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Commentary

Interrelationships Between Sleep, Obesity, and Glycemic Control in Children

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Insufficient sleep is highly prevalent among children, with fewer than half of youths currently meeting recommendations for optimal nightly sleep duration [1]. In the adult literature, there is extensive evidence of adverse effects of insufficient sleep on cardiometabolic outcomes. Recent data from epidemiological studies indicate that links between insufficient sleep and obesity extend to children and adolescents [2]. Given the key role of adiposity in the pathogenesis of type 2 diabetes, there is a need for rigorous investigation of the association of free-living sleep duration and glycemic control among children.

The recent study by Alves and colleagues provides evidence linking short sleep duration with risk for obesity and diabetes in a preadolescent population [3]. In 62 children aged 7 to 11 years, longer sleep duration as assessed via 7-day wrist actigraphy, was associated with lower adiposity levels and better insulin sensitivity, as quantified using data from oral glucose tolerance testing. Interestingly, percent body fat was a significant mediator of these relationships, explaining > 60% of the association between sleep duration and insulin sensitivity measures.

Maybe the most interesting finding of the study by Alves et al [3] is that in these children, sleep duration was associated with measures of insulin sensitivity but not beta cell function. This finding would suggest that the key driver of metabolic dysfunction in response to sleep debt is a shift toward positive energy balance and consequent accumulation of body fat, rather than a direct effect on insulin secretion. Indeed, experimental curtailment of sleep in preadolescent children increases energy intake [4], the key driver of weight change. Sleep restriction in children also increases consumption of total sugars [5], which is associated with positive energy balance and can adversely affect glycemic control. The mediation analysis in the current study by Alves et al [3] appears to confirm that adiposity lies in the pathway linking short sleep with insulin resistance measures in a free-living context.

The findings of this study are notable for several reasons. First, this study applies objective measures of free-living sleep over 7 days and glycemic outcomes were assessed with an oral glucose tolerance test, allowing for assessments of both insulin secretion and sensitivity. The comprehensive evaluation of glycemic control parameters and measures of adiposity with bioelectrical impedance analyses allows for important insights into the mechanisms underlying the increasingly robust association of short sleep with type 2 diabetes risk.

By providing insight into mechanisms underlying the relation of short sleep with glycemic control in children, this study can help to inform strategies to prevent future development of cardiometabolic diseases among children. Increasing children's total sleep time must be a priority. Recent studies have demonstrated that interventions to extend sleep duration in children and adolescents are feasible and effective [6], and, in adults, sleep extension reduces caloric intake [7]. While it remains to be determined whether extending sleep among children can improve dietary behaviors and reduce adiposity, the findings from Alves et al [3] would suggest that, if such behavioral modifications are achieved, children's risk for the development of diabetes could be mitigated.

While this investigation by Alves and colleagues [3] provides important information on the role of sleep duration in glycemic control among children, much work remains to be done. Most notably, replication of these findings in larger cohorts and incorporation of longitudinal data are needed. Furthermore, future research investigating associations of sleep with metabolic outcomes in children should strive to incorporate best practices for free-living assessments of sleep and physical activity. For example, Alves and colleagues used the Cole-Kripke algorithm to score sleep data with actigraphy, while the Sadeh algorithm is recommended for youths [8]. In addition, best practice for assessment of rest and activity with actigraphy is to wear the device on the nondominant wrist to reduce overestimation of activity. Studies aiming to assess both day and nighttime behaviors should also strive to achieve at least 22 hours of actigraph wear time in participants. Pairing sleep and glycemic control measures with reliable dietary assessments and precise measures of body composition, such as magnetic resonance imaging or dual-energy x-ray absorptiometry, should be included in future

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studies. Incorporating these measures across child-based cohorts would be key to better understanding of both the long-term health consequences of perturbations in sleep as well as the mechanisms underlying these associations. Ultimately, this information would allow for the development of strong, sustainable strategies to improve lifestyle behaviors and create favorable sleep health habits early on in life. This work certainly represents an important first step toward these goals.

In conclusion, there is growing evidence for a role of sleep in determining children's metabolic health. Sleep is a modifiable risk factor, and it is therefore imperative to investigate further the associations between sleep, obesity, and diabetes risk in children, with the goal of developing preventive strategies. Childhood and preadolescence might provide a unique window of opportunity for intervention, setting up healthy sleep hygiene and diet habits, before adolescence, when individuals might be more vulnerable to rash decision-making, emotional fragility, and risky behavior.

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Disclosures

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