Is Sleep Duration Associated With Childhood Obesity? A Systematic Review and Meta-analysis

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Obesity is a major public health epidemic worldwide in children and adults (1–6). The prevalence and severity of childhood obesity is dramatically increasing with a corresponding increase in the prevalence of obesity-related morbidities particularly those involving obstructive sleep apnea and metabolic and cardiovascular sequelae (7). Prevention of childhood obesity is an urgent issue for public health, in particular, in many industrialized countries and some transition societies. Nutrition and physical activity (PA) have been the major research focus on obesity prevention (8–11). However, most published findings of such interventions suggested little success in preventing childhood obesity (6,12), although some that focused on dietary or/and PA approaches have showed some desirable impact on BMI status (13). Other risk factors, such as sleep, may be related to obesity, although sleep behavior has received much less attention than dietary intake and PA (14,15).

Sleep, like PA and diet, plays an important role in the growth, maturation, and health of children and adolescents by allowing for the diurnal rhythm of hormones related to growth, maturation, and energy homeostasis (16). A number of biological mechanisms have been proposed to link sleep duration and obesity (17). For example, one theory posits that children with short sleep have low caloric intake and expenditure, given that sleep deprivation often leads to changes in the structure of sleep stage and results in fatigue, daytime sleepiness, somatic and cognitive problems, and low activity levels (18,19). Previous studies indicate that sleep deprivation results in changes in levels of several hormones including leptin, ghrelin, insulin, cortisol, and growth hormone (20–22). These hormonal changes may contribute to energy imbalance and then lead to overweight or obesity.

Recently, there is increasing epidemiological evidence suggesting a link between sleep duration and obesity in children (18,23–26), adolescents (27), and adults (28–30). In adults, some studies indicate a negative association between sleep duration and obesity/BMI in men and a U-shaped relation between sleep duration and BMI in women (29). Several epidemiological studies have examined the association between short sleep duration and obesity in children and adolescents (18,23–27,31–37), but the reported findings are inconsistent (18,32,35,37). A recent review summarized 13 studies that examined the association between short sleep duration and obesity among children and adolescents, and recommended more sleep to prevent obesity (17). However, the review suffered from several limitations including misclassification of the design of some studies and included a study with adults as the primary age group (28). Several related previous studies were not included. Furthermore, new studies have been reported since the publication of the review. A major gap in the related literature is that to our knowledge, no meta-analyses have been conducted to examine the overall association quantitatively and to test the possible differences by population groups such as age and gender.

This study aimed to assess epidemiologic evidence systematically on the relation between sleep duration and childhood obesity. We first performed a systematic review of all related studies and then conducted a meta-analysis based on cohort studies and cross-sectional studies in the general pediatric population.

Methods and procedures

Systematic search. We conducted a comprehensive literature search of the PubMed database using related MeSH keywords for papers published between January 1980 and May 2007. We first conducted the search using "sleep AND obesity AND child." The resulting number was 51, 10 of which were included in the systematic review. Further search with MeSH keywords: "sleep AND overweight AND child" yielded 42 papers from which few additional studies were selected. We also searched with "sleep AND obesity" and found 54 references, and found 46 references using "sleep AND overweight." Furthermore, we tried searching "paediatric" or "pediatric" as an MeSH keyword, but no matching references were found. Then, we checked articles using the "related article" option of PubMed. In addition, studies identified in the course of reading

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or brought to our attention by colleagues and experts consulted were checked. These yielded a total of 17 studies that met our inclusion criteria and were included in our systematic review; of which 11 (2 cohort studies and 9 cross-sectional studies) reported odds ratio (OR), relative risk or hazard ratio, and were included in our meta-analysis.

Study eligibility criteria and data extraction. Studies were included if they met the following criteria: (i) Observational studies, including cohort, randomized trial, cross-sectional, and case–control studies. Only those with a measure of OR (or relative risk or hazard ratio) and the 95% confidence interval (95% CI) were included in our meta-analysis; (ii) Enrolled children aged from 0 to 18 years; (iii) Included >100 subjects; and (iv) Had a measure of overweight/obesity using BMI. We chose to restrict our meta-analysis to those studies with sample size >100 subjects considering the studies' statistical power in detecting an association and the representativeness of their findings. Note that only one study with 60 children aged 10–16 years from a hospital-based pediatric weight-management clinic and 22 healthy controls was excluded, which found that the overweight children had shorter sleep time than their non-overweight counterparts (38). All the other studies were excluded because they did not meet our other inclusion criteria. Multiple studies based on the same cohort with different age group were also included. In our present review, 2 cross-sectional studies (age group: 3–4 and 6–7 years) based on the Toyama Cohort Study were included (18,24).

Using a standardized data extraction form, we extracted and tabulated related data. Information extracted included first author's name, year of publication, country of data collection, study design, and sample characteristics. A dataset based on this data extraction was created using a spreadsheet program (Microsoft Excel, Microsoft, 2003).

Classification of outcome and exposure. There are different standards to define childhood overweight and obesity in different population groups. Most of the studies followed the International Obesity Task Force's recommended age- and sex-specific BMI cutoff points (39) which were linked to adult cutoff points: overweight and obesity (equal to adult criterion, BMI ≥ 25 kg/m²) (18,24,31,33,35,37). Studies conducted in the United States used the 2000 Centers for Disease Control and Prevention Growth Chart 85th and 95th percentile to define overweight or obesity, respectively (27,32,40). All of included studies reported measured BMI.

As there are age-related shifts in children's sleep needs and the circadian timing of sleep, particularly as children grow older (40–42), in our meta-analysis we used different recommended sleep duration for different age groups based on previous research in which ideal sleep time or sleep requirement time was provided (**Table 1**). The standardized sleep duration reference was defined as ≥11 h for children aged <5 years (24,34). For children between 5 and 10 years of age, the sleep duration reference was treated as ≥10 h (18,43,44). As previous studies indicate that sleep time in kindergarten children is similar to that in elementary schoolchildren (43), thus we used one reference value for the 5- to 10-year-old. The corresponding figure was ≥9 h for children aged ≥10 years (40,42,45,46). In our meta-analysis, we considered children's sleep duration as a multi-level exposure variable classified as less than the recommended sleep time contrasted with the recommended level of sleep duration (e.g., <9 h for children aged ≥10 years).

Data analysis. In our meta-analysis, OR/relative risk/hazard ratio was used as the primary measure of the association between sleep duration and overweight/obesity. First, on the basis of each study's own reference group of sleep duration, we pooled the ORs for short sleep duration group. For example, a study might report two ORs for sleep duration of <9 h and 9–10 h when ≥10 h of sleep duration is treated as a reference group. Using the detailed information from that study, we pooled the ORs of <9 h and 9–10 h, and used the pooled OR to determine the risk for <10 h of sleep. Next, we created a database by assigning ORs extracted from each study to their corresponding (or matched) sleep duration categories according to the ranges or averages of the reported sleep duration categories. If one study used shorter sleep duration as a reference instead of the standardized sleep duration, we chose the suitable sleep duration group as reference and recalculated the OR and 95% CI.

We applied fixed- and random-effects models to estimate the pooled ORs and 95% CI of overweight/obesity for each sleep duration category compared with reference group. The pooled OR was obtained by averaging the natural logarithm of ORs, weighted by the inverse of their respective variances (47). DerSimonian and Laird's method was used in the random-effects model to further incorporate between-study variability (48). If heterogeneity was significant ($P < 0.05$), we used the random-effects models; otherwise, results from fixed-effects models were reported. In stratified meta-analyses, we examined potential sources of heterogeneity, including gender, study design, age group (<10 years vs. ≥10 years), BMI status (combined overweight/obesity vs. obesity), and sleep duration measure. We further conducted trend test between the three sleep duration categories. We used a non-parametric graphical method to assess linear trend in the $\text{Log}_e(\text{OR})$ point estimates, followed by a bivariate linear regression analysis in which sleep category was entered as an ordinal variable predicting $\text{Log}_e(\text{OR})$. The P value of the

Table 1 Classification of sleep duration category and recommended sleep duration

Age group (years)	Recommended sleep duration hours (ref.)	Sleep duration category (hours)[a]		
		Shortest	Much shorter	Shorter
<5	≥11 (24,34)	<9	9–10	10–11
5–10	≥10 (18,43,44)	<8	8–9	9–10
≥10	≥9 (40,42,45,46)	<7	7–8	8–9

[a]These cut points were used when we classified/converted reported odds ratios (ORs) in individual studies for our meta-analysis.

Table 2 Description and main findings of studies on the association between sleep duration and obesity among children, sorted by study design (cohort, cross-sectional, and case–control study), publication year, and country of data collection[a]

Source	Country	Study design	Follow-up (y)	Subject, age (y)	Men (%)	Classification of obesity and overweight	Sleep reference (hours/d)	Main findings: linear or logistic regression β (s.e.)/OR/RR/HR (95% CI)	Notes
Agras *et al.* (26)	USA	Cohort study	9 y	150, 0 y	49.3	Overweight and obesity: BMI > 85th percentile (59)		Sleep duration treated as continuous variable: Linear regression: −0.21*	Structural equation model was used
Reilly *et al.* (34)[b]	UK	Cohort study	3–5 y	7,758, 3 y	50.7	UK reference: Obesity: BMI ≥ 95th percentile (39)	>12 h	Boys and girls: <10.5 h: 1.45 (1.10–1.89) 10.5–10.9 h: 1.35 (1.02–1.79) 11–11.9 h: 1.04 (0.76–1.42)	Confounders were adjusted for: gender, maternal education, energy intake at age 3 y
Snell *et al.* (52)[b,c]	USA	Cohort Study	5 y	2,281, 3–12 y	50.3	IOTF: Overweight and obesity (60)	9–10 h	All children <8 h: −0.009 (0.073) 8–9 h: 0.014 (0.054) 10–11 h: −0.070 (0.044) ≥11 h: −0.171 (0.063)** Older children (8–13 y) <8 h: −0.093 (0.089) 8–9 h: 0.036 (0.072) 10–11 h: −0.084 (0.066) ≥11 h: −0.108 (0.115) Younger children (3–8 y) <8 h: 0.084 (0.126) 8–9 h: 0.041 (0.092) 10–11 h: −0.062 (0.060) ≥11 h: −0.158 (0.072)**	Confounders were adjusted for: gender, race, family income, parental education, age at times 1 and 2
von Kries *et al.* (25)[b]	Germany	Cross-sectional Study		6,862, 5–6 y	NA	German reference Overweight and obesity: BMI ≥ 90th percentile Obesity: BMI ≥ 97th percentile (61)	≤10 h	Overweight and obesity: 10.5–11 h: 0.77 (0.59–0.99) ≥11 h: 0.54 (0.40–0.73) Obesity 10.5–11 h: 0.52 (0.34–0.78) ≥11 h: 0.46 (0.28–0.75)	Confounders were adjusted for: parental education, parental BMI, birth weight, weight gain, screen time, snack taking
Sekine *et al.* (24)[b]	Japan	Cross-sectional study		8,941, 3–5 y	51.3	IOTF: Overweight and obesity (60)	≥11 h	Boys and girls: <9 h: 1.57 (0.90–2.75) 9–10 h: 1.34 (1.05–1.72) 10–11 h: 1.20 (0.97–1.49)	Confounders were adjusted for: gender, age, parental obesity, outdoor playing time
Sekine *et al.* (18)[b]	Japan	Cross-sectional study		8,274, 6–7 y	50.7	IOTF: Overweight and obesity (60)	≥10 h	Boys and girls: <8 h: 2.87 (1.61–5.05) 8–9 h: 1.89 (1.34–2.73) 9–10 h: 1.49 (1.08–2.14) Boys: <8 h: 5.49 (2.20–16.7) 8–9 h: 3.45 (1.86–6.37) 9–10 h: 2.28 (1.25–4.15) Girls: <8 h: 2.13 (1.01–4.48) 8–9 h: 1.28 (0.88–2.15) 9–10 h: 1.23 (0.81–1.87)	(i) Confounders were adjusted for: age, gender, parental obesity, physical activity, screen time, frequency of taking breakfast, frequency of taking snack; (ii) Risks were greater for boys than for girls
Gupta *et al.* (27)	USA	Cross-sectional study		383, 11–16 y	46.2	CDC ref: Overweight and obesity: BMI > 85th percentile (59)		Sleep duration treated as continuous variable: 0.20 (0.11–0.34)	(i) Confounders were adjusted for: gender, age, gender*age, race, sexual maturity; (ii) Sleep was assessed by wrist actigraphy
Gibson *et al.* (31)	UK	Cross-sectional study		1,294, 7–18 y	50.6	IOTF: Overweight and obesity (60)		Linear trend test: Boys: $P = 0.002$ Girls: $P > 0.05$	Quintiles of age-adjusted BMI were negatively related to sleep duration in boys, but not in girls

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Table 2 (continued)

Source	Country	Study design	Follow-up (y)	Subject, age (y)	Men (%)	Classification of obesity and overweight	Sleep reference (hours/d)	Main findings: linear or logistic regression β (s.e.)/ OR/RR/HR (95% CI)	Notes
Knutson (32)	USA	Cross-sectional study		4,486, 15–18 y	48.3	CDC ref: Obesity: BMI ≥ 95th percentile (59)		Linear regression: Boys: −0.08*; girls: −0.02 Logistic regression: Sleep duration treated as a continuous variable: Boys: 0.90 (0.82–1.00) Girls: 1.06 (0.96–1.17)	(i) Confounders were adjusted for: age, race, exercise, screen time, parental education; (ii) Obesity was negatively related to sleep duration in boys, but no in girls
Padez *et al.* (33)[b]	Portugal	Cross-sectional study		4,511, 7–9 y	49.6	IOTF: Overweight and obesity (60)	<9 h	Boys and girls Overweight: 9–10 h: 0.46 (0.40–0.51) ≥11 h: 0.44 (0.38–0.49) Obesity: 9–10 h: 0.44 (0.40–0.47) ≥11 h: 0.39 (0.35–0.42)	Confounders were adjusted for: gender and age
Chaput *et al.* (35)[b]	Canada	Cross-sectional study		422, 5–10 y	50.0	IOTF: Overweight and obesity (60)	≥12 h	Boys and girls 8–10 h: 3.45 (2.61–4.67) 10.5–11.5 h: 1.42 (1.09–1.98) Boys: 8–10 h: 5.65 (4.23–6.75) 10.5–11.5 h: 1.28 (0.98–1.65) Girls: 8–10 h: 3.15 (2.06–4.43) 10.5–11.5 h: 1.69 (1.22–2.78) Pearson correlation with sleep duration in boys: BMI: −0.12 Waist circumference: −0.24	(i) Confounders were adjusted for: age, gender, parental obesity, parental education, family income, single parenthood, screen time, exercise and breastfeeding time; (ii) Body weight and waist circumference were negatively related to sleep duration in boys, but not in girls
Chen *et al.* (36)[b]	Taiwan, China	Cross-sectional study		656, 13–18 y	53.2	Taiwan reference: Overweight: >85th percentile (62)	6–8 h	<6 h: 1.74 (1.3–2.4)	Confounders were adjusted for: gender, grade, number of visits to doctors
Eisenmann *et al.* (37)[b]	Australia	Cross-sectional study		6,324, 7–15 y	50.6	IOTF: Overweight and obesity (60)	≥10 h	7–16 y old Boys: ≤8 h: 3.06 (2.11–4.46) 8–9 h: 1.83 (1.30–2.58) 9–10 h: 1.61 (1.19–2.17) Girls: ≤8 h: 1.09 (0.68–1.20) 8–9 h: 1.31 (0.95–1.80) 9–10 h: 0.91 (0.71–1.69)	(i) Age was adjusted for. (ii) Sleep duration was negatively related to overweight/obesity in boys, but not in girls; (iii) Similar results were found in 7–10, 11–13, and 14–16 y age groups
Knutson and Lauderdale (40)[b]	USA	Cross-sectional study		1,546, 10–19 y	49.6	CDC ref: Obesity: BMI ≥ 95th percentile (59)	Self-reported: >9 h Time-diary: >10 h Both: >9 h	≤7 h: 0.88 (0.45–1.69) 7–8 h: 1.85 (1.01–3.38) 8–9 h: 1.93 (1.10–3.37) <8.4 h: 1.02 (0.57–1.84) 8.4–9.3 h: 1.56 (0.92–2.64) 9.3–10 h: 1.63 (0.96–2.78) ≤7 h: 0.87 (0.45–1.69) 7–8 h: 1.82 (0.99–3.32) 8–9 h: 1.83 (1.05–3.20)	(i) Confounders were adjusted for: gender, race, age, family SES (income, education), screen time, exercise; (ii) Sleep duration was assessed by self-reported questionnaire and time-diary
Seicean *et al.* (63)[b]	USA	Cross-sectional study		529, 14–18 y	49.1	CDC ref: Overweight and obesity: BMI ≥ 85th percentile	>8 h	<5 h: 7.65 (1.87–31.3) 5–6 h: 2.80 (1.00–7.79) 6–7 h: 2.55 (1.02–6.38) 7–8 h: 1.38 (0.54–3.53)	(i) Confounders were adjusted for: gender, age, irregular eating, health status, and caffeine intake; (ii) BMI was based on self-reported weight and height

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Table 2 (continued)

Source	Country	Study design	Follow-up (y)	Subject, age (y)	Men (%)	Classification of obesity and overweight	Sleep reference (hours/d)	Main findings: linear or logistic regression β (s.e.)/ OR/RR/HR (95% CI)	Notes
Locard *et al.* (23)	France	Case–control study		Cases: 327 controls: 704, 5 y	51.0	French reference: Obesity: Weight for height Z score	≥12 ≥11	<10 h: 4.9 (1.9–12.7) 10–11 h: 2.8 (1.2–6.3) 11–12 h: 2.0 (0.9–4.4) <11 h: 1.4 (1.1–1.9)	Confounders were adjusted for: parental overweight for the <11 h vs. ≥11 h OR
Hui *et al.* (53)	Hong Kong, China	Case–control study		343, 6–7 y	49.0	Hong Kong reference: Overweight: BMI ≥ 92nd percentile (64)	<9 h	9–11 h: 0.54 (0.30–0.97) ≥11 h: 0.31 (0.11–0.87)	Confounders were adjusted for: paternal and maternal obesity

CI, confidence intervals; HR, hazard ratio; NA, not available; OR, odds ratio; RR, relative risk.
[a]The International Obesity Task Force (IOTF) BMI reference was based on data collected in six populations, and the age–sex-specific BMI cut points correspond to those used in adults for the classification of overweight and obesity, BMI = 25 and 30, respectively (53). Centers for Disease Control and Prevention (CDC) ref: The US 2000 CDC Growth Charts, which define "at risk of overweight" (termed "overweight" in this study) using the 85th percentile, and "overweight" (termed "obesity") using the 95th percentile (52). [b]Studies were used in our meta-analysis. [c]OR and 95% CI were calculated based on β and s.e. of logistic regression models. *P < 0.05, **P < 0.01.

regression coefficient was presented and a significant P value reflected a linear trend.

Furthermore, treating sleep duration as a continuous variable, we estimated the OR and 95% CI associated with a 1 h increase in sleep using weighted meta-regression. The regression was weighted by the inverse variance of the Log_e(OR) for each category, and we used the average of each sleep duration category. In addition, we assessed publication bias using the Begg's funnel plots. The ORs were plotted on a logarithmic scale against their corresponding s.e. for each study (49,50). We also assessed publication bias by two formal tests, the Begg-adjusted rank correlation test, and the Egger's regression asymmetry test. Our meta-analysis was conducted using STATA 9.0 (StataCorp, College Station, TX) (51). Statistical significance was set at type I error of $P \le 0.05$.

Results

Systematic review. Table 2 summarizes the main characteristics and findings of 17 eligible observational studies, including 3 cohort studies, 12 cross-sectional studies, and 2 case–control studies. Of these studies, 6 were conducted in the United States, 5 were from Europe (France, Germany, Portugal, UK), 4 from Asia (Japan, China: Hong Kong, Taiwan), and 2 from other (Australia and Canada). The number of subjects included in these studies varied considerably, ranging from 150 to 8,941, though most were over 1,000. Despite the common use of self-reported questionnaire to assess sleep duration, one of our selected studies used wrist actigraphy (27), while another used both self-reported questionnaire and time-diary as measures of sleep duration (40).

Several important studies presented in Table 2 are worth highlighting as they provide strong evidence of the association between short sleep duration and childhood obesity. A prospective cohort study of 7,758 children aged 3 years at baseline conducted in the UK found that short sleep duration (<10.5 h) at age three was associated with obesity at age seven (OR = 1.45; 95% CI: 1.10, 1.89) (34). Early life shortness of sleep seems to be associated with an increased risk of childhood obesity. Sekine *et al.* found a clear dose–response relationship between sleep and obesity in Japanese children aged 6–7 years (18,24). Compared with sleep durations of 10 h or more, the adjusted OR was 1.49 (95% CI: 1.08, 2.14) for 9–10 h of sleep, 1.89 (95% CI: 1.34, 2.73) for 8–9 h, and 2.87 (95% CI: 1.61, 5.05) for <8 h of sleep, after adjustment for age, gender, PA, screen time, parental obesity, and other lifestyle factors (18). This association was stronger in boys than in girls. Gender difference seems to exist in the association between sleep duration and childhood obesity. Data on 6,324 Australian children aged 7–15 years found an inverse relationship between sleep and overweight in boys, but not in girls (37). For boys reporting ≤8 h of sleep, they had 3.1 times greater odds of overweight than those reporting ≥10 h of sleep (37). Another cohort study based on a

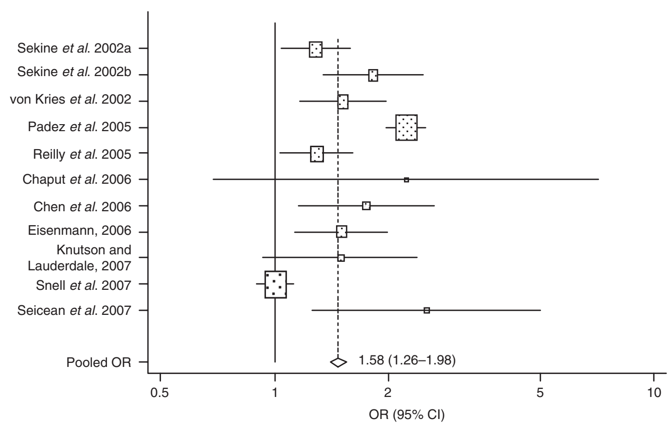

Figure 1 The association between short sleep duration and risk for overweight/obesity: Pooled odds ratio (OR) and 95% confidence interval (95% CI). Shorter vs. longer sleep duration (based on individual study's criteria); Test for heterogeneity: Q = 174.9, P < 0.001; pooled OR and 95% CI were based on random-effects model.

nationally representative sample of 2,281 children aged 3–12 years at baseline in the United States found that young children aged 3–8 years with shorter sleep duration were more likely to be overweight after 5 years of follow-up (52). However, no similar results were found in children aged 8–13 years (52).

In general, studies of children aged <10 years found an inverse association between sleep duration and overweight/obesity on the basis of parental report of sleep duration (18,23–26,33–35,40,53). Findings from studies in adolescents are somewhat inconsistent. In fact, although most studies of adolescents gave significant results in the expected direction (15,27,31,32,36), some reported no association among girls (32,37).

Meta-analysis. **Figure 1** shows the ORs and 95% CIs from each individual study and the pooled random-effects estimate of OR and 95% CI for the association between each study's own short sleep duration and overweight/obesity. The pooled OR and 95% CI, using each study's specific criterion for short sleep duration, was 1.58 (95% CI: 1.26, 1.98) for total general pediatric population.

Our analysis assessing the dose–response relationship between sleep duration and obesity was reported in **Figure 2** and **Table 3**. Overall, our findings did not show a clear dose–response relationship, though it existed in some groups. Compared with children having recommended sleep duration, those with much shorter sleep duration had significantly higher risk of overweight/obesity (OR = 1.60; 95% CI: 1.22, 2.10). The shortest sleep duration had much higher risk of overweight/obesity (OR = 1.92; 95% CI: 1.15, 3.20). A marginally significant dose–response relationship was found in the association between sleep duration categories and overweight/obesity (trend test: $P = 0.087$), and among young children aged <10 years (trend test: $P = 0.094$). We also tested the differences by gender, BMI status, and age group (**Table 3**). The association between shorter vs. longer sleep duration (based on individual studies' own criteria) and overweight/obesity was stronger in boys than in girls (OR = 2.50 (1.88, 3.34) vs. 1.24 (1.07, 1.45), $P < 0.001$), but we found no significant differences by age group or BMI status measurement (i.e., combined overweight/obesity vs. obesity). Finally, we treated sleep duration as a continuous variable through meta-regression analysis. For each 1 h unit increase in sleep duration, the pooled OR for overweight/obesity was 0.91 (0.84, 1.00), $P = 0.044$.

Our analysis shows no publication bias. The Begg's funnel plot (**Figure 3**) revealed slightly more data points below the horizontal line, but neither Begg's adjusted rank correlation test nor Egger's

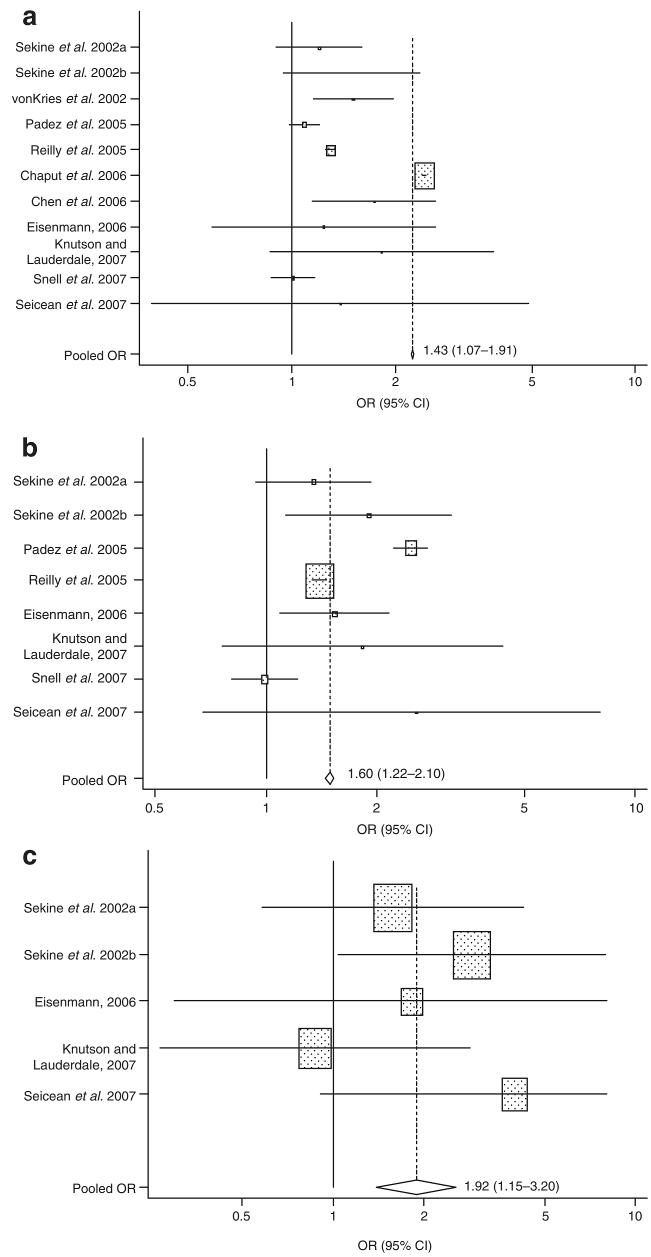

Figure 2 Test the dose–response association between different short sleep duration categories and overweight/obesity: Pooled odds ratio (OR) and 95% confidence interval (95% CI). (**a**) Shorter vs. suggested sleep duration. Test for heterogeneity: $Q = 3{,}043.8$, $P < 0.001$; pooled OR and 95% CI were based on random-effects model. (**b**) Much shorter vs. suggested sleep duration. Test for heterogeneity: $Q = 231.4$, $P < 0.001$; pooled OR and 95% CI were based on random-effects model. (**c**) Shortest vs. suggested sleep duration. Test for heterogeneity: $Q = 10.8$, $P = 0.028$; pooled OR and 95% CI were based on random-effects model. See **Table 1** for the age-specific cut points of the sleep duration categories; Pooled OR and 95% CI were based on random-effects model when tests for heterogeneity were significant ($P < 0.05$), and based on fixed-effects models when the tests were insignificant ($P \geq 0.05$).

Table 3 Pooled odds ratios (ORs) and 95% confidence intervals (95% CIs) of sleep duration and overweight/obesity, stratified by gender, BMI status, and age group

	Number of studies	Sleep duration[a] Shorter vs. longer		Sleep duration[b] (reference: longest) Shortest		Much shorter		Shorter		Trend test
		OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	*P* value
All studies	11	1.58	1.26, 1.98	1.92	1.15, 3.20	1.60	1.22, 2.10	1.43	1.07, 1.91	0.087
Gender difference										
Boys	3	2.50[c]	1.91, 3.26	3.28[c]	2.31, 4.46	2.13[c]	1.58, 2.87	2.57	1.19, 5.57	0.619
Girls	3	1.24	1.07, 1.45	1.19	0.91, 1.55	1.30	1.00, 1.69	1.33	0.83, 2.12	0.209
BMI status										
Overweight and obesity	9	1.64	1.25, 2.15	2.30[d]	1.64, 3.23	1.64	1.08, 2.50	1.41	0.97, 2.05	0.138
Obesity	4	1.76	1.20, 2.57	0.87	0.45, 1.69	1.87	1.12, 3.12	1.31	1.15, 1.50	0.641
Age group										
<10y	8	1.51	1.20, 1.92	2.09	1.49, 2.92	1.61	1.18, 2.19	1.38	1.00, 1.90	0.094
≥10y	4	1.62	1.39, 1.88	1.77	0.74, 4.25	1.47	1.14, 1.89	1.57	1.25, 1.97	0.561

[a]Shorter vs. longer sleep duration (based on individual study's own criteria). [b]Standardized sleep duration categories, see Table 1. [c]Gender difference, $P < 0.05$. [d]BMI status difference, $P < 0.05$.

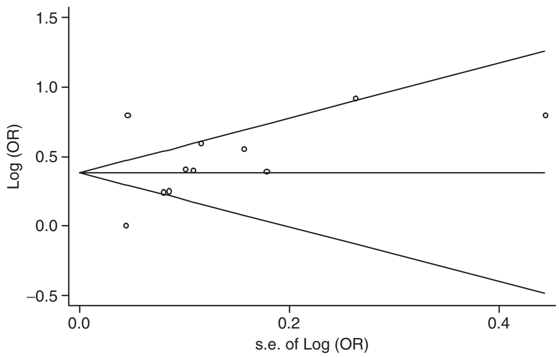

Figure 3 Assessment of publication bias: funnel plot regarding odds ratio (OR) on a log scale on its s.e. Horizontal lines indicate the random-effects pooled estimates of ORs and 95% CIs; sloping lines represent the expected 95% CIs for a given s.e., assuming heterogeneity between studies. Neither the Begg's adjusted rank correlation tests nor the Egger's regression asymmetry test is significant ($P = 0.139$, $P = 0.903$, respectively).

regression asymmetry test was significant ($P = 0.139$ and $P = 0.903$, respectively).

Discussion

Sleep deprivation is becoming increasingly prevalent in both adults and children (54). The decline in sleep duration has paralleled a dramatic increase in the prevalence of obesity (54). Our meta-analysis, based on previous epidemiologic studies in the general pediatric populations in different countries, provides strong evidence to help quantify the relationship between sleep duration and overweight/obesity in children and adolescents. First, we found that children with shorter sleep duration had a 58% (pooled OR = 1.58 (1.26, 1.98)) higher risk for overweight or obesity, and children with shortest sleep duration had an even higher risk (92%) when compared with children having longer sleep duration. For each hour increase in sleep, the risk of overweight/obesity was reduced on average by 9% (pooled OR = 0.91; 95% CI: 0.84, 1.00).

Second, our meta-analysis indicates that a significant linear dose–response relationship can be found only in young children (<10-year-old) or for combined overweight and obesity, but not for children as a whole group. For example, a large cross-sectional study of 8,274 children aged 6–7 years in Japan reported a clear dose–response relationship between short sleep duration and childhood obesity, using the International Obesity Task Force criterion of overweight/obesity (18).

Third, our meta-analysis supports a significant gender difference in the association between sleep and obesity, as boys had a stronger inverse association than girls (OR = 2.50 vs. 1.24), although findings of previous studies are inconsistent. In the Toyama Birth Cohort Study of 8,274 children aged 6–7 years, the association between sleep duration and overweight and obesity was stronger in boys than in girls (18), the adjusted OR with 9–10 h of sleep was 2.28 vs. 1.49 when compared with ≥10 h of sleep; for 8–9 h, were 3.45 vs. 1.89 and 5.49 vs. 2.87 for <8 h, respectively. Data of 4,486 adolescents from the US National Longitudinal Study of Adolescent Health indicated that every hour increase in sleep was associated with a 10% reduction in risk of overweight in boys, but no effect in girls (32). Similarly, another study of 6,324 Australian children aged 7–15 years found an inverse relationship between sleep and overweight/obesity in boys, but not in girls (37). Most other related studies did not report a gender difference. The explanation for the apparent gender difference remains unclear. Some researchers thought that from an evolutionary perspective, girls may be more resilient to environmental stressors and need greater sleep deprivation to be affected than boys (37,55).

Further studies are needed to clarify the gender difference.

All the studies included in our meta-analysis used questionnaires as the main tool to assess sleep duration, although various measures of sleep duration were used in other studies we reviewed. Sleep logs or time-diaries ask participants to record the exact time when they turn off the lights to try to fall asleep and when they wake up in the morning. Wrist actigraphy has not been used extensively in larger population-based studies as it is relatively expensive. A study of high school students compared results from questionnaires with both sleep log and actigraphy. Moderate correlations were found between self-reported questionnaire and sleep logs ($r = 0.61$) and between self-report questionnaire and actigraphy ($r = 0.53$) for weeknights (56). Studies in younger children aged 3–10 all used parental report of sleep duration, a measure whose validity has not been well established (18,23–26,34,35). Validation of self-reported questionnaires used to assess sleep duration was conducted in at least one study using a nationally representative sample of 1,546 adolescents. The two measures of sleep were not strongly correlated and self-reported sleep duration revealed a stronger association with obesity (40). It is possible that the 24-h diary is not representative of usual sleep behavior, while questionnaire may have higher construct validity.

In general, previous research shows that long sleep duration is associated with lower risk of childhood obesity, which is consistent with the findings of our meta-analysis. For example, a cohort study of 150 US children from birth to 9.5 years of age reported that children's sleep time at age 3–4 years was negatively related to overweight at age 9.5 years old (26). Another cohort study of 2,281 US children aged 3–12 years found that compared to children with 9–10 h of sleep, those with longer sleep (≥11 h) at baseline had lower risk for overweight at the 5-year follow-up (OR = 0.84, 95% CI = 0.74–0.95) (52). A case–control study in Hong Kong also indicated that children with sleep ≥11 h were less likely to be overweight (OR = 0.31, 95% CI = 0.11–0.87) compared to those with sleep <9 h (53).

Sleep deprivation may influence the development of obesity through several possible biological pathways including increased sympathetic activity, elevated cortisol and ghrelin levels, decreased leptin and growth hormone, and/or impaired glucose tolerance (15,20–22). Hormonal changes may contribute to selection of calorie-dense food, excessive food intake, changes in energy expenditure, and insulin resistance (17). Other potential mechanisms are effects of sleep deprivation on basal metabolic rate, thermic effect of food and non-exercise activity thermogenesis (17). Laboratory studies in adults have revealed a potential mechanism for the association between sleep deprivation and weight gain (20,21,57). Findings of our meta-analysis based on epidemiologic evidence provide more supportive data on the proposed biological mechanism.

Our meta-analysis has some limitations, which are common to these types of studies, such as potential selection bias and failure to adjust for some potential confounders. Differences in study population, assessment of covariates and exposure variables, and classification of overweight/obesity may result in heterogeneity across studies and thus affect the pooled estimates. Owing to the fact that no intervention studies have been available, our meta-analysis was conducted mainly based on cross-sectional studies and could not prove causality. Nevertheless, the bias from individual studies usually tends to be relatively small and not substantially influence the overall estimates in a meta-analysis as more studies being included. These cohort studies included in our systematic review and meta-analysis show a clear relationship between short sleep duration in early life and increased risk of obesity in childhood. In addition, it is likely that there are considerable measurement errors in quantifying sleep duration using the survey instruments in these epidemiological studies, as well as difficulties modeling sleep duration needs across age. Our meta-analysis established a dose–response relationship based on previous studies which included populations with various ages and sleep needs. Sleep duration needs may vary non-linearly across childhood and much information may have been lost in the original study analyses which did not consider the complex non-linear age associations as well as other potential confounders. In turn, these may affect our findings.

As the observed associations were remarkably consistent across most of reviewed studies, the likelihood that these findings are solely due to selective publication seems unlikely. Furthermore, most selected studies lacked mental health status such as depression as a potential confounder, which is well known to affect sleep (58). Most had fairly crude measurements of socioeconomic status and PA, and in addition to depression, other behavioral factors may influence both sleep and body weight. The validity of self- or proxy-reported sleep duration questionnaire, a primary measure of sleep used in epidemiologic studies, needs to be assessed. Further longitudinal studies are needed to determine the causality. Laboratory research is necessary to confirm the biological mechanisms in pediatric population.

In conclusion, our meta-analysis shows a clear association between short sleep duration and increased risk of childhood obesity based on previous studies conducted in diverse populations. Sleep may be an important factor to consider in the prevention of childhood obesity. The prevalence of childhood obesity may be decreased by increasing sleep duration, independent of other risk factors for childhood obesity. Our findings have some important public health implications for fighting the growing childhood obesity epidemic. A combination of strategies targeting both earlier bedtime and later wake time to increase sleep duration may help prevent childhood obesity. Desirable sleep behaviors may represent an important and relatively low-cost strategy to reduce childhood obesity. Future randomized intervention trials are needed to test the effectiveness of sleep extension for obesity prevention among children and adolescents. In addition to healthy diet and adequate PA, sleep should be considered in future comprehensive childhood obesity intervention studies.

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DISCLOSURE

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