Original Research Article

Short night sleeping is associated with higher risk of diabetes in older adults

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ABSTRACT

Background: It remains unclear how many hours of sleep are associated with the lowest risk of diabetes type II. This meta-analysis was completed to evaluate the dose-response relationship between sleep duration and risk of diabetes type II.

Methods: We conducted this meta-analysis using a comprehensive search of Medline, Pubmed, EMBASE, Cochrane database of systematic reviews, and Cochrane central register of controlled trials till 01 May 2017 for prospective observational studies that assessed the relationship of sleep duration and risk of type II diabetes. Both semiparametric and parametric methods were used.

Results: Ten articles with 7 reports were eligible for inclusion in the meta-analysis. A total of 16,123 incident cases of type II diabetes were ascertained among 402,397 participants with follow-up periods ranging from 3 to 17 years. The relationship was observed between sleep duration and risk of type II diabetes, with the lowest risk observed at a sleep duration category of 7–8 h per day. Compared with 7-h sleep duration per day, the pooled relative risks for type II diabetes were 1.11 (95% CI 1.06–1.17) for each 1-h shorter sleep duration among individuals who slept <7 h per day and 1.13 (1.05–1.31) for each 1-h increment of sleep duration among individuals with longer sleep duration.

Conclusions: Both short and long sleep duration are linked with a considerably increased risk of type II diabetes, underscoring the significance of appropriate sleep duration in the delay or prevention of type II diabetes.

Keywords: Sleep duration, Type II diabetes, Risk, Meta-analysis

INTRODUCTION

Given the significant burden of diabetes, it is imperative to identify modifiable lifestyle factors connected with lower risk of diabetes. Sleep is a biobehavioral phenomenon that is regulated by circadian, homeostatic, and neurohormonal processes.[1] In the past few years, suboptimal sleep duration, particularly short sleep, as a disorder character rising out of the 24 hours lifestyle of modern societies, has increasingly been shown to signify an extra behavioral factor undesirably affecting public health.[2,3]

Numerous studies have reported a U-shaped relationship between type II diabetes and sleep duration, but other studies have not found a uniform relationship.[4-6] One previous meta-analysis suggested that both short and long sleep duration were connected with risk of type II diabetes.[6] Though, the definitions of short and long sleep duration differed between studies, which complicated the interpretation of the pooled outcomes. Furthermore, without a dose-response analysis, it residues unidentified how many hours of habitual sleep are connected with the lowest risk of type II diabetes. In the last few years, several prospective studies with enough quantitative categories have nearly doubled.[7-13] Consequently, we conducted a meta-analysis of prospective studies to define the overall shape of the association between sleep duration and risk of diabetes type II.

METHODS

Data sources and searches

We conducted this meta-analysis using a comprehensive search of Medline, PubMed, EMBASE, Cochrane database of systematic reviews, and Cochrane central register of controlled trials till 01 May 2017 for prospective observational studies that assessed the relationship of sleep duration and risk of type II diabetes. Both semiparametric and parametric methods were used. No language restrictions were imposed. We followed the standard guidelines for conducting and reporting meta-analyses of observational studies.[14]

Selection criteria

Studies were included in this meta-analysis if they satisfied the following criteria: the study design was prospective, the exposure of interest was sleep duration, the outcome was type II diabetes, and the investigators reported relative risks (RRs) with 95% CIs for at least three quantitative categories of short sleep or long sleep duration. We excluded animal studies, clinical trials, cross-sectional studies, case-control studies, reviews, commentaries, letters, and studies that examined other associations. If study populations were reported more than once, we used the result with the longest follow-up duration.

Data extraction

Two reviewers independently reviewed studies, abstracted data, and resolved disagreements by consensus. Studies were evaluated for quality. A review protocol was followed throughout. We extracted the following data from each study: year of publication, authors, study location, study name, years of follow-up, sample size (number of participants and incident cases), participant characteristics (age and sex), sleep duration categories, type II diabetes assessment, covariates adjusted in the multivariable analysis, and RRs (95% CIs) for all categories of sleep duration. To achieve a dose-response meta-analysis, we allocated the median or mean sleep duration in each category of duration to the corresponding RR for each study. If the mean or median duration per category was not reported, the midpoint of the upper and lower boundaries in each category was allocated. When the shortest or the longest category was open-ended, we presumed that the open-ended interval length had the same length as the adjacent interval.

Statistical analysis

In this meta-analysis, the RRs and 95% CIs were considered as the effect size for all studies. Since the incidence of type II diabetes is sufficiently low, odds ratios were deemed equivalent to RRs. The heterogeneity between studies was assessed by the Cochran Q test and I^2 statistic. Heterogeneity was considered statistically significant at $p \leq 0.10$. The I^2 statistic defines the percentage of total variation in point estimates that can be attributed to heterogeneity. For the I^2 metric, we deliberated low, moderate, and high I^2 values to be 25, 50, and 75%, respectively. To explore the sources of heterogeneity among studies and to test the robustness of the associations, we conducted subgroup analyses by study location, number of participants, duration of follow-up, and study quality, as well as several sensitivity analyses. The possibility of publication bias was assessed using the Egger regression asymmetry test [15]. For sensitivity analysis, we likewise utilized the fixed-effects model for all the above analyses. Additional sensitivity analyses were completed by ignoring one study at a time and calculating a pooled estimate for the remainder of the studies to assess whether the results were affected markedly by a single study. All statistical analyses were performed with Stata version 12 (Stata Corp), and all tests were two sided with a significance level of 0.05.

RESULTS

The results of the literature research and study selection are shown in (Figure 1). We identified 966 articles. After exclusion of duplicates and studies that did not fulfill the inclusion criteria, 10 remaining articles seemed to be relevant for this meta-analysis. After evaluating the full texts of these 10 publications, we further excluded 3 articles. As a result of a lack of sufficient data for estimation of dose-response analysis. Furthermore, we

included the report of the control group, and not the intervention group, in the study by Tuomilehto et al.[12] In total, our meta-analysis included 7 articles.[7-13]
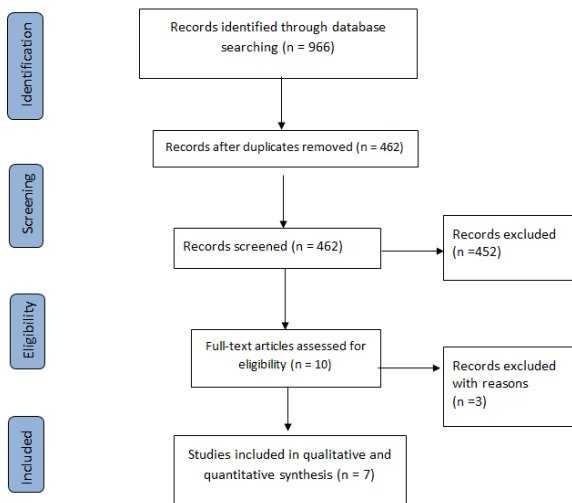

Figure 1: Flow diagram showing the selection criteria of assessed studies.

Characteristics of all 7 studies were shown in Table 1. The duration of follow-up for incident type II diabetes ranged from 3 to 17 years, with a median follow-up of 7.5 years. Sleep duration was evaluated by questionnaire in all studies. One study was a sub-study of a prospective lifestyle intervention trial [14], and the remaining six were prospective cohort studies. The mean NOS score was 6.8 (of a possible 9 points), suggesting the high quality of the studies included in the meta-analysis. All studies were included to explore the overall shape of the association between risk of type II diabetes and sleep duration.[9-15]

Short duration of sleep and risk of type II diabetes

The semiparametric analysis included 6 studies on short sleep and type II diabetes risk. Table 2 shows the RRs for type II diabetes with different levels of short sleep duration relative to the reference category. Compared with the reference category of sleep duration (7 h per day), the pooled RR for incident type II diabetes was 1.11 (95% CI 1.03–1.08, $I^2=8\%$, P for heterogeneity=0.41) for the second shortest (6 h per day) and 1.41 (1.21–1.62, $I^2=56\%$, p=0.019) for the shortest (≤5 h per day) category of sleep duration. Consequently, there was indication for substantial amid study heterogeneity in results for the shortest category of sleep duration. The above-mentioned studies were included in the dose-response analysis of short sleep duration and risk of type II diabetes. No indication was found of a curvilinear association among risk of type II diabetes and short sleep duration (p=0.19 for nonlinearity). Compared with 7 h sleep duration per day, the pooled RR for type II diabetes was 1.11 (95% CI 1.06–1.17) per 1-h reduction of sleep duration, with evidence of heterogeneity ($I^2=64\%$, p=0.005) (Table 2).

Table 1: Characteristics of prospective studies.

Author	year	Sample size	No. of diabetes cases	Follow up (years)	Covariates in fully adjusted model
Holliday[9]	2013	156,902	3,641	3	Age, smoking status, education, gender, marital status, residential remoteness, alcohol consumption, physical activity, health insurance status, income, BMI, and baseline health status
Boyko[10]	2013	47,093	871	6	Age, gender, education, BMI, and race/ethnicity
von Ruesten[11]	2012	23,620	841	7.8	Age, gender, sleeping disorders, alcohol intake from beverages, smoking status, walking, cycling, sports, employment status, education, BMI, waist-to-hip ratio, prevalent hypertension at baseline, history of high blood lipid levels at baseline, caffeinated beverages, satisfaction with life, satisfaction with health, and intake of antidepressants
Gangwisch[12]	2007	8,992	430	8–10	Age, depression, physical activity, education, alcohol consumption, ethnicity, marital status, overweight/obesity, and hypertension
Yaggi[13]	2006	1,139	90	15–17	Age, education, smoking, hypertension, self-rated health status, and waist circumference
Tuomilehto[14]	2009	252	107	7	Age, gender, BMI, smoking, study center, alcohol consumption, hypertension medication, leisure-time physical activity at baseline, and 1-year change in body weight
Xu[15]	2010	164,399	10,143	8	Age, race, gender, education, marital status, smoking, coffee and alcohol consumption, calorie intake, FHD, and general health status

Table 2: The dose-response relationship plot between sleep duration (per hour) and risk of type II diabetes for short sleep.

Study	RR (95% CI)	Weight (%)
Holliday[13]	1.07 (1.02–1.16)	21.42
Boyko[14]	1.26 (1.14–1.38)	16.42
von Ruesten[15]	1.02 (0.92–1.13)	15.52
Gangwisch[20]	1.16 (1.01–1.34)	11.85
Yaggi[21]	1.33 (1.01–1.77)	6.52
Xu[17]	1.05 (1.03–1.07)	28.27
Total	1.11 (1.06–1.17)	100,00

Long duration of sleep and risk of type II diabetes

Seven studies were included in the semiparametric analysis on long sleep duration and risk of type II diabetes. The pooled RR for type II diabetes was 1.12 (95% CI 0.98–1.31, I^2=61.0%, p=0.025) for the second longest (8 h per day) and 1.39 (1.11–1.78, I^2=76.4%, p<0.001) for the longest (≥9 h per day) category of sleep duration. Substantial between-study heterogeneity was shown in results for both categories of long sleep duration. No potentially nonlinear dose-response relationship was detected (p=0.88 for nonlinearity). The pooled RRs for type II diabetes were 1.13 (95% CI 1.05–1.31) per 1-h increment of sleep duration compared with 7 h, with evidence of heterogeneity (I^2=78.9%, p<0.001) (Table 3). The corresponding absolute risk difference was estimated to be 106 cases of type II diabetes per 100,000 individuals per year per 1 h increase of sleep duration compared with 7h per day.

Table 3: The dose-response relationship plot between sleep duration (per hour) and risk of type II diabetes for long sleep.

Study	RR (95% CI)	Weight %
Holliday[13]	1.01 (0.98–1.05)	22.34
Boyko[14]	1.11 (1.01–1.04)	17.22
von Ruesten[15]	1.01 (0.92–1.07)	20.05
Gangwisch[20]	1.18 (1.01–1.36)	16.01
Yaggi[21]	1.56 (1.17–2.08)	8.31
Tuomilehto[18]	1.33 (1.15–1.53)	16.08
Total	1.13 (1.05–1.31)	100,00

Begg and Egger regression tests provided no evidence of substantial publication bias (p>0.05 for both tests).

DISCUSSION

There has been some discussion about the association between type II diabetes and sleep duration, and the outcomes of published epidemiologic studies have been inconsistent. One potential reason is that categories of sleep duration differed across studies such as the definitions of short sleep duration ranged from ≤5 to ≤7 h per day. According to the current results, a previous meta-analysis specified both short and long sleep duration were associated with an increased risk of type II diabetes, however, they did not use a dose-response analysis to determine the overall shape of the association.[16] In the current meta-analysis, we found an association between sleep duration and risk of type II diabetes, with the lowest risk of type II diabetes at a sleep duration of 7–8 h per day in our analysis, which could contribute to recommendations concerning appropriate sleep duration for future intervention studies. A recent study has shown that both habitual short and long sleep duration were indicated to be associated with risk of obesity in younger (<40 years) but not in older women (>40 years).[17] Nevertheless, the association was still detected when we restricted to the studies that were conducted in subjects older than 40 years. Moreover, gender and ethnic differences have been described in the risk of type II diabetes by sleep duration, nonetheless, we could not examine these differences attributable to limited data from the original studies.

Possible mechanisms connecting sleep to diabetes might vary among short and long sleep. Numerous potential biologic mechanisms might contribute to the relation of short sleep duration and diabetes. First, laboratory studies have corroborated and extended the reductions in glucose tolerance and insulin sensitivity after sleep restriction, as shown by increased hepatic glucose production and decreased peripheral glucose disposal.[18,19] A recent study specified that slow wave sleep suppression but not rapid eye movement sleep disturbance during nocturnal sleep plays a key role in the regulation of glucose.[20] Changes in the activity of neuroendocrine systems seem to be major mediators of the detrimental metabolic effects of insufficient sleep. Increased sympathetic nerve activity can lead to reduced β-cell responsiveness and insufficient pancreatic insulin secretion, though increased uptake of glucose by total sleep or slow wave sleep deprived brain could result in increased circulating levels of glucose and postprandial insulin-to-glucose ratio. Both insufficient insulin and improved glucose can cause the development of insulin resistance and type II diabetes. In the meantime, sleep disruption throughout the night is related with decreased testosterone and melatonin secretion, and it was likely that sleep disruption was associated to diabetes via a mechanism of melatonin or testosterone.[21] Though, another study likewise found that restricting sleep could result in an insulin-resistant state in human adipocytes through decrease of phosphorylation of AKT, which specified that sleep can be a significant regulator of energy metabolism in peripheral tissues.[22] Reduced phosphorylation of AKT is linked to insulin resistance through negative insulin receptor substrate functions, reduced phosphatidylinositol 3-kinase (PI3K) activity, and impaired phosphorylation of the AKT substrate AS160.[23]

Second, associated with hunger/appetite and caloric intake due to increases in ghrelin and decreases in leptin, short sleep may lead to greater time to eat as well as

fatigue leading to lower physical activity levels, thereby increasing risk of weight gain and subsequent health risks.[24] In the meantime, short sleep duration was associated with depressive symptoms, low socioeconomic status, low education, and other risk factors of diabetes. Individuals with sleep loss, particularly shift workers, also have irregular sleep schedules, which could result in circadian misalignment and augment markers of insulin resistance and inflammation independently of sleep loss. Finally, short sleep duration has been associated with increases of inflammatory markers, such as C-reactive protein and interleukin-6 (IL-6), which indicate low-level systemic inflammation and play a role in diabetes development.[26] Short and long sleep duration were associated with increased levels of inflammation markers, and it is possible that sleep disruption is related to diabetes via a mechanism of low-grade systemic inflammation. However, it is also possible that long sleep is a consequence of the sleep-inducing effects of the inflammatory state.[26]

CONCLUSION

The current meta-analysis of prospective studies demonstrates the association between sleep duration and risk of type II diabetes, with the lowest type II diabetes risk at 7–8 h per day of sleep duration. Both short and long sleep duration are associated with an elevated risk of type II diabetes. Longer-term randomized controlled trials are needed to establish causality and to clarify the essential mechanisms.

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