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Transition of nighttime sleep duration and sleep quality with incident cardiovascular disease among middle-aged and older adults: results from a national cohort study

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Abstract

Background Sleep health has recently been incorporated into the Life's Essential 8 of the American Heart Association. Little is known about the associations between changes in nighttime sleep behavior and healthy outcomes, especially for the elderly. This study explores associations between transition of nighttime sleep duration and sleep quality and cardiovascular diseases (CVD) among middle-aged and older adults in China.

Methods Data were derived from the China Health and Retirement Longitudinal Study from 2011 to 2018, and a total of 7,905 participants age \geq 45 years were included. Participants were classified according to nighttime sleep duration (6–8, < 6 or > 8 h) and sleep quality assessed by the number of restless sleep days in the past week (< 3, 3–7 days). Four groups of the changing patterns in nighttime sleep duration and sleep quality between 2011 and 2015 were identified. CVD including heart disease and stroke was defined based on medical diagnosis. Robust Poisson regression and the restricted cubic spline were employed to evaluate the association between the transition of nighttime sleep behavior and the risk of CVD.

Results Compared to participants with consistently optimal nighttime sleep duration, those with consistently nonoptimal (incidence rate ratio [IRR]: 1.36, 95% confidence interval [CI]: 1.15–1.61, P < 0.001), optimal to non-optimal (IRR: 1.20, 95% CI: 1.02–1.43, P = 0.032), or non-optimal to optimal (IRR: 1.23, 95% CI: 1.02–1.48, P = 0.026) transition in nighttime sleep duration had higher risks of CVD. Additionally, those with a good to poor (IRR: 1.42, 95% CI: 1.20–1.68, P < 0.001) or a consistently poor (IRR: 1.55, 95% CI: 1.32–1.83, P < 0.001) changing pattern in nighttime sleep quality were associated with an increased risk of CVD compared to those with a consistently good changing pattern. There was a U-shaped association between changes in nighttime sleep duration and the incidence of CVD in sleepdeprived people. Changes in sleep quality and the risk of CVD exhibited a linear association.

Conclusions Persistent non-optimal nighttime sleep duration and poor sleep quality are associated with an increased risk of CVD in middle-aged and older adults. These findings highlight the importance of considering

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transitions in sleep behavior in CVD risk assessment for middle-aged and older adults, and emphasize the significance of long-term exposure to poor sleep behavior on their cardiovascular health.

Keywords Sleep duration, Sleep quality, Transition, Cardiovascular disease, Middle-aged and older adults

Text box 1. Contributions to the literature

 Healthy sleep is a crucial factor for cardiovascular health. However, studies on longitudinal changes in sleep behavior and their impact on cardiovascular disease risk among middle-aged and older adults remain limited.

• This study characterizes different changing patterns of nighttime sleep duration and sleep quality in middle-aged and older adults and examines their associations with future cardiovascular disease risk.

• Efforts aiming at maintaining optimal nighttime sleep duration and good sleep quality may contribute to a reduced risk of cardiovascular disease in middle-aged and older adults.

Introduction

Cardiovascular disease (CVD) is the leading cause of mortality worldwide [1]. Identifying modifiable risk factors is crucial for the prevention and control of CVD. In addition to traditional lifestyle behaviors, emerging evidence indicates that unhealthy sleep behaviors, such as short nighttime sleep duration and poor sleep quality, are significant risk factors for CVD [2, 3]. Given this accumulating evidence, sleep health has been incorporated into the latest Life's Essential 8 by the American Heart Association for CVD prevention [4].

Sleep patterns change with age, particularly during middle age, a period marked by increased susceptibility to stress and hormonal fluctuations [5-7]. Since the effects of behavioral factors on cardiovascular health are usually chronic, assessing sleep behavior at a single time point may not adequately capture its changing patterns related to CVD. Although the detrimental effects of poor sleep behaviors are recognized, there is often more interest in whether improving these behaviors can yield benefits. To our knowledge, only two studies have concurrently examined the longitudinal transition in nighttime sleep duration and sleep quality and their impact on CVD [8, 9]. One study involving 2,964 middle-aged American women found that persistent insomnia symptoms or insufficient sleep time are associated with a higher future risk of CVD [8]. However, it remains unknown whether these findings are applicable to males and individuals in a broader age range. Another study involving 11,347 Europeans showed that higher baseline healthy sleep scores and their improvement are associated with a lower risk of developing CVD [9]. This study, however, did not differentiate between the separate effects of nighttime sleep duration and sleep quality or identify any high-risk subgroups. Moreover, these associations have not yet been considered in Asian populations. Another outstanding question is the extent to which extending sleep duration in those with insufficient sleep could benefit CVD.

Therefore, based on the China Health and Retirement Longitudinal Study (CHARLS), we prospectively assessed the association between changing patterns in nighttime sleep duration and sleep quality, and the future risk of CVD. Additionally, we also quantitatively analyzed the impact of their transition on the risk of CVD.

Methods

Study design and participants

The CHARLS aims to collect high-quality data representative of the middle-aged and older population in China to analyze issues related to the aging population. The national baseline survey of CHARLS was conducted in 2011, employing a multi-stage probability sampling method to recruit more than 17,000 individuals from about 10,000 households across 150 county-level units and 450 village-level units. In the current analysis, we utilized data collected in the 2011, 2015, and 2018 waves. In the 2011 baseline survey, a total of 17,705 participants were recruited. We excluded participants who were under 45 years of age or lacked age information (N = 474), those with baseline CVD or missing CVD information (N=7,129), and those without sleep measurement data (N=1,199). Additionally, 998 participants who were lost to follow-up were further excluded. Ultimately, as shown in Figs. 1, 7,905 eligible participants were included in the current analysis. The ethics of CHARLS study was approved by the Biomedical Ethics Review Committee of Peking University (Approval number: IRB00001052-11014 and IRB00001052-11015). All participants have signed a written informed consent.

Exposure

Nighttime sleep duration and sleep quality data were collected in both the 2011 and 2015 waves of the survey. Participants were asked the following question: "During the past month, how many hours of actual sleep did you get at night (average hours for one night)?" Sleep duration was classified as optimal (6–8 h) or non-optimal (<6 or >8 h) [10]. Sleep quality was assessed by asking about the number of days of restless sleep (such as insomnia, waking up early, etc.) within the last week. The responses included four categories: (1) rarely or none of the time (<1 day); (2) some or a little of the time (1–2 days); (3) occasionally or a moderate amount of the time (3–4 days); (4) most or all of the time (5–7 days), each assigned scores from 1 to 4. Sleep quality was further categorized



Fig. 1 Study flowchart. CVD: cardiovascular disease

as good (<3 points) and poor (\geq 3 points). The changing patterns in nighttime sleep duration between 2011 and 2015 were used to identify four groups: consistent optimal, optimal to non-optimal, non-optimal to optimal, and consistent non-optimal. For sleep quality, the four groups were identified as: consistent good, good to poor, poor to good, and consistent poor.

Outcome

The primary outcome of the study was CVD, which included self-reported physician diagnoses of heart disease and/or stroke. The incidence of CVD was ascertained by asking the question in 2018 wave [11], "Did your doctor tell you that you have been diagnosed with a heart attack, angina pectoris, coronary heart disease, heart failure, or other heart problem?", and "Did your doctor tell you that you were diagnosed with a stroke?". If the participant gave at least one positive answer, they were considered to have CVD, and the time of diagnosis was also recorded.

Measurement of covariates

Sociodemographic and lifestyle information was collected using a standardized questionnaire. Current marital status was categorized into married and cohabiting, married but temporarily separated, and single. Residence was divided into rural and urban areas. Educational attainment was classified into three levels: primary school or below, middle school, and high school or above. Smoking status was categorized into never smokers and those with a history of smoking. Drinking status was divided into those who drink more than once a month, those who drink once a month or less, and non-drinkers. Household income was grouped into quartiles. Physical activity was defined as engaging in 30 min of moderate activity at least five times per week, or 20 min of vigorous activity at least three times per week [12]. Height was measured with the individual standing erect on the floor board of the stadiometer. Waist circumference was assessed while the individual was standing. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Systolic and diastolic blood pressures were measured using a digital blood pressure monitor (Omron[™] HEM-7200, Dalian, China). Hypertension was diagnosed if the participant had a previous diagnosis of hypertension, a systolic/diastolic blood pressure $\geq 140/90$ mmHg, or was taking antihypertensive medications. Diabetes was diagnosed if the participant had a previous diagnosis of diabetes, a fasting blood glucose level≥7 mmol/L, a glycated hemoglobin level \geq 6.5%, or was taking glucose-lowering medications.

Blood samples were stored at -70 °C. Bioassays were performed at national or county Centers for Disease Control that underwent uniform quality control. Blood glucose, creatinine levels, total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol were determined using the enzymatic colorimetric method. Hypersensitive C-reactive protein (hs-CRP) was measured using an immunoturbidimetric assay.

Statistical analysis

The study population was divided into two groups based on whether participants developed CVD. Descriptive statistics were expressed as mean±standard deviation for normally distributed continuous data, and as median (interquartile range) for skewed continuous data. Differences between the groups were assessed using Student's *t*-test or Kruskal-Wallis *H* test. Categorical data were expressed as frequency (percentage) and compared using the chi-square test.

A robust Poisson regression model was employed to ascertain the impact of baseline nighttime sleep duration and sleep quality, and their changing patterns on the incidence of CVD, reported as incidence risk ratios (IRR) with 95% confidence intervals (CI). Three multivariable models were utilized: Model 1, which adjusted for age, sex, and BMI; Model 2, which further adjusted for education, residence, smoking, alcohol consumption, and household income; Model 3, which additionally adjusted for hypertension, diabetes, and physical activity. In addition to analyzing the individual effects of nighttime sleep duration and sleep quality, the joint effect of baseline nighttime sleep duration and sleep quality on the risk of CVD was also assessed.

Initially, we analyzed the individual and joint effects of baseline nighttime sleep duration and sleep quality on the risk of CVD. Then, we explored the longitudinal changing patterns of nighttime sleep duration and sleep quality on the risk of CVD using two methods. First, a dose-response analysis was employed to assess the effects of transitions in nighttime sleep duration and sleep quality on CVD, using a restricted cubic spline model with four knots to quantitatively visualize the continuous relationship between these transitions and CVD events. Second, using 'consistent optimal' and 'consistent good' as reference categories, the association of the other categories with CVD, based on the changing patterns in nighttime sleep duration and sleep quality, was also explored. Subgroup analyses and effect modification were conducted in both predefined and exploratory subgroups across various factors, including age ($<65/\geq65$ years), sex (male/female), obesity status (BMI < $24/\ge 24$ kg/m²), diabetes status (yes/no), hypertension status (yes/no), and physical activity (yes/no). We conducted several sensitivity analyses to test the robustness of the results. First, logistic regression methods were used to clarify the association between changing patterns in nighttime sleep duration and sleep quality and CVD. Second, analyses excluding individuals with depressive symptoms were conducted. Finally, we included the 10-year CVD risk scores as a covariate for adjustment in the analysis. The method of the last observation carried forward, or the means and medians were used to interpolate the missing data (Supplementary Table 1). Stata (StataCorp LLC, version 15.0) and R software (version 4.2.2) were used for data analyses.

Results

Baseline information

Baseline characteristics are presented in Tables 1 and 2. The average age of the population was 57.87 ± 8.48 years,

with males comprising 48.84% of the population. Compared to participants with consistently optimal nighttime sleep duration, those with consistently non-optimal sleep duration were older, more likely to be female, more frequently single, predominantly rural residents, and had lower education levels and income. However, they had lower BMI and waist circumference, as well as lower smoking and alcohol consumption rates. Similar differences were observed among individuals with different transitions in nighttime sleep quality.

Baseline characteristics of participant with or without CVD events are presented in Supplementary Table 2. Compared to participants who did not experience CVD, those who did were older and had higher BMI and waist circumference, higher blood pressure, and lower levels of physical activity. Regarding biomarkers, participants with CVD had higher levels of blood glucose, total cholesterol, low-density lipoprotein cholesterol, and levels of hs-CRP. Details about nighttime sleep duration and sleep quality are also shown (Supplementary Table 3). Patients with CVD had significantly shorter baseline and followup nighttime sleep durations compared to those without CVD, and their sleep quality was also poorer. Consistently non-optimal nighttime sleep duration and poor sleep quality were significantly more common among patients with CVD than among those without.

The association of nighttime sleep duration, sleep quality, and cardiovascular disease

Over 55,335 person-years of follow-up, 873 CVD events were recorded. Non-optimal baseline nighttime sleep duration (IRR: 1.22, 95% CI: 1.07–1.38, P=0.003) and poor sleep quality (IRR: 1.19, 95% CI: 1.04–1.35, P=0.010) were both associated with the increased risk of CVD (Supplementary Fig. 1).

The association of transition of nighttime sleep duration and sleep quality and cardiovascular disease

As shown in Table 3, compared to those with consistently optimal nighttime sleep duration, the risk of CVD increased by 20% (IRR: 1.20, 95% CI: 1.02–1.43, P = 0.032) for the optimal to non-optimal changing pattern, 23% (IRR: 1.23, 95% CI: 1.02–1.48, P = 0.026) for the non-optimal to optimal pattern, and 36% (IRR: 1.36, 95% CI: 1.15–1.61, P < 0.001) for the consistently non-optimal pattern, after multivariable adjustments. For nighttime sleep quality, the IRR for CVD was 1.42 (95% CI: 1.20–1.68, P < 0.001) for the good to poor pattern and 1.55 (95% CI: 1.32–1.83, P < 0.001) for the consistently poor pattern, whereas the transition from poor to good did not significantly increase the risk of CVD (IRR: 1.05, 95% CI: 0.87–1.28, P = 0.604).

To further clarify the impact of quantitative changes in nighttime sleep duration on CVD, Fig. 2 shows a

Table 1 Baseline characteristic among different transition of nighttime sleep duration groups

| | All N=7905 | Consistent optimal | Optimal to non-optimal | Non-optimal to optimal | Consistent non-optimal | P- value |
|---|-------------------|-----------------------|---------------------------|---------------------------|---------------------------|-------------|
| | | N=3450 | N=1619 | N=1257 | N=1579 | |
| Age, year | 57.87±8.48 | 56.61 ± 7.92 | 58.29 ± 8.87 | 58.27±8.40 | 59.90 ± 8.85 | < 0.001 |
| Male sex, n (%) | 3861 (48.84%) | 1871 (54.23%) | 689 (42.56%) | 626 (49.80%) | 675 (42.75%) | < 0.001 |
| BMI, kg/m ² | 23.35 ± 3.50 | 23.60 ± 3.50 | 23.40 ± 3.48 | 23.14 ± 3.58 | 22.96 ± 3.42 | < 0.001 |
| Waist, cm | 83.75 ± 12.10 | 84.26 ± 12.00 | 83.95 ± 12.40 | 82.96 ± 12.19 | 83.07±11.89 | < 0.001 |
| Marriage status, n (%) | | | | | | < 0.001 |
| Married and living with spouse | 6820 (86.27%) | 3089 (89.54%) | 1368 (84.50%) | 1071 (85.20%) | 1292 (81.82%) | |
| Married but temporarily separated | 337 (4.26%) | 133 (3.86%) | 81 (5.00%) | 50 (3.98%) | 73 (4.62%) | |
| Single | 748 (9.46%) | 228 (6.61%) | 170 (10.50%) | 136 (10.82%) | 214 (13.55%) | |
| Residence, n (%) | | | | | | < 0.001 |
| Rural | 5254 (66.46%) | 2158 (62.55%) | 1106 (68.31%) | 861 (68.50%) | 1129 (71.50%) | |
| Urban | 2651 (33.54%) | 1292 (37.45%) | 513 (31.69%) | 396 (31.50%) | 450 (28.50%) | |
| Education, n (%) | | | | | | < 0.001 |
| Primary school and below | 5330 (67.43%) | 2040 (59.13%) | 1160 (71.65%) | 910 (72.39%) | 1220 (77.26%) | |
| Junior high school | 1707 (21.59%) | 897 (26.00%) | 306 (18.90%) | 249 (19.81%) | 255 (16.15%) | |
| High school and above | 868 (10.98%) | 513 (14.87%) | 153 (9.45%) | 98 (7.80%) | 104 (6.59%) | |
| Smoking, n (%) | 3129 (39.58%) | 1470 (42.61%) | 582 (35.95%) | 507 (40.33%) | 570 (36.10%) | < 0.001 |
| Alcohol consumption, n (%) | | | | | | < 0.001 |
| Drink more than once a month | 2157 (27.29%) | 1013 (29.36%) | 391 (24.15%) | 343 (27.29%) | 410 (25.97%) | |
| Drink less than once a month | 658 (8.32%) | 326 (9.45%) | 129 (7.97%) | 94 (7.48%) | 109 (6.90%) | |
| Never drink | 5090 (64.39%) | 2111 (61.19%) | 1099 (67.88%) | 820 (65.23%) | 1060 (67.13%) | |
| Household income, n (%) | | | | | | < 0.001 |
| Q1 | 1952 (24.69%) | 733 (21.25%) | 440 (27.18%) | 320 (25.46%) | 459 (29.07%) | |
| Q2 | 2024 (25.60%) | 880 (25.51%) | 410 (25.32%) | 334 (26.57%) | 400 (25.33%) | |
| Q3 | 1909 (24.15%) | 850 (24.64%) | 381 (23.53%) | 291 (23.15%) | 387 (24.51%) | |
| Q4 | 2020 (25.55%) | 987 (28.61%) | 388 (23.97%) | 312 (24.82%) | 333 (21.09%) | |
| Physical activity, n (%) | 2150 (27.20%) | 949 (27.51%) | 467 (28.84%) | 331 (26.33%) | 403 (25.52%) | 0.165 |
| Systolic BP, mmHg | 128.43±20.25 | 128.27±19.57 | 128.60±20.77 | 127.36±20.10 | 129.44±21.21 | 0.118 |
| Diastolic BP, mmHg | 75.25±11.75 | 75.86±11.74 | 74.99±11.66 | 74.51±11.73 | 74.81±11.83 | < 0.001 |
| Plasma glucose, mg/dL | 109.02±34.47 | 109.02±34.65 | 108.65±30.74 | 109.25±34.34 | 109.21±37.79 | 0.743 |
| Total cholesterol, mg/dL | 193.13±38.33 | 192.28±38.04 | 193.17±37.86 | 192.59±39.01 | 195.39±38.85 | 0.195 |
| Triglyceride, mg/dL | 132.75±114.11 | 134.78±126.14 | 129.83±100.09 | 132.54±107.17 | 131.57±105.44 | 0.793 |
| High-density lipoprotein cholesterol, mg/dL | 51.48±15.37 | 51.06±15.39 | 51.30±14.67 | 51.10±15.21 | 52.89±16.09 | 0.004 |
| Low-density lipoprotein cholesterol, mg/dL | 115.91±34.32 | 115.53±33.00 | 117.07±35.79 | 114.98±34.76 | 116.24±35.17 | 0.866 |
| Creatinine, mg/dL | 0.77±0.18 | 0.78±0.19 | 0.77±0.18 | 0.77±0.18 | 0.76±0.18 | < 0.001 |
| Hs-CRP, mg/L | 0.95 (0.52–1.94) | 0.95 (0.52–1.94) | 0.93 (0.52–1.94) | 0.98 (0.52–1.90) | 0.94 (0.52–1.98) | 0.993 |
| - Hypertension, n (%) | 2553 (32.30%) | 1067 (30.93%) | 523 (32.30%) | 395 (31.42%) | 568 (35.97%) | 0.004 |
| Diabetes mellitus, n (%) | 947 (11.98%) | 405 (11.74%) | 196 (12.11%) | 151 (12.01%) | 195 (12.35%) | 0.936 |

BMI: body mass index; Q: quantile; BP: blood pressure; Hs-CRP: hypersensitive C-reactive protein

U-shaped relationship between these changes and CVD events in individuals with insufficient sleep (<6 h). Increasing sleep duration by up to 4.5 h may benefit CVD, but further increases could be detrimental. Among all participants, there was a linear association between decreases in nighttime sleep quality scores and CVD.

Subgroup analysis

Results of the subgroup analyses are presented in Supplementary Fig. 2. Significant interactions between both nighttime sleep duration and sleep quality with age were identified. Consistently non-optimal nighttime sleep duration and poor sleep quality significantly increased the risk of CVD in individuals younger than 65 years (P for interaction: 0.005 for nighttime sleep duration and 0.013 for sleep quality).

Sensitivity analysis

Whether using the logistic model (Supplementary Table 4), excluding individuals with depression (Supplementary Table 5), or considering the 10-year CVD risk scores (Supplementary Table 6), the analysis results were consistent with the main findings.

Table 2 Baseline characteristic among different transition of nighttime sleep quality groups

| | Consistent good N=4138 | Good to poor N=1286 | Poor to good N=1178 | Consistent poor N=1303 | <i>P</i> -value |
|---|---------------------------|------------------------|------------------------|---------------------------|-----------------|
| Age, year | 57.73±8.47 | 57.70±8.88 | 58.15±8.26 | 58.25±8.27 | 0.026 |
| Male sex, n (%) | 2362 (57.08%) | 512 (39.81%) | 544 (46.18%) | 443 (34.00%) | < 0.001 |
| BMI, kg/m ² | 23.50 ± 3.48 | 23.38±3.43 | 23.21±3.57 | 22.99 ± 3.55 | < 0.001 |
| Waist, cm | 84.24±12.02 | 83.73±12.14 | 83.19±12.55 | 82.76±11.84 | < 0.001 |
| Marriage status, n (%) | | | | | < 0.001 |
| Married and living with spouse | 3641 (87.99%) | 1105 (85.93%) | 984 (83.53%) | 1090 (83.65%) | |
| Married but temporarily separated | 157 (3.79%) | 50 (3.89%) | 62 (5.26%) | 68 (5.22%) | |
| Single | 340 (8.22%) | 131 (10.19%) | 132 (11.21%) | 145 (11.13%) | |
| Residence, n (%) | | | | | 0.037 |
| Rural | 2696 (65.15%) | 872 (67.81%) | 785 (66.64%) | 901 (69.15%) | |
| Urban | 1442 (34.85%) | 414 (32.19%) | 393 (33.36%) | 402 (30.85%) | |
| Education, n (%) | | | | | < 0.001 |
| Primary school and below | 2611 (63.10%) | 893 (69.44%) | 840 (71.31%) | 986 (75.67%) | |
| Junior high school | 980 (23.68%) | 256 (19.91%) | 234 (19.86%) | 237 (18.19%) | |
| High school and above | 547 (13.22%) | 137 (10.65%) | 104 (8.83%) | 80 (6.14%) | |
| Smoking, n (%) | 1856 (44.85%) | 423 (32.89%) | 458 (38.88%) | 392 (30.08%) | < 0.001 |
| Alcohol consumption, n (%) | | | | | < 0.001 |
| Drink more than once a month | 1274 (30.79%) | 285 (22.16%) | 328 (27.84%) | 270 (20.72%) | |
| Drink less than once a month | 357 (8.63%) | 101 (7.85%) | 101 (8.57%) | 99 (7.60%) | |
| Never drink | 2507 (60.58%) | 900 (69.98%) | 749 (63.58%) | 934 (71.68%) | |
| Household income, n (%) | | | | | 0.275 |
| Q1 | 1023 (24.72%) | 316 (24.57%) | 281 (23.85%) | 332 (25.48%) | |
| Q2 | 1025 (24.77%) | 339 (26.36%) | 319 (27.08%) | 341 (26.17%) | |
| Q3 | 1024 (24.75%) | 279 (21.70%) | 284 (24.11%) | 322 (24.71%) | |
| Q4 | 1066 (25.76%) | 352 (27.37%) | 294 (24.96%) | 308 (23.64%) | |
| Physical activity, n (%) | 1143 (27.62%) | 367 (28.54%) | 310 (26.32%) | 330 (25.33%) | 0.230 |
| Systolic BP, mmHg | 128.97±20.00 | 128.30±20.61 | 127.90±20.13 | 127.33±20.72 | 0.005 |
| Diastolic BP, mmHg | 75.78±11.70 | 74.93±11.71 | 74.70±11.71 | 74.43±11.91 | 0.001 |
| Plasma glucose, mg/dL | 109.00±32.95 | 107.43±28.57 | 109.59±37.55 | 110.05 ± 40.62 | 0.442 |
| Total cholesterol, mg/dL | 192.32±38.68 | 192.44±36.24 | 194.60±37.74 | 194.92±39.65 | 0.237 |
| Triglyceride, mg/dL | 132.39±110.08 | 130.85±104.51 | 133.25±116.33 | 135.18±131.59 | 0.934 |
| High-density lipoprotein cholesterol, mg/dL | 50.91±15.17 | 52.34 ± 15.47 | 51.98±15.77 | 51.96±15.46 | 0.006 |
| Low-density lipoprotein cholesterol, mg/dL | 115.65±34.14 | 114.93±33.60 | 116.79±34.98 | 116.82±34.93 | 0.413 |
| Creatinine, mg/dL | 0.78±0.18 | 0.76±0.18 | 0.77±0.19 | 0.74±0.18 | < 0.001 |
| Hs-CRP, mg/L | 0.93 (0.52–1.88) | 0.95 (0.52-1.96) | 1.01 (0.53–1.90) | 0.98 (0.52-2.12) | 0.487 |
| Hypertension, n (%) | 1327 (32.07%) | 415 (32.27%) | 385 (32.68%) | 426 (32.69%) | 0.965 |
| Diabetes mellitus, n (%) | 478 (11.55%) | 151 (11.74%) | 147 (12.48%) | 171 (13.12%) | 0.443 |

BMI: body mass index; Q: quantile; BP: blood pressure; Hs-CRP: hypersensitive C-reactive protein

Discussion

From a national cohort study involving nearly 8,000 people followed over seven years, we found that: (1) consistently non-optimal nighttime sleep duration and poor sleep quality pose the greatest risk, while transitions from good to poor sleep behaviors can also increase the risk; (2) among those with insufficient nighttime sleep, changes in sleep duration show a U-shaped association with CVD events, with increases up to 4.5 h potentially reducing the risk; (3) changes in nighttime sleep quality show a linear relationship with the incidence of CVD. Our findings emphasize the importance of monitoring sleep behaviors in daily practice for CVD prevention. Most previous studies on the relationship between sleep and CVD focused solely on baseline sleep measurements, which did not adequately assess exposure to dynamic changes in sleep behaviors. In our current study, we considered the changing patterns in nighttime sleep duration and sleep quality, reflecting a more dynamic evaluation of sleep behaviors, which proved more effective than a single assessment. Consistent with our findings, several studies assessing sleep changing patterns found that individuals with persistent insufficient sleep duration or poor sleep quality were most prone to CVD [8, 9, 13]. However, many gaps still need to be addressed. Our results showed that persistent unhealthy nighttime

| Table 3 | The IRRs (95%CI) | of the association | between a | changing | patterns of | of nighttime | e sleep | duration | and sleep | o quality | and |
|----------|------------------|--------------------|-----------|----------|-------------|--------------|---------|----------|-----------|-----------|-----|
| cardiova | scular disease | | | | | | | | | | |

| | Events/N | Model 1 (IRR, 95%CI) | P-value | Model 2 (IRR, 95%CI) | P-value | Model 3 (IRR, 95%CI) | P-value |
|-----------------------------|----------|-------------------------|---------|-------------------------|---------|-------------------------|---------|
| Nighttime sleep changing p | oattern | | | | | | |
| Consistent optimal | 328/3450 | Reference | - | Reference | - | Reference | - |
| Optimal to non-optimal | 189/1619 | 1.18 (1.00–1.41) | 0.051 | 1.20 (1.01–1.42) | 0.038 | 1.20 (1.02–1.43) | 0.032 |
| Non-optimal to optimal | 146/1257 | 1.21 (1.01–1.45) | 0.043 | 1.23 (1.02–1.48) | 0.026 | 1.23 (1.02–1.48) | 0.026 |
| Consistent non-optimal | 210/1579 | 1.35 (1.14–1.59) | < 0.001 | 1.37 (1.16–1.62) | < 0.001 | 1.36 (1.15–1.61) | < 0.001 |
| Sleep quality changing patt | tern | | | | | | |
| Consistent good | 395/4138 | Reference | - | Reference | - | Reference | - |
| Good to poor | 172/1286 | 1.41 (1.19–1.67) | < 0.001 | 1.42 (1.20–1.68) | < 0.001 | 1.42 (1.20–1.68) | < 0.001 |
| Poor to good | 117/1178 | 1.05 (0.86–1.28) | 0.622 | 1.06 (0.87–1.29) | 0.558 | 1.05 (0.87–1.28) | 0.604 |
| Consistent poor | 189/1303 | 1.56 (1.32–1.83) | < 0.001 | 1.58 (1.34–1.86) | < 0.001 | 1.55 (1.32–1.83) | < 0.001 |

Model 1: adjusted for age, sex, and BMI

Model 2: adjusted for age, sex, BMI, education, residence, smoking, alcohol consumption, and household income

Model 3: adjusted for age, sex, BMI, education, residence, smoking, alcohol consumption, household income, hypertension, diabetes, and physical activity BMI: body mass index; IRR: incidence rate ratio; CI: confidence interval



Fig. 2 The dose-response relationship between change in nighttime sleep duration, sleep quality and cardiovascular disease. Note. The lines represent incidence rate ratios (IRRs, solid lines) and 95% confidence intervals (CIs, long dashed lines) after multivariable adjustment for Model 3 based on the RCS models. The reference values (IRR=1) were set at the 0 change point, and the knots were set at the 10th, 50th, and 90th percentiles of the x-axis variable. The histograms represent the distribution of concentrations of x-axis variable

sleep behaviors remained associated with an increased risk of CVD events even after adjusting for traditional risk factors. Shifts from good to poor sleep behaviors could elevate the risk of CVD. Conversely, improved patterns of sleep quality helped to reduce the risk. We also considered the 10-year CVD risk scores, validating the additional contribution of sleep behaviors. Adjustments for other cardiovascular risk factors did not significantly alter the effect sizes, demonstrating that the impact of sleep behaviors on CVD was not solely dependent on the traditional cardiovascular risk factors. Moreover, although poor sleep behaviors might be a characteristic of depressive symptoms, the associations remained present when analyses excluded individuals with depressive symptoms. This study provides a straightforward assessment model for transition in sleep behaviors applicable to the general population. Further, our quantitative analysis among populations with insufficient sleep found that an increase in sleep time of up to 4.5 h helped reduce the risk of CVD, while further decreases in sleep or excessively long sleep durations were detrimental. On the other hand, transition in sleep quality showed a linear association with CVD, indicating that greater improvements in sleep quality yield greater benefits. These results can be more easily translated into practice for public reference.

To our knowledge, this is the first prospective study in an Asian population to simultaneously assess the associations between transition in nighttime sleep duration and sleep quality and the risk of CVD. We observed statistically significant interactions between transition in sleep behaviors and age, indicating that the adverse effects of consistently non-optimal nighttime sleep duration and sleep quality were more pronounced in the middle-aged population under 65 years. This is consistent with the conclusions of previous studies [14, 15]. Middle-aged individuals often experience greater stress and have relatively unstable circadian rhythms [16], which may predispose them to cardiovascular damage. Interestingly, while the contribution of persistent poor sleep behaviors to CVD appeared greater in women than in men, we did not observe a significant interaction by sex, despite previous studies suggesting greater susceptibility in women [17, 18]. However, characteristics of sleep structure and cycles, such as shortened sleep-onset latency, may serve as protective factors against CVD in women [14]. Overall, these results provide critical public health insights, underscoring the importance of focusing on improving sleep behaviors in the middle-aged population under 65.

Previous research consistently indicates that nighttime sleep duration and sleep quality can impact the cardiovascular system, primarily due to disruptions in circadian rhythms and the accumulation of cardiovascular risk factors [19]. Insufficient sleep or poor quality sleep may lead to insulin resistance, activation of the sympathetic nervous system, chronic inflammation, and oxidative stress [20, 21]. Clinically, these conditions can manifest more specifically as increased blood pressure, metabolic dysregulation, and disturbances in the hypothalamicpituitary-adrenocortical axis [22, 23]. In addition, sleep disorders are positively correlated with the systemic inflammatory response [24]. Such conditions accelerate the development of atherosclerosis, leading to subsequent CVD events [25, 26].

There are several potential limitations to this study. Firstly, as an observational study, the association between sleep behaviors and risk of CVD cannot be interpreted as causal relationships. Secondly, the utilization of selfreported sleep behaviors in our analysis inevitably introduces misclassification of exposure. Thirdly, nighttime sleep quality was assessed using a simple scoring method without specific subdivision into different aspects of sleep quality, such as early awakening, daytime sleepiness, insomnia, etc., which may not allow for a more refined classification of sleep quality. Nevertheless, studies have that these specific aspects of sleep quality interact with each other; a change in one aspect usually leads to changes in others, making a holistic assessment of sleep quality potentially more effective. Notably, a recent study has also demonstrated the effectiveness of this simple sleep quality scoring method [8]. Finally, it should be noted that the current classifications of nighttime sleep duration and sleep quality may not be applicable to all populations. Further studies are needed to support the current findings.

Conclusion

After considering for traditional CVD risk factors, participants with consistently poor or a good to poor transition in nighttime sleep behaviors are associated with an increased risk of CVD. These findings highlight the importance of assessing dynamic sleep behaviors in CVD risk evaluation among middle-aged and older adults. In addition to efforts to improve sleep behaviors, maintaining healthy sleep habits over time is also essential.

Abbreviations

| BMI | Body Mass Index |
|--------|--|
| CHARLS | China Health and Retirement Longitudinal Study |
| CI | Confidence Interval |
| CVD | Cardiovascular Diseases |
| IRR | Incidence Rate Ratio |
| hs-CRP | Hypersensitive C-Reactive Protein |
| | |

Supplementary Information

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Supplementary Material 1

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Author contributions

Conceptualization, YZ, JG, XH and HX; Methodology, YZ, JG and XH; Validation, HX; Formal analysis, YZ, JG and XH; Data curation, XH; Writing-original draft preparation, YZ, JG and XH; Writing-review and editing, HX; Supervision, HX; All authors read and agreed to the published version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The ethics of CHARLS study was approved by the Biomedical Ethics Review Committee of Peking University (Approval number: IRB00001052-11014 and IRB00001052-11015). All participants have signed a written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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