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Long sleep duration and risk of increased arterial stiffness in a Chinese population

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Abstract

To examine the relationship between self-reported sleep duration and arterial stiffness in a large Chinese population from Kailuan.

From July 2010 to December 2015, a total of 17,018 participants aged 18 to 98 years were enrolled after excluding those with a history of cerebrovascular events and coronary artery disease. Participants were divided into 5 categories according to self-reported night sleep duration: ≤5.0, 6.0, 7 (ref), 8, and ≥9.0 hours. A brachial-ankle pulse wave velocity ≥1400 cm/s was considered to represent arterial stiffness. Multivariate logistic regression models were used to calculate the odds ratio (OR) and confidence interval (CI) for arterial stiffness according to the sleep duration.

Using 7 hours of sleep as the reference group, the multivariable adjusted ORs (95% CI) for arterial stiffness were 1.00 (0.87–1.16), 1.00 (0.90–1.11), 1.0 (ref), 1.03 (0.93–1.14), and 1.48 (1.05–2.08) from the lowest to highest category of sleep duration, respectively. Secondary analysis showed no evidence of interactions between sleep duration and age/sex on the risk of arterial stiffness (*P*-interaction = .390/.198).

A long night sleep duration was associated with increased arterial stiffness.

Abbreviations: ANOVA = one-way analysis of variance, baPWV = Brachial-ankle pulse wave velocity, BMI = body mass index, CAC = coronary artery calcification, CI = confidence interval, CVD = cardiovascular disease, DBP = diastolic blood pressure, OR = odds ratio, SBP = systolic blood pressure.

Keywords: arterial stiffness, cross-sectional study, pulse wave velocity, sleep duration

1. Introduction

Increasing evidence suggests that sleep plays an important role in the pathogenesis and progression of cardiovascular disease (CVD).[1–5] Previous epidemiological studies including data from our study also show that both short and long sleep duration are associated with an increased risk of hypertension, diabetes, metabolic syndrome, cardiovascular disease, and all-cause mortality.[5–9] However, the specific mechanisms underlying the association between sleep duration and cardiovascular disease remain unclear, meriting further investigation. Brachial-ankle pulse wave velocity (baPWV) is a well-known noninvasive indicator of arterial stiffness, and it has been shown to be closely linked to an increased risk of cardiovascular disease.[10–13] A relationship between sleep duration and arterial stiffness may exist, as both share common determinants; for example, both are associated with inflammation pathways,[14,15] hormonal changes, and metabolic alterations[16]; both share common cardiovascular risk factors; both show increased sympathetic nervous activity[17]; and both are associated with an increased risk of CVD. Arterial stiffness as assessed by baPWV can be seen as a link between sleep duration and cardiovascular outcomes.

To date, few studies have examined the relationship between sleep duration and arterial stiffness. The results from Japanese and Taiwanese studies revealed a positive association between longer sleep duration and increased arterial stiffness, as measured by baPWV, in men, but not women.[18,19] In contrast, Sunbul et al[20] found that sleep deprivation was associated with increased baPWV in healthy adults, whereas Anujuo et al[21] found that neither short nor long sleep was associated with increased arterial stiffness. Thus, further studies on the relationship linking sleep duration with baPWV stratified by sex and age are required. To address the aforementioned uncertainty in relating sleep duration to an increased risk of baPWV, we conducted a cross-sectional analysis focusing primarily on arterial stiffness assessed by baPWV using comprehensive data from the Kailuan study.

2. Methods

2.1. Study design and participants

The Kailuan study was a prospective cohort study designed to investigate the association of risk factors and chronic disease. The Kailuan community is located at the center of the Kailuan Coal

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XL and QS have contributed equally to this work.

The authors have no conflicts of interest.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Industry in Hebei Province, China, and has approximately 7.2 million inhabitants. In brief, between June 2006 and October 2007 (visit 1), a total of 101,510 adult participants (81,110 men and 20,400 women, aged 18–98 years) were recruited from 11 hospitals in the Kailuan community. All participants underwent questionnaire assessment, clinical examinations, and laboratory tests upon enrollment and were followed up every 2 years, that is, in the years 2008 and 2009 (visit 2) and in the years 2010 and 2011 (visit 3).

Among them, 22,622 participants underwent baPWV assessment in visit 3. In the current analysis, we excluded 477 participants who had a diagnosis of myocardial infarction or stroke at or prior to visit 3. We further excluded 5127 participants who had missing data for sleep duration and potential covariates. A total of 17,018 participants were included in our analysis, which investigated the association between night sleep duration and arterial stiffness. Because our study is a cross-sectional study, and the baPWV assessment started at visit 3, based on the adequate accuracy of all the covariates, especially the biological covariates that may influence baPWV, we selected visit 3 as the baseline time. The protocol for the study was approved by the Ethics Committee of Kailuan General Hospital in compliance with the Declaration of Helsinki, and all participants provided informed written consent with their signatures.[22,23]

2.2. Assessment of sleep duration

Sleep duration was assessed through a self-reported answer to the question "How many hours of sleep have you had on average night in the preceding 3 months?" Based on the responses, sleep durations were categorized into 5 groups: ≤5, 6, 7, 8, and ≥9 hours. Additionally, participants were asked to answer "yes" or "no" to the question "Do you generally snore when you sleep?"

2.3. Assessment of potential covariates

Demographic and clinical characteristics, including age, sex, alcohol use, education, and disease history, were collected via self-reported questionnaires. Educational attainment was categorized as "illiterate or primary school," "middle school," or "high school or above." Information on the physical activity level (minutes of moderate or vigorous activity per week) was obtained from questionnaires and categorized as follows: ≥80 minutes (active), 1 to 79 (intermediate), and 0 (negative) minutes of moderate or vigorous activity per week.[23] Smoking status and drinking status were classified as "never," "former," or "current" according to self-reported information. Body mass index (BMI) was calculated as kg/m^2. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured 3 times in the seated position using a mercury sphygmomanometer, and the average of the 3 readings was used in the analyses.

All blood samples were tested using a Hitachi 747 autoanalyzer (Hitachi; Tokyo, Japan) at the central laboratory of the Kailuan General Hospital. Fasting blood glucose, triglyceride, total cholesterol, high-density lipoprotein, low-density lipoprotein, serum creatinine, and high-sensitivity C-reactive protein levels were measured.

2.4. Assessment of brachial-ankle pulse-wave velocity and ankle brachial index

BaPWV and ABI were measured by a BP-203RPEIII networked arteriosclerosis detection device (OMRON Healthcare (China) Co., Ltd.). The temperature of the examination room was kept between 22 and 25 °C, and the study participants were told to refrain from smoking and rest for at least 5 minutes before the measurement. Blood pressure cuffs were attached to the upper arm and ankle: the balloon sign of the upper arm cuff was aligned with the brachial artery, with the lower edge of the cuff placed at 2 to 3 cm from the cubital fossa transverse, and the balloon sign of the leg cuff placed was preaxially, with the lower edge of the cuff positioned 1 to 2 cm from the medial malleolus. The cardiechema collecting device was placed at the precordial region, with the electrocardiography acquisition device clipped to the left and right wrists. The measurement was repeated twice for each subject and the second readings of each body side baPWV were recorded. The mean values of the left- and right-side baPWV were used in further analysis, and the smaller values of the left- and right-side ABI were used in further analysis. BaPWV ≥1400 cm/s was considered to represent arterial stiffness.[24]

2.5. Statistical analysis

Continuous variables are expressed as the means ± standard deviations, and categorical variables are expressed as percentages. We compared the parameters according to the self-reported sleep duration. One-way analysis of variance (ANOVA) was used for non-paired samples of normally distributed parameters and the Kruskal-Wallis test for non-parametric variables. A chi-squared test was used to compare categorical variables. A multivariate logistic regression analysis was performed using four models. Model 1 was adjusted for age and sex, and model 2 was adjusted for age, sex, education level, smoking status, drinking status, and physical activity. In addition to the independent parameters analyzed in model 2, a third model included BMI, systolic blood pressure, diastolic blood pressure, fasting blood glucose, triglyceride, total cholesterol, high-density lipoprotein, estimated glomerular filtration rate, and resting heart rate. Model 4 was stratified by hospital and adjusted for the variables in Model 3 plus high sensitive C-reactive protein; snoring status; and whether the participants were taking lipid-lowering drugs, antihypertensive drugs, or hypoglycemic agents. We used multivariable logistic regression modeling to calculate the odds ratios (OR) and 95% confidence intervals (CI) of increased baPWV (a group with a 7-hour sleep duration was used as the reference category). In secondary analyses, we evaluated whether there were statistically significant interactions between sleep duration and age/sex on the risk of arterial stiffness. All interactions were analyzed using multivariable logistic regression modeling. Statistical analyses were performed using SAS 9.4 (SAS Institute; Cary, NC). All statistical tests were 2-sided, and the significance level was set at 0.05.

3. Results

At baseline, participants were aged 18 to 98 years (mean 50.9). The percent of participants who reported sleeping for ≤5, 6, 7, 8, and ≥9 hours per night were 10.3%, 26.2%, 25.6%, 36.4%, and 1.5%, respectively. The characteristics of participants are reported in Table 1 by the categories of sleep duration. Significant associations were found among sleep duration and age, sex, education level, smoking status, drinking status, physical activity, BMI, systolic blood pressure, diastolic blood pressure, fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein, estimated glomerular filtration rate, high sensitive

Table 1

Baseline characteristics according to sleep duration.

Variable	Total (n = 17018)	Sleep duration, h)				
		≤5 (n = 1752)	6.0 (n = 4458)	7 (n = 4365)	8.0 (n = 6189)	≥9 (n = 254)
Age, y	50.94 ± 11.74	56.24 ± 11.81	52.09 ± 11.39	50.15 ± 11.55	49.18 ± 11.48	50.30 ± 13.75
Males, n (%)	10436 (61.32)	1191 (67.98)	2980 (66.85)	2811 (64.40)	3316 (53.58)	138 (54.33)
High school or above, n (%)	7330 (43.07)	595 (33.96)	2025 (45.42)	2067 (47.35)	2543 (41.09)	100 (39.37)
Current smokers, n (%)	5949 (34.96)	764 (43.61)	1871 (41.97)	1690 (38.72)	1543 (24.93)	81 (31.89)
Current alcohol use, n (%)	5725 (33.64)	709 (40.47)	1842 (41.32)	1611 (36.91)	1489 (24.06)	74 (29.13)
Active physical activity, n (%)	2610 (15.34)	360 (20.55)	805 (18.06)	751 (17.21)	654 (10.57)	40 (15.75)
Snoring, n (%)	2625 (15.42)	452 (25.80)	860 (19.29)	619 (14.18)	639 (10.32)	55 (21.65)
Drugs, n (%)	2164 (12.72)	355 (20.26)	669 (15.01)	564 (12.92)	537 (8.68)	39 (15.35)
Body mass index, kg/m^2	24.87 ± 3.33	25.11 ± 3.43	24.97 ± 3.22	24.72 ± 3.28	24.83 ± 3.38	24.86 ± 3.72
Resting heart rate, beat/min	73.15 ± 9.92	72.66 ± 10.40	72.50 ± 9.84	73.14 ± 9.96	73.76 ± 9.80	73.42 ± 9.20
Systolic blood pressure, mm Hg	127.57 ± 19.03	132.09 ± 19.83	128.53 ± 18.74	126.59 ± 18.54	126.37 ± 19.10	125.87 ± 19.69
Diastolic blood pressure, mm Hg	82.74 ± 11.17	83.99 ± 11.20	83.19 ± 11.21	82.29 ± 10.95	82.41 ± 11.23	81.67 ± 11.66
Fasting blood glucose, mmol/L	5.53 ± 1.61	5.65 ± 1.54	5.51 ± 1.35	5.51 ± 1.54	5.52 ± 1.84	5.57 ± 1.80
Total cholesterol, mmol/L	4.99 ± 1.47	5.20 ± 1.95	5.06 ± 1.36	4.99 ± 1.28	4.88 ± 1.53	5.07 ± 1.11
Triglycerides, mmol/L	1.72 ± 1.82	1.79 ± 1.77	1.78 ± 1.78	1.72 ± 2.11	1.64 ± 1.64	1.73 ± 1.72
High-density lipoprotein, mmol/L	1.60 ± 0.61	1.64 ± 0.46	1.64 ± 0.79	1.61 ± 0.68	1.55 ± 0.44	1.61 ± 0.45
Estimated glomerular filtration rate, mL/min	94.58 ± 18.97	93.93 ± 18.55	95.59 ± 18.67	95.63 ± 18.71	93.21 ± 19.44	96.55 ± 17.53
High sensitivity C-reactive protein, mg/L	0.95 (0.44–2.00)	0.97 (0.53–2.10)	0.95 (0.50–2.00)	0.95 (0.40–1.81)	0.95 (0.40–2.06)	1.00 (0.60–2.11)
Brachial-ankle pulse wave velocity	1482.03 ± 342.10	1538.17 ± 365.50	1457.94 ± 308.63	1429.46 ± 310.58	1415.89 ± 309.48	1449.13 ± 361.67

Drug, those taking lipid-lowering drugs, antihypertensive drugs, hypoglycemic agents.

C-reactive protein, snoring status, taking lipid-lowering drugs, taking antihypertensive drugs, and taking hypoglycemic agents.

Compared with participants without arterial stiffness, those with arterial stiffness were significantly older, contained a higher percentage of men, had higher BMI, higher systolic blood pressure, higher diastolic blood pressure, higher fasting blood glucose, higher resting heart rate, higher lipid level, higher sensitivity to C-reactive protein, higher proportion of smokers, and had a more elevated snoring status (Table 2).

Table 3 shows the odds ratios (ORs) for arterial stiffness according to sleep duration in the total population. A total of 8930 (52.5%) cases of arterial stiffness were observed: 6602 in men and 2328 in women. The crude prevalences of arterial stiffness were 64.8%, 55.4%, 50.7%, 48.0%, and 54.7% for people reporting average sleep durations of ≤5, 6, 7, 8, and ≥9 hours, respectively. Using 7 hours of sleep as the reference group (model 4), multivariable adjusted ORs (95% CI) for arterial stiffness were 1.00 (0.87–1.16), 1.00 (0.90–1.11), 1.0 (ref), 1.03 (0.93–1.14), and 1.48 (1.05–2.08) from the lowest to highest category of sleep duration, respectively (Table 3). Moreover, the association between sleep duration and the risk of arterial stiffness remained significant in the total population, even after excluding individuals taking lipid-lowering drugs, antihypertensive drugs, and hypoglycemic agents from the analysis.

We repeated the analysis stratified by different age and sex groups (Table 4). Women who slept ≥9 hours were associated with increased baPWV (OR, 1.85; 95% CI, 1.08–3.18). While this association was not significant among men (OR, 1.41; 95% CI, 0.90–2.22), a formal test for difference by sex also did not find statistical significance (*P*-interaction = .198). Participants aged <60 years and who slept ≥9 hours were associated with increased baPWV (OR, 1.52; 95% CI, 1.06–2.18). While this association was not significant among participants ≥60 years (OR, 1.24; 95% CI, 0.42–3.70), another test for differences by age also did not find statistical significance (*P*-interaction = .390).

Table 2

Differences in baseline characteristics between patients with and without arterial stiffness.

Variable	baPWV≥1400	baPWV<1400	*P* value
Age, y	56.23 ± 11.62	45.09 ± 8.69	<.001
Males, n (%)	6576 (73.64)	3860 (47.73)	<.001
High school or above, n (%)	3080 (34.49)	4250 (52.55)	<.001
Current smokers, n (%)	3717 (41.62)	2232 (27.60)	<.001
Current alcohol use, n (%)	3456 (38.70)	2269 (28.05)	<.001
Active physical activity, n (%)	1677 (18.78)	933 (11.54)	<.001
Snoring, n (%)	1773 (19.85)	852 (10.53)	<.001
Drug, n (%)	1852 (20.74)	312 (3.86)	<.001
Body mass index, kg/m^2	25.28 ± 3.30	24.41 ± 3.30	<.001
Resting heart rate, beat/min	74.34 ± 10.66	71.84 ± 8.84	<.001
Systolic blood pressure, mmHg	136.10 ± 18.72	118.17 ± 14.38	<.001
Diastolic blood pressure, mmHg	86.41 ± 11.15	78.68 ± 9.70	<.001
Fasting blood glucose, mmol/L	5.81 ± 1.77	5.23 ± 1.35	<.001
Total cholesterol, mmol/L	5.15 ± 1.62	4.82 ± 1.27	<.001
Triglycerides, mmol/L	1.94 ± 2.08	1.47 ± 1.45	<.001
High-density lipoprotein, mmol/L	1.58 ± 0.70	1.62 ± 0.51	<.001
Estimated glomerular filtration rate, ml/min	91.10 ± 18.77	98.42 ± 18.43	<.001
High sensitivity C-reactive protein, mg/L	1.00 (0.58–2.35)	0.80 (0.40–1.60)	<.001

Drug, those taking lipid-lowering drugs, antihypertensive drugs, hypoglycemic agents.

4. Discussion

In this cross-sectional, population-based cohort study, long sleep duration was associated with arterial stiffness assessed by baPWV in the general Chinese population, independent of cardiovascular risk factors (age, sex, smoking status, drinking status, physical activity, BMI, blood pressure, fasting plasma glucose, lipid, estimated glomerular filtration rate, and resting heart rate), socioeconomic characteristics (education level), inflammatory markers (C-reactive protein), and subjective sleep quality (snoring status). Participants who reported 7 hours of sleep had lower baPWV when compared with participants with a

Table 3

Odds ratios (95% CI) for arterial stiffness according to sleep duration in the Kailuan study.

	Sleep duration, h					
Total	**≤5**	**6**	**7**	**8**	**≥9**	***P* for interaction**
Case, prevalence	1136 (64.84)	2471 (55.43)	2212 (50.68)	2972 (48.02)	139 (54.72)	
Model 1	1.08 (0.95–1.24)	1.02 (0.92–1.12)	reference	1.08 (0.98–1.18)	1.47 (1.08–2.01)	
Model 2	1.05 (0.92–1.20)	1.01 (0.92–1.11)	reference	1.08 (0.98–1.18)	1.45 (1.06–1.98)	
Model 3	1.01 (0.88–1.17)	1.00 (0.90–1.12)	reference	1.01 (0.92–1.12)	1.48 (1.06–2.08)	
Model 4	1.00 (0.87–1.16)	1.00 (0.90–1.11)	reference	1.03 (0.93–1.14)	1.48 (1.05–2.08)	
Sensitivity analysis*	0.98 (0.84–1.14)	1.02 (0.91–1.14)	reference	1.04 (0.94–1.16)	1.53 (1.08–2.19)	

Model 1: Adjusted for age and sex.

Model 2: Adjusted for age, sex, level of education, smoking, alcohol, and physical activity.

Model 3: Adjusted for variables in Model 2 plus body mass index, systolic blood pressure, diastolic blood pressure, fasting blood glucose, triglyceride, total cholesterol, high-density lipoprotein, estimated glomerular filtration rate, and resting heart rate.

Model 4: Adjusted for variables in Model 3 plus high sensitive C-reactive protein, snoring status, and those taking lipid-lowering drugs, antihypertensive drugs, and hypoglycemic agents.

* Adjusted for variables in Model 4 with the further exclusion of individuals taking lipid-lowering drugs, antihypertensive drugs, or hypoglycemic agents.

longer sleep duration. A sensitivity analysis further confirmed these findings.

Results from Japanese civil servants indicate that, compared with the reference group of 7 hours of sleep, ≥9 hours is significantly associated with higher baPWV.[18] Other results from Taiwan also showed that only long sleep duration (>8 hours) was associated with a higher risk of increased arterial stiffness in middle-aged civil servants.[19] Both studies conducted in Asian populations found a positive association of long sleep duration with baPWV. Moreover, Wolff et al[25] reported that, compared with 8 hours sleep, both shorter (5 hours) and longer (≥10 hours) sleep durations are associated with higher carotid intima-media thickness in a combined study of men and women in Germany. Kim et al[26] also found that extreme sleep duration and poor subjective sleep quality were associated with an increased prevalence of coronary artery calcification (CAC) and higher baPWV. Our results are in line with these findings and showed that participants with long sleep duration also had increased baPWV compared with those who reported 7 hours of sleep. Furthermore, we obtained information on medication for hypertension, hyperlipidemia, and diabetes that could influence baPWV as a putative confounding factor, and a significant

Table 4

Odds ratios (95% CI) for arterial stiffness according to sleep duration stratified by sex and age in the Kailuan study.

	Sleep duration, h					
	≤5	**6.0**	**7**	**8.0**	**≥9**	***P* for interaction**
Women						.198
Case, prevalence	329 (59.39)	593 (40.56)	502 (32.70)	863 (30.13)	41 (35.34)	
Model 1	1.17 (0.91–1.50)	1.05 (0.87–1.27)	reference	1.18 (1.00–1.40)	1.90 (1.16–3.12)	
Model 2	1.14 (0.89–1.47)	1.06 (0.88–1.28)	reference	1.13 (0.96–1.34)	1.84 (1.12–3.04)	
Model 3	1.10 (0.84–1.46)	1.02 (0.83–1.26)	reference	1.09 (0.90–1.31)	1.78 (1.03–3.07)	
Model 4	1.09 (0.82–1.44)	1.02 (0.83–1.26)	reference	1.12 (0.92–1.35)	1.85 (1.08–3.18)	
Men						
Case, prevalence	807 (67.36)	1878 (62.68)	1710 (60.42)	2109 (63.43)	98 (71.01)	
Model 1	1.00 (0.86–1.16)	0.99 (0.89–1.11)	reference	1.10 (0.98–1.23)	1.40 (0.93–2.12)	
Model 2	0.97 (0.83–1.13)	0.98 (0.88–1.10)	reference	1.12 (1.00–1.26)	1.38 (0.92–2.10)	
Model 3	0.95 (0.80–1.12)	0.99 (0.88–1.12)	reference	1.04 (0.92–1.17)	1.45 (0.93–2.27)	
Model 4	0.94 (0.79–1.11)	0.98 (0.87–1.11)	reference	1.05 (0.93–1.18)	1.41 (0.90–2.22)	
Age <60						.390
Case, prevalence	619 (52.46)	1623 (46.46)	1511 (42.32)	2036 (39.83)	84 (43.08)	
Model 1	1.06 (0.92–1.22)	1.02 (0.92–1.14)	reference	1.11 (1.01–1.22)	1.49 (1.07–2.07)	
Model 2	1.02 (0.89–1.18)	1.02 (0.92–1.13)	reference	1.11 (1.01–1.23)	1.46 (1.05–2.03)	
Model 3	1.00 (0.86–1.17)	1.01 (0.90–1.13)	reference	1.04 (0.94–1.16)	1.52 (1.06–2.17)	
Model 4	0.99 (0.85–1.16)	1.00 (0.90–1.12)	reference	1.05 (0.94–1.17)	1.52 (1.06–2.18)	
Age ≥60						
Case, prevalence	517 (90.38)	848 (87.88)	701 (88.18)	936 (86.91)	55 (93.22)	
Model 1	1.03 (0.71–1.48)	0.92 (0.68–1.24)	reference	0.92 (0.69–1.23)	1.83 (0.63–5.30)	
Model 2	1.01 (0.70–1.46)	0.91 (0.67–1.23)	reference	0.89 (0.66–1.20)	1.76 (0.61–5.13)	
Model 3	0.97 (0.66–1.43)	0.92 (0.67–1.26)	reference	0.88 (0.64–1.20)	1.28 (0.43–3.81)	
Model 4	0.94 (0.64–1.39)	0.92 (0.67–1.27)	reference	0.92 (0.67–1.26)	1.24 (0.42–3.70)	

Model 1: Adjusted for age and sex.

Model 2: Adjusted for age, sex, level of education, smoking, alcohol, and physical activity.

Model 3: Adjusted for variables in Model 2 plus body mass index, systolic blood pressure, diastolic blood pressure, fasting blood glucose, triglyceride, total cholesterol, high-density lipoprotein, estimated glomerular filtration rate, and resting heart rate.

Model 4: Adjusted for variables in Model 3 plus high sensitive C-reactive protein, snoring status, and those taking lipid-lowering drugs, antihypertensive drugs, and hypoglycemic agents.

association between long sleep duration and increased baPWV was maintained after adjustment for these factors.

Previous studies conducted in Japan and Taiwan revealed a positive association between longer sleep duration and increased arterial stiffness, as measured by baPWV, in men, but not women.[18,19] Different with their studies, our study found an association between long sleep duration and increased baPWV that was significant in women and remained after the patients taking lipid-lowering drugs, antihypertensive drugs, and hypoglycemic agents were excluded from the analysis, as these conditions can affect baPWV. However, we did not find such a relationship in men. Differences in the age distribution of the study population, lifestyle, socioeconomic status, incomplete control for confounding factors, or different categorization and cutoff points of sleep duration may explain the different associations thus far. In addition, the possible reason for the differential association of long sleep duration with arterial stiffness between men and women is not fully understood. The differences in hormone secretion, health behaviors, and psychological factors between men and women might account for this differential association of long sleep duration with arterial stiffness. However, the lack of comprehensive information on the biological differences between men and women limits us in further investigating whether the relation could be modified or mediated by these factors. Furthermore, no evidence of the interaction between sleep duration and sex on the risk of baPWV was found (*P*-interaction = .198).

As we know, both structural and functional changes in the arteries occur with the aging process. Sympathetic nerve activity elevation,[27] endothelial dysfunction,[28] and proinflammatory status[29] induce vasoconstriction and smooth muscle cell hypertrophy, and then may lead to increased arterial stiffness with aging.[30] In addition, sleep behaviors that differ between younger and older participants could have biased the association; thus, we further repeated the analysis stratified by age group. However, no evidence was found of an interaction between sleep duration and age on the risk of baPWV (*P*-interaction = .390).

The mechanisms underlying sleep and arterial stiffness are not clear. There are several potential explanations for why prolonged sleep duration may be a risk factor for increased arterial stiffness. First, the relationship between long sleep duration with cardiovascular outcomes and all-cause mortality may be related to the following factors: sleep fragmentation; fatigue; immune function; depression; and underlying disease processes such as sleep apnea, heart disease, and failing health.[31–33] Second, inflammatory pathways may be another mechanism for the positive relationship between long sleep duration and increased arterial stiffness because the inflammatory process will cause the dysregulation of collagen and elastin fibers of the vascular wall, leading to arterial stiffness.[34] In addition, poor sleep quality was found to be associated with insulin resistance,[35] increased sympathetic activity,[36] and endothelial function impairment.[37] Further research is needed to explore their mechanism.

The strengths of our study include the large sample size, a broad spectrum of potential confounding parameters, and the use of a relatively healthy population without a history of CVD, in which the associations between sleep duration and markers of subclinical arterial disease are less likely to be confounded by comorbidities or medication use. However, the potential limitations of our study should also be discussed here. First, we only collected information on night sleep duration by self-reported questionnaires without polysomnographic assessments. Data on midday naps, excessive daytime sleepiness, sleep quality and the use of sleeping pills were also not collected in our study. Thus, night sleep duration may be different from whole day sleep duration, especially in China, where napping and poor sleep quality are not unusual. Furthermore, we did not exclude participants with obstructive sleep apnea syndrome, which is associated with disrupted sleep/sleep deprivation, excessive daytime sleepiness, and a high risk of arterial stiffness.[38] Therefore, we adjusted snoring status as a confounder in the statistical analysis, although the effect of obstructive sleep apnea may not have been fully controlled. Second, we did not collect sufficient information on the pre- or post-menopause status of women and whether the values of baPWV were lower in hormone therapy users.[39] Finally, arterial stiffness was only assessed by baPWV, without carotid intima-media thickness and coronary artery calcification, which limited our extensionality to other populations assessed by different assessment methods.

In conclusion, the results of our study demonstrate that long sleep duration was associated with elevated values of baPWV as a marker of arterial stiffness. It underscores the importance of adequate sleep quantity and quality to maintain cardiovascular health and supports the need to consider subjects with a long sleep duration or poor subjective quality of sleep to be at high risk for cardiovascular diseases. Therefore, regulating the night hours of sleep may contribute to a reduction in the incidence of cardiovascular diseases.

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Author contributions

Xiaoxue Liu and Qiaofeng Song wrote the manuscript and interpreted data. Xiaoxue Liu analyzed the data. Shouling Wu reviewed/edited the manuscript. Xizhu Wang contributed to the discussion and reviewed/edited the manuscript.

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