Review

Sleep Duration and Obesity in Children and Adolescents

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Key Messages

- Epidemiological and experimental research demonstrates that short sleep duration is a risk factor for obesity in children and adolescents.
- Evidence suggests short sleep duration may influence child and adolescent obesity through changes in appetite regulation, poor diet and reduced physical activity.
- Interventions that target child and adolescent obesity via sleep extension show some promise, but further research is needed.

ABSTRACT

Increased rates of obesity internationally have drawn significant attention. In particular, researchers and practitioners are seeking new information about risk factors for obesity that could be areas for preventive interventions. Given that obesity rates in children and adolescents are increasing worldwide, particular attention to child and adolescent obesity is needed. A large, and growing, body of research indicates that inadequate sleep duration is linked to obesity. The current article reviews the extant literature concerning sleep duration and obesity in children and adolescents by reviewing current theories of obesity as well as available literature specifically evaluating the relationship of obesity and sleep in children and adolescents, including epidemiologic, experimental and intervention research. Overall, our literature review concludes that the relationship between shortened sleep and increased risk for obesity has research support internationally, including in the few Canadian articles available that are discussed in our review.

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RÉSUMÉ

L'augmentation des taux d'obésité dans le monde a beaucoup retenu notre attention. Notamment, les chercheurs et les praticiens tentent d'obtenir de nouvelles informations sur les facteurs de risque d'obésité qui pourraient potentiellement constituer des domaines d'interventions préventives. Puisque les taux d'obésité chez les enfants et les adolescents augmentent dans le monde entier, l'obésité de l'enfant et de l'adolescent doit faire l'objet d'une attention particulière. Un corpus vaste et croissant de recherche montre qu'une durée inadéquate de sommeil est liée à l'obésité. Dans le présent article, nous passons en revue la littérature existante sur la durée du sommeil et l'obésité de l'enfant et de l'adolescent ainsi que les théories actuelles sur l'obésité et la littérature qui porte précisément sur l'évaluation de la relation entre l'obésité et le sommeil chez les enfants et les adolescents, y compris la recherche épidémiologique, expérimentale et interventionnelle. Dans l'ensemble, la revue de littérature permet de conclure que la relation entre le sommeil de courte durée et l'augmentation du risque d'obésité reçoit l'appui de la recherche dans le monde entier, y compris des rares articles canadiens sur lesquels porte notre revue.

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Introduction

Declining sleep time in children and adolescents has been attributed to various aspects of modern society (1). For example, the introduction of electricity and corresponding exposure to artificial light has been proposed as a plausible mechanism for extended waking hours (2). Concurrent with this decrease in sleeping time has been

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an increase in childhood obesity. Over the past 30 years, a growing body of experimental and epidemiologic research has linked short sleep duration with obesity. Given that sleep could be a modifiable risk factor, this sleep/obesity connection has stimulated significant research activity.

Although the function of sleep is not entirely understood, clues have emerged through examining what happens when people do not get enough sleep. Although to some degree, sleep requirements vary from individual to individual, most adults require between 7 and 9 hours of sleep (3). In children and youth, sleep requirements vary substantially by developmental stage. The most recent recommendations from the National Sleep Foundation are 9 to 10 hours of sleep per day for children 6 to 13 years of age and 8 to 10 hours for adolescents aged 14 to 17 years (3).

Sleep loss can also be categorized as partial or total. Partial sleep loss means getting fewer hours than needed to feel refreshed. In population-based research, people who chronically experience partial sleep loss can also be thought of as "short sleepers" or those with "short sleep duration." Partial sleep loss has been linked to declines in human functioning and health. For example, acute periods of partial sleep loss are associated with declines in cognitive functioning (4) and alterations in hormones involved in glucose regulation such as insulin and cortisol (5). Given that long-term experimental studies examining partial sleep loss would be difficult due to ethical and methodologic reasons, epidemiologic studies have been conducted to explore the effects of chronic partial sleep loss. Findings from these population-based studies have linked short sleep duration to poorer physical health (e.g. cardiometabolic diseases) (6) and declines in functioning (e.g. reaction time) (4). In comparison to partial sleep loss, total sleep deprivation during a period of time refers to a complete lack of sleep over that time frame. However, studies exploring the effects of total sleep deprivation in humans can be conducted only on a short-term basis because humans exhibit involuntary microsleeps after a few days of total sleep deprivation (7). Experimentally, short periods of total sleep deprivation are associated with declines in mental, emotional and physical health and functioning (4,5,7,8). For example, the results of a meta-analysis indicated that acute total sleep deprivation resulted in declines in cognitive function, including attention, processing speed, memory and reasoning (7). In a British study, a period of 17 to 19 hours without sleep induced declines in accuracy and response time comparable to measurements taken in people with blood alcohol levels of 0.05% (9). Short periods of total sleep deprivation have also been linked to deteriorations in indicators of cardiometabolic health (6) and immune function (10).

Evidence suggests that a bidirectional relationship links short sleep duration to obesity and other indicators of cardiometabolic disease (11). For example, carrying excess weight may adversely affect sleep quality; conversely, poor sleep quality could result in weight gain (12). Another example of this bidirectional effect is sleep-disordered breathing, a cause of sleep loss resulting from frequent interruptions in respiration during sleep (12). Sleep-disordered breathing is strongly associated with obesity and other indicators of cardiometabolic disease (13). It is thought that obesity plays a causal role in sleep-disordered breathing because it commonly occurs in obese individuals, and weight loss can result in improvements in sleep-disordered breathing as well as in cardiometabolic health (13). On the other hand, in studies that controlled for obesity, sleep-disordered breathing and concurrent sleep loss remain independently associated with poorer cardiometabolic health (14,15). Additionally, improvements in cardiometabolic health have been demonstrated after treatment of sleep-disordered breathing with continuous positive airway pressure (16) and tonsillectomy (17). However, continuous positive airway pressure does not result in weight loss (18), suggesting that the pathogenic mechanisms underlying the linkage between sleep-disordered breathing and

cardiometabolic risk are at least partly attributable to factors other than obesity.

Another way that sleep is connected to body-mass regulation is through the overlap in human genes that influence both circadian rhythms and endocrine hormones. Circadian Locomotor Output Cycles Kaput (CLOCK) genes are involved in orchestrating bodily circadian rhythms; their impact on neuroendocrine systems could influence obesity (19,20). CLOCK gene variants have been linked to sleep duration (21) and sleep timing (22) as well as to obesity (23), metabolic syndrome (24) and calorie intake (25). Patterns of CLOCK gene methylation have also been linked to body mass index, insulin resistance, total energy intake and carbohydrate intake (26).

Short Sleep Duration and Overweight/Obesity

Energy imbalance theory of obesity

Possible mechanisms for the observed connections between short sleep duration and obesity can be illuminated by the energy imbalance theory of obesity. For weight gain to occur, an imbalance between energy intake and energy expenditure must exist (27). Food consumption is mediated homeostatically (i.e. through sensations of appetite/hunger) as well as through hedonic, or pleasure-based, eating behaviour (28). Energy expenditure can occur voluntarily through physical activity or involuntarily through basal metabolism and thermogenesis. Short sleep duration could cause weight gain through both sides of this energy balance equation (27).

Short sleep duration dysregulates homeostatically mediated energy intake.

Homeostatic control over appetite is orchestrated through complex interactions among many neuroendocrine hormones. Key hormones thought to be involved in the sleep-obesity relationship include leptin, ghrelin, insulin and cortisol (28). Leptin and ghrelin have opposing functions in appetite regulation (29); leptin is released primarily from fat cells and is thought to suppress appetite, while ghrelin is secreted primarily from the stomach and stimulates hunger (29). Insulin plays an important role in the regulation of blood glucose levels (30). When blood glucose levels rise, insulin is secreted by the pancreas, and glucose is transported out of the blood and into the cells (30). Cortisol is part of the body's stress response and results in increased arousal, elevations in blood glucose and suppression of the immune system (30).

Experimental studies have demonstrated that partial sleep loss disrupts levels of leptin, ghrelin, cortisol and insulin (31–34). For example, in healthy young men, when sleep time was restricted to 4 hours of sleep per night in bed over 2 days, participants exhibited an 18% decrease in leptin and a 28% increase in ghrelin compared to when they spent 10 hours in bed (33). Participants also experienced a 24% increase in hunger after the period of sleep loss, with larger increases in appetite for high-carbohydrate foods (33). Similar findings were demonstrated by Schmid et al (32); a single night of partial sleep loss resulted in elevations in both ghrelin and sensations of hunger.

Increased waking time equals increased eating time

Initially, the idea that sleep loss could cause obesity might seem counterintuitive because sleep is a sedentary act, and having more time awake could provide more time for energy expenditure. Interestingly, research suggests that the opposite is true. In modern societies, people tend to consume more calories than they expend (35). In other words, spending more time awake tends to worsen the energy imbalance that underlies obesity because people tend to overcompensate for any increased energy expenditure that occurs during the additional hours spent awake (35). This observed increase in

energy intake resulting from sleep loss may be a consequence of increased appetite as well as nonappetite-based eating behaviour (35,36). For example, adults exposed to a sleep restriction period exhibited changes in responsivity to food cues in the reward and control regions of the brain (37). Further research is needed to examine sleep restriction and energy balance over the long term. For example, although research demonstrates that energy intake may increase acutely after periods of sleep restriction, it is uncertain whether people compensate by eating less later on.

Several aspects of modern lifestyles have been shown to stimulate nonappetite-mediated food consumption (27,38). Modern lifestyles facilitate obesity through the combination of easy access to calorie-dense foods and frequent engagement in sedentary activities that are associated with eating (35,39). Thus, short sleep (i.e. increased time awake) translates into increased exposure to the obesogenic modern environment. It is intuitive that sedentary activities, such as television watching and computer use, are associated with reduced energy expenditure, but research suggests that sedentary activities also result in increased food consumption independent of appetite (27). Sedentary activities such as use of electronic devices are thought to result in increased calorie intake due to disconnection from internal satiety signals, reducing conscious control over food consumption, and to exposure to food-related advertising (35). For example, Blass et al (40) found that meals consisting of macaroni and cheese or pizza consumed over a 30-minute period of television watching resulted in an additional 288 kcal consumed compared to meals consumed while not watching television. Similar findings have been demonstrated with increased food consumption during meals consumed after video game playing (+80 kcal) (35) and after cognitive working, such as reading and writing (+229 kcal) (35,41). Listening to music while eating has also been associated with increased calorie consumption (+107 kcal) (42).

Nonappetite-mediated eating behaviour also links short sleep duration to obesity. In a crossover study of 11 healthy participants, eating behaviour was compared between a sleep-restriction condition (5.5 hours of sleep per night for 14 days) and an adequate sleep condition (8.5 hours of sleep per night for 14 days) (43). Sleep restriction did not affect leptin or ghrelin levels in the study's participants, but increased calorie consumption was observed. Additionally, energy expenditure did not increase to compensate for the increased energy intake. It is interesting that although energy consumption during meals was similar in both conditions, increased snacking behaviour was observed during sleep restriction, especially during times when study participants would normally be sleeping. Several other studies support this finding that partial sleep loss is followed by increased calorie consumption (44,45). Short sleepers may also eat more in an attempt to offset the fatigue associated with sleep debt. Short sleepers frequently report fatigue and daytime sleepiness (7), and it has been hypothesized that sleep-restricted individuals might increase calorie intake in an attempt to counteract the fatigue associated with sleep loss (11).

Short sleep duration reduces voluntary energy expenditure

Sleep restriction may also contribute to the other side of the energy-balance equation through decreased energy expenditure. Energy expenditure occurs both voluntarily (e.g. through physical activity) and nonvoluntarily (e.g. to power basic metabolic processes). Fatigue consequent to sleep restriction could result in reduced motivation to participate in vigorous physical activity (11). Schmid et al (46) found that after 2 nights of sleep loss (4 hours of sleep per night), physical activity was reduced in healthy men during free-living conditions. Short sleep duration reduces involuntary energy expenditure.

Involuntary energy expenditure occurs to support basic metabolic processes, to maintain body temperature and to dispose of excess energy intake (47). Although research in this area is sparse, it has also been hypothesized that sleep duration could affect involuntary energy expenditure (48). Melatonin plays an important role in the regulation of involuntary energy expenditure (47). As such, the suppression of melatonin due to late-night light exposure is another proposed mechanism that could explain the linkages between sleep loss and obesity (49).

Melatonin also could influence involuntary energy expenditure via the growth and activity of brown adipose tissue. The human body contains 2 types of fat: white adipose tissue and brown adipose tissue (30). The function of white adipose tissue is energy storage and padding, whereas brown adipose tissue is metabolically active (30). Brown adipose tissue is thought to have several roles in human physiology, including heat generation to maintain body temperature (i.e. nonshivering thermogenesis) (30) and maintenance of energy balance by disposing of excess energy intake through heat production (i.e. diet-induced thermogenesis) (47). Through these mechanisms, brown adipose tissue burns energy and reduces white adipose tissue stores (47). In animals, melatonin stimulates the activity and growth of brown adipose tissue (47). As such, it is possible that melatonin suppression linked with late-night light exposure could result in decreased energy expenditure and an increase in white adipose tissue reserves (47). In humans, melatonin administration has a hypothermic effect on core body temperature but simultaneously occurs with proportionally larger increases in peripheral body temperature and peripheral heat loss (47). No studies thus far have examined the effect of sleep loss on brown adipose tissue activity in humans.

Epidemiologic Evidence for a Sleep-Obesity Link in Children and Adolescents

Review articles

A sizable body of research has explored the relationship between sleep duration and overweight/obesity in both children and adults (50–55). Meta-analyses of epidemiologic studies involving children and adolescents observed a significantly increased risk for overweight and obesity in short sleepers compared to longer sleepers (50,51). In a meta-analysis of 13 studies involving 30,002 children and adolescents aged 2 to 20 years, short sleepers had a pooled odds ratio of 1.89 for obesity (50). Similarly, in a systematic review of 11 studies, youth falling into the shortest category of sleep duration had an odds ratio of 1.92 for overweight/obesity compared to the longest sleepers (51). Reviews of longitudinal studies also demonstrate a link between sleep duration and overweight/obesity; in a meta-analysis of 25 studies and 56,584 children and adolescents, children with the shortest sleep durations had increased odds of being overweight or obese (odds ratio 1.76) (56). Similarly, Fatima et al (57) determined that short sleepers had higher odds of overweight/obesity (odds ratio 2.15) in their meta-analysis of 22 longitudinal studies involving 24,821 children and youth.

Cross-sectional studies: children and adolescents

Cross-sectional research examining associations between sleep duration and overweight/obesity in children and adolescent populations are more consistently positive than studies involving adults (51,53). A pediatric obesity-sleep research study completed in Canada was the Québec en Forme project, which involved 422 Québec children aged 5 to 10 years (58). After adjusting for age, sex and obesity risk factors, a negative linear relationship was observed between sleep duration and overweight/obesity (58). Compared to children

sleeping 12 to 13 hours per night, odds ratios for overweight/obesity were 1.42 for those with 10.5 to 11.5 hours of sleep per night and 3.45 for those with 8 to 10 hours of sleep per night (58). More recently, Sluggett et al evaluated data from the Canadian Health Measures Survey and found increased odds of overweight/obesity in short-sleeping boys (odds ratio 1.85) and in short-sleeping adolescents of both sexes (odds ratio 1.77) (59).

Cross-sectional studies from other countries support these findings from the few available Canadian studies. A recent study of 66,817 Chinese adolescents found that short sleepers had increased odds of overweight/obesity (60). Compared to the reference category, adolescents in the shortest sleep category had higher odds of overweight (odds ratio 1.26) and obesity (odds ratio 1.24). In an Australian study, Eisenmann et al (61) studied the relationship between sleep duration, waist circumference and body mass index in 6,324 youth 7 to 15 years of age. Compared to those sleeping more than 10 hours per night, those who slept fewer than 8 hours per night had an odds ratio for overweight/obesity of 3.1 (61). Morrissey et al (62) provided data showing a strong positive relationship for short sleep and obesity in a sample of 9- to 14-year-old Australian adolescents (n=157). In a cross-sectional study of 4,486 adolescent participants in the US National Longitudinal Study of Adolescent Health, Knutson (63) identified a negative relationship between sleep duration and body mass index in males only. In a study of Chinese children, Cao et al (64) concluded that short sleep duration should be considered a risk factor for obesity in girls and in 13- to 18-year-old boys but a protective factor for boys 6 to 12 years of age. Given the evidence that the link between sleep and obesity may differ according to age and sex (59,63,64), these variables should be considered carefully when drawing any conclusions regarding short sleep duration as a risk factor for obesity.

Longitudinal studies: children and adolescents

Longitudinal research demonstrates that short sleep duration is associated with obesity as well as an increase in body mass index over time. A Chinese prospective cohort study supported the oft-cited U-shaped distribution for this relationship: namely, they found that both oversleeping (≥13 hours) and undersleeping (≤10 hours) preschoolers were at high risk for obesity when compared to preschoolers sleeping 11 to 12 hours per night (65). In a British study, Reilly et al (66) studied 8,234 7-year-old children over 4 years; short sleep duration predicted higher body mass index at follow up (odds ratio 1.45). Similar associations have been observed in studies undertaken in the United States with 2,281 children aged 3 to 12 years over a 5-year period (67) as well as 9-year-olds over 4.5 years (68). A long-term prospective New Zealand cohort study had similar results: short sleep durations in childhood predicted a higher body mass index at age 32 after adjusting for confounders (69). Although the majority of research supports a link between short sleep duration and overweight/obesity, a few studies contradict these findings. For example, in a Danish cohort study of infants, sleep duration at 9 and 18 months did not predict adiposity at age 3 years (70). Similarly, in a Canadian study involving 597 children, short sleep duration at 12 to 36 months did not predict cardiometabolic risk cluster scores or obesity at 36 to 96 months (71). The comparatively young age of the participants and the relatively short follow-up time could be factors in the lack of findings in these studies.

Although the majority of the published literature has focused on sleep duration as a risk factor for obesity, research also has explored associations between obesity and other sleep-related variables, such as sleep timing, sleep variability and sleep quality. Sleep timing can be a particular problem in adolescents due to natural shifts in circadian rhythms that favour late bedtimes and a preference to sleep later in the mornings. As such, adolescents commonly exhibit "social jet lag" due to misalignment between their natural

circadian rhythms and social and environmental factors (72). In a study of adolescents 12 to 17 years of age, after controlling for sleep duration, later bedtimes and greater bedtime shift were positively associated with being overweight (73). Adolescents with late bedtimes are more likely to have higher body mass indexes and exhibit poor diet choices (74,75). In an Australian study of 2,200 children and adolescents 9 to 16 years of age, compared to participants with an early-bed/early-rise sleep pattern, participants with a late-bed/late-rise sleep pattern had higher body mass index z-scores, higher energy intakes and poorer diet quality (76). Participants with a late-bed/late-rise pattern also exhibit less moderate-to-vigorous physical activity and more screen time (77). Other sleep-related measures linked to obesity include the degree of regularity in sleeping schedules (78) and sleep quality (79).

The majority of the population-based studies discussed thus far used self-reported measures of sleep duration. Due to the expense and invasiveness of objective measures of sleep duration, such as actigraphy and polysomnography, epidemiologic studies involving children and adolescents have frequently utilized self-reported sleep durations. However, smaller studies using objective measures of sleep duration such as actigraphy also support these findings, providing even stronger evidence for the obesity-sleep duration relationship (74,80–82).

Experimental studies

Experimental studies provide strong evidence supporting a causal link between sleep restriction and obesity in children and adolescents, with increased calorie consumption evident under conditions of sleep restriction. In a randomized crossover trial, adolescents 14 to 17 years of age consumed more calories under a sleep-restriction condition compared to the normal sleep-length condition (83). The additional calories consumed during sleep restriction were skewed toward an increase in carbohydrates (83). In a study conducted by Hart et al, children 8 to 11 years of age consumed more calories and exhibited lower fasting leptin during a sleep-extension condition vs. a sleep-restriction condition (84). In another randomized crossover study of adolescents 14 to 17 years of age, calorie intake was 11% higher during sleep restriction compared to normal sleep duration with, again, a particular emphasis on increased consumption of sweets and dessert foods rich in carbohydrates (85). Further, when presented with pictures of various types of foods, participants exposed to sleep deprivation rated the sweet/dessert foods as more appealing (85). In contrast to this evidence supporting a link between sleep restriction and positive energy balance, in a randomized crossover trial of 21 adolescent boys involving sleep restriction over 3 nights, participants exhibited a small negative energy balance under the sleep-restriction condition compared to the long-sleep condition (86). Although participants exhibited reduced diet-induced thermogenesis during sleep restriction, overall energy balance was decreased due to reduced motivation to eat and decreased food intake as well as increased energy expenditure through physical activity (86). Leptin and ghrelin concentrations were not different under sleep restriction vs. normal sleep (86). Although the findings from this randomized controlled trial contradict other experimental studies that found a positive increase in energy balance under sleep restriction, this study took place inside a highly controlled laboratory setting. As such, the findings from this study may not be generalizable to participants in free-living conditions.

Time usage analysis

A relatively new approach in sleep-obesity research is the utilization of a compositional analysis approach to examine activity levels. Because increased time spent engaged in 1 activity reduces time spent doing something else, rather than considering the

absolute length of time spent in a single activity, it may be advantageous to consider the proportions of time spent in different activities over a 24-hour period (e.g. proportions of time spent in sleep, sedentary activity, light activity and moderate-to-vigorous activity). The results of compositional analyses published thus far suggest that replacing time spent in sedentary activities with time sleeping could reduce obesity in children and adolescents (87,88).

Supporting these compositional analyses are other studies demonstrating that short sleepers spend more time engaging in sedentary activities such as TV watching and use of smartphones, videogames and computers. Furthermore, short sleepers are also likely to have poorer diet quality and be less physically active. For example, screen time, especially in the evening, has been linked to reduced sleep duration in children (89–91) and adolescents (92). In adolescents, increased screen time is linked to inadequate sleep as well as to the consumption of sugar-sweetened beverages, inadequate physical activity and obesity (39). Late bedtimes in adolescents are also associated with unhealthful diet practices and less physical activity (73). Similar findings have been observed in children; in an multicountry study of 5,873 children 9 to 11 years of age, short sleepers consumed more sugar-sweetened beverages (93). These associations between short sleep duration and sweetened-beverage consumption are also supported by research in Canadian adolescents (94). Although evidence suggests an association between sugar-sweetened beverage consumption and short sleep duration, the cause-and-effect relationships are likely to be complicated. For example, although short sleepers may consume more sweet drinks in an attempt to offset fatigue, the sugar and caffeine present in these drinks could likewise lead to poorer sleep quality and/or delayed sleep onset.

Interventions

In recent years, interventions that aim to address obesity by improving sleep have been developed. These interventions typically involve educational programs targeted to parents and/or children and adolescents or policy changes, such as later school starting times. Strategies employed in educational programs include changing perceptions and beliefs about sleep, improving sleep hygiene (e.g. creating a dark, cool and quiet sleeping area), assisting participants in gradually moving toward earlier bedtimes, avoiding caffeine, alcohol and nicotine before bed, and limiting sleep-disturbing activities, such as technology use and vigorous physical activity close to bedtime (74).

In an intervention for obese adolescents, participants who achieved sleep extension also exhibited lowered body mass index after treatment (82). Interventions that aimed to extend sleep through earlier bedtimes resulted in consumption of fewer high-glycemic index foods post-treatment (95) and overall reduced caloric intake (96). Later school start times have also shown significant promise in extending sleep duration in adolescents and improving functioning (97). For example, in adolescents, delaying the school start time by 25 minutes resulted in improvements in sleep duration, with the number of students receiving 8 or more hours of sleep per night doubling from 18% to 44% (98). While later school start times show significant promise for successful sleep extension in adolescents, further research is needed to determine whether later school start times can affect obesity.

Although interventions that target sleep duration in children and adolescents can have beneficial effects on obesity, diet and physical activity, some research indicates the benefits may extend only to specific subgroups. For example, in the aforementioned intervention conducted by Beebe et al (96), the effectiveness of the intervention differed by chronotype (96). "Morning lark" chronotypes exhibited reduced calorie intake in response to sleep extension, but

no effect was observed in participants who exhibited the "night owl" chronotype (96). Similarly, in the intervention implemented by Valrie et al (82), despite achieving sleep extension, adolescents with the greatest sleep debt were less likely to achieve weight loss. Another challenge is the length of time an intervention remains effective. For example, when measured immediately postintervention, a sleep-education program improved parental knowledge, attitudes and self-efficacy for healthful child sleep habits, but this effect was not observed when measured again 1 month later (99).

Conclusions

A large collection of population-based studies, experimental studies and intervention studies provide evidence that short sleep duration is a risk factor for obesity in children and adolescents. Although there is some evidence suggesting that short sleep is linked to obesity through poor diet and less physical activity, further research is needed to investigate causal pathways. In particular, studies that utilize a compositional approach show promise in understanding these mechanisms. Finally, further research is needed to develop and evaluate interventions that can effectively improve both sleep duration and obesity over the long term in children and adolescents.

Funding

Author Disclosures

Conflicts of interest: None.

Author Contributions

The article was conceived and designed by LS, SLW and RLH. LS wrote the initial draft of the manuscript, while SLW and RLH provided critical edits and additions. All authors approved the final manuscript.

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