

# Poor Sleep Pattern Might Increase the Prevalence of Abdominal Aortic Calcification in US Adults: Data from the NHANES (2013-2014)

Yunqing Chen<sup>1</sup>, Shiyong Xu<sup>2</sup>, Mingxia Duan<sup>2</sup>, Liang Shen<sup>2</sup>

<sup>1</sup>Department of Infectious Diseases, Affiliated Hospital of Jiaying University, Jiaying, Zhejiang, People's Republic of China; <sup>2</sup>Department of Cardiology, Affiliated Hospital of Jiaying University, Jiaying, Zhejiang, People's Republic of China

Correspondence: Liang Shen, Affiliated Hospital of Jiaying University, No. 1882 Zhonghuan South Road, Jiaying, Zhejiang, People's Republic of China, Tel +86 18868042552, Email shenliang8011@126.com

**Background:** Abdominal aortic calcification (AAC) is a reliable predictor of cardiovascular events. Sleep is an essential component of cardiovascular health. This study aimed to assess the association between sleep patterns (including sleep duration, trouble sleeping, and sleep disorders) and the risk of AAC.

**Methods:** A total of 2801 participants from the 2013 to 2014 National Health and Nutrition Examination Survey (NHANES) 2013–2014 cycle were included in the study. AAC score was assessed using the Kauppila scoring system. Sleep patterns were defined according to the overall sleep score, which was calculated based on the sleep duration, self-reported trouble sleeping, and sleep disorders. Weighted multivariable linear regression models were used to analyze the association between sleep patterns and AAC.

**Results:** AAC prevalence was higher in older age groups, smokers, those with hypertension or diabetes, and those with less physical activity, lower eGFR, or higher levels of total 25-hydroxyvitamin D, serum uric acid, and serum calcium. Compared with the healthy sleep pattern group, participants in the poor sleep pattern group had a higher prevalence of AAC (odds ratio [OR] = 1.664, 95% CI: 1.286–2.152) and a higher prevalence of severe AAC (OR = 2.179, 95% CI: 1.539–3.087). After adjusting for potential confounders, the poor sleep pattern group remained significantly associated with a higher risk of AAC (OR = 1.657, 95% CI: 1.235–2.222) and severe AAC (OR = 2.374, 95% CI: 1.616–3.488).

**Conclusion:** Sleep patterns were related to the risk of AAC in middle-aged and elderly populations. Poor sleep patterns may increase AAC prevalence.

**Keywords:** abdominal aortic calcification, NHANES, sleep pattern, cardiovascular event

## Introduction

Cardiovascular disease (CVD) is the leading cause of mortality worldwide. Abdominal aortic calcification (AAC), defined as abnormal deposition of calcium phosphate complexes in the abdominal aorta, is a common manifestation of vascular calcification (VC).<sup>1</sup> Previous studies have demonstrated that AAC is a reliable marker for atherosclerotic CVD and can predict cardiovascular events.<sup>2–5</sup>

Sleep is an important component of cardiovascular health. In 2022, the American Heart Association (AHA) proposed the concept of “Life’s Essential 8” by adding sleep as one of the eight components that build optimal CV health.<sup>6</sup> Sleep disorders are associated with numerous CVDs including coronary artery disease, heart failure, and arrhythmias.<sup>7–10</sup>

Previous studies investigated the relationship between sleep and coronary artery calcification.<sup>11,12</sup> In addition, poor sleep quality is associated with aortic calcification in middle-aged women.<sup>13</sup> Recently, Wang et al reported that long sleep duration is associated with AAC in male adults with chronic kidney disease (CKD).<sup>14</sup> However, it remains unclear whether this relationship also applies to a broader population.

This study aimed to investigate the association between combined sleep factors and AAC in the US population using the 2013–2014 National Health and Nutrition Examination Survey (NHANES).

## Methods

All data are publicly available and can be accessed from NHANES (<https://www.cdc.gov/nchs/nhanes/index.htm>). The National Center for Health Statistics Research Ethics Review Board approved all NHANES protocols, and this study was also approved by the Ethics Committee of the First Hospital of Jiaying, Jiaying, China.

## Study Population

In this study, we analyzed data from the NHANES, focusing on the 2013–2014 cycle. In total, 10175 participants were enrolled, after excluding those with missing AAC data ( $n=7035$ ), missing sleep data ( $n=15$ ), and missing relevant covariate data ( $n=324$ ), a total of 2801 participants were included (Figure 1).

## Assessment of Sleep Pattern

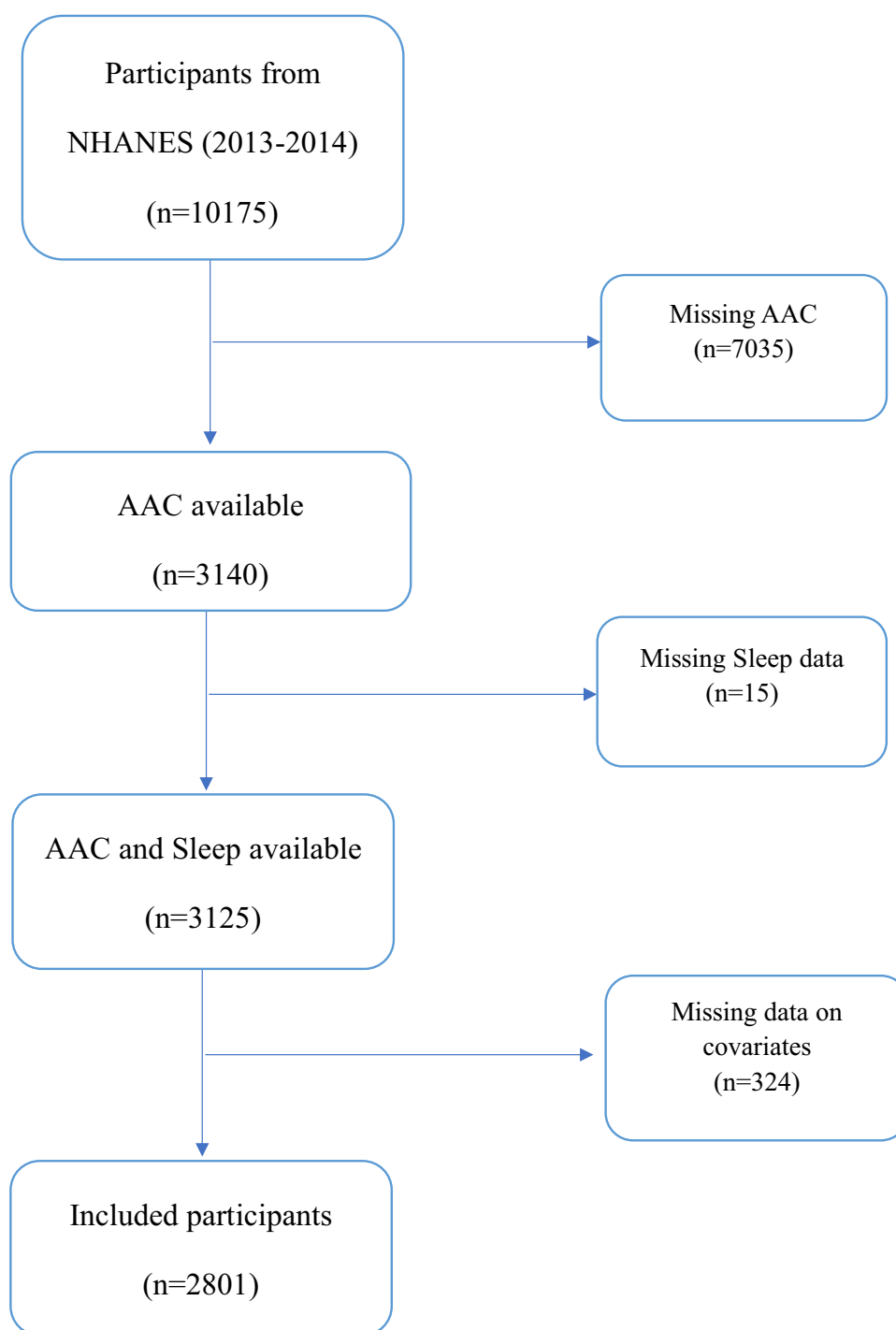
Sleep patterns were assessed, as previous study.<sup>15</sup> Briefly, the sleep duration was divided into three groups: short (< 7 h per night), normal (7–9 h per night), and long (>9 h per night). The answers to Have you ever told a doctor or other health professionals that you have trouble sleeping? and “Have you/Has SP ever been told by a doctor or other health professional that you have a sleep disorder?” was used to assess trouble sleeping and other sleep disorders. A score of 1 or 0 was assigned to each sleep factor according to a low or high risk, respectively. Low-risk sleep was defined as sleeping 7–9 hours per night without trouble sleeping or sleep disorders. Finally, the following three sleep patterns were obtained: poor sleep ( $0 \leq \text{sleep score} \leq 1$ ), intermediate sleep (sleep score = 2), and healthy sleep (sleep score = 3).

## Evaluation of AAC and Severe AAC

AAC was scanned using dual-energy X-ray absorptiometry (DXA) and semi-quantified using the Kauppila score system.<sup>16</sup> Briefly, the anterior and posterior aortic walls were divided into four segments, and each segment was assigned a score (0–6) according to the degree of calcium deposition. AAC was diagnosed when the total AAC score was >0 and severe AAC was defined as a total AAC score >6.

## Assessment of Covariates

Other covariates including age, sex, race, marital status, education level, ratio of family income to poverty (RIP), lifestyle risk factors (smoking, alcohol, and physical activity), general health condition (BMI, hypertension, and diabetes), and biochemical parameters (total cholesterol, total triglycerides, total 25-hydroxyvitamin D, serum alkaline phosphatase, eGFR, serum uric acid, serum phosphorus, and serum calcium) were obtained through interviews and physical examinations. In brief, demographic characteristics and lifestyle risk factors were collected using household interview and mobile examination center (MEC) questionnaires. Physical examination information, biochemical parameters were derived from medical examinations and laboratory assessments in the MEC. The selection of all covariates was based on previous studies.<sup>17–19</sup> Race was categorized into five groups: Mexican American, Non-Hispanic White, Non-Hispanic Black, Other Hispanic, and Other others. Educational level was divided into less than high school, high school and higher than high school. Marital status included marriage or living with a partner or a single. PIR was classified into three groups: <1.30, 1.31, 3.50, and  $\geq 3.50$ .<sup>18</sup> Smokers were defined as those who had smoked more than 100 cigarettes in their lifetime. Drinkers were defined as those who drank at least 12 alcoholic drinks per year. BMI was categorized as under/normal weight (<25.0 kg/m<sup>2</sup>), overweight (25.0–30.0 kg/m<sup>2</sup>), and obesity ( $\geq 30.0$  kg/m<sup>2</sup>).<sup>20</sup> Physical activity was classified into two groups, active and inactive, according to the Physical Activity Guidelines recommendation of  $\geq 75$  min/week of vigorous or  $\geq 150$  min/week of moderate physical activity.<sup>21</sup> The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration creatinine equation.<sup>22</sup> Hypertension was defined as a blood pressure measurement of  $\geq 140/90$  mmHg, self-reported physician-diagnosed hypertension, or the use of antihypertensive medications.<sup>23</sup> Diabetes was defined as a fasting glucose level of  $\geq 7.0$  mmol/L, or a plasma glucose level of  $\geq 11.1$  mmol/L 2 h after an oral glucose tolerance test, self-reported physician-diagnosed diabetes, or taking oral hypoglycemic agents or insulin.<sup>17</sup>



**Figure 1** Flow chart of the participants' enrollment.

**Abbreviations:** NHANES, National Health and Nutrition Examination Survey; AAC, abdominal aortic calcification.

## Statistical Analysis

In accordance with the NHANES analytic guidelines, all statistical analyses were conducted using appropriate sampling weights. We adjusted for the complex, multistage, stratified, cluster-sampling design of the sample by applying the survey weight from the MEC (*wtmec2yr*). Continuous variables are presented as mean  $\pm$  standard deviation or median (quartiles: Q1, Q3) depending on whether the data was normal distribution, and weighted linear regression or Mann-Whitney U or Kruskal-Wallis H were used to examine the differences between the two groups. Categorical variables

were expressed as weighted percentages (95% CI) and compared using the chi-square test. Multivariate regression models were used to assess the association between sleep pattern and AAC. Model 1 shows the unadjusted results. Model 2 was adjusted for age, sex, race, marital status, and educational level. Model 3 was further adjusted for PIR, BMI, smoking and drinking status, diabetes, hypertension, physical activity status, eGFR, serum uric acid, serum phosphorus, total 25-hydroxyvitamin D, and total serum calcium levels.

All statistical analyses were performed using STATA (version 16.0; StataCorp, College Station, TX, USA) and SPSS 25.0 (IBM SPSS, Turkey). Two-tailed P value less than 0.05 was considered statistically significant.

## Results

### Baseline Characteristics

The baseline characteristics of the participants are summarized in Table 1 and Table 2. Significant differences were found between participants with and without AAC in terms of age, race, marital status, PIR, smoking status, physical activity,

**Table 1** Characteristics of the Study Population

Characteristic	All (n=2801)	AAC		p-value	Severe AAC		p-value
		Yes (n=976)	No (n=1825)		Yes (n=316)	No (n=2485)	
Age, year	59.1 ± 11.9	63.9 ± 11.9	56.4 ± 11.0	< 0.001	68.3 ± 11.6	57.9 ± 11.4	< 0.001
Gender, n (%)				0.168			0.074
Male	1347 (48.1)	452 (46.3)	895 (49.0)		137 (43.4)	1210 (48.7)	
Female	1454 (51.9)	524 (53.7)	930 (51.0)		179 (56.6)	1275 (51.3)	
Race, n (%)				< 0.001			< 0.001
Mexican American	373 (13.3)	113 (11.6)	260 (14.2)		32 (10.1)	341 (13.7)	
Non-Hispanic White	269 (9.6)	85 (8.7)	184 (10.1)		20 (6.3)	249 (10.0)	
Non-Hispanic Black	1268 (45.3)	514 (52.7)	754 (41.3)		192 (60.8)	1076 (43.3)	
Other Hispanic	532 (19.0)	150 (15.4)	382 (20.9)		41 (13.0)	491 (19.8)	
Others	359 (12.8)	114 (11.6)	245 (13.5)		31 (9.8)	328 (13.2)	
Education level, n (%)				0.300			0.157
Less than high school	614 (21.9)	214 (21.9)	400 (21.9)		75 (23.7)	539 (21.7)	
High school	638 (22.8)	238 (24.4)	400 (21.9)		82 (25.9)	556 (22.4)	
More than high school	1549 (55.3)	524 (53.7)	1025 (56.2)		159 (50.3)	1390 (55.9)	
Marital status, n (%)				0.001			0< 0.001
Married/Living with partner	1789 (63.9)	583 (59.7)	1206 (66.1)		165 (52.2)	1624 (65.4)	
Single	1012 (36.1)	393 (40.3)	619 (33.9)		151 (47.8)	861 (34.6)	
PIR				0.044			0.223
<1.30	764 (27.3)	282 (28.9)	482 (26.4)		96 (30.4)	668 (26.9)	
1.31 to 3.50	907 (32.4)	335 (34.3)	572 (31.3)		109 (34.5)	798 (32.1)	
≥3.50	916 (32.7)	288 (29.5)	628 (34.4)		92 (29.1)	824 (33.2)	
Smoker, n (%)	1295 (46.2)	525 (53.8)	770 (42.2)	< 0.001	190 (60.1)	1105 (44.5)	< 0.001
Alcohol drinker, n (%)	2005 (71.6)	699 (71.6)	1306 (71.6)	0.974	226 (71.5)	1779 (71.6)	0.979
BMI status, n (%)				0.340			0.145
Under/normal weight	763 (27.2)	264 (27.0)	499 (27.3)		89 (28.2)	674 (27.1)	
Overweight	1026 (36.6)	378 (38.7)	648 (35.5)		128 (40.5)	898 (36.1)	
Obesity	996 (35.6)	329 (33.7)	667 (36.5)		99 (31.3)	897 (36.1)	
Physical activity, n (%)	963 (34.4)	284 (29.1)	679 (37.2)	< 0.001	81 (25.6)	882 (35.5)	0.001
Sleep patterns				< 0.001			< 0.001
Poor	612 (21.8)	276 (28.3)	336 (18.4)		106 (33.5)	506 (20.4)	
Intermediate	958 (34.2)	299 (30.6)	659 (36.1)		88 (27.8)	870 (35.1)	
Healthy	1231 (44.0)	401 (41.1)	830 (45.4)		122 (38.6)	1109 (44.6)	
Hypertension, n (%)	1577 (56.3)	669 (68.5)	908 (49.8)	< 0.001	249 (78.8)	1328 (53.4)	< 0.001
Diabetes, n (%)	659 (23.5)	319 (32.7)	340 (18.6)	< 0.001	124 (39.2)	535 (21.5)	0< 0.001
TC, mmol/L	4.99 (4.29, 5.74)	4.91 (4.26, 5.74)	5.04 (4.34, 5.74)	0.054	4.76 (4.06, 5.72)	4.95 (4.12, 5.74)	0.059

(Continued)

**Table 1** (Continued).

Characteristic	All (n=2801)	AAC		p-value	Severe AAC		p-value
		Yes (n=976)	No (n=1825)		Yes (n=316)	No (n=2485)	
TG, mmol/L	1.43 (0.95, 2.24)	1.51 (1.06, 2.34)	1.40 (0.91, 2.17)	0.001	1.56 (1.09, 2.53)	1.42 (0.95, 2.19)	0.003
Total 25-hydroxyvitamin D, nmol/L	67.4 (51.0, 86.2)	70.0 (53.1, 91.6)	66.5 (50.1, 84.1)	< 0.001	75.7 (57.6, 95.3)	66.7 (50.5, 85.0)	< 0.001
Serum ALP, IU/L	64 (53, 79)	65 (53, 79)	64 (53, 79)	0.234	65 (51, 79)	64 (53, 78)	0.705
eGFR, mL/min per 1.73m <sup>2</sup>	76.9 (65.4, 91.0)	80.9 (64.5, 93.5)	76.9 (65.4, 90.2)	0.036	77.8 (64.5, 98.8)	73.9 (63.2, 90.2)	0.042
Serum uric acid, μmol/L	321.2 (267.7, 374.7)	327.1 (273.6, 380.7)	315.2 (261.7, 374.7)	0.006	327.1 (279.6, 380.7)	315.2 (267.7, 374.7)	0.117
Serum calcium, mmol/L	2.35 (2.30, 2.43)	2.38 (2.30, 2.43)	2.35 (2.30, 2.40)	0.009	2.38 (2.30, 2.43)	2.35 (2.30, 2.42)	0.116
Serum phosphorus, mmol/L	1.23 (1.10, 1.36)	1.23 (1.10, 1.36)	1.23 (1.09, 1.34)	0.313	1.24 (1.13, 1.36)	1.23 (1.10, 1.35)	0.033

**Abbreviations:** AAC, abdominal aortic calcification; RIP, ratio of family income to poverty; BMI, body mass index; TC, total cholesterol; TG, total triglycerides; ALP, alkaline phosphatase; eGFR, estimated glomerular filtration rate.

**Table 2** General Characteristics of Participants Stratified by Sleep Pattern

Characteristic	Sleep patterns			p-value
	Poor (n=612)	Intermediate (n=958)	Healthy (n=1231)	
Age, year	58.7 ± 11.5	58.7 ± 11.8	59.5 ± 12.2	0.034
Gender, n (%)				< 0.001
Male	253 (41.3)	457 (47.7)	637 (51.7)	
Female	359 (58.7)	501 (52.3)	594 (48.3)	
Race, n (%)				< 0.001
Mexican American	68 (11.1)	123 (12.8)	182 (14.8)	
Non-Hispanic White	60 (9.8)	99 (10.3)	110 (8.9)	
Non-Hispanic Black	297 (48.5)	400 (41.8)	571 (46.4)	
Other Hispanic	133 (21.7)	207 (21.6)	192 (15.6)	
Others	54 (8.8)	129 (13.5)	176 (14.3)	
Education level, n (%)				0.052
Less than high school	130 (21.2)	203 (21.2)	281 (22.8)	
High school	165 (27.0)	217 (22.7)	256 (20.8)	
More than high school	317 (52.8)	538 (56.1)	694 (56.4)	
Marital status, n (%)				< 0.001
Married/Living with partner	348 (56.9)	590 (61.6)	851 (69.1)	
Single	264 (43.1)	368 (38.4)	380 (30.9)	
PIR				< 0.001
<1.30	208 (33.9)	260 (27.1)	296 (24.0)	
1.31 to 3.50	201 (32.8)	320 (33.4)	386 (31.4)	
≥3.50	161 (26.3)	312 (32.5)	443 (36.0)	
Smoker, n (%)	321 (52.5)	445 (46.4)	529 (43.0)	0.001
Alcohol drinker, n (%)	438 (71.6)	693 (72.3)	874 (71.0)	0.789
BMI status, n (%)				< 0.001
Under/normal weight	129 (21.1)	187 (19.5)	372 (30.2)	
Overweight	187 (30.1)	359 (37.5)	480 (39.0)	
Obesity	294 (48.0)	480 (50.1)	371 (30.1)	
Physical activity, n (%)	222 (36.3)	353 (36.8)	388 (31.5)	0.018
Hypertension, n (%)	393 (64.2)	544 (56.8)	640 (52.0)	< 0.001
Diabetes, n (%)	183 (29.9)	223 (23.3)	253 (20.6)	< 0.001
TC, mmol/L	4.91 (4.22, 5.72)	4.99 (4.27, 5.74)	5.04 (4.34, 5.74)	0.256
TG, mmol/L	1.57 (1.00, 2.46)	1.42 (0.95, 2.10)	1.39 (0.94, 2.19)	0.011

(Continued)

**Table 2** (Continued).

Characteristic	Sleep patterns			p-value
	Poor (n=612)	Intermediate (n=958)	Healthy (n=1231)	
Total 25-hydroxyvitamin D, nmol/L	68.5 (49.8, 87.7)	65.4 (48.6, 85.3)	68.7 (53.7, 86.3)	0.014
Serum ALP, IU/L	67 (54, 81)	65 (53, 79)	63 (52, 77)	0.001
eGFR, mL/min per 1.73m <sup>2</sup>	76.9 (66.3, 92.8)	76.9 (64.5, 90.4)	76.9 (65.4, 91.1)	0.031
Serum uric acid, μmol/L	321.2 (267.7, 380.7)	315.2 (267.7, 374.7)	321.2 (267.7, 374.7)	0.012
Serum calcium, mmol/L	2.35 (2.30, 2.43)	2.35 (2.30, 2.40)	2.35 (2.30, 2.43)	0.761
Serum phosphorus, mmol/L	1.26 (1.13, 1.39)	1.23 (1.10, 1.32)	1.23 (1.10, 1.32)	< 0.001
AAC	276 (45.1)	299 (31.2)	401 (32.6)	< 0.001
Severe AAC	106 (17.3)	88 (9.2)	122 (9.9)	< 0.001

**Abbreviations:** AAC, abdominal aortic calcification; RIP, ratio of family income to poverty; BMI, body mass index; TC, total cholesterol; TG, total triglycerides; ALP, alkaline phosphatase; eGFR, estimated glomerular filtration rate.

sleep pattern, hypertension, diabetes, eGFR, total 25-hydroxyvitamin D level, serum uric acid level, and serum calcium level (all  $p < 0.05$ ). Participants with AAC were older and more likely to be black, single, smokers, and less physically active; had less PIR; lower eGFR; and had a high prevalence of hypertension, diabetes, and higher levels of total 25-hydroxyvitamin D, serum uric acid, and serum calcium. Participants with poor sleep patterns were older, more likely to be female, single, smokers, and engaged in less physical activity. In addition, these participants had a lower PIR, lower eGFR, higher prevalence of hypertension and diabetes, and higher BMI, serum uric acid, and serum phosphorus levels.

## Sleep Pattern and AAC

Multiple logistic regression in [Table 3](#) showed that compared with the healthy sleep pattern group, the odds ratios (ORs) for AAC and severe AAC were 1.664 (95% CI, 1.286–2.152) and 2.179 (95% CI, 1.539–3.087), respectively, in the poor sleep pattern group before adjusted for any covariates (Model 1). After adjusting for age, sex, race, education level, and marital status, the ORs for AAC and severe AAC in the poor sleep pattern group were 1.891 (95% CI, 1.423–2.512) and 2.747 (95% CI, 1.882–4.010), respectively, compared to those in the healthy sleep pattern group (Model 3). After adjusting for all covariates, the ORs for AAC and severe AAC in the poor sleep pattern group were 1.657 (95% CI, 1.235–2.222) and 2.374 (95% CI, 1.616–3.488), respectively (Model 3).

**Table 3** Associations Between Sleep Pattern and AAC or Severe AAC

	Model 1		Model 2		Model 3	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Sleep pattern*						
Poor	1.664 (1.286–2.152)	<0.001	1.891 (1.423–2.512)	<0.001	1.657 (1.235–2.222)	0.001
Intermediate	1.017 (0.806–1.285)	0.884	1.064 (0.830–1.364)	0.623	1.012 (0.786–1.303)	0.927
Healthy	Reference	/	Reference	/	Reference	/
Sleep pattern <sup>#</sup>						
Poor	2.179 (1.539–3.087)	<0.001	2.747 (1.882–4.010)	<0.001	2.374 (1.616–3.488)	<0.001
Intermediate	0.983 (0.691–1.399)	0.923	1.034 (0.718–1.487)	0.858	0.981 (0.678–1.420)	0.921
Healthy	Reference	/	Reference	/	Reference	/

**Notes:** Model 1 was unadjusted for any covariates, and Model 2 included adjustments for age, gender, race, education levels, and marital status. Model 3 was further adjusted for poverty income ratio, BMI, smoking and drinking status, diabetes, hypertension, physical activity status, eGFR, serum uric acid, serum phosphorus, total 25-hydroxyvitamin D and serum total calcium.\*The association between sleep pattern and AAC. <sup>#</sup>The association between sleep pattern and severe AAC.

**Abbreviations:** AAC indicates abdominal aortic calcification, OR, odds ratio.

## Discussion

In this cross-sectional study, we investigated the relationship between sleep patterns and AAC and found that poor sleep patterns may increase the prevalence of AAC. We also found that several factors, such as age, smoking, physical activity, hypertension, diabetes, eGFR, and serum uric acid, were simultaneously associated with AAC and poor sleep patterns, which further supported the conclusion that poor sleep patterns are associated with higher AAC risk. Since AAC a reliable marker for atherosclerotic CVD and cardiovascular events, improve the quality of sleep might reduce the risk of CVD and cardiovascular events.

Although previous studies have explored the association between sleep and AAC risk, the number of enrolled patients was relatively limited. Our study further expanded the research population and validated previous findings. In addition, our study integrated sleep duration, self-reported trouble sleeping, and sleep disorders into a single sleep measurement, which differs from previous studies. In the future, sleep assessment should be integrated into cardiovascular risk evaluations. Sleep interventions, such as cognitive behavioral therapy, may reduce the risk of AAC.

Vascular calcification is a complex process similar to bone formation. Several mechanisms may have contributed to the association between poor sleep patterns and AAC. Poor sleep patterns may promote the release of inflammatory cytokines such as interleukin-1 $\beta$  (IL-1 $\beta$ ) and interleukin-6 (IL-6),<sup>24,25</sup> which play crucial roles in the transformation of vascular smooth muscle cells (VSMC) into osteoblast-like cells, followed by calcification.<sup>26,27</sup> In addition, poor sleep patterns may also facilitate oxidative stress,<sup>28,29</sup> which stimulates the osteoblastic transition of VSMC.<sup>30</sup> Bone mineral density (BMD) has been reported to be associated with AAC.<sup>31,32</sup> BMD has also been found to be related to sleep quality,<sup>33</sup> it is reasonable to speculate that sleep quality might be associated with AAC. Endothelial dysfunction is an important pathophysiological basis for vascular calcification, and sleep disorders may cause endothelial dysfunction and promote AAC.<sup>34,35</sup> Maybe we can perform some basic researches by constructing models of sleep disorder and AAC to further explore the relationship between sleep and AAC and the underline mechanisms.

Our study may provide important guidance for the prevention of AAC; however, several limitations should be considered. First, this was a cross-sectional study, and we could not determine a causal relationship between sleep patterns and AAC. Future longitudinal studies are needed to explore the temporal relationships and causality. Second, sleep-related data were collected from self-reports, which might have led to estimation errors. Objective sleep measures such as actigraphy or polysomnography may further validate these findings. Third, we excluded many missing data, which may have influenced our results. Finally, AAC measurement was performed only in the US population aged  $\geq 40$  years; whether the association existed in other age groups or populations from other countries remains unclear and warrants additional research.

## Conclusion

Our study indicated that poor sleep patterns are associated with a higher risk of AAC. Therefore, sleep may be relevant for primary prevention of AAC.

## Data Sharing Statement

The data of the current study are available in the NHANES repository, [https:// www.cdc.gov/nchs/nhanes/](https://www.cdc.gov/nchs/nhanes/).

## Ethics Approval and Consent to Participate

This study analyzed de-identified information downloaded from the National Health and NHANES public database. This study was approved by the National Center for Health Statistics Research Ethics Review Board. Informed consent was obtained from each participant before the survey.

## Acknowledgments

The authors gratefully acknowledge all staff and participants of the NHANES for their participation and contribution.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

## Funding

This study was supported by the Pioneer Innovation Team of the Jiaying Institute of Atherosclerotic Diseases (Grant No. XFCX–DMYH), Jiaying Science and Technology Program (Grant No.2024AD30032), and Jiaying Key Laboratory of Virus-mediated Infectious Diseases (Grant No. 2021-bdzsys).

## Disclosure

The authors declare no competing interests in this work.

## References

1. Golledge J. Abdominal aortic calcification: clinical significance, mechanisms and therapies. *Curr Pharm Des.* 2014;20:5834–5838. doi:10.2174/1381612820666140212195309
2. Bastos Goncalves F, Voûte MT, Hoeks SE, et al. Calcification of the abdominal aorta as an independent predictor of cardiovascular events: a meta-analysis. *Heart.* 2012;98:988–994. doi:10.1136/heartjnl-2011-301464
3. Bolland MJ, Wang TK, van Pelt NC, et al. Abdominal aortic calcification on vertebral morphometry images predicts incident myocardial infarction. *J Bone Miner Res.* 2010;25:505–512. doi:10.1359/jbmr.091005
4. Golestani R, et al. Abdominal aortic calcification detected by dual X-ray absorptiometry: a strong predictor for cardiovascular events. *Ann Med.* 2010;42:539–545. doi:10.3109/07853890.2010.515604
5. Bartstra JW, Mali W, Spiering W, de Jong PA. Abdominal aortic calcification: from ancient friend to modern foe. *Eur J Prev Cardiol.* 2021;28:1386–1391. doi:10.1177/2047487320919895
6. Lloyd-Jones DM, Allen NB, Anderson CAM, et al. Life's essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. *Circulation.* 2022;146:e18–e43. doi:10.1161/CIR.0000000000001078
7. Jaspan VN, Greenberg GS, Parihar S, et al. The role of sleep in cardiovascular disease. *Curr Atheroscler Rep.* 2024;26:249–262. doi:10.1007/s11883-024-01207-5
8. Kadier K, et al. Association of sleep-related disorders with cardiovascular disease among adults in the United States: a cross-sectional study based on national health and nutrition examination survey 2005-2008. *Front Cardiovasc Med.* 2022;9:954238. doi:10.3389/fcvm.2022.954238
9. Fan T, Su D. Interaction effects between sleep disorders and depression on heart failure. *BMC Cardiovasc Disord.* 2023;23:132. doi:10.1186/s12872-023-03147-5
10. Li X, Zhou T, Ma H, et al. Healthy sleep patterns and risk of incident arrhythmias. *J Am Coll Cardiol.* 2021;78:1197–1207. doi:10.1016/j.jacc.2021.07.023
11. King CR, et al. Short sleep duration and incident coronary artery calcification. *JAMA.* 2008;300:2859–2866. doi:10.1001/jama.2008.867
12. Kowall B, Lehmann N, Mahabadi A-A, et al. Sleep characteristics and progression of coronary artery calcification: results from the Heinz Nixdorf Recall cohort study. *Atherosclerosis.* 2018;271:45–52. doi:10.1016/j.atherosclerosis.2018.02.013
13. Matthews KA, Everson-Rose SA, Kravitz HM, et al. Do reports of sleep disturbance relate to coronary and aortic calcification in healthy middle-aged women?: study of women's health across the nation. *Sleep Med.* 2013;14:282–287. doi:10.1016/j.sleep.2012.11.016
14. Wang Y, et al. Long sleep duration is associated with abdominal aortic calcification among male adults with chronic kidney disease: NHANES 2013-2014. *Sci Rep.* 2024;14:22076. doi:10.1038/s41598-024-72879-x
15. Li C, Shang S. Relationship between sleep and hypertension: findings from the NHANES (2007-2014). *Int J Environ Res Public Health.* 2021;18. doi:10.3390/ijerph18157867.
16. Kauppila LI, Polak JF, Cupples LA, et al. New indices to classify location, severity and progression of calcific lesions in the abdominal aorta: a 25-year follow-up study. *Atherosclerosis.* 1997;132:245–250. doi:10.1016/s0021-9150(97)00106-8
17. Cai C, et al. Association between hemoglobin A1c and abdominal aortic calcification: results from the national health and nutrition examination survey 2013-2014. *BMC Cardiovasc Disord.* 2024;24:26. doi:10.1186/s12872-023-03700-2
18. Cai Z, et al. Associations between life's essential 8 and abdominal aortic calcification among middle-aged and elderly populations. *J Am Heart Assoc.* 2023;12:e031146. doi:10.1161/JAHA.123.031146
19. Li W, Wang Z, Li M, et al. Association between a body shape index and abdominal aortic calcification in general population: a cross-sectional study. *Front Cardiovasc Med.* 2022;9:1091390. doi:10.3389/fcvm.2022.1091390
20. Xie ZQ, Li H-X, Tan W-L, et al. Association of Serum Vitamin C With NAFLD and MAFLD Among Adults in the United States. *Front Nutr.* 2021;8:795391. doi:10.3389/fnut.2021.795391
21. Piercy KL, Troiano RP, Ballard RM, et al. The physical activity guidelines for Americans. *JAMA.* 2018;320:2020–2028. doi:10.1001/jama.2018.14854
22. Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. *Ann Intern Med.* 2009;150:604–612. doi:10.7326/0003-4819-150-9-200905050-00006

23. Heffernan KS, Barreira TV. Association between pulse pressure and aortic calcification: findings from the national health and nutrition examination survey 2013-2014. *J Clin Hypertens*. 2020;22:879–885. doi:10.1111/jch.13853
24. Zhang Y, Zhao W, Liu K, et al. The causal associations of altered inflammatory proteins with sleep duration, insomnia and daytime sleepiness. *Sleep*. 2023;46. doi:10.1093/sleep/zsad207
25. Morales-Munoz I, Marwaha S, Upthegrove R, Cropley V. Role of inflammation in short sleep duration across childhood and psychosis in young adulthood. *JAMA Psychiatry*. 2024;81:825–833. doi:10.1001/jamapsychiatry.2024.0796
26. Kurozumi A, Nakano K, Yamagata K, et al. IL-6 and sIL-6R induces STAT3-dependent differentiation of human VSMCs into osteoblast-like cells through JMJD2B-mediated histone demethylation of RUNX2. *Bone*. 2019;124:53–61. doi:10.1016/j.bone.2019.04.006
27. Han L, Zhang Y, Zhang M, et al. Interleukin-1beta-induced senescence promotes osteoblastic transition of vascular smooth muscle cells. *Kidney Blood Press Res*. 2020;45:314–330. doi:10.1159/000504298
28. Lei X, Xu Z, Chen W. Association of oxidative balance score with sleep quality: NHANES 2007-2014. *J Affect Disord*. 2023;339:435–442. doi:10.1016/j.jad.2023.07.040
29. BinHeyat MB, Akhtar F, Sultana A, et al. Role of oxidative stress and inflammation in insomnia sleep disorder and cardiovascular diseases: herbal antioxidants and anti-inflammatory coupled with insomnia detection using machine learning. *Curr Pharm Des*. 2022;28:3618–3636. doi:10.2174/1381612829666221201161636
30. Huang M, et al. Oxidative stress contributes to vascular calcification in patients with chronic kidney disease. *J Mol Cell Cardiol*. 2020;138:256–268. doi:10.1016/j.yjmcc.2019.12.006
31. Naves M, Rodriguez-Garcia M, Diaz-Lopez JB, Gomez-Alonso C, Cannata-Andia JB. Progression of vascular calcifications is associated with greater bone loss and increased bone fractures. *Osteoporos Int*. 2008;19:1161–1166. doi:10.1007/s00198-007-0539-1
32. Paccou J, Mentaverri R, Renard C, et al. The relationships between serum sclerostin, bone mineral density, and vascular calcification in rheumatoid arthritis. *J Clin Endocrinol Metab*. 2014;99:4740–4748. doi:10.1210/jc.2014-2327
33. Xiong M, Liu X, You L, Chen X. Relationship between sleep quality and bone mineral density in urban residents. *Zhejiang Da Xue Xue Bao Yi Xue Ban*. 2020;49:431–438. doi:10.3785/j.issn.1008-9292.2020.08.01
34. Calvin AD, Covassin N, Kremers WK, et al. Experimental sleep restriction causes endothelial dysfunction in healthy humans. *J Am Heart Assoc*. 2014;3:e001143. doi:10.1161/JAHA.114.001143
35. Farabi SS, et al. Endothelial dysfunction is related to glycemic variability and quality and duration of sleep in adults with type 1 diabetes. *J Cardiovasc Nurs*. 2018;33:E21–E25. doi:10.1097/JCN.0000000000000485

## Vascular Health and Risk Management

### Publish your work in this journal

Vascular Health and Risk Management is an international, peer-reviewed journal of therapeutics and risk management, focusing on concise rapid reporting of clinical studies on the processes involved in the maintenance of vascular health; the monitoring, prevention and treatment of vascular disease and its sequelae; and the involvement of metabolic disorders, particularly diabetes. This journal is indexed on PubMed Central and MedLine. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/vascular-health-and-risk-management-journal>

**Dovepress**  
Taylor & Francis Group