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The association of the non-HDL-cholesterol to HDL-cholesterol ratio (NHHR) with obstructive sleep apnea among adults aged ≥ 40 years: results from NHANES 2015–2018

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Abstract

Background The ratio of the non-HDL-cholesterol to HDL- cholesterol (NHHR) is a newly proposed lipid metric. Currently, few studies have explored the relationship between NHHR and obstructive sleep apnea (OSA) among middle-aged and elderly people. This study aims to investigate the potential association between NHHR and OSA.

Methods This study included participants from the NHANES 2015–2018 cycles, focusing exclusively on individuals aged 40 and above. OSA data were estimated based on questionnaire responses. NHHR was estimated as the ratio of non-HDL-C to HDL-C. Multivariable logistic regression, adjusted for covariates, subgroup analysis, and smoothing spline fittings were utilized to assess the link between NHHR and OSA.

Results A total of 5,858 participants were analyzed. Multivariable logistic regression highlighted a statistically significant positive relationship between NHHR and OSA [OR 1.06 (95% CI: 1.02, 1.11), $p = 0.0053$]. As NHHR increased by tertiles, the risk of OSA also showed an upward trend. Subgroup and interaction analyses confirmed the overall association was robust across most confounding factors except for gender and diabetes status. Further nonlinear analyses identified a significant inverted U-shaped curve (p-nonlinearity < 0.05) with peak risk at an NHHR of 5.3. This pattern was particularly evident in males (turning point = 3.97) and diabetics (turning point = 5.61).

Conclusion From 2015 to 2018, among the NHANES population aged over 40, NHHR showed a significant and independent positive association with OSA risk. The consistency of this relationship across various subgroups suggests NHHR's potential as a complementary biomarker for metabolic risk assessment in OSA.

Keywords Non-high density lipoprotein cholesterol, High density lipoprotein cholesterol, Obstructive sleep apnea, Middle-aged, Elderly

Introduction

Obstructive Sleep Apnea (OSA) [1] is a critical public health issue characterized by recurrent interruptions in breathing during sleep due to partial or complete blockages of the upper airway [2–4]. These interruptions disturb sleep patterns and trigger systemic consequences, including oxidative stress and endothelial dysfunction,

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with cascading effects on cardiometabolic health. Contemporary data from the latest Global Sleep Apnea Collaboration meta-analysis estimate OSA prevalence at 9–38% in adults(≥ 18 years) [5], with prevalence escalating to up to 49% in adults aged 40–85 years [6]. Untreated OSA is strongly linked to metabolic dysfunction [7] (e.g., insulin resistance, type 2 diabetes) and cardiovascular diseases (e.g., hypertension [8], stroke [9]). Recent estimates suggest that nearly 1 billion people worldwide have OSA [10], a significant portion of whom require medical intervention. The prevalence of OSA increases with age [11], due to physiological changes related to aging, such as loss of upper airway muscle tone [12], increased parapharyngeal fat deposition [13], and altered sleep regulation [14]. In the United States, over 25 million middle-aged adults are affected, yet underdiagnosis persists due to nonspecific symptoms and low public awareness [10].

Previous research has connected obstructive sleep apnea (OSA) to multiple risk factors such as obesity, alcohol use, and tobacco usage, underscoring its association with dyslipidemia [15]. For instance, animal models have demonstrated that intermittent hypoxia, a common occurrence in OSA, is closely related to dyslipidemia and liver oxidative stress [16]. Research indicates that this condition exacerbates lipid metabolism, reduces lipoprotein clearance, and increases lipid output, underscoring the biochemical link between dyslipidemia and OSA [15, 17]. A recent large-scale study demonstrated significant synergistic effects between diet quality and physical activity on OSA risk [18], underscoring the importance of considering multiple metabolic pathways in OSA development. The NHHR (non-HDL-cholesterol to HDL-cholesterol ratio) is a novel lipid marker that includes lipids associated with both promoting and inhibiting atherosclerosis [19]. Recent evidence confirms NHHR's superiority over conventional lipid ratios in predicting cardiometabolic outcomes, including its dose–response relationship with vascular inflammation [20], making it particularly relevant for OSA pathophysiology. It is used not only for assessing cardiovascular disease risk [21] but also shows promise in predicting and diagnosing non-alcoholic fatty liver disease [15], chronic renal failure [22], glucose intolerance [23], and emotional disorders [24, 25].

Although existing studies have revealed correlations between NHHR and various health conditions [21–23], understanding of its connection to the risk of OSA in middle-aged and older populations remains insufficient. This exploratory study examines the possible link between NHHR and OSA, which may contribute to developing supplementary indicators for OSA risk assessment in older populations. The findings could offer preliminary evidence for future research on early OSA detection, particularly for cases where traditional diagnostic methods present challenges. This study aims to examine the association between NHHR and OSA risk in middle-aged and older adults using a nationally representative sample. It also evaluates the potential of NHHR as a supplementary biomarker for OSA risk stratification, explores variations across demographic subgroups, and provides preliminary evidence to support future personalized interventions.

Methods

Sample population

The samples and data were sourced from the National Health and Nutrition Examination Survey (NHANES), an extensive, layered, multistage probability cross-sectional study designed to evaluate the health and nutritional condition of the non-institutionalized [26]. This study analyzed data from the 2015–2016 and 2017–2018 cycles, covering 19,225 participants. All participants had signed consent forms and obtained authorization from the National Center for Health Statistics (NCHS) Ethics Review Board. This study focused on adults aged ≥40 years by selecting eligible individuals from the NHANES dataset. After excluding participants with incomplete data, the study included 5,858 qualified subjects for the final analysis. Detailed information on the selection process is illustrated in Fig. 1. Additional details about the survey methodologies are available on the NHANES website.

The focus on middle-aged and older adults (≥ 40 years) was based on: (a) OSA prevalence rises sharply after age 40 [27].(b) Alignment with clinical guidelines for lipid screening from age 40 in adults with risk factors [28] and (c) As people age, they experience a natural decrease in muscle tone, along with an increase in fat deposition around the neck and upper airway, which may not be as pronounced in younger individuals [29]. By excluding participants under 40, researchers can focus on the more common demographic and potentially reduce confounding variables.

Assessment of OSA and NHHR

The diagnosis of OSA in this study was based on participants' self-reported affirmative responses to at least one of three key questions. Therefore, all OSA cases in our study population were self-reported. Below are the detailed questionnaire items: (1) whether snoring occurs three or more nights per week; (2) whether there are three or more nights per week of snoring, gasping, or breathing pauses; (3) reporting excessive daytime sleepiness despite sleeping for at least 7 h each night, observed 16–30 times [30]; The frequency of these symptoms was assessed through three dichotomous questions, involving

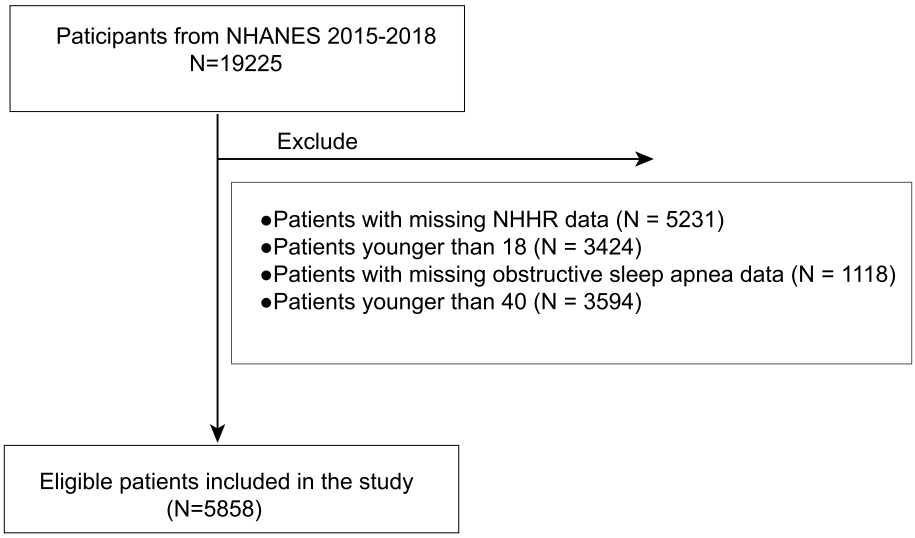

Fig. 1 The selection process of NHANES 2015–2018

snoring, snoring/breathing pauses, and excessive daytime sleepiness. Individuals reporting any of these symptoms three or more times per week or frequent daytime sleepiness were categorized as having OSA symptoms. We employed the methodology outlined in previous studies to calculate NHHR, specifically utilizing the non-HDL-C/HDL-C ratio [24, 25, 31]. NHHR was computed as (Total Cholesterol—High-Density Lipoprotein Cholesterol)/High-Density Lipoprotein Cholesterol, expressed as (TC—HDL-C)/HDL-C, and we analyzed the lipid profiles of fasting individuals.

Selection of covariates

In this analysis, a variety of factors were taken into account as covariates, such as gender (male/female), age (years), ethnicity (Mexican American/other Hispanic/non-Hispanic white/non-Hispanic black/other), levels of physical activity (none/moderate/intense/moderate and intense), education level (below high school/high school or above), poverty income ratio (PIR), body mass index (BMI, kg/m2), smoking habits (never/former/current smoker), alcohol use (yes/no), presence of liver disease (yes/no), diabetes (yes/no), heart failure (yes/no), history of stroke (yes/no), and hypertension (yes/no).

Statistical analysis

Analyses were performed using R software (version 4.3.3) and EmpowerStats (version 4.1) for statistical evaluation to determine the connection between NHHR and OSA. Continuous variables were displayed as means and standard deviations (SD), while categorical variables were represented as percentages.Initially, t-tests were employed to compare mean differences between groups with and without OSA, and Chi-square tests were utilized to assess percentage discrepancies among categorical variables.A multivariate binary logistic regression model was constructed to explore the correlation between NHHR and OSA, utilizing odds ratios (OR) and 95% confidence intervals (CI) to quantify the degree of association. Model 1 did not adjust for any factors. Model 2 adjusted for certain variables,such as age, gender, and race. Model 3 further adjusted for all the variables,including PIR, BMI, physical activity, alcohol status, smoking status, educational level, marital status, hypertension, diabetes, heart failure, liver disease, and stroke.Additionally, subgroup analysis was conducted to evaluate disparities in the correlation between NHHR and OSA among various populations, incorporating variables such as age (under 65 and 65 and over), gender, BMI (under 25, 25 to 30, over 30), race, diabetes, hypertension, educational level, and marital status. Multicollinearity among predictor variables was assessed using variance inflation factors (VIFs). A VIF value > 10 was considered indicative of significant multicollinearity. Variables with high VIFs were considered for removal or transformation to improve model stability. To assess the potential nonlinear association between NHHR and OSA, smooth spline fitting was applied within the logistic regression framework. This method enabled the identification of non-linear trends and potential inflection points in the relationship. Additionally, subgroup analyses were conducted to examine whether the association between NHHR and OSA was consistent across different population groups. Stratified logistic regression models were used, and interaction terms were included to assess potential effect modification. P for interaction were calculated to

determine whether the relationship between NHHR and OSA differed significantly among subgroups. For categorical data, the mode was utilized to fill in missing entries, while the median was applied to complete missing continuous data. All NHANES study protocols were sanctioned by the NCHS Ethics Review Board. Each participant provided written authorization. Moreover, all research methods adhered to relevant industry guidelines and regulatory requirements.

Results

Foundational traits of the study participants

A total of 5,858 individuals were included in this study. The mean age was 60.25 ± 11.66 years, and 50.61% were female. Participants were categorized based on self-reported OSA status: 3,351 with OSA and 2,507 without. The overall prevalence of OSA was 57.2%. Mean NHHR was 2.86 ± 1.42. Compared to participants without OSA, those with OSA had significantly higher BMI (31.00 ± 6.96 vs. 28.45 ± 6.22, $p < 0.001$) and NHHR (2.97 ± 1.40 vs. 2.70 ± 1.43, $p < 0.001$). They also had higher proportions of hypertension (51.63% vs. 44.36%, $p < 0.001$), diabetes (30.77% vs. 24.65%, $p < 0.001$), heart failure (5.55% vs. 4.31%, $p = 0.031$), and liver disease (7.55% vs. 5.35%, $p < 0.001$).Non-Hispanic whites were less prevalent in the OSA group ($p < 0.001$). Additionally, participants with OSA were more likely to be male, to be current or former smokers ($p < 0.001$), and to engage in both moderate and vigorous physical activity ($p < 0.001$). Marital status also differed significantly between groups ($p < 0.001$). Participants with OSA were slightly less likely to have attained education beyond high school ($p = 0.037$). No significant differences were observed in stroke prevalence ($p = 0.217$) or alcohol use status ($p = 0.399$) (Table 1).

Linear relationship between NHHR and OSA

Our results indicate a substantial positive relationship between NHHR and obstructive sleep apnea (Table 2). This association remained statistically meaningful in both the unadjusted model(OR = 1.15; 95% CI, 1.11–1.20; $p < 0.001$) and the partially adjusted model (OR = 1.12; 95% CI, 1.07–1.16; $p < 0.001$). Furthermore, this robust positive relationship continued even after extensive adjustments for all variables in Model 3(OR = 1.06; 95% CI, 1.02–1.11; $p = 0.0053$).For sensitivity analysis, we categorized the continuous variable NHHR into tertiles. Compared to individuals in the lowest NHHR tertile, those in the highest tertile exhibited a 30% increased risk of OSA (OR = 1.30; 95% CI: 1.13–1.50; $p < 0.001$), confirming a statistically significant positive association. In terms of absolute risk, the prevalence of OSA rose from 49.9% in T1 to 63.0% in T3, yielding an absolute risk difference (ARD) of 13.1%. This highlights the clinical relevance of

Table 1 Analysis of variance (ANOVA) tests or Kruskal–Wallis tests were used to analyze differences between continuous variables

Variables	Without OSA	With OSA	*p*-value
N	2507	3351	
AGE	60.67 ± 12.10	59.93 ± 11.32	0.016
BMI	28.45 ± 6.22	31.00 ± 6.96	< 0.001
PIR	2.65 ± 1.53	2.59 ± 1.53	0.108
NHHR	2.70 ± 1.43	2.97 ± 1.40	< 0.001
Gender			< 0.001
Male	1132 (45.15%)	1761 (52.55%)	
Female	1375 (54.85%)	1590 (47.45%)	
RACE			< 0.001
Mexican American	315 (12.56%)	535 (15.97%)	
Other Hispanic	242 (9.65%)	459 (13.70%)	
Non-Hispanic White	930 (37.10%)	1144 (34.14%)	
Non-Hispanic Black	561 (22.38%)	689 (20.56%)	
Other Race	459 (18.31%)	524 (15.64%)	
Marital			< 0.001
Married/Living with partner	1563 (62.35%)	2275 (67.89%)	
Widowed/Divorced/Separated/ Never married	944 (37.65%)	1076 (32.11%)	
Education level			0.037
< High school	276 (11.01%)	418 (12.47%)	
Completed high school	267 (10.65%)	404 (12.06%)	
> High school	1964 (78.34%)	2529 (75.47%)	
Physical activity			< 0.001
Inactive	1559 (62.19%)	1937 (57.80%)	
Moderate	521 (20.78%)	702 (20.95%)	
Vigorous	90 (3.59%)	122 (3.64%)	
Both moderate and vigorous	337 (13.44%)	590 (17.61%)	
Hypertension			< 0.001
YES	1112 (44.36%)	1730 (51.63%)	
NO	1395 (55.64%)	1621 (48.37%)	
Diabetes			< 0.001
YES	618 (24.65%)	1031 (30.77%)	
NO	1889 (75.35%)	2320 (69.23%)	
Heart failure			0.031
YES	108 (4.31%)	186 (5.55%)	
NO	2399 (95.69%)	3165 (94.45%)	
Stroke			0.217
YES	135 (5.38%)	206 (6.15%)	
NO	2372 (94.62%)	3145 (93.85%)	
Liver disease			< 0.001
YES	134 (5.35%)	253 (7.55%)	
NO	2373 (94.65%)	3098 (92.45%)	
Smoke status			< 0.001
Never	1465 (58.44%)	1782 (53.18%)	
Former	663 (26.45%)	975 (29.10%)	
Current	379 (15.12%)	594 (17.73%)	
Alcohol status			0.399
YES	208 (8.30%)	299 (8.92%)	
NO	2299 (91.70%)	3052 (91.08%)	

Categorical variables were compared using chi-square tests

NHHR Non high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio, *BMI* Body mass index, *PIR* Poverty income ratio, *OSA* Obstructive sleep apnea

Table 2 Association of NHHR with obstructive sleep apnea

Exposure	Model 1 OR (95% CI) *p*-value	Model 2 OR (95% CI) *p*-value	Model 3 OR (95% CI) *p*-value
NHHR	1.15 (1.11, 1.20) < 0.0001	1.12 (1.07, 1.16) < 0.0001	1.06 (1.02, 1.11) 0.0053
NHHR Tertile			
Low	Reference	Reference	Reference
Middle	1.37 (1.20, 1.55) < 0.0001	1.31 (1.15, 1.50) < 0.0001	1.17 (1.02, 1.34) 0.0255
High	1.71 (1.50, 1.94) < 0.0001	1.54 (1.35, 1.76) < 0.0001	1.30 (1.13, 1.50) 0.0002

NHHR in identifying individuals at higher risk for OSA (Table 3).

Multicollinearity was assessed using variance inflation factors (VIFs), and no significant collinearity was detected, with all VIF values below 10.

Nonlinear relationship between NHHR and OSA

We investigated the nonlinear relationship between NHHR and OSA using a smoothing curve fitting method. The results showed an inverted U-shaped relationship between NHHR and OSA among middle-aged and elderly Americans, with a turning point at an NHHR value of 5.3. The *p*-value for non-linearity was 0.002, indicating a statistically significant non-linear relationship. The related results and their visual representations are shown in Fig. 2 and Table 4. Further stratified analysis revealed that this inverted U-shaped relationship was also significant in subgroups with diabetes and in male subgroups, with turning points at NHHR values of 5.61 and 3.97 (Fig. 3), respectively. These findings suggest significant variability in the association between NHHR and OSA across different health conditions and gender backgrounds, providing important reference information for the design of future clinical interventions and management strategies.

Subgroup analysis

The results of subgroup analyses showed that, aside from gender and diabetes status, other demographic characteristics did not significantly influence the relationship between NHHR and OSA. As illustrated in Fig. 4, the positive correlation between NHHR and OSA remained consistent across subgroups defined by age, ethnicity, hypertension status, educational level, marital status, physical activity, and smoking status, except for gender and diabetes status, which showed significant interactions (all other interaction *p*-values > 0.05). This indicates that the association between NHHR and OSA is generally consistent across most of the examined population levels, while gender and diabetes status may be key factors affecting this relationship.

Table 3 Absolute risk differences in OSA prevalence across NHHR tertiles

NHHR Tertile	OSA Cases/Total	OSA Prevalence (%)	Absolute Risk Difference (vs T1)
T1 (Low)	887/1778	49.9%	Reference
T2 (Middle)	1137/1973	57.6%	+ 7.7%
T3 (High)	1327/2107	63.0%	+ 13.1%

Discussion

This study examined the correlation between NHHR and OSA in the middle-aged and elderly population,utilizing data from a nationwide survey of the U.S. population from 2015 to 2018. Our findings reveal a consistent positive link between NHHR and the occurrence of OSA. This relationship remained stable even when adjusting for factors like age, race, hypertension, level of education, marital status, physical activity, and smoking habits. Specifically, for each unit increase in NHHR, the likelihood of middle-aged and older adults developing OSA increases by 6%, a statistically significant result. The analysis utilizing smoothing curve fitting and saturation threshold effects revealed an inverted U-shaped relationship between NHHR and OSA, with a notable inflection point at NHHR 5.3. Subgroup and interaction analyses indicate that this correlation holds steady across various subpopulations.

Recent research suggests that NHHR is a comprehensive lipid ratio reflecting both pro-atherogenic and anti-atherogenic lipids [19, 24, 32]. Moreover, NHHR surpasses traditional lipid markers in assessing non-alcoholic fatty liver disease (NAFLD) and disease severity related to diabetes [33]. While there are few studies directly linking NHHR with OSA, the relationship between lipid abnormalities and OSA has been extensively studied. For instance, research by Koseoglu et al.found that the monocyte to HDL ratio could indicate the severity of OSA and its associated cardiovascular risks [34]. Additionally, a recent study by Guscoth et al. pointed out a correlation between triglycerides and the

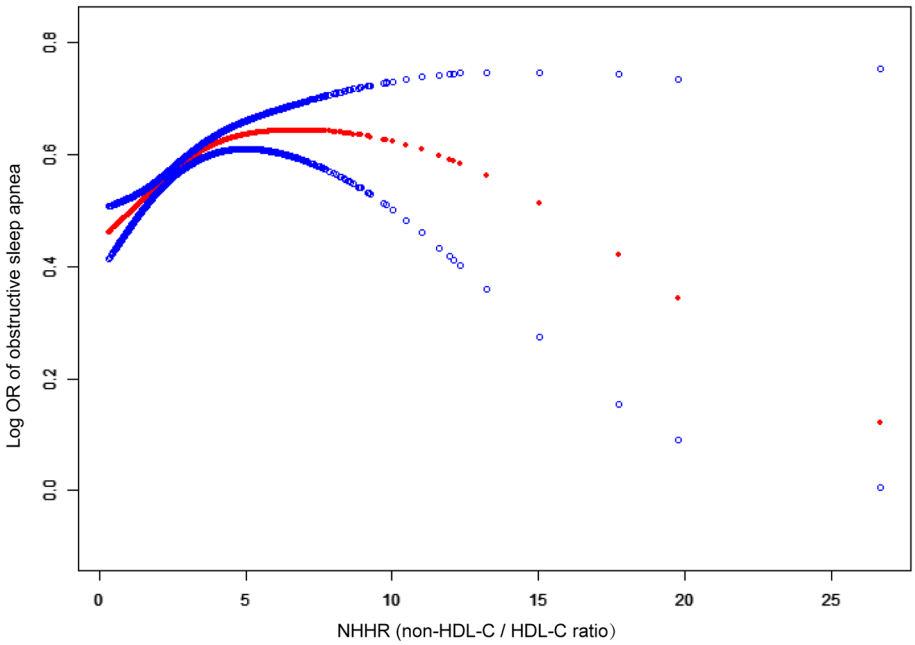

Fig. 2 The relationship analysis between NHHR and OSA among middle-aged and elderly people. The red solid line represents the log odds ratio [log(OR)] of OSA, blue shaded region represents 95% CI. NHHR: Non high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio, OSA: obstructive sleep apnea

Table 4 The threshold effect of NHHR on OSA among the Middle-aged and elderly people was analyzed using a two-part linear regression model

Outcome	Obstructive Sleep Apnea
Fitting by standard linear model	
OR (95% CI)	1.1 (1.0, 1.1)
p-value	0.005
Fitting by two-piecewise linear model	
5.3	
OR1(< K)	1.1 (1.1, 1.2) < 0.001
OR2(> K)	0.9 (0.8, 1.0) 0.058
Logarithmic likelihood ratio test P-value	0.002

Age, gender, race, marital, education level, BMI, income-to-poverty ratio, alcohol status,smoking status, diabetes, hypertension, physical activity,heart failure,stroke and liver disease were adjusted. 95% CI, 95% confidence interval; OR, odds ratio

severity of OSA, importantly, this relationship holds even in patients with normal waist circumference, challenging the traditional view that associates high BMI exclusively with elevated OSA risk [35]. Studies have shown that there is a significant association between high-density lipoprotein (HDL) levels and the severity of OSA, with patients exhibiting lower HDL levels having higher apnea–hypopnea indexes (AHI) [16, 36].

These insights highlight the importance of considering a wider range of biomarkers, such as NHHR, in the clinical assessment and management of OSA, especially given the potential for these markers to reflect broader metabolic and cardiovascular risks associated with this condition. Multiple studies have provided insights into the relationship between OSA and lipid metabolism abnormalities. OSA has been associated with the development of atherosclerosis through intermittent hypoxia, oxidative stress, endothelial dysfunction, heightened sympathetic nervous activity, and systemic inflammation [37–39]. Recurrent hypoxic episodes in OSA increase the production of reactive oxygen species (ROS), which damage vascular endothelial cells and impair normal vascular function, thereby accelerating atherosclerosis progression [39]. Moreover, OSA has been shown to adversely affect lipid metabolism. It can reduce lipoprotein lipase (LPL) activity—a key enzyme regulated by insulin and inhibited by stress hormones such as cortisol and adrenaline—through increased insulin resistance and sympathetic overactivity [40, 41]. This disruption in lipid regulation promotes abnormal lipid accumulation, elevating non-HDL cholesterol and reducing HDL cholesterol, thus increasing NHHR. These lipid abnormalities, in turn, exacerbate systemic inflammation and oxidative stress, which further impair respiratory muscle function and contribute to fat accumulation around the upper airway. Such changes narrow the airway and increase the likelihood of airway collapse during sleep, perpetuating a vicious cycle of worsening OSA and lipid dysregulation [42].

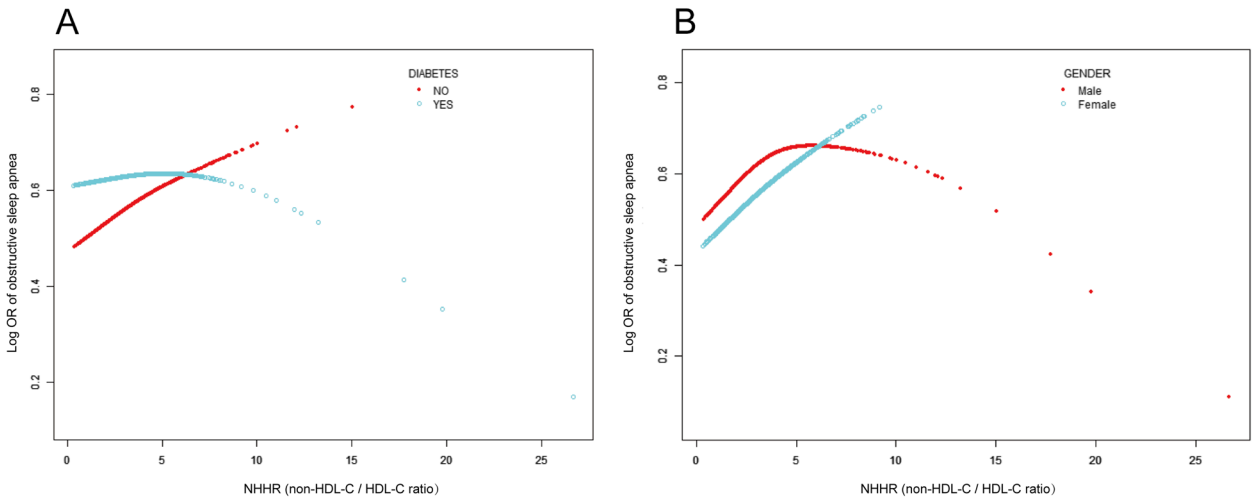

Fig. 3 **A** The relationship analysis between NHHR and OSA is stratified by diabetes status. **B** The relationship analysis between NHHR and OSA is stratified by gender

Compared to individual lipid components, NHHR has shown greater predictive power for cardiovascular disease and has also been validated in relation to OSA. This aligns with findings from Kollar et al. [43], who observed increased levels of atherogenic lipids and decreased levels of protective lipids in OSA patients. Thus, monitoring changes in NHHR levels could be beneficial for the prevention and treatment of OSA. The initial linear model of our study demonstrated a consistent positive correlation between NHHR and OSA. While NHHR is positively associated with OSA risk at lower and moderate levels of NHHR, this relationship changes at higher NHHR values. More sophisticated nonlinear analyses revealed a significant threshold effect—the positive association between NHHR and OSA remained strong until reaching a specific critical value (5.3), after which the relationship plateaued and and slightly declined. Our observed threshold effect aligns with emerging reports of nonlinear lipid-OSA associations [44]. The subsequent risk decline could hypothetically reflect inflammatory pathway exhaustion at extreme dyslipidemia levels [45], though this speculative mechanism requires experimental validation.

In summary, while the linear model provides a useful overview of the general trend, the nonlinear model captures more nuanced variations at specific ranges of NHHR. Both models offer complementary insights. These findings suggest that maintaining NHHR levels below 5.3 may represent an potential strategy for OSA risk reduction.

Interestingly, our findings indicate that gender and diabetes status significantly affect the relationship between NHHR and OSA. Several studies report that being male and having impaired glucose tolerance are risk factors for OSA, with men being more susceptible to OSA partly due to typically larger neck circumferences and higher central obesity, which increase the risk of airway collapse [46, 47]. The incidence of OSA in diabetic patients is notably higher than in the general population, related to chronic inflammation and enhanced sympathetic nervous system activity common among diabetics [47–49]. These abnormalities in inflammation and neural activity can increase OSA risk by affecting the function of neck and respiratory muscles [48, 50]. Moreover, OSA itself might exacerbate diabetes through potential effects on sleep quality and worsening insulin resistance. Through stratified analysis by gender and diabetes status, we observed the same inverted U-shaped relationship between NHHR and OSA. This finding suggests that maintaining lipid levels within an optimal range may help reduce OSA risk in high-risk groups, particularly men and individuals with diabetes. It underscores the need for personalized treatment strategies and targeted prevention. From a public health perspective, incorporating NHHR screening into routine assessments—especially in settings with limited access to polysomnography—may facilitate early risk identification, reduce the burden of undiagnosed OSA, and enable more efficient, risk-based allocation of healthcare resources. If validated by prospective studies, NHHR could serve as a valuable supplementary tool for OSA detection in such contexts.

This study has certain limitations. As an illustration,we utilized NHANES data from 2015 to 2018; therefore, its results may be limited by the timing of data collection, and correlations may change over time. Moreover, as a cross-sectional study, it cannot establish causality but only indicates correlations between variables. Despite attempts to

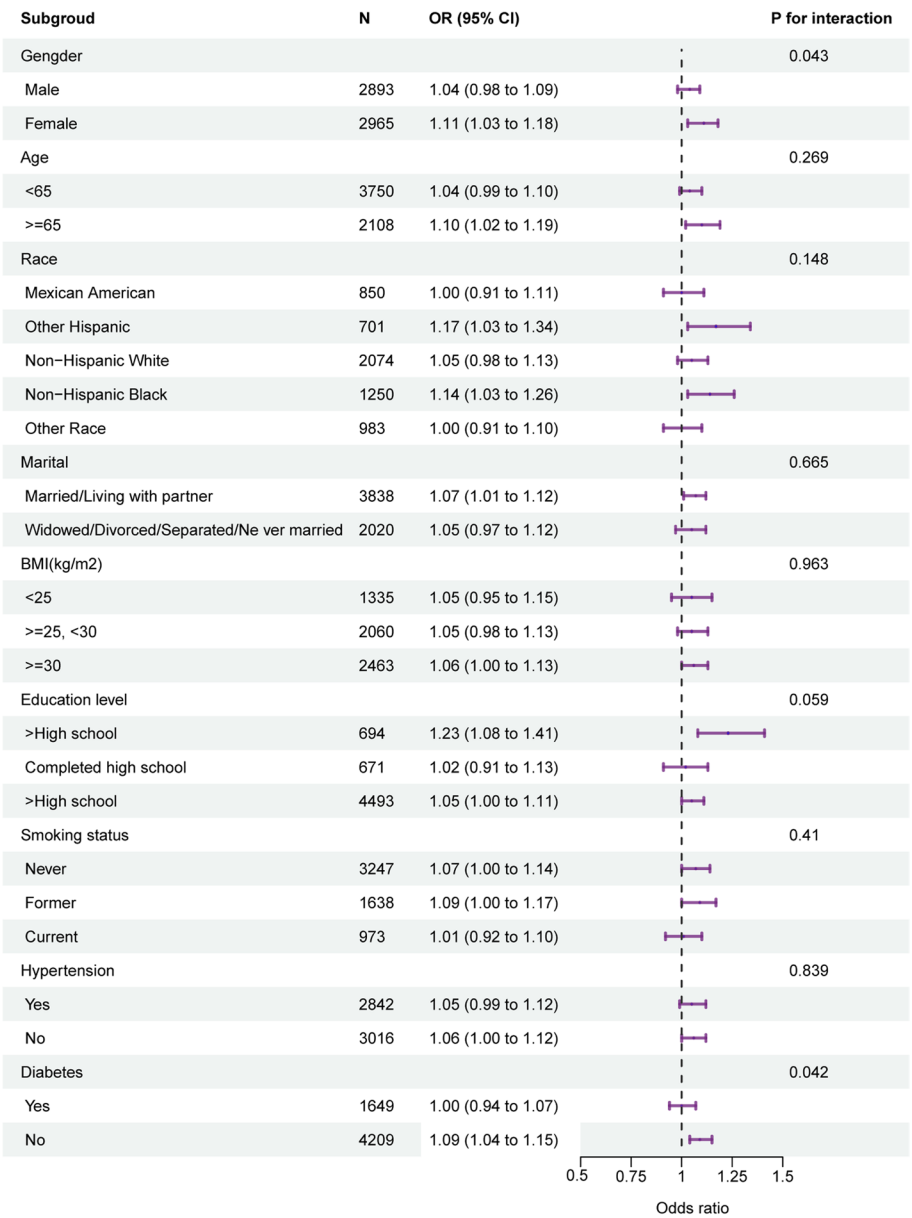

Fig. 4 Subgroup analysis for the relationship between NHHR and OSA among middle-aged and elderly people. Analyses were adjusted for all presented covariates except the effect modifier

control for several confounders, the inherent limitations of a cross-sectional study may not allow for comprehensive control of these factors. In addition, OSA status in this study was based on self-report rather than objective diagnostic tools such as polysomnography, which may lead to misclassification bias and affect the accuracy of OSA prevalence estimates. Future research should involve larger samples and longer durations, possibly using prospective or cohort study designs, to further validate and deepen the understanding of the relationship between NHHR and OSA. This will help more accurately assess the linkage between lipid levels and OSA risk and provide stronger evidence to support clinical interventions.

Conclusion

This study investigated the association between NHHR and OSA among adults aged 40 years and older using data from the NHANES 2015–2018 cycles. The results demonstrated a significant and independent positive association between NHHR and the risk of OSA. Nonlinear analysis further revealed an inverted U-shaped relationship, with OSA risk increasing with NHHR up to

an inflection point at 5.3, after which the risk plateaued and showed a slight decline. This pattern was consistent across key subgroups, particularly among males and individuals with diabetes, where similar turning points were observed. These findings indicate a statistically significant association between NHHR and OSA risk and highlight the need for further investigation into NHHR as a potential biomarker for identifying individuals at an elevated risk of OSA, especially within specific metabolic ranges. However, its clinical applicability remains to be validated in prospective longitudinal studies.

Abbreviations

AHI	Higher apnea–hypopnea indexes
BMI	Body mass index
HDL	High-density lipoprotein
NAFLD	Non-alcoholic fatty liver disease
NCHS	The National Center for Health Statistics
NHANES	The National Health and Nutrition Examination Survey
NHHR	Non-HDL-cholesterol to HDL- cholesterol ratio
OSA	Obstructive Sleep Apnea
PIR	Poverty income ratio
TC	Total cholesterol

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-23274-2.

Supplementary Material 1.

Supplementary Material 2.

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Not applicable.

Authors' contributions
CYC, CLA, CSK and CJM contributed to the collection of records, data analysis, CYC and CLA drafted the manuscript. All authors read and approved the final manuscript.

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Data availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study analyzed secondary data in the NHANES database. The original NHANES study was approved by the National Center for Health Statistics (NCHS) Research Ethics Review Board, with all participants providing written informed consent. As this analysis utilized exclusively anonymous existing data, no additional ethics approval was required.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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