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ORIGINAL ARTICLE

The Association between NHHR and Nonalcoholic Fatty Liver Disease: Result from NHANES 2017 - 2023

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SUMMARY

Background: **The non-high-density lipoprotein cholesterol-to-high-density lipoprotein cholesterol ratio (NHHR) has emerged as a novel lipid biomarker with potential relevance to nonalcoholic fatty liver disease (NAFLD), yet its role remains underexplored.**
Methods: **Utilizing data from the National Health and Nutrition Examination Survey (NHANES) spanning 2017 through 2023, we investigated the relationship between NHHR and NAFLD. NHHR values were log-transformed (LnNHHR) to achieve normal distribution. Multivariate logistic regression and restricted cubic spline (RCS) models were applied to examine the association between NHHR and both NAFLD and hepatic fibrosis. Robustness was evaluated through subgroup and sensitivity analyses.**
Results: **NHHR levels were significantly elevated in individuals with NAFLD and hepatic fibrosis compared to those without ($p < 0.001$). Multivariate logistic regression indicated a positive correlation between increased LnNHHR and NAFLD risk [odds ratio (OR): 2.94, $p < 0.001$], whereas no significant association was found with hepatic fibrosis (OR: 1.02, $p = 0.870$). Participants in the highest LnNHHR quartile (Q4) had a 3.09-fold higher likelihood of NAFLD compared to those in the lowest quartile (Q1) [95% confidence interval (CI): 2.60 - 3.67, $p < 0.001$]. However, no significant trend was observed for hepatic fibrosis across quartiles ($p > 0.05$). RCS analysis revealed a J-shaped relationship between LnNHHR and both NAFLD ($p_{interaction} < 0.001$) and hepatic fibrosis ($p_{interaction} = 0.006$). Stratified analyses further demonstrated that NHHR's impact on NAFLD varied across age groups ($p_{interaction} = 0.024$), while its effect on hepatic fibrosis differed by education level ($p_{interaction} = 0.048$).**
Conclusions: **NHHR is strongly linked to an increased risk of NAFLD, suggesting that improving NHHR levels may help mitigate hepatic steatosis.**
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KEYWORDS

NHHR, NAFLD, hepatic fibrosis, NHANES, cross-sectional study

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is characterized by hepatic steatosis affecting at least 5% of liver tissue in the absence of significant alcohol consumption or other identifiable causes of fat accumulation [1]. The global prevalence of NAFLD was estimated at 25% in 2016, rising to over 30% by 2019 [2], and is projected to become the leading cause of chronic liver disease

worldwide [3]. This increase is driven by the growing prevalence of metabolic risk factors and an aging population, which are expected to more than double the burden of advanced NAFLD-related conditions between 2016 and 2030 [4]. As a progressive disorder, NAFLD can advance to non-alcoholic steatohepatitis (NASH), liver fibrosis, cirrhosis, and hepatocellular carcinoma, often coexisting with diabetes, cardiovascular disease, and chronic kidney disease, significantly increasing mortality risk [5,6]. Its pathogenesis is complex and influenced by metabolic dysfunction, gut microbiota, genetic and epigenetic factors, and environmental exposures [7]. Early detection and evaluation of NAFLD and liver fibrosis are crucial for tracking disease progression and guiding therapeutic decisions [8]. Given the current lack of effective pharmacological treatments, non-invasive approaches for diagnosing and staging liver fibrosis are particularly valuable.

Dyslipidemia is a common feature in NAFLD, typically presenting as elevated triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C) alongside reduced high-density lipoprotein cholesterol (HDL-C). Hepatic cholesterol accumulation is also recognized as a key driver of NAFLD progression [9]. Additionally, the disease is often linked to increased circulating atherogenic lipids and lower HDL-C levels [9,10]. Previous studies have established associations between NAFLD and lipid parameters such as remnant cholesterol [11], HDL-C [12], and non-HDL-C [13], suggesting that the ratio of non-HDL-C to HDL-C (NHHR) may serve as a potential marker for assessing NAFLD and hepatic fibrosis risk. Notably, NHHR has demonstrated superior predictive value over traditional lipid metrics in evaluating abdominal aortic aneurysm [14] and cardiovascular disease [15]. Furthermore, it has been identified as an independent risk indicator for conditions such as diabetes [16] and metabolic syndrome [17], underscoring its relevance in metabolic disease assessment. However, the relationship between NHHR and liver steatosis or fibrosis remains inadequately explored.

To address this gap, our study leveraged data from the National Health and Nutrition Examination Survey (NHANES) to investigate the association between NHHR and both hepatic steatosis and fibrosis in adults.

MATERIALS AND METHODS

Study population

This analysis utilized data from the combined NHANES survey cycles spanning 2017 through 2023, encompassing 27,493 participants. After applying exclusion criteria - including minors, individuals lacking vibration-controlled transient elastography (VCTE) results, those with hepatitis B surface antigen positivity, missing NHHR or covariate data, hepatitis C antibody or RNA positivity, a history of chronic or autoimmune hepatitis, and heavy alcohol consumption (> 4 drinks/day) - a total of 8,350 participants were included in the final analysis. The detailed participant selection process is illustrated in Figure 1.

NHHR calculation

NHHR was derived from lipid profile measurements. Non-HDL-C levels were calculated by subtracting HDL-C from total cholesterol (TC). The NHHR was then obtained by dividing non-HDL-C by HDL-C.

Assessment of NAFLD and hepatic fibrosis

VCTE was performed using the FibroScan®-equipped model 502 touch, operated by trained NHANES personnel. Hepatic steatosis was assessed via the controlled attenuation parameter (CAP), while liver stiffness measurement (LSM) was used to evaluate hepatic fibrosis. Based on prior research, NAFLD was diagnosed when CAP values met or exceeded 274 dB/m, with severe steatosis defined at a threshold of 302 dB/m. Fibrosis severity was categorized as F2, F3, or F4, corresponding to LSM thresholds of 8.2, 9.7, and 13.6 kPa, respectively. An LSM value of ≥ 8.2 kPa was considered indicative of hepatic fibrosis (stage ≥ F2) [18].

Covariates

Covariates were carefully selected based on their established relevance in prior literature. Demographic variables included gender, age, and race, while socioeconomic and lifestyle factors encompassed education level, poverty-to-income ratio (PIR), body mass index (BMI), and waist circumference. Additionally, smoking status and medical histories of diabetes mellitus (DM) and hypertension were extracted from NHANES to ensure a comprehensive analytical framework.

Statistical analyses

Baseline characteristics of the study population were stratified by NAFLD and hepatic fibrosis status. Continuous variables were summarized as medians with interquartile ranges (IQR), while categorical variables were expressed as frequencies and percentages.

Weighted multivariable logistic regression models were employed to assess the relationship between NHHR and the presence of NAFLD and hepatic fibrosis. Odds ratios (OR) and their corresponding 95% confidence intervals (CI) were reported to quantify the strength of these associations. To evaluate linear trends, NHHR was divided into quartiles, with the lowest quartile serving as the reference group. NHHR values were log-transformed (LnNHHR) to achieve normal distribution. Three regression models were constructed: Model 1 was unadjusted, Model 2 was adjusted for age, gender, and race, while Model 3 incorporated additional adjustments for PIR, education level, BMI, waist circumference, smoking status, DM, and hypertension.

To explore potential non-linear associations between NHHR and NAFLD or hepatic fibrosis, restricted cubic spline (RCS) regression analysis was conducted. Subgroup analyses and interaction tests were performed to assess variations across demographic and clinical sub-

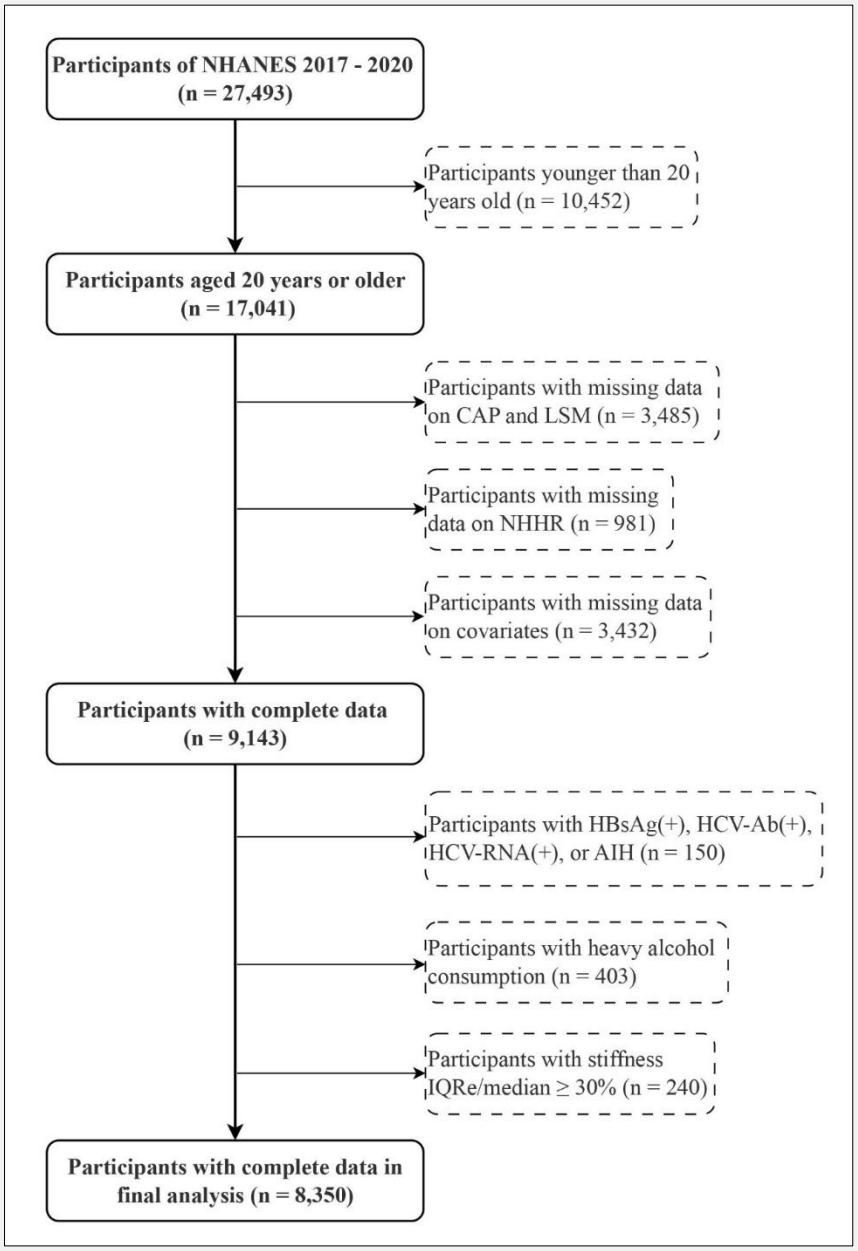

Figure 1. The flowchart of the participants screened from the NHANES.

groups.

Sensitivity analyses were conducted to validate the robustness of our findings. First, the receiver operating characteristic (ROC) curve analysis (data not shown) determined LnNHHR cutoff values of 0.838 and 0.897, which were subsequently treated as binary variables.

In a secondary sensitivity analysis, participants with hypercholesterolemia (defined as non-HDL-C ≥ 160 mg/dL) were excluded.

All statistical analyses were performed using R version 4.3.3 (R Foundation for Statistical Computing, Vienna, Austria), with a two-sided p-value < 0.05 considered statistically significant.

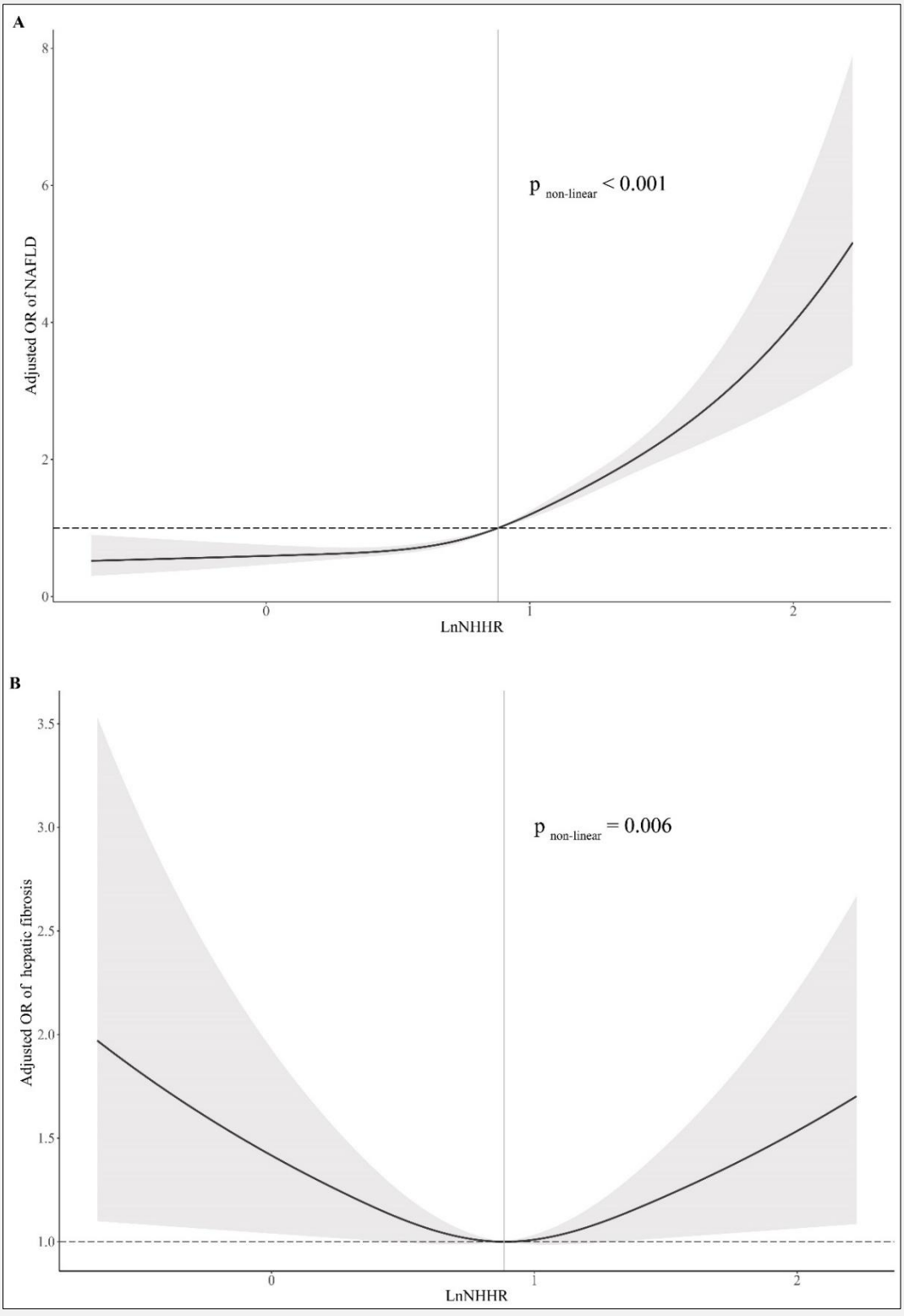

Figure 2. Dose-response relationships of NHHR and the risk of NAFLD and hepatic fibrosis.

A Dose-response of NHHR and NAFLD. B Dose-response of NHHR and hepatic fibrosis. The odds ratio and 95% confidence interval were adjusted for gender, age, poverty-to-income ratio, education level, body mass index, waist circumference, smoking status, diabetes mellitus, and hypertension.

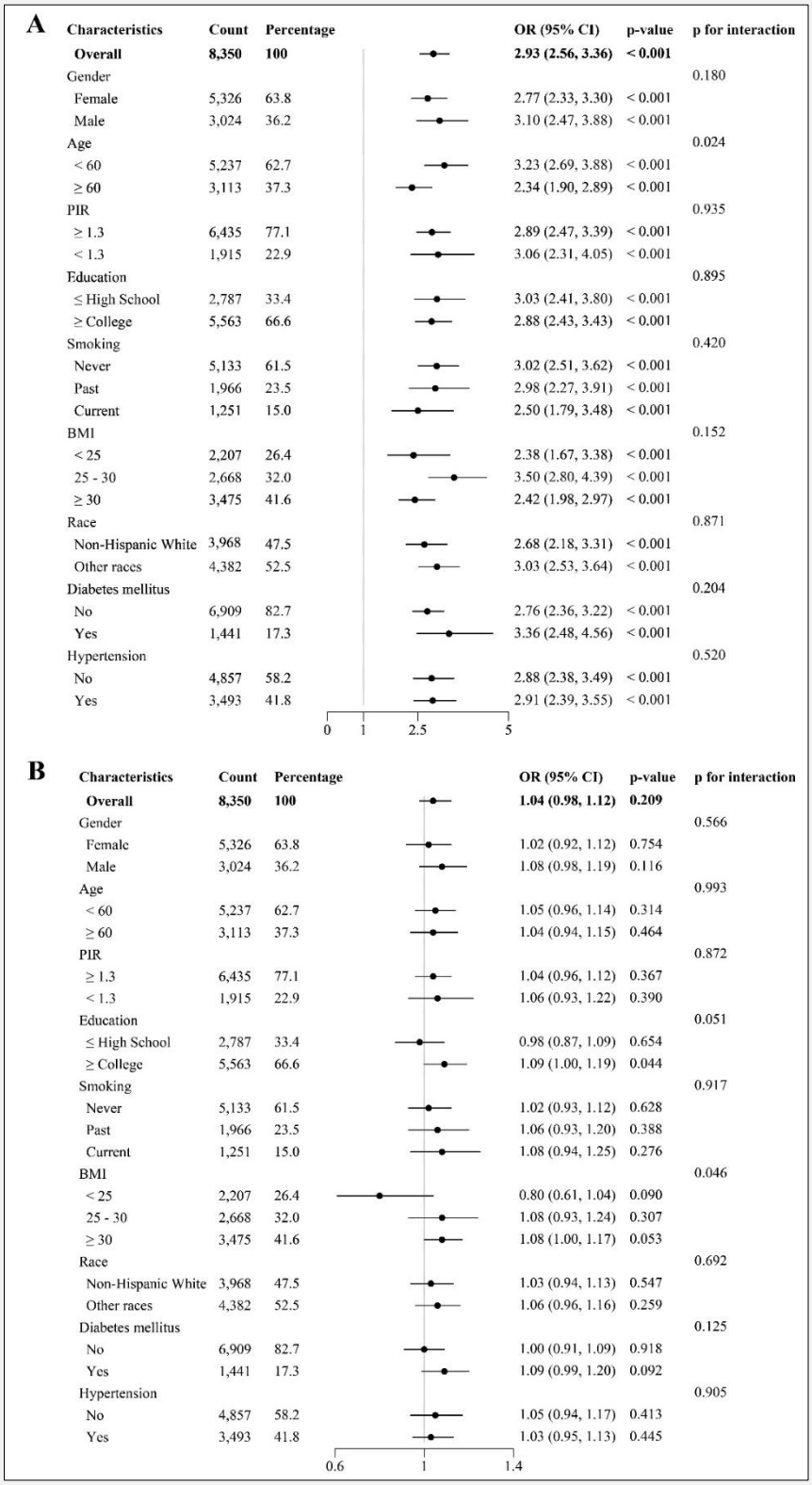

Figure 3. Subgroup analysis of the association between NHHR with NAFLD (A) and hepatic fibrosis (B).

Each stratification was adjusted for gender, age, poverty-to-income ratio, education level, body mass index, waist circumference, smoking status, diabetes mellitus, and hypertension.

RESULTS

Population characteristics

Table S1 provides a comprehensive summary of the demographic and clinical characteristics of the study population, stratified by NAFLD and hepatic fibrosis status. The median age of participants was 52 years (IQR: 37 - 65), with 36.2% being male. NAFLD was diagnosed in 3,464 individuals (41.5% of the cohort), while hepatic fibrosis was present in 798 individuals (9.6%). The median NHHR was significantly elevated in individuals with NAFLD (2.84, IQR: 2.15 - 3.72) and hepatic fibrosis (2.66, IQR: 1.94 - 3.52) compared to those without these conditions (NAFLD: 2.15, IQR: 1.64 - 2.84; hepatic fibrosis: 2.39, IQR: 1.78 - 3.19; both $p < 0.001$).

Participants with NAFLD or hepatic fibrosis tended to be older (NAFLD: 55 vs. 49 years; hepatic fibrosis: 58 vs. 51 years) and were more frequently female (NAFLD: 59.6% vs. 40.4%; hepatic fibrosis: 58.9% vs. 41.1%). Additionally, these individuals exhibited higher BMI, greater waist circumference, and an increased prevalence of DM and hypertension compared to those without NAFLD or hepatic fibrosis.

Association between NHHR and NAFLD and hepatic fibrosis

Table S2 summarizes the relationship between NHHR and the risks of NAFLD and hepatic fibrosis. In continuous model analyses, NHHR was significantly associated with an increased risk of NAFLD in the unadjusted (OR: 4.77), partially adjusted (OR: 4.98), and fully adjusted models (OR: 2.94; all $p < 0.001$). However, the association between NHHR and hepatic fibrosis was statistically nonsignificant in the fully adjusted model (OR: 1.02, $p = 0.870$).

Stratified analysis by NHHR quartiles indicated that individuals in the highest quartile (Q4) had a 3.09-fold greater risk of NAFLD in the fully adjusted model (95% CI: 2.60 - 3.67, $p < 0.001$) compared to those in the lowest quartile (Q1). Conversely, NHHR in Q4 did not significantly differ from Q1 regarding hepatic fibrosis risk (95% CI: 0.77 - 1.30, $p > 0.990$).

To further explore the potential dose-response relationship, an RCS model was applied. The analysis revealed a J-shaped association between NHHR and NAFLD risk, with a significant deviation from linearity ($p_{non\text{-}linear} < 0.001$) (Figure 2A). The threshold analysis (Table S3) identified an inflection point at 0.885 for LnNHHR. Below this threshold, an increase in LnNHHR was significantly associated with NAFLD risk ($p = 0.012$), while above this threshold, each unit increase in LnNHHR corresponded to a 3.16-fold rise in NAFLD risk (OR: 3.16, 95% CI: 2.40 - 4.17, $p < 0.001$).

A similar J-shaped trend was observed for hepatic fibrosis risk, with significant non-linearity ($p_{non\text{-}linear} = 0.006$) (Figure 2B). Below the inflection point of 0.885, LnNHHR was not significantly linked to hepatic fibrosis risk ($p = 0.160$). However, above this threshold, each unit increase in LnNHHR was associated with a 54% increase in hepatic fibrosis risk (OR: 1.54, 95% CI: 1.04 - 2.29, $p = 0.031$).

Subgroup and sensitivity analyses

To ensure the robustness of our findings, stratified analyses and interaction tests were conducted to evaluate the association between NHHR and NAFLD/hepatic fibrosis across various demographic subgroups, including gender, age, PIR, education level, smoking status, BMI, race, DM, and hypertension. The association between NHHR and NAFLD remained consistent across all subgroups (all $p < 0.05$). Notably, significant interactions were observed between NHHR and age for NAFLD risk ($p_{interaction} < 0.05$) (Figure 3A) and between NHHR and education level for hepatic fibrosis risk (Figure 3B).

A sensitivity analysis was performed by excluding participants with hypercholesterolemia. The association between NHHR and NAFLD remained statistically significant (OR: 1.95, 95% CI: 1.72 - 2.20, $p < 0.001$), whereas no significant relationship was observed for hepatic fibrosis (OR: 1.02, 95% CI: 0.87 - 1.19, $p = 0.980$) in the fully adjusted model when NHHR was treated as a binary variable (Table S4). Similarly, in the highest NHHR quartile (Q4), a strong association with increased NAFLD risk persisted (OR: 2.78, 95% CI: 2.22 - 3.49, $p < 0.001$), while no significant link was found for hepatic fibrosis (OR: 0.96, 95% CI: 0.77 - 1.21, $p = 0.720$) in the fully adjusted model.

DISCUSSION

This large-scale study, encompassing 8,350 adults, examined the relationship between NHHR and the risk of NAFLD and hepatic fibrosis in a nationally representative U.S. population. Our findings suggest that elevated NHHR is significantly associated with an increased risk of NAFLD, even after adjusting for multiple covariates, whereas no significant link was observed with hepatic fibrosis. This association remained robust across various demographic subgroups, including gender, age, PIR, education level, smoking status, BMI, race, DM, and hypertension. Furthermore, sensitivity analyses excluding individuals with hypercholesterolemia reaffirmed the strong correlation between NHHR and NAFLD risk.

Previous studies have also identified a positive association between NHHR and NAFLD, although they were primarily conducted outside the U.S. In a cross-sectional analysis of 7,759 Chinese children and adolescents (aged 2 - 8 years), Yang et al. [19] reported a significant correlation between NHHR and NAFLD prevalence ($p < 0.001$), with an OR of 1.83 for individuals in the highest NHHR tertile compared to the lowest. Additionally, NHHR demonstrated superior discriminatory power in diagnosing NAFLD, with AUC values of 0.787 for boys and 0.763 for girls, both significantly higher than those of non-HDL-C and HDL-C ($p < 0.001$). Similarly,

in a longitudinal study of 16,173 initially NAFLD-free participants, Gao et al. [20] found that higher NHHR levels were associated with increased cumulative NAFLD incidence, with HRs of 1.3 and 1.5 for the second and third tertiles, respectively, over a five-year follow-up. Notably, NHHR also exhibited stronger predictive capability than non-HDL-C, with an AUC of 0.705 versus 0.656. These findings, in conjunction with our own, underscore NHHR's potential as a predictive marker for NAFLD. Unlike prior studies that relied on single-center cohorts, our analysis leveraged a nationally representative dataset, enhancing the generalizability of our results to the broader U.S. population.

Despite the well-established role of hepatic fibrosis in NAFLD progression, our study did not detect a significant association between NHHR and hepatic fibrosis risk. Fibrosis results from an imbalance between fibrogenesis and fibrolysis, ultimately leading to extracellular matrix accumulation when fibrogenesis predominates [8]. The limited research on NHHR's relationship with hepatic fibrosis makes direct comparisons challenging. However, prior studies have linked lower HDL-C levels, a component of NHHR, to adverse outcomes in chronic liver disease. Rao et al. [21] reported that decreased HDL-C levels indicate hepatic fibrosis and are associated with poor prognoses in liver disease patients. Similarly, in individuals with NAFLD and liver fibrosis, low HDL-C has been implicated in an increased risk of hepatocellular carcinoma [22]. Given HDL-C's antioxidant properties and role in reverse cholesterol transport, it is a key factor in NAFLD pathogenesis [23]. Variations in study design, diagnostic criteria for NAFLD, sample size, and ethnic differences may contribute to the inconsistencies observed across studies.

In our study, age, gender, PIR, education level, smoking status, BMI, race, DM, and hypertension were considered as potential stratification factors when assessing NHHR's association with NAFLD and hepatic fibrosis. Notably, age was the only factor that significantly modified the NHHR-NAFLD relationship. Participants younger than 60 years exhibited a higher OR for NAFLD, consistent with prior research [20]. While hypercholesterolemia has been linked to increased NAFLD risk [24], excluding individuals with hypercholesterolemia in our sensitivity analysis did not alter the significance of our findings, reinforcing the robustness of NHHR as an independent predictor of NAFLD.

Strengths and limitations

This study has several key strengths. The use of a large, nationally representative sample from NHANES enhances the reliability of our findings, minimizing random variability and improving generalizability. Furthermore, the comprehensive adjustment for a wide range of demographic and lifestyle factors strengthens the validity of our conclusions.

Nonetheless, certain limitations must be acknowledged. First, the cross-sectional study design precludes causal inferences regarding NHHR's role in NAFLD development. Second, despite controlling for multiple confounders, the possibility of residual confounding due to unmeasured variables cannot be entirely excluded. Third, as our sample consisted of U.S. adults, the findings may not be directly generalizable to other populations or ethnic groups. Future studies involving diverse cohorts are warranted to confirm these results.

CONCLUSION

Our findings establish a positive correlation between NHHR and NAFLD risk, highlighting NHHR's potential as a novel predictive marker. However, no significant association was found between NHHR and hepatic fibrosis. Strategies to regulate NHHR may have clinical implications in NAFLD prevention. Further longitudinal studies and randomized controlled trials are needed to validate our findings and explore NHHR's utility as a predictive biomarker for NAFLD.

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Ethical Approval and Consent to Participate:

The portions of this study involving human participants, human materials, or human data were conducted in accordance with the Declaration of Helsinki and were approved by the NCHS Ethics Review Board. Participants provided written informed consent to participate in this study.

Availability of Data and Materials:

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Declaration of Interest:

The authors declare that they have no competing interests.

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