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Sleep Characteristics and Cardiovascular Risk in Children and Adolescents: An Enumerative Review

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Abstract

Cardiovascular risk factors develop in childhood and adolescence. This enumerative review addresses whether sleep characteristics, including sleep duration, continuity, quality, and daytime sleepiness, are associated with cardiovascular risk factors in young people. Thirty-nine studies were identified that examined the following risk factors: metabolic syndrome, glucose and insulin, lipids, blood pressure, and cardiovascular responses to psychological stressors. Due to the availability of other reviews, 16 longitudinal studies of obesity published in 2011 and later were also included in this report. Excluded from the review were studies of participants with suspected or diagnosed sleep disorders and reports from sleep deprivation experiments. Combining studies, evidence was strongest for obesity, followed by glucose, insulin, blood pressure (especially ambulatory blood pressure) and parasympathetic responses to psychological stressors. There was little evidence for metabolic syndrome cluster, lipids, and blood pressure responses to psychological stressors. The more positive associations were obtained for studies that incorporated objective measures of sleep and included adolescents. The foundational evidence is almost entirely cross-sectional, except for work on obesity. In summary, available evidence suggests that the associations between sleep characteristics and cardiovascular risk vary by risk factor. It is time to conduct studies to determine antecedent and consequent relationships and to expand risk factors to include markers of inflammation.

Keywords

sleep; children; cardiovascular risk factors; lipids; blood pressure; obesity; glucose; insulin

1. Introduction

Elevated risk factors for cardiovascular diseases are apparent in children and adolescents and relate to subclinical cardiovascular disease (CVD) later in life. For example, 6% of female adolescents and 20% of male adolescents had high fasting blood glucose (≥ 100

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mg/dl) in the National Health Administration Examination Survey (NHANES) study.[1] Up to 9.8% of children and adolescents had systolic hypertension, and up to 7.1% had diastolic hypertension in an analysis of 58,698 children and adolescents enrolled in 11 studies.[2] Blood pressure (BP) at age 13 predicted adulthood BP at age 24, in addition to elevated lipids and glucose.[3] Autopsy studies of young adults who died from traumas reported a linear relationship between number of cardiovascular risk factors and intima surface covered with fatty streaks in the coronary arteries: 0, 1, 2, and 3/4 risk factors had, respectively, 1.3%, 2.5%, 7.9%, and 11.0%; the extent of fibrous-plaque lesions in the coronary arteries was 12 times as great in persons with 3 or 4 risk factors, compared to those with none.[4] The greater the number of risk factors (cigarette smoking, elevated lipids, BP, and body mass index (BMI)) in adolescence the greater carotid intima medial thickness in both men and women in adulthood.[5] A combination of risk factors among children was associated with reduced carotid artery elasticity and increased stiffness.[6;7] The metabolic syndrome, a combination of elevated BP, triglycerides, waist circumference, glucose, and low high density lipoprotein levels (HDL-C), in childhood and adolescence predicted CVD in adulthood.[8]

A burgeoning epidemiological literature suggests that sleep patterns are related to CVD morbidity and mortality in adulthood.[9–13] In particular, either short or very long sleep duration, fragmented sleep, and insomnia-like symptoms have been connected to risk for CVD. Supporting these epidemiological data are a series of experiments depriving healthy participants of sleep for varying lengths of time and observing acute changes in cardiovascular risk factors, including BP, heart rate, glucose and insulin metabolic indices, and inflammation.[14–16] In recognition of the early origins of CVD, the extent to which sleep patterns are related to cardiovascular risk factors in children and adolescents has recently been a focus of investigation. The primary purpose of the present paper is to synthesize evidence on the association between sleep characteristics of young people and their cardiovascular risk factors, in particular, metabolic syndrome, glucose and insulin, lipids, BP, and heart rate and BP responses to stressful tasks, e.g., mental arithmetic or giving a speech. Stress-induced cardiovascular responses are included because of their association with risk with incident hypertension and CVD.[17]

Another purpose of the paper is to update prior reviews on the relationship between obesity and sleep characteristics. A 2008 meta-analysis of 17 studies found that the risk of being overweight or obese decreased by 9% for each additional hour of sleep, with effects stronger in boys than in girls.[18] Two literature reviews of sleep and obesity have been published since that time. Magee and Hale reported that all 7 longitudinal studies they reviewed observed a relationship between short sleep and increased weight and/or adiposity,[19] while Guidolin and Gradisar[20] reported that neither of the 2 longitudinal studies they identified observed a relationship. In the current review, we summarize the longitudinal studies that were published after January 2011 and were not included in the two previous reviews.

We chose to synthesize available evidence based on an enumerative or descriptive review, as opposed to using meta-analytic techniques, for several reasons. First, we did not want to combine all cardiovascular risk factors into one quantitative analysis because associations may vary by risk factor and by sleep characteristic. Even within one type of cardiovascular

risk factor or sleep characteristic, there can be quite different approaches to assessment. Although the study findings could be pooled initially to test for heterogeneity across studies, our review is aimed at identifying which risk factors seem to be linked. A compelling reason for meta-analysis is the increased power that results in combining individual studies with relatively small sample sizes. In this review multiple studies have large sample sizes and should have sufficient power to be considered in an enumerative review.

The sleep characteristics we reviewed are those considered to be the major dimensions of sleep health, i.e., duration, continuity, perceived quality, and daytime sleepiness.[21] We also included studies on sleep architecture when available and did not include sleep disordered breathing (SDB). Another major dimension of sleep health, timing, was not reviewed because of few studies in children and adolescence. Our general hypothesis is that short sleep, less continuous sleep, poorer quality, and more daytime sleepiness would be associated with elevated levels of cardiovascular risk factors. Because long sleep has also been associated with elevated CV risk in adulthood, we also identified studies that tested for a curvilinear relationship with risk factors. After summarizing the evidence for each risk factor, the review identifies subgroups that have stronger associations with sleep characteristics. The paper also highlights methodological issues and identifies directions for future research.

2. Methods

We used PubMed and PsycInfo to search for articles. We first searched for articles examining sleep and cardiovascular risk factors, excluding obesity, using the following combination of search terms: (“*sleep*” OR “*actigraphy*”) joined by “AND” with a cardiovascular risk factor (“*metabolic syndrome*,” OR “*lipids*,” OR “*cholesterol*,” OR “*blood pressure*,” OR “*insulin*,” OR “*glucose*,” OR “*heart rate variability*,” OR “*cardiovascular*”). “NOT” qualifiers included “*apnea*” and “*breathing*.” Age limiters (0 years – 29 yrs) were used to refine the search. Reference lists were used to identify additional articles. We included studies that a) had a mean sample age of 24 or younger, in accordance with the Centers for Disease Control and Prevention’s[22] definition of youth, and b) investigated sleep duration, continuity, quality, or sleepiness in relation to one or more of the cardiometabolic risk factors identified above. We also included studies on sleep architecture when available. Sleep continuity refers to the consolidation of one’s sleep throughout the night (e.g., sleep latency, sleep efficiency, wake after sleep onset), and sleep quality refers to the subjective assessment of how good or poor one’s sleep is.[21] Excluded from review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical samples (e.g., psychiatric) or participants with sleep disorders. Figure 1a displays the number of records identified, screened, and excluded. Thirty-nine studies met criteria for inclusion. These studies are listed in Table 1 according to category of risk factors, with those reporting multiple risk factors listed first. Within risk factor category, studies are listed by year of publication.

We then searched PubMed and PsycInfo for longitudinal studies on sleep and obesity, using the following combination of search terms: [(“*sleep*” OR “*actigraphy*”) AND (“*body mass index*” OR “*overweight*” OR “*obesity*” OR “*waist circumference*”) AND (“*prospective*” OR

"*longitudinal*")]. "NOT" qualifiers included "*apnea*" and "*breathing*." Age limiters (0 years – 29 yrs) were used to refine the search. Reference lists were used to identify additional articles. We included only those studies that a) had a mean sample age of 24 or younger, b) were published in 2011 or later, c) were not included in recent reviews of sleep and obesity in youth[19;20] and d) used a longitudinal design to examine sleep as a predictor of BMI or adiposity. Excluded from review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical or sleep-disordered samples. Figure 1b displays the number of records identified, screened, and excluded. Sixteen studies met criteria for inclusion. These studies are listed in Table 2 by year of publication.

We first describe the overall pattern of results within risk factor category below and discuss whether the pattern of results varies according to the sleep construct (duration, quality, continuity, and architecture; see Table 3). Then we discuss whether the findings vary by study characteristics: sleep measure (polysomnography (PSG), actigraphy, parent/self report), samples from United States vs. other, obesity within the sample, and age of participants. We considered the study as positive if any of the sleep characteristics in a given report were related to the risk factor in the expected direction and noted when the findings were in subgroups only. For example, if short sleep duration but not sleep continuity was related to BP, we considered it as a positive study; or if short sleep duration was related to BP in boys, but not girls, we considered it positive in subgroups. For the summaries where we characterized findings by study characteristics, e.g., comparing studies of children vs. adolescents, we considered the study positive if a sleep measure and multiple cardiovascular risk factors within a report were related as expected, and mixed, if it was less than a majority of the risk factors but at least one relationship in the expected direction. Thus, for example, if a report concerned BP, glucose, and total cholesterol in relation to sleep duration in elementary school aged children, and found expected associations for 2 of the 3, it would be considered positive; and mixed if there was one association, and 0 null. These judgments relied on the multivariate analyses where available. About one-third of the studies (14/38) that examined sleep duration in relation to cardiovascular risk markers reported that curvilinear or other statistical tests were used to investigate the potential association between long sleep and risk factors.

3. Results

3.1 Multiple risk factors

There are 10 studies that reported multiple risk factors in relation to sleep, with 5 specifically reporting results for the combined index comprising the metabolic syndrome. The individual components of the metabolic syndrome are considered in the sections below. Of the 5 on the metabolic syndrome, samples ranged from 37 to 1187. Participant ages ranged from 6 to 18 years. One reported that shorter sleep duration was associated with higher risk for the metabolic syndrome among 37 obese adolescents, [23] whereas three studies reported null effects for sleep duration.[24–26] The fifth study combined multiple characteristics of sleep into a latent factor and reported no direct effect of sleep on metabolic syndrome;[27] however, there was an indirect effect of overall sleep characteristics being

associated with lower aerobic fitness, which was, in turn, related to the metabolic syndrome. In sum, evidence is weak that sleep characteristics are related to the metabolic syndrome.

3.2 Glucose and insulin metabolism

There are 13 studies that reported associations between sleep characteristics and measures reflecting glucose and insulin metabolism. These measures included a variety of outcomes: fasting glucose, homeostasis model assessment of insulin resistance (HOMA-IR), 2-hour glucose tolerance test (OGTT), hemoglobin A1c (HbA1c; a measure of glycated hemoglobin), Matsuda index of insulin sensitivity, acute insulin response test (AIRg), insulinogenic index of insulin secretion (IGI), and whole body insulin sensitivity index (WBISI). Sample sizes ranged from 133 to 2053. Participant age ranged from 3 to 26 years. All studies were cross-sectional. Of the 13, 8 reported the hypothesized associations with sleep characteristics: with short duration,[28–35] with decreased continuity,[28;32;33] and with sleep stages.[28;31] Of the 8 positive studies, 2 reported a curvilinear association with sleep duration: adjustment for waist circumference resulted in only long duration being related to higher HOMA-IR,[30] whereas adjustment for obesity resulted in the curvilinear association of short sleep time with glucose and HbA1c remaining.[31] One of the 8 studies tested the association of a combined index of shorter sleep duration, more screen time, and higher sugary drink consumption with HOMA-IR, but did not perform a separate analysis of sleep duration. This study did adjust for a number of important covariates, including waist circumference, gender, parental BMI, and birth weight.[29] Two studies reported hypothesized associations in subgroups. Short sleep duration was associated with insulin, HOMA-IR, and leptin in girls only but these associations became nonsignificant with adjustment for waist circumference.[35] The other found that short sleep was related to high fasting glucose in obese but not nonobese children.[33] Five studies reported no associations;[24;26;36–38] all of these were studies of the metabolic syndrome and 1 was from a sample in a weight management clinic. In sum, the majority of the evidence suggests that sleep characteristics are related to indices of glucose and insulin metabolism.

3.3 Lipids

There were 7 studies that reported associations between lipids and sleep characteristics. These measures included total cholesterol, low density lipoprotein cholesterol (LDL-C) or non-high density lipoprotein cholesterol (HDL-C), HDL-C, and triglycerides; sample sizes ranged from 1198 to 4104. Participant age ranged from 10 to 26 years. For high triglycerides, there were 2 null studies[24;37] and 2 studies opposite to prediction.[26;38] For high LDL-C or non HDL-C, there was a positive association with poor sleep quality but not with short sleep duration,[39] another with short sleep duration in secondary students but not in primary students.[25] For low HDL-C, there was one positive study with high daytime sleepiness in girls but not boys,[36] and another with short parent-reported sleep duration but not with actigraphy measured sleep duration,[38] and 3 null studies.[26;37;39] One study with no direct measures of lipids found that female adolescents who reported short sleep duration on several occasions indicated 6–7 years later that they had been diagnosed by a physician as having high cholesterol.[40] The same relationship was not apparent in males. In sum, the evidence does not support sleep characteristics being associated with lipids.

3.4 Blood pressure

There were 21 studies that examined sleep characteristics and BP. Sample sizes ranged from 49 to 6940, participant ages ranged from 3 to 19, and studies varied widely in terms of using cutoffs for prehypertension/hypertension, continuous BP readings, or a combination of both. Only one of the 21 studies used a longitudinal design. Archbold et al.[41] performed in-home PSG assessment of children ages 6–11 and reported that shorter total sleep time (TST) at baseline predicted an increase in resting systolic (S) BP over 5 years, after adjusting for age, sex, ethnicity, SDB, and change in obesity. There was also a marginal effect of SDB on increase in SBP at follow-up.

Of the cross-sectional studies, 7 reported a relationship between decreased sleep duration *or* continuity and higher BP.[26;42–47] One study reported that poorer sleep quality was associated with hypertension.[39] In addition, 1 study that examined sleep stages reported that percentage of REM and slow wave sleep were each inversely associated with BP measurements taken the next morning.[48] Eight studies observed no relationship between sleep and BP,[24;35–38;49–51] and 2 studies reported that longer sleep was associated with higher BP.[52;53] One study reported mixed results, such that shorter parent- reported sleep was associated with increased BP in 11–14 year-old boys and girls, but with decreased diastolic (D) BP in younger boys.[54] In sum, the evidence linking sleep characteristics to BP in children and adolescents is mixed.

Of the 20 cross-sectional studies listed above, 4 used ambulatory BP monitoring over a period of 24 hours or longer. Three of these reported that decreased sleep duration or decreased sleep continuity was associated with elevated BP for a portion or all of the ambulatory monitoring period.[43;45;46] In contrast, Martikainen et al. reported that neither self-reported sleep disturbance nor actigraphy-assessed sleep characteristics were associated with ambulatory BP in a sample of Finnish 8-year-olds.[50;55] Thus, it is possible that multiple measurements of BP collected over an extended time period may reveal more robust associations with sleep than a limited number of clinic assessments.

3.5 Cardiovascular responses to stress and heart rate variability

Four studies examined sleep in relation to heart rate or BP responses to stress. Sample sizes ranged from 79 to 489, and participant age ranged from 8 to 29 years. Each study used a different stress task to measure cardiovascular responses. Results across the studies were mixed. Two studies reported that shorter sleep was associated with elevated or prolonged DBP response to stress;[51;56] however, one of these failed to control for a number of important covariates.[51] In a third study, poor sleep quality was associated with blunted DBP reactivity during a semi-structured stress interview in college students, and no other associations between sleep and reactivity were observed.[57] The fourth study reported no association between actigraphy-measured sleep or self-reported sleep disturbances and heart rate or BP responses to stress in young children.[50;55]

Six studies examined sleep in relation to high-frequency heart rate variability (HF-HRV) or respiratory sinus arrhythmia (RSA), which are markers of parasympathetic nervous system activity. The studies varied in whether they investigated parasympathetic activity during rest

and/or during psychological stress tasks. Sample sizes ranged from 29 to 334. Participant ages ranged from 3 to 29 years. The one prospective study reported that both decreased actigraphy- assessed sleep time and decreased sleep continuity, but not parent reports of sleep, predicted a higher resting ratio of low-frequency (sympathetic) to high-frequency (parasympathetic) power at 1-year follow-up in 165 children.[58] Of the five cross-sectional studies, 3 reported positive results, such that shorter or less continuous sleep was linked to lower HF-HRV/RSA either at rest or during stress,[56;59;60] while 2 studies reported null effects or effects in the opposite direction.[50;55;61] In sum, 4 of 6 studies supported a relationship between decreased sleep duration or continuity and decreased parasympathetic activity.

3.6 Comparisons of studies by sleep measure, country of origin, obesity as a covariate, and age

3.6.1 Sleep characteristics—Sleep duration or TST was examined in the majority of studies. Across cardiovascular risk factors, 25 findings were reported showing a relationship between shorter sleep and elevated risk in the whole sample or in sub-groups. Thirty findings were null. There were 7 reported associations between longer sleep and elevated risk. Of the 11 reports of sleep continuity (including wake after sleep onset, sleep efficiency, sleep latency, and/or fragmentation), there were 7 instances of decreased continuity being associated with elevated risk and 4 null reports. Of the 12 reports of sleep quality, disturbance, or daytime sleepiness; 4 were positive, 5 were null, and 3 indicated that more sleepiness or worse quality was associated with elevated risk. In sum, there were no clear differences in cardiometabolic risk by type of sleep characteristic.

3.6.2 Subjective vs. objective report of duration or continuity—Eighteen studies assessed sleep duration or continuity using PSG or actigraphy, while 20 studies used a self-report measure of sleep duration or continuity. Regardless of cardiovascular risk factor, results tended to be more positive for studies that used an objective sleep assessment, with 11 positive, 4 mixed, 2 null, and 1 reporting longer sleep and elevated risk. Studies that used self-reports of duration or continuity were more likely to report null results, with 3 positive, 8 mixed, and 7 null, and 2 reporting longer sleep and elevated risk.

3.6.3 Country of origin—Eighteen papers reported on data collected in the United States and 21 papers reported on data collected in other countries, including Belgium, Brazil, Canada, China, Finland, Germany, Greece, Hong Kong, India, Iran, Japan, Korea, Lithuania, Netherlands, and Portugal. Regardless of cardiovascular risk factor, reports were similar in terms of those supporting the hypothesized direction of effect: for studies originating in the U.S., 10 were positive, 1 null, 6 mixed, and 1 opposite to hypotheses, vs. elsewhere 7 were positive, 4 null, 8 mixed, and 2 opposite to hypothesis.

3.6.4 Obesity—Adiposity measured as BMI or waist circumference served as a covariate in 27 studies; 11 studies did not adjust for measures of adiposity. In general the studies that adjusted for adiposity did not differ in the extent of support for associations with risk factors from those studies that did not adjust for adiposity. Four studies only recruited obese adolescents and children; of these 2 were positive and 2 were mixed. One study reported in

subanalyses that shorter sleep was related to fasting glucose > 100 mg/dl only in obese children.

3.6.5 Age—Age of participants ranged from preschool to young adulthood (early 20's). Nineteen studies reported a mean age of 14 years or older or conducted age-stratified analyses in older adolescents. Nine of these studies were positive, 7 were mixed, and 3 were null. Ten studies reported a mean sample age of 10 or younger or conducted age-stratified analyses in younger adolescents. Of these, 3 reported positive results, 1 reported mixed results, 5 reported null results, and 1 was negative. Thus, full or partial support for relationships between sleep and cardiovascular risk was more likely in older versus younger youth.

3.7 Longitudinal Studies of Obesity

We identified 16 longitudinal studies of sleep and obesity published since 2011 and not included in previous reviews.[19;20] Note that the cross-sectional studies outlined in Table 1 that included measures of weight are not included in this review. All studies focused on sleep duration, with only 1 study also including a measure of sleep disturbance. In terms of sleep duration, 8 studies reported that shorter sleep predicted higher BMI, greater increases in BMI, or greater risk of overweight/obesity over time compared to longer sleep duration, in either the full sample or in both boys and girls.[62–69] Three studies reported that shorter sleep was predictive of higher BMI or greater weight increases in boys but not girls.[70–72] Three studies reported that associations between sleep duration and anthropometric outcomes varied by other sub-groups, such as age or outcome of interest.[73–75] Two studies reported that sleep duration was not a significant predictor of BMI over time.[76;77]

4. Discussion

Our enumerative review of the association of cardiovascular risk factors and sleep characteristics in children and adolescents revealed several findings. First, evidence for associations with sleep characteristics is most consistent for obesity, then glucose and insulin metabolism, followed by BP (especially 24 hour ambulatory BP), and parasympathetic responses to psychological stressors . The evidence suggesting that short sleep leads to increased risk for obesity is particularly striking, especially given the longitudinal designs of the studies, and that obesity increases risk for other cardiovascular risk factors and tracks over time in young people.

On the other hand, evidence suggests null or weak associations for metabolic syndrome cluster, lipids, and BP responses to psychological stressors. These conclusions should not be considered definitive in light of many more reports regarding obesity, glucose and insulin metabolism, and BP than for metabolic syndrome, lipids, and BP responses to stress. Further examination of these parameters may reveal a different pattern of results using longitudinal designs and more thorough assessment of sleep characteristics. In that regard, it is noteworthy that the only longitudinal study other than those related to obesity did find that short sleep predicted increases in blood pressure across five years; in contrast to the number of cross-sectional studies of blood pressure that found no associations.[41]

Second, given the large number of studies with a variety of participants from many developed countries, it is not surprising that the strength of associations varied by several key covariates or descriptors of the study. It appears that the associations with cardiovascular risk factors are somewhat more consistent in older than younger children and in studies that used "objective" measures of sleep as opposed to self- or parent-report. The finding that objective measures may reveal stronger associations than subjective measures indicates that future studies should preferentially use objective measures. It may be that child- or parent-report measures of sleep are less accurate for children because they usually do not sleep in the room with their parents and because of the challenges of obtaining reliable self-report data regarding any characteristics from younger children. Objective measures, e.g., actigraphy or in home polysomnography, usually accompanied by sleep diaries, are feasible in large scale studies but do require compliant participants and are more expensive and labor intensive than self- or parent-report measures.

Surprisingly the results did not vary substantially by the specific sleep characteristic (i.e., duration, continuity, quality) or by whether obesity was introduced as a covariate. The latter may be due to the number of studies with only obese participants, as well as the substantial number of studies in countries that do not have epidemics of obesity as is occurring in the United States.

Third, with the exception of studies of obesity, almost all studies reviewed herein were cross-sectional in nature. Given the foundation of research summarized in this review, it is now time to examine antecedent and consequent relationships among sleep characteristics and cardiometabolic factors. All the risk factors considered are impacted by weight gain so longitudinal studies must examine how weight change affects the relationships between sleep and change in cardiovascular risk factors. Otherwise, relationships that may be attributed to the risk factors may be secondary to weight gain. Finally, it also time to conduct randomized trials to address whether improving sleep can lead to improving cardiovascular risk factors.

Fourth, only two studies included indices of mental health as covariates. A very large literature suggests that depression and anxiety are consistently related to future cardiovascular morbidity, mortality, and subclinical CVD.[78] Poor sleep is also intertwined with depression and anxiety,[79] and the mechanisms accounting for their associations may be similar to the mechanisms accounting for associations between depression and anxiety and cardiovascular risk.[80] It is important to include indices of depression and anxiety in future studies to examine whether poor sleep leads to mental health problems, which, in turn, lead to cardiometabolic risk later in life, or whether mental health problems lead to poor sleep, which then leads to cardiovascular risk.

Fifth, although timing of sleep is a key characteristic of sleep health, we only found one relevant study. Scharf and DeBoer[66] noted that a later bedtime in 4-year-olds predicted an increase in BMI by age 5. Previous studies not included in this review also suggest an association between variability in sleep timing and risk factors, including higher BMI,[81] inflammatory activity, [82] and sympatho-adrenal- medullary activity. [83] The sleep-wake cycle, as well as many of the physiological systems implicated in cardiometabolic disease

risk, including glucose metabolism, adipocyte function, and vascular function, are closely tied to and may be influenced by circadian rhythms[84;85] or circadian preference.[86] Thus, it is possible that that circadian dysregulation is an underlying cause of both disrupted sleep and variations in CVD risk markers.

We did not include inflammatory risk factors because of the few studies available, other than those concerning SDB. However, elevated levels of a generic marker of inflammation, C-reactive protein (CRP), were related to shorter sleep duration in several cross-sectional studies of adolescents.[87–89] Inflammation is an important arena for investigation, especially given substantial literature showing that CRP and interleukin 6 predict the early development of CVD.[90] As investigations move forward in this arena, it is important to measure SDB, especially in populations of overweight children and adolescents.

Finally, the review does not address whether effects are stronger in lower SES or minority children and adolescence because rarely did papers report moderation effects by SES or race. More generally, it is worthwhile to consider that poor sleep may inform the relationship between low SES and minority status with cardiovascular disease risk. Supporting this notion is ample evidence that low SES in childhood is related to elevated cardiovascular morbidity and mortality in adulthood,[91;92] although low SES is not consistently related to cardiovascular risk factors in childhood.[93] Black children and adolescents have shorter sleep than their white counterparts but they report fewer insomnia-like symptoms.[81;94–96] This pattern of results by race is consistent with meta-analyses documenting similar associations in adults.[97;98] Evidence regarding the influence of SES on sleep of children and adolescence is less clear but it is reasonable to hypothesize adverse effects of low SES because environmental factors, such as inadequate heating and cooling, noise, irregular routines, and stress, do covary with SES and impact sleep.

The present review has a number of strengths and weaknesses. The strengths are clear specification of inclusion criteria for studies summarized in the review; detailed description of sleep measures and cardiovascular risk factors; and identification of types of studies and participants that show more positive vs. null associations. The weaknesses of the review are primarily a consequence of the current status of the literature: absence of a sufficient number of longitudinal studies that would permit conclusions regarding antecedent and consequent relationships; the heterogeneity of the studies making meta-analysis less attractive; key information lacking in some papers, e.g., adjustments for obesity, age stratification that is not comparable across studies; few studies examining the impact of long sleep duration; and few studies of inflammatory markers. Furthermore, little is known about variability in sleep patterns or sleep timing. Addressing these weaknesses in future research, however, provides guidelines for additional investigation.

Given the state of the field, it may be premature to recommend health policy and service delivery changes at this time. The exception is obesity, with the caveat that no clinical trials are yet available. Should short, discontinuous, and low quality sleep be related to prevention of obesity, there are many ways that sleep can be improved. For example, school start times can be delayed to permit more opportunity to sleep. Health care providers can provide

materials on improving sleep hygiene and parents and students educated regarding the negative effects of poor sleep. Sleep interventions could target obese children.

5. Conclusions

Cardiovascular risk factors emerge in childhood and adolescence and impact long-term cardiovascular health. The extent to which sleep characteristics play a role in understanding cardiovascular risk in young people is an area of active international investigation. While an important topic, substantial challenges exist in addressing the roles of sleep characteristics. Sleep patterns change dramatically during childhood and adolescence. Rates of maturation vary across boys and girls as well as within gender such that age adjustments and grouping by age are not sufficient proxies. Demands of school and home superimpose constraints that entrain circadian and sleep patterns that vary by culture. Although general standards for optimal sleep exist according to developmental stages, they do not take into account the sleep “needs” of the individual based on their diet, activity pattern, environment, and genetic make-up. Thus far, cross-sectional evidence provides the bulk of the relevant data we have reviewed and no clinical trials are available. Yet hypothesized relationships may be obtained in longitudinal data even when they are not obtained in cross-sectional data. Although the current state of evidence varies by risk factor, there are enough positive findings, particularly in studies employing the more objective measures of sleep and including adolescent samples, to provide support for substantial future efforts to understand the links between sleep and cardiovascular risk in young people.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

M	mean
NS	nonsignificant
MetS	metabolic syndrome
BMI	body mass index
BP	blood pressure, S systolic, D diastolic
MAP	mean arterial blood pressure
HR	heart rate
CO	cardiac output
HF-HRV	high frequency heart rate variability
LF-HRV	low frequency heart rate variability

RSA	respiratory sinus arrhythmia
HbA1c	hemoglobin A1c
HDL-C	high density lipoprotein cholesterol
TC	total cholesterol
LDL-C	low density lipoprotein cholesterol
HOMA-IR	homeostasis model assessment of insulin resistance
IGI	insulinogenic index of insulin secretion
WBISI	whole body insulin sensitivity index
AIRg	acute insulin response to glucose
OGTT	oral glucose tolerance test
SDB	sleep disordered breathing
TST	total sleep time
REM	rapid eye movement
N3	sleep stage #3
SWS	show wave sleep
PSG	polysomnography
IOTF	International Obesity Task Force
CVD	cardiovascular disease
SES	socioeconomic status

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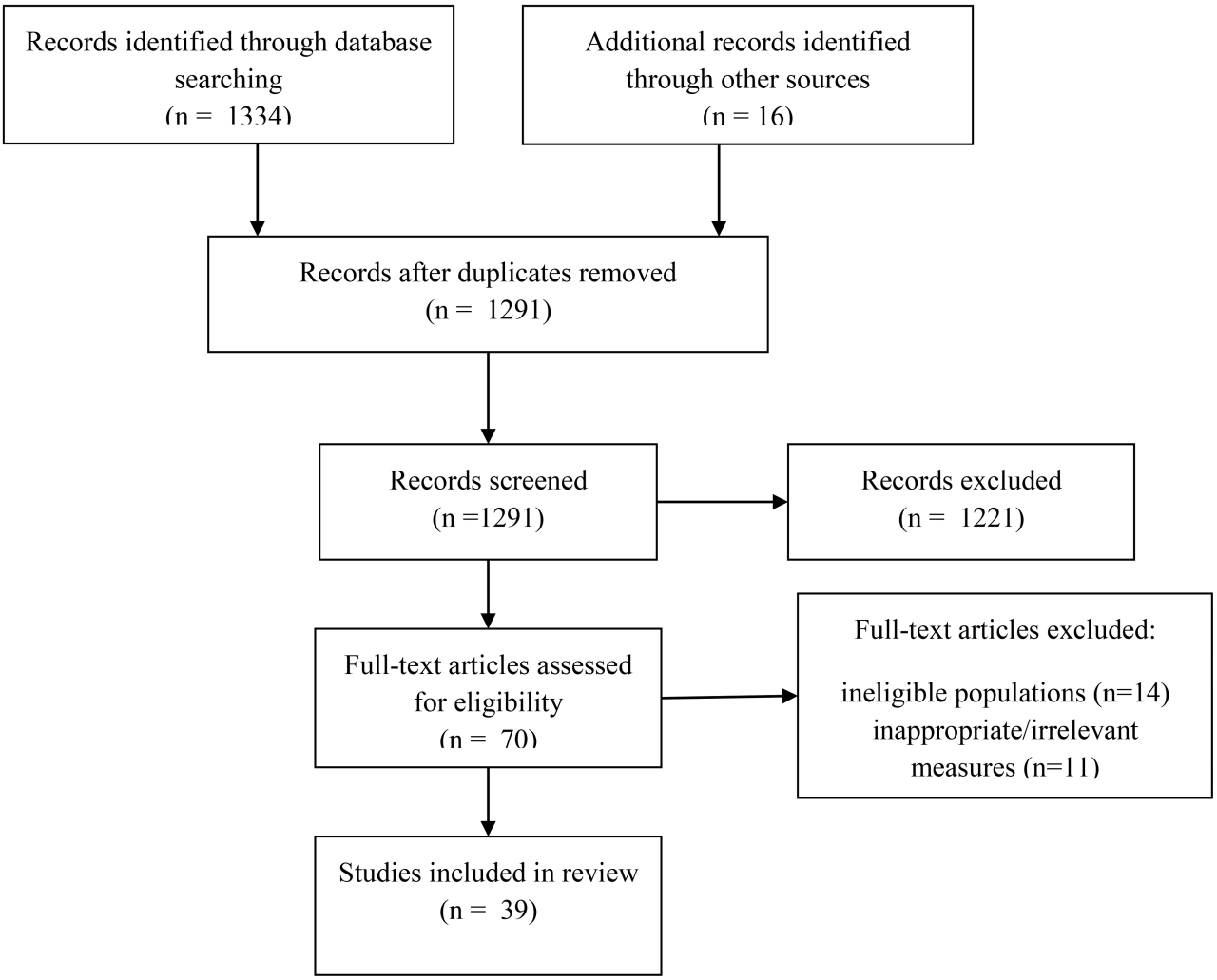
Highlights

- We review 55 studies of cardiovascular risk factors, obesity, and sleep in youth.
- There are 39 studies of sleep and risk factors, most of which are cross-sectional.
- In cross-sectional studies, the most consistent evidence links sleep to glucose/insulin.
- Data from 16 longitudinal studies suggest that short sleep predicts obesity.
- More longitudinal studies that use objective sleep measures are needed.

a)



b

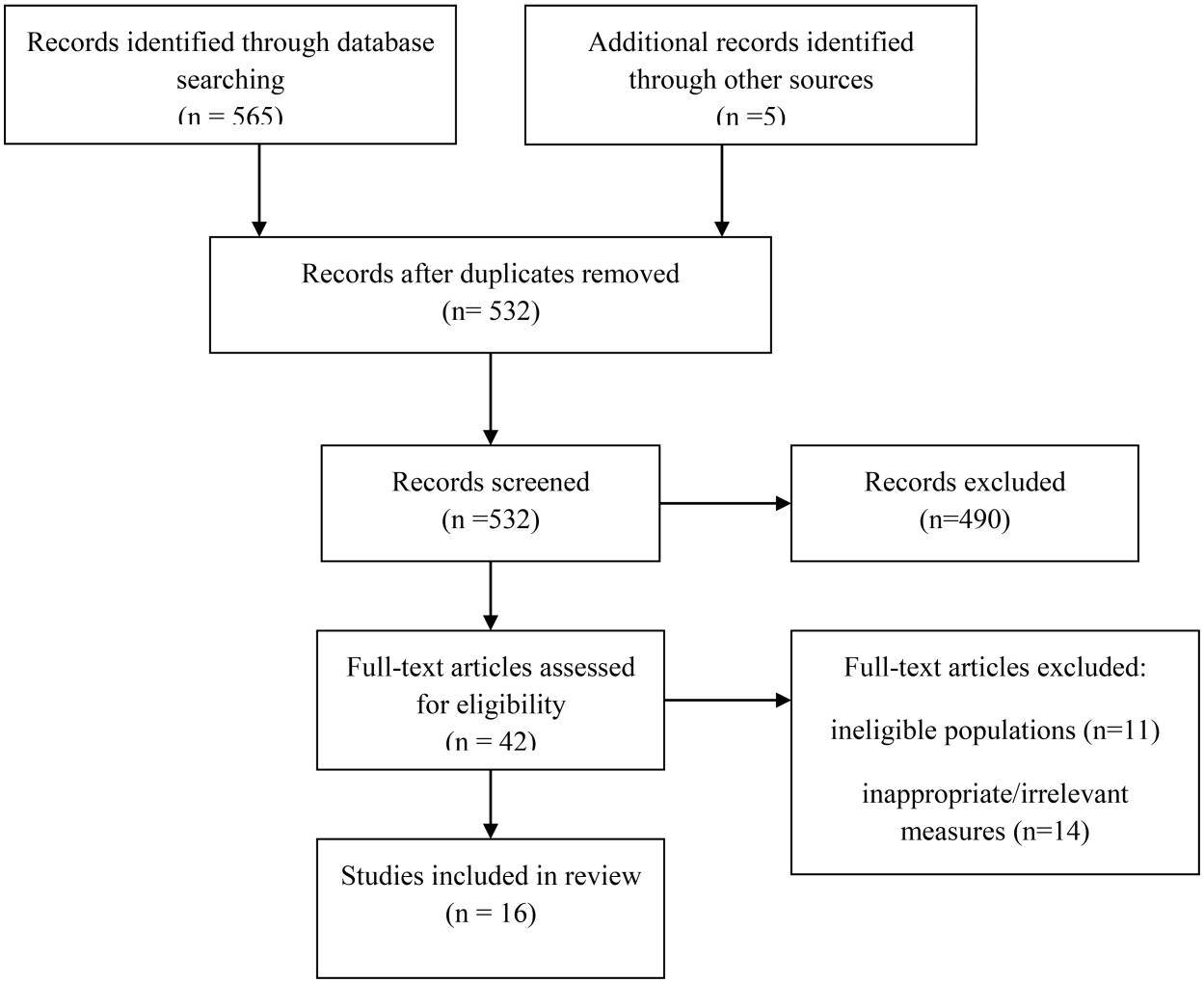


Figure 1.

a. Flow diagram of study selection for sleep and cardiovascular risk factors.

Note: Included studies had a) had a mean sample age of 24 or younger, and b) investigated sleep duration, continuity, quality, timing, or sleepiness in relation to one or more of the cardiometabolic risk factors (metabolic syndrome, glucose/insulin, lipids, blood pressure, cardiovascular stress responses). We also included studies on sleep architecture when available. Excluded from review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical samples or participants with sleep disorders.

b. Flow diagram of longitudinal study selection for sleep and obesity.

Note: Included studies a) had a mean sample age of 24 or younger, b) were published in 2011 or later c) were not included in recent reviews of sleep and obesity in youth, and d) used a longitudinal design to examine sleep as a predictor of body mass index or adiposity.

Excluded from review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical samples or participants with sleep disorders.

Table 1

Sleep and Cardiovascular Risk Factors organized by Category of Risk

First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
IglayReger[23]	37 obese U.S. adolescents, 54.1% female, ages 11–17; M =14.0 ± 0 yrs	cross-sectional	≥ 5 nights actigraphy-assessed sleep duration	MetS composite risk score	BMI, physical activity duration and intensity	↓sleep duration ↑ MetS composite risk score
Lee [26]	1187 Korean adolescents, 46.9% female, ages 12–18; M =15.0 ± .1 yrs	cross-sectional	self-reported sleep duration	MetS and MetS components	age, sex, household income, caloric intake, physical activity	↓sleep duration ↑BP, ↑BMI, ↑waist circumference ↓ triglycerides NS glucose, HDL-C, MetS
Azadbakht[24]	5528 Iranian children ages 10–18; M =14.7 yrs	cross-sectional	parental report of sleep duration	BP, lipids, glucose, BMI %, physical activity	SES, family history, physical activity, BMI, age	all NS for multivariate analyses
Berentzen[36]	1481 Dutch children ages 11–12; M =12.7 ± .4 yrs	cross-sectional	self-report time in bed on school day, sleep pattern, nighttime awakenings, trouble falling asleep, daytime sleepiness	cholesterol, HbA1c, BP	age, height, maternal education, puberty, screen time	↓ time in bed ↑ BMI, waist circumference ↑daytime sleepiness ↓HDL-C, ↑TC/HDL-C in girls only. NS boys no effects for BP, HbA1c
Rey-Lopez[37]	699 European adolescents, 51.6% female, ages 12.5-17.5; M =14.8 yrs	cross-sectional	self-reported sleep duration	HOMA-IR, triglycerides, TC, HDL-C, systolic BP	age, sex, SES, physical activity	all NS
Countryman[27]	367 U.S. adolescents 27% female, 45.8% Hispanic, 30.8% Black, ages 15–17; M =16.1 ± .7 yrs	cross-sectional	latent sleep factor (composed of self-reported sleep duration over past 7 days, fatigue, & sleep quality)	latent MetS factor (composed of obesity, insulin resistance, lipids, and BP)	gender, parent education	In structural equation model, sleep was indirectly associated with increased risk of MetS via decreased aerobic fitness
Narang[39]	4104 Canadian adolescents;51% male, M =14.6 ± .5 yrs	cross-sectional	self-reported sleep quality self-reported sleep duration	TC, HDL-C, non-HDL-C, prehypertension (≥90 - <95th % based on age, sex, height) or hypertension (≥99th %)	sex, family history of CVD, adiposity, nutrition, physical activity, screen time	↓sleep quality ↑ non-HDL-C ↑ hypertension NS TC NS HDL-C ↓self-report duration all NS

First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
Kong[25]	2053 Hong Kong children ages 6–18 years, M = 13.3 yrs	cross-sectional	self-reported sleep duration in full sample, actigraphy for 24 hrs in 138 children selected on obesity	lipids, metabolic syndrome	age, gender, BMI, pubertal stage	↓ self-reported duration ↑ TC and LDL-C in multivariate analyses in secondary students, NS for primary students; no report for actigraphy sleep, BP, glucose
Sung[38]	133 obese U.S. adolescents in tertiary care weight management clinic, 66% female; ages 10–16 yrs. M=13.2 ± 1.8 yrs	cross-sectional	self-reported sleep duration, parent-reported sleep duration, 7 nights actigraphy-assessed sleep duration	MetS, MetS components (waist circumference, triglycerides, BP, HDL-c, glucose), HOMA-IR	age, gender, race, SES, BMI, obstructive apnea	↓self-report duration ↓ triglycerides NS for all others ↓parent-report duration ↓ HDL cholesterol NS for all others ↓actigraphy duration ↓ triglycerides NS for all others
Hitze[35]	414 German children ages 6–20; M =13.0 ± 3.4 yrs	cross-sectional	self-report after 11, parent report before 11, cutoffs based on age	manual BP, lipids, glucose, leptin, adiponectin, HOMA-IR	age	↓sleep duration ↑insulin, HOMA-IR, leptin in girls. NS after adjustment for waist circumference; NS in boys
Gangwisch[40]	14257 U.S. adolescents in ADD Health, 51% female, grades 7–12 at baseline	longitud-inal	self-report sleep duration at 2 times averaged	self-report of doctor diagnosing high cholesterol 6–7 years later	physical activity, emotional distress, BMI groups, age, sex, race, alcohol and smoking	↓sleep duration ↑cholesterol in females; NS in males. Test for sex interaction NS
Glucose and Insulin Metabolism Studies						
Androutsos[29]	2026 Greek children, 50.1% female, ages 9–13	cross-sectional	parental report of sleep duration	HOMA-IR	gender, Tanner stage, waist circumference, parent BMI, SES, birth weight	children with an unhealthy "lifestyle pattern," consisting of ↓ sleep duration, ↑ screen time, and ↑sugary drink consumption, had ↑HOMA-IR
Zhu[34]	118 healthy Chinese children and adolescents; 55.1% female, moderate-to-severe OSA excluded M=13.1 ± 3.3 yrs	cross-sectional	1 night PSG -TST -sleep efficiency	2-hr oral glucose tolerance test, insulin sensitivity (Matsuda index)	age, gender, BMI, pubertal status, AHI	↓TST ↑ glucose levels ↓ insulin sensitivity ↓sleep efficiency ↑ glucose levels ↓ insulin sensitivity ↓% stage 3 ↑glucose ↓insulin sensitivity
Matthews [32]	245 healthy U.S. adolescents, 56% African American, 53% female; ages	cross-sectional	diary & actigraphy sleep duration for 1 week,	HOMA-IR, glucose	age, race, gender, BMI, waist circumference	↓ nocturnal sleep ↑HOMA-IR stronger in males, effect due to weekday sleep ↑fragmentation

First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
	14–19 M = 15.7±1.3 yrs		fragmentation			↑glucose
Javaheri [30]	471 U.S. adolescents in Cleveland Children's Sleep and Health study; 50.7% female, 42.7% minority race; ages 13–19 M =15.7 ± 2.2 yrs	cross-sectional	actigraphy-assessed sleep duration	HOMA-IR	Model 2: age, sex, race, physical activity, preterm history Model 3: above + waist circumference	Model 2: curvilinear association of sleep duration with ↑HOMA-IR Model 3: only long sleep duration related to ↑HOMA-IR
Koren [31]	62 obese U.S. adolescents, 54.8% African American, 37.1% White, 55% female; ages 8–17.5 M = 14.4 ± 2.1 yrs	cross-sectional	in clinic PSG TST, sleep stages	HOMA-IR, OGTT, IGI, WBISI	extent of obesity, OSA	curvilinear association of TST with ↑glucose and HbA1c; NS with HOMA-IR, WBISI, IGI; ↑N3 ↑IGI and AIRg (i.e., beta cell function)
Tian[33]	619 obese & 617 nonobese Chinese children, matched by age; ages 3–6 M = 5.3 ± .9 yrs	cross-sectional	parent-reported sleep duration	fasting glucose, hyperglycemia (fasting glucose ≥ 100 mg/dL)	BMI, age, sex, birth weight, gestational age, systolic BP, parents' education and BMI, breast-feeding, timing of food introduction, disease in past month, diet, sweetened beverage consumption, TV viewing, physical activity	↓ sleep duration ↑ glucose ↑ hyperglycemia in obese only Glucose NS after adjusting for waist circumference
Flint[28]	40 obese U.S. children from weight clinic (32 with SDB), ages 3.5–18.5 M =12.3 ± 4.2 yrs	cross-sectional	in clinic PSG for sleep duration, efficiency, AHI, % stages	OGTT insulin and glucose, HOMA-IR, WBISI	None	<6 hr sleep ↑fasting and peak insulin, HOMA-IR, WBISI, and ↓ % REM in univariate analyses; did not report sleep efficiency
Blood Pressure Studies						
Kuciene [44]	6940 Lithuanian children ages 12–15; M =13.4 yrs	cross-sectional	self-report TST	SBP, DBP (oscillometric) ≥90th, ≥95th %ile based on age, sex, height	BMI groups, physical activity, smoking, age, sex	Compared to ≥8hr, ↓ sleep ↑ risk for ≥90th, ≥95th % BP
Meininger[45]	366 U.S. adolescents 53.6% female, 37% Black, 31% Hispanic, 29%	cross-sectional	24-hr actigraphy-assessed sleep (nighttime and	24-hr ambulatory SBP and DBP on a school day	age, sex, racial/ethnic group, mother's education, BMI,	↓nighttime sleep duration ↑ ambulatory SBP NS ambulatory DBP ↓daytime sleep duration

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First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
	White, ages 11–16		daytime sleep duration)		sexual maturation, physical activity, position and location during BP reading	↑ ambulatory SBP ↑ ambulatory DBP
Paciência[52]	1771 Portuguese 13 year-olds, 53.5% female	cross-sectional	self-reported sleep duration	prehypertension (BP > 90th %ile for sex, age, and height)	females: BMI, caffeine intake, depression males: BMI, caffeine intake, sports	↑sleep duration females: ↑ BP males: NS
Archbold[41]	334 U.S. Hispanic & white children, 6–11 years; M =9.03 ± 1.63	longitud-inal for 5 years	in-home PSG-based SDB, TST	Obesity, BP ?	sex, ethnicity, age, change in obesity	↑obesity and ↓TST related to ? SBP; NS DBP
Mezick[46]	246 healthy U.S. adolescents; 53.3% female, 56.5% Black; ages 14–19, M =15.7 ± 1.3 yrs	cross-sectional	7 nights actigraphy-assessed sleep duration; efficiency	24 hr ambulatory BP, nighttime ambulatory BP, daytime ambulatory BP	age, sex, race, BMI	↓sleep duration ↑ 24-hr SBP ↑ 24-hr DBP ↑ nighttime SBP ↑ nighttime DBP NS daytime SBP, DBP efficiency not related to BP
Guo[54]	4902 Chinese children, ages 5–18, M = 10.9 ± 2.7 yrs	cross-sectional	parental report of TST	BP (mercury column) ≥ 90% for age, sex, height, or ≥ 120/80, hypertensive ≥ 95%	age, BMI, waist circumference, physical activity	↓TST ↑ SBP, DBP levels among boys 11–14 ↑ DBP among girls 11–14 ↓ DBP among boys 5–10 years. NS in other age groups
Bayer[49]	7701 German children, 49% female, ages 3–10	cross-sectional	parental report of sleep duration standardized within age group	MAP (oscillometric)	BMI (age-, sex-specific), parental report of physical activity	↓ sleep duration ↑MAP, NS in multivariate analysis
Javaheri [42]	238 U.S. adolescents without clinical sleep apnea in Cleveland Children's Sleep and Health study; 48.3% female, 45% White; M = 13.7± .7 yrs	cross-sectional	5–7 nights of actigraphy -sleep duration -sleep efficiency	prehypertension defined as BP ≥ 90th percentile for sex, age, and height, continuous resting SBP and DBP	age, BMI, socio-economic status, models examining continuous BP outcomes also adjusted for sex, race, term status	↓sleep duration prehypertension, SBP, DBP all NS ↓ sleep efficiency ↑ prehypertension ↑ SBP ↑DBP associations replicated using PSG
Wells[47]	4452 Brazilian adolescents, ages 10–12	cross-sectional	self-report bedtime & wake up time during week	oscillometric BP ≥120/80	physical activity, BMI groups, sex, SES, birth weight, length, maternal health habits	↓ sleep duration ↑SBP NS DBP

First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
Sampei[53]	117 Japanese children, ages 5–6	cross-sectional	parental report of sleep + teacher report of naps	mercury column BP	age, sex, BMI, school	↓TST ↓ SBP
Au[43]	143 normal weight Chinese children and adolescents, 42% female, AHI ≥ 5 excluded, ages 10-17.9; M = 14.3 ± 1.8 yrs	cross-sectional	1-night PSG -sleep time -sleep efficiency 7-day sleep diary duration	24-hr ambulatory BP -prePSG BP -in-bed BP -postPSG BP	age, gender, BMI, AHI, parental hypertension	↓PSG sleep time ↑ postPSG SBP NS other BP outcomes ↓PSG sleep efficiency ↑ in-bed SBP ↑ in-bed DBP ↑ postPSG DBP NS other BP outcomes ↓sleep diary duration ↑ prePSG SBP ↑ prePSG ↑ in-bed SBP ↑ in-bed DBP ↑ postPSG SBP NS postPSG DBP
Hannon[48]	49 obese, nondiabetic U.S. adolescents, 48.1% female, 57.1% White, 40.7% Black, ages 12–18; M = 14.4 yrs	cross-sectional	1-night PSG -REM % -SWS % -TST -sleep latency -time to REM	resting oscillometric BP assessed morning after PSG.	age or pubertal stage, sex, race, BMI, AHI	↓REM % ↑ SBP ↑ DBP ↓SWS % ↑ SBP ↑ DBP NS for other sleep parameters
Reactivity/HRV Studies						
Mezick[56]	79 healthy normal weight U.S. college students, 100% male, ages 18–29, M = 19 ± 2.0 yrs	cross-sectional	7 nights actigraphy-assessed sleep duration	HR reactivity, BP reactivity, HRV reactivity, HR recovery, BP recovery, HRV recovery	age, race, BMI, daily caffeine, daily nicotine, stress task appraisals, naps	↓ duration NS HR reactivity NS SBP reactivity NS DBP reactivity ↑HRV-HF reactivity ↑ HR recovery NS SBP recovery ↑ DBP recovery NS HRV-HF recovery
Martikainen[55]	241–274 Finnish 8-year-olds (number varied by outcome)	cross-sectional	sleep disturbance, Scales: 6 subscale scores (yes/no at least 1 sleep problem 2-3x per week)	Cardiovascular reactivity, Ambulatory BP 24 hours	sex, age, height, BMI, start time, education, maternal licorice pregnancy yes/no	excessive somnolence ↑HF-HRV at rest SDB ↑ CO + HR reactivity
Williams[57]	98 U.S. college students; 50% female, 74% White, 7% Latino/a, M =	cross-sectional	prior month sleep quality, prior night sleep quality & TST	HR reactivity, SBP reactivity, DBP reactivity	baseline CV parameters	↓prior month quality NS HR reactivity NS SBP reactivity ↓ DBP reactivity

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First Author	Sample	Study Design	Sleep Measures	CV Risk Factors	Covariates	Results
Metabolic Syndrome and Multiple Risk Factors						
	23 ± 5.8 yrs					↓prior night quality all NS ↓prior night TST all NS (marginal effect on SBP reactivity)
Michels[58]	334 Belgian children, 47% female, ages 5–11, actigraphy in subsample of N = 165	cross-sectional and longitudinal	parent-reported sleep duration, actigraphy-assessed latency, TST, efficiency	resting HRV, (HF-HRV and HF/LF ratio)	age, sex, physical activity, parental education, stress	↓parent-reported sleep duration NS HF-HRV NS LF/HF ratio ↑actigraphy latency ↓HF-HRV ↑LF/HF ratio ↓actigraphy efficiency NS HF-HRV ↑ LF/HF ratio ↓actigraphy TST NS HF-HRV ↑ LF/HF ratio longitudinal results replicated cross-sectional results
El-Sheikh[59]	224 U.S. children, 64% European American, 36% African American, 46% female, ages 8–10	cross-sectional	7 nights actigraphy- TST, sleep activity, wake after sleep onset	resting RSA, vagal withdrawal during stress	age, sex, race, BMI, asthma	↑ Wake after sleep onset ↑ vagal withdrawal during stress all other main effects NS ↓ resting RSA and ↑ vagal withdrawal during stress interacted to predict ↑ wake after sleep onset, ↑ sleep activity TST NS
Elmore-Staton[60]	29 U.S. preschoolers; 31% female, 64% European American, ages 3–5, M=3.99 ± .69 yrs	cross-sectional	actigraphy-assessed TST, efficiency, sleep activity	resting RSA	age, sex, ethnicity	↓ sleep efficiency & ↑ sleep activity ↓ RSA trend for ↓ TST, ↓ RSA
Martikainen[50]	231–265 Finnish 8 year olds (number varied by outcome)	cross-sectional	actigraphy- TST, efficiency, fragmentation	CV reactivity, ambulatory BP	sex, age, height, BMI, maternal licorice during pregnancy, parental education	all NS
Shaikh[51]	489 Gujarti Indian adolescents; 42% female, ages 16–19	cross-sectional	self-reported sleep duration (≥ 7 vs. < 7 hrs)	resting BP, DBP reactivity	none reported	those sleeping <7 hrs had higher DBP reactivity vs. those sleeping ≥ 7 hrs; resting SBP and DBP were NS
El-Sheikh[61]	41 U.S. children, 44% female, ages 6–12; M = 10.06 ± 1.74 yrs	cross-sectional	4 nights actigraphy- TST, sleep efficiency, self-reported sleep-wake problems and sleepiness	resting RSA, vagal withdrawal during stress	gender, age, puberty status	↓TST ↓ vagal withdrawal during stress ↑ sleep-wake problems ↑ resting RSA, ↓ vagal withdrawal during stress sleep efficiency NS

Table 2

Longitudinal Studies of Sleep and Obesity

First Author	Sample	Study Design	Sleep Measures	Obesity Measures	Covariates	Results
El-Sheikh[63]	273 children, mean age of 9.4 yrs at Time 1	longitudinal, Time 1: 2009/2010, Time 2: 2010/2011, Tim 3: 2011–2012	sleep-wake problems (School Sleep Habits Survey), actigraphy-assessed sleep duration	BMI	sex, ethnicity, puberty, income-to-need ratio, asthma, medication use	↑ sleep problems at Time 1 = ↑ BMI at Time 3 in girls. ↓ sleep duration at Time 1 = ↑ BMI at Time 3 in boys and girls, and ↑increase in BMI in girls.
Scharf[66]	10,700 4–5 yr olds in Early Childhood Longitudinal Study-Birth Cohort (N=7000 at age 5)	longitudinal, measures assessed at ages 4 and 5	parent-reported weeknight sleep duration, based on bedtime and waketime	BMI z-score	sex, race/ethnicity, SES, TV viewing	↓ sleep duration at age 4 =↑ BMI increase by age 5. Later bedtime at age 4 = ↑ BMI increase by age 5.
Taveras[69]	1046 children in Project Viva, 6 months old at baseline	longitudinal, yearly assessments from infancy until 7 years	sleep duration score, based on parent reports at each yearly assessment vs. published norms for sleep duration	BMI z-score, total fat mass index, trunk fat mass index, skinfold thickness, waist and hip circumference at age 7	age, gender, race/ethnicity, maternal age, maternal BMI, maternal education, maternal parity, household income, TV viewing time	lowest sleep duration group = ↑ BMI, total and trunk fat mass index, skinfold thickness, waist and hip circumference at age 7 vs. reference group sleep duration after age 2 = NS with BMI at age 7
Chang[73]	6220 children in 5th grade at baseline, in the Early Childhood Longitudinal Study-Kindergarten Cohort	longitudinal, children followed from 5th through 8th grade	sleep duration derived from parent-reported bedtime and official school start time	3 BMI groups: healthy weight (<85th % for age, gender), overweight (≥ 85th% - <95th %) or obese (≥95th %)	gender, age, parental health, child health, race/ethnicity, parent education, family structure, poverty level	"obese" in 5th grade ↑ sleep duration predicted moving into "overweight" or "healthy" category by 8th grade (vs. staying in obese category) "healthy weight" in 5th grade ↑ sleep duration predicted moving into "overweight" or "obese" category by 8th grade (vs. staying in healthy category)
Magee[75]	1079 children 4–5 yrs at Wave 1 in the Longitudinal Study of Australian Children	longitudinal, 4 waves of data across 6 yrs	parent-reported diary duration for first 3 waves; self-reported duration at Wave 4	3 BMI groups based on weight at 4 waves: healthy weight (↓ IOTF overweight at all waves), early onset obesity (↑ IOTF at all waves), later onset obesity (↓ IOTF at first wave but ↑ at later	mother and father BMI, mother education, child birth weight	healthy weight trajectory: mixed associations between sleep duration and change in BMI later onset obesity: longitudinal associations NS early onset obesity: ↓ sleep duration at age 6–7 =↑ BMI at age 8–9; ↓ sleep duration at age 8–9 predicted ↑ BMI at age 10–11

First Author	Sample	Study Design	Sleep Measures	Obesity Measures	Covariates	Results
				waves)		
Mitchell[64]	1390 adolescents in 9th grade (Philadelphia suburban high school) at baseline	longitudinal, assessments every 6 months thru 12th grade	self-reported sleep duration (weighted average for school and weekend night)	self-reported BMI	study wave, gender, race, self-reported physical activity, maternal education, screen time	↓ sleep duration = ↑ increases in BMI from age 14 to 18
Lytle[77]	723 adolescents age 14.7 yrs at baseline	longitudinal, first cohort: baseline, 12 mos, 24 mos second cohort: baseline, 24 mos	self-reported sleep duration averaged across each assessment	BMI, % body fat measured by bioelectrical impedance	grade, race, parent education, school lunch, 24 hr energy intake, depression, pubertal status, physical activity, screen time/sedentary behavior	all longitudinal relationships NS in boys and girls
Araujo[70]	1171 Portuguese adolescents, 13 yrs old at Time 1	longitudinal, 4-year follow-up	self-reported weeknight sleep duration	BMI z-score and body fat % measured by bioelectrical impedance	parental education, Mediterranean Diet Quality Index	boys: ↓ sleep duration at age 13 = ↑ BMI, ↑ body fat % at age 17. girls: ↑ sleep duration at age 13 = ↑ BMI change by age 17 All NS after adjusting for baseline adiposity
O'Dea[65]	939 children in New South Wales, ages 7–12 at baseline	longitudinal, 4 annual assessments starting in 2007	self-reported weeknight sleep duration	BMI	gender, school SES (physical activity not related to BMI and therefore not included as covariate)	univariate analysis: upper tertile of sleep at year 1 = ↓ weight gain and ↓ increase in BMI between years 1 and 4 vs. lower tertile of sleep. multivariate analysis: consistently ↑ sleep = ↓ BMI
Storfer-Isser[71]	313 children ages 8–11 at baseline in the Cleveland Children's Sleep and Health Study	longitudinal, 3 assessments approx 4 yrs apart	parent-reported sleep duration at Times 1 and 2; self-reported sleep duration at Time 3 (weighted mean for weekday/weekend)	BMI z-score	age, race, birth weight, SES	boys: ↓ sleep duration at age 8–11 predicted ↑ BMI at ages 12–15 and 16–19; NS after adjusting for baseline BMI. girls: all NS
Tatone-Takuda[72]	>1000 Canadian children, approx 2.5 yrs at baseline	longitudinal, annual assessments across 4–5 yrs	parent-reported sleep duration, assessed annually, used to create 3 groups from 2.5 to 6 yrs: 1) short-persistent/increasing 2) 10-hr persistent, 3) 11-hr persistent	BMI at ages 6 and 7	child overweight or obese at 2.5 yrs, mother's immigrant status, mother overweight or obese, household income, vegetable/fruit consumption	boys: ↑ sleep group, ↓ BMI at age 6 (shortest 2 groups had ↑ BMI than longest group) and at age 7 (shortest group had ↑ BMI than longest group) girls: sleep, BMI NS
Carter[62]	244 children in New Zealand, age 3 at baseline	longitudinal, assessments every 6 mos from age 3 to age 7	actigraphy-assessed sleep duration	BMI z-score, various adiposity measures from bioelectrical impedence	age, sex, maternal education and income, maternal BMI, birth weight, smoking in pregnancy, ethnicity,	↑ sleep duration at ages 3–5 = 1) ↓ BMI at age 7, 2) ↓ increase in BMI from age 3 to 7, and 3) ↓ risk of overweight at age 7. each hr of sleep = 61% reduction in risk of overweight or obese at age

First Author	Sample	Study Design	Sleep Measures	Obesity Measures	Covariates	Results
					behavioral variables assessed at ages 3–5 (diet, activity, TV)	7
Diethelm[74]	481 German children in the DONALD study	longitudinal, multiple assessments in first 2 yrs after birth; annual assessments from ages 2–7	parent-reported sleep duration at age 1.5 and 2 yrs used to create 3 groups: consistently long, consistently short, inconsistent	BMI, fat mass index, fat free mass index from ages 2 to 7 yrs (mass indices based on skinfold thickness)	sex, birth year, birth weight, rapid weight gain	Inconsistent & consistently short sleepers at age 1.5–2 yrs = ↑ odds of excess body fat at age 7; consistently short sleepers showed progressively higher fat mass index levels until age 7 vs consistently long sleepers. BMI and fat free mass NS between sleep groups
Hiscock[76]	3857 infants (3–18 months) and 3844 preschoolers (4.3–5.6 yrs) at Wave 1 in the Longitudinal Study of Australian Children	longitudinal, Wave 1: 2004, Wave 2: 2006	parent-reported diary sleep duration	infants: weight-for-age, adjusted for birth length preschoolers: BMI z-score	sex, Wave 1 weight	NS; Sleep duration at Wave 1 did not predict BMI z-score at Wave 2 for either cohort
Seegers[67]	1916 children in Quebec Longitudinal Study of Kindergarten Children, 10 yrs at baseline	longitudinal, annual assessments across 3 yrs	parent-reported weekday sleep duration, assessed annually, used to calculate 3 trajectories: 1) short sleepers, 2) 10.5 hr sleepers, 3) 11 hr sleepers	BMI, based on annual parent reports of height and weight, used to create 3 groups: 1) normal BMI 2) overweight 3) obese	sex, immigrant status, family income, birth weight, parent education, pubertal status (ages 11–13), television and physical activity (age 13)	short sleepers and 10-hr sleepers had ↑ risk of overweight and obese at age 13 versus 11 hr sleepers ↑ sleep duration at age 10 predicted ↓ BMI at age 13
Silva[68]	304 children in the Tucson Children's Assessment of Sleep Apnea study, 6–12 yrs at baseline	longitudinal, baseline and 5-year follow-up	PSG-assessed TST at baseline and follow-up, used to create 3 groups: 1) ≤ 7.5 hr/night 2) > 7.5 - < 9 hr/night 3) ≥ 9 hr/night daytime sleepiness	BMI z-score at baseline and follow-up	ethnicity, sleep disordered breathing at baseline and follow-up, age	≤ 7.5 hr/night vs. ≥ 9 hr/night at baseline = ↑ odds of obesity at 5-yr follow-up. ≤ 7.5 hr/night vs. ≥ 9 hr/night at baseline = ↑ increase in BMI over 5 yrs daytime sleepiness NS

Table 3

Results by Sleep Characteristic

Risk Factor	Sleep Duration	Sleep Quality/Sleepiness	Sleep Continuity	Sleep Architecture
Metabolic Syndrome	1 positive	1 null/nonsignificant	--	--
	1 partial			
	3 null/nonsignificant			
Glucose/Insulin	8 positive (6 short sleep, 2 curvilinear)[1]	1 null/nonsignificant	2 positive	2 positive
	5 null/nonsignificant			
Total Cholesterol, LDL,	3 positive[2]	3 positive[3]	--	--
HDL	5 null/nonsignificant			
Triglycerides	2 opposite (↓ sleep, ↓ triglycerides)	--	--	--
	2 null/nonsignificant			
Blood Pressure	8 positive	1 positive	2 positive	1 positive
	2 opposite (↓sleep, ↓BP)	2 null/nonsignificant	2 null/nonsignificant	
	1 partial/mixed			
	11 null/nonsignificant			
Cardiovascular	2 positive	1 null/nonsignificant	1 null/nonsignificant	--
Reactivity	2 null/nonsignificant	1 opposite		
Heart Rate Variability	2 positive	2 opposite	3 positive	--
	2 null/nonsignificant		1 null/nonsignificant	
	1 opposite			

[1] 2 of the 8 positive findings in subgroups only

[2] 3 of the 3 positive findings in subgroups only

[3] 2 of the 3 positive findings in subgroups only