

HHS Public Access

Sleep Epidemiol. Author manuscript; available in PMC 2022 December 01.

Published in final edited form as:

Author manuscript

Sleep Epidemiol. 2022 December; 2: . doi:10.1016/j.sleepe.2022.100037.

Associations between sleep apnea risk and cardiovascular disease indicators among Chinese and Korean Americans

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Abstract

Study objectives: While sleep apnea has been associated with cardiovascular disease (CVD) risk factors in white individuals in the U.S., these associations in Chinese and Korean Americans are less well-understood, particularly how these associations vary by age, gender, Asian origin, obesity, chronic conditions, and daytime sleepiness.

Methods: We used a sample of Chinese and Korean Americans ages 50–75 (n = 394) from the Baltimore-Washington DC Metropolitan Area to examine the associations of high risk (HR) sleep apnea with diagnoseable hypercholesterolemia and diabetes, as well as the following biomarkers: total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), total cholesterol/HDL-C ratio, triglycerides, and glucose (non-fasting). Poisson models included demographic factors, socioeconomic status, and body mass index (BMI). We tested for potential effect modifiers.

Results: HR-sleep apnea was associated with higher LDL-C level ($\beta = 14.56$, p < 0.05) and higher total cholesterol/HDL ratio ($\beta = 0.64$, p < 0.01). Younger respondents had higher levels of triglycerides associated with HR-sleep apnea than older respondents. For men, HR-sleep apnea was associated with higher total cholesterol, total cholesterol/HDL-C ratio, and triglycerides. Obese and overweight respondents had positive associations between HR-sleep apnea and total cholesterol, total cholesterol/HDL ratio, and triglycerides, while underweight/normal weight individuals did not. The interactions between snoring and daytime sleepiness were associated with hypercholesterolemia and diabetes.

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Keywords

Sleep apnea; Cardiovascular disease; Hypercholesterolemia; Diabetes; Cholesterol; Asian Americans

1. Introduction

Left untreated, obstructive sleep apnea—defined as the repeated col-lapse of the upper airway during sleep which induces disruptions to breathing and, often times, arousal from sleep—increases risk of cardiovascular disease (CVD) morbidity and mortality [1, 2]. Past longitudinal research shows that sleep apnea increases incidence of coronary disease, stroke, and heart failure [3, 4]. The pathways linking sleep apnea to CVD include increasing oxidative stress and inflammation [5, 6]. Furthermore, sleep apnea is associated with metabolic syndrome and insulin resistance, which also heighten risk of end-stage CVD [2, 5]. Epidemiological studies have demonstrated that sleep apnea increases risk of dyslipidemia and hyperglycemia. [7–12] This research overall indicates the importance of intervening to treat sleep apnea as a means of preventing CVD in the population [2, 3, 5].

While the links between sleep apnea and CVD risk have been demonstrated previously, most of these studies were conducted with samples of white populations of European descent [7–9], and very few studies have examined these associations for Asian Americans. Asian Americans are often not viewed as having CVD disparities, with age-adjusted prevalence of all heart diseases among Asian American adults being lower (4.4%) compared to non-Hispanic white Americans (5.8%) [13]. In addition, Asian Americans overall have lower rates of overweight and obesity—a risk factor of both sleep apnea and CVD [14]. Nevertheless, heart disease and stroke remain the second and third leading causes of death among Asian Americans [15]. More detailed mortality data reveal important cardiovascular health disparities: mortality rates for hypertensive disease and CVD are proportionately higher for Asian Americans compared to non-Hispanic white Americans [16]. Furthermore, Asian Americans are 40% more likely to be diagnosed with diabetes compared to white Americans [17], with certain origin groups such as Koreans being diagnosed with diabetes at earlier ages [18]. Research finds that Asian Americans start to have metabolic problems -including higher blood sugar, higher blood pressure, and abnormal cholesterol and triglyceride levels-at lower body mass indices (BMIs) compared to other populations, highlighting the importance of addressing CVD risk for Asian Americans who may not be considered overweight or obese according to the standard BMI cut-off points commonly used in the United States (U.S.) [19]. Research on Asian American health usually combines all people of Asian descent together, but it is important to note that Asian Americans are extremely diverse with various countries of origin, histories of immigration, and health behavior practices. More research is needed to highlight the unique health needs of specific Asian groups.

Although studies on sleep among Asian American groups are limited, initial evidence show that they experience sleep disparities as well. Population research combining Asian Americans together shows that they have overall shorter mean sleep duration than white Americans [20]. Furthermore, one study reported greater odds of daytime sleepiness among Chinese American men compared to white men [21]. Evidence indicates Asian Americans as a whole have higher risk of sleep apnea compared to white Americans [22], with Chinese men in particular experiencing higher rates of sleep apnea compared to white men, adjusting for age and BMI [21, 23]. Chen et al. (2016) found that compared to white, Black, and Hispanic individuals, Chinese individuals had the strongest association between BMI and sleep disordered breathing, and this association was more pronounced among women and younger (67 years-old) Chinese people [24]. In fact, for every increment of higher BMI, Chinese participants experienced an increase in sleep disordered breathing score at a rate almost double that of white, Black, and Hispanic participants. So far, there has not been research on sleep health for other Asian origin groups in the U.S. besides Chinese Americans.

Preliminary research shows that Chinese Americans are at risk for sleep apnea [21–23], and other research demonstrates Asian Americans are at high risk of CVD, hypertension, and diabetes [16, 18, 19], no study to date has examined the associations between sleep apnea and CVD risk among Asian Americans. This study fills this gap by examing these associations for Chinese and Korean Americans, the two largest East Asian populations in the U.S. [25]. Some studies have found sleep apnea to be associated with higher rates of hypercholesterolemia, dyslipidemia, metabolic syndrome, and diabetes in Asian countries such as China and Taiwan [26–28], but studies of Chinese and Korean populations in the U.S. are lacking. It is possible that associations between sleep apnea and CVD among Chinese and Korean Americans are different than in other populations due to higher risk of both conditions at lower BMIs. Furthermore, as 62% and 59% of Chinese and Korean Americans, respectively, were born outside of the U.S., they likely experience unique stressors including occupational/economic strain, limited healthcare access, and cultural/ language barriers that can subsequently impact both sleep and CVD risk. Multi-ethnic samples in the U.S. that include white, Black, and Native American participants have demonstrated associations between sleep apnea and CVD risk factors [10, 11]. However, these studies did not have sufficient sample sizes to examine Asian Americans separately. Only one multi-ethnic study examining the associations between sleep apnea and glucose level included only Chinese Americans, but this study found no statistically significant associations for this Asian American subgroup [29]. Therefore, more research is needed to clarify the potential association between sleep apnea and CVD, which will inform whether treating sleep apnea among Chinese and Korean Americans would be an appropriate intervention to decrease risk of CVD and diabetes. If so, which Chinese and Korean American patients may benefit most from intervention with regards to age, gender, and other comorbidities?

To address this gap in the literature, our study examines the associations between risk of sleep apnea and CVD in a sample of Chinese and Korean American adults. First, we test whether risk of sleep apnea is associated with diagnoseable hypercholesterolemia and diabetes, as well as with several biomarkers of metabolic abnormalities linked to

CVD risk (total cholesterol, low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol [HDL-C], total cholesterol-to-HDL-C [total cholesterol/HDL-C] ratio, triglycerides, and glucose). Second, we test whether these associations between sleep apnea and metabolic abnormalities are moderated by age, gender, Asian origin group, obesity, and number of chronic conditions [10, 26, 27]. Third, we examine whether the associations between snoring (a prominent indicator of sleep apnea) and CVD varies by daytime sleepiness [30, 31]. The results of the current study will determine whether there is a greater risk of CVD among Chinese and Korean Americans at high risk of sleep apnea and will further identify which segments of Chinese and Korean American populations may benefit most from intervention to decrease future CVD risk.

2. Methods

2.1. Sample

We used data from a randomized controlled trial to increase colorectal cancer screening among Chinese and Korean Americans. Study participants were between the ages of 50 and 75 living in the Baltimore-Washington DC Metropolitan Area, and they were recruited from primary care physicians' clinics in Maryland and Northern Virginia. The baseline survey data were collected from August 2018 to June 2020. After signing informed consent forms, 400 participants (200 Chinese and 200 Korean Americans) completed the survey either in-person or by phone in their preferred language (Mandarin, Korean, or English). Eighty-nine percent of the participants (155 Chinese and 200 Korean Americans) completed a self-administered questionnaire in-person, while 11% of the participants (45 Chinese Americans) completed a research assistant-led phone survey because of the COVID-19 outbreak in March 2020. This study was approved by the Institutional Review Boards of the University of California, Irvine and the University of Maryland, College Park.

We have different analytic samples depending on measure. For the 355 participants who completed the in-person baseline survey, anthropometric measurements (e.g., height and weight) and blood samples were collected by trained research assistants. Two out of these 355 participants refused to provide their weight and height. For the 45 participants who completed phone surveys, self-reported weight and height were collected during the 6-month follow-up phone survey, and four out of these 45 participants refused to provide their weight and height. Thus, our first analytic sample was n = 394 (excluding six participants without weight and height information). The second analytic sample was based on biomarker measurements (total cholesterol, LDL-C, HDL-C, total cholesterol/HDL-C ratio, triglycerides, and glucose) from random blood samples. Study participants were not required to fast prior to blood sample collection, which occurred anytime between midmorning to afternoon. Information on biomarker measurements were not available for the 45 people who completed a phone survey and could not provide blood samples. For the 355 people who completed an in-person survey, 25 participants refused to participate in blood sample collection, and two participants had missing values for LDL-C and triglycerides due to machine errors. Therefore, the analytic sample for biomarker measurement outcomes was n = 328.

2.2. Dependent variables

As dependent variables, we used six biomarker measurements of CVD risk (total cholesterol, LDL-C, HDL-C, total cholesterol/HDL ratio, triglycerides, and glucose) and two chronic conditions (hypercholesterolemia and diabetes). The biomarker measurements were based on objective measurement from blood samples. A blood measuring device— CardioChek Analyzer, Polymer Technology Systems, Inc.—was used to estimate total cholesterol, LDL-C, HDL-C, triglycerides, and glucose. This device has been shown to meet the accuracy guidelines established by the National Cholesterol Education Program of the National Institutes of Health in published research and has been used in several other studies [32, 33]. The lowest possible cholesterol level reading was 100 mg/dL, and the highest possible triglyceride level reading was 500 mg/dL. We had five participants who had total cholesterol values of 100 mg/dL and nine participants who had triglycerides values of 500 mg/dL. We used total cholesterol, LDL-C, HDL-C, HDL-C, total cholesterol/HDL-C ratio, triglycerides, and glucose as continuous variables.

Hypercholesterolemia and diabetes were based on a combination of objective biomarker measurement and two self-reported variables on diagnosis by a doctor or other healthcare professional (in the past year and ever) and medication usage. Hypercholesterolemia was defined as having one or more of the following: total cholesterol measurement 240 mg/dL [34], self-report of currently taking anti-hypercholesterolemia medication, or self-report of having been diagnosed with high cholesterol by a doctor or other healthcare professional in the past year or ever. Diabetes was defined as having one or more of the following: random glucose measurement 200 mg/dL [35], self-report of currently taking anti-diabetes medication, or self-report of having been diagnosed with diabetes by a doctor or other healthcare professional in the past year or ever. Both hypercholesterolemia and diabetes were used as binary variables in the analyses. As self-reported information on diagnosis and medication usage were collected for all participants, analyses for hypercholesterolemia and diabetes measurement outcomes (n = 328).

2.3. Sleep apnea risk

To identify the risk of having sleep apnea, we used the Berlin questionnaire—a valid clinical screening test and epidemiological tool to identify people with sleep apnea [36]. As the Berlin sleep apnea scale includes questions about obesity, which is one of the independent variables of interest in this study, we used a modified high-risk (HR) sleep apnea score that excluded obesity [37]. Three subcategories were used from the questionnaire: *Subcategory 1*, snoring, provided a positive score when snoring intensity was "louder than talking" and/or snoring frequency was "3–4 times a week" or "almost every day"; *Subcategory 2*, breathing pauses (anyone noticing you stop breathing during sleep) provided a positive score when the response was "3–4 times a week" or "almost every day"; and *Subcategory 3*, sleepiness (feeling tired or fatigued in the morning and/or daytime) provided a positive score when the response was "3–4 times a week" or "almost every day." Participants were classified as HR-sleep apnea when they scored positive on two or more subcategories, and the participants who did not score positive in any or only one subcategory were categorized

as low-risk (LR)-sleep apnea. The risk of having sleep apnea was used as a binary variable (HR-sleep apnea or LR-sleep apnea) in the analysis.

2.4. Covariates

Sociodemographic characteristics included age, gender, Asian origin group, marital status, education, household income, employment status, and health insurance based on self-report in the survey. Age was used as a continuous variable in years, gender was classified as male and female, and Asian origin group was categorized as Chinese and Korean. Marital status was used as a binary variable: married/cohabiting and not currently married. We classified education into five categories: less than high school, high school graduate or GED, business/vocational school/some college, college graduate, and attended graduate/ professional school. We categorized household income into six categories: <\$20,000, \$20,000-\$39,999, \$40,000-\$59,999, \$60,000-\$79,999, \$80,000-\$99,999, and \$100,000. Employment status was categorized as full time, part time, and not employed. Health insurance was classified as private health insurance, Medicare/Medicaid, and no health insurance. Continuous BMI was also included as a control variable, as it may be associated with both sleep apnea and CVD risk factors.

Potential effect modifiers included age, gender, Asian origin, obesity, number of chronic conditions, and daytime sleepiness. When testing age as a potential effect modifier, we dichotomized the variable by median age (< 58 years-old or 58 years-old). Obesity was a three-category variable determined using cut-off points for BMI that are specific for Asian populations: underweight/normal (< 23 kg/m²), over-weight (23–26.9 kg/m²), or obese (27 kg/m²) [38, 39]. Number of chronic conditions was determined by counting the number of positive responses when participants were asked whether or not they had been told by a doctor in the past year that they had any of the following nine conditions: high blood pressure, high cholesterol, heart attack, cancer, stroke, diabetes, anxiety or depression, breathing problems such as asthma or emphysema, and any other health problems. This variable was dichotomized at the median of one (> 1 chronic condition, or 1 chronic condition). Daytime sleepiness was determined from subcategory 3 of the Berlin questionnaire and used as a dichotomous variable (no sleepiness or positive sleepiness).

2.5. Statistical analysis

First, we conducted descriptive analysis for the sample overall and stratified by the risk of sleep apnea. We calculated the mean and standard error for all continuous variables and reported frequencies and percentages for all categorical variables. To compare the differences between origin groups, we conducted two sample t-tests for continuous variables and chi-square tests for categorical variables. Second, we used Poisson regression models with robust error variance to estimate associations between sleep apnea risk and chronic conditions (hypercholesterolemia and diabetes) and linear regression models to estimate associations between sleep apnea risk and cholesterol, LDL-C, HDL-C, total cholesterol/HDL-C ratio, triglycerides, and glucose). Three regression models were conducted for each outcome. Model 1 included sleep apnea risk, age, and gender as independent variables. Model 2 added Asian origin, marital status, education, household income, employment status, and health insurance to Model 1. Model 3 added

continuous BMI to Model 2. Then we tested age, gender, Asian origin, obesity, and number of chronic conditions as potential effect modifiers of the associations between sleep apnea risk and the outcomes, accounting for covariates of Model 2 and Model 3. We conducted stratified analyses by the effect modifiers if the p-values of effect modifications were less than 0.05. Finally, to capture a potentially more severe sleep apnea subtype associated with excessive daytime sleepiness, we conducted stratified analyses by sleepiness (subcategory 3 of sleep apnea risk) to estimate the associations between snoring (subcategory 1 of the sleep apnea risk) and all dependent variables, accounting for covariates in Model 2 and Model 3. All statistical analyses were computed using SAS, version 9.4.

3. Results

Table 1 presents the characteristics of the participants. Of the 394 participants, 345 (87.6%) were classified as LR-sleep apnea, while 49 (12.4%) were classified as HR-sleep apnea. There were statistically significant differences between the two sleep apnea groups by gender, HDL-C, total cholesterol/HDL-C ratio, and triglycerides. Individuals with a HR-sleep apnea were more likely to be male (63.3%) relative to those with a LR-sleep apnea (male: 44.9%). Respondents with HR-sleep apnea had significantly lower HDL-C (49.0 mg/dL), higher total cholesterol/HDL-C ratio (4.2), and higher triglycerides (229.2 mg/dL) as compared to respondents with LR-sleep apnea (HDL-C: 54.7 mg/dL, total cholesterol/HDL-C ratio: 3.5, triglycerides: 193.1 mg/dL).

Table 2 displays the results of Poisson regression models to estimate associations between sleep apnea risk, hypercholesterolemia, and diabetes. The prevalence ratios (PRs) and 95% confidence intervals (CI) are reported. Having HR-sleep apnea was positively associated with having hypercholesterolemia in Model 1 (p < 0.05). Participants with HR-sleep apnea were at 1.28 (95% CI: 1.00–1.62) times greater risk of having hypercholesterolemia compared to those with LR-sleep apnea, accounting for age and gender in Model 1. The significance was attenuated in Model 2 and Model 3 with further adjustment for Asian origin, marital status, socioeconomic status, health insurance, and BMI. Among the covariates, age (aPR: 1.03, 95% CI: 1.01–1.05, *p* < 0.01) and BMI (aPR: 1.04, 95% CI: 1.02-1.07, p < 0.01) were positively associated with having hypercholesterolemia. Having HR-sleep apnea was positively associated with having diabetes in Model 1 (aPR: 1.47, 95% CI: 0.94-2.31, p < 0.1). The association remained similar in Model 2, but the association was slightly attenuated when BMI was further adjusted in Model 3 (aPR: 1.44, 95% CI: 0.90-2.31, p > 0.1). Chinese participants were slightly less likely to have diabetes than Korean participants in Model 2 (aPR: 0.68, 95% CI: 0.43-1.06, p < 0.1), but this association was attenuated after accounting for BMI in Model 3. Among the covariates in Model 3, age (aPR: 1.06, 95% CI: 1.02–1.09, p < 0.01), working part time relative to working full time (aPR: 1.62, 95% CI: 1.04–2.51, p < 0.05), and BMI (aPR: 1.05, 95% CI: 1.00–1.10, p < 0.05) were positively associated with having diabetes.

Table 3 shows the results of linear regression models to estimate associations between sleep apnea risk and biomarkers, adjusting for covariates. Participants with HR-sleep apnea had significantly higher LDL-C and total cholesterol/HDL-C ratio, which was robust to the adjustment for all covariates in Model 3. When all covariates were adjusted for, HR-sleep

apnea was associated with higher level of LDL-C by an average of 14.56 mg/dL (p < 0.05), while it was associated with higher total cholesterol/HDL-C ratio by 0.64 (p < 0.01). Additionally, individuals with HR-sleep apnea were more likely to have lower HDL-C by 3.99 (p < 0.1) and higher triglycerides by 30.32 mg/dL (p < 0.1) compared to those with LR-sleep apnea in Model 2. However, these associations were attenuated when BMI was included in Model 3. We did not find any significant associations between HR-sleep apnea and total cholesterol and glucose, although trends suggest a positive association.

We tested age, gender, Asian origin, obesity, and number of chronic conditions as potential effect modifiers of the association between sleep apnearisk and CVD risk factors. The stratified analyses of the effect modifications with p-values less than 0.05 are presented in Table 4. Age was a significant modifier of the association between sleep apnea and triglycerides. Younger participants (age < 58) with HR-sleep apnea had a higher level of triglycerides by 77.73 mg/dL (p < 0.01), while the association between sleep apnearisk and triglycerides was not significant among older participants (age 58). Gender and obesity were significant modifiers for three outcomes: total cholesterol, total cholesterol/HDL-C ratio, and triglycerides. Men with HR-sleep apnea had higher levels of total cholesterol, total cholesterol/HDL-C ratio, and triglycerides compared to those with LR-sleep apnea by an average of 25.50 mg/dL (p < 0.05), 1.07 (p < 0.01), and 51.56 mg/dL (p < 0.05), respectively. The associations between sleep apnea risk score and these three outcomes were not significant among women. Models stratified by obesity indicate that obese participants with HR-sleep apnea had 45.68 mg/dL higher total cholesterol (p < 0.05), while sleep apnea was not strongly associated with total cholesterol for underweight/normal or overweight people. Overweight individuals had 1.36 higher total cholesterol/HDL-C ratio and 81.64 mg/dL higher triglycerides (p < 0.01) when they had HR-sleep apnea compared to LR-sleep apnea. However, we did not find any significant associations between sleep apnea risk and these two outcomes for individuals who were underweight/normal or obese. The number of chronic conditions was significant modifier of sleep apnea for three outcomes: total cholesterol, LDL-C, and total cholesterol/HDL-C ratio. For participants with more than one chronic condition, HR-sleep apnea was associated with higher level of total cholesterol, LDL-C, and total cholesterol/HDL-C ratio by an average of 38.28 mg/dL, 46.58 mg/dL, and 1.00, respectively (p < 0.01). The associations between sleep apnearisk and the three outcomes were not significant among those with zero or only one chronic condition.

Table 5a shows the results of Poisson regression models to estimate associations between snoring, hypercholesterolemia, and diabetes, stratified by sleepiness. Among individuals without sleepiness, those with positive snoring were at 1.46 (95% CI: 1.15–1.84) and 1.39 (95% CI: 1.09–1.79) times greater risk of having hypercholesterolemia in Models 2 and 3, compared to those without snoring (p < 0.01). However, the association between snoring and hypercholesterolemia was not significant among participants with positive sleepiness. Among individuals with positive sleepiness, positive snoring was associated with greater risk of having diabetes (Model 2: PR: 2.06, CI: 1.08–3.93, p < 0.05; Model 3: PR: 2.05, CI: 1.07–3.91, p < 0.05). For those without sleepiness, there was not a significant association between positive snoring and diabetes (Model 3: PR: 0.65, CI: 0.39–1.08, p < 0.1).

Table 5b displays the results of linear regression models to estimate associations between snoring and biomarkers stratified by sleepiness. Among participants without sleepiness, those with positive snoring had lower HDL-C level by 5.28 mg/dL in Model 2 (p < 0.01) compared to those without snoring, but the association decreased after further adjustment for BMI in Model 3 ($\beta = -3.64$, p < 0.1). The association between snoring and HDL-C was not significant for those with positive sleepiness. Individuals with positive snoring had higher total cholesterol/HDL-C ratio (p < 0.05) in Model 2, no matter what sleepiness status they were, but the association was stronger for those with positive sleepiness (positive sleepiness: $\beta = 0.86$, p < 0.05; no sleepiness: $\beta = 0.34$, p < 0.05). When we further adjusted for BMI in Model 3, the significance was attenuated for both those with and without sleepiness. In models stratified by sleepiness, we did not find any significant associations between snoring and total cholesterol, LDL-C, triglycerides, and glucose at p < 0.05.

Asian origin was not a significant effect modifier in any of the analyses.

4. Discussion

In this paper, we examined the associations between sleep apnea and CVD risk among Chinese and Korean Americans across multiple metabolic parameters. Our study suggests that overall Chinese and Korean Americans at high risk of sleep apnea have a higher burden of metabolic abnormalities than those at low risk of sleep apnea, with the strongest associations found among younger (< 58 years-old), men, overweight or obese individuals, and among people with existing chronic conditions. Associations also varied by the interaction between snoring and daytime sleepiness. This is the first study to our knowledge to demonstrate these associations with a sufficient sample for Asian American subpopulations.

We found evidence of sleep apnea among Chinese and Korean Americans being associated with dyslipedemia in the sample overall. Adjusting for age and gender, Chinese and Korean Americans at high risk of sleep apnea were at elevated risk of hypercholesterolemia. Although this association was attenuated after including BMI in the regression model, the trend suggests that people at high risk of sleep apnea may have 22% greater risk of hypercholesterolemia than those with low risk of sleep apnea, accounting for all else. Furthermore, we found that high risk of sleep apnea was strongly associated with higher LDL-C and total cholesterol/HDL ratio, even after adjustment for demographic factors, socioeconomic status, and BMI. These findings indicate greater risk for CVD associated with sleep apnea taken from reliable measures from random blood samples. Our results confirm prior studies showing strong associations between sleep apnea and higher levels of LDL-C and higher total cholesterol/HDL-C ratio [12, 27]. However, these previous studies were conducted in samples in China and the United Kingdom, and this is the first study to show this association among Chinese and Korean Americans.

Chinese and Korean Americans with high risk of sleep apnea may be more likely to have diabetes. Although the association between high risk of sleep apnea and diagnosed diabetes was positive, this association was only marginally significant (p < 0.1) when adjusting for age and gender alone. The association was attenuated after including other covariates.

Overall, the trend suggests that those at high risk of sleep apnea might have on average 44% greater risk of diabetes, accounting for all else, but these trends should be interpreted with caution. High risk of sleep apnea did not have a statistically strong association with random glucose measurement. As with other studies, our current sample may have been underpowered to demonstrate strong associations between sleep apnea, diabetes, and glucose [10, 12, 26, 29]. Previous studies have been able to demonstrate associations between sleep apnea and glucose intolerance [8, 9, 28, 29].

Notably, our study found that the associations between sleep apnea and metabolic abnormalities were moderated by age, gender, obesity, and number of chronic conditions. Younger people (< 58 years-old) in our sample had higher levels of triglycerides when they had high risk of sleep apnea in comparison to those who were older. The apparently stronger associations in younger compared to older individuals is also consistent with evidence that associations between sleep apnea and incident coronary heart disease is stronger in younger individuals [40]. While the reasons for this are not well understood, sleep apnea in younger individuals may cause greater autonomic nervous system dysfunction, with secondary effects on inflammation and metabolic abnormalies. Furthermore, our study found that the associations between high risk of sleep apnea and higher total cholesterol, total cholesterol/HDL-C ratio, and triglycerides were strong in the positive direction for men, but not for women. These results coincide with findings by Geovanini et al. (2018) that similarly found that the associations between sleep apnea and CVD risk factors were stronger among younger people and men [10]. Stronger associations in men may reflect differences in underlying sleep apnea endotypes in men and women. Specifically, recent data indicate that men tend to have longer apneas and hypopneas, which would lead to more profound oxyhemoglobin desaturation-a key driver for metabolic disturbances and cardiovascular disease [41, 42].

Stratified analyses also revealed that the association between high risk sleep apnea and total cholesterol was strongest among obese people (BMI 27 kg/m²), and the associations between high risk sleep apnea with total cholesterol/HDL ratio and triglycerides were strongest among overweight people (BMI 23 to 26.9 kg/m²). These findings may reflect the additive or synergistic effects of are similar to other studies in China and Taiwan that found associations between sleep apnea and cholesterol were greater in higher BMI groups [26, 27]. Our study is the first to show stronger associations between sleep apnea and total cholesterol, LDL-C, and total cholesterol/HDL ratio among primarily people with more than one chronic conditions. In sum, associations between sleep apnea and biomarkers for CVD risk varied by age and gender. Furthermore, people with high risk of sleep apnea in combination with other comorbidities, such as overweight/obesity and other chronic conditions, may be particularly at risk for higher cholesterol levels.

This study further explored a potential interaction between snoring and daytime sleepiness on CVD risk factors. Excessive daytime sleepiness is a marker of more severe-potentially more chronic sleep apnea, as well as a marker for potentially elevations in inflammatory cytokines [43, 44]. Accordingly, excessive daytime sleepiness has been studied as a potential marker of increased CVD incidence in individuals with sleep apnea [45]. We found that Chinese and Korean Americans who reported snoring but did not report excessive daytime

sleepiness had about 40% higher risk of hypercholesterolemia than people who did not snore. This is in contrast to study by Endeshaw et al. (2013) that found higher incidence of CVD when participants reported both snoring and daytime sleepiness [31]. However, our study also found that respondents who were positive for both snoring and daytime sleepiness were at over two times greater risk for diabetes than those who did not snore. This finding seems similar to the Endeshaw et al. (2013) study of 70 to 79 year-old Black and white Americans, as well as findings from Lindberg et al. (2007) in a sample of adult women in Sweden [30, 31]. Based on our data, it is not clear whether excessive daytime sleepiness moderates associations between sleep apnea and cardiometabolic abnormalities in individuals of Chinese or Korean descent.

Our study found similar associations between sleep apnea and CVD risk factors among people of Chinese compared to those of Korean descent. Many scholars and community advocates have stressed the importance of disaggregating Asian American data to look at health differences by group defined by country of origin, due to different contexts of immigration, socioeconomic indicators, and cultural practices. Although we did not find significant differences between Chinese and Korean Americans in the current study, a major strength of our sample was the ability to explore potential differences by country of origin.

This study has limitations that should be noted. The current analysis is cross-sectional, so longitudinal studies with follow-up over longer period of time could determine whether sleep apnea increases risks for CVD and diabetes over the life course. In this study, we demonstrate associations, but we cannot make causal inferences about the directionality of sleep apnea with CVD risks and biomarkers. Furthermore, this study was conducted among Chinese and Korean American immigrants living in the Balitmore-Washington DC Metropolitan Area. The findings in this study may not be generalizable to Chinese and Korean American populations outside of this geographic area, nor to other Asian Americans with origins other than China or Korea. Future studies should examine associations between sleep and CVD among other subpopulations, as the Asian American grouping is extremely diverse. Respondents in our sample were between the ages of 50 to 75, so only represent this older age group. Future sleep studies among Asian Americans should include a broader range of ages. Blood samples were collected at random, so we could not measure fasting glucose-a more accurate indicator of diabetes risk. Since respondents were assessed at different times of the day, it was unfortunately not possible to capture fasting glucose in this sample. The findings for glucose in our study should be interpreted accordingly. Our measure of sleep apnea relied on self-reported modified Berlin questionnaire, rather than objectively measured sleep apnea, which is more ideal. Lastly, 11% of our sample's data were collected during the COVID-19 pandemic leading to some missing biomarker data. Furthermore, prior studies have demonstrated the effect of the COVID-19 pandemic on sleep health, but the sample size collected for our study during lockdowns was too small to do a separate analysis of the effect of the pandemic [46, 47].

Nevertheless, the major strength of this study is the ability to examine associations between sleep and risk factors of CVD and diabetes in a sample of Chinese and Korean Americans, with surveys conducted in the participants' preferred language of either Mandarin, Korean, or English. Notably, the response rate for blood sample collection was quite high in this

study (93% response rate among in-person participants), especially compared to populationbased studies that also collect blood samples, such as the National Health and Nutrition Examination Survey (49% final examination response rate in 2017–2018) [48]. This is an indication of the value of samples collected through engagement with communities to improve data quality for groups commonly underrepresented in research [49, 50].

These findings demonstrated associations between sleep apnea risk and cardiometabolic abnormalities among Asian American groups. Health practitioners should recognize the potential links between sleep and cardiovascular health for their Chinese and Korean American patients. Future research should examine the efficacy of addressing sleep apnea as a way to prevent CVD burden in Asian American populations. Efforts to identify and treat sleep apnea to prevent CVD may especially be beneficial for Chinese and Korean American patients who are younger, who are men, who have existing chronic conditions, and who are overweight or obese according to the lower BMI cut-off points suggested for Asian populations (normal 18.5–22.9 kg/m², overweight 23–27.5 kg/m², and obese 27.5 kg/m²) [19, 38].

Declaration of Competing Interest

This research was supported by the National Institute on Minority Health and Health Disparities of the National Institutes of Health under award number R01MD012778 (PI: Sunmin Lee, Sc.D., M.P.H.). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The authors have no other conflicts of interest to disclose. This research was performed at the University of Maryland and the University of California, Irvine. All authors have seen and approved this manuscript.

Abbreviations:

CVD	cardiovascular disease	
BMI	body mass index	
LDL-C	low-density lipoprotein cholesterol	
HDL-C	high-density lipoprotein cholesterol	
HR-sleep apnea	high-risk sleep apnea	
LR-sleep apnea	low-risk sleep apnea	
PR	prevalence ratio	
CI	confidence interval	
SE	standard error	

References

- [1]. Drager LF, McEvoy RD, Barbe F, Lorenzi-Filho G, Redline S. Sleep apnea and cardiovascular disease. Circulation 2017; 136 (19):1840–50. doi:10.1161/CIRCULATION-AHA.117.029400.
 [PubMed: 29109195]
- [2]. Drager LF, Polotsky VY, O'Donnell CP, Cravo SL, Lorenzi-Filho G, Machado BH. Translational approaches to understanding metabolic dysfunction and cardiovascular consequences of

obstructive sleep apnea. Am J Physiol Heart Circ Physiol 2015; 309 (7):H1101–11. doi:10.1152/ajpheart.00094.2015. [PubMed: 26232233]

- [3]. Monahan K, Redline S. Role of obstructive sleep apnea in cardiovascular disease. Curr Opin Cardiol 2011; 26 (6):541–7. doi:10.1097/HCO.0b013e32834b806a. [PubMed: 21993356]
- [4]. Gami Apoor S, Olson Eric J, Shen Win K, et al. Obstructive sleep apnea and the risk of sudden cardiac death. J Am Coll Cardiol 2013; 62 (7):610–16 2013/08/13. doi:10.1016/ j.jacc.2013.04.080. [PubMed: 23770166]
- [5]. Javaheri S, Barbe F, Campos-Rodriguez F, et al. Sleep apnea: types, mechanisms, and clinical cardiovascular consequences. J Am Coll Cardiol 2017; 69 (7):841–58 Feb 21. doi: 10.1016/ j.jacc.2016.11.069. [PubMed: 28209226]
- [6]. Baltzis D, Bakker JP, Patel SR, Veves A. Obstructive sleep apnea and vascular diseases. 10.1002/ cphy.c150029. Compr Physiol 2016:1519–28 2016/06/13Major Reference Works. doi:10.1002/ cphy.c150029.
- [7]. Gunduz C, Basoglu OK, Hedner J, et al. Obstructive sleep apnoea independently predicts lipid levels: data from the European sleep apnea database. Respirology 2018; 23 (12):1180–9 Dec. doi:10.1111/resp.13372. [PubMed: 30133061]
- [8]. Huang TAO, Lin BM, Stampfer MJ, Tworoger SAO, Hu FB, Redline S. A population-based study of the bidirectional association between obstructive sleep apnea and type 2 diabetes in three prospective U.S. Cohorts. Diabetes Care 2018; 41 (10):2111–19. [PubMed: 30072403]
- [9]. Seicean S, Kirchner Hl Fau, Gottlieb DJ, Gottlieb Dj Fau, Punjabi NM, et al. Sleep-disordered breathing and impaired glucose metabolism in normal-weight and overweight/obese individuals: the sleep heart health study. Diabetes Care 2008; 31 (5):1001–6. [PubMed: 18268072]
- [10]. Geovanini GR, Wang R, Weng J, et al. Association between obstructive sleep apnea and cardiovascular risk factors: variation by age, sex, and race. the multi-ethnic study of atherosclerosis. Ann Am Thorac Soc 2018; 15 (8):970–7. doi:10.1513/AnnalsATS.201802-1210C [PubMed: 29742365]
- [11]. Newman AB, Nieto Fj Fau Guidry U, Guidry U, Fau Lind BK, et al. Relation of sleepdisordered breathing to cardiovascular disease risk factors: the sleep heart health study. Am J Epidemiol 2001; 154 (1):50–9. [PubMed: 11434366]
- [12]. Coughlin SR, Mawdsley L, Fau Mugarza JA, Mugarza Ja Fau Calverley PMA, Calverley Pm Fau - Wilding JPH, Wilding JP. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. Eur Heart J 2004; 29 (5):735–41.
- [13]. Centers for Disease Control and Prevention Summary health statistics: national health interview survey. Centers for Disease Control and Prevention; 2018,. Table A-1a http://www.cdc.gov/nchs/ nhis/shs/tables.htm.
- [14]. National Center for Health Statistics. Health. United States, 2018. Table 26. Hyattsville, MD: National Center for Health Statistics; 2019 https://www.cdc.gov/nchs/data/hus/hus18.pdf.
- [15]. Heron M Deaths: leading causes for 2017. Natl Vital Stat Rep 2019; 68 (6):1–77 June 24, 2019.
- [16]. Jose PO, Frank AT, Kapphahn KI, et al. Cardiovascular disease mortality in Asian Americans. J Am Coll Cardiol 2014; 64 (23):2486–94 Dec 16. doi:10.1016/j.jacc.2014.08.048. [PubMed: 25500233]
- [17]. Centers for Disease Control and Prevention Summary health statistics: national health interview survey. Centers for Disease Control and Prevention; 2018. Table A-4a http://www.cdc.gov/nchs/ nhis/shs/tables.htm.
- [18]. Becerra MB, Becerra BJ. Disparities in age at diabetes diagnosis among asian americans: implications for early preventive measures. Prev Chronic Dis 2015; 12 E146–E146. doi:10.5888/ pcd12.150006.
- [19]. Yi SS, Kwon SC, Wyatt L, Islam N, Trinh-Shevrin C. Weighing in on the hidden Asian American obesity epidemic. Prev Med 2015; 73 :6–9 2015/04/01/. doi:10.1016/j.ypmed.2015.01.007.
 [PubMed: 25602909]
- [20]. Song Y, Ancoli-Israel S, Lewis CE, Redline S, Harrison SL, Stone KL. The association of race/ ethnicity with objectively measured sleep characteristics in older men. Behav Sleep Med 2011; 10 (1):54–69 Dec 28. doi:10.1080/15402002.2012.636276. [PubMed: 22250779]

- [21]. Chen X, Wang R, Zee P, et al. Racial/ethnic differences in sleep disturbances: the multi-ethnic study of atherosclerosis (MESA). Sleep 2015; 38 (6):877–88 Jun 1. doi:10.5665/sleep.4732.
 [PubMed: 25409106]
- [22]. Mehra R, Stone KL, Blackwell T, et al. Prevalence and correlates of sleep-disordered breathing in older men: osteoporotic fractures in men sleep study. J Am Geriatr Soc 2007; 55 (9):1356–64 Sep. doi:10.1111/j.1532-5415.2007.01290.x. [PubMed: 17767677]
- [23]. Hnin K, Mukherjee S, Antic NA, et al. The impact of ethnicity on the prevalence and severity of obstructive sleep apnea. Sleep Med Rev 2018; 41 :78–86 2018/10/01/. doi:10.1016/ j.smrv.2018.01.003. [PubMed: 30149931]
- [24]. Chen X, Wang R, Lutsey PL, et al. Racial/ethnic differences in the associations between obesity measures and severity of sleep-disordered breathing: the multi-ethnic study of atherosclerosis. Sleep Med 2016; 26 :46–53 10//. doi:10.1016/j.sleep.2015.06.003. [PubMed: 26459687]
- [25]. Budiman A, Ruiz N Key facts about Asian origin groups in the U.S. Pew Research Center. 2021. Accessed May 23rd, 2021. https://www.pewresearch.org/fact-tank/2021/04/29/key-factsabout-asian-origin-groups-in-the-u-s/
- [26]. Wu WT, Tsai SS, Shih TS, et al. The association between obstructive sleep apnea and metabolic markers and lipid profiles. PLoS One 2015; 10 (6):e0130279. doi:10.1371/journal.pone.0130279.
- [27]. Zhang N, Chen Y, Chen S, et al. Self-reported snoring is associated with dyslipidemia, high total cholesterol, and high low-density lipoprotein cholesterol in obesity: a cross-sectional study from a rural area of China. LID - 10.3390/ijerph14010086 [doi] LID - 86. Int J Environ Res Public Health 2017; 14 (1):1–10.
- [28]. Liu CL, Wu CS. Assessing whether the association between sleep apnea and diabetes is bidirectional. Can J Diabetes 2017; 41 (2):197–203 [PubMed: 27989495]
- [29]. Bakker JP, Weng J, Wang R, Redline S, Punjabi NM, Patel SR. Associations between obstructive sleep apnea, sleep duration, and abnormal fasting glucose. The multi-ethnic study of atherosclerosis. Am J RespirCrit Care Med 2015; 192 (6):745–53
- [30]. Lindberg E, Berne C, Franklin KA, Svensson M, Janson C. Snoring and day-time sleepiness as risk factors for hypertension and diabetes in women—a population-based study. Respir Med 2007; 101 (6):1283–90 2007/06/01/. doi:10.1016/j.rmed.2006.10.015. [PubMed: 17127049]
- [31]. Endeshaw Y, Rice TB, Schwartz AV, et al. Snoring, daytime sleepiness, and incident cardiovascular disease in the health, aging, and body composition study. Sleep 2013; 36 (11):1737–45. doi:10.5665/sleep.3140. [PubMed: 24179308]
- [32]. Bastianelli K, Ledin S, Chen J. Comparing the accuracy of 2 point-of-care lipid testing devices. J Pharm Pract 2016; 30 (5):490–7 2017/10/01. doi:10.1177/0897190016651546. [PubMed: 27313238]
- [33]. Szablowski CJ, Mercer KR, Manalang M, et al. Differences between point-of-care blood glucose analyzers in identifying prediabetes. Diabetes 2018; 67 (1) Supplement2370-PUB. doi:10.2337/ db18-2370-PUB.
- [34]. He J, Gu D, Reynolds K, et al. Serum total and lipoprotein cholesterol levels and awareness, treatment, and control of hypercholesterolemia in China. Circulation 2004; 110 (4):405–11 Jul 27. doi:10.1161/01.CIR.0000136583.52681.0D. [PubMed: 15238453]
- [35]. ADA Understanding A1C: diagnosis. American Diabetes Association; 2021. Accessed 03/06/ https://www.diabetes.org/a1c/diagnosis.
- [36]. Senaratna CV, Perret JL, Matheson MC, et al. Validity of the Berlin questionnaire in detecting obstructive sleep apnea: a systematic review and meta-analysis. Sleep Med Rev 2017; 36 :116–24 2017/12/01/. doi:10.1016/j.smrv.2017.04.001. [PubMed: 28599983]
- [37]. Peker Y, Celik Y, Arbatli S, et al. Effect of high-risk obstructive sleep apnea on clinical outcomes in adults with coronavirus disease 2019: a multicenter, prospective, observational cohort study. Ann Am Thorac Soc 2021:2325–6621 (Electronic). doi:10.1513/AnnalsATS.202011-1409OC.
- [38]. World Health Organization Expert ConsultationAppropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet 2004; 363 (9403):157–63 Jan 10. doi:10.1016/s0140-6736(03)15268-3. [PubMed: 14726171]

- [39]. Lim JU, Lee JH, Kim JS, et al. Comparison of world health organization and asia-pacific body mass index classifications in COPD patients. Int J Chronic Obstr Pulm Dis 2017; 12 :2465–75. doi:10.2147/COPD.S141295.
- [40]. Gottlieb DJ, Yenokyan G, Newman AB, et al. Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: the sleep heart health study. Circulation 2010; 122 (4):352–60 Jul 27. doi:10.1161/CIRCULATION-AHA.109.901801. [PubMed: 20625114]
- [41]. Won CHJ, Reid M, Sofer T, et al. Sex differences in obstructive sleep apnea phenotypes, the multi-ethnic study of atherosclerosis. Sleep 2020; 43 (5) May 12. doi:10.1093/sleep/zsz274.
- [42]. Azarbarzin A, Sands SA, Stone KL, et al. The hypoxic burden of sleep apnoea predicts cardiovascular disease-related mortality: the osteoporotic fractures in men study and the sleep heart health study. Eur Heart J 2019; 40 (14):1149–57 Apr 7. doi: 10.1093/eurheartj/ehy624. [PubMed: 30376054]
- [43]. Kheirandish-Gozal L, Gozal D. Obstructive sleep apnea and inflammation: proof of concept based on two illustrative cytokines. Int J Mol Sci 2019; 20 (3):459
- [44]. Vgontzas AN, Papanicolaou DA, Bixler EO, et al. Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. J Clin Endocrinol Metab 2000; 85 (3):1151–8. doi:10.1210/jcem.85.3.6484. [PubMed: 10720054]
- [45]. Mazzotti DR, Keenan BT, Lim DC, Gottlieb DJ, Kim J, Pack AI. Symptom subtypes of obstructive sleep apnea predict incidence of cardiovascular outcomes. Am J Respir Crit Care Med 2019; 200 (4):493–506 2019/08/15. doi: 10.1164/rccm.201808-1509OC. [PubMed: 30764637]
- [46]. Alimoradi Z, Broström A, Tsang HWH, et al. Sleep problems during COVID-19 pandemic and its' association to psychological distress: a systematic review and meta-analysis. EClinicalMedicine 2021; 36 :100916 Jun. doi:10.1016/j.eclinm.2021.100916.
- [47]. Alimoradi Z, Gozal D, Tsang HWH, et al. Gender-specific estimates of sleep problems during the COVID-19 pandemic: systematic review and meta-analysis. 10.1111/jsr.13432. J Sleep Res 2022; 31 (1):e13432 2022/02/01. doi:10.1111/jsr.13432.
- [48]. Fakhouri THI, Martin CB, Chen TC, et al. An investigation of nonresponse bias and survey location variability in the 2017–2018 national health and nutrition examination survey. Vol.. Vital Health Stat 2020; 2 (185):1–36.
- [49]. Tilley BC, Mainous AG, Amorrortu RP, et al. Using increased trust in medical researchers to increase minority recruitment: the RECRUIT cluster randomized clinical trial. Contemp Clin Trials 2021; 109 :106519 2021/10/01/. doi:10.1016/j.cct.2021.106519.
- [50]. George S, Duran N, Norris K. A systematic review of barriers and facilitators to minority research participation among African Americans, Latinos, Asian Americans, and Pacific Islanders. Am J Public Health 2013; 104 (2):e16–31 2014/02/01. doi:10.2105/ AJPH.2013.301706. [PubMed: 24328648]

Table 1

Characteristics of the study participants (n = 394).

		Sleep apnea risk	nea risk	
	Total	Low-risk	High-risk	<i>P</i> -value
	<i>n</i> = 394 (100.0%)	<i>n</i> = 345 (87.6%)	<i>n</i> = 49 (12.4%)	
Age, mean (SE)	58.37 (0.32)	58.30 (0.34)	58.84 (0.90)	0.580
Gender, n (%)				
Female	208 (52.8)	190 (55.1)	18 (36.7)	0.016
Male	186 (47.2)	155 (44.9)	31 (63.3)	
Asian origin, n (%)				
Chinese	194 (49.2)	174 (50.4)	20 (40.8)	0.208
Korean	200 (50.8)	171 (49.6)	29 (59.2)	
Marital status, n (%)				
Not currently married	58 (14.7)	50 (14.5)	8 (16.3)	0.735
Married/living as married	336 (85.3)	295 (85.5)	41 (83.7)	
Education, n (%)				
Less than high school	42 (10.7)	35 (10.1)	7 (14.3)	0.230
High school graduate or GED	88 (22.3)	78 (22.6)	10 (20.4)	
Business/vocational school/some college	67 (17.0)	57 (16.5)	10 (20.4)	
College graduate	101 (25.6)	85 (24.6)	16 (32.7)	
Attended graduate/professional school	96 (24.4)	90 (26.1)	6 (12.2)	
Household income, n (%)				0.791
<\$20,000	61 (15.5)	51 (14.8)	10 (20.4)	
\$20,000–39,999	61 (15.5)	55 (15.9)	6 (12.2)	
\$40,000–59,999	84 (21.3)	72 (20.9)	12 (24.5)	
\$60,000–79,999	49 (12.4)	42 (12.2)	7 (14.3)	
\$80,000–99,999	32 (8.1)	29 (8.4)	3 (6.1)	
> = \$100,000	107 (27.2)	96 (27.8)	11 (22.5)	
Employment, n (%)				
Working full time	229 (58.1)	200 (58.0)	29 (59.2)	0.683
Working part time	81 (20.6)	73 (21.2)	8 (16.3)	

		Sleep apnea risk	nea risk	
	Total	Low-risk	High-risk	- P-value
	$n = 394 \ (100.0\%)$	<i>n</i> = 345 (87.6%)	<i>n</i> = 49 (12.4%)	
Not currently working	84 (21.3)	72 (20.9)	12 (24.5)	
Health insurance, n (%)				
Private health insurance	240 (60.9)	210 (60.9)	30 (61.2)	0.888
Medicare/Medicaid	73 (18.5)	63 (18.3)	10 (20.4)	
No health insurance	81 (20.6)	72 (20.9)	9 (18.4)	
Total Cholesterol, mean (SE) a	182.9 (2.59)	182.0 (2.49)	188.6 (11.02)	0.400
LDL-C, mean (SE) a	89.7 (2.38)	88.4 (2.12)	98.5 (11.73)	0.155
HDL-C, mean (SE) a	54.0 (0.88)	54.7 (0.94)	49.0 (2.30)	0.029
Total Cholesterol/HDL-C ratio, mean (SE) a	3.6 (0.07)	3.5 (0.06)	4.2 (0.32)	0.001
Triglycerides, mean $(SE)^{\ a}$	197.7 (5.84)	193.1 (5.94)	229.2 (20.65)	0.039
Glucose, mean (SE) ^a	134.3 (2.91)	132.8 (2.90)	144.3 (11.16)	0.187
BMI, mean (SE)	24.6 (0.18)	24.5 (0.19)	25.4 (0.56)	0.110
Number of chronic conditions, mean (SE)	1.27 (0.07)	1.22 (0.07)	1.61 (0.20)	0.052
Hypercholesterolemia, n (%)				
No	205 (52.0)	185 (53.6)	20 (40.8)	0.093
Yes	189 (48.0)	160 (46.4)	29 (59.2)	
Diabetes, n (%)				
No	305 (77.4)	272 (78.8)	33 (67.4)	0.072
Yes	89 (22.6)	73 (21.2)	16 (32.7)	
Obesity, n (%)				
Underweight/Normal	134 (34.0)	120 (34.8)	14 (28.6)	0.433
Overweight	174 (44.2)	153 (44.4)	21 (42.9)	
Obese	86 (21.8)	72 (20.9)	14 (28.6)	

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Table 2

Poisson regression analysis of hypercholesterolemia and diabetes on sleep apnea risk and covariates (n = 394).

		Hypercholesterolemia			Diabetes	
		PR (95% CI)			PR (95% CI)	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Sleep apnea risk						
Low-risk	1.00	1.00	1.00	1.00	1.00	1.00
High-risk	$1.28\left(1.00{-}1.62 ight)^{*}$	$1.25\ (0.96{-}1.62)^{\dagger}$	1.22 (0.95–1.58)	$1.47~(0.94{-}2.31)^{\dagger}$	1.47 (0.92–2.34)	1.44 (0.90–2.31)
Age	$1.03 \left(1.02 {-} 1.05 \right)^{**}$	$1.03 \ (1.01{-}1.05)^{**}$	$1.03 \left(1.01 {-} 1.05 \right)^{**}$	$1.06\left(1.03{-}1.08 ight)^{**}$	$1.06\left(1.02{-}1.09 ight)^{**}$	$1.06(1.02{-}1.09)^{**}$
Gender						
Male	1.00	1.00	1.00	1.00	1.00	1.00
Female	1.12(0.91 - 1.37)	$1.08\ (0.86-1.35)$	1.15(0.91 - 1.45)	0.85 (0.59–1.22)	0.82 (0.55–1.23)	$0.88\ (0.59{-}1.33)$
Asian origin						
Korean		1.00	1.00		1.00	1.00
Chinese		$0.84\ (0.67{-}1.06)$	$0.86\ (0.68{-}1.08)$		$0.68~(0.43{-}1.06)^{\dagger}$	$0.69\ (0.44{-}1.08)$
Marital Status						
Married/cohabit		1.00	1.00		1.00	1.00
Not currently married		1.09 (0.83–1.43)	1.16(0.88 - 1.53)		0.70 (0.39–1.28)	0.76 (0.42–1.36)
Education						
Less than high school		1.18 (0.78–1.78)	1.17 (0.78–1.78)		1.43 (0.64–3.21)	1.41 (0.63–3.16)
High school graduate or GED		1.15 (0.81–1.64)	1.14 (0.80–1.62)		1.36 (0.68–2.71)	1.33 (0.67–2.66)
Business/vocational school/some college		0.96 (0.65–1.41)	0.95 (0.65–1.39)		1.49 (0.75–2.97)	1.46 (0.73–2.93)
College graduate		1.10 (0.79–1.54)	1.12 (0.80–1.56)		1.49 (0.77–2.89)	1.52 (0.79–2.93)
Attended graduate/professional school		1.00	1.00		1.00	1.00
Household income						
<\$20,000		0.78 (0.52–1.15)	$0.76\ (0.51{-}1.14)$		0.73 (0.38–1.42)	$0.73\ (0.38{-}1.41)$
\$20,000–39,999		0.94 (0.66–1.36)	0.92 (0.64–1.33)		0.64(0.33 - 1.24)	0.62 (0.31–1.22)
\$40,000–59,999		0.84 (0.61–1.17)	0.82 (0.59–1.15)		$0.87\ (0.49{-}1.53)$	0.85 (0.48–1.52)
\$60,000–79,999		0.98 (0.67–1.43)	$0.99\ (0.68{-}1.45)$		0.66 (0.32–1.36)	0.67 (0.32–1.39)
\$80,000–99,999		$0.59~(0.35{-}1.01)^{\circ}$	$0.60\ (0.35{-}1.02)^{\dagger}$		1.02 (0.51–2.06)	1.03 (0.51–2.08)

		Hypercholesterolemia	iia		Diabetes	
		PR (95% CI)			PR (95% CI)	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
>=\$100,000		1.00	1.00		1.00	1.00
Employment status						
Working full time		1.00	1.00		1.00	1.00
Working part time		1.07 (0.82–1.41)	1.09 (0.83–1.43)		$1.62 \left(1.04 - 2.54 \right)^{*}$	$1.62 \left(1.04 - 2.51 \right)^{*}$
Not currently working		1.13 (0.85–1.49)	1.11 (0.84–1.47)		1.15 (0.69–1.93)	1.14 (0.68–1.92)
Health Insurance						
Private health insurance		1.00	1.00		1.00	1.00
Medicare/Medicaid		0.90 (0.66–1.23)	0.93 (0.68–1.25)		0.83(0.49 - 1.40)	$0.84\ (0.50{-}1.41)$
No health insurance		0.96 (0.73–1.28)	0.97 (0.73–1.29)		1.00 (0.61–1.64)	1.03 (0.62–1.70)
BMI			$1.04 (1.02 - 1.07)^{**}$			$1.05\ (1.00{-}1.10)^{*}$
$\dot{r} p < 0.1.$						
$* \\ p < 0.05.$						
p < 0.01.						

Model 1: Sleep apnea risk + age, gender. Model 2: Model 1 + Asian origin, marital status, health insurance, education, household income, employment status. Model 3: Model 2 + BMI. *Note:* Both hypercholesterolemia and diabetes are based on three measures: blood measurement, diagnosis from a doctor, and medication usage. PR=prevalence ratio, CI=confidence interval.

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B (S)	10tal Cholesterol LDL-C	LDL-C	HDL-C	Total Cholesterol/HDL ratio Triglycerides	Triglycerides	Glucose
	<i>β</i> (SE)	<i>β</i> (SE)	$\boldsymbol{\beta}(\mathrm{SE})$	<i>β</i> (SE)	<i>β</i> (SE)	β (SE)
Model 1						
Sleep apnea risk (reference: Low-risk)						
High-risk 8.97	8.97 (7.66)	11.33 (7.12)	-3.81 (2.38)	-3.81 (2.38) 0.59 (0.20) **	33.44 (17.45) [†] 9.15 (8.64)	9.15 (8.64)
Model 2						
Sleep apnea risk (reference: Low-risk)						
High-risk 12.5	12.54 (7.70)	$15.00\left(7.18 ight)^{*}$	-3.99 (2.41)	$15.00(7.18)^{*}$ -3.99(2.41) 0.67(0.20) ^{**}	$30.32~(17.85)^{\dagger}$	8.14 (8.83)
Model 3						
Sleep apnea risk (reference: Low-risk)						
High-risk 11.9	11.95 (7.70)	$14.56\left(7.18 ight)^{*}$	-3.60 (2.38)	-3.60 (2.38) 0.64 (0.19) **	28.21 (17.78)	7.32 (8.81)
, p < 0.1.						
* <i>p</i> < 0.05.						
p < 0.01.						

Sleep Epidemiol. Author manuscript; available in PMC 2022 December 01.

Model 2 + BMI. Note: All outcomes are based on blood measurement. SE=standard error. Author Manuscript

Table 4

Linear regression analysis of biomarkers on sleep apnearisk stratified by age, gender, obesity, and number of chronic conditions (n = 328).

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	β (SE)	$\boldsymbol{\beta}(SE)$	$\boldsymbol{\beta}(\mathrm{SE})$	$\boldsymbol{\beta}(\mathrm{SE})$
Stratified by age[1]				
Age<58 ($n = 153$)				
Sleep apnea risk (reference: Low-risk)				
High-risk				77.73 (27.92) ^{**}
Age 58 $(n = 175)$				
Sleep apnea risk (reference: Low-risk)				
High-risk				-14.07 (22.29)
Stratified by gender[2]				
Female $(n = 173)$				
Sleep apnea risk (reference: Low-risk)				
High-risk	-8.51 (10.82)		-0.04 (0.26)	-20.63 (25.87)
Male $(n = 155)$				
Sleep apnea risk (reference: Low-risk)				
High-risk	25.50 (11.32) [*]		$1.07 (0.30)^{**}$	51.56 (25.18) [*]
Stratified by obesity[3]				
Underweight/normal $(n = 111)$				
Sleep apnea risk (reference: Low-risk)				
High-risk	-5.11 (12.13)		-0.10 (0.29)	-33.27 (25.61)
Overweight $(n = 144)$				
Sleep apnea risk (reference: Low-risk)				
High-risk	19.06 (12.91)		$1.36\ (0.30)^{**}$	81.64 (29.23) ^{**}
Obese $(n = 73)$				
Sleep apnea risk (reference: Low-risk)				
High-risk	$45.86\left(18.39 ight)^{*}$		0.70 (0.49)	57.78 (44.70)

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	Total Cholesterol LDL-C	LDL-C	Total Cholesterol/HDL ratio Triglycerides	Triglycerides
	<i>b</i> (SE)	<i>β</i> (SE)	<i>β</i> (SE)	<i>β</i> (SE)
Sleep apnea risk (reference: Low-risk)				
High-risk	-8.05 (8.78)	$-14.61(7.81)^{\dagger}$ 0.35(0.24)	0.35 (0.24)	
>1 (<i>n</i> = 119)				
Sleep apnea risk (reference: Low-risk)				
High-risk	38.28 (14.45) ^{**}	$46.58 (13.58)^{**}$ 1.00 (0.35) ^{**}	$1.00\ (0.35)^{**}$	
t^{\pm} $p < 0.1$.				
p < 0.05.				
p < 0.01.1				

measurement. SE=standard error. Median of age is 58, median of number of chronic conditions is 1. BMI is categorized into 3 groups according to the cut-off points for Asian populations: underweight/ education, household income, employment status, health insurance, BMI.3 Model: Sleep apnea risk + age, gender, Asian origin, marital status, education, household income, employment status, health Model: Sleep apnea risk + gender, Asian origin, marital status, education, household income, employment status, health insurance, BMI.2 Model: Sleep apnea risk + age, Asian origin, marital status, insurance.4 Model: Sleep apnea risk + age, gender, Asian origin, marital status, education, household income, employment status, health insurance, BMI. Note: All outcomes are based on blood normal (< 23 kg/m²), overweight (23–26.9 kg/m²), obese (>=27 kg/m²).

Table 5a

Poisson regression analysis of hypercholesterolemia and diabetes on snoring, stratified by daytime sleepiness (n = 394).

	Hypercholestero	Hypercholesterolemia Sleepiness	Diabetes	Diabetes Sleepiness
	No sleepiness	Positive sleepiness	No sleepiness	Positive sleepiness
	n = 303	n = 91	n = 303	n = 91
	PR (95% CI)	PR (95% CI)	PR (95% CI)	PR (95% CI)
Model 2				
Snoring (reference: No snoring) 1.00	1.00	1.00	1.00	1.00
Positive snoring	$1.46\left(1.15{-}1.84 ight)^{**}$	1.34 (0.90–2.01)	0.74 (0.45–1.23)	$2.06\left(1.08{-}3.93 ight)^{*}$
Model 3				
Snoring (reference: No snoring) 1.00	1.00	1.00	1.00	1.00
Positive snoring	$1.39 \left(1.09 {-}1.79\right)^{**}$	$1.29\ (0.86 - 1.93)$	$0.65~(0.39{-}1.08)^{\not \tau}$	$0.65 (0.39 - 1.08)^{\dagger}$ 2.05 (1.07 - 3.91) [*]
*				
p < 0.1.				
$_{P < 0.05.}^{*}$				
p < 0.01.				
Model 2: Snoring + age, gender, Asian origin, marital status, health insurance, education, household income, employment status. Model 3: Model 2 + BMI. Note: Both hypercholesterolemia and diabetes are based on three measures: blood measurement, diagnosis from a doctor, and medication usage. PR=prevalence ratio, CI=confidence interval.	sian origin, marital stat I measurement, diagnos	us, health insurance, ec sis from a doctor, and n	lucation, household i redication usage. PR-	ncome, employment stat =prevalence ratio, CI=co
4 odel 2: Snoring + age, gender, As re based on three measures: blood	sian origin, marital stat 1 measurement, diagnos	us, health insurance, eo sis from a doctor, and n	lucation, household i hedication usage. PR=	ncome, emi =prevalence

Table 5b

Linear regression analysis of biomarkers and snoring, stratified by day-time sleepiness (n = 328).

	Sle	epiness
	No sleepiness	Positive sleepiness
	<i>n</i> = 251	<i>n</i> = 77
	B (SE)	B (SE)
Total Cholesterol		
Model 2		
Snoring (reference: No snoring)		
Positive snoring	-0.07 (5.79)	23.63 (15.53)
Model 3		
Snoring (reference: No snoring)		
Positive snoring	-0.57 (6.08)	17.98 (15.72)
LDL-C		
Model 2		
Snoring (reference: No snoring)		
Positive snoring	3.20 (5.00)	29.27 (15.66) [†]
Model 3		
Snoring (reference: No snoring)		
Positive snoring	2.52 (5.25)	27.05 (16.15) [†]
HDL-C		
Model 2		
Snoring (reference: No snoring)		
Positive snoring	-5.28 (2.00) **	-3.47 (3.77)
Model 3		
Snoring (reference: No snoring)		
Positive snoring	-3.64 (2.07) [†]	-3.85 (3.90)
Total Cholesterol/HDL ratio		
Model 2		
Snoring (reference: No snoring)		
Positive snoring	0.34 (0.14)*	0.86 (0.38)*
Model 3	. *	
Snoring (reference: No snoring)		
Positive snoring	0.23 (0.15)	0.76 (0.39) [†]
Triglycerides		× ·/
Model 2		
Snoring (reference: No snoring)		
Positive snoring	13.21 (14.69)	11.36 (28.33)
Model 3		
Snoring (reference: No snoring)		

	Sle	eepiness
	No sleepiness	Positive sleepiness
	<i>n</i> = 251	<i>n</i> = 77
	β (SE)	\$ (SE)
Positive snoring	7.15 (15.36)	0.79 (28.66)
Glucose		
Model 2		
Snoring (reference: No snoring)		
Positive snoring	-2.15 (7.38)	23.09 (14.27)
Model 3		
Snoring (reference: No snoring)		
Positive snoring	-8.00 (7.64)	25.03 (14.73) [†]

 $^{\dagger} p < 0.1.$

**		
$D \leq$	0.01.	

Model 2: Snoring + age, gender, Asian origin, marital status, education, household income, employment status, health insurance Model 3: Model 2 + BMI. *Note:* All outcomes are based on blood measurement. SE=standard error.