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Association between the non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio and cardiovascular mortality among US adults with diabetes: a prospective cohort study of NHANES 1999–2018

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Abstract

Background: The non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR) integrates information on atherogenic and anti-atherogenic lipoproteins. Its association with cardiovascular mortality in adults with diabetes remains unclear.

Methods: We conducted a prospective cohort analysis of ten National Health and Nutrition Examination Survey (NHANES) cycles (1999–2018) linked to mortality follow-up through December 31, 2019. Diabetes was defined by self-reported diagnosis, insulin use, oral glucose-lowering medication use, or hemoglobin A1c (HbA1c) $\geq 6.5\%$. NHHR was calculated as (total cholesterol – HDL-C)/HDL-C. Survey-weighted cause-specific Cox models and restricted cubic splines were used to examine the association between NHHR and cardiovascular mortality.

Results: The complete-case cohort comprised 6,581 adults with diabetes, including 622 cardiovascular deaths. Compared with Q1, the fully adjusted hazard ratios for cardiovascular mortality were 0.89 (95% confidence interval [CI], 0.67–1.19) for Q2, 1.07 (95% CI, 0.84–1.36) for Q3, and 1.31 (95% CI, 0.95–1.80) for Q4. The hazard ratio per one-quartile increase was 1.11 (95% CI, 1.00–1.22; P for trend=0.043). Restricted cubic spline analysis suggested a nonlinear association (P for overall association=0.024; P for nonlinearity=0.020), with the lowest estimated hazard at an NHHR of approximately 2.5–3.0. The direction of association was similar in sensitivity analyses, although statistical significance was attenuated.

Conclusions: NHHR was nonlinearly associated with cardiovascular mortality among US adults with diabetes. Higher NHHR values above the apparent nadir were associated with a tendency toward higher cardiovascular mortality; however, the association was modest and less precise in sensitivity analyses. These findings warrant cautious interpretation and external confirmation.

Keywords: NHHR; diabetes mellitus; cardiovascular mortality; NHANES; cohort study; nonlinear association

1 Introduction

Diabetes mellitus is a major global health challenge and is associated with a substantially increased risk of cardiovascular disease and premature mortality [1, 2]. Diabetic dyslipidemia, characterized by abnormalities in triglyceride-rich lipoproteins, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol (HDL-C), contributes to this excess cardiovascular risk [3]. Although conventional lipid measures remain central to cardiovascular risk assessment, residual risk persists in many individuals with diabetes [4].

NHHR is calculated by dividing non-HDL cholesterol by HDL-C. Non-HDL cholesterol captures cholesterol in potentially atherogenic apolipoprotein B-containing lipoproteins, whereas HDL-C reflects anti-atherogenic lipid transport pathways. Accordingly, NHHR may provide a more integrated measure of lipid balance than individual lipid indices [5, 6, 7, 8]. Previous studies have linked NHHR to insulin resistance, metabolic syndrome, carotid atherosclerosis, and coronary artery disease [9, 10, 11, 12].

However, the association between NHHR and cardiovascular mortality among adults with diabetes remains uncertain. Previous NHANES-based studies have reported heterogeneous associations, including positive and nonlinear patterns [13, 14]. Differences in study populations, diabetes definitions, exposure categorization, covariate adjustment, and handling of the complex survey design may contribute to these inconsistent findings. In particular, assuming a linear association may obscure clinically relevant nonlinear exposure-response patterns.

We therefore examined the association between NHHR and cardiovascular mortality among US adults with diabetes using ten NHANES cycles conducted from 1999 to 2018 and linked mortality follow-up through 2019. Survey-weighted Cox models and restricted cubic splines were used to characterize categorical, linear-trend, and nonlinear associations.

2 Materials and Methods

2.1 Study design and population

This prospective cohort study used data from ten consecutive NHANES cycles conducted between 1999 and 2018. NHANES uses a complex, multistage probability sampling design to represent the noninstitutionalized civilian US population. The original numbers of participants who completed the household interview in each cycle were 9,965 (1999–2000), 11,039 (2001–2002), 10,122 (2003–2004), 10,348 (2005–2006), 10,149 (2007–2008), 10,537 (2009–2010), 9,756 (2011–2012), 10,175 (2013–2014), 9,971 (2015–2016), and 9,254 (2017–2018), for a total of 101,316 participants. All participants provided written informed consent, and study protocols were approved by the National Center for Health Statistics Research Ethics Review Board.

Participants were eligible if they were aged 18 years or older, attended the mobile examination center, and had available total cholesterol and HDL-C measurements. This restriction yielded 52,764 participants for diabetes ascertainment and subsequent eligibility assessment. Diabetes was defined by any of the following: self-reported physician-diagnosed diabetes, current insulin use, current use of oral glucose-lowering medication, or HbA1c $\geq 6.5\%$ [15]. Participants with missing or nonpositive total cholesterol or HDL-C values, ineligibility for mortality linkage, or unavailable follow-up time were excluded.

A total of 7,570 adults with diabetes were eligible for mortality follow-up. The primary complete-case analysis included 6,581 participants after exclusion of individuals with missing covariate data.

2.2 Exposure assessment

Serum total cholesterol and HDL-C were measured using standardized NHANES laboratory procedures. NHHR was calculated as follows:

$$\text{NHHR} = \frac{\text{Total cholesterol} - \text{HDL-C}}{\text{HDL-C}}. \quad (1)$$

For categorical analyses, NHHR was classified into survey-weighted quartiles. The weighted cut-points were 2.19, 3.03, and 4.13, defining Q1 (<2.19), Q2 (2.19–3.03), Q3 (3.03–4.13), and Q4 (>4.13).

2.3 Ascertainment of mortality

Mortality status was ascertained by linkage of NHANES records to the National Death Index through December 31, 2019, using the publicly available 2019 NHANES Linked Mortality File [17]. Follow-up time, expressed in months, was calculated from the examination date until death or the end of follow-up.

The primary outcome was cardiovascular mortality. Using the leading-cause recode in the linked mortality file, cardiovascular deaths were defined as deaths from diseases of heart (UCOD_LEADING=1) or cerebrovascular diseases (UCOD_LEADING=5). Deaths from other causes were treated as competing events and censored at the time of death in the cause-specific Cox models.

2.4 Covariates

Covariates were selected a priori based on their established associations with lipid metabolism and cardiovascular mortality. They included age, sex, race/ethnicity, educational attainment, poverty-income ratio (PIR), body mass index (BMI), ever-smoking status, and hypertension.

Race/ethnicity was categorized as Mexican American, Other Hispanic, Non-Hispanic White, Non-Hispanic Black, and Other race. Educational attainment was categorized as below high school, high school graduate, and college or above. Ever smoking was defined as having smoked at least 100 cigarettes during the lifetime. Hypertension was defined as self-reported physician-diagnosed hypertension or use of antihypertensive medication.

HbA1c was not included in the primary model because it contributed to the diabetes definition; it was additionally adjusted for in a sensitivity analysis.

2.5 Statistical analysis

All analyses accounted for the complex NHANES sampling design, including examination weights, strata, and primary sampling units. For the combined 1999–2018 analysis, mobile examination center weights were recalculated according to NHANES guidance [16]. The 4-year examination weight was divided by 5 for the 1999–2002 cycles, and the 2-year examination weight was divided by 10 for each 2003–2018 cycle. Sampling strata and primary sampling units were nested within survey cycle.

Baseline characteristics were summarized across NHHR quartiles as weighted means with standard errors for continuous variables and weighted percentages for categorical variables. Group differences were evaluated using survey-weighted regression models for continuous variables and design-based Rao–Scott tests for categorical variables.

Survey-weighted cause-specific Cox proportional hazards models estimated hazard ratios (HRs) and 95% CIs for cardiovascular mortality. Model 1 was unadjusted; Model 2 was adjusted for age, sex, race/ethnicity, educational attainment, and PIR; and Model 3 was additionally adjusted for BMI, ever-smoking status, and hypertension. Linear trends were assessed by entering the ordinal NHHR quartile variable as a continuous term.

Restricted cubic spline analysis with four knots located at the 5th, 35th, 65th, and 95th percentiles of NHHR was conducted in the fully adjusted model to assess potential nonlinearity. The median NHHR value (2.95) was used as the reference. Overall and nonlinear associations were evaluated using design-based Wald tests.

Sensitivity analyses comprised: (1) exclusion of participants with follow-up of 24 months or less, with follow-up reset at the 24-month landmark; (2) additional adjustment of Model 3 for HbA1c; and (3) modeling NHHR continuously per one standard deviation increase. The proportional hazards assumption was assessed using Schoenfeld residuals from an unweighted Cox model fitted to the same complete-case cohort. Neither the global test ($P=0.801$) nor the NHHR quartile term ($P=0.825$) indicated a violation of the assumption.

All analyses were conducted in R version 4.4.0. Two-sided P values <0.05 were considered statistically significant.

3 Results

3.1 Study population

Of 52,764 adults with available total cholesterol and HDL-C measurements, 7,579 met the diabetes definition. After exclusion of participants who were ineligible for mortality linkage or had unavailable follow-up time, 7,570 were eligible for mortality analysis. The complete-case primary analytic cohort included 6,581 participants and represented approximately 20.54 million US adults with diabetes (Figure 1).

Participants were categorized into survey-weighted NHHR quartiles: Q1, <2.19 ; Q2, 2.19–3.03; Q3, 3.03–4.13; and Q4, >4.13 . The unweighted sample sizes were 1,746, 1,689, 1,606, and 1,540 in Q1 through Q4, respectively.

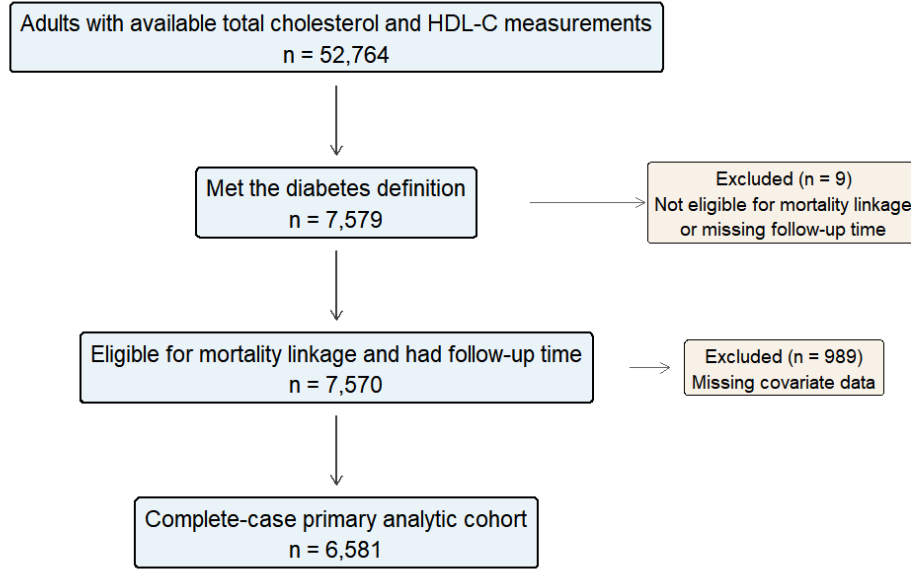


Figure 1: Flowchart of participant selection. HDL-C, high-density lipoprotein cholesterol; NHANES, National Health and Nutrition Examination Survey.

3.2 Baseline characteristics

Baseline characteristics by NHHR quartile are shown in Table 1. Weighted mean age decreased from 62.6 years in Q1 to 55.0 years in Q4 ($P < 0.001$). Higher NHHR quartiles were associated with higher weighted mean BMI and a greater prevalence of male sex and ever smoking (all $P < 0.001$). The prevalence of hypertension decreased from 67.9% in Q1 to 60.3% in Q4 ($P = 0.007$). Educational attainment did not differ significantly across quartiles ($P = 0.082$).

The weighted mean NHHR values were 1.66, 2.60, 3.53, and 5.56 in Q1, Q2, Q3, and Q4, respectively. The weighted mean poverty-income ratio decreased across NHHR quartiles ($P < 0.001$).

Table 1: Weighted baseline characteristics of adults with diabetes by NHHR quartiles (N=6,581).

Characteristic	Q1 (n=1,746)	Q2 (n=1,689)	Q3 (n=1,606)	Q4 (n=1,540)	P value
NHHR, weighted mean (SE)	1.66 (0.01)	2.60 (0.01)	3.53 (0.01)	5.56 (0.06)	<0.001
Age, years, weighted mean (SE)	62.55 (0.48)	60.14 (0.45)	57.59 (0.43)	55.01 (0.41)	<0.001
Male, weighted %	41.7	46.7	56.7	58.9	<0.001
Race/ethnicity, weighted %					<0.001
Mexican American	7.0	8.5	10.4	11.6	
Other Hispanic	4.6	5.0	6.8	6.6	
Non-Hispanic White	60.9	61.5	61.7	62.3	
Non-Hispanic Black	18.2	17.0	13.7	10.3	
Other race	9.2	8.0	7.3	9.2	
Educational attainment, weighted %					0.082
Below high school	22.9	24.1	27.9	27.0	
High school graduate	24.6	26.3	25.3	25.4	
College or above	52.6	49.6	46.8	47.6	
Poverty-income ratio, weighted mean (SE)	2.90 (0.06)	2.71 (0.05)	2.71 (0.07)	2.55 (0.06)	<0.001
BMI, kg/m ² , weighted mean (SE)	31.19 (0.24)	33.46 (0.30)	33.94 (0.31)	34.01 (0.28)	<0.001
Ever smoker, weighted %	46.5	49.5	49.5	59.7	<0.001
Hypertension, weighted %	67.9	65.3	61.4	60.3	0.007

Values are weighted means (standard errors) or weighted percentages, unless otherwise indicated. *P* values were calculated using survey-weighted regression models for continuous variables and design-based Rao–Scott tests for categorical variables. BMI, body mass index; NHHR, non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio; SE, standard error.

3.3 Association between NHHR and cardiovascular mortality

During follow-up, 622 cardiovascular deaths and 1,189 non-cardiovascular deaths occurred in the complete-case cohort. The unweighted numbers of cardiovascular deaths were 151, 145, 156, and 170 in Q1 through Q4, respectively.

NHHR quartiles were not associated with cardiovascular mortality in the unadjusted model (*P* for trend=0.656). After adjustment for age, sex, race/ethnicity, educational attainment, and poverty-income ratio, a positive trend emerged (*P* for trend=0.040). In the fully adjusted model, the HRs for cardiovascular mortality were 0.89 (95% CI, 0.67–1.19) for Q2, 1.07 (95% CI, 0.84–1.36) for Q3, and 1.31 (95% CI, 0.95–1.80) for Q4, relative to Q1. The HR per one-quartile increase was 1.11 (95% CI, 1.00–1.22; *P* for trend=0.043) (Table 2).

When NHHR was modeled as a continuous variable, the association per one standard deviation increase was not statistically significant (HR, 1.08; 95% CI, 0.97–1.20; *P*=0.148).

Table 2: Association of NHHR quartiles with cardiovascular mortality in survey-weighted Cox proportional hazards models (N=6,581; 622 cardiovascular deaths).

NHHR category	Model 1, HR (95% CI)	Model 2, HR (95% CI)	Model 3, HR (95% CI)
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	0.83 (0.62–1.10)	0.90 (0.67–1.19)	0.89 (0.67–1.19)
Q3	0.86 (0.67–1.10)	1.07 (0.84–1.35)	1.07 (0.84–1.36)
Q4	0.91 (0.68–1.22)	1.32 (0.96–1.80)	1.31 (0.95–1.80)
Per one-quartile increase	0.98 (0.89–1.07)	1.11 (1.00–1.22)	1.11 (1.00–1.22)
<i>P</i> for trend	0.656	0.040	0.043

Model 1 was unadjusted. Model 2 was adjusted for age, sex, race/ethnicity, educational attainment, and poverty-income ratio. Model 3 was additionally adjusted for body mass index, ever-smoking status, and hypertension. HR, hazard ratio; CI, confidence interval; NHHR, non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio.

Restricted cubic spline analysis provided evidence of an overall nonlinear association between NHHR and cardiovascular mortality (P for overall association=0.024; P for nonlinearity=0.020) (Figure 2). The estimated hazard was lowest at an NHHR of approximately 2.5–3.0 and increased at higher NHHR values. Confidence intervals widened at the extremes of the NHHR distribution.

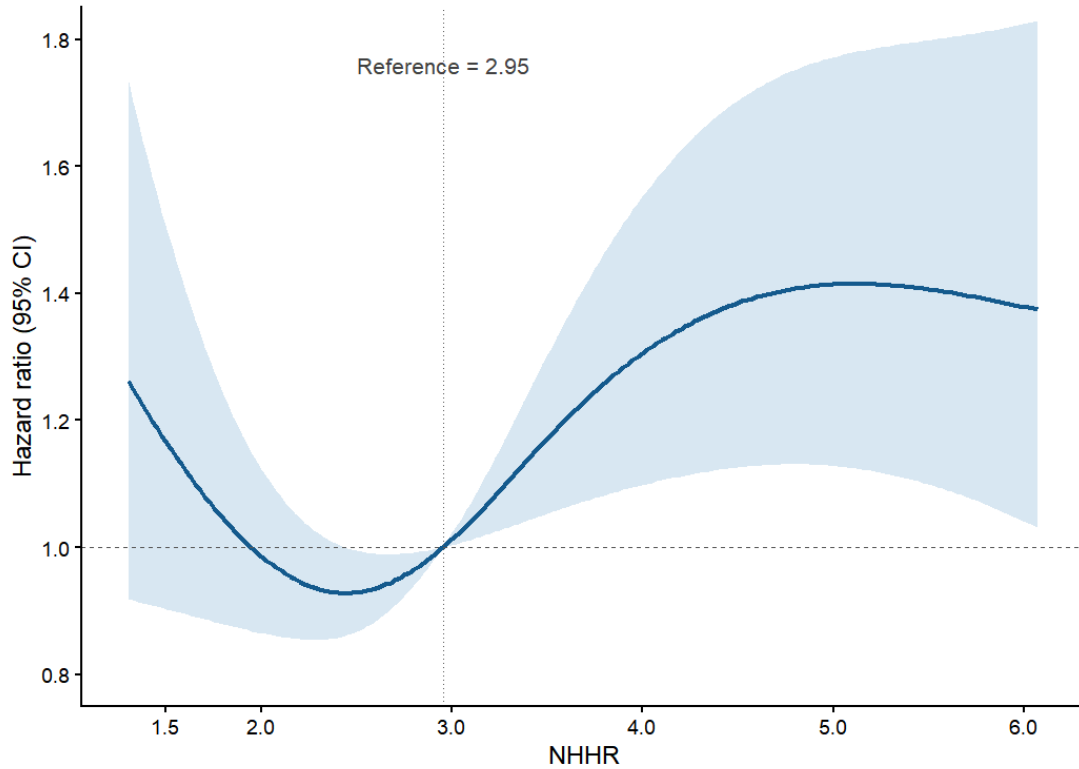


Figure 2: Restricted cubic spline analysis of the association between NHHR and cardiovascular mortality. Hazard ratios were adjusted for age, sex, race/ethnicity, education, poverty-income ratio, body mass index, smoking status, and hypertension. The solid line represents the adjusted hazard ratio and the shaded area represents the 95% confidence interval. The reference value was NHHR=2.95. P for overall association=0.024; P for nonlinearity=0.020.

3.4 Sensitivity analyses

In the 24-month landmark analysis, the direction and magnitude of association were similar to those in the primary analysis. The HR per one-quartile increase in NHHR was 1.10 (95% CI, 0.99–1.22), although the trend did not reach conventional statistical significance ($P=0.064$).

After additional adjustment for HbA1c, the association was also attenuated but remained directionally consistent. The HR per one-quartile increase in NHHR was 1.09 (95% CI, 0.98–1.21; $P=0.098$). In this analysis, 6,568 participants and 620 cardiovascular deaths were included (Supplementary Table S1).

3.5 Proportional hazards assumption

In the diagnostic Cox model fitted to the complete-case cohort, the global Schoenfeld residual test did not indicate a violation of the proportional hazards assumption ($P=0.801$). The NHHR quartile term also satisfied the proportional hazards assumption ($P=0.825$).

4 Discussion

In this nationally representative cohort of US adults with diabetes, NHHR was nonlinearly associated with cardiovascular mortality. In the fully adjusted survey-weighted Cox model, the hazard of cardiovascular mortality increased by 11% per one-quartile increase in NHHR; however, the estimate was modest, with a lower 95% CI bound of 1.00. The Q4-versus-Q1 comparison was not statistically significant. Restricted cubic spline analysis indicated the lowest estimated hazard at an NHHR of approximately 2.5–3.0, followed by higher estimated risk at higher NHHR values.

This pattern should be interpreted cautiously. The continuous analysis per one standard deviation increase was not statistically significant, and spline-based estimates were imprecise at low and high NHHR values. Moreover, the positive quartile trend was attenuated after exclusion of the first 24 months of follow-up and after additional adjustment for HbA1c. Taken together, these findings are suggestive rather than definitive and may be sensitive to modeling choices and residual confounding.

Our findings add to the mixed literature on NHHR and mortality in people with diabetes. Previous NHANES-based studies have reported heterogeneous dose-response patterns, including positive, U-shaped, and L-shaped associations between NHHR and mortality outcomes [13, 14]. Variations in study populations, diabetes definitions, outcome ascertainment, covariate adjustment, handling of the complex survey design, and exposure parameterization may explain some of this inconsistency. In the present analysis, higher NHHR quartiles were associated with younger age but with a greater prevalence of male sex, ever smoking, and higher BMI. This age gradient is important because age strongly predicts cardiovascular mortality and materially influenced the association after multivariable adjustment.

The nonlinear pattern is biologically plausible but does not establish a clinical threshold. NHHR reflects the balance between non-HDL cholesterol, which represents atherogenic lipoprotein burden, and HDL-C. Thus, a higher NHHR may indicate a less favourable lipid profile. Conversely, very low NHHR values can arise from different combinations of low non-HDL cholesterol and high HDL-C, with potentially distinct clinical implications. The present study could not distinguish these mechanisms; therefore, the apparent nadir should not be regarded as a treatment target.

This study has several strengths. It pooled ten NHANES cycles, incorporated nationally representative sampling weights, used standardized lipid measurements, and included linked mortality follow-up. The complex survey design was accounted for, and the association was evaluated using both categorical and nonlinear exposure parameterizations. Landmark and HbA1c-adjusted sensitivity analyses further assessed the potential influence of early events and glycemic control.

Several limitations warrant consideration. First, the observational design precludes causal inference, and residual confounding by diet, physical activity, diabetes duration, comorbidities, medication use, and socioeconomic factors cannot be excluded. Second, lipid measurements and covariates were assessed only at baseline, and changes during follow-up were unavailable. Third, lipid-lowering medication use, particularly statin therapy, was not included in the primary model. Fourth, complete-case analysis may have introduced selection bias. Fifth, cardiovascular mortality was defined with the publicly available leading-cause recode rather than detailed individual-level underlying ICD-10 codes. Finally, the number of cardiovascular deaths was limited, and estimates at the tails of the NHHR distribution were imprecise.

5 Conclusion

Among US adults with diabetes, NHHR was nonlinearly associated with cardiovascular mortality in survey-weighted analyses. NHHR values above approximately 2.5–3.0 were associated with a tendency toward higher cardiovascular mortality; however, the magnitude of association was modest and statistical significance was attenuated in sensitivity analyses. These findings alone do not support NHHR as an independent clinical prognostic marker. Studies with repeated lipid measurements, medication data, and external validation are needed.

Supplementary material

Supplementary Table S1: Sensitivity analyses for the association between NHHR and cardiovascular mortality.

Analysis	N	CVD deaths	Q4 vs Q1, HR (95% CI)	Per one-quartile increase, HR (95% CI)	<i>P</i> for trend
Primary fully adjusted model	6,581	622	1.31 (0.95–1.80)	1.11 (1.00–1.22)	0.043
24-month landmark analysis	5,912	537	1.29 (0.92–1.81)	1.10 (0.99–1.22)	0.064
Additional adjustment for HbA1c	6,568	620	1.25 (0.90–1.73)	1.09 (0.98–1.21)	0.098

All models were adjusted for age, sex, race/ethnicity, educational attainment, poverty-income ratio, body mass index, ever-smoking status, and hypertension, unless otherwise specified. The 24-month landmark analysis excluded participants with follow-up of 24 months or less and reset follow-up time after the landmark. HbA1c, hemoglobin A1c; HR, hazard ratio; CI, confidence interval.

Supplementary Table S2: Continuous-exposure result.

Analysis	Exposure scale	HR (95% CI)	<i>P</i> value
Fully adjusted continuous model	Per 1 SD increase in NHHR	1.08 (0.97–1.20)	0.148

Declarations

Ethics approval and consent to participate

NHANES study protocols were approved by the National Center for Health Statistics Research Ethics Review Board, and all participants provided written informed consent. This study was a secondary analysis of publicly available, de-identified data and did not require additional ethical approval.

Consent for publication

Not applicable.

Availability of data and materials

The datasets analysed during the current study are publicly available from the National Health and Nutrition Examination Survey (NHANES; <https://www.cdc.gov/nchs/nhanes/>) and the NHANES Linked Mortality Files (<https://www.cdc.gov/nchs/data-linkage/mortality.htm>). The R code used to harmonise survey cycles, construct the analytic cohort, and perform the analyses is archived in the Zenodo repository at <https://doi.org/10.5281/zenodo.21962806>.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Rui Lin conceived the study. Zexiong Li, Shangbo Xu, and Yongjian Lin curated the data and performed the statistical analyses. Rui Lin and Youde Liu created the figures and drafted the manuscript. Yangbo Chen supervised the study and critically revised the manuscript. All authors interpreted the data, approved the final manuscript, and agree to be accountable for the work.

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