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Associations of Framingham Risk Score Profile and Coronary Artery Calcification with Sleep Characteristics in Middle-aged Men and Women: Pittsburgh SleepSCORE Study

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Background: Short and less efficient sleep may be risk factors for atherosclerosis. Few studies have investigated the associations between sleep characteristics and early cardiovascular disease (CVD) risk.

Objective: Evaluate the associations between coronary artery calcification (CAC) and Framingham risk score profile with sleep characteristics in middle-aged men and women with no history of diagnosed myocardial infarction, interventional cardiology procedures, stroke, diabetes, or sleep disorders.

Method: 224 participants enrolled in an epidemiological study of disparities in CVD risk were recruited for a 9-night assessment of sleep, with 2 nights of polysomnography (PSG) and 9 nights of actigraphy and sleep diaries. Of the 224 participants, 110 had high/moderate Framingham risk scores and 114 had low scores; 195 had computed tomography measures of CAC.

Results: Individuals who had any CAC or higher Framingham risk scores had elevated apnea/hypopnea index (AHI) values, independent of age, race, and gender. The AHI association with CAC was nonsignificant in analyses adjusting for body mass index (BMI). Those with higher Framingham risk score profiles had shorter PSG sleep duration and less percent stage 3-4 and delta power sleep. High blood pressure and left ventricular hypertrophy were related to AHI and sleep duration, independent of BMI. Neither sleep duration nor efficiency was associated with CAC.

Conclusions: CAC was not associated with AHI, independent of BMI in a community-based sample of middle-aged men and women. Framingham risk score profiles were related to poor sleep. Sleep duration may not be related to early plaque burden in relatively healthy individuals.

Key Words: apnea, sleep duration, coronary calcification, atherosclerosis, Framingham risk score profile

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INTRODUCTION

Poor sleep may be a risk factor for cardiovascular disease (CVD) morbidity and mortality. A recent review summarizing 23 mortality studies concluded that among both males and females, short and long sleepers are at increased risk for all-cause mortality, compared to individuals who report a medium amount of sleep.[1] Short sleep among men and long sleep among men and women are associated with CVD mortality. The few prospective studies of sleep efficiency, e.g., difficulties maintaining sleep, in relation to all-cause or CVD mortality show that sleep disturbances may be associated with CVD mortality, with weaker evidence for women.[2-4] To our knowledge, the only study using objective sleep measures found that early mortality was not related to sleep duration measured by polysomnography but was related to long sleep latency, decreased sleep efficiency, and either very high or very low percent REM sleep.[5]

In the aggregate, the findings are intriguing, but they are difficult to interpret. Most epidemiological studies of sleep and health do not include measures of apnea, which is a well-established risk factor for hypertension and heart disease.[6-8] Self-report measures of typical sleep may be poorly correlated with objectively measured sleep.[9] Furthermore, both self-reported short and long sleepers tend to have elevated symptoms of depression and anxiety and have medical comorbidities.[10] Finally, poor sleep may be a consequence of rather than an antecedent to CVD.[11]

One way to approach the temporal relationship between sleep characteristics and cardiovascular risk is to examine the associations of sleep and atherosclerosis prior to the onset of diagnosed heart disease. Coronary artery calcification (CAC) can be considered a measure of atherosclerotic plaque burden; it predicts later CVD morbidity and mortality in both initially healthy samples and patient populations.[12,13] We are aware of only a few studies of CAC in relation to sleep characteristics. In a sample of 202 patients with suspected sleep disorders but no diagnosed heart disease, extent of CAC increased as apnea/hypopnea index (AHI) values increased; there was no association with overnight oxygen saturation.[14] Among 258 healthy middle-aged Korean men selected from a population-based study, greater AHI values were associated with having any CAC; adjustments for obesity reduced the association to nonsignificant levels.[15] In the CARDIA Sleep ancillary study, sleep duration and fragmentation measured by actigraphy and self-reported sleep quality, typical duration, and daytime sleepiness were measured in 495 middle-aged black and white participants who had no calcification 3 years prior to the sleep study; 61 participants developed any coronary calcification in the 2 years following the sleep study.[16] Shorter sleep duration measured by

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actigraphy was the only variable related to having any calcification. Apnea was not measured.

The objectives of the present study were several-fold. First, we examined the associations of AHI and extent of CAC in a middle-aged community sample of black and white men and women.[17] The study sample was recruited to be free of history of diagnosed heart disease, stroke, diabetes, or sleep disorders and represented a range of Framingham risk score profiles, i.e., their 10-year absolute risk of CHD events. Thus, we also had the opportunity to examine the association between AHI and the Framingham risk score profiles. Second, we examined in-home objective (polysomnography and actigraphy) measures of sleep duration, continuity, and architecture, and subjective sleep quality with CAC and Framingham risk score profiles. Third, we explored the impact of gender and race/ethnicity on the pattern of results because of gender and race differences in sleep reported in the present sample[18] and in other samples.[19,20] Our overall hypotheses were that greater CAC and the Framingham risk score profiles would be associated with higher AHI scores, shorter sleep duration, less efficient and deep sleep, and poorer sleep quality.

METHODS

Participants

Participants in the present study were recruited from the Heart Strategies Concentrating on Risk Evaluation (HeartSCORE) study, a prospective, community-based cohort study investigating mechanisms for racial disparities in cardiovascular risk.[17] HeartSCORE eligibility criteria included age 45 to 75 years, residence in the greater Pittsburgh metropolitan area, ability to undergo baseline and annual follow-up visits, and absence of known comorbidities expected to limit life expectancy to less than 5 years. Data collection included demographics, medical history, anthropometrics, calcification, lipids/lipoproteins, physical activity, and psychological status. HeartSCORE classified participants into one of 3 groups: prior diagnosis of stroke, myocardial infarction, or interventional cardiology procedures; moderate/high (> 10%) probability of CHD event in next 10 years; or low probability of CHD events, based on the Framingham risk score profiles.[21]

From HeartSCORE, the current study, called SleepSCORE, recruited 97 African American, 123 Caucasian, and 4 Asian men and women, with approximately half from the moderate/high and half from the low risk Framingham risk score profile groups. Those with a prior diagnosis of stroke, myocardial infarction, or interventional cardiology procedures were ineligible. Additional SleepSCORE exclusionary criteria included pregnancy, use of continuous positive airway pressure treatment for sleep disordered breathing, regular use of medications for sleep problems, nighttime work schedule, and medication for diabetes. These additional exclusionary criteria were based on their associations with sleep characteristics. Both HeartSCORE and SleepSCORE protocols were approved by the University of Pittsburgh Institutional Review Board, and all participants provided written informed consent for both protocols.

Assessment of Framingham Risk Score Profiles

Framingham risk score profiles were calculated based on age (separately for males and females), systolic blood pressure, smoking status, high-density lipoprotein (HDL) and total cholesterol, history of diabetes, and left ventricular hypertrophy (LVH) based on ECG.[21] Resting blood pressure measurement was based on the average of 2 seated blood pressures by trained nurses. Laboratory assessment of lipoprotein levels (Cholestech) was performed on venous blood drawn in the fasting state. LVH was scored using the Cornell criteria by a certified nurse supervised by 2 cardiologists. Age, gender, and smoking status (yes/no to current nicotine use) were determined by self-report. Participants were classified as above or below a 10% risk of a CHD event in the next 10 years.

SleepSCORE Data Collection

Eligible participants were recruited sequentially during regular HeartSCORE assessment visits and were scheduled for the SleepSCORE protocol within approximately 3 months of initial contact, provided that the equipment dedicated to this protocol was available. The study protocol lasted 10 days. On all 9 nights of the study, actigraphy was used to track rest and activity patterns via physical movement. On nights 1 and 2, in-home polysomnography (PSG) sleep studies were conducted by trained sleep technicians, and on day 2 participants completed self-report measures of global sleep quality and daytime sleepiness. Morning and evening diary reports, as well as 2 days and nights of ambulatory blood pressure monitoring and two 15-h periods of urine collection, were also included as part of the study; these data are reported elsewhere.[22,23]

Assessment of Sleep

Polysomnography

Two nights of PSG recording were conducted in participants' homes using a Compumedics Siesta monitor (Charlotte, NC). The PSG montage included bilateral central and occipital electroencephalogram channels, bilateral electroculograms (EOG), bipolar submentalis electromyograms (EMG), and one channel of electrocardiogram (EKG) recording. On the first night of PSG, participants were monitored for sleep disordered breathing using nasal pressure, oral-nasal thermistors, inductance plethysmography, and fingertip oximetry. High-frequency filter settings were 100 Hz for EEG and EOG and 70 Hz for EMG. Low-frequency filter settings were 0.3 Hz for EEG and 10 Hz for EMG. Trained PSG technologists scored sleep records using Rechtschaffen and Kales[24] sleep stage scoring criteria for each 20-sec epoch. American Academy of Sleep Medicine Task Force[25] definitions were used to identify apneas and hypopneas; oximetry readings were used to quantify average and minimum oxygen saturation levels. Sleep records were scored prior to the updated guidelines for sleep scoring published in 2007.[26] PSG sleep variables included sleep duration (total recording period – [sleep latency + wakefulness after sleep onset]), sleep efficiency ([time spent asleep/total recording period] × 100), parameters of sleep architecture (percentage of total sleep time scored as stage 3-4 [% Stage 3-4]), and indices of physiological arousal (mean absolute power in the beta band (16.0–32.0 Hz), and mean absolute power in the delta band (0.5–4.0 Hz) during NREM sleep). Values from the 2 nights of the study were averaged for each sleep variable. The apnea/hypopnea index (AHI) was defined as number of apneas and hypopneas per hour of sleep measured on

Night 1. Variables with skewed distributions were transformed using either a log (sleep efficiency, beta power, delta power, AHI) or square root (% Stage 3-4) transformation. In addition, we classified participants into AHI groups of ≤ 5; 5-29; 30+.

Actigraphy

Participants wore an Actiwatch-16 (Phillips Respironics, Inc.) on the non-dominant wrist for 10 days to provide behavioral data regarding sleep and wake patterns. Data were stored in 1-min epochs, and MiniMitter software (Phillips Respironics, Inc.) algorithms were used to estimate sleep parameters. The 2 variables used in analyses included sleep duration (defined as actual sleep time excluding periods of wakefulness during the night) and sleep efficiency (percentage of time in bed spent sleeping). Sleep duration and sleep efficiency values were averaged across the nine nights. Sleep efficiency was log-transformed due to a skewed distribution.

Self-report

Participants completed the Pittsburgh Sleep Quality Index (PSQI), which measures subjective sleep quality over the previous month.[27] The 7 component subscores on the PSQI are summed to generate a global score between 0 and 21, with higher scores indicating worse sleep quality. Usual self-reported sleep duration estimates were obtained using the PSQI item: "During the past month, how many hours of actual sleep did you get at night?"

Table 1—Sample characteristics

	No. (%)	Mean (SD)	Range
Current smokers	19 (8.5)		
Hypertension based on readings and/or medications	94 (42.0)		
Left ventricular hypertrophy	28 (12.5)		
Systolic blood pressure		135.72 (20.79)	102–214
High-density lipoprotein cholesterol		53.29 (16.98)	22–101
Total cholesterol		208.54 (40.23)	108–311
Lipid-lowering medication	52 (23.2)		
Coronary calcification total score		115.1 (269.1)	0.0–1519.1
AHI (events/h of sleep)		13.25 (14.89)	0.00–92.88
< 5	63 (28.5)		
5-29	134 (60.6)		
≥ 30	24 (10.9)		
Sleep duration (h)			
PSG		6.07 (1.01)	2.17–8.43
Actigraphy		5.79 (0.88)	2.93–8.74
Self-report		6.47 (1.21)	2.50–10.00
Sleep efficiency			
PSG		76.97 (11.22)	24.97–93.00
Actigraphy		80.41 (7.99)	45.25–93.16
Architecture			
% Stage 3-4		5.21 (6.33)	0.00–39.44
Beta power		0.147 (0.070)	0.037–0.456
Delta power		25.68 (16.09)	6.35–105.08
Self-report			
PSQI score		5.54 (3.21)	0–15

Assessment of coronary calcification

Electron beam tomography (EBT) image acquisition was obtained with an Imatron C150 scanner (GE Imatron Inc, South San Francisco, CA). During coronary scanning, 30 to 40 contiguous 3-mm thick transverse images were obtained from the level of the aortic root to the apex of the heart during an inspiratory breath-hold. Each image (100-millisecond exposure) was acquired during the same phase of the cardiac cycle (80% of the RR interval) using electrocardiogram triggering. The Agatston method was used to derive a total coronary calcium score, based on the detection of ≥ 3 contiguous pixels > 130 Hounsfield units.[28] Due to a change in the HeartSCORE protocol, EBT scans were not performed on all participants. Thus, coronary calcification data were available for a total of 195 participants (n = 101 in the low Framingham risk group and n = 94 in the moderate/high risk group). The 195 with CAC data did not differ from the 29 who did not participate on age, sex, race, or Framingham risk group (Ps ≥ 0.30).

Statistical Analysis

We used ANCOVAs to determine differences in sleep by Framingham risk group, after adjusting for race (not age and gender, which are incorporated into the Framingham equation).

Significant associations were followed by partial correlations with total and HDL-C cholesterol, SBP, and by ANCOVA with smoking, hypertensive, and LVH status; in these analyses we adjusted for race, age, and gender to ensure that any obtained effects were not simply due to their influence on sleep characteristics. We also adjusted for being on lipid-lowering agents for total and HDL-C effects that were significant.

We classified participants into 3 groups according to untransformed coronary calcium scores (those with no calcium; scores 1-99; scores ≥ 100). To evaluate CAC group differences in sleep, we conducted analyses of covariance (ANCOVA) adjusted for age, race, and gender with tests for linear trend, followed by planned contrasts for pairwise comparisons. AHI was treated as a transformed and categorical variable. Tests for interactions with race and/gender were also evaluated in relation to CAC groups. Additional analyses adjusted for BMI.

RESULTS

Sample Characteristics

Sample was comprised of 113 men and 111 women, with a mean age about 60 years; 97 were African American and 127 were Caucasian or Asian (Caucasian and the 4 Asian participants were combined for the purpose of analysis). There were few smokers; a substantial number had measured blood pressure > 140/90 or were on anti-hypertensive medications; and nearly a quarter were on lipid lowering agents (Table 1). Forty percent (N = 88) had a BMI ≥ 30, with only 20% (N = 43) considered to be normal weight. About half of the sample was classified as moderate/high risk for a future CHD event within 10 years according to Framingham risk score profile (a simi-

Table 2—Unadjusted mean (SD) sleep characteristics by coronary calcification group

	Coronary Calcification Group			P-value adjusted for	P-value Contrast	P-value Contrast
Group (N)	0 (62)	1-99 (90)	100+ (43)	age, race, gender	0-99 vs. 100	0 vs. > 0
AHI[T]	10.48 (13.93)	13.03 (12.75)	14.47 (12.19)	0.04	0.88	0.04
Sleep duration						
PSG	6.11 (0.91)	6.16 (1.00)	5.90 (1.04)	0.89	0.63	0.79
Actigraphy	5.78 (0.98)	5.80 (0.82)	5.92 (0.79)	0.64	0.35	0.62
Self-report	6.41 (1.25)	6.51 (1.18)	6.48 (1.19)	0.88	0.73	0.96
Sleep efficiency[T]						
PSG	76.20 (9.67)	78.82 (11.27)	74.03 (11.92)	0.21	0.73	0.27
Actigraphy	80.51 (8.20)	80.35 (7.45)	80.89 (8.33)	0.73	0.59	0.93
Architecture						
% Stage 3-4[T]	4.62 (5.41)	6.40 (6.91)	4.15 (6.10)	0.45	0.44	0.77
Beta power[T]	0.14 (0.07)	0.16 (0.07)	0.13 (0.06)	0.55	0.82	0.34
Delta power[T]	25.40 (14.39)	27.57 (16.50)	21.40 (12.92)	0.89	0.66	0.89
Self-report						
PSQI	6.61 (3.28)	6.19 (3.32)	6.42 (3.44)	0.18	0.11	0.75

[T]Analyses based on transformed variables.

Table 3—Number (percentage) of participants in apnea-hypopnea index (AHI) groups by coronary calcification and Framingham risk score profile groups

	AHI Group			Chi-Square
Group	< 5	5-29	≥ 30	P-value
Calcification Group				
0 (n = 62)	25 (46.3)	33 (27.5)	4 (21.1)	
1-99 (n = 88)	22 (40.7)	56 (46.7)	10 (52.6)	
≥ 100 (n = 43)	7 (13.0)	31 (25.8)	5 (26.3)	0.08[T]
Framingham Risk Group				
Low (n = 113)	45 (71.4)	59 (44.0)	9 (37.5)	
High (n = 108)	18 (28.6)	75 (56.0)	15 (62.5)	0.001

[T]P-value determined using Fisher exact test.

lar proportion was obtained in HeartSCORE). In the 195 participants who had EBT scans, the mean coronary calcification score was 115.09 (SD = 269.12). These data were skewed, as 31.8% of the sample had no measured calcification, 46.2% had scores from 1-99, and 22.1% had scores > 100. The percentages of participants with moderate/high Framingham risk scores in the low to high CAC groups were 33.9%, 52.2%, and 72.1%, respectively, chi square (2) = 15.0, P < 0.001. Sleep characteristics are indicated in Table 1.

Associations between Sleep and Coronary Calcification

The calcification groups differed in AHI, after adjusting for age, race, and gender ($F_{2,187} = 3.38$, P = 0.04; Table 2). Subsequent pairwise comparisons showed that persons with any calcification had higher AHI scores than persons with no measurable calcification (P = 0.04). After adjustment for BMI the association between AHI and CAC was no longer significant, ($F_{2,186} = 2.11$, P = 0.12). The highest AHI group had twice the likelihood of being in the highest CAC group than had the lowest AHI group, but the effect only approached conventional level of statistical significance (Table 3).

Measures of sleep duration, sleep efficiency, and sleep architecture, as well as self-report measures, did not differ by CAC (Ps > 0.15). Tests for interactions of CAC group by race or by gender yielded one significant association. PSQI scores increased, indicative of poorer sleep quality, with increasing CAC group in women only, Gender by Group interaction, $F_{2,187} = 3.75$, P = 0.03.

Associations between Sleep Characteristics and Framingham Risk Score Profile Groups

Differences in sleep by Framingham risk group are shown in Table 4. Persons in the moderate/high Framingham risk group had higher AHI scores compared to the low risk group ($F_{1,219} = 22.38$, P < 0.001). Adjustments for BMI did not alter the association ($F_{1,218} = 20.94$, P < 0.001). The AHI group analyses showed that 62.5% of those in the highest AHI group and 28.6% of the lowest AHI group were in the high Framingham risk score profile group (Table 3). Logistic regression analyses with adjustments for BMI showed that relative to the lowest AHI group, the highest and middle AHI groups were 4.1 times and 3.2 times more likely to be in the moderate/high Framingham risk group than in the low risk group.

In analyses adjusted for age, gender, and race, higher AHI scores were associated with lower HDL-C (r = 0.17, P = 0.01), higher SBP (r = 0.17, P = 0.01), and presence of LVH (Ms = 18.2 vs 12.6, $F_{1,216} = 8.90$, P = 0.003), and of hypertension (Ms = 16.2 vs 9.5, $F_{1,216} = 21.97$, P < 0.001). The associations remained significant when also adjusting for BMI for SBP (r = 0.14, P = 0.04) and LVH ($F_{1,215} = 8.78$, P = 0.003), and when adjusting for lipid-lowering medications for HDL-C (r = −0.17, P = 0.01) (adjusting for lipid-lowering medications and BMI, r = −0.13, P = 0.06). The association between AHI and LVH remained significant after adjusting for either SBP (P = 0.01) or hypertensive status (P = 0.04) (in separate models). Test for an interaction between Framingham risk group and CAC group was significant:

$F_{2,187} = 4.35$, P = 0.01: those in the low Framingham risk group and no measurable CAC had the lowest AHI values.

Persons in the moderate/high Framingham risk group had a shorter sleep duration as measured by PSG than those in the low Framingham risk group ($F_{1,219} = 5.09$, P = 0.03). The association remained significant when BMI was entered into the model ($F_{1,218} = 4.99$, P = 0.03). Subsequent analyses adjusting for gender, race, and age showed that hypertensives (Ms = 5.87 vs 6.31, $F_{1,217} = 6.20$, P = 0.01) and those with LVH had shorter sleep duration (Ms = 5.69 vs 6.12, $F_{1,217} = 3.74$, P = 0.05); the effects remained after further adjustment for BMI (hypertensives: $F_{1,216} = 5.76$, P = 0.02; LVH: $F_{1,216} = 3.67$, P = 0.06). The 2 risk groups did not differ in actigraphy or self-report measures of sleep duration (Ps > 0.40).

The moderate/high risk group had lower absolute (NREM) delta power ($F_{1,208} = 4.55$, P = 0.03) and spent a lower percentage of time in stage 3-4 sleep ($F_{1,219} = 4.06$, P = 0.05) than the low risk group. Further adjustments for BMI showed a similar pattern of results, Ps = 0.05 and 0.03, respectively. Subsequent analyses showed that current smokers had less absolute delta power (19.45 vs 26.37, $F_{1,206} = 6.19$, P = 0.01) and spent a lower percentage of time in stage 3-4 sleep (3.23 vs 5.43, $F_{1,217} = 4.84$, P = 0.03). Framingham risk groups did not differ in sleep efficiency, absolute beta power, or subjective sleep quality (Ps > 0.10).

Table 4—Unadjusted mean (SD) sleep characteristics by Framingham risk score profile groups

	Risk Group		P-value
Group (N)	Low (114)	Moderate/High (110)	adjusted for Race
AHI [T]	10.46 (13.97)	16.16 (15.33)	< 0.001
Sleep duration			
PSG	6.20 (0.97)	5.92 (1.04)	0.03
Actigraphy	5.82 (.88)	5.77 (0.88)	0.49
Self-report	6.41 (1.22)	6.53 (1.20)	0.57
Sleep efficiency[T]			
PSG	78.24 (9.46)	75.64 (12.72)	0.16
Actigraphy	80.78 (7.89)	80.04 (8.12)	0.37
Architecture			
% Stage 3-4 [T]	5.86 (6.38)	4.53 (6.23)	0.05
Beta power[T]	0.15 (0.07)	0.14 (0.07)	0.23
Delta power[T]	27.84 (18.02)	23.32 (13.38)	0.03
Self-report			
PSQI	6.53 (3.58)	6.25 (3.11)	0.60

[T]Analyses based on transformed data.

DISCUSSION

This study evaluated the associations between extent of coronary calcium and Framingham risk score profiles with AHI, sleep duration, efficiency, architecture, and sleep quality in a multi-ethnic sample of middle-aged adults. By design, participants had no past history of myocardial infarction, stroke, or interventional cardiology procedures, and represented a range of Framingham risk scores. Results showed that higher AHI was associated with having any measurable CAC, relative to no CAC. These findings extend prior results based on a sample with individuals with suspected sleep disorders[14] and a sample of Korean men[15] to a sample of relatively healthy black and white men and women with no suspected sleep disorders. Note that our results are very similar to those obtained In the Korean sample, where AHI was associated with having any measurable CAC, and that adjustments for BMI reduced the association between AHI and CAC to nonsignificant levels. Although sleep disordered breathing may have adverse effects on the development of coronary atherosclerosis, our findings suggest that obesity may be a common pathway for both, or perhaps a confounder of the association.

With regard to the Framingham risk score profiles, participants with moderate/high scores had higher AHI, lower sleep duration (PSG only) and less delta power, and lower percent stage 3-4 sleep, indicative of less restful sleep. Framingham risk score profiles remained associated with AHI after separate adjustment for BMI. These findings confirm that those at moderate to high risk on the Framingham risk score profiles are likely to evidence poorer overall sleep. Taken together with the above results on CAC, patients with high Framingham scores or any CAC in mid-life should be evaluated for apnea. Sleep apnea can be treated with a number of modalities, and early evidence suggests that it may benefit cardiovascular health.[29,30]

Of interest were subsequent analyses that addressed which components of the Framingham risk score profiles were related to sleep characteristics. The presence of LVH and hypertension, and lower HDL-C and higher SBP were related to AHI, the presence of LVH and hypertension to short sleep duration, and current smoking status to less delta power and lower percent stage 3-4 sleep. The last finding suggests that smoking in particular disturbs restorative sleep. Prior research on sleep duration and hypertension has yielded conflicting findings, perhaps because the effect is weaker in older individuals and large studies often used subjective reports.[31] The present report supports the importance of high blood pressure and LVH in understanding sleep disturbance. Furthermore, that the association of AHI and LVH remained significant after adjustment for SBP is consistent with some evidence that sleep disordered breathing promotes development of LVH, independent of blood pressure.[32,33] Although SBP did not account for this relationship in the present sample, it is conceivable that acute elevations in nocturnal pressure associated with sleep disordered breathing events may play a role. From a clinical perspective, these findings suggest that apnea patients should be evaluated for LVH.

In contrast to the Framingham risk groups, CAC groups did not differ in sleep duration or sleep architecture. While the null association for sleep architecture was not unexpected, we did anticipate that sleep duration would be shorter in those with any measurable CAC as reported in CARDIA.[16] The difference may be due in part to differences in the proportions of individuals with any measurable CAC in the two samples. In the present sample, it was substantial (68%), with 22% with CAC scores > 100, in contrast to the 12% proportion of any new CAC in CARDIA. It also may be due to the different outcome measure of CAC, i.e., concurrent CAC in our sample vs. any CAC among those who had no CAC 5 years earlier in CARDIA. Also, our actigraphy measures of sleep duration were based on 9 days of recording, whereas CARDIA was based on 3 days of recording in the 2 years prior to the CAC measure.

Study limitations include the small sample size, ranging from 195-224, and relatively few with AHI ≥ 30. Studies were unobserved and in-home, with the attendant irregularities in the sleep environment. On the other hand, participants were in their usual sleep environment, which could be argued to yield more valid measures of sleep patterns than laboratory-based measures. Participants who had prior diagnosed sleep disorders or risk factors for sleep disorders were excluded, so findings cannot be generalized to clinic samples.

In summary, we found that AHI is elevated among individuals with any CAC or moderate/high risk for a future CHD death event as measured by the Framingham risk score profiles. The AHI-CAC association was nonsignificant after adjustment for BMI. We did not find that sleep duration and efficiency were related to CVD risk in the early stages of atherosclerosis.

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