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Sleep duration and overweight among Australian children and adolescents

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Abstract
Aim: To examine the association between sleep and overweight and waist circumference (WC) in children and adolescents. *Methods:* Data were from a nationally representative sample of 6324 7–15-y-old males and females from the Australian Health and Fitness Survey. Associations between sleep duration and body mass index (BMI) and WC were examined by analysis of covariance, linear regression, and logistic regression. *Results:* In the total sample, there was a significant main effect across sleep-duration categories ($\leq$8 h, 8–9 h, 9–10 h, and $\geq$10 h) for BMI. Linear regression showed significant age, sex, age-sleep, and age-sex-sleep interactions in the total sample. There was an inverse graded relationship between sleep and BMI and WC in boys. In boys, there was a 1.6–1.8 times greater odds of overweight for those who reported 8–9 h or 9–10 h of sleep compared to those reporting $\geq$10 h of sleep. For boys reporting $\leq$8 h of sleep, there was about 3.1 times greater odds of overweight compared to those reporting $\geq$10 h of sleep. In contrast, no significant associations between sleep and overweight were found in girls.

Conclusion: Sleep duration was inversely related with overweight in young males but not females.

Key Words: *BMI, obesity, puberty, risk factors*

Introduction

The secular increase and current epidemic of pediatric obesity are well documented in several nations of the world [1]. The convincing evidence of the adverse medical [2], psychosocial [3], and economic burden [4] of pediatric obesity identifies the magnitude and significance of this health problem in contemporary society. Like other chronic diseases, pediatric obesity is a complex, multifactorial phenotype determined by genes, the environment, gene–gene interactions, and gene–environment interactions.

Although the underlying cause of obesity is simply characterized by an imbalance of energy expenditure and energy intake, resulting in positive energy balance, several peripheral factors are associated with these two main elements. The two risk factors that are usually focused upon are habitual physical activity and diet. The results examining the relationship between physical activity and diet with measures of adiposity and/or obesity in children and adolescents are inconsistent and produce only modest associations [5]. However, the strength of the associations between physical activity and diet variables and obesity may be attenuated due to measurement issues. Regardless, given the unexplained variance in the risk of obesity, it is also warranted to explore additional hypotheses, which may be independent or interact with physical activity and diet, to better understand the etiology of pediatric obesity.

Currently, there is interest in the possible association between sleep and obesity. Although the number of studies is limited, there is some epidemiological evidence suggesting a link between sleep duration and obesity in children [6–10], adolescents [11], and adults [12,13]. Pathophysiologically, sleep deprivation could influence the development of obesity through several possible pathways, including increased sympathetic activity, elevated cortisol and ghrelin levels, decreased leptin, and/or impaired glucose tolerance [14,15]. Furthermore, these perturbations may affect other common pathophysiologic mechanisms associated with atherosclerosis and insulin resistance.

The present study contributes to the fledgling knowledge base on the sleep–overweight relationship by introducing the following two unique elements: a) an examination of sex differences, and b) an examination of waist circumference (WC), in addition to BMI,

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as an index of overweight. Considering sex differences, particularly when studying overweight among adolescents, is critically important given the sex-associated variation in adiposity between boys and girls during this period. Likewise, given the limitations of BMI as an index of overweight, the co-examination of WC allows the examination of the sleep–overweight relationship through a second index and, importantly, one that is an independent risk factor for several chronic disease states.

Subjects and methods

Study design and subjects

Data for this analysis are taken from the 1985 Australian Health and Fitness Survey (AHFS). The study design and procedures have been described in detail elsewhere [16], but will be summarized here. The AHFS was a cross-sectional population study of approximately 8500 schoolchildren 7 to 15 y of age that assessed health-related fitness and self-reported health behaviors. The design of the survey was a two-stage probability sample. The first stage was the selection of schools, and the second stage consisted of a random sample of boys and girls of each year of age from the total school enrollment. The overall response rate was 67.5% for those participating in the field and technical measures portion of the study. The study was approved by local ethics committees, informed consent was obtained prior to participation in the study, and procedures were in accordance to those outlined by the Declaration of Helsinki. Measurements were recorded May through October 1985. In the present analysis, 3203 males and 3121 females are included based on complete data for sleep and anthropometry.

Sleep

A questionnaire was administered to the students by data collectors. The question pertaining to sleep asked, "What time did you go to bed last night and wake up this morning?" Sleep duration (hours) was calculated as the difference between wake-up time and bedtime. Sleep duration was categorized as follows: $\leq$8 h, 8–9 h, 9–10 h, and $\geq$10 h. These categories were chosen so that comparisons could be made to a similar study [7].

Anthropometry

BMI was calculated from measures of stature and body mass, which were measured by standard procedures [17]. BMI was used to classify overweight and obesity based on the age- and sex-specific international reference values corresponding to the adult reference values at 18 y of age (i.e., overweight >25 kg/m^2 and obesity >30 kg/m^2) [1]. WC was measured in the standing position at the level of the umbilicus to the nearest 0.1 cm using a constant-tension tape in duplicate. The average of the two measures was used in the analysis. Data collectors were trained in anthropometric techniques by the same project director, and a member of the steering committee made site visits to ensure that the standardized protocol was being followed during data collection. However, data on the within- or between-observer reliability are not available.

Statistical analysis

Initial exploratory statistics yielded the elimination of 27 outliers with sleep values $>\pm 3$ SD (subjects who reported sleeping less than 4 h ($n=5$) and those reporting greater than 14 h ($n=27$)). Furthermore, commonsense informs that 7–15-y-olds do not have sleep habits of <4 h or >14 h per night, particularly during the school year. Subjects were stratified into three age groups (7.5–10.9 y, 11.0–13.9 y, and 14.0–16.5 y), which essentially encompassed 3-y age spans. All analyses were conducted in the three age groups and for the total sample by sex. Differences in the descriptive characteristics of the sample across age groups by sex were examined by analysis of variance. The χ^2 test was used to examine the sex difference in percentage of overweight and obese subjects within age groups and for the total sample. Subsequent analyses were adjusted for age given the association between age and sleep, BMI, and WC in both boys ($r=-0.38$, 0.42, and 0.55, respectively) and girls ($r=-0.46$, 0.46, and 0.46, respectively). The relationships between sleep duration and BMI and WC were evaluated as continuous variables using partial correlations, controlling for age. Linear regression was also conducted with BMI or WC as outcome variables, and age, sex, sleep duration, and their interactions as predictors. Since the results showed significant age, sex, age-sleep, and age-sex-sleep interactions (and sex-sleep interaction for WC only), separate analyses were carried out by age and sex groups. Furthermore, since age was associated with sleep duration, we controlled for age even with the age groups to show the age-independent effects of sleep on BMI and WC. BMI and WC were compared across levels of sleep duration by analysis of covariance (ANCOVA), with age as the covariate. Least significant difference *post hoc* tests were conducted to examine differences between sleep categories. Logistic regression models were used to examine the relationship between sleep duration and overweight, adjusting for chronological age. Odds ratios (OR) and 95% confidence intervals were calculated using

binary logistic regression for each model. All statistical analyses were performed using SPSS version 11.0.

Results

Descriptive statistics of the sample are shown in Table I. As expected, the body-size variables increased across chronological age groups in males and females ($p < 0.0001$). Sleep duration significantly decreased across age groups ($p < 0.0001$). Although small in magnitude, there were several significant sex differences (see Table I). The prevalence of overweight and obesity were 9.0% and 1.8%, respectively, in males and 10.6% and 1.5%, respectively, in females. Approximately 35% of males and 40% of females reported sleeping 10 or more hours (Table II).

Age-adjusted means for BMI and WC within each sleep-duration category are shown in Table II. In the total sample of both boys and girls, there was a significant main effect across sleep-duration categories for BMI. In boys, there was an inverse graded relationship between sleep duration and BMI and WC with *post hoc* tests showing significant differences between all sleep-duration categories. In girls, the only significant *post hoc* differences were between those who reported 8–9 h and those who reported either 9–10 h or ≥10 h. The pattern between sleep duration and BMI and WC was consistent in all age groups in boys, with a 0.8–1.3 kg/m^2 and 2.1–3.3 cm, respectively, difference between the lowest and highest sleep categories. In girls, the pattern between sleep duration and BMI and WC was less clear.

Scatterplots fit with a lowess curve depicting the relationship between sleep duration and BMI and WC are shown in Figures 1 and 2, respectively. Regression coefficients (β) in Table III represent the association of sleep duration with BMI and WC. For boys, there was a significant association between sleep duration and BMI and WC in all age groups. Adjusted r^2 values ranged from 0.005 to 0.018. In girls, the coefficients were lower and not significant in the older girls for both BMI and WC, and also for WC in the 11–13-y-old group. Adjusted r^2 values ranged from 0.001 to 0.006.

Results of the logistic regression analysis are shown in Table IV. In the total sample, boys who reported less than 10 h of sleep were significantly at an increased odds of being classified as overweight. In boys, there was about 1.6–1.8 times greater odds of being classified as overweight/obese for those who reported 9–10 h or 8–9 h of sleep compared to those reporting >10 h of sleep. For boys reporting <8 h of sleep, there was about 3.1 times greater

Table I. Descriptive characteristics for Australian youth from the Australian Health and Fitness Survey, 1985.

	7.5–10.9 y		11.0–13.9 y		14.0–16.5 y		Total		
Variable	Males (n = 919)	Females (n = 953)	Males (n = 1360)	Females (n = 1350)	Males (n = 924)	Females (n = 818)	Males (n = 3203)	Females (n = 3121)	Total (n = 6324)
Age (y)	10.0 (0.6)	10.0 (0.6)	12.4 (0.9)	12.4 (0.9)	14.9 (0.6)	15.0 (0.6)	12.5 (2.0)	12.3 (2.0)	12.4 (2.0)
Height (cm)	138.4 (6.4)	138.0 (7.1)	151.5 (9.4) [a]	152.2 (8.3)	167.2 (9.0) [a]	161.2 (6.1)	152.3 (13.9) [a]	150.3 (11.6)	151.3 (12.8)
Weight (kg)	33.0 (5.8)	33.3 (6.6)	43.1 (9.9) [a]	44.0 (9.3)	56.6 (10.4) [a]	53.8 (8.4)	44.1 (12.8) [a]	43.3 (11.3)	43.7 (12.1)
BMI (kg/m^2)	17.1 (2.2) [a]	17.4 (2.3)	18.6 (2.8) [a]	18.8 (2.8)	20.1 (2.6) [a]	20.7 (2.8)	18.6 (2.8) [a]	18.9 (2.9)	18.7 (2.9)
WC (cm)	60.8 (6.0) [a]	60.2 (6.9)	66.8 (8.2) [a]	64.6 (7.4)	72.3 (7.0) [a]	69.1 (7.3)	66.7 (8.5) [a]	64.4 (7.9)	65.6 (8.3)
Sleep duration (h)	9.9 (1.2) [a]	10.2 (1.0)	9.4 (1.1)	9.5 (1.1)	8.8 (1.0)	8.9 (1.0)	9.4 (1.2) [a]	9.5 (1.2)	9.5 (1.2)
Overweight (%)	9.7	11.7	8.6	9.5	8.8	11.0	9.0	10.6	9.8
Obesity (%)	1.2	1.9	2.5	1.3	1.3	1.1	1.8	1.5	1.6

Values are mean (SD).
[a] $p < 0.05$ for sex difference within age group.

Table II. Mean body mass index (BMI) and waist circumference (WC) in relation to the number of hours spent sleeping among Australian youth aged 7–15 y.

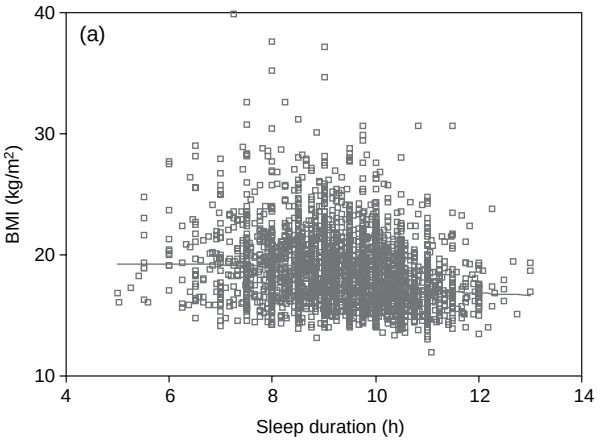
	≤8 h	8–9 h	9–10 h	≥10 h	F	*p* value
7.5–10.9 y						
BMI (kg/m²)						
Males	17.7 (0.3) [c]	17.6 (0.2) [e]	17.4 (0.1) [f]	16.9 (0.1)	6.3	0.0001
n	56	99	238	526		
Females	17.4 (0.4)	18.0 (0.3) [e]	17.5 (0.2)	17.2 (0.1)	2.3	0.08
n	27	71	218	637		
WC (cm)						
Males	63.2 (0.8) [b,c]	61.6 (0.6) [e]	61.1 (0.4)	60.3 (0.3)	5.4	0.001
n	56	99	238	526		
Females	59.6 (1.3)	62.3 (0.8) [e]	60.5 (0.5)	59.8 (0.3)	3.0	0.03
n	27	71	218	637		
11.0–13.9 y						
BMI (kg/m²)						
Males	19.5 (0.3) [a,b,c]	18.8 (0.2) [e]	18.7 (0.1) [f]	18.2 (0.1)	8.0	0.0001
n	124	264	506	466		
Females	19.3 (0.3) [c]	19.1 (0.2) [e]	18.8 (0.1)	18.6 (0.1)	2.9	0.03
n	108	240	505	497		
WC (cm)						
Males	68.8 (0.7) [c]	67.3 (0.5) [e]	67.3 (0.3) [f]	65.5 (0.4)	7.7	0.0001
n	124	264	506	466		
Females	64.8 (0.7)	65.1 (0.5)	64.6 (0.3)	64.4 (0.3)	0.6	0.66
n	108	240	505	497		
14.0–16.5 y						
BMI (kg/m²)						
Males	20.9 (0.2) [a,b,c]	20.2 (0.1) [e]	19.9 (0.1)	19.6 (0.2)	7.1	0.0001
n	145	333	313	133		
Females	20.4 (0.2)	21.0 (0.2) [d]	20.4 (0.2)	21.0 (0.3)	2.7	0.04
n	128	269	307	114		
WC (cm)						
Males	73.4 (0.6) [b,c]	72.6 (0.4)	72.0 (0.4)	71.3 (0.6)	2.7	0.05
n	145	333	313	133		
Females	68.3 (0.6) [c]	69.3 (0.4)	68.5 (0.4) [f]	70.7 (0.7)	3.2	0.02
n	128	269	307	114		
Total						
BMI (kg/m²)						
Males	19.5 (0.1) [a,b,c]	18.8 (0.1) [d,e]	18.7 (0.1) [f]	18.2 (0.1)	20.5	0.0001
n	325	694	1057	1125		
Females	19.0 (0.2)	19.2 (0.1) [d,e]	18.8 (0.1)	18.7 (0.1)	5.2	0.001
n	263	580	1030	1248		
WC (cm)						
Males	68.4 (0.4) [a,b,c]	67.3 (0.3) [e]	67.0 (0.2) [f]	65.6 (0.2)	14.8	0.0001
n	325	694	1057	1125		
Females	64.2 (0.4)	65.1 (0.3) [d,e]	64.3 (0.2)	64.3 (0.2)	1.8	0.138
n	263	580	1030	1248		

Values are age-adjusted means (SE).
[a] <8 h significantly different from 8–9 h.
[b] <8 h significantly different from 9–10 h.
[c] <8 h significantly different from >10 h.
[d] 8–9 h significantly different from 9–10 h.
[e] 8–9 h significantly different from >10 h.
[f] 9–10 h significantly different from >10 h.

odds of being classified as overweight/obese compared to those reporting ≥10 h of sleep. However, this result was not found in girls. The results shown in the total sample of boys were also apparent in the age groups. The highest risks occurred in the oldest age group. There were no significant trends in any age group of girls.

Discussion

This study extends our knowledge of the association between sleep duration and overweight by examining sex differences and including WC. Another major strength of this investigation was the large representative sample and age range studied representing mid-to-late childhood through adolescence. The results



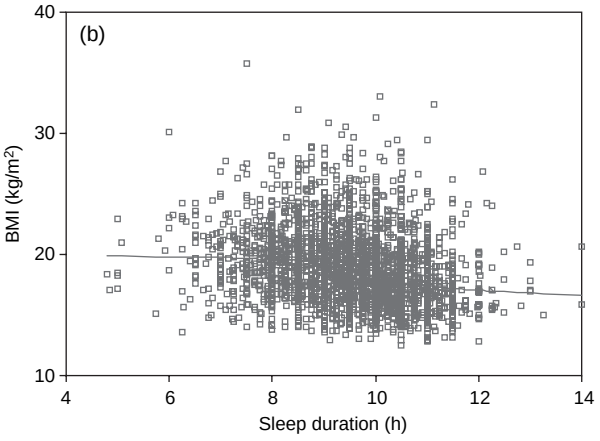
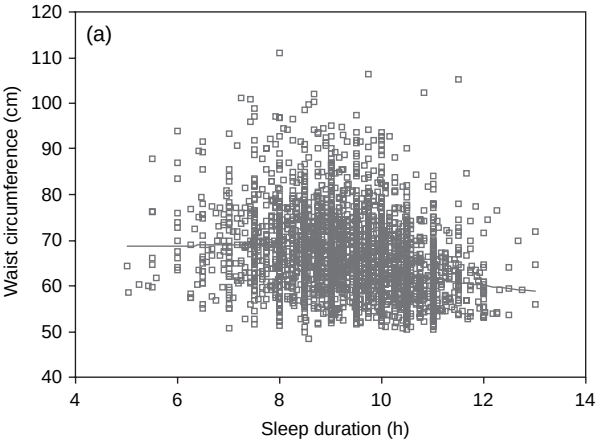


Figure 1. Scatterplot of sleep duration and body mass index (BMI) among 7–15-y-old Australian males (A) and females (B).



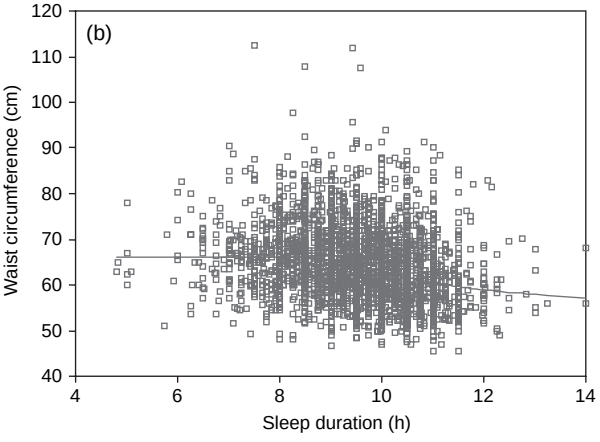


Figure 2. Scatterplot of sleep duration and waist circumference (WC) among 7–15-y-old Australian males (A) and females (B).

indicate significant associations between sleep duration and mean BMI and WC in both sexes; however, a stronger association and an inverse graded response between sleep duration and mean BMI and WC and risk of overweight was found in males but not females.

The main findings of this study support previous epidemiological investigations examining the relationship between sleep duration and obesity. The best evidence supporting the sleep–obesity hypothesis in children is from the Toyama Birth Cohort Study [7]. In this study, a dose-response relationship was shown between late bedtime and short sleeping hours and obesity in 8274 6–7-y-old Japanese children. The odds ratio for children sleeping less than 8 h was 2.87 compared to those sleeping 10 or more hours, which is similar to our findings in boys. Others have also found similar results in young children [8,9] and adolescents [11].

Results from the linear regression are less impressive than those from the logistic regression, and the adjusted r^2 is similar to a previous study of adolescents [11]. In a prospective study of 150 children from birth to 9.5 y of age, overweight children slept 30 min less than normal-weight children [10]. Recent findings from the U.S. NHANES I showed that adults who reported less than 7 h of sleep were at an increased risk of overweight in both cross-sectional and prospective analyses [12]. Based on the reported findings across the lifespan, there is consistent epidemiological evidence to support the hypothesis that sleep duration is inversely related to risk of overweight.

To our knowledge, this is the first study to examine the relationship between sleep duration and WC in children and adolescents. Results indicate significant differences in WC across sleep-duration categories in both sexes. In boys, there is an inverse graded relationship between sleep duration and WC. The magnitude of the difference between those reporting <8 h of sleep compared to those reporting >10 h was 2–3 cm. The clinical significance of such a difference is unknown in children and adolescents. Since WC is a simple, inexpensive measurement that is strongly correlated with trunk fat [19], and has immediate [20] and long-term effects [21] on atherosclerotic and metabolic risk factors, it is recommended that the assessment of WC, along with BMI, be considered in clinical and public health assessments of children and adolescents.

Table III. Linear regression models predicting BMI (kg/m^2) and WC (cm) from sleep duration (h) among Australian youth.

	7.5–10.9 y		11.0–13.9 y		14.0–16.5 y	
	Male ($n=919$)	Female ($n=953$)	Male ($n=1360$)	Female ($n=1350$)	Male ($n=924$)	Female ($n=818$)
BMI						
Sleep duration	−0.235 [a] (−0.357, −0.113)	−0.173 [a] (−0.319, −0.028)	−0.366 [a] (−0.505, −0.228,)	−0.189 [a] (−0.325, −0.053)	−0.349 [a] (−0.518, −0.179)	0.023 (−0.166, 0.213)
WC						
Sleep duration	−0.659 [a] (−0.330, −0.988)	−0.504 [a] (−0.082, −0.926)	−0.957 [a] (−1.350, −0.564)	−0.255 (−0.619, 0.10,)	−0.513 [a] (−0.959, −0.067)	0.403 (−0.094, 0.90)

Values are beta coefficient and 95% confidence intervals. Age- and sex-specific models are adjusted for age.
BMI: body mass index; WC: waist circumference.
[a] $p<0.05$.

Table IV. Associations between sleep duration and overweight among Australian youth.

	7.5–10.9 y		11.0–13.9 y		14.0–16.5 y		Total	
Sleep duration	Male ($n=919$)	Female ($n=953$)	Male ($n=1360$)	Female ($n=1350$)	Male ($n=924$)	Female ($n=818$)	Male ($n=3203$)	Female ($n=3121$)
≥10 h	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
9–10 h	1.60 (0.98, 2.61)	1.14 (0.71, 1.82)	1.62 (1.04, 2.50)	0.90 (0.59, 1.37)	1.94 (0.78, 4.83)	0.57 (0.31, 1.08)	1.61 (1.19,2.17)	0.91 (0.71, 1.69)
8–9 h	1.88 (0.98, 3.60)	1.80 (0.97, 3.35)	1.68 (1.01, 2.81)	1.15 (0.70, 1.89)	2.56 (1.04, 6.24)	0.85 (0.46, 1.56)	1.83 (1.30, 2.58)	1.31 (0.95, 1.80)
≤8 h	2.42 (1.14, 5.15)	1.41 (0.47, 4.25)	2.73 (1.53, 4.85)	1.24 (0.65, 2.35)	4.85 (1.92, 12.2)	0.61 (0.28, 1.30)	3.06 (2.11, 4.46)	1.09 (0.68, 1.20)

Values represent odds ratios (95% confidence intervals) adjusted for age.

As noted, there are clear sex differences in the results. One explanation of the null results in girls is that they may need to incur greater sleep deprivation to be affected, since from an evolutionary perspective they are more resilient to environmental stressors than males [22]. In an attempt to explore this hypothesis, we reanalyzed the data for girls by creating new categories of sleep duration (e.g., $\leq$6 h of sleep). Although we found a BMI difference of 1 unit (19.7 vs 18.7 kg/m^2) between those who slept $\leq$6 h compared to those who slept $\geq$10 h, the results are limited since only 14 girls slept less than 6 h. Future studies should consider the potential lower threshold for sleep debt in young females and the biological mechanisms that explain the sex difference.

The proposed mechanisms underlying the relationship between sleep, obesity, and atherosclerotic and metabolic risk factors appear to be complex and have yet to be fully elucidated. Several papers support the notion that sleep curtailment is related to metabolic and endocrine regulation, immune function, and sympathovagal stimulation—each a potential cause and consequence of the obesity-atherosclerosis-insulin resistance phenotypes. For example, in a recent study [14], glucose tolerance was lower and cortisol and sympathetic nervous system activity were higher in sleep-deprived (4 h per night for 6 d) men compared to those allowed 12 h per night. In addition, elevated evening cortisol levels may reflect an impairment in the negative feedback loop of the hypothalamic–pituitary–adrenal axis, which stimulates food intake [23] and further exacerbates the metabolic, vascular, and inflammatory state of obesity. Sleep deprivation has also been shown to lead to hyperphagia [24]. The molecular basis for the coupling of increased appetite and sleep wakening is supported by the identification of hypothalamic excitatory peptides (hypocretins or orexins) [25]. Furthermore, recent findings indicate that leptin levels decrease during sleep restriction (4 h per night for 6 d) compared with sleep extension (12 h per night for 6 d) [15], which may be associated with a concomitant increase in sympathovagal stimulation that is known to inhibit leptin release [26]. Thus, the putative pathways relating sleep debt to obesity and related atherosclerotic and metabolic co-morbidities occur as a result of the complex interactions of the psychoneuroendocrinology of energy and metabolic homeostasis.

The perturbations in sleep and the neuroendocrine cascade may also be related to psychosocial factors. Shorter sleep duration has been associated with lower self-esteem and higher depressive symptoms among 11–14-y-olds [27]. In turn, obese children and adolescents generally have a lower quality of life [3], which may be related to depressive symptoms. In a prospective cohort study of 5399 Swedish children followed up from birth through 15 y of age, an increment in relative weight of more than 15% was associated with psychosocial stress [28]. Several studies have shown the relationships between psychosocial indices, obesity and the metabolic syndrome in adults [29]. Therefore, the antecedents of sleep also need to be considered in the context of the sleep-obesity and atherosclerotic and metabolic co-morbidities hypothesis.

The co-occurrence of sleep deprivation, physical inactivity, television viewing (TV), and obesity should also be considered. The association between sleep and TV has been shown among adolescents and young adults. Adolescents who watched 3 or more hours of TV per day were at increased risk for sleep problems during young adulthood [30]. Another study of adolescents found that sleep was related to physical activity, with a 3% lower physical activity for every hour increase in sleep disturbance [11]. Decreased sleep could also be caused by increased caffeine and/or high simple sugar intake, common among contemporary youth. In turn, this pattern of dietary intake may be related to TV advertisements. It has also been shown that both physical activity and TV are associated with the risk of overweight in U.S. adolescents [31,32]. When these findings are considered together, it appears the inter-relationships between sleep, physical activity, and TV are all important considerations in preventing and managing pediatric obesity.

Although this is a cross-sectional study, and therefore cannot indicate a causal relationship between sleep and obesity, there appears to be a growing body of evidence to support the notion that sleep curtailment is in the causal pathway of obesity and related atherosclerotic and metabolic co-morbidities. Among the causal criteria, there is strength of association, biological plausibility, experimental evidence [14,15], consistency in results, a dose-response relationship [7,12], and time-order [10,12]. Further consideration of sleep deprivation as a risk factor for obesity in children and adults is warranted.

Conclusions

The results of this study indicate a significant association between sleep duration and BMI-based overweight and WC in boys, but not girls. Since physical activity and diet were not included as covariates, it is difficult to establish a direct link between sleep duration and overweight; therefore, it is important to consider that sleep duration may be in the causal chain to overweight, possibly by affecting diet and physical activity. Alternatively, sleep deprivation has also been shown to cause neurohumoral changes in key biomarkers related to weight gain. Regardless of the causal pathway, sleep should be considered in the

establishment of effective prevention, treatment, and intervention programs for pediatric obesity and related metabolic disorders.

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