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Review Article

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, which 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 that 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.

1. Introduction

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

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, slow-wave sleep; PSG, polysomnography; CVD, cardiovascular disease; SES, socioeconomic status.

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of 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 aim 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, for example, mental arithmetic or giving a speech. Stress-induced cardiovascular responses are included because of their association with risk of incident hypertension and CVD [17].

Another aim of this 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 [19] reported that all seven longitudinal studies they reviewed observed a relationship between short sleep and increased weight and/or adiposity, whereas Guidolin and Gradisar [20] reported that neither of the two longitudinal studies they identified observed a relationship. In the current review, we summarize the longitudinal studies that were published after January 2011, and these were not included in the two previous reviews.

We chose to synthesize available evidence based on an enumerative or a 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 they 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, that is, duration, continuity, perceived quality, and daytime sleepiness [21]. We also included studies on sleep architecture when available, and we did not include sleep-disordered breathing (SDB). Another major dimension of sleep health, timing, was not reviewed because of few studies in children and adolescents. 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. This paper also highlights methodological issues, and it 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 (0–29 years) 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 earlier. We also included studies on sleep architecture when available. Sleep continuity refers to the consolidation of one's sleep throughout the night (eg, 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 the review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical samples (eg, psychiatric) or participants with sleep disorders. Fig. 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 the category of risk factors, with those reporting multiple risk factors listed first. Within risk factor category, studies are listed by the 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 (0–29 years) 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 the review were total or partial sleep deprivation experiments and studies that focused exclusively on clinical or sleep-disordered samples. Fig. 1b displays the number of records identified, screened, and excluded. Sixteen studies met criteria for inclusion. These studies are listed in Table 2 by the year of publication.

We first describe the overall pattern of results within risk factor category subsequently, and we 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 versus 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 we 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 in girls, we considered it positive in subgroups. For the summaries where we characterized findings by study characteristics, for example, comparing studies of children versus 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 two of the three, 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.