Development of a Behavioral Sleep Intervention as a Novel Approach for Pediatric Obesity in School-aged Children

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KEYWORDS

• Obesity • Pediatrics • Sleep • Behavioral intervention

KEY POINTS

- Use of multiple methodological approaches for determining the potential efficacy of a novel approach for pediatric obesity prevention and treatment can provide a strong foundation and rationale for refining approaches and current treatment targets.
- Systematic study of how sleep duration may affect eating and activity pathways suggests that sleep may be an important modifiable risk factor for obesity prevention and treatment.
- Several future directions are warranted, including further refinement of behavioral interventions, and further delineation of the mechanisms through which sleep may affect obesity risk.

INTRODUCTION

Despite being the focus of widespread public health efforts, childhood obesity remains an epidemic worldwide. The most recent US estimates show that 17.7% (95% confidence interval [CI], 14.5–21.4) of children 6 to 11 years old are obese (body mass index for age ≥95th Centers for Disease Control and Prevention [CDC]

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percentile), whereas a further 16.5% are overweight and at risk for becoming obese.[1] Given the now well-documented consequences of obesity for childhood health and psychosocial functioning, as well as associated morbidity in adulthood, identifying novel, modifiable behaviors that can be targeted to improve weight control is imperative.

The observation that while obesity levels were increasing, the duration of children's nighttime sleep was decreasing,[2] accompanied by compelling evidence for the potential role of sleep in both intake and expenditure aspects of energy balance,[3–7] suggests that nighttime sleep might be one such modifiable factor. Numerous cross-sectional and prospective observational studies have supported the association between sleep duration and obesity risk in children.[8–10] A recent meta-analysis found that, across 22 prospective observational studies of children aged 6 months to 18 years at baseline, from diverse backgrounds, children with a shorter sleep duration had twice the risk of overweight/obesity (odds ratio [OR], 2.15; 95% CI, 1.64–2.81) compared with their longer-sleeping peers.[10] The association was stronger among younger (OR, 1.88; 95% CI, 1.26–2.81) compared with older children (OR, 1.55; 95% CI, 1.22–1.97).[10]

Several pathways have been suggested that may link short sleep with obesity risk,[11] (**Fig. 1**). Experimental studies in healthy adults have provided evidence that sleep restriction or deprivation results in several neuroendocrine and inflammatory changes: impaired glucose metabolism, reduced insulin sensitivity, and increased levels of inflammatory mediators such as interleukin-6 and tumor necrosis factor.[12–14] Of particular interest are the changes that occur in hormones related to hunger and appetite; sleep restriction has been shown to reduce levels of leptin, a hunger inhibitor, and increase levels of the hunger hormone ghrelin.[6,7,15–17] Additional pathways proposed, and supported at least in part by adult experimental studies, include poorer food choices among those who are sleep deprived as well as reduced activity levels related to daytime tiredness.[3–7] Pediatric observational studies[18–23] are consistent with adult experimental studies, suggesting that similar pathways may be responsible for associations between short sleep and obesity risk in children as well. However, the pediatric literature remains limited by the observational nature of most of the existing studies.

To build on previous work, our group developed Project SLEEP, a series of studies designed to determine whether changes in children's sleep lead to changes in eating

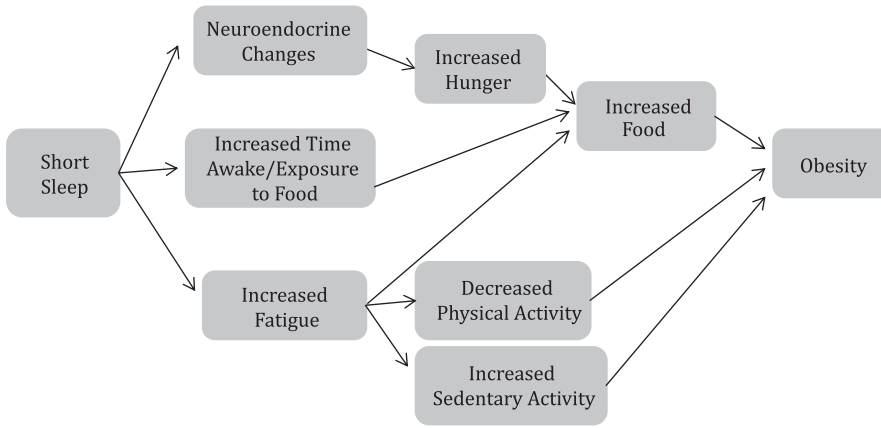

Fig. 1. Pathways through which sleep duration could affect obesity risk.