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Impact of sleep habits on life expectancy free of cardiovascular disease in the Chinese population: a prospective cohort study

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Abstract

Background

Abnormal sleep duration and poor sleep quality have been associated with increased risks of cardiovascular disease (CVD) incidence and mortality. However, little is known about how these sleep problems affect total life expectancy (LE) and LE free of CVD and its subtypes.

Methods

We included 483,384 adults from the China Kadoorie Biobank (CKB) who were free of heart disease, stroke, cancer, major depressive disorder, and generalized anxiety disorder at baseline. Sleep duration was categorized as <6 h/d, 6-9 h/d, and >9 h/d. Three sleep disturbance symptoms were considered: self-reported difficulties initiating and maintaining sleep, early morning awakening, and daytime dysfunction. Participants with at least one of the three symptoms were considered to have a sleep disturbance. We estimated sex-specific LE with and without CVD at age 40 by using multistate Markov models, with separate models specified for total CVD, ischemic heart disease (IHD), ischemic stroke (IS), and hemorrhagic stroke (HS) as the disease state.

Results

During a median follow-up of 12.1 years, we documented 135,429 incident CVD events, including 46,479 IHD events, 47,562 IS events, and 10,844 HS events. Overall, there

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were 48,372 deaths. Compared to other sleep problems, longer sleep duration (>9 h/d) had the greatest impact on total LE and LE free of CVD and its subtypes, with the impact on total LE greater than that of disease-free LE. In men, the reduction in total LE and LE without CVD (95% confidence intervals) at age 40 associated with longer sleep duration was 2.11 (-2.50, -1.71) and 1.29 (-1.68, -0.98) years, respectively. The corresponding values for women were 1.37 (-1.81, -0.98) and 0.43 (-0.75, -0.07) years. In contrast, sleep disturbance had a stronger impact on disease-free LE than on total LE, thereby reducing the proportion of life spent in a healthy state. Compared with participants without sleep disturbance, the total LE at age 40 was 0.46 (-0.77, -0.15) and 0.22 (-0.47, 0.06) years lower in men and women with sleep disturbance, respectively, and the LE without CVD was 0.99 (-1.23, -0.73) and 1.05 (-1.27, -0.85) years lower.

Conclusions

In this Chinese population, abnormal sleep duration, especially long sleep, and sleep disturbance were linked with lower total LE and LE free of CVD. This study confirmed the importance of good sleep habits in health management for both those without and with CVD.

Keywords

Sleep duration, sleep quality, disease-free life expectancy, cardiovascular disease, cohort study

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Introduction

Sleep accounts for one-third of a person's life, and getting adequate and good-quality sleep is critical for health and well-being. In recent years, insufficient sleep and poor sleep quality have emerged as a global health concern. For example, more than one-quarter of the population in most European countries suffers from insomnia,[1] while at least one-third of Americans sleep less than 7 hours per day.[2] Such sleep problems are common in the Chinese population as well.[3]

Previous studies found that abnormal sleep duration, whether short or long, and poor sleep quality were both associated with an increased risk of total cardiovascular disease (CVD), coronary heart disease, and stroke, but the impact on coronary heart disease and stroke may differ.[4-7] A few studies conducted separately in patients with different types of CVD have also found an association between abnormal sleep duration and an increased risk of mortality in patients with CVD.[8,9] Furthermore, the aforementioned sleep problems have been linked to an increased risk of all-cause mortality in the general population.[10-12]

Unlike commonly used ratio-based estimates such as relative risk, life expectancy (LE) free of disease measures the average number of years lived without disease by combining both morbidity and mortality, providing an intuitive metric for evaluating the burdens related to specific risk factors. For example, if we define disease state of interest as having CVD, total life years can be divided into those lived without and with CVD, termed CVD-free LE and LE with CVD, respectively. The CVD-free LE is

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determined by the CVD incidence risk and mortality risk in the absence of CVD, whereas LE with CVD is determined by the CVD incidence risk and mortality risk in the presence of CVD.[13] However, because study populations, exposure definitions or classifications, and statistical analysis methods vary among existing studies, we could not clarify the impact of the aforementioned sleep problems on total LE and LE free of CVD and its subtypes simply by comparing the results of different studies.

To date, only one study using the UK Biobank (UKB) has evaluated the impact of sleep problems on total LE and LE free of CVD.[14] The study defined shorter sleep duration (<7 h/d) or longer sleep duration (≥9 h/d) as suboptimal sleep duration. Participants with different sleep characteristics had similar total LE, but those with suboptimal sleep duration and/or insomnia had a significant reduction in LE free of CVD. Nevertheless, this study did not report the strength of the association between sleep problems and each stage of CVD development and progression, making it hard to determine whether sleep problems contributed to the reduction of CVD-free LE by influencing which stage of disease progression. Furthermore, it is unclear whether longer and shorter sleep durations have different impacts on CVD-free LE, whether the impact of various sleep problems on LE free of different CVD subtypes are consistent, and whether the findings in the UK population are applicable to the Chinese population.

Thus, in this study, based on the China Kadoorie Biobank (CKB) of 0.5 million adults, we aimed to examine the impact of sleep duration and various sleep disturbance symptoms on the development and progression of total CVD and its subtypes, as well

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as differences in CVD-free LE and its share of total LE in participants with different sleep characteristics.

Methods

Study design and participants

The CKB is a nationwide population-based prospective cohort study. The study design and implementation are detailed elsewhere.[15] In brief, the 2004-2008 baseline survey enrolled 512,723 participants aged 30-79 from five urban and five rural areas. Following the completion of the baseline survey, all participants were followed up for mortality, morbidity, and hospitalization events. Two periodic resurveys were conducted in 2008 and 2013-2014, each with a cluster random sample of about 5% surviving participants. To avoid missing items and minimize logic errors, information collected at baseline and resurveys was directly entered into a laptop-based data entry system with built-in functions. All participants signed an informed consent form. The Ethical Review Committee of the Chinese Center for Disease Control and Prevention (Beijing, China), the Peking University Health Science Center (Beijing, China), and the Oxford Tropical Research Ethics Committee, University of Oxford (UK) approved the study.

In the present study, we excluded participants with self-reported clinician diagnoses of coronary heart disease (n=15,472), stroke (n=8,884), or cancer (n=2,578) at baseline. We also excluded participants who had major depressive disorder (n=3,354)

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or generalized anxiety disorder (n=1,278), as determined by section A and section B of the Chinese version of the computerized Composite International Diagnostic Inventory-short form, respectively.[16] This exclusion was implemented to minimize potential confounding by psychological conditions. Reasons for exclusion were not mutually exclusive. Forty-eight participants with missing values for body mass index (BMI) or women's menopause status were also excluded, leaving 483,384 participants in the primary analysis.

Assessment of sleep behaviors and other covariates

Sleep behaviors were assessed by an interviewer-administered questionnaire. By asking, "How many hours do you typically sleep per day (including naps)?" sleep duration was recorded as the number of reported hours. Based on the American National Sleep Foundation's sleep time recommendation, sleep duration was categorized as <6 h/d, 6-9 h/d, and >9 h/d.[17] Participants were asked if they had any of the following symptoms of sleep disturbance for at least 3 days per week in the past month (yes or no): (1) sleep onset latency of ≥30 min after going to bed or waking up in the middle of the night: if yes, considered as having difficulties initiating and maintaining sleep (DIMS); (2) waking up early and not being able to go back to sleep: if yes, considered as having disorders of early morning awakening (EMA); (3) having difficulty staying alert while at work, eating or meeting people during daytime: if yes, considered as having daytime dysfunction (DDF). Participants who reported having one or more of the three aforementioned conditions were considered to have a sleep

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disturbance.[18]

Other covariates, including socio-demographic characteristics, lifestyle behaviors, personal and family medical history, and women's menopausal status, were assessed by questionnaire and physical measurements. Participants who had at least one first-degree relative, including biological parents and siblings, having a heart attack or stroke were considered as having a CVD family history. The physical activity level was calculated by multiplying the metabolic equivalent of task (MET) value of each type of activity and the hours spent on that activity per day and then summarizing the MET-hours for all activities.[19] Habitual dietary intake in the past year was assessed by a qualitative food frequency questionnaire.[20] Weight and height were measured by trained staff using well-calibrated instruments. BMI was calculated as weight in kilograms divided by height in meters squared. Prevalent hypertension was defined as measured systolic blood pressure ≥140 mmHg, measured diastolic blood pressure ≥90 mmHg, self-reported diagnosis of hypertension, or self-reported use of antihypertensive medication at baseline. Prevalent diabetes was defined as self-reported diabetes or a measured fasting blood glucose ≥7.0 mmol/L or random blood glucose ≥11.1 mmol/L at baseline.

Ascertainment of disease onset and deaths

Incident cases of interest and the vital status of each participant were identified chiefly by linking to local disease and death registries and national health insurance database, supplemented with annual active follow-up to minimize loss to follow-up. All events

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7 Diseases (ICD-10) by trained staff blinded to the baseline information.
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10 The disease outcomes of interest in this study were CVD (I00-I99) and its major
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13 component endpoints -- ischemic heart disease (IHD, I20-I25), ischemic stroke (IS,
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16 I63), and hemorrhagic stroke (HS, I61). Case adjudication for IHD and stroke has
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18 been ongoing since 2014. Qualified cardiovascular specialists who were not aware of
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21 the baseline exposures reviewed the medical records of incident cases. Of the 33,515
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24 medical records of IHD cases and 40,465 medical records of stroke cases retrieved by
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26 October 2018, 87.9% of IHD cases and 91.8% of stroke cases had their diagnosis
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29 confirmed.[21]
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32 **Statistical analysis**
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36 Changes in sleep behaviors between the 2004-2008 baseline and 2013-2014 resurvey
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39 by the occurrence of CVD during this period were analyzed with adjustments for age
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42 at enrollment, sex, and study area, as appropriate.
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45 The following analyses were performed for men and women separately. A multistate
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48 Markov model was used to describe how participants moved between states in
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50 continuous time. To estimate associations between sleep behaviors and the
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53 instantaneous risk of movement between states, we constructed a non-recoverable
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56 illness-death model, with participants starting free of disease and moving (1) from
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58 disease-free to presence of disease; (2) from disease-free to death without
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experiencing the disease. Participants could also move from a disease state to death. Only the first entry into a state was considered. For participants who died on the same date as their first diagnosis, we used the date of death minus 0.5 days as the date of disease onset.[22] Separate models were specified for CVD, IHD, IS, and HS as the disease state.

Age was used as the time scale for all models. Participants were considered at risk from enrollment to death, loss to follow-up, or 31 December 2018, whichever came first. The parameters of the three-state model were estimated as a log-linear parametric model with an exponential distribution using the msm R package.[23] The analytical model included sleep behaviors and covariates, including attained age, study areas, education, marital status, family history of CVD, smoking status, alcohol intake, physical activity, dietary factors (intake frequencies of fresh vegetables, fresh fruits, and red meat), BMI, and menopausal status (only in women). All covariates were time-constant and influenced the instantaneous risk of movement between states in a proportional hazard manner.

With the estimated parameters of the multistate model above, LE at age 40 years was calculated using the Estimating Life Expectancies in Continuous Time (ELECT) R package (version 1.2).[24] The upper limit was set as 90 years since few participants reached this age during the follow-up period. For all the LE estimations, covariates used in analytic models were specified to the baseline average levels of the cohort. The confidence intervals (CIs) for LE were estimated using simulation 500 times.[25]

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To examine the robustness of the results, we performed several sensitivity analyses: (1) further adjusting for prevalence of hypertension and diabetes, and usage of statin at baseline; (2) excluding participants with chronic obstructive pulmonary disease (COPD), asthma, or type 2 diabetes (T2D) at baseline; (3) excluding participants who experienced disease onset or died within the first five years of follow-up.

Subgroup analyses were performed by residence (urban or rural), baseline disease status (with hypertension and/or diabetes, or none), family history of CVD (with or without), smoking status (never, former, and current; only in men), and physical activity level (age-specific [<50, 50-59, and ≥60 years] and sex-specific median or higher level, or lower level). All statistical analyses were performed using R version 4.0.3.

Results

Characteristics of the study population

The mean age of the included 483,384 participants was 51.5 ± 10.6 years, and 41.0% were men. At baseline, 86.9% of participants slept between 6 and 9 hours per day, while 8.0% slept less than 6 hours and 5.1% slept more than 9 hours; additionally, 16.1% of participants reported at least one symptom of sleep disturbance. Overall, participants who were older, less-educated, or lived in rural areas were more likely to have shorter or longer sleep duration, as well as sleep disturbance (**Table 1**). Of the 23,932 participants who participated in the 2013-2014 resurvey, over 70% had not

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changed their sleep habits since baseline (**Supplementary Table 1**). In addition, there were no discernible differences in sleep habit changes between participants who experienced CVD events and those who did not.

During a median follow-up of 12.1 years (5.69 million person-years, PYs), 135,429 incident CVD events were documented, with 46,479 being IHD events, 47,562 being IS events, and 10,844 being HS events. We recorded 48,372 deaths, 18,740 of which had no documented CVD between baseline and death.

Sleep behaviors and transitions between states

After controlling for potential confounders, shorter sleep duration was not associated with the incidence risks of CVD or any of the subtypes, nor with the mortality risk in the absence of disease, except that the risks of CVD and IHD onset were slightly increased in women with shorter sleep duration (**Figures 1 and 2**). In contrast, shorter sleep duration was associated with increased mortality risks in the presence of CVD or any of the subtypes in men and HS in women. The strongest association was observed for HS, with HRs (95% confidence intervals [CIs]) of 1.28 (1.13, 1.43) and 1.21 (1.09, 1.36) for men and women, respectively.

Longer sleep duration was associated with elevat risks of almost all transitions. In men, compared with participants with a sleep duration of 6-9 h/d, the HRs (95% CIs) of those with longer sleep duration were 1.13 (1.09, 1.18), 1.07 (0.99, 1.16), and 1.29 (1.22, 1.37) for baseline to CVD onset, baseline to death, and disease to death,

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respectively. The corresponding values for women were 1.03 (0.99, 1.06), 1.16 (1.05, 1.29), and 1.21 (1.12, 1.31). In general, the association of longer sleep duration with mortality risk in the presence of disease was stronger than that with incidence risk. Except in men, longer sleep duration was associated with an elevated incidence risk of HS, with an HR (95% CI) of 1.33 (1.21, 1.48), but was not with the mortality risk in the presence of disease.

Except for HS, sleep disturbance was associated with increased incidence risks of CVD and all subtypes. However, there was no statistically significant association between sleep disturbance and most transitions to death. Compared with participants without sleep disturbance, the HRs (95% CIs) for CVD onset in men and women with sleep disturbance were 1.10 (1.07, 1.13) and 1.10 (1.08, 1.13), respectively. When individual symptoms of sleep disturbance were considered, the results were largely consistent; except that EMA was not associated with the incidence risks of CVD or any of the subtypes in men (**Supplementary Table 2**).

Sleep behaviors and LE with and without disease

Longer sleep duration basically had a greater impact on total LE and disease-free LE for CVD, IHD, and IS at age 40 than shorter sleep duration in both men and women (**Figures 3 and 4**). In men, the reduction in total LE caused by longer or shorter duration was greater than the reduction in disease-free LE. In the analysis of CVD, the total LE of 40-year-old men with longer or shorter sleep duration was reduced by 2.11 (-2.50, -1.71) and 0.72 (-1.09, -0.39) years, respectively, with the reduction in LE

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4 without CVD being 1.29 (-1.68, -0.98) and 0.27 (-0.61, 0.05) years. In women, only
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7 longer sleep duration was similarly linked to a reduction in total LE and CVD-free LE
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9 at age 40 of 1.37 (-1.81, -0.98) and 0.43 (-0.75, -0.07) years, respectively. In contrast,
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23 of disease-free LE to total LE. Compared with participants without sleep disturbance,
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29 men and women with sleep disturbance, respectively, and LE without CVD was 0.99
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31 (-1.23, -0.73) and 1.05 (-1.27, -0.85) years lower.
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35 The impacts of longer and shorter sleep duration, as well as sleep disturbance, on LE
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38 without HS were similar to total LE at age 40 in both men and women. Compared
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40 with the reference group, the number of years lived free of HS at age 40 for
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43 participants with longer and shorter sleep duration and sleep disturbance was 2.08 (-
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45 2.60, -1.68), 0.49 (-0.91, -0.11) and 0.47 (-0.80, -0.15) years lower in men,
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48 respectively. The corresponding estimates in women were 1.43 (-1.89, -0.96), 0.24 (-
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51 0.58, 0.05), and 0.29 (-0.58, -0.03) years.
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54 Sensitivity analyses revealed no significant changes in the results (**Supplementary**
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57 **Tables 3 and 4**). Similar findings were found for CVD-free LE at ages 40, 50, and 65
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60 (**Supplementary Table 5**). When symptoms of sleep disturbance were considered

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individually, only DIMS had a statistically significant impact on total LE and disease-free LE in men; in women, all symptoms of sleep disturbance had impacts on disease-free LE, except for HS (**Supplementary Figures 1 and 2**).

In the joint analysis, compared with those with a sleep duration of 6-9 h/d and no sleep disturbance, participants with longer sleep duration and sleep disturbance had the greatest reduction in LE without CVD or any of the subtypes at age 40 (**Figure 5**). The CVD-free LE was reduced by 1.99 (-3.50, -0.45) years in men and 1.86 (-3.22, -0.64) years in women.

In the subgroup analysis by residence, the impact of longer sleep duration and sleep disturbance on LE without disease was slightly greater among urban residents than rural residents for most outcomes (**Supplementary Figure 3**). Longer and shorter sleep duration also had a greater impact on disease-free LE in participants with prevalent hypertension or diabetes than in those without (**Supplementary Figure 4**). Otherwise, the associations were broadly similar across subgroups defined by CVD family history, smoking status, and physical activity level (**Supplementary Figures 5-7**).

Discussion

This study found that longer sleep duration was associated with elevated risks of incident CVD and its subtypes, as well as mortality risks either in the absence or in the presence of disease, with the impact on mortality risks being stronger. Shorter sleep

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duration was predominantly associated with an increased risk of mortality in the presence of CVD, particularly in men. Sleep disturbance was associated with an increased risk of CVD and its subtype incidence. Thus, longer sleep duration had a bigger impact on total LE and LE free of CVD and its subtypes than both shorter sleep duration and sleep disturbance; meanwhile, its impact on total LE was greater than that of disease-free LE. In contrast, sleep disturbance had a stronger impact on LE free of CVD and its subtypes than on total LE, with a consequent higher proportion of life years lived with disease in participants with sleep disturbance.

Only one study has evaluated the association between sleep behaviors and LE free of CVD. Based on the UKB, the study found that men and women with only 0-1 healthy sleep habits had a reduced CVD-free LE of 2.31 (95% CI: 1.46, 3.29) and 1.80 (0.96, 2.75) years at age 40, compared with those with 4-5 healthy sleep habits (sleep duration of 7 to <9 h/d, no difficulty initiating or maintaining sleep, no snoring, morning chronotype, no frequent daytime sleepiness).[14] Despite the slight difference in the dimensions of sleep due to a different questionnaire design, the current study yielded similar results. Compared with those with a sleep duration of 6-9 h/d and no symptoms of sleep disturbance, the LE free of CVD at age 40 of participants with longer sleep duration and any symptom of sleep disturbance was reduced by 1.99 (-3.50, -0.45) years in men and 1.86 (-3.22, -0.64) years in women.

In the current study, longer sleep duration was associated with an increased risk of CVD and its subtype incidence, as well as a deleterious impact on mortality risks in the

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absence or presence of disease, with a consequent significant reduction in both total LE and LE free of disease. A meta-analysis of 95 prospective cohort studies showed that participants with longer sleep duration had a 39% higher risk of all-cause mortality and a 25% higher risk of CVD incidence risk compared with those with normal sleep duration.[26] In this study, conducted in a single cohort, we also observed that those with longer sleep duration had a greater increase in mortality risk than CVD incidence risk, with a consequent bigger reduction in total LE than in CVD-free LE. It has been postulated that the above association may be subjected to residual confounding or reverse causation, as participants with longer sleep duration may be more fatigued, have undiagnosed mental disorders, or be in a sub-healthy state.[27,28] However, in our primary analysis, we only included participants without major chronic diseases, major depressive disorder, or generalized anxiety disorder at baseline. Further excluding participants with respiratory diseases or those who experienced disease onset or died within the first 5 years of follow-up did not change the findings. This suggests that the previously mentioned bias cannot fully explain the association between longer sleep duration and the risk of CVD incidence or mortality. An earlier Mendelian randomization (MR) study in UKB concluded that genetically predicted long sleep duration might not be a causal risk factor for CVD, but this finding was likely influenced by weak instrument bias.[29] There is still a need to discover more sleep-related single nucleotide polymorphisms, and MR analysis in populations with different genetic backgrounds is required for additional validation.

Shorter sleep duration had a somewhat different impact on men and women, with men

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having an increased risk of death after CVD disease and women having a little higher incidence risk of CVD and IHD. As a result, women had a higher proportion of life years lived with the disease. A few earlier studies also observed that shorter sleep duration was associated with the elevated risks of CVD and its intermediate risk factors, such as hypertension, atherosclerosis, and myocardial infarction, but only in women.[30-33] It was suggested that hormonal influences may play a role; for example, shorter sleep duration may cause hormonal fluctuations in women, particularly around menopause, when women are more vulnerable.[32] Nevertheless, midday napping might compensate for the detrimental impact of shorter sleep duration at night in women.[6] The impact of shorter sleep duration on mortality risk in the presence of CVD or its subtypes was only seen in men, which was consistent with the findings of a recent study of 2,846 coronary artery disease patients with a mean age of 64 years. In this US study, after a median follow-up of 2.8 years, the all-cause mortality risk was only significantly increased in men with shorter sleep duration compared with those with sleep duration of 6.5-7.4 h/d, but not in women.[9] The reasons for this sex disparity are not fully understood.

In line with meta-analyses of the association between sleep disturbance and CVD incidence risk and all-cause mortality,[34,35] we observed that sleep disturbance and all its three symptoms were primarily associated with increased incidence risks of CVD, IHD, and IS, but not with risk of most transitions to death. As a result, participants with sleep disturbance developed CVD at a younger age and lived with it for a longer period of time. In the above-mentioned UKB study, only women with DIMS or DDF showed a significant reduction in CVD-free LE, with CVD-free LE at age 40 reduced by 0.30

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(0.18, 0.43) and 0.92 (0.04, 2.06) years, respectively.[14] In the current study, both men and women suffered from the negative consequences. The LE free of CVD at age 40 was 0.67 (0.42, 0.93) and 1.29 (0.87, 1.74) years lower for participants with DIMS or DDF, respectively in women; the corresponding values in men were 1.11 (0.79, 1.38) and 0.74 (0.00, 1.49) years. Sleep disturbance had a greater impact on our population, which may be partly related to the low treatment rate for insomnia in the Chinese population. According to a survey conducted in northern China in 2018, only 2.1% of participants with insomnia symptoms were treated, while only 6.2% of those with clinical insomnia diagnosis.[36] However, in Western developed countries, the treatment rate for insomnia symptoms reached 30% over a decade ago.[37] [38] Proper insomnia therapy may reduce the adverse effects on cardiovascular health.

In the current study, except for longer sleep duration, which had a significant association with an increased risk of HS incidence in men, none of the other sleep problems was linked to the HS risk. The observed slight decrease in LE free of HS among participants with shorter sleep duration or sleep disturbance was owing to an increased mortality risk in the absence of HS. Only three previous studies investigated the relationship between sleep duration and the risk of HS incidence, with two showing no association,[5,39] and one revealing a link between longer sleep duration and a higher risk of HS in women.[40] Because the number of HS cases in all these three studies was less than 1,000, statistical power may be inadequate.

This study has several strengths. First, using the same population and a uniform

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analytical strategy, we conducted the first comprehensive assessment of the impact of longer and shorter sleep duration, as well as various symptoms of sleep disturbance, on the development and progression of CVD and its subtypes. This could help us understand how sleep problems affect total LE and LE free of disease. Second, the study population was from areas with different economic development levels, having diverse socioeconomic characteristics. Third, because of the large sample size and long-term follow-up, the current study had greater statistical power to estimate disease-free LE for CVD and its subtypes, as well as perform several subgroup analyses.

Potential limitations should also be acknowledged. First, information on sleep duration and sleep disturbance symptoms was self-reported. Thus, the results could be influenced by information bias. Nevertheless, questionnaires are the most common and feasible tool for large population-based studies, and self-reported information is also the basis for assessing sleep quality in sleep clinics.[41] Second, we only used one baseline measurement of sleep habits, and possible changes in sleep patterns during follow-up were not considered. However, reverse causality could be avoided by using only baseline information. Furthermore, among participants who attended both the baseline survey and the second resurvey (a mean interval of 8 years), the changes in sleep patterns were similar between those who developed CVD during this period and those who did not. Third, limited by the questionnaire design, we focused on sleep duration and quality while not including other characteristics of sleep, such as chronotype, which is also an important factor in cardiovascular health.[42] However, some studies have shown that chronotype was closely correlated with sleep duration.[43,44] Fourth, there is

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a potential bidirectional relationship between sleep habits and CVD onset. However, the study participants' inclusion criteria, as well as the sensitivity analyses used in this study, suggest a robust temporal relationship between sleep habits and CVD onset. Nevertheless, the observational nature of this study limits causal inference, and future MR studies with proper instrumental variables and adequate power are needed to test the association.

In conclusion, based on a large prospective cohort of Chinese adults, we revealed that longer and shorter sleep duration, as well as sleep disturbance, had varied degrees of negative influence on the incidence of CVD and its subtypes, as well as mortality in the absence or the presence of CVD. Among them, longer sleep duration had the greatest impact on total LE and LE free of CVD. The findings of this study confirm the importance of good sleep habits in health management for individuals without and with CVD. Further research could be performed to detect whether there are any mechanistic pathways that mediate the adverse health effects of longer sleep duration. If their association was causal, more effective interventions for longer sleep duration should be developed.

Contributors

JL and LL conceived and designed the study, contributed to the interpretation of the results and critical revision of the manuscript for valuable intellectual content. LL, ZC, and JC: as the members of the CKB steering committee, designed and supervised the conduct of the whole study, obtained funding, and together with CY, DS, PP, LY,

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4 YC, HD, XZ, Dan S, acquired the CKB data. QS and JH accessed, verified, and analyzed the data. QS drafted the manuscript. All authors had access to the data and have read and approved the final manuscript. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted. JL and LL are the guarantors.

Declaration of interests

We declare no competing interests.

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Ministry of Science and Technology (2011BAI09B01).

Data availability

Details of how to access China Kadoorie Biobank data and details of the data release schedule are available from www.ckbiobank.org/site/Data+Access.

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Table 1. Baseline characteristics of the study participants by sleep duration and sleep disturbance.

	Sleep duration			Sleep disturbance*	
	<6 h/d	6-9 h/d	>9 h/d	No	Yes
Men (n=198,190)					
No. of participants	13,682	174,257	10,251	171,958	26,232
Age, years	56.4 (10.6)	52.0 (10.7)	52.6 (11.3)	52.1 (10.7)	54.2 (11.0)
Urban area	5,794 (42.3)	76,472 (43.9)	2,371 (23.1)	75,150 (43.7)	9,487 (36.2)
Middle school and above	6,234 (52.9)	103,359 (58.5)	4,984 (52.6)	101,248 (58.1)	13,329 (55.6)
Married	11,993 (89.6)	163,014 (93.4)	9,421 (92.3)	160,873 (93.5)	23,555 (90.7)
Current smoker†	9,255 (68.1)	118,080 (67.8)	7,128 (68.7)	116,614 (67.8)	17,849 (68.3)
Ever-regular drinker‡	6,578 (44.6)	72,330 (41.6)	4,087 (42.4)	71,165 (41.5)	11,830 (44.3)
Total physical activity, MET-h/d	22.9 (14.8)	22.7 (15.2)	20.1 (16.5)	22.6 (15.2)	22.8 (15.5)
Daily consumption of					
Fresh vegetables	12,988 (94.1)	165,133 (94.9)	9,861 (95.6)	163,243 (95.0)	24,739 (93.8)

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	Sleep duration			Sleep disturbance*	
	<6 h/d	6-9 h/d	>9 h/d	No	Yes
Fresh fruits	1,598 (11.1)	25,251 (14.3)	964 (14.4)	24,869 (14.2)	2,944 (12.7)
Red meat	4,234 (29.8)	58,397 (33.0)	2,456 (35.5)	57,236 (32.9)	7,851 (32.5)
Body mass index, kg/m^2	23.1 (3.2)	23.4 (3.2)	23.4 (3.3)	23.4 (3.2)	23.0 (3.2)
Prevalent hypertension	5,134 (34.7)	62,110 (35.9)	3,982 (37.7)	61,491 (36.0)	9,735 (35.6)
Prevalent diabetes	880 (5.7)	8,692 (5.0)	513 (5.9)	8,624 (5.0)	1,461 (5.5)
Family history of heart attack	390 (3.1)	5,490 (3.1)	317 (3.3)	5,281 (3.0)	916 (3.8)
Family history of stroke	2,271 (17.5)	30,994 (17.9)	1,965 (16.9)	30,245 (17.6)	4,985 (19.3)
Women (n=285,194)					
No. of participants	25,032	245,860	14,302	233,720	51,474
Age, years	56.4 (10.1)	50.6 (10.2)	48.9 (10.4)	50.3 (10.3)	54.0 (10.3)
Urban area	10,682 (42.7)	110,215 (44.8)	3,389 (23.7)	104,466 (44.7)	19,820 (38.5)
Middle school and above	8,044 (39.4)	109,563 (43.8)	5,796 (41.0)	105,440 (43.5)	17,963 (42.0)

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	Sleep duration			Sleep disturbance*	
	<6 h/d	6-9 h/d	>9 h/d	No	Yes
Married	20,517 (87.9)	221,770 (89.8)	13,146 (89.5)	211,306 (89.9)	44,127 (88.3)
Current smoker†	1,494 (2.7)	5,887 (2.7)	301 (3.2)	5,538 (2.7)	2,144 (2.8)
Ever-regular drinker‡	1,239 (3.1)	6,725 (2.9)	375 (3.1)	6,301 (2.8)	2,038 (3.2)
Total physical activity, MET-h/d	21.0 (11.8)	20.9 (12.9)	18.4 (12.5)	20.8 (12.8)	20.9 (12.6)
Daily consumption of					
Fresh vegetables	23,663 (93.9)	232,315 (94.6)	13,775 (94.9)	221,426 (94.7)	48,327 (94.1)
Fresh fruits	4,523 (17.9)	53,828 (21.5)	2,260 (23.6)	51,723 (21.5)	8,888 (20.2)
Red meat	6,153 (23.9)	66,734 (26.7)	2,775 (29.9)	63,242 (26.6)	12,420 (26.4)
Body mass index, kg/m^2	23.4 (3.5)	23.8 (3.4)	23.9 (3.5)	23.8 (3.4)	23.3 (3.4)
Prevalent hypertension	9,468 (31.3)	77,576 (32.2)	4,900 (34.5)	73,369 (32.4)	18,575 (31.7)
Prevalent diabetes	1,902 (5.8)	13,135 (5.5)	802 (6.4)	12,465 (5.5)	3,374 (5.8)
Family history of heart attack	710 (3.0)	7,900 (3.2)	400 (3.1)	7,309 (3.1)	1,701 (3.7)

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	Sleep duration			Sleep disturbance*	
	<6 h/d	6-9 h/d	>9 h/d	No	Yes
Family history of stroke	4,461 (17.5)	42,663 (17.5)	2,777 (17.3)	40,392 (17.2)	9,509 (19.0)
Postmenopausal	18,354 (52.5)	120,489 (50.6)	5,876 (50.0)	111,459 (50.4)	33,260 (52.2)

MET-h/d indicates the metabolic equivalent of tasks hours/day.

All variables were presented as mean (standard deviation) or number (percentage).

Baseline characteristics were adjusted for age and study area, except in the cases where age or urban/rural residence was the variable of interest.

*Sleep disturbance includes having difficulties initiating and maintaining sleep, early morning awakening, and daytime dysfunction. Participants who reported having one or more of the three aforementioned conditions were classified as having sleep disturbance.

†Participants who had stopped smoking due to illness were included.

‡Regular drinking refers to weekly consumption of any volume of alcohol.

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Figure legends

Figure 1. Multivariable-adjusted hazard ratios (95% confidence intervals) for each transition by sleep duration and sleep disturbance in men.

PYs indicate person-years. HR, hazard ratio; CI, confidence interval.

The definition of sleep disturbance was the same as in Table 1.

Multivariable models were adjusted for attained age, study areas (10 groups), education (no formal school, primary school, middle school, high school, college or university or higher), marital status (married, separated/divorced/widowed/never married), family history of cardiovascular disease (presence, absence), smoking status (never, former, current), alcohol intake (never, former, current weekly), physical activity (metabolic equivalent of tasks hours/day), dietary factors (intake frequency of fresh vegetables, fresh fruits, and red meat; the midpoint value of each frequency category was used in the model and treated as continuous), body mass index (kg/m^2), and mutually adjusted for sleep duration and sleep disturbance.

Figure 2. Multivariable-adjusted hazard ratios (95% confidence intervals) for each transition by sleep duration and sleep disturbance in women.

PYs indicate person-years. HR, hazard ratio; CI, confidence interval.

The definition of sleep disturbance was the same as in Table 1.

Multivariable models were adjusted for attained age, study areas, education, marital status, family history of cardiovascular disease, menopausal status, smoking status, alcohol intake, physical activity, dietary factors, body mass index, and mutually

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adjusted for sleep duration and sleep disturbance.

Figure 3. Life expectancy at age 40 years with and without disease by sleep duration and sleep disturbance in men.

LE indicates life expectancy; CI, confidence interval; DFLE, disease-free life expectancy; %DFLE represents the percentage of disease-free life expectancy to total life expectancy. The definition of sleep disturbance was the same as in Table 1.

Figure 4. Life expectancy at age 40 years with and without disease by sleep duration and sleep disturbance in women.

LE indicates life expectancy; CI, confidence interval; DFLE, disease-free life expectancy; %DFLE represents the percentage of disease-free life expectancy to total life expectancy. The definition of sleep disturbance was the same as in Table 1.

Figure 5. Life expectancy at age 40 years with and without disease by joint categories of sleep duration and sleep disturbance in men and women separately.

LE indicates life expectancy. The definition of sleep disturbance was the same as in Table 1.

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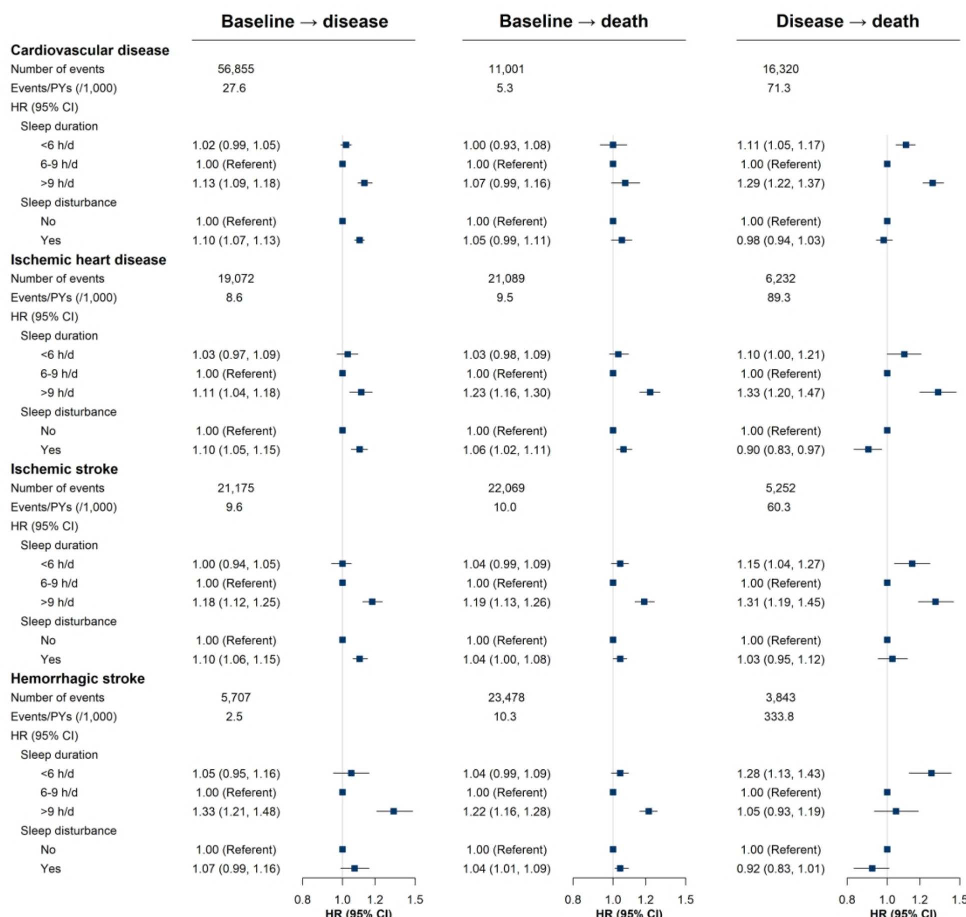

Figure 1. Multivariable-adjusted hazard ratios (95% confidence intervals) for each transition by sleep duration and sleep disturbance in men.
PYs indicate person-years. HR, hazard ratio; CI, confidence interval.
The definition of sleep disturbance was the same as in Table 1.
Multivariable models were adjusted for attained age, study areas (10 groups), education (no formal school, primary school, middle school, high school, college or university or higher), marital status (married, separated/divorced/widowed/never married), family history of cardiovascular disease (presence, absence), smoking status (never, former, current), alcohol intake (never, former, current weekly), physical activity (metabolic equivalent of tasks hours/day), dietary factors (intake frequency of fresh vegetables, fresh fruits, and red meat; the midpoint value of each frequency category was used in the model and treated as continuous), body mass index (kg/m2), and mutually adjusted for sleep duration and sleep disturbance.

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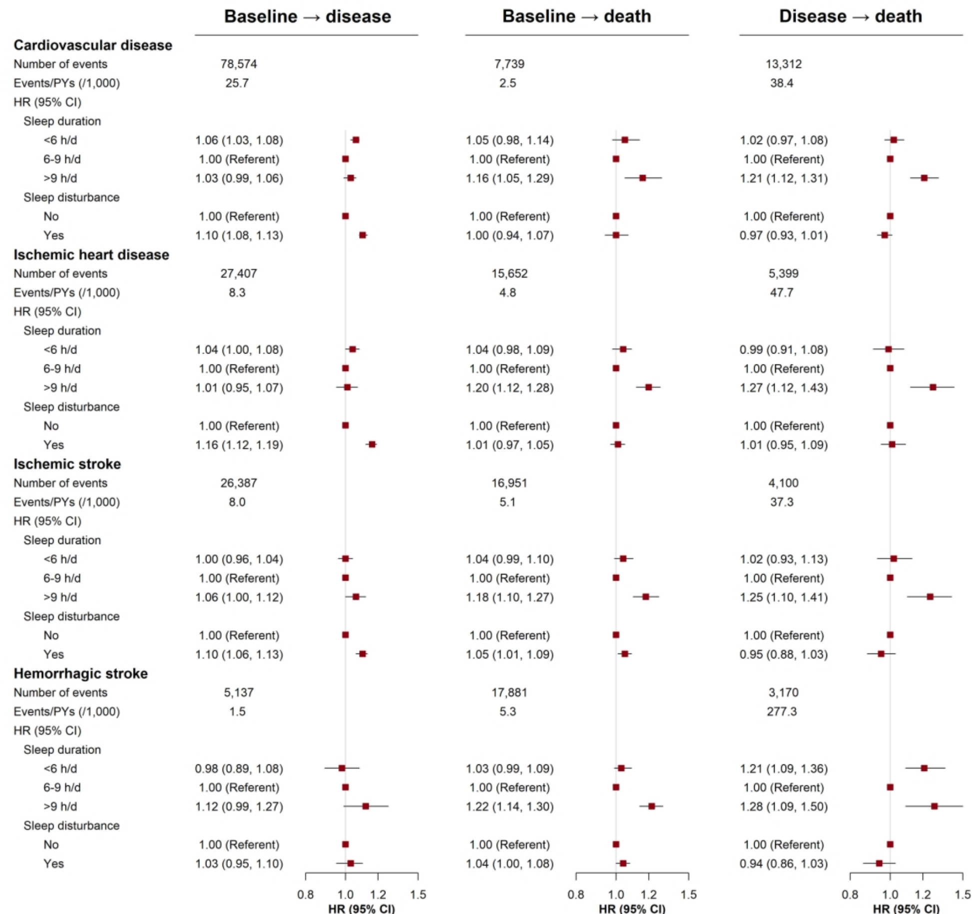

Figure 2. Multivariable-adjusted hazard ratios (95% confidence intervals) for each transition by sleep duration and sleep disturbance in women.
PYs indicate person-years. HR, hazard ratio; CI, confidence interval.
The definition of sleep disturbance was the same as in Table 1.
Multivariable models were adjusted for attained age, study areas, education, marital status, family history of cardiovascular disease, menopausal status, smoking status, alcohol intake, physical activity, dietary factors, body mass index, and mutually adjusted for sleep duration and sleep disturbance.

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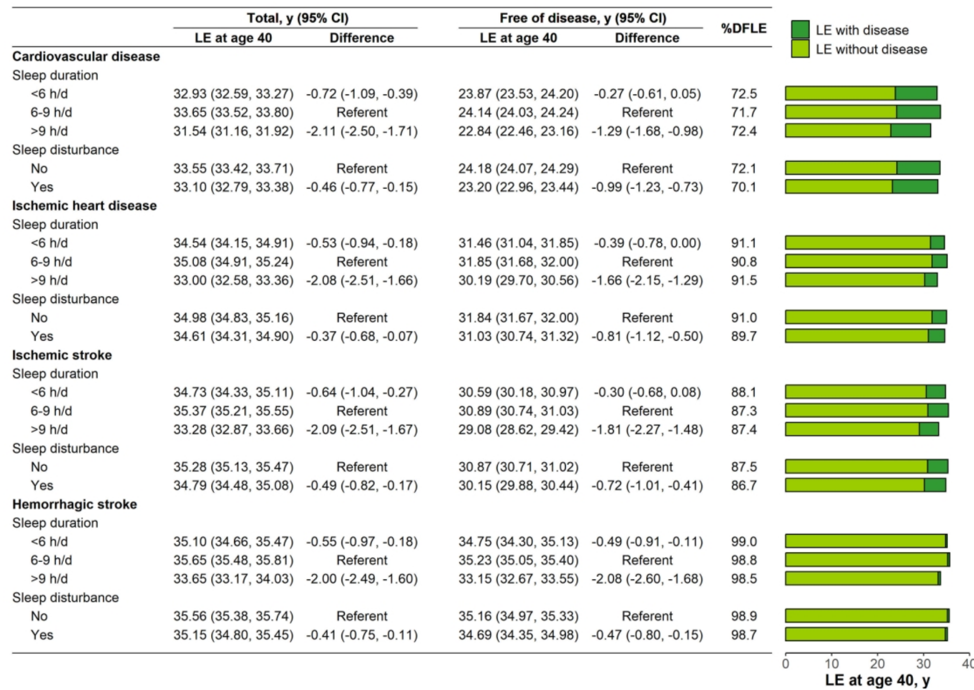

	Total, y (95% CI)		Free of disease, y (95% CI)		%DFLE
	LE at age 40	Difference	LE at age 40	Difference	
Cardiovascular disease					
Sleep duration					
<6 h/d	32.93 (32.59, 33.27)	-0.72 (-1.09, -0.39)	23.87 (23.53, 24.20)	-0.27 (-0.61, 0.05)	72.5
6-9 h/d	33.65 (33.52, 33.80)	Referent	24.14 (24.03, 24.24)	Referent	71.7
>9 h/d	31.54 (31.16, 31.92)	-2.11 (-2.50, -1.71)	22.84 (22.46, 23.16)	-1.29 (-1.68, -0.98)	72.4
Sleep disturbance					
No	33.55 (33.42, 33.71)	Referent	24.18 (24.07, 24.29)	Referent	72.1
Yes	33.10 (32.79, 33.38)	-0.46 (-0.77, -0.15)	23.20 (22.96, 23.44)	-0.99 (-1.23, -0.73)	70.1
Ischemic heart disease					
Sleep duration					
<6 h/d	34.54 (34.15, 34.91)	-0.53 (-0.94, -0.18)	31.46 (31.04, 31.85)	-0.39 (-0.78, 0.00)	91.1
6-9 h/d	35.08 (34.91, 35.24)	Referent	31.85 (31.68, 32.00)	Referent	90.8
>9 h/d	33.00 (32.58, 33.36)	-2.08 (-2.51, -1.66)	30.19 (29.70, 30.56)	-1.66 (-2.15, -1.29)	91.5
Sleep disturbance					
No	34.98 (34.83, 35.16)	Referent	31.84 (31.67, 32.00)	Referent	91.0
Yes	34.61 (34.31, 34.90)	-0.37 (-0.68, -0.07)	31.03 (30.74, 31.32)	-0.81 (-1.12, -0.50)	89.7
Ischemic stroke					
Sleep duration					
<6 h/d	34.73 (34.33, 35.11)	-0.64 (-1.04, -0.27)	30.59 (30.18, 30.97)	-0.30 (-0.68, 0.08)	88.1
6-9 h/d	35.37 (35.21, 35.55)	Referent	30.89 (30.74, 31.03)	Referent	87.3
>9 h/d	33.28 (32.87, 33.66)	-2.09 (-2.51, -1.67)	29.08 (28.62, 29.42)	-1.81 (-2.27, -1.48)	87.4
Sleep disturbance					
No	35.28 (35.13, 35.47)	Referent	30.87 (30.71, 31.02)	Referent	87.5
Yes	34.79 (34.48, 35.08)	-0.49 (-0.82, -0.17)	30.15 (29.88, 30.44)	-0.72 (-1.01, -0.41)	86.7
Hemorrhagic stroke					
Sleep duration					
<6 h/d	35.10 (34.66, 35.47)	-0.55 (-0.97, -0.18)	34.75 (34.30, 35.13)	-0.49 (-0.91, -0.11)	99.0
6-9 h/d	35.65 (35.48, 35.81)	Referent	35.23 (35.05, 35.40)	Referent	98.8
>9 h/d	33.65 (33.17, 34.03)	-2.00 (-2.49, -1.60)	33.15 (32.67, 33.55)	-2.08 (-2.60, -1.68)	98.5
Sleep disturbance					
No	35.56 (35.38, 35.74)	Referent	35.16 (34.97, 35.33)	Referent	98.9
Yes	35.15 (34.80, 35.45)	-0.41 (-0.75, -0.11)	34.69 (34.35, 34.98)	-0.47 (-0.80, -0.15)	98.7

Figure 3. Life expectancy at age 40 years with and without disease by sleep duration and sleep disturbance in men.
LE indicates life expectancy; CI, confidence interval; DFLE, disease-free life expectancy; %DFLE represents the percentage of disease-free life expectancy to total life expectancy. The definition of sleep disturbance was the same as in Table 1.

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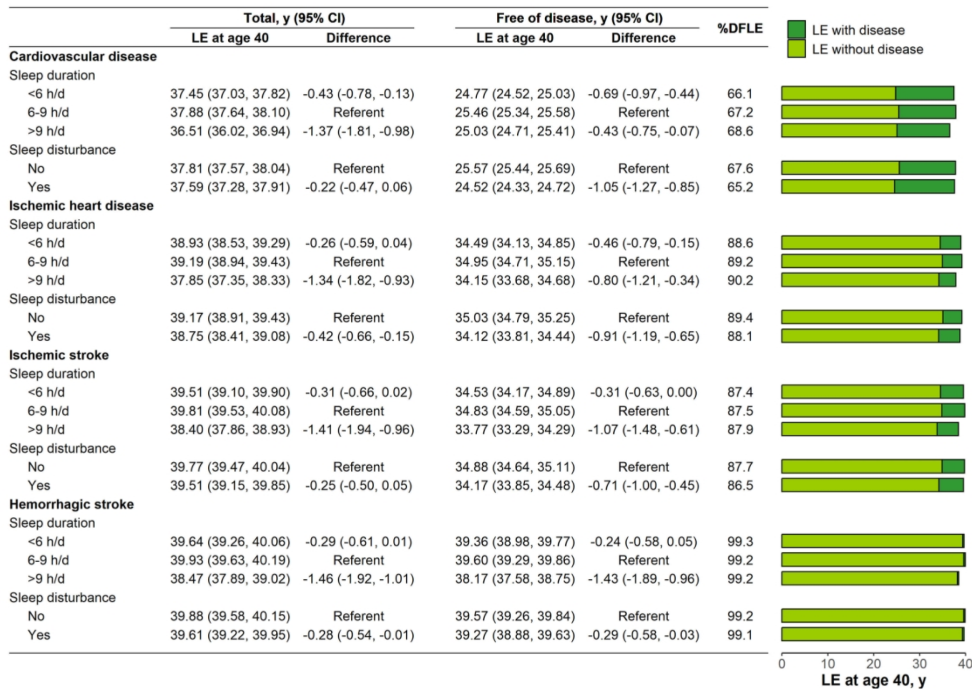

	Total, y (95% CI)		Free of disease, y (95% CI)		%DFLE
	LE at age 40	Difference	LE at age 40	Difference	
Cardiovascular disease					
Sleep duration					
<6 h/d	37.45 (37.03, 37.82)	-0.43 (-0.78, -0.13)	24.77 (24.52, 25.03)	-0.69 (-0.97, -0.44)	66.1
6-9 h/d	37.88 (37.64, 38.10)	Referent	25.46 (25.34, 25.58)	Referent	67.2
>9 h/d	36.51 (36.02, 36.94)	-1.37 (-1.81, -0.98)	25.03 (24.71, 25.41)	-0.43 (-0.75, -0.07)	68.6
Sleep disturbance					
No	37.81 (37.57, 38.04)	Referent	25.57 (25.44, 25.69)	Referent	67.6
Yes	37.59 (37.28, 37.91)	-0.22 (-0.47, 0.06)	24.52 (24.33, 24.72)	-1.05 (-1.27, -0.85)	65.2
Ischemic heart disease					
Sleep duration					
<6 h/d	38.93 (38.53, 39.29)	-0.26 (-0.59, 0.04)	34.49 (34.13, 34.85)	-0.46 (-0.79, -0.15)	88.6
6-9 h/d	39.19 (38.94, 39.43)	Referent	34.95 (34.71, 35.15)	Referent	89.2
>9 h/d	37.85 (37.35, 38.33)	-1.34 (-1.82, -0.93)	34.15 (33.68, 34.68)	-0.80 (-1.21, -0.34)	90.2
Sleep disturbance					
No	39.17 (38.91, 39.43)	Referent	35.03 (34.79, 35.25)	Referent	89.4
Yes	38.75 (38.41, 39.08)	-0.42 (-0.66, -0.15)	34.12 (33.81, 34.44)	-0.91 (-1.19, -0.65)	88.1
Ischemic stroke					
Sleep duration					
<6 h/d	39.51 (39.10, 39.90)	-0.31 (-0.66, 0.02)	34.53 (34.17, 34.89)	-0.31 (-0.63, 0.00)	87.4
6-9 h/d	39.81 (39.53, 40.08)	Referent	34.83 (34.59, 35.05)	Referent	87.5
>9 h/d	38.40 (37.86, 38.93)	-1.41 (-1.94, -0.96)	33.77 (33.29, 34.29)	-1.07 (-1.48, -0.61)	87.9
Sleep disturbance					
No	39.77 (39.47, 40.04)	Referent	34.88 (34.64, 35.11)	Referent	87.7
Yes	39.51 (39.15, 39.85)	-0.25 (-0.50, 0.05)	34.17 (33.85, 34.48)	-0.71 (-1.00, -0.45)	86.5
Hemorrhagic stroke					
Sleep duration					
<6 h/d	39.64 (39.26, 40.06)	-0.29 (-0.61, 0.01)	39.36 (38.98, 39.77)	-0.24 (-0.58, 0.05)	99.3
6-9 h/d	39.93 (39.63, 40.19)	Referent	39.60 (39.29, 39.86)	Referent	99.2
>9 h/d	38.47 (37.89, 39.02)	-1.46 (-1.92, -1.01)	38.17 (37.58, 38.75)	-1.43 (-1.89, -0.96)	99.2
Sleep disturbance					
No	39.88 (39.58, 40.15)	Referent	39.57 (39.26, 39.84)	Referent	99.2
Yes	39.61 (39.22, 39.95)	-0.28 (-0.54, -0.01)	39.27 (38.88, 39.63)	-0.29 (-0.58, -0.03)	99.1

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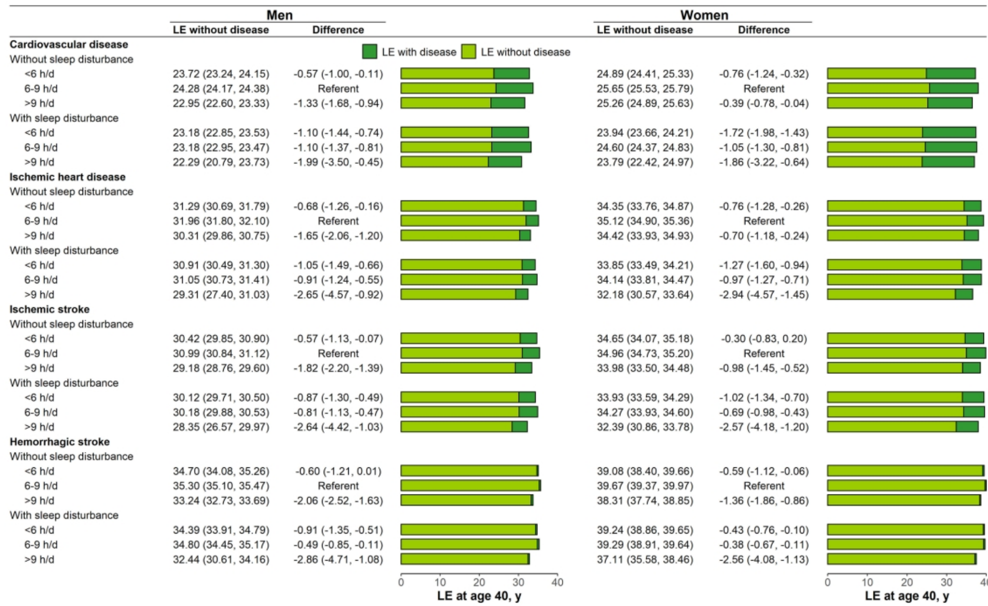

Figure 5. Life expectancy at age 40 years with and without disease by joint categories of sleep duration and sleep disturbance in men and women separately.
LE indicates life expectancy. The definition of sleep disturbance was the same as in Table 1.

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