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Associations Between Self-Reported Sleep Duration and Mortality in Employed Individuals: Systematic Review and Meta-Analysis

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Abstract

Objective: Sleeping less or more than the 7-8 h has been associated with mortality in the general population, which encompasses diversity in employment status, age and community settings. Since sleep patterns of employed individuals may differ to those of their unemployed counterparts, the nature of their sleep-mortality relationship may vary. We therefore investigated the association between self-reported sleep duration and all-cause mortality (ACM) or cardiovascular disease mortality (CVD) in employed individuals.

Data sources: Based on Preferred Reporting Items for Systematic Reviews and Meta-Analyses, searches between January 1990 and May 2020 were conducted in PubMed, Web of Science and Scopus. Inclusion/exclusion criteria: Included were prospective cohort studies of 18–64-year-old disease-free employed persons with sleep duration measured at baseline, and cause of death recorded prospectively as the outcome. Gray literature, case-control or intervention design studies were excluded.

Data Extraction: Characteristics of the studies, participants, and study outcomes were extracted. The quality and risk of bias were assessed using the Newcastle-Ottawa Scale.

Data synthesis: The pooled relative risks (RR) with 95% confidence intervals (CI) were obtained with a random-effects model and results presented as forest plots. Heterogeneity and sensitivity analysis were assessed.

Results: Shorter sleep duration (≤ 6 h) was associated with a higher risk for (ACM) (RR: 1.16, 95% CI: 1.11–1.22) and CVD (RR: 1.26, 95% CI: 1.12–1.41) compared to 7-8 h of sleep, with no significant heterogeneity. The association between longer sleep (≥ 8 h) and ACM (RR: 1.18, 95% CI: 1.12–1.23, $P < 0.001$) needs to be interpreted cautiously owing to high heterogeneity ($I^2 = 86.0\%$, $P < 0.001$).

Conclusion: Interventions and education programs targeting sleep health in the workplace may be warranted, based on our findings that employed individuals who report shorter sleep appear to have a higher risk for ACM and CVD.

Keywords

sleep quantity, short sleep, heart disease, employed, workplace

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Objective

Non-communicable diseases (NCDs) account for about 71% of deaths globally among working-aged adults (35–70 years), with cardiovascular disease (CVD) being the leading cause of death (31%).¹ Modifiable risk factors for NCDs include an unhealthy diet, physical inactivity, alcohol use and tobacco smoking. The World Health Organization's (WHO) global action plan aims to manage these risks so that NCD-related deaths can be reduced by 25% by the year 2025.² The workplace has been identified as a key setting for public health action, in which diet and physical activity interventions are primarily used to address NCD risk.³

Suboptimal sleep (poor quality, insufficient duration and/or mis-timed) has emerged as an important modifiable risk factor for cardiovascular and metabolic diseases.^{3–8} Notwithstanding the importance of sleep timing and quality, we will focus on sleep duration in this study given that much of the prospective work relating sleep to mortality relies on sleep duration as an outcome variable. Guidelines have been published recommending that adults attain 7–9 h⁹ or at least 7 h¹⁰ of sleep per night for optimal physical and mental health. Acknowledging that not all studies conclusively observe associations between sleep duration and health outcomes, longitudinal evidence from systematic reviews and meta-analyses indicates that individuals with shorter and longer sleep durations are more likely to experience poorer health outcomes compared to those clustered around the group median (typically 7–8 h).^{11–15} While organizations such as the American Heart Association and the Center for Disease Control and Prevention promote healthy sleep awareness,^{16,17} sleep is yet to be included as one of the WHO's targeted modifiable risk factors that could enhance health and prevent NCDs.² Thus it is unsurprising that although workplace health programs, typically delivered in urban settings, are becoming more commonplace, sleep improvement rarely feature as an intervention.

Working hours, commuting time and even the nature of urban employment may impact sleep. Work has been shown to be the dominant waking activity in employees who sleep ≤ 6 h per night.¹⁸ Adults with longer working hours are more likely to have shorter sleep durations^{4,6} such that for every additional hour spent working beyond the standard eight-hour day, sleep is shortened by about 2 hours.⁴ Commuters traveling more than 75 minutes may also sleep less compared to those whose commute is under 45 minutes^{6,7} and one study showed that for every hour of commuting, sleep was shortened by 15 minutes.¹⁹ Furthermore, employees in the manufacturing sector, routine and semi-routine occupations (e.g. cleaners, receptionists) and in managerial roles have been shown to sleep less than those in other occupational groups.^{6,8} Therefore, extended working hours and fixed work schedules that demand consistent wake-times may largely contribute to shorter sleep duration in employed adults. On the other hand, unemployment has been linked to at least a 2-fold increase in longer sleep duration (>9 h) compared to those sleeping 7–8 h. These studies postulated that the sleeping patterns of unemployed individuals

are not impinged upon by a fixed work schedule; thus having the opportunity to sleep more than employed individuals.^{20,21}

One might speculate that employees working longer hours might sleep less and be at increased risk for CVD, all-cause mortality (ACM) or CVD mortality (CVDM). In a meta-analysis including data from 24 occupational cohort studies, employees working >54 h per week had a 30% higher risk for stroke and a 13% higher risk for CVD compared to those who worked 35–40 h.²² Similarly, the incidence of diabetes was higher among women working >45 h per week compared to those working 35–40 h.²³ One study has shown a J-shaped relationship between sleep duration and number of health risk factors in employees, with those reporting ≤ 5 h per night likely to present with the most risk factors, and those sleeping 7–8 h per night with the fewest risk factors.²⁴ It was interesting to note that employees sleeping ≥ 9 h per night were at greater risk for hypertension, depression, heart disease and cancer compared to those sleeping 7–8 h,²⁴ possibly suggesting a link between underlying disease and sleep duration. Since, to the best of our knowledge, no studies have included working hours, sleep duration and NCD, CVDM or ACM risk in their analyses, it is unclear whether shorter sleep associated with employment translates to increased risk for NCDs or mortality.

Although systematic reviews examining the literature pertaining to sleep duration and mortality have been published,^{25–30} none have presented the magnitude of these associations exclusively in employed populations. Since the sleep patterns of employed individuals may well differ from those of unemployed persons, which may in turn alter the nature of the sleep-CMD or sleep-mortality relationship, we see value in focusing exclusively on employed individuals, living in urban areas and younger than 65 years. Even though studies have shown that sleep health can be promoted in the workplace,^{31–33} some organizations feel there is insufficient evidence to make sleep a key component of their workplace policies and health promotion programs. Data emanating from studies like ours may help advocate for the inclusion of sleep health in employee wellness programs. Therefore, the aim of this systematic review and meta-analysis was to investigate the association between sleep duration and risk for all-cause mortality (ACM) and cardiovascular disease mortality (CVDM) in employed individuals.

Methods

Data Sources

The study was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.³⁴ A standardized search strategy was developed *a priori* to identify published cohort studies that quantified the relationship between sleep duration and risk for ACM and CVDM in employed populations. Systematic literature searches were conducted in 3 databases: PubMed, Web of Science and Scopus. The search strategy included terms characterizing the exposure (e.g. sleep), the outcome (e.g. mortality), and the type of study design (e.g. cohort studies). Details of the

complete search terms can be found in Supplementary Table S1. Reference lists of included articles were also searched for eligible studies.

Inclusion and Exclusion Criteria

The following inclusion criteria were applied: (i) original articles published between January 1990 and May 2020 in English, (ii) prospective cohort study designs, (iii) employed individuals (where employment and unemployment data were presented separately, only data from employed groups were included), 18–65 years and disease-free at baseline from urban settings (iv) sleep duration assessed at baseline, measured as either total sleep duration over a 24 h period (nocturnal and daytime sleep) or exclusively during night-time (nocturnal sleep), (v) cause of death recorded prospectively as outcome, (vi) follow-up of at least 4 years, and (vii) at least 3 sleep duration categories were reported, e.g. ≤ 6 h, 7–8 h and ≥ 9 h per night. CVDM was defined in accordance with ICD-9 codes 390–459 or ICD-10 codes I00–I99. If insufficient data were reported (e.g. absence of relative risk (RR), confidence intervals (CI) or number of cases) additional information was requested from the authors of studies eligible for inclusion. Studies were excluded if (i) a case-control or intervention design was used, (ii) relevant statistical data could not be accessed or (iii) the outcome was not adjusted for potential confounders (examples include: age, ethnicity, gender, marital status, education, social class (i.e. education level and job position,³⁵ or the individual's own current occupation³⁶), employment grade (i.e. administrative, professional and executive, or clerical job), lifestyle characteristics (e.g. smoking, physical activity, alcohol consumption), medical conditions (e.g. diabetes, depression, hypertension, cancer, obesity) or medications. Conference abstracts, other published abstracts, and gray literature (e.g. reports, white papers, academic theses or dissertations) were not included in this meta-analysis.

Data Extraction

Three authors (DER, TKA and PRP) independently screened titles and abstracts, assessed full-texts for eligibility, and extracted the following data from included studies: (i) study characteristics (author name, publication date, cohort name, design, setting, sleep exposure, start date, end date and duration of follow-up period, inclusion and exclusion criteria, confounders), (ii) participant characteristics (sample size, age range, gender, race and/or ethnicity, disease status at baseline), and (iii) study outcomes (ACM, CVDM, total number of participants and number of deaths at each level of exposure, multivariate adjusted effect estimates, e.g. RR and 95% CIs).

DER, TKA and PRP assessed the quality and risk of bias of included studies using the Newcastle-Ottawa Scale (NOS).³⁷ The NOS has 3 broad categories (patient selection, comparability of study groups, and assessment of the outcome) and provides a maximum total score of 9. Disagreements were resolved through discussion and consensus and NOS scores for each study are included in Table 1.

Data Synthesis

The exposure variable used in this meta-analysis was self-reported sleep duration, measured at baseline of each included study. The manner in which each included study assessed sleep duration is defined in Table 1. The included studies varied in their sleep duration categories for which ACM or CVDM statistics were reported (Supplementary Table S2), with 3 studies presenting statistics on sleep duration in 3 categories^{35,36,38} and 2 studies presenting data on 5 categories.^{39,40} Four of the 5 studies used a reference sleep duration category in the range of 6–8 h (Ferrie, Garde, Heslop, Patel) while Rhee et al (2012) originally used their ≥ 8 h group as their reference category. For our meta-analysis, we used the ACM and CVDM statistics in their original form for the 3 studies reporting on 3 sleep categories (Garde, Heslop, Rhee), but we re-referenced the data for Rhee et al to their 6–7 h category, to better match the other studies.³⁸ For the 2 studies in which authors reported 5 sleep duration categories, we collapsed those categories less than the reference group into a single “shorter sleep” category and those longer than the reference group into a single “longer sleep” category (Supplementary Table S2). Primary outcome variables were ACM and/or CVDM RR with 95% CIs per exposure. The most covariate-adjusted risk estimates were used in the meta-analysis. Men and women were considered independently among studies that reported gender-specific results. The pooled RRs with 95% CIs were obtained with a random-effects model and results presented as forest plots.

Heterogeneity among studies was assessed by the I^2 statistic, which represents the total variability that is attributed to between-study variability.^{41,42} When heterogeneity was significant, sensitivity analyses was performed to identify the influence of individual studies on the magnitude of the pooled estimate. This was achieved by omitting the studies with the greatest weight, one at a time, and examining the extent to which inferences were dependent on a particular study. Publication bias was not assessed as there were inadequate numbers of included studies to properly measure funnel plot asymmetry. Data were analyzed using Stata v15 (StataCorp, Texas, USA). Significance was assumed for $P < 0.05$.

Results

Search Results and Study Characteristics

After identification, exclusion of duplicates and screening, 89 full text articles were assessed for eligibility. Eighty-four studies failed to meet the inclusion criteria and were excluded, leaving 5 studies eligible for inclusion and analysis (Figure 1). The general characteristics of the included studies are presented in Table 1. The 5 studies analyzed for ACM comprised 116 969 participants from 5 countries: 2 studies included only men,^{35,38} one included only women³⁹ and 2 reported data on men and women combined or separately.^{36,40} The 3 studies analyzed for CVDM comprised 19 467 participants from 3 countries. One of these studies combined their analysis to include men and women,⁴⁰ one study reported only on a male

Table 1. Description of Studies Included in the Meta-Analysis.

Author, year	Country	Cohort	Year at baseline	Gender	Sample size	Deaths (ACM)	Deaths (CVD ^M)	Follow-up (y)	Mean age (range, y)	Quality score [†]	Sleep exposure assessment	Outcome assessment	Adjusted variables
Ferrie, 2007	England	Whitehall II	1985-1988	Men & Women	7729	292	89	4-8	35-55	7	Questionnaire: "How many hours of sleep do you have on an average week night?" Response options: ≤5 h, 6, 7, 8 and ≥9 h.	Death certificate	age, sex, marital status, employment grade, smoking status, PA, alcohol consumption, self-rated health, BMI, SBP, TC, physical illness, modified GHQ score, prevalent CHD
Garde, 2013	The Netherlands	Copenhagen Male Study	1970-1971	Men	4941	2663	587	30	48.9 (40-59)	8	Questionnaire: Participants reported their daily number of hours of sleep Response options: <6 h, 6-7, 8-9, and >9 h.	National Registers	age, BMI, SBP, DBP, diabetes, HT, physical fitness, alcohol use, smoking, LTPA, social class
Heslop, 2002	Scotland	Scottish workplaces	1970-3	Men Women	5819 978	2303 262	1182 117	25	≤65	7	Questionnaire: "How many hours in 24 do you sleep?" Response option: Hours as a continuous variable	Death certificate	age, marital status, social class, risk
Patel, 2004	USA	Nurses' Health Study	1986	Women	82969	5409	1084	14	53.4 (30-55)	6	Questionnaire: "Indicate total hours of actual sleep in a 24-hour period." Response options: ≤5 h, 6 h, 7 h, 8 h, 9 h, 10 h, or ≥11 h.	National Death Index	age, smoking, alcohol consumption, PA, depression, snoring, BMI, history of cancer, CVD, HT, diabetes, and shift work
Rhee, 2012	South Korea	The Seoul Male Cohort Study	1993	Men	14533	990	—	15	49.5 (49-59)	7	Questionnaire: For sleep hours, respondents were asked to report the usual number of sleep hours, including naps. Response options: Hours as a continuous variable	Death certificate	age, education, smoking, alcohol, self-reported hypertension and diabetes

ACM: all-cause mortality; CVD^M: cardiovascular disease mortality; PA: physical activity; BMI: body mass index; SBP: systolic blood pressure; TC: total cholesterol; GHQ: general health questionnaire; CHD: coronary heart disease; DBP: diastolic blood pressure; HT: hypertension; LTPA: leisure time physical activity; CVD: cardiovascular disease; † Newcastle-Ottawa Scale.

Figure 1. Flowchart of the records retrieved, screened and included in the systematic review.

cohort³⁵ and another analyzed men and women separately.³⁶ The studies used either death certificates or national death registries to determine number of deaths in each cohort. Follow-up years for ACM and CVDM ranged from 4-30 years, and total NOS scores ranged from 6-8 (average of 7) out of a possible score of 9. All studies originated from the USA, Europe or South Korea and used questionnaires to obtain self-reported 24h^{35,36,38,39} or night-time⁴⁰ sleep duration (h). The reference sleep duration ranged from 6-8 h, shorter sleep ranged from <6 to <7 h and longer sleep from ≥ 8 to >8 h (Supplementary Table S2).

Sleep Duration and All-cause Mortality

In the pooled analysis, shorter sleep duration was associated with a 16% greater risk for ACM relative to the reference sleep duration ($P < 0.001$, Figure 2). Although long sleep duration appeared to be associated with an 18% greater risk for ACM compared to the 6-8 h sleep duration ($P < 0.001$, Figure 3), high

heterogeneity ($I^2 = 86.0\%$, $P < 0.001$) and limited number of studies suggest that this pooled estimate was not robust and therefore unlikely to be associated with ACM.

Sleep Duration and Cardiovascular Disease Mortality

In the pooled analysis, shorter sleep duration was associated with a 26% greater risk for CVDM relative to the reference sleep duration ($P < 0.001$, Figure 4) and heterogeneity was non-significant (45.8%, $P = 0.136$). There was no association between longer sleep duration and risk for CVDM ($P = 0.193$, Figure 5).

Sensitivity Analyses

All-cause mortality. Sensitivity analyses were conducted by omitting the study with the largest weight to the pooled analyses. Removing Patel et al (2004) (relative weight: 61.09%) from the analysis between shorter sleep duration and ACM increased the

Figure 2. Forest plot of the risk of death (all-cause) associated with shorter sleep duration compared to the reference of 6-8 h in 5 population cohorts from 4 published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

relative risk (RR: 1.19, 95% CI: 1.11 -1.27, $P < 0.001$) with no heterogeneity. Patel et al had the greatest relative weight (53.3%) for the longer sleep duration analysis and when omitted, relative risk for ACM (RR: 1.02, 95% CI: 0.96 - 1.09, $P = 0.558$) and heterogeneity decreased and were no longer significant.

Cardiovascular disease mortality. Sensitivity analysis was not conducted for the association between shorter sleep duration and CVDM, because heterogeneity was not significant ($I^2 = 45.8\%$, $P = 0.136$). Furthermore, sensitivity analysis was not needed for longer sleep duration as it was not associated with CVDM.

Discussion

To the best of our knowledge, this is the first systematic review to describe the association between self-reported sleep duration and ACM and CVDM in cohorts of employed persons younger than 65 years of age living in urban settings. The main finding was that compared to the reference sleep duration group, those reporting shorter sleep duration had a 16% greater risk for ACM and a 26% greater risk for CVDM.

The association between short sleep duration and ACM has previously been reported in the general population.^{25-27,29,30} Compared to such systematic reviews in which the same reference of 7-8 h was used, shorter sleep had lower relative risks than the present study. For example, one review reported a RR

of 1.13 (95% CI: 1.09 -1.17) for short sleep relative to a reference of 7-8 h, but had an extreme definition for short sleep duration (≤ 4 h).³⁰ In contrast, the present review showed that by sleeping as little as 1 hour less than the recommended sleep duration of 7-9 h, employees may have a significantly increased risk for ACM. In another review using the same reference range of 7-8 h, shorter sleep duration was significantly associated with ACM, but the RR of 1.07 (95% CI: 1.03 -1.11) included only elderly individuals,²⁸ while the present study excluded all cohorts older than 65 years.

Our finding that shorter, but not longer, sleep duration is associated with higher risk for CVDM contrasts with one previous study²⁸ that showed a 43% increased risk for long sleep duration and CVDM but no association between short sleep and CVDM. Perhaps age contributes to these disparate findings since we report on working-aged adults (<65 years) while da Silva et al (2016) reported on older individuals (> 60 years). Given the relationship between age and mortality, it is unsurprising that longer sleep duration is more strongly associated with ACM and CVDM in older cohorts.^{29,43,44}

Another study found an association between short sleep and CVDM in women, but not men, although both sexes showed an association between long sleep and CVDM compared to 7 h. However, extreme sleep duration criteria of ≤ 4 h and ≥ 10 h were used, and participants were exclusively of Asian ethnicity.⁴⁵ Socioeconomic status (SES) may affect the sleep duration and CVDM relationship. Studies report that disadvantaged

Figure 3. Forest plot of the risk of death (all-cause) associated with longer sleep duration compared to the reference of 6-8 h in 5 population cohorts from 4 published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

groups who are influenced by factors such as poverty and unemployment are more likely to have shorter or longer sleep compared to the recommended 7-9 h.^{46,47} Grandner et al (2010) hypothesized that sleep may be the mediator between SES and mortality.⁴⁸ We were not able to account for SES in the present study, but future work on this topic would benefit from sub-analyses accounting for SES.

Potential pathways through which short sleep duration may increase risk for ACM include disruption of physiological regulatory pathways,⁴⁹ including those that regulate metabolism,^{50,51} immunity, inflammation, appetite and cardiovascular health.⁵² These disruptions may manifest as CMD risk factors, such as hypertension, hyperglycaemia and obesity, all of which are associated with mortality.^{48,53,54}

The observation that longer sleep duration was associated with an 18% greater risk for ACM compared to an average of 6-8 h must be interpreted cautiously since there was high heterogeneity within the pooled estimate, heavily influenced by the Patel et al (2004) study. Despite the high heterogeneity, the present findings show that the direction of the associations are predominantly toward increased risk for most of the analyzed cohorts. Identifying heterogeneity can be helpful in interpreting and explaining study results, and for the planning of future studies. We therefore view this finding as exploratory, as presently there are insufficient eligible studies to investigate the sources of such wide variation through sub-group analyses.

One might speculate that the high heterogeneity may be attributed to factors such as sex or the definition of long sleep duration. For example, there were 4 times more women than men in this meta-analysis (women: $n = 83\,947$; men: $n = 19\,474$) for long sleep duration and ACM. We can only speculate that this disproportionate representation of sex may have contributed to the significantly increased risk for ACM among the long sleepers, as previous studies have shown that women sleeping longer than 9 h may have a higher risk for ACM compared to men.⁵⁵ In contrast, 3 other systematic reviews found no effect for sex on the sleep duration and ACM risk relationship,^{29,43,56} apart from a greater risk for obesity in longer sleeping women.⁴³ Since long sleep duration may impair whole-body metabolism, increasing risk for obesity and T2DM,⁵⁷ the nature of risk may vary between men and women and requires further investigation.

Among the 5 included studies, longer sleep was defined as either ≥ 8 h or ≥ 9 h per night. Given that current sleep duration guidelines indicate that 7-9 h of sleep are recommended for optimal health,^{9,10} longer sleep defined as ≥ 8 h may not be long enough to adversely affect health. Explanations for why longer sleep may increase risk for ACM are debatable. Long sleep may be a response to poor sleep quality (i.e. fragmented sleep), high daytime sleepiness, elevated inflammatory markers or underlying conditions such as sleep disorders (e.g. obstructive sleep apnoea) or CVD.⁵⁸ One might hypothesize that perhaps individuals with longer sleep (≥ 9 h) unwittingly

Figure 4. Forest plot of the risk of death (cardiovascular disease) associated with shorter sleep duration compared to the reference of 6-8 h in 4 population cohorts from 3 published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

increased their sleep duration in response to a pre-existing, undiagnosed underlying health condition predisposing them to premature mortality. In support of this hypothesis, it has been suggested that prolonged sleep might be a consequence of underlying diseases, frailty and worse health status, or be a part of the dying process.⁵⁹

The results of this study emphasize the need for sleep health management in the workplace, since the sleep health of employees may play as important a role on their future health outcomes as traditional CVD risk factors. Health screenings serve as a first step in workplace health promotion, where the awareness of health risks can lead to lifestyle change for the management and prevention of CVD. Of interest is that most workplace health programs focus on physical activity, smoking and diet, despite the evidence showing that employees averaging ≤ 6 h sleep were found to have a higher average number of health risks compared to those averaging 7-8 h.^{24,60} It is also clear that short sleep duration is associated with decreased workplace and public safety, sickness-related absenteeism,⁶¹ lower information processing,⁶² impaired cognition,⁶³ reduced task performance⁶⁴ and diminished job performance.⁶⁵

Given that many sequelae of insufficient sleep duration may adversely impact on cardiovascular health, safety and productivity of employees, employers have a vested interest in providing workplace health programs to support the workforce in achieving healthy sleep. Since our findings, and other recent evidence show a high prevalence of shorter sleep duration in

working adults,⁶⁶ there is an urgency to address sleep as a public health issue in workplace settings. Obstacles that prevent employees from having sufficient time to obtain the recommended 7-9 h of sleep, such as the duration and scheduling of work hours, and time taken to commute to work, are important focal points that may aid in the development and implementation of policies to improve employee sleep health. Strategies to promote sleep in the occupational health setting have been proposed, and include sleep education programs that can be provided at workplaces; behavioral interventions to promote sleep; setting limits on the number of hours worked; where possible, encouraging teleworking to facilitate more time for sleep; and modifying workplace environmental characteristics such as lighting.⁶⁷⁻⁶⁹

Another avenue for supporting sleep enhancement include workplace policies that allow for more flexibility, whereby employees are given autonomy of when and where they work, as this has been shown to improve sleep health.⁷⁰ Ultimately, employers and policy-makers are called to maximize the opportunity for employees to optimize their sleep health. One of the lessons learned from the global coronavirus pandemic in 2020 relates to sleep, work hours and workplace flexibility, since many employees were required to work from home. From a sleep perspective, there were reports of initial sleep extension^{71,72} while maintaining work hours, which suggests catch-up sleep in response to chronic sleep deprivation. Anecdotal reasons for longer sleep when working from home related to

Figure 5. Forest plot of the risk of death (cardiovascular disease) associated with longer sleep duration compared to the reference of 6-8 h in 4 population cohorts from 3 published prospective studies. RR: Relative risk; CI: confidence interval. Risk estimates were determined using a random effects model and heterogeneity is reflected by the I^2 statistic.

reduced travel time and more flexible work schedules. Another observation was that some people significantly delayed their sleep timing and work hours when working from home. This indicated that under routine work conditions, some people were sleeping and working at a time of day that may not have suited their circadian rhythms and chronotype. More flexibility in sleep and work timing may thus afford employees the opportunity to adjust their sleep and work activities to a time of day that best suits their biology, a known factor in improving both sleep health and productivity.

Strengths

All studies included in this meta-analysis used a prospective study design, thus the misclassification of sleep duration attributable to recall bias was minimized. Most of the included studies were of relatively high quality, based on the NOS quality assessment (average of 7) and only included employed adults from urban settings.

This review provides rationale for the inclusion of further studies representative of employees exclusively, particularly of studies representing the workforce of the African continent, which appear to be absent in the literature. The risk of dying from NCDs is highest in low- and middle-income countries, especially in sub-Saharan Africa,⁷³ therefore the impact of sleep duration in such countries, and its association with mortality are urgently needed to improve our knowledge of sleep health and disease in such populations.

Limitations

Although our findings are internally consistent with previous studies, we acknowledge several limitations. The primary limitation of these analyses is the small number of eligible studies available, which limited our statistical power. However, our study has highlighted a gap in the literature and underscores the importance of more sleep research needed in the field of occupational health. A second limitation is that in all but 2 studies,^{36,40} sleep duration was assessed at one point in time, and a single measurement of exposure may not capture the sustained effects of sleep duration over time, particularly if the reported period was shortly before death. Third, sleep duration in all studies was based on self-report and all but one study used 24 h sleep, thus making it difficult to distinguish time asleep from time-in-bed, estimating the number and duration of naps or capturing sleep quality data. The consistency of assessments (all self-reported questionnaires) and outcomes (all obtained from official national registries) across studies, however, attenuates heterogeneity which would otherwise occur due to differences in methods. We acknowledge the limitations of self-reported sleep duration as an outcome variable. It has, however, routinely been used in similar studies^{25,28-30,74} and shown to correlate well with more objective measures (sleep diaries, actigraphy, and polysomnography).^{39,75,76} It is also worth considering that from a translational-research and practical perspective, self-reported sleep duration provides a more cost-effective measure to implement in workplace health

So What?

What is Already Known on this Topic?

Suboptimal sleep has been identified as an important modifiable risk factor for cardiovascular and metabolic diseases and linked to mortality in the general population which include all ages, the unemployed, and people from rural communities. Since sleep patterns of employed individuals may differ to those of their unemployed counterparts, the nature of their sleep-mortality relationship may vary.

What does this Article Add?

This review considered only studies of employed urban individuals younger than 65 years, and found that self-reported shorter sleep was associated with a higher risk for both all-cause and cardiovascular disease mortality. These findings highlight the role of adequate sleep in the urban labor force required to mitigate the increased risk of all-cause and cardiovascular disease mortality.

What are the Implications for Health Promotion Practice or Research?

Workplace health programs are encouraged to include education, screening and support to employees about improving sleep health, and to minimize sleep deprivation of employees. Our findings suggest that aspects of employment, such as working hours, commuting time and industry, which likely impact sleep duration or quality, need to be addressed as part of workplace health programs.

programs and can be more widely utilized. Fourth, the included studies all presented mortality data based on different sleep duration categories (Supplementary Table S2) making it difficult to precisely match shorter, reference and longer sleep duration categories between studies. Finally, we recognize that a small number of occupations were represented by the participants in the reviewed studies, and the nature of the employment hours (e.g. night-time or rotational vs daytime shift work) was not recorded, thus limiting the generalisability of our results.

Conclusions

In this cohort of employed, urban individuals, self-reported shorter sleep was associated with a higher risk for both ACM and CVD. Whether or not longer sleep is associated with a higher ACM risk, requires further investigations to unravel the mechanisms by which this association may contribute to increased mortality risk. Specifically, studies in which sleep is measured objectively and aspects of sleep quality are required.

In the meantime, interventions such as workplace health programs which include education, screening and support to improve sleep health and ensure an adequate sleep opportunity on an individual basis are warranted. Conversations around aspects of employment, such as working hours, commuting and industry, which likely limit sleep duration or reduce sleep quality, should be held. Finally, sleep needs to be considered in the management and treatment of comorbid factors such as obesity, hypertension, and T2DM, all of which are known to increase the risk for mortality.

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Declaration of Conflicting Interests

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Supplemental Material

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References

1. World Health Organization. *Global Status Report on Non-Communicable Diseases, 2014*. World Health Organization; 2014.
2. World Health Organization. *Global Action Plan for the Prevention and Control of Noncommunicable Diseases 2013-2020*. World Health Organization. WHO Document Production Services; 2013.
3. World Health Organization. *Preventing Noncommunicable Diseases in the Workplace Through Diet and Physical Activity*. World Health Organization; 2008.
4. Basner M, Fomberstein KM, Razavi FM, et al. American time use survey: sleep time and its relationship to waking activities. *Sleep*. 2007;30(9):1085-1095.
5. Bin YS, Marshall NS, Glozier NS. Secular changes in sleep duration among Australian adults, 1992-2006. *Med J Aust*. 2011; 195(11-12):670-672.

6. Chatzitheochari S, Arber S. Lack of sleep, work and the long hours culture: evidence from the UK time use survey. *Work Employ Soc.* 2009;23(1):30-48.
7. Walsleben JA, Norman RG, Novak RD, O'malley EB, Rapoport DM, Strohl KP. Sleep habits of Long Island rail road commuters. *Sleep.* 1999;22(6):728-734.
8. Luckhaupt SE, Tak S, Calvert GM. The Prevalence of short sleep duration by industry and occupation in the National Health Interview Survey. *Sleep.* 2010;33(2):149-159.
9. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health.* 2015;1(1):40-43.
10. Watson NF, Badr MS, Belenky G, et al. Recommended amount of sleep for a healthy adult: a joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep.* 2015;38(6):843-844.
11. Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J.* 2011;32(12):1484-1492.
12. Guo X, Zheng L, Wang J, et al. Epidemiological evidence for the link between sleep duration and high blood pressure: a systematic review and meta-analysis. *Sleep Med.* 2013;14(4):324-332.
13. Shan Z, Ma H, Xie M, et al. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. *Diabetes Care.* 2015;38(3):529-537.
14. Gangwisch JE, Heymsfield SB, Boden-Albala B, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. *Hypertension.* 2006;47(5):833-839.
15. Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. *Diabetes Care.* 2006;29(3):657-661.
16. St-Onge M-P, Grandner MA, Brown D, et al. Sleep duration and quality: impact on lifestyle behaviors and cardiometabolic health: a scientific statement from the American Heart Association. *Circulation.* 2016;134(18):e367-e386.
17. Liu Y, Croft JB, Wheaton AG, et al. Peer reviewed: clustering of five health-related behaviors for chronic disease prevention among adults, United States, 2013. *Prev Chronic Dis.* 2016;13:160054.
18. Basner M, Spaeth AM, Dinges DF. Sociodemographic characteristics and waking activities and their role in the timing and duration of sleep. *Sleep.* 2014;37(12):1889-1906.
19. Petrov ME, Weng J, Reid KJ, et al. Commuting and sleep: results from the Hispanic community health study/study of Latinos Sueno ancillary study. *Am J Prev Med.* 2018;54(3):e49-e57.
20. Yoon H-S, Yang JJ, Song M, et al. Correlates of self-reported sleep duration in middle-aged and elderly Koreans: from the Health Examinees Study. *PLoS One.* 2015;10(5):e0123510.
21. Patel SR, Malhotra A, Gottlieb DJ, White DP, Hu FB. Correlates of long sleep duration. *Sleep.* 2006;29(7):881-889.
22. Kivimaki M, Jokela M, Nyberg ST, et al. Long working hours and risk of coronary heart disease and stroke: a systematic review and meta-analysis of published and unpublished data for 603,838 individuals. *Lancet.* 2015;386(10005):1739-1746.
23. Gilbert-Ouimet M, Ma H, Glazier R, Brisson C, Mustard C, Smith PM. Adverse effect of long work hours on incident diabetes in 7065 Ontario workers followed for 12 years. *BMJ Open Diabetes Res Care.* 2018;6(1):e000496.
24. Burton WN, Chen C-Y, Schultz AB, Li X. Association between employee sleep with workplace health and economic outcomes. *J Occup Environ Med.* 2017;59(2):177-183.
25. Liu T-Z, Xu C, Rota M, et al. Sleep duration and risk of all-cause mortality: a flexible, non-linear, meta-regression of 40 prospective cohort studies. *Sleep Med Rev.* 2017;32:28-36.
26. Garcia-Perdomo HA, Zapata-Copete J, Rojas-Ceron CA. Sleep duration and risk of all-cause mortality: a systematic review and meta-analysis. *Epidemiol Psychiatr Sci.* 2018;28(5):578-588.
27. Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. *J Sleep Res.* 2009;18(2):148-158.
28. da Silva AA, de Mello RG, Schaan CW, Fuchs FD, Redline S, Fuchs SC. Sleep duration and mortality in the elderly: a systematic review with meta-analysis. *BMJ Open.* 2016;6(2):e008119.
29. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. *Sleep.* 2010;33(5):585-592.
30. Yin J, Jin X, Shan Z, et al. Relationship of sleep duration with all-cause mortality and cardiovascular events: a systematic review and dose-response meta-analysis of prospective cohort studies. *J Am Heart Assoc.* 2017;6(9):e005947.
31. Olson R, Crain TL, Bodner TE, et al. A workplace intervention improves sleep: results from the randomized controlled work, family, and health study. *Sleep Health.* 2015;1(1):55-65.
32. Montagni I, Dehman A, Yu Z, et al. Effectiveness of a blended web-based intervention to raise sleep awareness at workplace: the WarmUapp™ pilot study. *J Occup Environ Med.* 2019;61(6):e253-e259.
33. Robbins R, Jackson CL, Underwood P, Vieira D, Jean-Louis G, Buxton OM. Employee sleep and workplace health promotion: a systematic review. *Am J Health Promot.* 2019;33(7):1009-1019.
34. Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. *PLoS Med.* 2009;6(7):e1000100.
35. Garde AH, Hansen AM, Holtermann A, Gyntelberg F, Suadicani P. Sleep duration and ischemic heart disease and all-cause mortality: prospective cohort study on effects of tranquilizers/hypnotics and perceived stress. *Scand J Work Environ Health.* 2013;39(6):550-558.
36. Heslop P, Smith GD, Metcalfe C, Macleod J, Hart C. Sleep duration and mortality: the effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Med.* 2002;3(4):305-314.
37. Wells G. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analysis. Published 2004. Accessed February 2 2021. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp
38. Rhee CW, Kim JY, Park BJ, Li ZM, Ahn YO. Impact of individual and combined health behaviors on all causes of premature mortality among middle aged men in Korea: the Seoul Male Cohort Study. *J Prev Med Public Health.* 2012;45(1):14-20.

39. Patel SR, Ayas NT, Malhotra MR, et al. A prospective study of sleep duration and mortality risk in women. *Sleep*. 2004;27(3):440-444.
40. Ferrie JE, Shipley MJ, Cappuccio FP, et al. A prospective study of change in sleep duration: associations with mortality in the Whitehall II cohort. *Sleep*. 2007;30(12):1659-1666.
41. Higgins J, Thompson SG. Quantifying heterogeneity in a meta-analysis. *Stat Med*. 2002;21(11):1539-1558.
42. Jackson D, White IR, Thompson SG. Extending DerSimonian and Laird's methodology to perform multivariate random effects meta-analyses. *Stat Med*. 2010;29(12):1282-1297.
43. Jike M, Itani O, Watanabe N, Buysse DJ, Kaneita Y. Long sleep duration and health outcomes: a systematic review, meta-analysis and meta-regression. *Sleep Med Rev*. 2018;39:25-36.
44. Suzuki E, Yorifuji T, Ueshima K, et al. Sleep duration, sleep quality and cardiovascular disease mortality among the elderly: a population-based cohort study. *Prev Med*. 2009;49(2-3):135-141.
45. Ikehara S, Iso H, Date C, et al. Association of sleep duration with mortality from cardiovascular disease and other causes for Japanese men and women: the JACC study. *Sleep*. 2009;32(3):295-301.
46. Stamatakis KA, Kaplan GA, Roberts RE. Short sleep duration across income, education, and race/ethnic groups: population prevalence and growing disparities during 34 years of follow-up. *Ann Epidemiol*. 2007;17(12):948-955.
47. Grandner MA, Williams NJ, Knutson KL, Roberts D, Jean-Louis G. Sleep disparity, race/ethnicity, and socioeconomic position. *Sleep Med*. 2016;18:7-18.
48. Grandner MA, Hale L, Moore M, Patel NP. Mortality associated with short sleep duration: the evidence, the possible mechanisms, and the future. *Sleep Med Rev*. 2010;14(3):191-203.
49. Grandner MA, Patel NP, Gehrman PR, Perlis ML, Pack AI. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies. *Sleep Med Rev*. 2010;14(4):239-247.
50. Kim TW, Jeong J-H, Hong S-C. The impact of sleep and circadian disturbance on hormones and metabolism. *Int J Endocrinol*. 2015;2015:591729.
51. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med*. 2004;1(3):e62.
52. Tobaldini E, Costantino G, Solbiati M, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neurosci Biobehav Rev*. 2017;74:321-329.
53. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. *Ann N Y Acad Sci*. 2008;1129(1):287-304.
54. Deng HB, Tam T, Chung-Ying Zee B, et al. Short sleep duration increases metabolic impact in healthy adults: a population-based cohort study. *Sleep*. 2017;40(10).
55. Amagai Y, Ishikawa S, Gotoh T, et al. Sleep duration and mortality in Japan: The Jichi Medical School Cohort Study. *J Epidemiol*. 2004;14(4):124-128.
56. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes: a systematic review, meta-analysis, and meta-regression. *Sleep Med*. 2017;32:246-256.
57. Tan X, Chapman CD, Cedernaes J, Benedict C. Association between long sleep duration and increased risk of obesity and type 2 diabetes: a review of possible mechanisms. *Sleep Med Rev*. 2018;40:127-134.
58. Grandner MA, Drummond SP. Who are the long sleepers? Towards an understanding of the mortality relationship. *Sleep Med Rev*. 2007;11(5):341-360.
59. Burazeri G, Gofin J, Kark JD. Over 8 hours of sleep—marker of increased mortality in Mediterranean population: follow-up population study. *Croat Med J*. 2003;44(2):193-198.
60. Chen C-Y, Schultz AB, Li X, Burton WN. The association between changes in employee sleep and changes in workplace health and economic outcomes. *Popul Health Manag*. 2018;21(1):46-54.
61. Reynolds AC, Appleton SL, Gill TK, et al. Sick leave absenteeism is associated with sleep problems independent of sleep disorders: results of the 2016 Sleep Health Foundation National Survey. *Sleep Health*. 2017;3(5):357-361.
62. Hsieh S, Cheng IC, Tsai LL. Immediate error correction process following sleep deprivation. *J Sleep Res*. 2007;16(2):137-147.
63. Kuppermann M, Lubeck DP, Mazonson PD, et al. Sleep problems and their correlates in a working population. *J Gen Intern Med*. 1995;10(1):25-32.
64. Dean B, Aguilar D, Shapiro C, et al. Impaired health status, daily functioning, and work productivity in adults with excessive sleepiness. *J Occup Environ Med*. 2010;52(2):144-149.
65. Hafner M, Stepanek M, Taylor J, Troxel WM, Van Stolk C. Why sleep matters—the economic costs of insufficient sleep. *Rand Health Q*. 2017;6(4):11.
66. Khubchandani J, Price JH. Short sleep duration in working American adults, 2010-2018. *J Community Health*. 2020;45(2):219-227.
67. Redeker NS, Caruso CC, Hashmi SD, Mullington JM, Grandner M, Morgenthaler TI. Workplace interventions to promote sleep health and an alert, healthy workforce. *J Clin Sleep Med*. 2019;15(4):649-657.
68. Barnes CM, Watson NF. Why healthy sleep is good for business. *Sleep Med Rev*. 2019;47:112-118.
69. Magnavita N, Garbarino S. Sleep, health and wellness at work: a scoping review. *Int J Environ Res Public Health*. 2017;14(11):1347.
70. Grzywacz JG, Casey PR, Jones FA. The effects of workplace flexibility on health behaviors: a cross-sectional and longitudinal analysis. *J Occup Environ Med*. 2007;49(12):1302-1309.
71. Leone MJ, Sigman M, Golombek DA. Effects of lockdown on human sleep and chronotype during the COVID-19 pandemic. *Curr Biol*. 2020;30(16):R930-R931.
72. Staller N, Randler C. Changes in sleep schedule and chronotype due to COVID-19 restrictions and home office. *Somnologie*. 2020:1-7.
73. Bennett JE, Stevens GA, Mathers CD, et al. NCD Countdown 2030: worldwide trends in non-communicable disease mortality

- and progress towards sustainable development goal target 3.4. *Lancet*. 2018;392(10152):1072-1088.
74. Zhong G, Wang Y, Tao T, Ying J, Zhao Y. Daytime napping and mortality from all causes, cardiovascular disease, and cancer: a meta-analysis of prospective cohort studies. *Sleep Med*. 2015; 16(7):811-819.
75. Signal TL, Gale J, Gander PH. Sleep measurement in flight crew: comparing actigraphic and subjective estimates to polysomnography. *Aviat Space Environ Med*. 2005;76(11):1058-1063.
76. Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *J Sleep Res*. 1999;8(3):175-183.