Weighing the Causal Evidence That Associates Short Sleep Duration With Obesity

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A substantial body of epidemiologic data has demonstrated associations between obesity and shorter sleep duration.1–6 These associations have been found in both adults and children and in diverse populations. These findings are particularly relevant because insufficient sleep is a widespread problem in the United States.7,8 Depending on the cutoff used to define inadequate sleep duration, anywhere from 30% to 90% of American children sleep fewer than the recommended number of hours per night.7,8 Adolescents meet recommended targets for sleep duration less frequently than younger children.7–9 The pressure of homework or extracurricular activities, the distraction of electronics, the mismatch between adolescents’ biological clocks and school schedules, and the noisy environments of many inner cities may be contributing factors for the relative sleeplessness of these children and adolescents.

To date, most pediatric studies investigating relationships between sleep and obesity have been strictly observational and therefore do not provide information to make inferences about causation. Decreased sleep duration of children aged 16 months to 17 years is associated with a risk ratio between 1.86 and 2.06 for obesity, as well as increased calorie and sugar intake in many10–13 but not all studies.14,15 Although a causal relationship between inadequate sleep and obesity is possible, numerous noncausal relationships might also be hypothesized. For example, less active children might require less sleep, or children who are allowed more screen time might tend to stay awake later. Lack of adequate sleep might even reflect lower levels of parental vigilance about all aspects of health (such as diet and exercise).

In the current issue of the journal, Rudnicka et al16 provide information that brings us a step closer to understanding the relationship between sleep, obesity, and the metabolic syndrome. These investigators conducted comprehensive metabolic evaluations of a large, diverse sample of 9- to 10-year-old children and correlated these with sleep duration. They document inverse associations between sleep duration and risk markers for type 2 diabetes and demonstrate that these associations persist after accounting for differences in adiposity. These findings also were unchanged after adjusting for differences in physical activity. These data are important because they confirm that the relationship between sleep, obesity, and the metabolic syndrome is unlikely to simply reflect lifestyle variables (such as activity level, screen time, or parental vigilance) and instead reflect more complex relationships that must be explored.

The Rudnicka study reveals a correlation in which 1 hour increased mean weekday sleep duration is associated with ∼0.2 lower BMI and 3% reduction in insulin resistance.
The recent Endocrine Society Clinical Practice Guideline on assessment, treatment, and prevention of pediatric obesity states that every 1 U reduction of BMI in kg/m² is associated with a decrease in systolic BP of 1.25 mm Hg and a decrease in serum triglyceride concentration of 1.55 mg/day, which is a proxy measure of insulin resistance.\(^1\) Although all of these associations indicate small effects, the development of obesity and type 2 diabetes is due to multiple factors. Each influence may exert a small effect, but multiple influences combined may lead to a more robust drive to these conditions.

The data from Rudnicka et al.\(^16\) reinforce those from epidemiologic studies of adults that similarly demonstrate associations between shorter sleep duration and insulin resistance, type 2 diabetes, and the metabolic syndrome.\(^1,8,19\) Given the substantial evidence linking inadequate sleep and aspects of the metabolic syndrome, it is necessary to consider how these factors might be related. A causal relationship remains to be confirmed, and it is possible that short sleep duration and risk factors for type 2 diabetes might reflect separate but related alterations in hypothalamic functions controlling sleep and those modulating neuroendocrine regulators of appetite and insulin sensitivity. To disprove this hypothesis and support a causal relationship, it is necessary to demonstrate that reductions in sleep duration increase risk factors for type 2 diabetes and that increasing sleep duration has the reverse effect. Of course, such an intervention would have to extend over a considerable time period and presents ethical challenges.

Data from studies in adults, and limited data in adolescents, suggest that experimental sleep deprivation may indeed cause insulin resistance and (in some studies) increased calorie intake and more rapid weight gain.\(^20\)–\(^25\) Adults subjected to severe sleep restriction (4 hours/night) have increased daily cortisol and catecholamine production,\(^21\)\(^,\)\(^22\) suggesting that elevated stress hormone levels may cause or contribute to insulin resistance in this setting. These data support the possibility of a causal relationship between shorter sleep duration and insulin resistance and/or type 2 diabetes risk; however, experimentally-imposed sleep deprivation may not equate with voluntary short sleep duration. Severe, experimental sleep deprivation induces a physiologic stress state, and similar physiologic alterations may or may not occur in individuals with habitual, apparently asymptomatic, short sleep duration. Interventions studies involving an increase in sleep duration are unfortunately few, and pediatric studies have typically involved interventions that combined sleep promotion with other lifestyle modifications, making it unclear which factors were most associated with the outcomes.\(^26\)–\(^28\) The study by Rudnicka et al.\(^16\) suggests that the influence of sleep duration on insulin resistance is relatively modest, particularly in relation to other, nonmodifiable factors such as ethnicity. Therefore, a future interventional study would need to be large, with sufficient statistical power to detect clinically-relevant changes in type 2 diabetes risk factors. The data from Rudnicka et al.\(^16\) provide ideal information on which to base a future clinical trial, which will be essential to resolve the question of whether relationships between sleep, obesity, and the metabolic syndrome are causal or associated via related but independent pathways.

**REFERENCES**


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