



A Model of Adolescent Sleep Health and Risk for Type 2 Diabetes

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Abstract

Purpose of Review This paper presents a review of the current literature in support of a model explaining the relationships between sleep health and risk for type 2 diabetes in adolescents.

Recent Findings Short sleep duration is associated with risk of developing obesity in youth. Sleep restriction increases energy expenditure, but also increases hunger, appetite, and food intake, causing positive energy balance, impacting appetite-regulating hormones, and leading to increased eating late at night. Insufficient sleep may lead to reduced physical activity and greater sedentary behaviors. In addition, short sleep duration is associated with reduced insulin sensitivity. The cumulative negative consequences of insufficient sleep increase risk for type 2 diabetes. Applications to clinical care, public policy, and future research are discussed.

Summary Insufficient sleep in adolescence increases risk for type 2 diabetes directly through impact on insulin sensitivity and indirectly through increased dietary intake, sedentary activity, and weight gain.

Keywords Insufficient sleep · Pediatrics · Glucose metabolism · Insulin resistance

Introduction

An overwhelming majority (78%) of high school-age adolescents report sleeping less than the recommended 8–10 h per night [1, 2]. Simultaneously, rates of type 2 diabetes (T2D) are

increasing among youth [3]. One contributor to insufficient sleep is the physiological shift in circadian rhythm that occurs mid-adolescence, causing later sleep onset [4]. When coupled with academic, extra-curricular, and social demands, light exposure from evening electronics use, and imposed early school start times, it is difficult for adolescents to obtain sufficient sleep [4]. Because insufficient sleep is linked to insulin resistance (IR) [5], the high incidence of insufficient sleep in adolescents may be a factor underlying the increasing rates of T2D in youth [3].

IR peaks during puberty, is frequently greater in adolescents with obesity than those of normal weight, and contributes significantly to T2D development and cardiometabolic risk [6, 7]. T2D presents aggressively in adolescents, with rapid onset of β cell failure compared with adults [3, 8]. Given that obesity currently impacts approximately 1/3 of adolescents in the USA [9], alternative T2D prevention and treatment targets for youth are urgently needed to prevent future morbidity and mortality.

Adults that habitually maintain insufficient sleep schedules demonstrate IR, and findings from laboratory-based studies show that experimentally induced insufficient sleep in adults induces IR [10–15]. For example, 5 days of insufficient sleep led to a 20% decrease in insulin sensitivity in healthy adults in one study [16], and emerging evidence suggests a similar relationship between insufficient sleep and IR in adolescents

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[5]. Yet, multiple unique developmental and physiological changes occur during adolescence, limiting the generalizability of adult studies to adolescents. Thus, we developed a model to explain the relationship between sleep health and risk for T2D in adolescents based on the current literature.

Model of Sleep and Risk for Diabetes in Adolescents

Our model (Fig. 1) posits that the pubertally related circadian delay that leads to a propensity for delayed bedtimes, combined with forced early morning school day rise times, results in insufficient sleep. Insufficient sleep may then lead to decreased physical activity, increased evening food intake, and increased weight gain and obesity risk. Thus, in addition to pubertally induced IR, insufficient sleep both directly and indirectly, through its impact on activity, diet, and weight, results in IR, which in turn, increases future risk for developing T2D. Here, we discuss the evidence base to date in support of this model.

Definition of Sleep Health in Adolescents

Sleep health is a multidimensional construct, including sleep duration, continuity or efficiency, timing, sleepiness, and subjective satisfaction or quality [17]. For the purpose of the current manuscript, we will focus on sleep duration, while acknowledging the impact and importance of future research in these and other domains, including circadian misalignment, and obstructive sleep apnea, insomnia, and other sleep disorders.

Insufficient Sleep in Adolescents

Chronic insufficient sleep is endemic in today's adolescents, with typical sleep duration well below recommendations for age [1, 2]. A pubertal shift in circadian rhythms results in a delayed bedtime, which is compounded by increased academic, extracurricular, and social demands [4]. These factors drive late sleep onset, which, when combined with imposed early school start times, result in high rates of insufficient sleep in

adolescents [4]. Data from the 2019 Youth Risk Behavior Survey reveal that 83% of 12th grade students report obtaining < 8 h sleep per night, and as many as 43% of teens obtain < 6 h sleep per night [1].

Adolescence is a developmental stage during which youth have more autonomy over their bedtimes, which has been shown to result in later bedtimes and shorter sleep duration [18]. In addition, high rates of screen-based electronics use in adolescents increase evening light exposure, which may further delay circadian rhythms and have been shown to promote later bedtimes and insufficient sleep [18, 19]. Over 70% of adolescents report using at least one electronic device at least 1 h before sleep, and 30% of adolescents use mobile devices throughout the night [19].

Negative consequences of insufficient sleep in adolescents are numerous and include attention difficulties [20], mood and behavior problems, and poor academic performance [21]. As a result, the American Academy of Pediatrics recommends that high schools start no earlier than 8:30 am to provide adolescents with the opportunity to obtain sufficient sleep aligned with their biological clock [22]. Yet, the majority of middle and high schools start much earlier (average start time is 8:03 am), indicating that most youth are at risk for short sleep duration and highlighting that examination of the impact of short sleep on T2D risk is vital.

Sleep Health and Association with Obesity

The link between sleep and risk of obesity is well established. Correlational data from a meta-analysis of an international sample of 2–20-year-olds suggest that for each hour less sleep obtained, there is a corresponding 80% increase in obesity risk to youth [23]. In adolescents, ages 10–16 years, each additional hour of sleep obtained was associated with a decrease in BMI percentile by 3.6 points as well as lower body fat [24]. Longitudinal research affirms these findings: one longitudinal study found that adolescent self-reported sleep problems (e.g., difficulty sleeping, daytime sleepiness) were associated with an increased risk of developing overweight/obesity in young adulthood [25]. Additionally, age and sex may be important factors in this association: in a longitudinal assessment of adolescents biannually at six timepoints, shorter sleep at baseline was found to be associated with an increase in BMI over time for older adolescent females (16–18 years), whereas baseline sleep was associated only with baseline BMI, not weight gain over time, for males [26]. Finally, findings from a meta-analysis of prospective studies revealed a causal relationship between short sleep duration and subsequent weight gain and obesity in youth across development, including adolescence [27]. Specifically, short sleep duration was associated with a two-fold increased risk of developing obesity in youth [27].

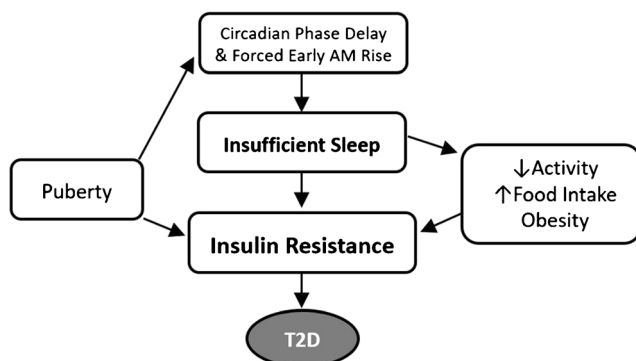


Fig. 1 A model of sleep and risk for diabetes in adolescents

Sleep Health and Association with Dietary Intake

Findings from laboratory studies in adults show that sleep restriction (4–5 h per night) increases total energy expenditure by ~5% but also increases hunger, appetite, and food intake, causing positive energy balance [28•, 29]. It is hypothesized that the function of this additional food intake is to compensate for the additional energy expenditure of increased wakefulness, but intake is excessive when food is readily available [29]. This increased energy intake following insufficient sleep may have implications for obesity and T2D risk [30, 31].

Similar results have been found in studies of adolescents. Adolescents ages 14–16 years consumed 10% more calories and 110% more servings of sweet/dessert foods during a week of experimental insufficient sleep (6.5 h time in bed) compared with longer sleep (10 h time in bed) [30, 31]. Additionally, teens rated images of sweets/desserts as more appealing after insufficient sleep than longer sleep, while no difference was found in ratings of hunger or non-sweet foods [31]. Similarly, adolescents performed more poorly on a food-related inhibitory control task and had higher food reward following a week of insufficient sleep (5 h time in bed) compared with a week of habitual sleep (9 h time in bed) [32]. These findings suggest that insufficient sleep may impact dietary choices by increasing the reward value of foods, particularly foods that are carbohydrate dense.

Conflicting evidence exists as to whether insufficient sleep may influence levels of appetite-regulating hormones leptin and ghrelin [33]. In adults, increased evening ghrelin was associated with greater energy intake following sleep restriction in laboratory studies [34]. Similarly, a meta-analysis of 21 adult studies found that insufficient sleep duration was associated with increased ghrelin levels [35]. In a study of children 8–11 years of age, however, youth reported lower energy intake and had lower fasting morning leptin levels but no difference in ghrelin was observed following 1 week of increased sleep duration (1.5 h more time in bed compared with typical sleep) compared with a week of decreased sleep (1.5 h less time in bed) [36]. In an experimental study, adolescent boys had no difference in leptin and ghrelin concentrations and actually consumed fewer calories during an ad libitum meal opportunity controlling for dietary intake on previous days following 3 nights of insufficient sleep (4 h time in bed) compared with 3 nights of longer sleep (9 h time in bed) [37]. Thus, data regarding the role of insufficient sleep on appetite-regulating hormones in adolescents are equivocal and research specifically designed to address impacts of insufficient sleep on appetitive hormones rather than as an adjunct to studies with other primary outcomes and using high-quality methods are needed.

Timing of eating in relation to sleep may be another important factor. Adults with later timing of sleep (later bed and wake time) consumed more protein, fat, and carbohydrates

in the evening (after 8 pm), and dietary intake after 8 pm was associated with higher BMI [38]. Evidence suggests that dietary intake during times of high circulating melatonin levels (late at night, or early in the morning) may contribute to weight and metabolic dysregulation [39, 40]. The consequences of late eating timing have potential negative implications for adolescents, given the circadian phase delay that occurs in this developmental stage, and adolescents, particularly those with late chronotype (diurnal preference), demonstrate most eating occasions clustered late in the day [41, 42]. Indeed, later chronotype was associated with greater evening caloric intake in one sample of adolescents [42]. Moreover, later bedtime was associated with insufficient sleep duration and both decreased intake of fruits and vegetables and increased intake of foods such as pizza, soda, and sweets/desserts in youth 12–18 years of age [43]. However, specific research in adolescents is needed on the timing of eating in relation to sleep and glucose metabolism or obesity risk.

Sleep Health and Association with Physical Activity

It has been hypothesized that insufficient sleep duration may reduce adolescent's participation in physical activity, in part due to increased daytime sleepiness and fewer activity opportunities in the evening hours. However, the existing literature is limited both in number of studies and reliance on self-report measures, and findings are mixed. Contrary to this hypothesis, in one study of Saudi adolescents, longer self-reported sleep duration was associated with high screen time and low-to-medium physical activity levels, whereas insufficient sleep was associated with high levels of physical activity [44]. In contrast, several other studies have shown that shorter sleep duration and poor sleep quality were associated with less physical activity, more electronics use, and higher sedentary behaviors in adolescents using both subjective and objective assessments [45–49].

In an experimental study with objectively measured sleep and activity, adolescents increased time spent in sedentary behaviors during 2 weeks of habitual short sleep duration (5–7 h per night) when compared with 2 weeks of sleep extension (1.5 h increased time in bed), with no differences in light or moderate-to-vigorous physical activity [50]. Thus, the authors posited that increasing sleep in short-sleeping adolescents reduces sedentary time without impacting physical activity behaviors, implying that extra wakefulness during insufficient sleep is sedentary time [50]. Similarly, an association between short sleep and sedentary behaviors was consistently observed in a systematic review, but the impact of sleep on physical activity and electronics use was not able to be determined, primarily due to few studies and poor quality of the existing research [33].

The combined health benefit of meeting both sleep and physical activity recommendations has been reported in the

literature. Accelerometer data from adolescents ages 10–16 years revealed that, compared with those with insufficient sleep and low physical activity, adolescents that met guidelines for adequate sleep duration and physical activity had lower cardiometabolic risk factors and adiposity [51]. A review of studies in which physical activity was objectively measured in youth (ages 5–17 years) found that those with a combination of high physical activity, longer sleep duration, and low sedentary behaviors had lower adiposity and better measures of cardiometabolic health (e.g., waist circumference, HOMA-IR, triglycerides) compared with those with low physical activity, short sleep, and high sedentary time [52]. This suggests that evaluating healthy lifestyle behaviors in combination rather than in isolation may be important.

Sleep Health and Association with Insulin Resistance

Evidence is mounting that insufficient sleep duration is associated with IR in youth. Self-reported insufficient sleep duration is associated with IR assessed with fasting measures and measures from a hyperglycemic clamp in youth ages 10–19 years [53, 54]. Further, actigraphy-estimated sleep was found to be significantly associated with insulin sensitivity measured with fasting glucose and insulin in adolescents ages 13–19 years, but this relationship was attenuated when weight status was included in the model [55]. This was examined further in another study of youth ages 2–15 years in which it was found that abdominal adiposity mediated the relationship between insulin sensitivity (HOMA-IR) and self-reported sleep duration [56]. Finally, actigraphy-assessed longer sleep duration and higher sleep efficiency were associated with lower metabolic risk scores, composed of waist circumference, systolic blood pressure, high density lipoprotein cholesterol (HDL-C), triglycerides, and HOMA-IR [57]. However, each of these studies was limited by either using only fasting glucose and insulin rather than more sensitive assessments of IR or subjective assessment of sleep.

The sleep and IR relationship has been examined in few studies of adolescents using both objectively measured sleep and more sensitive assessment of IR. In a sample of adolescents ages 14–19 years with overweight/obesity during the academic year, shorter actigraphy-estimated sleep duration on weekends and weekdays, as well as later weekday bedtimes, was significantly associated with IR assessed with an oral glucose tolerance test [5]. In a randomized cross-over in-laboratory study design, adolescent boys demonstrated lower HOMA-IR after three nights of insufficient sleep opportunity (4 h time in bed) compared with three nights of longer sleep opportunity (9 h time in bed) [58]. In sum, the extant literature provides evidence that short sleep is associated with IR in adolescents but additional research using both objective measurement of sleep and more rigorous stimulated measures of insulin sensitivity and secretion are needed.

Sleep Health and Risk for Type 2 Diabetes

The negative metabolic consequences of insufficient sleep increase risk for T2D. In adults, a U-shaped relationship has consistently been found with both short and long sleep associated with risk for developing T2D [59, 60]. An analysis of the pooled related risks of multiple sleep variables found that the diabetes risk conferred by insufficient sleep, as well as other sleep factors such as obstructive sleep apnea, poor sleep quality, and shift work, was comparable with traditional risk factors such as weight, family history, and sedentary activity [61]. Less research has been conducted in adolescents, but a narrative review of the pediatric literature found that there was “compelling evidence” for a link between both objectively and subjectively assessed sleep duration with biomarkers of T2D in youth (e.g., adiposity, glucose homeostasis), even after accounting for weight, pubertal status, age, and sex (though physical activity and dietary intake were not controlled for) [62].

Clinical Applications

Health care providers working with adolescents, particularly those with obesity or other risk factors for T2D, should consider including assessment and treatment of sleep health in their routine care. Specifically, querying about bed and wake times on both weekdays and weekends and encouraging adolescents to schedule adequate time in bed to allow for enough time to obtain the recommended 8–10 h of sleep per night are important [2]. Additionally, delayed school start times have been shown to result in increased sleep duration and less variability between weekday and weekend sleep in high school students [63, 64]. Thus, policy changes to delay school start times may be beneficial for the metabolic health of adolescents [22].

Future Research

Extending sleep duration may reverse the negative metabolic consequences of insufficient sleep, though findings are mixed. In habitually short-sleeping young-adult men, three nights of 10 h in-lab time in bed resulted in improved insulin sensitivity [65] and two nights of in-lab “recovery sleep” (10 h per night) reversed the effect of insufficient sleep on IR [66]. Conversely, 2–5 nights of recovery sleep (9–10 h time in bed or ad libitum sleep opportunity) were not sufficient to improve insulin sensitivity following insufficient sleep in other studies with adults [16, 67, 68]. It may be that improved sleep must be sustained in order to benefit metabolic health: adult participants demonstrated improved insulin sensitivity following 6 weeks of in-home sleep extension [69]. Sleep extension with youth has been shown to be feasible by changing sleep onset times [70, 71], and increased sleep was found

to improve attention, memory, problem-solving, emotional regulation, and depression [71–73]. Thus, research investigating the impact of experimental sleep extension and sleep health intervention specifically on IR in adolescents is warranted. Given that individual school start times are not usually individually modifiable, focusing on sleep onset times may be most feasible.

Beyond sleep duration, variability in sleep timing, or social jetlag, defined as the difference between weekday and weekend sleep times, is another variable worthy of investigation. A social jet lag > 1 h was reported by 87% of adolescents in one study, with nearly 60% of youth endorsing a > 2-h difference between weekday and weekend sleep times [74]. While studies in adults consistently report that greater social jetlag is associated with metabolic risk factors and diabetes/pre-diabetes diagnosis [75, 76], findings are mixed on the impact of social jetlag and diabetes risk factors in adolescents. One study found an increase in adiposity corresponding to each additional hour of social jetlag in adolescent girls, but not boys [77]. Another study found that social jetlag was associated with lower odds of healthy dietary behaviors (e.g., breakfast consumption, fruit and vegetable intake, and fast food consumption, and higher BMI in a study of over 3000 adolescents) [78]. Conversely, social jetlag was associated with healthier lifestyle behaviors (physical activity and dietary intake) and lower BMI in two other studies of youth [74, 79]. Given the high prevalence, additional research clarifying the impact of social jetlag on metabolic risk factors in adolescents is needed.

Circadian misalignment is another factor that must be examined in future research. Circadian rhythms are near-24-h patterns of behavior and physiology regulated by the suprachiasmatic nucleus of the hypothalamus and synchronized by light/dark changes [80]. Evidence suggests that circadian misalignment is associated with IR in adults [16, 81]. Compared with adults and younger children, however, adolescents demonstrate a puberty-related delay in melatonin secretion and the forced early rise times common for high school students results in adolescents being awake at inappropriate circadian times [82]. Indeed, morning circadian misalignment, or wakefulness while melatonin levels remained elevated, was associated with IR (HOMA-IR) in adolescent girls with obesity [83]. Further research examining the impact of circadian misalignment on IR is needed in other adolescent populations particularly given the increased risk for delayed circadian rhythm in this age group.

Evidence suggests that racial and ethnic background and sex may also be important to consider [84]. In adults, racial disparities in cardiometabolic risk were explained in part by the shorter sleep duration and poorer sleep efficiency obtained by Black compared with White individuals [85]. Racial disparities in sleep have been documented with Black individuals demonstrating shorter sleep duration compared with White

individuals [86, 87]. The incidence rate of T2D is increasing at a faster rate for non-White youth, and racial differences in beta-cell responsiveness have been found in adolescents [3, 88]. To our knowledge, racial and ethnic differences have not been examined in the adolescent sleep and T2D literature but are important factors to consider for future study.

Sex differences are also an area in need of further exploration. Adolescent girls are at increased risk for insufficient sleep duration: compared with boys, adolescent girls report more daytime sleepiness, and a later chronotype [89, 90]. Despite similar bedtimes, adolescent girls wake significantly earlier than boys on weekdays and shift their bed and wake times significantly later than boys on weekends [90, 91]. Additionally, limited studies suggest sex differences with regards to glucose metabolism and exposure and influence of obesogenic factors [92, 93]. However, findings regarding sex differences in sleep and T2D risk are unclear. In some studies, a relationship between sleep duration and BMI has been found for male but not female adolescents [94], while in others, insufficient sleep was associated with increased obesity risk for females but not males [95, 96]. Thus, further research to better understand these relationships is needed.

Finally, research on the unintended consequences of the COVID-19 pandemic on adolescent sleep in relation to T2D risk is important. Recent findings show that adolescents report longer sleep duration since the start of the pandemic and the shift to online learning [97], but it is unclear whether this equates to changes in risk for T2D. Additional data on sleep timing, physical activity, sedentary behaviors, and dietary habits during the COVID-19 time period are also needed.

Conclusions

Our model proposes that insufficient sleep in adolescence increases risk for T2D both directly through its impact on IR and indirectly through increased dietary intake, sedentary behavior, and weight gain. This is alarming given that a majority of adolescents are not achieving the recommended sleep duration, particularly on school nights [98–101]. Moreover, adolescents are already at increased risk for abnormal glucose metabolism as puberty is strongly associated with IR [6]. Health behaviors established during adolescence often persist into adulthood, making adolescence a critical window for intervention [102]. Adolescents self-direct their food choices more than younger children [103] and poor eating habits are prevalent in teens [104]. Rates of screen-time surge, while physical activity levels plummet during this high-risk developmental stage [18, 19]. Thus, prioritizing sleep health promotion clinically and through public health changes such as delaying high school start times is urgently needed. Moreover, additional research in adolescents examining the impact of

improving sleep health on insulin sensitivity and T2D risk factors is imperative.

Authors' Contributions SLS had the idea for the article, performed the literature search, and drafted the manuscript; JH, EM, KPW, and KJN helped develop the model and critically revised the work.

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Compliance with Ethical Standards

Conflict of Interest Kenneth P. Wright Jr. reports during the conduct of the study being a consultant to/and/or receiving personal fees from Circadian Therapeutics, Inc., Circadian Biotherapies, Inc., Philips, Inc.; and receiving research support from the NIH, the Office of Naval Research, the PAC-12 Conference, and Somalogic, Inc., outside the submitted work. EM is supported by resources from the Geriatric Research, Education, and the Clinical Center at the Denver VA Medical Center. The contents do not represent the views of the U.S. Department of Veterans Affairs or the United States Government.

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Human and Animal Rights All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its amendments, institutional/national research committee standards, and international/national/institutional guidelines).

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