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REVIEW

Sleep Duration and All-Cause Mortality

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Sleep, as a behavioural lifestyle factor, has so far received limited attention in medical risk assessment. Nonetheless, behavioural lifestyle factors can offer valuable insights into the health status of applicants. Health trackers enable the continuous recording of lifestyle factors such as physical activity and sleep patterns. Currently, there is a dearth of experience in incorporating such data when calculating premiums, as well as in understanding the correlation between continuously recorded lifestyle factors and mortality/morbidity. Hence, the literature was reviewed to examine the association between sleep duration and all-cause mortality to derive dose-response rates. Relative risks were calculated by pooling data from 10 selected studies comprising over 3 million study participants. The findings suggest that both short (<6 hours) and long sleep duration (>9 hours) are associated with an increased risk of all-cause mortality.

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Lifestyle choices can significantly impact health, quality of life, and life expectancy. Unhealthy lifestyle factors, such as poor diet, lack of physical activity, and smoking, have been linked to the development of various diseases including cancer, coronary heart disease, and diabetes, which are among the primary causes of death in developed countries.[1] Life insurance companies use standardized procedures to assess risks and determine rate calculations, which take unhealthy lifestyle factors such as smoking, alcohol consumption, and higher BMI into account. Yet, how much consideration is given to behavioural factors like adequate sleep and exercise? These have received little attention in the application process to date. One reason for this is the challenge of reliably documenting and collecting relevant data. However, health trackers/smart watches are opening new possibilities; healthy lifestyles can be determined by continuously recording heart rate, O_2 consumption, sleep duration or step count. In recent years, insurance companies have started implementing approaches that utilize "wearables" to incentivize healthy lifestyles among their policyholders, often by offering vouchers as rewards.[2] The challenge for insurers lies in accurately translating policyholders' healthy lifestyle information into tangible numerical adjustments that can be made to their premium calculations. Currently, there is a shortage of experience in utilizing this data, as well as insufficient quantified information on the correlation between continuously recorded lifestyle factors and mortality/morbidity. Numerous scientific observational studies have been conducted over the past decades to explore the correlation between lifestyle factors and mortality/morbidity, providing a general overview and preliminary estimation of the association between selected lifestyle factors and mortality/morbidity. Quantifiable lifestyle factors such as sleep duration, physical activity and step count are most suitable for summarizing available evidence in a short report. This article examines one such factor: sleep duration and its impact on mortality.

Sleep plays a crucial role in maintaining mental and physical health, as essential processes such as muscle repair, tissue growth, hormone release occur during this period, and for optimal cognitive performance.[3-5] Adequate sleep is necessary to boost the immune system, fight inflammation, and infections.[6] The National Sleep Foundation recommends for young adults and adults between 7 to 9 hours of sleep per night, and for older adults 7 to 8 hours.[7] Growing evidence over the past few decades links inappropriate sleep duration to adverse health outcomes, including coronary heart disease, cognitive disorders, and stroke.[8] The aim of this brief report is to summarize the available evidence and establish a dose-response relationship between nighttime sleep duration and all-cause mortality.

METHOD

Eligible systematic reviews and meta-analyses were searched in NCBI PubMed between January 2010 and January 2023. The reported results were summarized following a process similar to an umbrella review.[9] A total of 97 reports were identified in the initial search. After title and abstract screening and reviewing full-text papers for eligibility, 15 reviews[10-24] were identified based on the following criteria:

- The exposure of interest was nighttime sleep duration.
- The outcome measure was all-cause mortality.
- Reported results were expressed as relative risk, hazard ratio, or odds ratio.
- Reported results were presented as dose-response ratios.

The studies utilized in the 15 systematic reviews identified are consistent by more than 80%. This overlap made it impractical to conduct an umbrella review (defined as a review of systematic reviews or meta-analyses in medical research, allowing comparison of results between individual reviews, useful for developing guidelines), as the same findings would have been accounted for multiple times in the final analysis, leading to a biased outcome. For this reason, the systematic review with the most prospective cohort studies included was selected.[9] Considering this, an additional literature search was carried out in NCBI PubMed to identify further large prospective cohort studies (>10,000 study participants) published between January 2010 and January 2023. A total of 250 publications were initially identified. After abstract screening, title review, and full-text assessment, 9 additional prospective cohort studies[25-33] were selected, which are not cited as primary literature in the selected systematic review.[10] All studies included in the final analysis utilized self-reported data to document sleep duration, gathered through either questionnaires or interviews. For each eligible study, detailed information was extracted including the publication year, effect size, sex and age of participants, follow-up period (FUP) in years, population size, number of all-cause mortality cases, and the country where the study was conducted. The extracted effect sizes (and 95% CIs) were weighted based on the reported study population size to calculate the pooled effect size. Moreover, only the reported adjusted values, such as age, sex, socioeconomic status, education level, underlying diseases (eg, diabetes, hypertension, mental health), and smoking status, were considered while computing the pooled effect size.

RESULTS

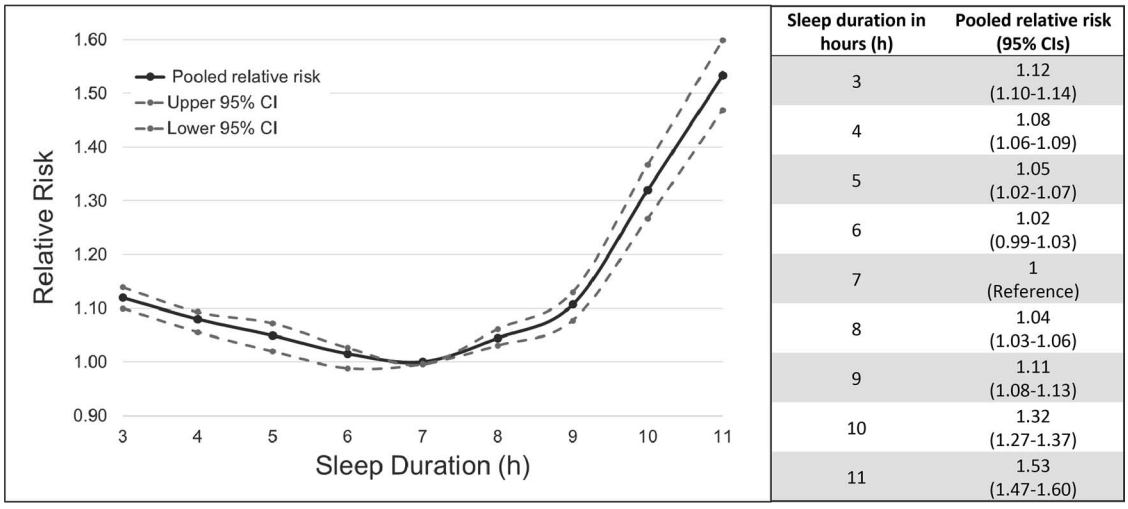
Table 1 provides an overview of the key characteristics of the 10 studies included in the analysis, comprising 9 prospective cohort studies (PCS) and 1 systematic review (SR). In total, these studies enrolled 3,268,565 participants, and documented a total of 341,254 cases of all-cause mortality, as outlined in Table 1. Notably, the follow-up period varied considerably across the included studies, spanning from 3 to 35 years, and the age range of participants at the onset of the study was also

Table 1. Detailed Information of Included Publications

Author	Study Type	Population Size	Sex	FUP in Years	Number of Cases	Age in Years	Country
Yin et al. 2017	SR*	2,446,116	m/f	3.0–35.0	241,107	>18	Europe, Australia, US, Asia
Kabat et al. 2018	PCS	158,203	f	17.8 (median)	30,400	50–79	US
Svensson et al. 2021	PCS	144,179	m	14.0 (mean)	19,419	53.6 (mean)	East Asia**
Svensson et al. 2021	PCS	178,542	f	13.4 (mean)	13,768	54.5 (mean)	East Asia**
Omichi et al. 2022	PCS	81,382	m/f	9.1 (mean)	3,376	35–69	Japan
Bai et al. 2021	PCS	13,441	m/f	6.0	7,849	>65	China
Ren et al. 2020	PCS	15,092	m/f	4.5 (mean)	10,768	89.3 (mean)	China
Kwon et al. 2020	PCS	36,264	m/f	9.5 (mean)	1,028	>20	Korea
Wang et al. 2019	PCS	116,632	m/f	7.8 (mean)	4,381	35–70	WW
Akerstedt et al. 2017	PCS	39,191	m/f	13 (mean)	3,548	>18	Sweden
Soh et al. 2018	PCS	39,523	m/f	12.7 (mean)	5,610	54.8 (mean)	Singapore
Total		**3,268,565**			**341,254**		

SR: systematic review; PCS: prospective cohort study; m: male; f: female, FUP: follow up period; WW: worldwide.
* includes 43 articles providing statistical effects relevant to the meta-analyses on all-cause mortality; **Japan, China, Singapore, and Korea.

quite diverse. The geographic distribution of the studies was global in nature, with the SR encompassing various countries worldwide. Similarly, the PCS were conducted worldwide, with well represented East Asia regions. Figure 1 displays the results of the analysis, with the pooled relative risk (RR) plotted on the y-axis and sleep duration in hours (h) on the x-axis. The reference value for the analysis was set at 7 h of sleep (RR of 1.00). Notably, a "J-shaped" association was observed between sleep duration and all-cause mortality risk, with individuals who slept for less than 7 h having a lower relative risk compared to those who slept for longer periods (>7 h). In comparison with the reference value (7 h), a more pronounced increase in the mortality risk was observed among those who slept for a longer duration [11 h: RR of 1.53 (95% CI: 1.47-1.60)] relative to those who slept for a shorter period [3 h: RR of 1.12 (95% CI: 1.10-1.14)].



Sleep duration in hours (h)	Pooled relative risk (95% CIs)
3	1.12 (1.10-1.14)
4	1.08 (1.06-1.09)
5	1.05 (1.02-1.07)
6	1.02 (0.99-1.03)
7	1 (Reference)
8	1.04 (1.03-1.06)
9	1.11 (1.08-1.13)
10	1.32 (1.27-1.37)
11	1.53 (1.47-1.60)

Non-linear dose-response between the risk of all-cause mortality and sleep duration (h). Relative risks and 95% CIs are weighted according to the population size of the studies included in the analysis. 7 h is the reference value.

Table 2. Reported Effects of Abnormal Sleep Duration on Metabolic and Endocrine Hormones, and Inflammatory Markers

Short Sleep Duration (<6)	Long Sleep Duration (>9)
Higher levels of: • total cholesterol[22] *(associated with increased risk of all-cause mortality)* • calcium[22] *(associated with increased risk of all-cause mortality)* • ghrelin[10,20,29] *(associated with obesity, diabetes, impaired glycemic control)* • interleukin-6[15] *(associated with inflammation, infection, CVD)* • cortisol[20,29] *(associated with altered growth hormone metabolism and activation of low-grade inflammation, increase CVD)*	**Higher levels of:** • glucose[22] *(associated with increased risk of all-cause mortality)* • calcium[22] *(associated with increased risk of all-cause mortality)* • creatinine[22] *(associated with increased risk of all-cause mortality)* • interleukin-6[15,24] *(associated with inflammation, infection, CVD)* • tumor necrosis factor[23] *(associated with inflammation, increased risk of all-cause mortality)* • c-reactive protein[15,24] *(associated with chronic inflammatory processes)*
Lower levels of: • leptin[10,20] *(associated with obesity, diabetes, impaired glycemic control)* • testosterone[10] *(associated with increased risk of all-cause mortality and CVD)* • melatonin[10,11,34] *(associated with increased risk of all-cause mortality and CVD, cancer risk)*	**Lower levels of:** • lactate dehydrogenase[22] *(associated with increased risk of all-cause mortality)* • cholinesterase[22] *(associated with increased risk of all-cause mortality)* • alkaline phosphatase[22] *(associated with increased risk of all-cause mortality)*

DISCUSSION

Various scientific publications have discussed the potential mechanisms that could contribute to the relationship between sleep duration and mortality. Table 2 summarizes the adverse effects of sleep duration on metabolic and endocrine hormones, as well as inflammatory markers. Further details about some reported associations are briefly explained in the following section.

Insufficient sleep was reported to be linked to lower leptin (regulates hunger by signaling feelings of satiety) and higher ghrelin (stimulates appetite by signaling the stomach is empty, leading to increased food intake and promoting fat storage) serum levels. These hormonal changes can lead to alterations in appetite and contribute to the development of obesity, which is a significant predictor of cardiovascular disease (CVD).[10] In addition, short sleep duration is associated with impaired glucose metabolism (insulin resistance) and an increased risk of weight gain, obesity, and diabetes.[25]

Both prolonged and short sleep durations have been found to affect the levels of inflammatory markers such as interleukin, tumor necrosis factor, and C-reactive protein, which are known risk factors for cancer and CVD.[10] Li et al investigated the relationship between sleep duration and multiple blood biochemical parameters (listed in Table 2). The authors found that sleep duration had a significant impact on these biochemical levels.[22] However, it is currently unclear how these altered biochemical parameters are associated with all-cause mortality. In addition, the level of the sleep hormone, melatonin, is also influenced by sleep duration, with short sleepers having decreased levels of melatonin,[11,34] which may be linked to mortality or cardiovascular events.[10] Conversely, long sleepers tend to have increased melatonin levels, which may contribute to longer sleep durations.[34]

The potential mechanisms underlying the relationship between long sleep duration and mortality remain speculative. It is likely that poor sleep quality, waking after sleep onset,

or sleep latency induce sleep extension.[12,34] Additionally, sleep fragmentation has been linked to excessive time spent in bed and has been associated with various negative health outcomes.[10,26,34,35] Feelings of fatigue and lethargy are also commonly reported by long sleepers, which may increase susceptibility to stress-related disorders such as depression.[10-12,26,27,34,35] Another possibility is that long sleepers experience a shortened light-day cycle (photoperiod), which has been linked to mortality in other mammalian species.[10,12,26,27,34] In addition to these potential biological mechanisms, it is also believed that the observed associations between long sleep duration and mortality may be influenced by residual confounding[28,34] as well as mediators for subhealthy status,[10] subclinical markers for undiagnosed diseases[29,35] or a mediator between socioeconomic status and mortality.[13]

LIMITATIONS

In the context of this short report, the effect size was weighted based on population size, but this approach has certain limitations. For instance, this method did not consider the quality of the studies, the distribution of sex and age, or the frequency of countries, meaning that the study with the largest population group influence the outcome most. Furthermore, different studies used varying cutoff levels of sleeping hours (<4 h, <5 h, >8 h, >9 h, etc.) and defined different reference values for sleep duration (eg, 7 h, 7-8 h, or 8 h). Therefore, an exact derivation of the dose-response relation is only possible with certain restrictions. It is important to consider that the data collected relied on self-reported questionnaires or interviews, which could lead to misclassification due to participants reporting time spent in bed instead of actual hours of sleep.[34] This could result in an over- or underestimation of mortality risk. Furthermore, studies have reported that sleep duration measured by polysomnography or actigraphy is often shorter than self-reported sleep.[36]

This analysis employs a semi-systematic literature search, considering only publications from NCBI PubMed. As a result, publications that met the search criteria may not have been included in the analysis, potentially distorting the results.

Another aspect to consider is that this report solely focuses on nighttime sleep, without taking daytime sleep into account. Further investigations into this association, such as the umbrella review conducted by Sun et al (2022),[37] indicate that long daytime napping (>1 hour per day) is associated with higher all-cause mortality among adults compared to short daytime napping (<1 hour per day).

It would also be of interest to conduct a more stratified analysis of the association between sleep duration and mortality across different age groups, as the recommendations of the National Sleep Foundation vary depending on age. As part of these analyses, only adjusted estimators (eg, age and gender) were extracted from the literature.

CONCLUSION & OUTLOOK

Emerging evidence supports the association of both long (>9 h) and short (<6 h) sleep durations with various adverse health outcomes. However, further high-quality, large cohort studies are essential to establish the relationship between changes in biochemical parameters and sleep duration.

What are the implications of this evidence for underwriters? As discussed in the ReCent Medical News published in 2019,[38] the focus in medical risk assessment is primarily on biomedical data and less on behavioural health factors. However, recent evidence suggests that sleep duration can provide an indication of an applicant's current health status. Accordingly, with further investigations into the association between sleep duration and biomarkers, it could be suggested that sleep may serve as a proxy for the current state of health. This could simplify risk assessment by reducing health-related questions, medical examinations, and analysis of diverse biomarkers.

How could such data be collected? As mentioned, smartwatches are suitable data sources. It should be noted here that the reliability and validity of each tracker differ greatly and must be taken into account when using wearable data.[39] Studies have explicitly investigated the reliability and validity of sleep duration as measured by health trackers.[39-43] The sample size in the respective studies is relatively modest; however, the results suggest that the quality of the data is contingent upon the specific device utilized. However, a recent prospective study analysed the association of insufficient sleep quantity/quality with chronic disease incidence by using both, data from commercial wearable devices and electronic health record (EHR).[44] The results demonstrate that the integration of EHR and wearable data enables the examination of the interrelationships between behavioural lifestyle factors and health outcomes. Furthermore, this approach has the potential to serve as an early diagnostic indicator. An additional study examined the optimal duration for which sleep behaviour should be recorded consecutively to ensure the reliability for estimating the weekly total sleep time (3 to 5 nights of tracking) and for monthly mean sleep duration (5 to 10 nights of tracking).[45]

However, collecting and analysing such data involves a great deal of effort, such as building data infrastructure (eg, databanks, data pipelines). Instead of collecting data prospectively, a simple approach could be to summarize the data retrospectively: consider already recorded data and derive the average sleep duration, for example, over the last 2 weeks based on wearable data. This information could then be compared with the relative risks listed in Figure 1.

How could sleep duration be rated? The values presented in Figure 1 can serve as reference points for assessment. An average sleep duration between 5 to 8 hours could be rated as preferred standard, or a discount (in whatever form) can be offered. Another option could be to assign a loading of 25% to applicants with an average sleep duration above 9 hours and below 5 hours. However, it is probably more appropriate to reward a healthy lifestyle than to punish an unhealthy one.

Given the limited experience in this area, the primary goal should be to expand knowledge and gather data on lifestyle factors alongside insurance data.

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