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Sleep patterns and obesity in childhood

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Abstract

Purpose of review—To highlight the recent findings on sleep–obesity associations in children. We focus on sleep duration, sleep timing and chronotype, and describe the potential mechanisms underlying sleep–obesity associations.

Recent findings—Poor sleep is increasingly common in children and associations between short sleep duration in early childhood and obesity are consistently found. Less is known about the infancy period, and the findings in adolescents are inconsistent. Sleep timing patterns may also contribute to obesity risk. Variable and shifted sleep schedules and evening chronotypes have recently been linked to adiposity in adults; less is known about children. Further, there is little understanding regarding the mechanisms of association. The timing of eating, dietary intake, obesogenic eating behaviors, and changes in appetite-regulating hormones have been identified as possible mechanisms for sleep–obesity associations and may be promising avenues for future research. Longitudinal and experimental work with children is needed to determine the nature of associations.

Summary—Beyond sleep duration, sleep timing patterns may contribute to obesity risk. Biological and behavioral processes have been proposed as mechanisms that may explain the association. Understanding the pathways through which poor sleep patterns could increase obesity risk in children may provide novel avenues for intervention.

Keywords

BMI; child obesity; chronotype; eating behavior; sleep timing

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Conflicts of interest

None.

INTRODUCTION

Obesity is widespread in the USA, with a prevalence rate of 35% in adults and 17% in children [1]. Short sleep duration is also common [2] – more than 30% of adults sleep less than 6 h per night [3], only 20% of adolescents get their optimal 9 h of sleep on school nights, and one-third of 2–3 year olds sleep less than recommended [4]. An association between short sleep duration and obesity or adiposity during early childhood has emerged in numerous epidemiological studies over the last decade; however, sleep–obesity associations at other points in development are less well studied. Further, understanding the multiple aspects of sleep patterns, rather than sleep duration alone, may identify the contributing mechanisms and provide new opportunities for sleep-focused interventions to prevent obesity. This review will provide updates in three areas, focusing on studies published since 2013: sleep duration–obesity associations during infancy and adolescence; associations of sleep timing and chronotype with obesity; and selected biobehavioral mechanisms of association between poor sleep patterns and obesity risk.

DEVELOPMENTAL DIFFERENCES IN SLEEP DURATION–OBESITY ASSOCIATIONS

Studies have consistently identified an association between short sleep during early childhood (age 3–7 years) and either concurrent or later obesity (see [5] for review). In comparison, a gap exists in understanding the sleep duration in relation to adiposity during infancy and toddlerhood (prior to age 3 years), even though sleep problems and sleep loss are common at this age [4]. A recent longitudinal study found that chronic short sleep from infancy through school age was associated with increased adiposity indicators and obesity [6▪▪]; associations between short sleep in infancy and adiposity at age 3 years had been previously reported in this cohort [7]. Similar to most epidemiological studies, this analysis relied on parent-reported sleep. Results from the few studies using actigraphy to measure infant sleep have been mixed: one study found the expected association between short nighttime (though not daytime) sleep and greater weight-for-length at age 6 months [8], and another found no association between infant sleep (parent reported at 9 and 18 months, actigraphy at 36 months) and adiposity at 36 months [9▪▪]. Incorporating the objective measurements of sleep such as actigraphy in future work may be important in clarifying the nature of the association (if any) and better articulate the mechanism underlying early-emerging sleep–obesity associations.

Later school age and adolescent periods also remain understudied compared with early childhood, despite adolescents' known short sleep duration because of interactions between sleep bio-regulatory processes (i.e., circadian phase delay and attenuation of the buildup of sleep pressure) and external constraints such as school start times [10]. Studies of sleep duration–obesity associations during adolescence have yielded mixed results, perhaps in part because of the methodological differences (see [11▪] for a recent review). A recent cross-sectional study found short sleep–adiposity associations for both boys and girls at age 9 [12▪▪] and 10 years [13▪], whereas a longitudinal study of 9–11 year olds found associations between short sleep and BMI for girls, but not for boys [14▪▪]. Among older adolescents, a cross-sectional study has shown the expected inverse relationship between short sleep

duration and adiposity, with frequent sex differences. For example, the association was present for younger boys (age 13 [15] and 16 years [16▪,17▪]) and to some degree for older girls (age 17 years [15]). Longitudinal studies across this age range have been inconclusive. In boys, short sleep at 13 years predicted higher BMI *z*-score at age 17 [15], though no association was observed for boys or girls between changes in sleep duration and changes in adiposity across age 14–18 years in another study [18▪]. Still another study found that short sleep at age 14 predicted weight gain across age 14–18 years, with the strongest associations found among heavier adolescents [19▪▪]. Thus, the mechanisms through which short sleep may confer obesity risk during adolescence are complex and may differ from the early childhood period. Longitudinal work that can identify sleep–growth associations at multiple timepoints is vital (see [20▪▪]). Furthermore, as child sex may moderate these associations during the adolescent period, it will be important to consider and assess pubertal status in future work.

BEYOND SLEEP DURATION: SLEEP TIMING AND PATTERN

Sleep timing is emerging as an important mechanism through which poor sleep patterns may confer obesity risk in adults [21], but less is known about sleep timing–obesity associations in infants, children, or adolescents. Aspects of poor sleep timing include late bedtimes and more variable patterns, including variability throughout the week and between weekday and weekend sleep schedules, which indicate a ‘shifted’ or ‘delayed’ sleep phase.

Late bedtimes

Two reports in the last year specifically examined sleep timing and obesity in early childhood, finding that late bedtimes (after 9 p.m.) magnified [22▪▪] and independently predicted [23▪▪] the association between short sleep duration and obesity. Studies of school-age children and adolescents (age 8–17 years) also found that late bedtimes were associated with adiposity, independent of sleep duration [24▪▪,25▪▪]. In a small study of obese 13 year olds (n = 26), late bedtimes were related to increased cumulative energy intake and more screen time, even though total sleep duration was the same for ‘late-sleeping’ (mean bedtime 10 p.m., mean waketime 7 a.m.) and ‘early-sleeping’ (mean bedtime 11 p.m., mean waketime 8 a.m.) groups [26▪▪]. Although this finding needs to be replicated in a larger sample, it suggests that late bedtime may be a unique contributor to obesity risk.

Variable and shifted schedules

Earlier work found that greater weekday-to-weekend variability in sleep timing, in combination with shorter sleep duration, was associated with obesity and poorer metabolic health in 4–10-year-old children [27], and that school-age children and adolescents (9–16 years) demonstrating a late-bedtime/late-rise time pattern were more likely than those with an early-bedtime/early-rise time pattern to be overweight and engage in more screen time and less physical activity [28]. More recently, variability between weekend and weekday sleep schedules has been associated with adiposity in school-age children and adolescents, independent of sleep duration. One study [29] found that more overweight adolescents (14 years of age) had late weekend compared with weekday bedtimes, but there was no concurrent association between sleep duration and weight status. In slightly younger

children (12 years of age), oversleeping on the weekends was associated with increased adiposity, and the association with sleep duration was attenuated after accounting for sleep timing [25▪▪]. A study of 8–11-year-old Danish children found that more variable sleep timing during a given week was associated with greater energy intake, independent of total sleep duration, screen time, and demographic confounders [30▪▪].

Sleep timing and obesity risk: complex associations across development

If sleep timing patterns contribute to obesity risk independent of sleep duration, identifying when such associations first emerge is important in determining when and how to intervene. Very young children meet their 24-h sleep need in part via daytime naps [2], for example, yet most studies examining daytime napping in relation to adiposity in young children have not found the associations (e.g., [22▪▪]). As children transition to formal school settings (e.g., kindergarten), their sleep patterns change; children as young as age 5 years may ‘shift’ their bedtimes late on weekends compared with weekdays [31]. Maintaining a regular and early bedtime schedule during the week, as well as across weekdays and weekends, may be an important obesity prevention strategy by reducing the behavioral and metabolic changes that occur as a result of shifted sleep timing [32,33▪]. This strategy may be particularly critical for school-age children and adolescents, who can face challenges to optimal sleep timing; yet, considering how such sleep timing patterns may relate to obesity risk in younger children is also important.

CHRONOTYPE: AN UNDERLYING DIURNAL PREFERENCE THAT MAY SHAPE TIMING OF SLEEPING, EATING, AND ACTIVITY PATTERNS RELEVANT FOR OBESITY

It has recently been suggested that ‘chronotype’, a construct reflecting individual differences in diurnal preference (e.g., being an early bird or a night owl), may independently contribute to obesity risk by influencing the time(s) of day when an individual is more likely to sleep, eat, or be physically active [34▪▪–36▪▪]. In addition, ‘social jetlag’ (i.e., weekday sleep restriction followed by a weekend delayed sleep phase) has been proposed as a direct cause of obesity through metabolic changes [32] and indirectly through behavioral pathways such as eating behavior [35▪▪,36▪▪,37]. A social jetlag pattern may arise from repeatedly shifting sleep timing, and is characterized by a misalignment of circadian-regulated sleep and appetite that can disrupt the underlying metabolism [32,33▪]. Studies of college students and adults have found that a preference for evening alertness and late sleeping was associated with unhealthy diet [35▪▪,38] and weight gain [35▪▪,39▪]. Furthermore, among adolescents (11–16 years) evening chronotype was also associated with higher BMI [34▪▪,36▪▪] and more unhealthy eating behaviors [34▪▪,37].No study has examined the emergence of these associations in younger children; however, recent work [40▪▪] determined that parent reports of chronotype were associated with a reliable marker of the timing of the circadian clock, salivary dim light melatonin onset. The early emergence of chronotype and associated sleep, eating, and activity timing patterns as they relate to childhood obesity risk is an exciting new research direction.

BIOLOGICAL AND BEHAVIORAL MECHANISMS PROPOSED TO UNDERLIE THE SLEEP–OBESITY ASSOCIATION

The mechanisms linking sleep and obesity are not fully understood, and a comprehensive update on all such relevant findings is beyond the scope of this review. Recent experimental work on the biological and behavioral mechanisms underlying sleep–obesity associations, including some studies with children, has nonetheless uncovered intriguing possibilities that could greatly inform new research and intervention efforts. We focus on the studies that consider how the timing of sleep and eating behaviors, in addition to sleep duration (or restriction) alone, may contribute to these pathways.

Biological mechanisms

For over a decade, short sleep duration has been suggested to lead to increased weight gain through hormonal changes associated with appetite regulation, specifically leptin and ghrelin secretion (e.g., [41]). Much less is known about how such processes operate in children, and recent work has found conflicting results. One large epidemiological study showed that chronically short sleep duration in school-age children was associated with lower leptin levels [42▪▪], whereas a smaller study that experimentally manipulated sleep duration found higher leptin levels associated with short sleep [43▪▪]. Methodological differences across the studies may account for different results, and neither study considered sleep timing or circadian cycle, which can regulate metabolism [44▪]. Thus, it may be that sleep timing and chronotype as well as sleep duration shape the physiological processes relevant for weight gain [45▪]. Investigating how sleep loss and sleep timing patterns may interact and independently contribute to the biological changes that could increase obesity risk, particularly in children, is an area ripe for research.

Behavioral mechanisms

Although appetite-regulating hormones and other biologically mediated pathways have been the focus of significant research, it has been recently noted that adults tend to increase their energy intake after sleep restriction even in the absence of hormonal changes [46▪,47▪▪], suggesting the importance of behaviorally mediated pathways. Although multiple behavioral mechanisms such as screen time and physical activity are clearly important, we focus here on the pathways related to eating behavior.

Dietary intake and quality

Experimental studies in adults have shown that increased energy consumption is a primary mechanism for weight gain following sleep restriction [48▪▪,49▪,50]. Experimental work with very young children has not been conducted. Yet, shorter nocturnal sleep at 16 months of age was associated with greater intake of energy 5 months later in a large birth cohort study [51▪▪]. Observational work with adolescents has also found associations between short sleep duration and poor dietary quality [52▪]. The few extant experimental studies with children are consistent with such results, finding that adolescents ate more dessert after five nights of sleep restriction [53▪▪], and that school-age children consumed more calories overall the day following sleep restriction [43▪▪]. Understanding how dietary intake may be

altered as a result of sleep restriction (or extension) in young children should be further investigated in future work using experimental designs [46▪].

Timing of eating

Behavioral mechanisms that promote obesity may be related to what time during the day children sleep, eat, and engage in sedentary versus active activities. Sleep timing and chronotype may thus contribute to obesity in part through shaping the timing of eating behavior [45▪,54▪,55▪▪,56]. Snacking outside of mealtimes has increased in recent years and is proposed as a contributor to excessive weight gain in children [57▪]. Young children may also wake at night and eat to soothe themselves back to sleep, and seek to delay bedtime by asking for food. Yet, little is known about how the timing of sleep and the timing of eating behavior relate to obesity risk in children; in one observational study, overweight school-age chldren ate more later in the day, whereas overweight adolescents ate less [58]. In experimental work with adults, participants who were sleep-restricted compared with well rested gained weight and consumed more calories specifically because of eating late at night [48▪▪,49▪,50], with foods eaten during the evening more likely to be high in carbohydrates [48▪▪,50] and fat, salt, and sugar [59▪]. Correlational studies have also found an association between adiposity indicators and the late-night eating of fats and carbohydrates [60▪].

Obesogenic eating behavior

Finally, dietary intake is the outcome of several increasingly well characterized obesity-promoting eating behaviors such as lack of satiety responsiveness, food responsiveness, enjoyment of food, emotional eating, eating in response to external versus internal cues, and restrained eating [61,62▪▪]. Recent work suggests that such eating behaviors may moderate or mediate the association between short sleep duration and obesity. In a small study of children (age 5–12 years), short sleep duration, poor sleep quality, and late bedtimes were associated with emotional eating, eating in response to external cues, and restrained eating respectively [63▪], suggesting that such eating behaviors may mediate sleep–obesity associations. Among adult women, eating behavior moderated the association, such that the effect of short sleep on food intake was greater for women who reported being emotional eaters [64]. Articulating the role of such eating behaviors is an important direction for future work.

CONCLUSION

Identifying how sleep patterns (i.e., timing and chronotype), above and beyond the sleep duration, may contribute to obesity risk across different developmental periods can help us understand the longitudinal and mechanistic associations and design effective interventions. Establishing a regular, consistent sleep schedule early in childhood may enhance metabolic regulation and prime healthier sleep and eating habits in the future. Sleep schedules shift increasingly with development and implications of sleep timing for obesity appear to begin at least during early childhood [20▪▪], a time when children shift from a biphasic to amonophasic sleep schedule [31]. Adolescents may be particularly vulnerable to the effects of poor sleep timing, perhaps as a function of their sex and pubertal status. Experimental work with adults has articulated specific biological and behavioral pathways connecting

sleep and obesity, many of which appear to operate through the timing of sleep and eating behavior, and mechanistic research on how such processes operate in children is critical.

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KEY POINTS

- Short sleep duration during early childhood is associated with concurrent and later obesity, but little is known about sleep duration–obesity associations in children under the age of 3 years, and associations in adolescents are inconsistent.
- Beyond sleep duration, sleep timing and chronotype may confer unique risk for obesity. Links between sleep timing, chronotype, and obesity risk in children and adolescents are understudied.
- Eating behavior – specifically the timing of eating, nature of dietary intake, and presence of obesogenic eating behavior styles – is a primary behavioral pathway that may confer obesity risk and may be influenced by sleep pattern (duration, timing, and chronotype).
- Longitudinal, experimental, and mechanistic work focused on young children and adolescents is needed to identify the early emergence and trajectories of sleep pattern–obesity risk associations in order to design developmentally appropriate obesity prevention strategies.