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CLINICAL REVIEW

Sleep and cardiometabolic risk in children and adolescents

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SUMMARY

The evidence for a link between sleep and cardiometabolic risk factors in children and adolescents is accumulating; however, the literature has not yet been reviewed. Seventy-five studies investigating associations between sleep variables and measures of abdominal adiposity, glucose homeostasis, blood lipids, blood pressure (BP), and inflammatory markers were included in the present review. The current evidence indicates that inadequate sleep may play a role in cardiometabolic risk at a later age for children and adolescents. Most compelling is the evidence for an association between inadequate sleep and abdominal adiposity, decreased insulin sensitivity as well as high BP, whereas the evidence for potential links between sleep and blood lipids as well as inflammatory markers is less convincing. It should, however, be noted that the majority of studies linking sleep with cardiometabolic outcomes are cross-sectional in nature, and sleep is often assessed using parent or self-report. We suggest that future studies should investigate longitudinal associations between sleep and cardiometabolic risk factors with the use of objective sleep measurements conducted for several days, including weekdays and weekend days, at multiple time points over time. Meanwhile, based on the available evidence, we recommend that children and adolescents get adequate amounts of good sleep in a regular pattern.

Introduction

The prevalence of childhood obesity has increased worldwide over the past decades [1], which is of serious concern, since being overweight or obese in childhood carries into adulthood and increases the risk of related comorbidities, including type 2 diabetes (T2D) and cardiovascular diseases [2,3]. Although changes in sleep durations have not been uniform across regions, some data suggest that average sleep durations in children and adolescents have declined over the last century [4]. Among the reasons for reduced, disturbed, or variable sleep patterns in youth is the increased use and availability of electronic entertainment and communication devices, especially during the late evening, which is reported to delay bedtimes and to be associated with shortened and disturbed sleep in children and adolescents [5]. Moreover, children's sleep-wake pattern may be influenced by their parents as observed from associations between parental socioeconomic status and childhood sleep duration [6].

Adequate sleep is particularly important during childhood and adolescence since inadequate sleep is associated with atypical physical and cognitive development [7]. Although the evidence for what can be defined as an optimal sleep duration in children and adolescents has been questioned [8], a growing body of evidence suggests that inadequate sleep could be a contributing factor for weight gain and the increased prevalence of overweight and obesity among children and adolescents [9,10]. Physical activity (PA) and diet have traditionally been considered the "Big Two" factors affecting energy balance homeostasis. However, recent research suggests that sleep may also be an important modifiable lifestyle component that can affect eating and activity behaviors, and ultimately energy balance and body weight regulation [11], as well as other cardiometabolic risk factors in youth.

Although experimental studies in children and adolescents are scarce, several studies have examined the association between sleep and cardiometabolic risk factors in the pediatric population; however, this literature has not yet been reviewed. Given that cardiometabolic risk can track from childhood to adulthood, a better understanding of the possible adverse effects of inadequate

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Abbreviations

BMI	body mass index
BP	blood pressure
CI	confidence interval
CRP	C-reactive protein
DBP	diastolic blood pressure
HDL	high density lipoprotein
HOMA-IR	homeostatic model assessment of insulin resistance
HPA	hypothalamic-pituitary-adrenal
HT	hypertension
IR	insulin resistance
LDL	low density lipoprotein
MAP	mean arterial blood pressure
MetS	metabolic syndrome
OSA	obstructive sleep apnea
PA	physical activity
PSG	polysomnography
REM	rapid eye movement
SBP	systolic blood pressure
SDB	sleep disordered breathing
SNS	sympathetic nervous system
SWS	slow wave sleep
T2D	type 2 diabetes
TG	triglycerides
WC	waist circumference

sleep at an early stage in life is important and can help implement interventions aimed at improving sleeping habits in children.

Therefore, the aim of the present review is to give an overview of the literature investigating associations between sleep and cardiometabolic risk factors in children and adolescents. In the present paper, measures of sleep include sleep duration, sleep quality as well as sleep architecture and assessment of cardiometabolic risk is based on studies focusing on abdominal adiposity, glucose homeostasis, blood lipids, blood pressure (BP), and inflammation.

Methods

We performed literature searches for publications using PubMed until August 10, 2015. Search strategies included the following key words: “children”, “adolescents”, “sleep”, “cardiometabolic”, “metabolic health”, “abdominal obesity”, “glucose”, “insulin”, “lipids”, “dyslipidemia”, “triglycerides”, “cholesterol”, “blood pressure”, “hypertension”, and “inflammation”.

Only publications in English were considered. Reference lists of the relevant publications were cross-checked for additional publications. Studies conducted in children and adolescents with obstructive sleep apnea (OSA)/sleep disordered breathing (SDB) were not included, except for one study in which OSA was present in a subgroup of the study population [12]. As most children below the age of three take daytime naps, and some metabolic variables such as waist circumference (WC) in that age group are poorly translated into later health, this literature review includes children and adolescents aged 3–20 y, except for two studies that investigated longitudinal associations between sleep curtailment from infancy to mid-childhood adiposity [13] and cardiometabolic risk [14]. Measures of sleep quality and quantity included parent/self-report, actigraphy/accelerometry and polysomnography (PSG). Given the pronounced heterogeneity among the available studies we put more emphasis on the relative amount of sleep within each study and less on the absolute sleep duration. To evaluate the studies that categorize sleep duration, we present the cut-points and proportion of participants in each category. The majority of studies assessed sleep by subjective methods and we, therefore, only specified the method used within each study when objective sleep measures of sleep duration have been used. Furthermore, sleep duration decline from infancy throughout adolescence, hence the wide age range within many studies may affect the associations between sleep and the outcome variables [15]. We therefore discussed whether associations are dependent on age and pubertal status (e.g., Tanner stage) when available and appropriate.

After screening, 75 studies were included in the present article out of which 38 studies (n = 71,806) included measures of abdominal adiposity, 21 studies (n = 15,561) included measures of glucose homeostasis, 14 studies (n = 30,098) included measures of blood lipids, 30 studies (n = 62,146) included measures of BP, and nine studies (n = 8,266) included measures of inflammatory markers. Cross-sectional studies reporting beta coefficients and 95% confidence intervals (CI) or standard errors were included in Figs. 1–4. Studies reporting logarithmic transformed homeostatic model assessment of insulin resistance (HOMA-IR) were selected for presentation in Fig. 2. Information about logarithmic transformations was obtained by correspondance with the authors of the specific articles. Figures on associations between sleep duration and measures of blood lipids and inflammatory markers were not included due to a limited number of studies reporting sleep duration as a continuous variable. Continuous associations were selected for graphic presentation since the majority of studies adjusted for important covariates and due to the heterogeneity in the definition of sleep categories among the different studies. Calculations of overall estimate and 95% CI were performed by weighted least squares regression with standard error of the mean in the weighting equation in STATA/IC 11.2 (Houston, TX, USA) and forest plots illustrating beta coefficients, overall estimate, 95% CI, and weights were created in SigmaPlot 13.0 (Systat Software Inc., San Jose, CA, USA).

Sleep and abdominal adiposity in children and adolescents

The association between short sleep duration and overweight/obesity in youth is well supported by the literature [9,10]. Although childhood obesity is associated with comorbidities such as T2D and hypertension (HT) [2], evidence in adults indicates that abdominal adiposity is the strongest determinant of the metabolic syndrome (MetS) [16]. Therefore, WC is now included in the International Diabetes Federation's criteria for MetS in children and adolescents [17]. The main results and methodologies used in the 38 observational studies investigating the associations between sleep and abdominal adiposity in children and adolescents are presented in Table 1.

Longitudinal age-independent associations between shorter sleep duration and abdominal adiposity have been identified in four studies [13,14,18,19], whereas change in accelerometer-determined sleep duration was not associated with changes in WC during 200 d in children 8–11 y old in our recent study [20]. However, changes in sleep duration and cardiometabolic risk profile over the 200-d follow-up period were negatively associated [20], which is in line with a cross-sectional study by Iglayreger et al. [21] in which sleep duration inversely predicted cardiometabolic risk in a small sample of obese adolescents. In line with our observation, reduction in time in bed was not associated with changes in WC over a 1 y period in 6th grade adolescents [22].