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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • REVIEW

Dose-response association between sleep duration and obesity risk: a systematic review and meta-analysis of prospective cohort studies

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Abstract

The association between sleep duration and obesity risk remains unclear. We performed an updated meta-analysis to quantify a potential dose-response relation between sleep duration and risk of obesity. PubMed and Embase were searched for prospective cohort studies examining the association between sleep duration and risk of obesity that were published up to October 28, 2017. Random effects models were used to evaluate the pooled relative risks (RRs) and 95% confidence intervals (CIs) for the association of sleep duration and obesity. Restricted cubic splines were used to model the dose-response association. The meta-analysis included 12 studies (16 reports). We found a reverse J-shaped relation between sleep duration and obesity, with the lowest risk at 7–8-h sleep per day. Compared with 7-h sleep duration per day, the pooled relative risks for obesity were 1.09 (95% CI 1.05–1.14) for each 1-h decrement among individuals who slept < 7 h per day and 1.02 (95% CI 0.99–1.05) for each 1-h increment of sleep duration among individuals with longer sleep duration. Short sleep duration significantly increased the risk of obesity. Compared with 7-h sleep duration per day, the risk of obesity increases 9% for each 1-h decrease in sleep duration.

Keywords Sleep duration · Obesity · Cohort studies meta-analysis · Dose-response relation

Introduction

The prevalence of overweight and obesity is increasing at an alarming rate. Worldwide, the number of adults with obesity increased between 1975 and 2016, from 31 to 281 million for men and from 69 to 390 million for women [1]. In addition, 213 million children and adolescents and 1.30 billion adults were considered overweight [1]. Overweight and obesity are important risk factors for some diseases, including diabetes, cardiovascular disease, cancer, and premature death [2]. Given the severe consequences and profound burden of obesity, identifying modifiable lifestyle factors is imperative to prevent obesity.

Sleep is a complex set of brain processes that supports several physiological needs [3]. Adequate sleep duration is important for maintaining good health. Several systematic reviews have reported associations between sleep duration and ill health, including mortality [4–6], diabetes [7, 8], hypertension [9, 10], cardiovascular disease [11], stroke [12], and coronary heart disease [13]. These studies suggest that moderate sleep duration promotes good health and long-term sleep deprivation may be related to various chronic diseases. However, the prevalence of short sleep has increased over the last century [14–16]. The trend of short sleep duration coincides with a striking increase in obesity prevalence [17]. Thus, to prevent obesity and hence many obesity-related chronic diseases, many studies have focused on modifiable lifestyle factors such as sleep duration involved in the development of obesity.

An increasing number of prospective studies in adults found extreme sleep duration related to risk of obesity, with inconsistent results. Some studies found a U-shaped relation

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[18–20] and others a negative linear relation [21–26] or no relation [27–30]. A previous meta-analysis [31] reported the relation between short sleep duration and obesity in children and adults. However, the pooled studies are cross-sectional and cannot establish the directionality of the association. Another meta-analysis by Wu et al. [32] in adults simply pooled the relative risks (RRs) values for short or long sleep duration and failed to clarify the dose-response relation between sleep duration and obesity risk.

We performed a systematic review and dose-response meta-analysis of prospective cohort studies of healthy adults to determine the overall shape of the relation between sleep duration and risk of obesity.

Methods

Search strategy

We searched PubMed and Embase for English reports of prospective cohort studies examining the association between sleep duration and risk of obesity in human adults that were published up to October 28, 2017. Key words included sleep, overweight, obesity, and body mass index (BMI) (details in Table S1). Moreover, we manually screened the reference lists of relevant papers and review articles for additional publications.

Study selection

Cohort studies were included if (1) they were a prospective study conducted in adults (> 18 years old at baseline); (2) they provided multivariate-adjusted RRs, odds ratios (ORs), or hazard ratios (HRs) with 95% confidence intervals (CIs) and number of cases, person-years, or participants for each category of sleep duration; (3) the exposure of interest was sleep duration; and (4) the outcome was new-onset obesity (BMI ≥ 30 kg/m^2 for Americans and Europeans or ≥ 25 kg/m^2 for Asians and Canadians). We excluded cross-control studies, reviews, comments, letters, editorials, and studies without enough quantitative data for dose-response relation. We selected the longest follow-up duration or the largest sample size if study data were published more than once.

Data extraction and quality assessment

A standard form was used to extract the first author, year of publication, study name (if applicable), location, follow-up years, sample size (number of participants and incident cases) per category, participant characteristics (age and gender), sleep duration categories, obesity assessment, the adjusted covariates, and ORs or RRs or HRs with 95% CIs (adjusted by the most confounders) for all categories of sleep duration.

Quality was evaluated by the Newcastle-Ottawa Scale (NOS) [33], with scores ranging from 0 to 9 summarizing eight aspects of each study, higher scores representing higher study quality.

For a dose-response analysis, the number of participants, exposed person-years, incident cases, and mean or median sleep duration for each category were further extracted. We contacted the corresponding authors when data were missing.

Statistical analysis

We used the RRs and 95% CIs as the effect size for all studies; we assumed that the HRs and ORs reported for obesity risk in each original study were approximately RRs [34]. If the number of incident cases in a sleep duration category was missing, these data were calculated by using the number of total cases and the reported effect size; the groups were assumed to be equal in size when the exposed person-years or participant numbers were not provided for each sleep duration category [35]. Additionally, we assigned the median or mean sleep duration per category to the corresponding RRs for each study. If the median or mean duration per category was not provided, we used the midpoint of the lower and upper bounds for that category. When the shortest or longest category was open-ended, we assumed that the open-ended interval length had the same length as the adjacent interval.

In most of the included studies, 7 to 8 h per day was considered as the reference category. When the reference category was not 7 to 8 h, we used the method proposed by Hamling and colleagues to convert risk estimates [36]. We calculated pooled RRs and 95% CIs for the shortest and longest sleep duration versus the reference category. In addition, the studies with at least three quantitative categories of short or long sleep duration were included in dose-response analyses. We adopted restricted cubic splines with three knots (25th, 50th, and 75th percentiles) of exposure distribution to evaluate possible nonlinear dose-response relations between sleep duration and obesity. Finally, if a nonlinear association was observed, we treated the slope of the curve as two piecewise and used the DerSimonian and Laird random effects model [37] to pool the study-specific dose-response RRs for 1-h decrement or increment compared with the reference category in sleep duration.

Heterogeneity was assessed by the Q test and I^2 statistic [38]. For the Q statistic, $P < 0.1$ was considered statistically significant, then we used a random effects model; otherwise, a fixed effects model was used. $I^2 > 50\%$ was considered statistically significant heterogeneity. Subgroup analyses were conducted by sex, age, nationality, cutoff of short sleep duration, study quality, and adjustment for confounders. We also conducted sensitivity analyses by omitting 1 study at a time to assess the effects of a single study on the pooled results. Publication bias was assessed by funnel plots and by Egger's and Begg's tests [39].

All analyses involved use of Stata 12.0 (Stata Corp, College Station, TX), and all tests were two-sided with a statistical significance level of 0.05.

Results

Literature search

We identified 2658 studies from PubMed and 5984 from Embase that were published up to October 28, 2017 (Fig. 1). After screening titles and abstracts, 86 full-text studies were eligible for further assessment. Finally, we included results for 12 prospective cohort studies (16 reports) in the meta-analysis [19, 23, 24, 26, 28, 29, 40–45]. Some studies reported results by sex, so we treated them as two independent reports [24, 28, 29, 43].

Study characteristics

The sample sizes ranged from 151 to 67,344 (total 198,579 participants) and the duration of follow-up for incident obesity ranged from 1 to 12 years (Table 1). Three studies were conducted in the USA [19, 26, 42], five in Japan [24, 29, 40, 41, 44], two in Spain [23, 43], and one each in Canada [45] and the UK [28]. Sleep duration was self-reported in 11 articles, measured objectively by using polysomnography (PSG) in only one article [42]. Obesity was defined as BMI ≥ 30 kg/m^2 in the studies from the USA [19, 26, 42] and Europe [23, 28, 43] and BMI ≥ 25 kg/m^2 in studies from Asia [24, 29, 40, 41, 44] and Canada [45]. The mean NOS score was 6.9/9 (Table S2), indicating that the articles as reported were high quality.

Short sleep duration and risk of obesity

We included results from 12 studies (16 reports) involving 198,579 participants in the analysis of short sleep duration and obesity. The pooled RR of the shortest sleep duration versus reference sleep duration was 1.48 (95% CI 1.29–1.72), with high heterogeneity ($I^2 = 66.3\%$, $P = 0.000$) (Fig. 2a). On sensitivity analysis, none of the single studies altered the pooled RR substantially (Figure S1). A possible publication bias was found between short sleep duration and obesity risk (Egger's test, $P = 0.037$; Begg's test, $P = 0.558$) (Figure S2A). With the trim-and-fill method used to adjust for publication bias, the main result was not altered significantly (RR 1.23; 95% CI 1.16–1.31) (Figure S2B).

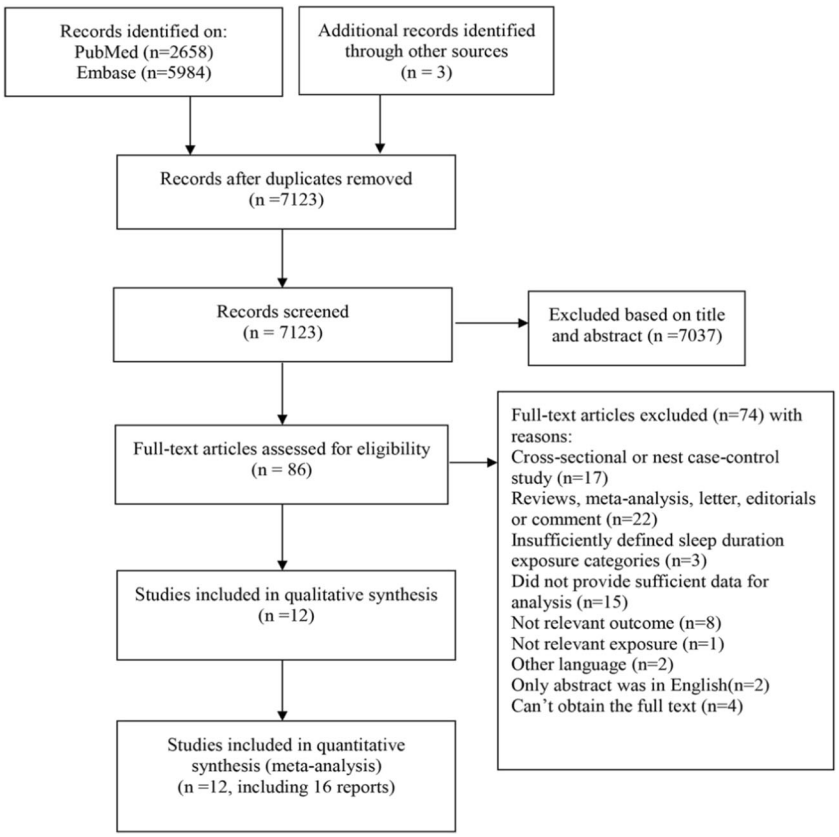


Fig. 1 Flowchart of study screening and selection

Table 1 Characteristics of the included studies

Author, year	Country	Follow-up (years)	Sex	Age (years)	Cohort size	Exposure categories	Reported OR/RR/HR	Adjustment	NOS
	Korea	2.6	W/M	40–70	2579	< 6	1.30 (0.98–1.69)	Age, sex, education, smoking, alcohol intake, total calorie intake, and exercise	7
						6–7.9	1		
						≥ 10	0.97 (0.62–1.50)		
Vgontzas et al., 2014 [42]	USA	7.5	W/M	48.9	815	≤ 5	1.08 (0.48–2.41)	Objective sleep duration, sleep difficulty, emotional stress, gender, race, age, AHI, BMI, age × BMI and depression	7
						5–6	1.27 (0.89–2.76)		
						6–7	1.03 (0.46–2.34)		
						≥ 7	1		
Gutiérrez-Repiso et al., 2014 [23]	Spain	14	W/M	39.2	673	≤ 7	2.73 (1.47–5.04)	Age, sex, leisure physical activity, and weight gain	6
Xiao et al., 2013 [26]	USA	7.5	M	51–72	35,319	< 5	1.45 (1.06–1.99)	Age, baseline BMI, race/ethnicity, marital status, educational level, self-reported health, smoking status, alcohol consumption, and coffee consumption	6
						5–6	1.03 (0.93–1.14)		
						7–8	1		
						≥ 9	1.12 (0.84–1.49)		
Xiao et al., 2013 [26]	USA	7.5	W	51–72	32,025	< 5	1.37 (1.04–1.79)	Age, baseline BMI, race/ethnicity, marital status, educational level, self-reported health, smoking status, alcohol consumption, and coffee consumption	6
						5–6	1.15 (1.05–1.28)		
						7–8	1		
						≥ 9	0.91 (0.60–1.20)		
Sayón-Orea et al., 2013 [43]	Spain	6.5	M	39.1	4689	< 5	2.09 (1.18–3.69)	Age, sex, smoking status, physical activity, time spent sitting down, fast food, sugared soft drinks, snacking between meals, total energy intake, caffeine, alcohol, snoring, insomnia, siesta hours, and baseline BMI	7
						5–7	1.03 (0.91–1.45)		
						7–8	1		
						≥ 8	0.88 (0.64–1.21)		
Sayón-Orea et al., 2013[43]	Spain	6.5	W	39.1	5843	< 5	1.26 (0.44–3.57)	Age, sex, smoking status, physical activity, time spent sitting down, fast food, sugared soft drinks, snacking between meals, total energy intake, caffeine, alcohol, snoring, insomnia, siesta hours, and baseline BMI	7
						5–7	1.34 (0.90–2.00)		
						7–8	1		
						≥ 8	1.43 (0.97–2.10)		
Nagai et al., 2013 [29]	Japan	12	M/W	57.9	6162	≤ 5	0.96 (0.59–1.57)	Sex; age; body mass index; education; smoking status; alcohol drinking; time spent walking/day; job status; marital status; menopause; coffee; self-rated health	6
						6	1.09 (0.87–1.38)		
						7	1		
						8	1.08 (0.90–1.28)		
						≥ 9	1.06 (0.82–1.37)		
Itani et al., 2011 [24]	Japan	7	M	≥ 8	11,424	< 5	1.20 (1.09–1.32)	Age class, eating habits, alcohol consumption, smoking habit, exercise habit, mental complaints, hypertension, hyperglycemia, hypertriglyceridemia, and hypo-HDL, cholesterolemia	8
						5–7	1		
						> 7	1.00 (0.51–1.94)		
Itani et al., 2011 [24]	Japan	7	W	≥ 8	899	< 5	1.71 (1.11–2.87)	Age class, eating habits, alcohol consumption, smoking habit, exercise habit, mental complaints, hypertension, hyperglycemia, hypertriglyceridemia, and hypo-HDL, cholesterolemia	8
						5–7	1		
Kobayashi et al.,2011[44]	Japan	3	W/M	47.5	11,136	≤ 5	1.50 (1.10–2.00)	Age, gender, baseline BMI, alcohol drinking, exercise, hypertension, dyslipidemia,	8
						6	1.10 (0.90–1.40)		

Table 1 (continued)

Author, year	Country	Follow-up (years)	Sex	Age (years)	Cohort size	Exposure categories	Reported OR/RR/HR	Adjustment	NOS
						7	1	diabetes, cerebral infarction, and	
						≥ 8	1.30 (0.90–1.80)	myocardial infarction	
Watanabe et al., 2010 [41]	Japan	1	M	39.9	20,023	< 5	1.91 (1.36–2.67)	Age, shift-worker, smoking, alcohol	7
						5–6	1.50 (1.25–1.80)	consumption, and physical activity,	
						6–7	1.14 (0.98–1.32)	depressive symptoms	
						7–8	1		
						8–9	1.17 (0.88–1.55)		
						≥ 9	1.42 (0.69–2.96)		
Watanabe et al., 2010 [41]	Japan	1	W	39.9	3189	< 5	0.35 (0.05–2.69)	Age, shift-worker, smoking, alcohol	7
						5–6	0.82 (0.40–1.68)	consumption, and physical activity,	
						6–7	1.04 (0.59–1.84)	depressive symptoms	
						7–8	1		
						8–9	0.64 (0.15–2.79)		
						≥ 9	0		
Nishiura et al., 2010 [40]	Japan	4	M	47.8	2632	< 6	2.46 (1.41–4.31)	Age, baseline BMI, current medication,	8
						6–6.9	1.03 (0.68–1.58)	family history of disease, smoking,	
						7–7.9	1	drinking, exercise, preference for fatty	
						≥ 8	0.75 (0.35–1.62)	food, skipping breakfast, snacking, and	
								eating out	
Chaput et al., 2010 [45]	Canada	6	W/M	18–64	151	< 6	2.97 (1.68–4.34) 1	Age, sex, baseline BMI, length of follow-up,	6
						7–8		and socioeconomic status.	
Stranges et al., 2008 [28]	England	4	M/W	55.4	3786	≤ 5	1.05 (0.60–1.82)	Age, sex, employment, alcohol consumption,	7
						6	1.21 (0.89–1.64)	smoking, physical activity and	
						7	1	cardiovascular drugs, mental and physical	
						8	0.97 (0.64–1.47)0.91	scores (SF-36), depressive symptoms, and	
						≥ 9	(0.27–3.01)	use of hypnotics	
Patel et al., 2006 [19]	USA	12	W	52.4	59,813	≤ 5	1.15 (1.04–1.27)	Age, year of follow-up, and baseline BMI,	7
						6	1.06 (1.01–1.12)	smoking, alcohol consumption, caffeine	
						7	1	intake, spousal level of education,	
						8	1.03 (0.97–1.09)1.03	medication use, menopausal status, snoring	
						≥ 9	(0.93–1.14)	status, shift-working history, physical	
								activity, total caloric intake, dietary fiber,	
								ratio of polyunsaturated to saturated fat,	
								trans-fat intake, and servings of fruits and	
								vegetables	

NOS, Newcastle-Ottawa Scale; *RR*, relative risk; *OR*, odds ratio; *HR*, hazard ratio; *CI*, confidence interval; *BMI*, body mass index; *AHI*, apnea-hypopnea index; *HDL*, high-density lipoprotein; *SF-36*, Medical Outcomes Study Short Form 36

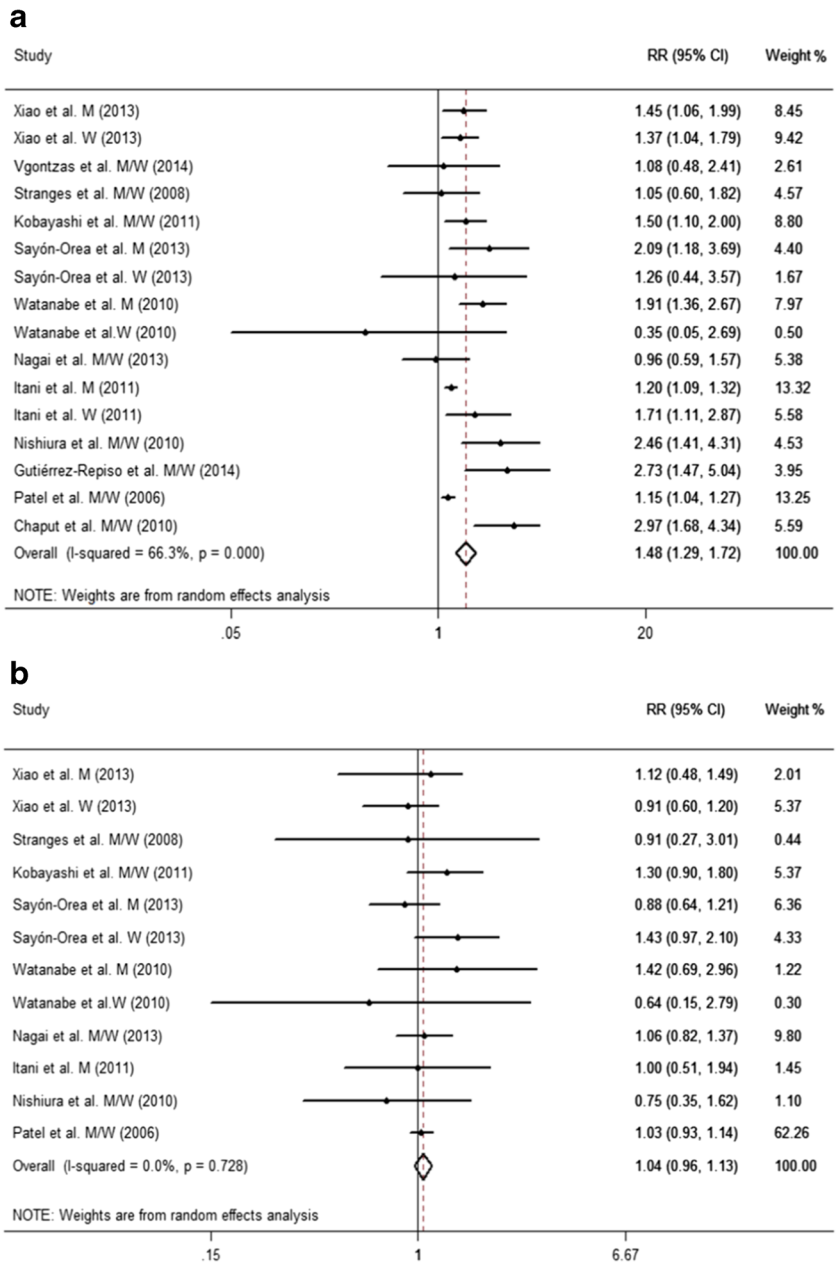


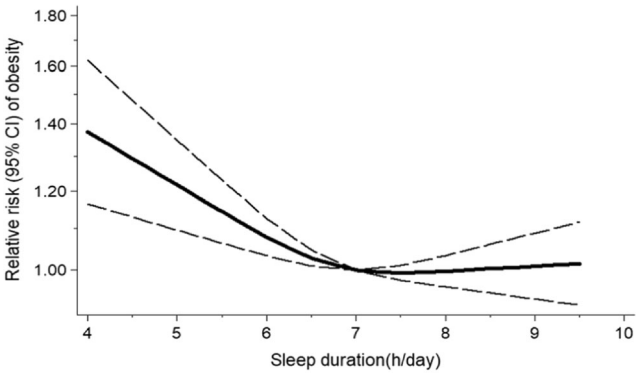
Fig. 2 The forest plots between sleep duration and risk of obesity for short sleep (**a**) and long sleep (**b**). RR relative risk, CI confidence interval

Long sleep duration and risk of obesity

Three studies reported insufficient data on long sleep duration and obesity risk [23, 24, 45]. We included results from 9 studies (12 reports) involving 196,041 participants in the analysis of long sleep duration and obesity. The pooled RR of the longest sleep duration versus reference sleep duration was 1.04 (95% CI 0.96–1.13), without heterogeneity ($I^2 = 0.0\%$, $P = 0.728$) (Fig. 2b). On sensitivity analysis, none of the single studies altered the pooled RR substantially (Figure S3). In addition, no publication bias was observed (Egger's test, $P = 0.944$; Begg's test, $P = 0.891$) (Figure S4).

Dose-response relation between sleep duration and risk of obesity

Eight studies with 11 reports were included to explore the overall shape of the association between sleep duration and risk of obesity. We found a reverse J-shaped curvilinear relation between sleep duration and risk of obesity ($P < 0.01$ for nonlinearity), with the lowest risk of obesity at a sleep duration of 7–8 h per day (Fig. 3). Short sleep duration increased risk of obesity significantly, but long sleep did not. Results from the cubic spline model suggested that when compared with 7 h per day, those who slept shorter had higher risk of obesity. For example, for participants with sleep duration at



Sleep duration	Obesity
4h	1.37 (1.16-1.62)
5h	1.22 (1.09-1.35)
6h	1.08 (1.03-1.13)
7h	1.00
8h	1.00 (0.96-1.03)
9h	1.01 (0.94-1.09)

Fig. 3 Nonlinear dose-response association between sleep duration and incident obesity modeled by using restricted cubic splines. Data are RR (95% CI)

5 h per day and 4 h per day, the risk of obesity was increased by 22% (RR, 1.22; 95% CI 1.09–1.35) and 37% (RR, 1.37; 95% CI 1.16–1.62), respectively. The reverse J-shaped association remained when analyzing only studies with a subjective measurement of sleep duration (Figure S5).

For short sleep, the pooled RR for risk of obesity was 1.09 (95% CI 1.05–1.14) per 1-h decrement of sleep duration compared with 7–8 h, with moderate heterogeneity ($I^2 = 50.7\%$, $P = 0.027$; Fig. 4a). On sensitivity analysis, none of the single studies altered the pooled RR substantially (Figure S6). No publication bias was detected (Egger's test, $P = 0.567$; Begg's test, $P = 0.876$) (Figure S7).

For long sleep, the pooled RR for risk of obesity was 1.02 (95% CI 0.99–1.05) per 1-h increment of sleep duration compared with 7–8 h, without heterogeneity ($I^2 = 0.0\%$, $P = 0.456$; Fig. 4b). On sensitivity analysis, none of the single studies altered the pooled RR substantially (Figure S8). No publication bias was detected (Egger's test, $P = 0.430$; Begg's test, $P = 0.371$) (Figure S9).

Subgroup analysis

To test the sources of heterogeneity across studies, we performed subgroup dose-response analysis by sex, age, nationality, duration of follow-up, study quality, number of participants, number of cases, and adjustment for confounders (Table S3). In general, the association between sleep duration and risk of obesity was similar in most analyses, but it implied that gender might be the major possible source of heterogeneity. The pooled result for men was borderline (1.12 [0.99–1.27]) when we stratified studies by gender. No significant changes of heterogeneity occurred in other subgroup analyses.

Discussion

This meta-analysis of prospective cohort studies revealed a reverse J-shaped curvilinear relation between sleep duration and risk of obesity, with the lowest risk at 7–8-h sleep per day. Short sleep duration significantly increased the risk of obesity, but long sleep duration did not increase the risk. Compared with 7 h per day, each 1-h decrease led to an increase of 9% in risk of obesity.

Whether short or long sleep duration is a risk factor for obesity is debated. Two meta-analyses [31, 32], both exploring the relation between sleep duration and risk of obesity among adults, showed short sleep duration significantly related to increased incident risk of obesity, which agrees with our results. However, for the study by Cappuccio et al. [31], most of the pooled articles were cross-sectional and could not determine temporal sequence and therefore causality. Different from the study by Wu et al. [32], we quantitatively assessed the relation between sleep duration and risk of obesity by a dose-response analysis. Additionally, our study included more comprehensive original literature and had a more rigorous methodological strength to improve the accuracy and reliability of the findings.

The mechanisms linking short sleep duration and obesity remain complex. Several potential pathways have been proposed [46, 47]. First, laboratory-based studies have shown sleep restriction related to low levels of leptin and high levels of ghrelin, which may increase hunger and thus caloric intake [48–51]. Also, lack of sleep may be associated with irregular eating habits such as eating snacks between meals [52] and lower consumption of fruits and vegetables [53]. Second, sleep deprivation increases fatigue, resulting in decreased levels of physical activity and increased sedentary behavior, thereby reducing energy expenditure [46]. Third, sleep restriction could activate the hypothalamic-pituitary-adrenal axis, increasing cortisol levels, which increases food intake and leads to the accumulation of visceral fat [46, 54, 55]. In addition, melatonin may mediate the effects of sleep on body weight [56]. In animal studies [57–59], short sleep duration reduced melatonin levels, decreasing the metabolic activity of brown adipose tissue and thus causing weight gain. Our meta-analysis showed no association between long sleep duration and subsequent obesity.

We conducted subgroup analyses to explore the sources of heterogeneity among studies and found the association between sleep duration and obesity similar in most analyses. The heterogeneity seemed to be lower in studies of women.

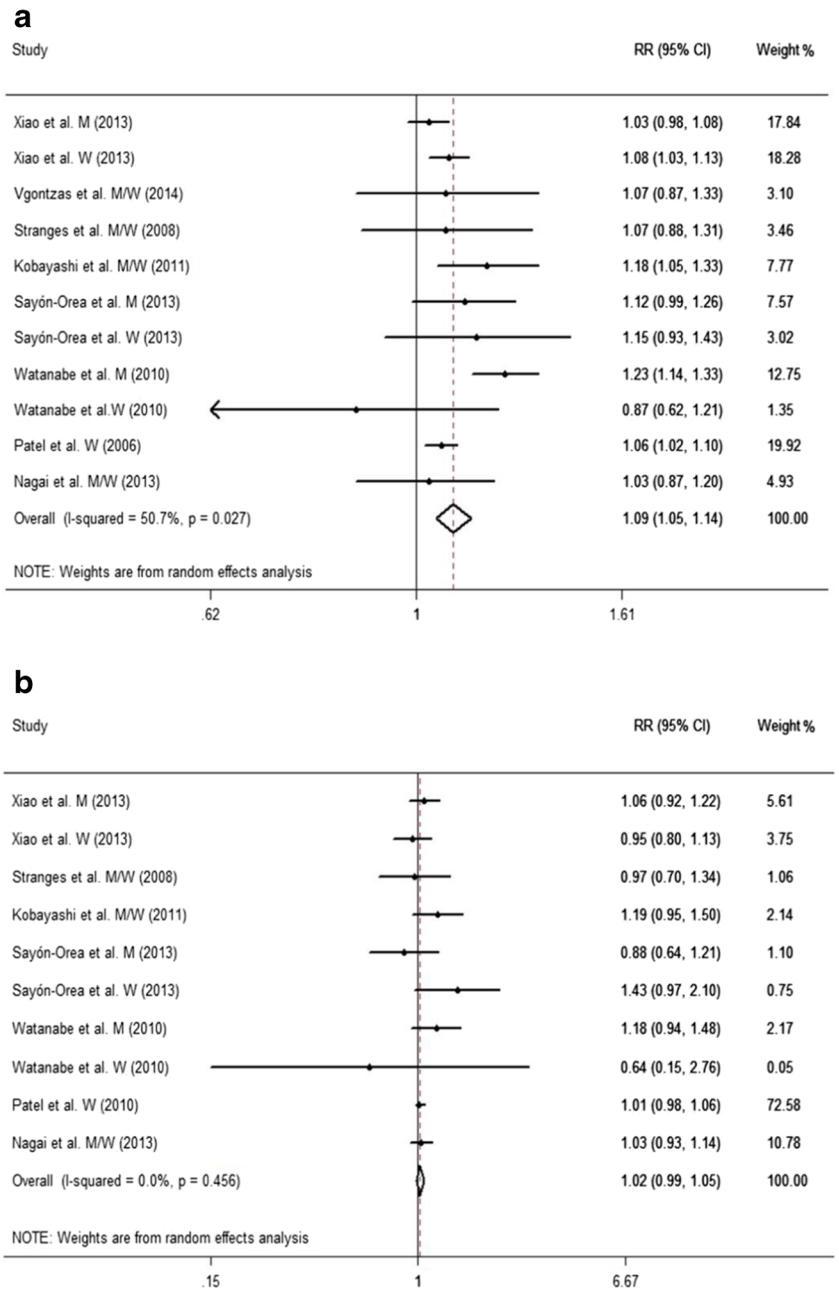


Fig. 4 The forest plots between sleep duration (per hour) and risk of obesity for short sleep (**a**) and long sleep (**b**). RR relative risk, CI confidence interval

Women seemed to be more vulnerable to the impacts of short sleep duration on obesity than men because of differences in endocrine and socioeconomic status. Gender differences in the risk of obesity by sleep duration have been reported [60]. The heterogeneity that seemed to be higher in Asian populations may be due to an imbalanced gender ratio. Given limited research in this area, we need more studies estimating possible gender-specific differences in the relation between sleep restriction and obesity.

Our meta-analysis has several strengths. Only prospective cohort studies were included, so the causal association was somewhat rigorous. The mean quality score of 6.9/9 suggested high credibility of our study. Moreover, we used restricted cubic splines and a piecewise linear function to investigate the potential nonlinear and linear relation between sleep duration and risk of obesity, which made full use of the available data and had higher statistical power than traditional binary meta-analysis. Therefore, the present study might provide useful and robust evidence to explore the association between sleep duration and obesity.

Several limitations of this study should be considered. First, in nearly all studies, sleep duration was self-reported; the gold standard of actigraphy and polysomnography may provide more objective measures. However, polysomnography may not be feasible in large prospective cohort studies due to its high expenditure [61]. Besides, the

results were consistent in the present meta-analysis after excluding one study with an objective measurement of sleep duration. Second, most studies assessed sleep variables at one time, but sleep duration is a dynamic process, so they may not fully capture the continuing effect of sleep duration over time when correlated with long-term disease onset. Third, although extracted risk estimates were adjusted by the most confounders, we cannot rule out residual or unmeasured confounding, and we did not assess some possible covariates (sleep quality, sleep apnea). Finally, we excluded studies that were unpublished or published in other languages. Despite the limitations, findings from prospective cohort studies remain the best evidence to evaluate the longitudinal effect of sleep duration on obesity.

In conclusion, our dose-response systematic review and meta-analysis of cohort studies found a reverse J-shaped curvilinear relation between sleep duration and risk of obesity, with the lowest obesity risk at 7–8-h sleep per day. Short sleep duration significantly increased the risk of obesity. Given the relatively small number of studies included in this study, more studies are needed to confirm this result. In addition, randomized controlled trials with larger sample sizes and longer follow-up are needed to explore the underlying mechanisms and causal associations.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval Not applicable for this meta-analysis in which all analyses were based on published literature.

Informed consent Not applicable for this meta-analysis in which all analyses were based on published literature.

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