



Sleep duration and risk of stroke events and stroke mortality: A systematic review and meta-analysis of prospective cohort studies



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ABSTRACT

Background: Numerous studies have suggested the relationship between sleep duration and risks of stroke mortality and morbidity, however, the effect estimates varied substantially across studies and it remains unknown how many hours of habitual sleep are associated with the lowest risk of stroke outcomes. Therefore, we performed a dose–response meta-analysis of prospective cohort studies to evaluate the relation of sleep duration with risk of total mortality and stroke events.

Methods: PubMed and Embase databases were searched through January 2016, and multivariate-adjusted relative risks were pooled by using fixed-effects models. Semiparametric and dose–response methods were used to assess the relationship of sleep duration and risk of stroke and stroke mortality.

Results: Eleven articles with 16 independent reports were included in our meta-analysis. An approximate J-shaped relationship was detected between sleep duration and risk of stroke and stroke mortality. No evidence of a curve linear relationship was seen between sleep duration and risk of stroke or stroke mortality. Compared with 7-h sleep duration per day, the pooled relative risks for stroke events were 1.07 (95% CI 1.02–1.12) for each 1-h shorter sleep duration among individuals who slept <7 h per day and 1.17 (1.14–1.20) for each 1-h increase of sleep duration among individuals with longer sleep duration and the pooled RR for stroke mortality was 1.17 (95% CI 1.13–1.20) per 1-h increase of sleep duration.

Conclusions: Both short and long duration of sleep are predictors of stroke outcomes, and long sleep duration is significant marker of stroke mortality.

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1. Introduction

Stroke is the third commonest cause of death and a major public-health burden worldwide, which caused 3% of the world's disability burden in 1990. By 2020, stroke mortality will have almost doubled [1, 2]. Stroke is also a main cause of death in developed countries, and it is expected to become a rising health problem in developing countries as well. Given the severe consequences and accompanying burden, recognition of risk factors may have a significant role in preventing the incidence of stroke, and sleep duration has long been considered as a main contributing factor to the risk of stroke.

Increasing numbers of prospective studies have suggested that sleep duration are associated with stroke and a meta-analysis by Cappuccio et al. [3] reported that both short and long sleep durations were

associated with increased stroke events. However, the number of included studies was small, and the dose–response analysis was not conducted. Since 2010, the number of prospective studies with enough quantitative categories has rapid increased [4–14]. Therefore, we conducted a dose–response meta-analysis of prospective studies to describe the relationship between sleep duration and risk of stroke events and stroke mortality.

2. Methods

2.1. Search strategy and selection criteria

In accordance with the PRISMA guidelines [15], we identified published studies through PubMed and Embase from inception to January 29, 2016, with the following search terms without restriction: (cardiovascular diseases[MeSH] or stroke[MeSH]) and sleep. In addition, we searched the reference lists of all identified relevant original publications and relevant reviews.

Studies were identified on the basis of predefined inclusion criteria: the study design was prospective, the exposure of interest was sleep duration, the outcome was stroke or stroke mortality, and the investigators reported relative risks (RRs) with 95% CIs for at least three quantitative categories of short sleep or long sleep duration. Additionally,

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non-human studies, clinical trials, cross-sectional studies, case–control studies, reviews, letters without sufficient data, and studies that examined other associations were excluded. If study populations were reported more than once and the outcome was the same, we used the result with the longest follow-up duration. After exclusion of duplicate studies, two investigators (WZL and DMW) independently reviewed all remaining articles by titles and abstracts and then by full texts. Any discrepancy between the two investigators was solved by discussion with the senior investigator (ZXL).

2.2. Data extraction and quality assessment

We extracted the following information from each eligible article: name of the first author, study location (country), number of participants, number of stroke events or stroke mortality, mean follow-up time, participant characteristics (age and sex), method of stroke ascertainment (assessed by self-reports, death certificates, medical records, or clinical examinations), sleep duration categories, covariates included in the adjusted models, and RRs (95% CIs) for all categories of sleep duration.

Quality assessment was performed according to the Newcastle–Ottawa Quality Assessment Scale (NOS), which is a validated scale for nonrandomized studies in meta-analyses. This scale awards a maximum of 9 points to each study: 4 for selection of participants and measurement of exposure, 2 for comparability of cohorts on the basis of the design or analysis, and 3 for assessment of outcomes and adequacy of follow-up. We assigned scores of 0–3, 4–6, and 7–9 for low, moderate, and high quality of studies, respectively. When studies had several adjustment models, we extracted those that reflected the maximum extent of adjustment for potentially confounding variables.

To perform a dose–response meta-analysis, we assigned 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 assigned. When the shortest or the longest category was open-ended, we assumed that the open-ended interval length had the same length as the adjacent interval.

2.3. Statistical analysis

In this meta-analysis, the relative risk (RR) and 95% confidence intervals (CIs) were considered as the effect size for all studies, and the hazard ratios were deemed equivalent to RRs. Any results stratified by sex were treated as two separate reports. Forest plots were produced to visually assess the RRs and corresponding 95% CIs across studies. To analyze the trend of sleep duration and risk of stroke, we first used the semiparametric methods [16], in which three groups (namely longest/shortest, second longest/shortest, and reference) were generated. For each included study, the longest/shortest and the reference categories corresponded to the longest/shortest and reference groups, respectively. For studies with three categories, the second categories were considered to the second longest/shortest group. If the study had more than three categories for long or short duration, one group, other than the longest/shortest and reference, was chosen on the basis of their similarity of the duration of sleep in that category to the second longest/shortest group. For each group, a fixed/random-effects model was used to pool the RR of stroke.

And then we used a fixed-effects dose–response meta-analysis described by Greenland and Longnecker [17] to calculate the trend from the correlated estimates for log RR across categories of sleep duration. The distributions of cases and participants, and RRs and 95% CIs, in each sleep duration category were extracted according to this method. Additionally, we tested for potential nonlinearity in the association between sleep duration and stroke using restricted cubic splines with three knots at percentiles 10, 50, and 95% of the distribution [18].

The heterogeneity among studies was estimated by the Cochran Q test (reported as I^2 statistic, ranges from 0% to 100% with lower values representing less heterogeneity). According to the estimated I^2 values, inconsistency across studies was considered to be high (75% or greater), moderate (25%–75%), and low (25% or less) [19]. We used the Mantel–Haenszel fixed-effect model to pool results across studies when heterogeneity was negligible and the Mantel–Haenszel random-effect model was used when heterogeneity was significant [20].

We also conducted subgroup analyses stratified by sex, study location, number of participants and cases, duration of follow-up, and history of stroke at baseline. Publication bias was evaluated by Egger's regression test, Begger's funnel plot and nonparametric trim-and-fill method, which estimates the number and outcomes of potentially missing studies resulting from publication bias [21,22]. All statistical analyses were performed with Stata version 11 (Stata Corp, College Station, TX), and all tests were two sided with a significance level of 0.05.

3. Results

3.1. Literature search

Fig. 1 shows the results of our literature research and study selection. We identified 11,296 articles from PubMed and 10,489 articles from Embase prior to 29 January 2016. After exclusion of duplicates and studies that did not fulfill the inclusion criteria, 15 remaining articles seemed

to be relevant for this meta-analysis. After evaluating the full texts of these 15 publications, we excluded four articles as follows. One article was excluded owing to lack of sufficient data [23], and another two articles were excluded because of fewer than three categories of short sleep or long sleep duration [24,25], and one article was excluded owing to lack of 95%CI [26]. Finally, 11 articles with 16 independent reports were included in our meta-analysis. It was noted that the same study populations were reported in two articles [6,12], but the outcomes were not same, one was stroke risk [6] and another was stroke mortality [12]. When we conducted the meta analysis on risk of stroke, only the study of stroke risk was included, and when the meta analysis on risk of stroke mortality was conducted, the other was only included.

3.2. Study characteristics

Supplementary Table S1 shows the information extracted from the 11 included studies from ten cohorts, all of which were prospective cohort design. The duration of follow-up for incident stroke ranged from 3 to 18 years. Five cohorts were conducted in the Japan [6,8,9,11, 14], one in Hawaii and Los Angeles [10], one in Singapore [13], one in Europe [4], and two in USA [5,7]. Sleep duration was assessed by questionnaire in all studies. Except one article [5] assessed death through self-reported, other studies assessed death through death certificates. And for non-fatal incident cases of stroke events, one study was recorded through self-reported, others were recorded through disease registers or medical diagnosis.

3.3. Overall sleep duration and risk of stroke and stroke mortality

Ten studies with 14 reports were included to explore the overall shape of the relationship between sleep duration and risk of stroke [4–11,13,14] and six studies with 11 reports were included to explore the overall shape of the relationship between sleep duration and risk of stroke mortality [9–14]. An approximate J-shaped association was shown, with the lowest total risk of stroke and risk of stroke mortality at a sleep duration of 6–7 h per day (Fig. 2).

3.4. Short duration of sleep and risk of stroke and stroke mortality

The semiparametric analysis included seven articles with 11 reports on short sleep and stroke risk and four articles with 7 reports on short sleep and stroke risk. Fig. 3 shows the RRs for stroke with different levels of short sleep duration relative to the reference category. Compared with the reference category of sleep duration (7 h or 7–8 h per day), the pooled RR for incident stroke was 1.00 (95% CI 0.92–1.11, $I^2 = 0.0%$, P for heterogeneity = 0.86) for the second shortest (6 h or 6–7 h per day) and 1.26 (1.12–1.42, $I^2 = 0.6%$, $P = 0.44$) for the shortest (≤ 5 h per day) category of sleep duration (Supplementary Fig. 1).

The seven articles were included in the dose–response analysis of short sleep duration and risk of stroke. We found no evidence of a curvilinear association between short sleep duration and risk of stroke ($P = 0.23$ for nonlinearity). Compared with 7 h sleep duration per day, the pooled RR for stroke was 1.07 (95% CI 1.02–1.12) per 1-h reduction of sleep duration, with low heterogeneity ($I^2 = 33.8%$, $P = 0.13$) (Fig. 3).

The RRs for stroke mortality with different levels of short sleep duration relative to the reference category were shown in Supplementary Fig. 2. Compared with the reference category of sleep duration (7 h or 7–8 h per day), the pooled RR for incident stroke was 1.00 (95% CI 0.91–1.09, $I^2 = 0.0%$, P for heterogeneity = 0.86) for the second shortest (6 h or 6–7 h per day) and 1.19 (1.05–1.36, $I^2 = 0.0%$, $P = 0.78$) for the shortest (≤ 5 h per day) category of sleep duration.

The four articles were included in the dose–response analysis of short sleep duration and risk of stroke mortality. We found no evidence

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