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

Pienaar, P. R., Kolbe-Alexander, T. L., van Mechelen, W., Boot, C. R. L., Roden, L. C., Lambert, E. V. & Rae, D. E.

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ABSTRACT

Objective: Sleeping less or more than the 7-8h 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 (CVDM) 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. Grey 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 (≤6h) was associated with a higher risk for (ACM) (RR: 1.16, 95% CI: 1.11-1.22) and CVDM (RR: 1.26, 95% CI: 1.12-1.41) compared to 7-8h of sleep, with no significant heterogeneity. The association between longer sleep (≥8h) 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 CVDM.

Keywords: sleep quantity; short sleep; heart disease; employed; workplace

OBJECTIVE

Non-communicable diseases (NCDs) account for about 71% of deaths globally among working-aged adults (35–70y), 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 Organisation'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 7-9h[9] or at least 7h[10] 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 7-8h).[11-15] While organisations 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 programmes, 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 ≤6h 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 two 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 two-fold increase in longer sleep duration (>9h) compared to those sleeping 7-8h. 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 >54h per week had a 30% higher risk for stroke and a 13% higher risk for CVD compared to those who worked 35-40h.[22] Similarly, the incidence of diabetes was higher among women working >45h per week compared to those working 35-40h[23] One study has shown a J-shaped relationship between sleep duration and number of health risk factors in employees, with those reporting ≤5h per night likely to present with the most risk factors, and those sleeping 7-8h per night with the fewest risk factors.[24] It was interesting to note that employees sleeping ≥9h per night were at greater risk for hypertension, depression, heart disease and cancer compared to those sleeping 7-8h[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 65y. Even though studies have shown that sleep health can be promoted in the workplace[31-33], some organisations feel there is insufficient evidence to make sleep a key component of their workplace policies and health promotion programmes. Data emanating from studies like ours may help advocate for the inclusion of sleep health in employee wellness programmes. 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 standardised 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 three databases: PubMed, Web of Science and Scopus. The search strategy included terms characterising 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 24h 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 four years, and (vii) at least three sleep duration categories were reported, e.g. ≤6h, 7-8h and ≥9h 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 occupation[36]), 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 grey 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 three 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 three studies presenting statistics on sleep duration in three categories[35, 36, 38] and two studies presenting data on five categories.[39, 40] Four of the five studies used a reference sleep duration category in the range of 6-8h (Ferrie, Garde, Heslop, Patel) while Rhee et al (2012) originally used their ≥8h group as their reference category. For our meta-analysis, we used the ACM and CVDM statistics in their original form for the three studies reporting on three sleep categories (Garde, Heslop, Rhee), but we re-referenced the data for Rhee et al to their 6-7h category, to better match the other studies.[38] For the two studies in which authors reported five 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 analysed 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 five studies eligible for inclusion and analysis (Figure 1). The general characteristics of the included studies are presented in Table 1. The five studies analysed for ACM comprised 116 969 participants from five countries: two studies included only men,[35, 38] one included only women[39] and two reported data on men and women combined or separately.[36, 40] The three studies analysed for CVDM comprised 19 467 participants from three countries. One of these studies combined their analysis to include men and

women,[40] one study reported only on a male cohort[35] and another analysed 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-time[40] sleep duration (h). The reference sleep duration ranged from 6-8h, shorter sleep ranged from <6 to <7h and longer sleep from ≥8 to >8h (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 6-8h 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 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 7-8h 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 7-8h, but had an extreme definition for short sleep duration (≤4h)[30]. In contrast, the present review showed that by sleeping as little as one hour less than the recommended sleep duration of 7-9h, employees may have a significantly increased risk for ACM. In another review using the same reference range of 7-8h, 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 65y.

Our finding that shorter, but not longer, sleep duration is associated with higher risk for CVDM contrasts with one previous study[28] 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 (<65y) while Da Silva et al. (2016) reported on older individuals (>60y). 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 7h. However, extreme sleep duration criteria of ≤4h and ≥10h 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 groups who are influenced by factors such as poverty and unemployment are more likely to have shorter or longer sleep compared to the recommended 7-9h.[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 6-8h 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 analysed 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 subgroup 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 four 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 than women sleeping longer than 9h may have a higher risk for ACM compared to men.[55] In contrast, three 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 five included studies, longer sleep was defined as either ≥8h or ≥9h per night. Given that current sleep duration guidelines indicate that 7-9h of sleep are recommended for optimal health[9, 10], longer sleep defined as ≥8h 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 hypothesise that perhaps individuals with longer sleep (≥9h) unwittingly 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 emphasise 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 programmes focus on physical activity, smoking and diet, despite the evidence showing that employees averaging ≤6h sleep were found to have a higher average number of health risks compared to those averaging 7-8h.[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 performance[64] 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 programmes 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 7-9h 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 programmes that can be provided at workplaces; behavioural 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 optimise 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 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 two 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 24h 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 programmes 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 recognise 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 CVDM. 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 programmes 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.

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 labour 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 programmes are encouraged to include education, screening and support to employees about improving sleep health, and to minimise 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 programmes.

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