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Association between neutrophil to high-density lipoprotein cholesterol ratio and hearing loss: a cross-sectional study from NHANES

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Abstract

Background This study aimed to investigate the relationship between the neutrophil-to-HDL cholesterol ratio (NHR) and the risk of hearing loss, as well as to evaluate the potential of the NHR as a biomarker for hearing loss.

Methods The U.S. National Health and Nutrition Examination Survey (NHANES) data covering 2005–2012 and 2015–2020 were analyzed. A weighted multivariate logistic regression model assessed the correlation between NHR and speech-frequency hearing loss (SFHL) and high-frequency hearing loss (HFHL). Restricted cubic spline (RCS) regression analysis was utilized to investigate the nonlinear correlation. Additionally, subgroup analysis was performed to identify differences among subgroups. A receiver operating characteristic (ROC) analysis was conducted to evaluate the efficacy of NHR in predicting hearing loss.

Results A total of 10,436 participants were involved. After comprehensive adjustments for confounding factors, NHR was linearly correlated with SFHL and HFHL. Subgroup analysis revealed that race and the poverty index ratio (PIR) significantly modified the association between NHR and hearing loss. ROC analysis demonstrated the predictive capability of NHR for hearing loss.

Conclusion NHR is positively correlated with the risk of hearing loss. This study suggests that NHR may serve as a potential biomarker for predicting and assessing hearing loss, demonstrating significant clinical application value. However, this cross-sectional study limits the ability to establish causality. Future longitudinal studies are needed to confirm these findings and explore potential mechanisms.

Keywords Speech-frequency hearing loss, High-frequency hearing loss, neutrophil-to-HDL cholesterol ratio, NHANES

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Background

Hearing loss is a significant public health concern. According to the World Health Organization (WHO), more than 1.5 billion individuals are affected by hearing loss, of whom approximately 430 million worldwide experience moderate or higher levels of hearing loss [1]. Hearing loss is not merely a sensory impairment that ranks as the fifth leading cause of disability worldwide and is linked to various comorbidities, including cognitive decline, social isolation, and increased medical expenses [2]. In 2017, the WHO estimated that unaddressed hearing loss incurred an annual cost of approximately $750 billion globally, underscoring its significant economic burden [3]. Hearing loss can be classified into three categories: conductive, sensorineural, and mixed. Conductive hearing loss is generally more amenable to improvement, whereas sensorineural hearing loss currently lacks effective medication approved by the FDA [4]. As the population ages, the incidence is anticipated to increase further [1]. Consequently, the development of effective interventions for prevention and treatment is essential to address this escalating global health challenge.

The risk factors for hearing loss are diverse, including demographic characteristics, health conditions, environmental exposures, and lifestyle choices. Age and sex are significant demographic determinants, as the prevalence of hearing loss increases with age, and men are at a higher risk than women [5]. Risk factors for cardiovascular diseases, such as obesity, hypertension, and diabetes are significantly associated with hearing impairment [6]. Lifestyle factors such as noise exposure, smoking, and poor dietary habits can adversely affect hearing health [5, 7]. Furthermore, individuals with a family history of hearing impairment are at a greater risk of developing hearing loss, indicating a potential genetic influence [8]. In recent years, dyslipidemia, chronic low-grade inflammation, and oxidative stress have emerged as contributing factors to hearing loss [10–12].

Previous studies have demonstrated associations between inflammatory markers and hearing impairment. Cross-sectional studies reported correlations of white blood cell count (WBCC), neutrophil count, interleukin-6 (IL-6), and C-reactive protein (CRP) with hearing loss. Large longitudinal cohorts, including studies by Lassale C et al. and Nash SD et al., further confirmed that systemic inflammation, reflected by WBCC and CRP, predicts hearing decline in the elderly [12, 13]. Additionally, dyslipidemia-related impaired cochlear blood flow may contribute to hearing loss [14]. Therefore, we aimed to identify composite indicators that capture the combined impact of inflammation and dyslipidemia on hearing.

It was reported that the NHR has emerged as a potential cost-effective biomarker of systemic inflammation [15, 16], which has been proven associated with cardiovascular disease, metabolic syndrome, and certain cancers [18–20]. These findings indicate that NHR possesses considerable predictive validity for various systemic diseases. The potential impact of inflammation and dyslipidemia on hearing loss prompted us to investigate whether NHR could serve as a biomarker.

This research aimed to explore the utility of the NHR as a potential biomarker for hearing loss. We focused our study on the hypothesis that an elevated NHR is associated with an increased likelihood of developing hearing loss. Rigorous statistical approaches were used to analyze cross-sectional data collected from the NHANES during 2005–2012 and 2012–2020 to evaluate this hypothesis and account for potential confounding factors.

Methods

Study population

Data were collected from the NHANES which utilizes intricate multistage probability sampling techniques and reflects the health of the U.S. population. Using a cross-sectional study approach, we analyzed NHANES data from 2005 to 2012 and 2015–2020, focusing on participants with complete NHR and hearing information to investigate the association between them. Of the original 75,575 participants, they were excluded for following conditions: (1) were younger than 20 years old or pregnant ($N = 33,093$), (2) were missing binaural speech frequencies (0.5, 1, 2, and 4 kHz) and binaural pure tone average (PTA) data at high frequencies (3, 4, 6, and 8 kHz) ($N = 31,413$), or (3) lacked NHR data ($N = 663$). Thus, the final cohort analyzed consisted of 10,436 individuals (Fig. 1).

Data assessment

The neutrophil count (10^3 cells/μL) is divided by the HDL-C value (mmol/L). Two metrics were obtained from laboratory tests, to derive the NHR. These test results adhered to procedure manuals specified in the NHANES, thereby ensuring the accuracy and reproducibility of measurement results.

Audiometry in NHANES employed pure-tone air conduction audiometry by trained examiners in a calibrated soundproof room using an audiometer equipped with standard earphones and inserted earphones. Participants' hearing thresholds were assessed at frequencies 0.5, 1, 2, 3, 4, 6, and 8 kHz, with intensities ranging from − 10 to 120 dB. Each ear was examined twice at 1 kHz. If the difference between the two measurement results exceeded 10 dB, the result was excluded. All subjects' left and right pure tone average (PTA) results were compared, and the ear with better hearing was designated the hearing

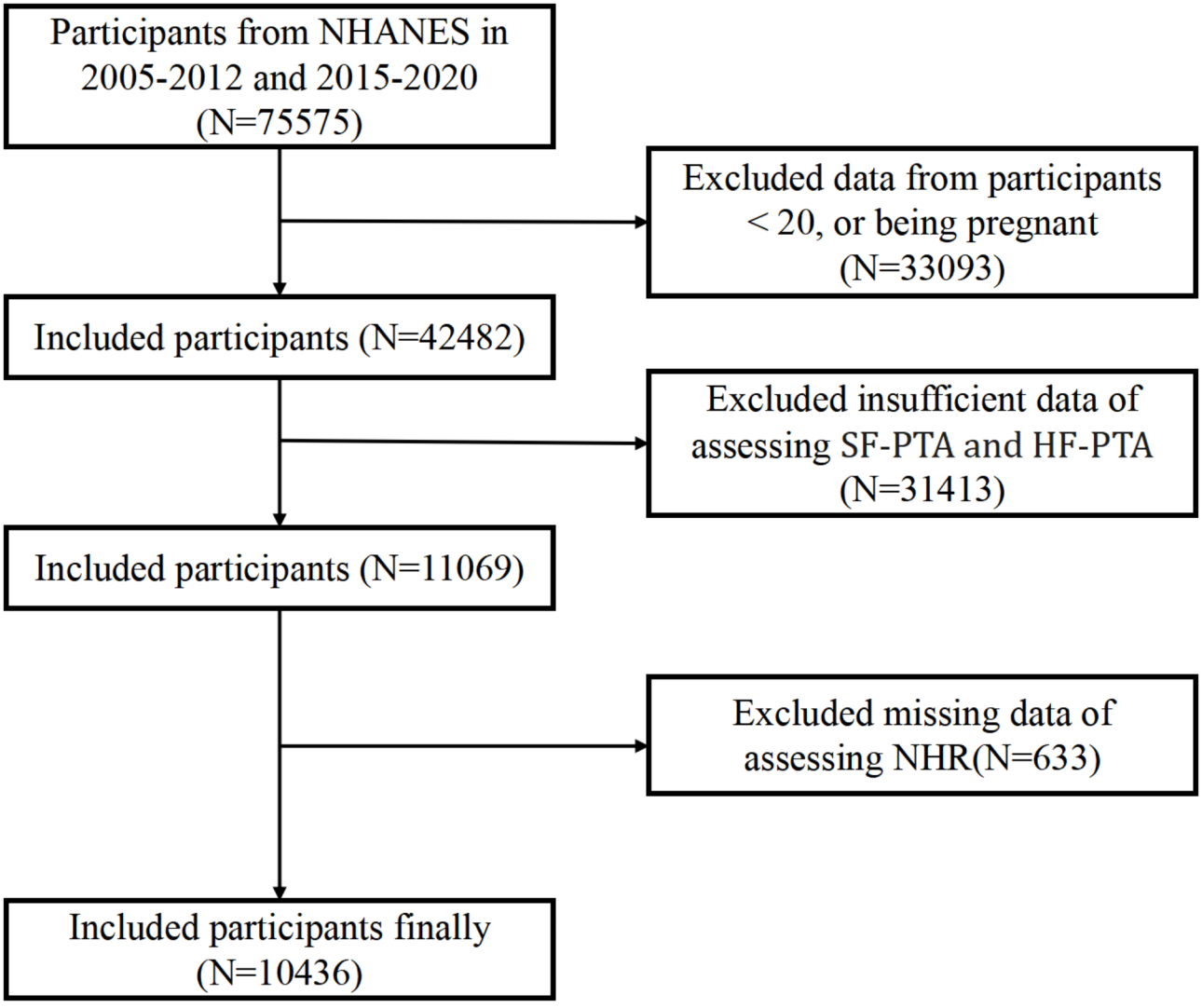

Fig. 1 Inclusion and exclusion criteria for participants in this study

index. According to the WHO's report [1], hearing loss is defined as PTA ≥ 20 dB. Therefore, PTA values of ≥ 20 dB for speech frequencies (0.5, 1, 2, and 4 kHz) are classified as speech-frequency hearing loss (SFHL), while those for high frequencies (3, 4, 6, and 8 kHz) are categorized as high-frequency hearing loss (HFHL) [20]. Audiometric testing in NHANES was conducted in sound-isolated booths using standardized protocols consistent with guidelines from the American Speech-Language-Hearing Association. The audiometers were regularly calibrated to meet ANSI S3.6 specifications for audiometric equipment. Quality control procedures included daily biological checks and regular monitoring of equipment function to ensure the accuracy and reliability of hearing threshold measurements [21, 22].

Covariates

Covariates included sociodemographic data, such as age, sex, race, educational level, poverty income ratio (PIR), and marital status. Lower socioeconomic status is associated with higher systemic inflammation, making PIR a relevant factor in assessing the relationship between NHR and hearing loss [23]. Additionally, the covariates included risk factors for hearing loss, such as body mass index (BMI), smoking habits, alcohol consumption, and noise exposure. The classification of all covariates can be found in Additional file 1 [24]. Notably, an individual's noise exposure (yes or no) was determined by whether they were exposed to noise during work, after-work activities, or when using firearms.

Statistical analysis

R software (V4.3.1) was used to perform the statistical analyses. P-values < 0.05 (two-tailed) were defined as

statistically significant. For data with missing covariates, multiple imputation was performed using the "MICE" package. In descriptive data for baseline characteristics, categorical variables were presented as frequencies (n) and weighted percentages, and continuous variables were expressed as weighted means accompanied by standard deviations. The t-test and chi-square test were used to evaluate differences between diseased and non-diseased. Multiple weighted logistic regression examined the relationship between NHR and hearing loss. Three models were constructed based on adjustment for confounders: model 1 (unadjusted for any covariates), model 2 (adjusted for sociodemographic characteristics), and model 3 (adjusted for all covariates). Continuous variable NHR was divided into four quartiles to create categorical variables and calculated p-values for trend. Subsequently, RCS regression was utilized to illustrate the nonlinear relationship. In addition, we executed interaction tests and subgroup analyses for various covariates. Finally, we used the ROC curve to determine AUC values to evaluate NHR as a predictor of hearing loss. The Youden index (sensitivity + specificity − 1) was used method to determine the best cutoff. This assessment illustrated the balance between sensitivity and specificity of the model at different NHR thresholds. Values of AUC close to 1 indicated a higher predictive accuracy of the model.

Results

Baseline characteristics

A total of 10,436 individuals were included, with outcomes categorized into the SFHL and HFHL groups (Additional file 1). Among them, 3,018 (28.9%) individuals were diagnosed with SFHL, while 5,227 (50.1%) were diagnosed with HFHL. Notably, there were no significant differences in smoking habits between patients with two types of hearing loss and those without. However, all other covariates exhibited significant differences between hearing loss and normal groups. Patients with SFHL or HFHL were generally older and tended to be male, non-Hispanic white, and widowed/divorced/separated. They were inclined to lower levels of education, lower household incomes, and a personal history of smoking and alcohol consumption. In terms of noise exposure, individuals with a history of it were more likely to experience HFHL. In particular, participants with both types of hearing loss exhibited higher neutrophil counts, while levels of HDL-C did not differ significantly between hearing loss and normal participants. Furthermore, participants with hearing loss generally demonstrated higher NHR, which was particularly significant among those with SFHL.

The relationship between NHR and hearing loss

The weighted multiple logistic regression indicated that NHR was associated with SFHL and HFHL (Table 1). In Model 3, NHR was positively correlated with both SFHL and HFHL, with ORs of 1.06 (95% CI: 1.01 to 1.11) for both, $p = 0.017$ and $p = 0.021$, respectively. The positive associations observed between the Q2 and Q3 of NHR and SFHL did not demonstrate statistical significance compared to the Q1 (Q2: OR = 1.02, 95% CI: 0.83 to 1.24; Q3: OR = 1.26, 95% CI: 0.99 to 1.61). However, the Q4 was significantly positively related to the risk of SFHL, with an OR of 1.48 (95% CI: 1.18 to 1.86). Regarding the HFHL, the positive correlations observed in the Q2 and Q3 of NHR did not reach statistical significance (Q2:

Table 1 The associations of NHR with SFHL and HFHL in weighted logistic regression models

	Model 1		Model 2		Model 3	
	OR (95% CI)	***P***	**OR (95% CI)**	***P***	**OR (95% CI)**	***P***
SFHL						
Continuous	1.04 (1.01, 1.06)	**0.004**	1.13 (1.07, 1.19)	**< 0.001**	1.06 (1.01, 1.11)	**0.017**
Q1	Reference				Reference	
Q2	1.03 (0.89, 1.20)	0.682	1.06 (0.88, 1.28)	0.541	1.02 (0.83, 1.24)	0.873
Q3	1.22 (1.03, 1.44)	**0.020**	1.31 (1.08, 1.60)	**0.008**	1.26 (0.99, 1.61)	0.059
Q4	1.27 (1.10, 1.47)	**0.001**	1.38 (1.13, 1.69)	**0.002**	1.48 (1.18, 1.86)	**0.001**
P for trend	**< 0.001**		**< 0.001**		**< 0.001**	
HFHL						
Continuous	1.04 (1.01, 1.07)	**0.011**	1.10 (1.04, 1.15)	**< 0.001**	1.06 (1.01, 1.11)	**0.021**
Q1	Reference				Reference	
Q2	1.04 (0.90, 1.21)	0.567	1.03 (0.85, 1.25)	0.763	1.00 (0.83, 1.20)	0.998
Q3	1.18 (1.00, 1.39)	**0.045**	1.29 (1.05, 1.59)	**0.018**	1.20 (0.99, 1.45)	0.060
Q4	1.17 (0.99, 1.40)	0.072	1.52 (1.26, 1.84)	**< 0.001**	1.21 (1.00, 1.46)	0.054
P for trend	**0.039**		**0.001**		**0.036**	

Model 1 was not adjusted for any covariate

Model 2 was adjusted for age, gender, race, education, PIR, marital status

Model 3 was adjusted for age, gender, race, education, PIR, marital status, smoking status, drinking status, BMI and noise exposure

OR = 1.00, 95% CI: 0.83 to 1.20; Q3: OR = 1.20, 95% CI: 0.99 to 1.45), and Q4 could be considered as marginally significant (Q4: OR = 1.21, 95% CI: 1.00 to 1.46, $p = 0.054$). Additionally, adjusted models consistently showed an increasing trend in NHR quartiles associated with SFHL and HFHL (p for trend < 0.001, $p = 0.036$, respectively).

Restricted cubic spline regression (RCS) analysis

We used the RCS model to investigate the nonlinear correlation between NHR and hearing loss (Fig. 2). RCS regression analysis did not detect a non-linear association, suggesting that the risk of hearing loss increases linearly with higher NHR levels within the observed range.

Subgroup analysis

Participants were categorized based on all covariates mentioned above. Subgroup analysis and interaction tests were consequently conducted. The results showed a marked correlation between NHR and hearing loss, in speech-frequency subgroups based on race ($P = 0.016$) (Table 2). Among HFHL, race and PIR exhibited statistically significant differences ($P = 0.023$ and $P = 0.041$) (Table 3). Other confounding factors did not show significant differences. This underscores the importance of accounting for these variables in future studies to enhance predictive accuracy.

Receiver operating characteristic (ROC) analysis

The ROC curve illustrated the predictive capability of NHR for SFHL (Fig. 3a) with an area under the curve (AUC) of 0.871. This indicated good predictive performance when NHR was combined with demographic and other clinical variables, consistent with previous weighted multivariate logistic regression results, suggesting a significant association between elevated NHR and SFHL occurrence. The ROC curve for NHR in predicting HFHL showed a higher AUC of 0.906 (Fig. 3b) which suggested that NHR combined with other variables was more effective in predicting the occurrence of HFHL. Additionally, analysis using NHR alone yielded AUC values of 0.533 for predicting SFHL and 0.521 for HFHL, with cutoff values of 2.934 and 2.475, respectively. (Additional file 2). In summary, the ROC curve analysis further validated NHR as a potential biomarker for assessing the risk of hearing loss.

Discussion

In this study, we conducted a range of statistical analyses of NHR as predictors of hearing loss. Weighted multiple logistic regression showed that an increased NHR was significantly related to SFHL and HFHL. Additionally, independent NHR levels demonstrated a negative impact on SFHL after adjusting for key confounding variables. As the NHR quartiles increased, the effect of NHR on HFHL showed marginal significance. RCS regression analysis showed no nonlinear correlation between NHR and hearing loss. Subgroup analyses indicated that factors like race and PIR influenced the correlation. ROC analysis illustrated the trade-off relationship between the model's sensitivity and specificity across various classification thresholds.

Longitudinal cohort studies highlighted the impact of inflammation on hearing loss. Nash SD et al. measured CRP, IL-6, and hearing impairment at multiple time points over 22 years. They found that inflammatory markers at a single time point were not associated with increased hearing loss risk. However, adults under 60 with persistently elevated CRP levels had twice the risk of developing hearing impairment [13]. Another prospective study further demonstrated the association between CRP and hearing loss risk in older adults. Additionally,

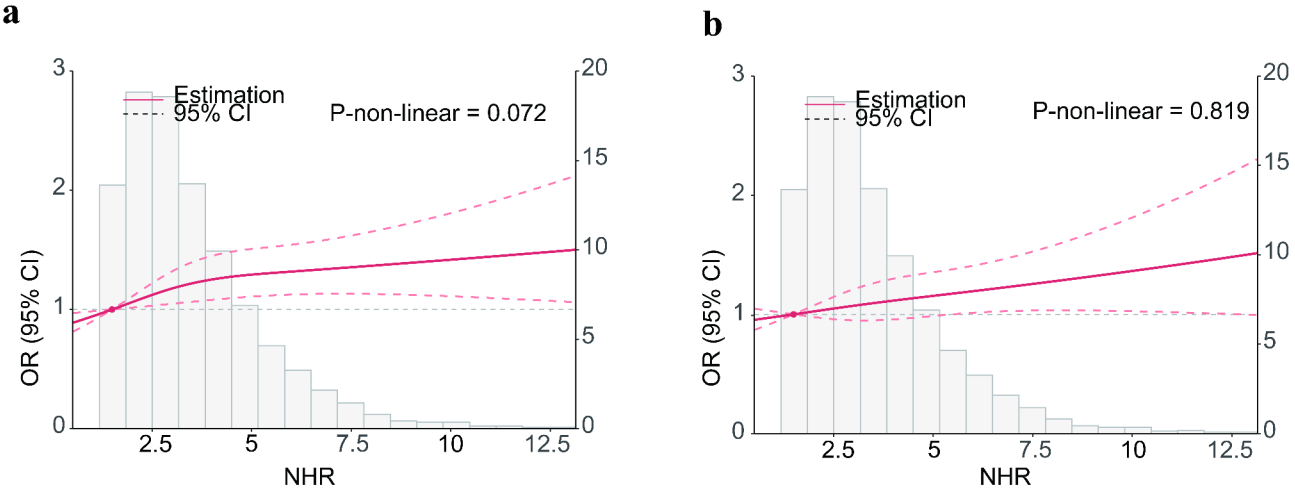

Fig. 2 RCS analysis. (**a**) The nonlinear association between NHR and SFHL. (**b**) The nonlinear association between NHR and HFHL

Table 2 Subgroup analysis for the association between NHR and SFHL with different clinical characteristics

Characteristic	OR(95%CI)	*P* for interaction
Age		0.775
20–39	1.14 (0.98, 1.33)	
40–59	1.05 (0.97, 1.15)	
≥ 60	1.06 (0.99, 1.14)	
Gender		0.37
Male	1.07 (1.00, 1.14)	
Female	1.06 (0.99, 1.13)	
Race		**0.016**
Mexican American	0.98 (0.90, 1.06)	
Other Hispanic	1.18 (1.09, 1.28)	
Non-Hispanic White	1.06 (0.99, 1.13)	
Non-Hispanic Black	1.00 (0.96, 1.03)	
Other Race - Including Multi-Racial	1.22 (1.08, 1.37)	
Education		0.386
Less than high school	1.13 (1.04, 1.23)	
High school grad/GED or equivalent	1.02 (0.93, 1.13)	
Higher than high school	1.05 (0.98, 1.12)	
Marital status		0.564
Married/living with partner	1.07 (0.99, 1.15)	
Widowed/divorced/separated	1.05 (0.97, 1.13)	
Never married	1.07 (0.99, 1.16)	
PIR		0.187
≤ 1.3	1.02 (0.96, 1.09)	
1.3–3.5	1.09 (1.02, 1.16)	
≥ 3	1.19 (1.02, 1.38)	
BMI		0.083
≤ 25 kg/m2	1.13 (1.03, 1.24)	
25–30 kg/m2	1.11 (1.02, 1.22)	
≥ 30 kg/m2	1.01 (0.96, 1.06)	
Smoking status		0.083
Current non smokers	1.08 (1.02, 1.14)	
Current smokers	1.02 (0.94, 1.11)	
Drinking status		0.469
Current non drinkers	1.05 (0.96, 1.15)	
Current moderate drinkers	1.08 (1.02, 1.13)	
Current heavy drinkers	0.98 (0.84, 1.14)	
Noise exposure		0.368
Yes	1.06 (0.99, 1.13)	
No	1.06 (1.00, 1.12)	

Table 3 Subgroup analysis for the association between NHR and HFHL with different clinical characteristics

Characteristic	OR(95%CI)	*P* for interaction
Age		0.112
20–39	1.02 (0.92, 1.14)	
40–59	1.04 (0.97, 1.11)	
≥ 60	1.16 (1.05, 1.28)	
Gender		0.075
Male	1.10 (1.04, 1.17)	
Female	1.00 (0.94, 1.07)	
Race		**0.023**
Mexican American	1.00 (0.93, 1.08)	
Other Hispanic	1.08 (0.97, 1.21)	
Non-Hispanic White	1.07 (1.00, 1.14)	
Non-Hispanic Black	0.97 (0.94, 1.00)	
Other Race - Including Multi-Racial	1.09 (1.00, 1.20)	
Education		0.092
Less than high school	1.04 (0.92, 1.17)	
High school grad/GED or equivalent	1.03 (0.93, 1.13)	
Higher than high school	1.06 (1.01, 1.12)	
Marital status		**0.041**
Married/living with partner	1.05 (0.97, 1.13)	
Widowed/divorced/separated	1.01 (0.94, 1.08)	
Never married	1.10 (1.03, 1.19)	
PIR		0.958
≤ 1.3	1.06 (0.99, 1.12)	
1.3–3.5	1.07 (0.98, 1.17)	
≥ 3	1.00 (0.84, 1.20)	
BMI		0.392
≤ 25 kg/m2	1.06 (0.96, 1.18)	
25–30 kg/m2	1.11 (1.03, 1.20)	
≥ 30 kg/m2	1.02 (0.95, 1.10)	
Smoking status		0.875
Current non smokers	1.05 (1.00, 1.10)	
Current smokers	1.09 (0.98, 1.20)	
Drinking status		0.801
Current non drinkers	1.04 (0.96, 1.12)	
Current moderate drinkers	1.07 (1.01, 1.13)	
Current heavy drinkers	1.09 (0.91, 1.32)	
Noise exposure		0.287
Yes	1.07 (1.02, 1.13)	
No	1.02 (0.96, 1.09)	

elevated WBCC appeared to have predictive value for hearing impairment in the elderly [12].

Cochlear hair cells in the inner ear play a crucial role in efficiently converting sound energy into nerve signals and transmitting them to the brain during the normal auditory perception process. However, once these cells are damaged, they cannot regenerate [25]. Inflammatory factors can impair the microcirculatory function of the inner ear through various mechanisms. Firstly, they may increase capillary permeability, resulting in the obstruction of nutrient transport to inner ear tissues, which can lead to hypoxia and malnutrition of hair cells and neurons [26]. Additionally, inflammatory factors such as IL-1β and TNF-α can exert direct effects on the vascular endothelial cells within the inner ear, leading to their dysfunction and promoting apoptosis of hair cells [27, 28]. The activation of the NLRP3 inflammasome pathway is associated with noise-induced cochlear damage. This activation results in the production of additional inflammatory cytokines which exacerbate the inflammatory response and contribute to the destruction of cochlear hair cells [29]. Furthermore, chronic inflammatory states

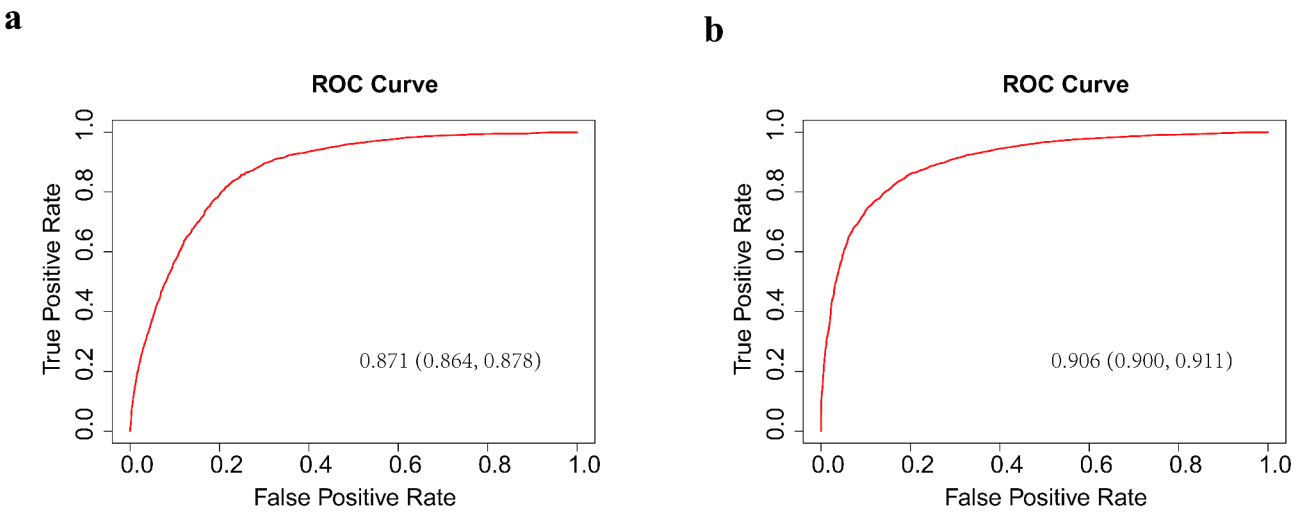

Fig. 3 ROC analysis. (**a**) ROC curve and diagnostic value of NHR in SFHL. (**b**) ROC curve and diagnostic value of NHR in HFHL

can induce neuroinflammation along the central auditory pathway, thereby playing a role in the development of sensorineural hearing loss [30]. Chan J et al. found that a long-term high-fat diet can trigger a localized inflammatory response in the cochlea, characterized by increased expression of inflammatory markers and the recruitment of macrophages to the cochlea [31]. This suggests that changes in systemic metabolic and inflammatory status may indirectly influence hearing health by altering the microenvironment of the inner ear. Additionally, oxidative stress is also a significant mechanism contributing to hearing loss. Both inflammation and lipid metabolism disorders trigger excessive production of reactive oxygen species (ROS), leading to oxidative stress (OS) [32, 33]. OS can cause hair cell apoptosis and necrosis by destroying intracellular macromolecules and impairing mitochondrial function, ultimately leading to sensorineural hearing loss [9, 34]. In short, systemic inflammatory status plays a role in sensorineural hearing loss through various pathways and mechanisms. According to our results, participants with hearing loss exhibited elevated neutrophil counts. As an important cell type of the innate immune system, the increased number of neutrophils often indicates a rise in systemic inflammation levels and tissue injury [35, 36]. A study by Bae SH et al. found that neutrophils were recruited in the spiral ligament instead of the stria vascularis (SV) region and Corti organ in LPS-induced cochlear inflammation [37]. Previous studies have shown that HDL possesses anti-inflammatory effects [38, 39]. However, in this study, no significant difference in HDL levels was observed between participants with and without hearing loss. Therefore, single biochemical marker may not adequately represent the intricate influence of systemic metabolic and inflammation on hearing loss. Considering that the anti-inflammatory effect of HDL may influence neutrophils, NHR reflects the level of systemic inflammation more comprehensively than a single indicator [18]. From a physiological perspective, an increase in NHR may indicate that the body is experiencing chronic hyperinflammation [40]. As previously mentioned, inflammatory factors contribute to the decline in hearing function through multiple pathways.

Our data demonstrated a positive linear correlation between NHR and hearing loss. This suggests a focus not only on the nutritional supply to the inner ear's neurovascular system but also on the levels of systemic inflammation when preventing and treating hearing loss. This finding offers significant reference value for the clinical identification of high-risk populations for hearing loss and the implementation of targeted intervention strategies. Subgroup analysis showed that the association between higher NHR and hearing loss was more pronounced in non-Hispanic White participants. This finding aligns with a large population-based epidemiological study reporting a lower prevalence of hearing loss among Black participants compared to Whites [41]. Such differences may be attributed to racial variations in baseline systemic inflammation, genetic susceptibility, environmental exposures, and access to healthcare. Mechanistically, cochlear melanin deposition has been suggested to play a protective role against hearing loss [42]. We also observed that low PIR, reflecting lower socioeconomic status, significantly strengthened the association between NHR and hearing loss. This may result from the combined effects of chronic stress, poor nutrition, limited access to healthcare, and higher environmental risk exposures commonly faced by socioeconomically disadvantaged populations. These factors could exacerbate systemic inflammation and amplify the biological impact of elevated NHR on cochlear and auditory function. Consequently, future research should investigate the role of

sociodemographic characteristics in the pathogenesis of hearing impairment to inform the development of targeted intervention measures.

Using nationally representative NHANES data, we applied weighted multivariable logistic regression, RCS regression, and subgroup analyses to minimize the inherent limitations of cross-sectional studies and enhance the reliability of the observed associations while controlling for potential confounders. Although causality cannot be established, cross-sectional studies are well-suited for identifying strong associations and generating hypotheses for future research. Before the NHR can be recommended as a screening tool for hearing loss risk, further longitudinal studies are necessary to validate its predictive value over time and to assess its clinical utility in early detection and prevention strategies.

The study has the following limitations. On the one hand, the cross-sectional design employed did not allow for the determination of a causal relationship between the two factors, highlighting the need for longitudinal cohort studies to track the dynamic relationship between changes in inflammatory indicators and changes in hearing. On the other hand, the study focused exclusively on NHR as a comprehensive inflammation indicator and did not thoroughly evaluate other inflammatory cytokines, oxidative stress, and additional biomarkers, which could help elucidate the specific mechanisms of inflammation. Participants primarily consist of individuals from the U.S., resulting in a relatively concentrated distribution of race and socioeconomic status. Expanding the sample range could enhance the generalizability of the results. Finally, the study did not fully account for potential confounding factors, such as past medical history, medication use, and genetic predisposition, which may interfere with an accurate assessment of the relationship between inflammatory indicators and hearing loss through their potential impact on hearing. While this study contributes new evidence regarding the significant role of systemic inflammation in hearing health, the research design requires further optimization, and the observation indicators and population range should be expanded to better elucidate the intrinsic relationship.

Conclusion

This cross-sectional study showed a positive correlation between NHR and two types of hearing loss, including SFHL and HFHL. This association was consistently confirmed across diverse population characteristics. These findings underscore the potential of controlling systemic inflammation to reduce the risk of hearing damage. Moreover, they provide new evidence for exploring factors affecting hearing health from the perspectives of inflammation and metabolism, while also seeking potential biomarkers for the early identification and prevention of hearing loss.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s41043-025-00851-0.

Additional file 1

Additional file 2

Acknowledgements

We acknowledge the workers who contributed to the collection of NHANES, making it possible for scientists and doctors worldwide to share these data.

Author contributions

Y. Y. led the study's conception, overall design, main data analysis, and paper writing. Z. S. contributed to data analysis and provided advice on paper revision. Y. L. checked and revised the manuscript. Z. X. made significant contributions to study initiation, supervision, project, management, manuscript writing, and final revision. The final manuscript was read and approved by all authors.

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Data availability

This study analyzes publicly available datasets. These data can be found here: National Health and Nutrition Examination Survey Database (https://www.cdc.gov/nchs/nhanes/?CDC_AAref_Val=https://www.cdc.gov/nchs/nhanes/index.htm).

Declarations

Ethics approval and consent to participate

Data for this study was collected from the NHANES database. Approval was obtained from the Research Ethics Review Board of the National Center for Health Statistics (NCHS). All participants provided written informed consent, ensuring compliance with relevant local laws and regulations to uphold the legality of the research.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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