et al. [37] also quantified the effects of 5 days of insufficient sleep (5 h of sleep per night), equivalent to a work week, on weight gain in 16 adults. They found that insufficient sleep led to a 0.82 kg weight gain ($P < 0.05$), and concluded that increased food intake during insufficient sleep is a physiological adaptation to provide energy needed to sustain additional wakefulness. They also observed that transitioning from an insufficient to adequate/recovery sleep schedule decreased energy intake, especially the intake of fats and carbohydrates, and led to weight loss [37].

However, the ecological validity of these laboratory experiments is poor because they generally involve abnormally large sleep restrictions, unnatural environments without medications or caffeine, and a limited behavioral repertory for handling restricted sleep (for example it is usually not allowed to go for a walk outside in the fresh air). Because laboratory studies have used large restrictions in sleep, we do not have a good sense of dose–response relationships and perhaps small sleep restrictions do not matter. Another limitation of experimental studies in this field of research is that they are short-term, lasting only a few days. This raises the question of whether these effects can persist outside the laboratory setting when sleep restriction is chronic. Unfortunately, experimental evidence that sleep restriction induces obesity is not possible in humans for both ethical and logistic reasons. The "gold standard" would be to conduct a randomized controlled trial in which we would have to restrict sleep duration in a group of lean individuals in order to be able to compare them with a control group. However, the slow development of obesity implies that such a study would have to run for years in a large sample of individuals. Accordingly, we need to rely on short-term intervention studies as well as epidemiologic studies. Although the proof-of-concept will never be complete in this field of research, the evidence taken as a whole suggests that insufficient sleep plays a role in the risk of developing obesity. Therefore, there is minimal risk in taking a pragmatic approach and encouraging a good night's sleep as an addition to other health promotion measures [38–40].

3. Influence of insufficient sleep on energy balance

3.1. Insufficient sleep and energy expenditure

Sleep influences energy metabolism, and one of its main functions is to conserve energy [41]. One of the proposed mechanisms that associate insufficient sleep to weight gain is a decrease in energy expenditure. A recent well-designed study examining the impact of 5 days of insufficient sleep (5 h of sleep/night) using a whole room calorimetric chamber found an average increase of ~5% (~111 kcal/d or ~464 kJ/d) in 24-h energy expenditure during sleep loss compared with a control session (9 h of sleep/night), which is similar to the energy cost of a 70-kg adult performing aerobic exercise for ~24 min [37]. This increase in total daily energy expenditure with sleep loss was predominantly driven by the energy cost of additional wakefulness. However, the increased fatigue and tiredness associated with sleeping too little could result in reduced voluntary physical activity in some individuals. For example, short-term sleep restriction (2 nights of 4 h in bed) was accompanied by a decrease in daytime spontaneous physical activity in healthy men [31]. Interestingly, the reduction in overall physical activity was a shift towards less intense activities under free-living conditions. However, while sleep restriction may decrease physical activity energy expenditure in