Nutrición Hospitalaria

Asociación entre Life’s Essential 8 y fibrosis hepática: NHANES 2017-2018

Association between Life’s Essential 8 and liver fibrosis: NHANES 2017-2018

10.20960/nh.05710

09/05/2025

OR 5710

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Fang Qi, Jiahui Yang

Department of Infection. Tongxiang First People's Hospital. Tongxiang, Zhejiang. People's Republic of China

Received: 13/01/2025
Accepted: 01/06/2025
Correspondence: Jiahui Yang. Department of Infection. Tongxiang First People's Hospital. Jiaochang Road 1918. 314500 Tongxiang, Zhejiang. People's Republic of China
e-mail: 18324356574@163.com

Authors´ contribution: Fang Qi: conceptualization, data analysis, and writing - original draft. Jiahui Yang: data collection and curation, and writing - review and editing. All authors have read and approved the final version of the manuscript.

Acknowledgments: The authors sincerely thank all of the NHANES staff and research participants for their invaluable contributions.

Ethics approval and consent to participate: The study was approved by the National Center for Health Statistics Research Ethics Review

Board, and all treatment of human subjects was compliant with the Declaration of Helsinki's ethical standards. Informed written consent was provided by patients/participants to take part in this research.

Data availability statement: All original data in this study were obtained from the publicly available database NHANES: https://wwwn.cdc.gov/nchs/nhanes/Default.aspx.

Conflict of interest: The authors declare no conflict of interest.

Artificial intelligence: The authors declare not to have used artificial intelligence (AI) or any AI-assisted technologies in the elaboration of the article.

ABSTRACT

Objective: liver fibrosis and cardiovascular disease (CVD) are intimately linked. This study aimed to investigate the relationship between liver fibrosis and cardiovascular health (CVH), as assessed by the Life's Essential 8 (LE8) scale.

Methods: 3,609 adult participants from the United States participated in this cross-sectional study, which used information from the National Health and Nutrition Examination Survey (NHANES) database for the years 2017–2018. Three categories were established for CVH and its eight metrics: high (80-100), moderate (50-79), and low (0-49). To screen for liver fibrosis, liver stiffness measurement (LSM) was assessed using vibration-controlled elastography (VCTE) with a threshold of 8.0 kPa. Linear and logistic regression were used to evaluate the relationship between CVH and liver fibrosis, smoothed curve fitting was used to investigate the nonlinear relationship, and

subgroup analysis was performed to test the robustness of the relationship.

Results: total CVH and its subcomponents (diet, PA, BP, blood glucose, and BMI) had an inverse relationship with hepatic fibrosis. In the fully adjusted model, there was a stronger negative connection between the high CVH score group and hepatic fibrosis [odds ratios, OR = 0.10, 95 % confidence intervals, CI (0.05, 0.19)]. Sleep health, nicotine exposure, blood lipids, and liver fibrosis had no statistically significant differences, and the negative link between LE8 and liver fibrosis was stronger in young and middle-aged people.

Conclusions: there was a substantial negative connection between LE8 and its subcomponents and liver fibrosis. According to our research, keeping appropriate CVH levels may help lower the prevalence of liver cirrhosis and fibrosis, which would have a major effect on public health.

Keywords: Liver cirrhosis. Heart disease risk factors. Healthy lifestyle. Nutrition surveys.

RESUMEN

Objetivo: la fibrosis hepática y las enfermedades cardiovasculares (CVD) están íntimamente relacionadas. Este estudio se propuso investigar la relación entre la fibrosis hepática y la salud cardiovascular (CVH), evaluada mediante la escala Life's Essential 8 (LE8).

Métodos: 3609 participantes adultos de Estados Unidos participaron en este estudio transversal, que utilizó información de la base de datos de la Encuesta Nacional de Examen de Salud y Nutrición

(NHANES) de los años 2017-2018. Se establecieron tres categorías para la CVH y sus ocho parámetros: alta (80-100), moderada (50-79) y baja (0-49). Para detectar la fibrosis hepática, se evaluó la medición de la rigidez hepática (LSM) mediante elastografía controlada por vibración (VCTE) con un umbral de 8.0 kPa. Se utilizaron la regresión lineal y logística para evaluar la relación entre la CVH y la fibrosis hepática, el ajuste suavizado de curvas para investigar la relación no lineal y el análisis de subgrupos para comprobar la solidez de la relación.

Resultados: la CVH total y sus subcomponentes (dieta, AF, PA, glucemia e IMC) tenían una relación inversa con la fibrosis hepática. En el modelo totalmente ajustado, hubo una conexión negativa más fuerte entre el grupo de puntuación alta de CVH y la fibrosis hepática [odds ratios, OR = 0,10; intervalos de confianza del 95 %, IC (0,05, 0,19)]. La salud del sueño, la exposición a la nicotina, los lípidos sanguíneos y la fibrosis hepática no presentaron diferencias estadísticamente significativas, y la relación negativa entre el LE8 y la fibrosis hepática fue mayor en las personas jóvenes y de mediana edad.

Conclusiones: se observó una relación negativa sustancial entre el LE8 y sus subcomponentes y la fibrosis hepática. Según nuestra investigación, mantener unos niveles adecuados de CVH puede ayudar a reducir la prevalencia de cirrosis y fibrosis hepáticas, lo que tendría un efecto importante en la salud pública.

Palabras clave: Cirrosis hepática. Factores de riesgo de cardiopatías. Estilo de vida saludable. Encuestas sobre nutrición.

INTRODUCTION

Liver disease accounts for 4 % of all yearly fatalities globally, killing about 2 million people (1). Cirrhosis and its consequences are responsible for 50 % of these fatalities (2). Advanced liver disease is a significant contributor to personal, family, and global financial problems (3,4). Liver fibrosis is an unavoidable step on the path to cirrhosis, and early identification of causal variables and screening for liver fibrosis has been and will continue to be challenging (5). According to several researches, liver fibrosis and exposure to cardiovascular disease (CVD) were independently connected, and the two influenced and impacted each other (6,7).

The American Heart Association updated and revamped cardiovascular health (CVH) in 2022, naming it "Life's Essential 8" (LE8), based on the "Life's Simple 7 (LS7)" recommendation issued in 2010, including the inclusion of a new sub-score (sleep health score), refinement of nutritional metrics, updating of blood lipids and blood glucose metrics, and the adoption of a whole new evaluation approach to give more assistance on decreasing the risk of CVD and other unfavorable consequences (8-11). Previous research has indicated a negative relationship between LS7 and liver fibrosis, with a higher LS7 score related to a decreased risk of liver fibrosis (12-14). However, all of these studies excluded persons with hepatitis B, hepatitis C, and significant alcohol consumption. It is uncertain whether the recently disclosed LE8 connects with liver fibrosis.

To explore the hypothesis of an association between updated CVH and liver fibrosis, we used data from the 2017-2018 National Health and Nutrition Examination Survey (NHANES), a continuous cycle, for this study.

MATERIALS AND METHODS

Data source and study population

The data that we used came from the NHANES 2017-2018 cycle. NHANES is a national cross-racial comprehensive survey study approved by the National Center for Health Statistics Ethics Review Board that includes interviews, standardized examinations, and biospecimen collection; the survey is nationally representative (15). All those surveyed have submitted informed permission forms. The NHANES official webpage contains relevant data information.

During the current cycle, a total of 9,254 respondents completed the survey. Previous research has revealed that CVH indicators for pregnant women need to be changed by pregnancy recommendations, which have yet to be standardized (16). In the beginning, 5,493 participants with missing CVH data and pregnant women were excluded, and the remaining 3,761 participants aged 20-80 years. One hundred fifty-two patients were eliminated because they did not have liver stiffness measurement (LSM), and a total of 3,609 subjects were eventually included in the study (Fig. 1).

LSM and liver fibrosis

In the current study, liver fibrosis was utilized as an outcome variable. The current gold standard for diagnosing liver fibrosis is liver biopsy, which is limited in its widespread use due to its invasive nature, and noninvasive approaches for measuring hepatic fibrosis are now routinely employed in clinical practice (17). A widely used technique in clinical and prospective investigations for evaluating liver cirrhosis and fibrosis is vibration-controlled elastography (VCTE) (18). The FibroScan® model 502 V2 Touch was used to collect data on LSM from all responders. The equipment is calibrated annually, and workers are

educated and qualified, ensuring accurate data. Based on 2021 revised European standards, LSM < 8 kPa may rule out patients with advanced liver fibrosis, but the threshold for distinguishing between liver fibrosis and cirrhosis remains unclear, and the criteria for determining cirrhosis due to different etiologies are inconsistent (19). Previous studies have also set the LSM cutoff at 8 kPa for screening patients with liver fibrosis (13). To account for the above, the continuous variable of LSM was separated into two groups using 8 kPa as a threshold, with LSM < 8 kPa omitting liver fibrosis and LSM ≥ 8 kPa including liver fibrosis.

LE8 Metrics

The LE8 is made up of eight health-related indicators: diet, sleep health, physical activity (PA), nicotine exposure (all of which are referred to as health behaviors), body mass index (BMI), blood pressure (BP), blood lipids, and blood glucose (all of them are referred to as health factors) (8). To determine the overall CVH score, the unweighted mean of eight health markers was employed (8). Diet score could be calculated from the Dietary Approaches to Stop Hypertension (DASH) diet score, sleep health, PA, and nicotine exposure scores from the NHANES questionnaire, BMI (weight divided by height squared) and BP from the physical examination, and fasting glucose, glycosylated hemoglobin, total cholesterol, and high-density lipoproteins (HDL) could be collected from laboratory test data to calculate blood glucose and blood lipids scores. Prior research included the complete method (8,20,21). CVH was classified as high (80-100), moderate (50-79), and low (0-49) by the American Heart Association's presidential advisory (8). To further study the association between CVH subcomponents and liver fibrosis, we

employed the same strategy to stratify the 8 sub-scores.

Covariates

As covariates, we used basic demographic information from the NHANES, such as gender, age, race, education level, The ratio of family income to poverty (PIR), and marital status. Age and PIR were continuous variables in the baseline table, while marital status was taken into account as a categorical variable in two groups. Based on high school graduation, the education level is split into three categories. Race used the same style of grouping as the NHANES. At the 60-year threshold, age was split into two groups in the subgroup analysis, while PIR was split into three groups: < 1.3, 1.3-3.5, and > 3.5. Education level and marital status subgroups were the same as in the baseline table.

Statistical analysis

Software R (version 4.1.3) and EmpowerStats (version 2.0) were utilized to do all computations, and p values < 0.05 were determined to be statistically significant. The statistical process was as follows: First, the baseline characteristics of every participant were compared. Grouping by total CVH level investigated variations in all covariates, LE8 sub-scores, LSM, and liver fibrosis among categories. T-tests (mean ± standard deviation) were applied for evaluating continuous variables, while chi-square tests [number (%)] were employed to test categorical variables. Second, various regression equations were employed to understand the link between the total CVH/sub-scores and liver fibrosis/LSM, using multiple linear regression [β (95 % confidence intervals, CI)] when LSM was the dependent variable and multiple logistic regression [odds ratios, OR (95 % CI)] while liver

fibrosis was the outcome variable. The nonlinear connection between the independent and dependent variables was further investigated using smoothed curve fitting. Finally, the robustness of the association between liver fibrosis and CVH when variables were taken into consideration was investigated using subgroup analysis.

RESULTS

Baseline characteristics

The fundamental traits of the individuals are listed in table I. There were 3,609 people in total, with an approximate mean age of 51, 1,786 (49.49 %) males and 1,823 (50.51 %) females, 431 people (11.94 %) were identified with liver fibrosis, 807 (22.36 %) in the low-level CVH group (LE8 < 50), 2,277 (63.09 %) in the moderate-level CVH group (50 ≤ LE8 < 80), and 525 (14.55 %) in the high-level CVH group (LE8 ≥ 80). The results revealed that the high-level group was more likely to be female, younger, have a high family income, and be highly educated. In contrast, the low-level group was more likely to be Mexican-American with a lower CVH score, higher LSM, and higher risk of hepatic fibrosis. Marital status did not differ significantly between the groups.

Association between LE8 and LSM

Table II shows the multiple linear regression associations of CVH and 8 health markers with LSM. In all three models, CVH and LSM had a substantial negative connection. The fully adjusted model showed that compared to the low level of CVH, the moderate level of CVH was associated with a 1.6 KPa decrease in LSM [95 % CI (-2.06, -1.15)]; the high level of CVH showed a higher inversed association with LSM [β = -2.37, 95 % CI (-3.03, -1.70)]. Meanwhile, in all three models, there

was a negative correlation between diet score, PA score, BMI score, blood glucose score, BP score, and LSM, with LSM not statistically different between the low and moderate levels of diet and PA scores, and LSM decreasing with increasing scores for the remaining four health indicators. There was no association between sleep health score, nicotine exposure score, and LSM, and only in unadjusted models did LSM and blood lipids score exhibit a negative correlation [β = -0.01, 95 % CI (-0.01, -0.00)]. As illustrated in figure 2A, a nonlinear, smooth curve fitting of the CVH and LSM was conducted, and a negative correlation relationship between the two could be noticed.

Association between LE8 and liver fibrosis

From table III, it could be observed that in unadjusted, partially adjusted, and fully adjusted models, the multivariate logistic regression showed a significant negative correlation between liver fibrosis and CVH, with ORs and 95 % CIs of [0.96 (0.95, 0.96)], [0.96 (0.95, 0.96)], and [0.96 (0.95, 0.97)], respectively. After adjusting for all covariates, the prevalence of liver fibrosis was 53 % [OR = 0.47, 95 % CI (0.38, 0.59)] and 90 % [OR = 0.10, 95 % CI (0.05, 0.19)] lower in participants with moderate and high levels of CVH, respectively, compared with those lower CVH. Meanwhile, there was a statistically significant inverse relationship between diet, PA, BMI, blood glucose, and BP scores with liver fibrosis. Notably, at low to moderate diet and PA score levels, the odds of developing hepatic fibrosis were not substantially different across all models ($p > 0.05$). Additionally, we failed to identify any relationship between nicotine exposure score, sleep health score, and blood lipids score with liver fibrosis. Figure 2B depicted a smoothed curve fit between CVH and

hepatic fibrosis, revealing yet another negative association between CVH and liver fibrosis.

Subgroup analysis

We did a subgroup analysis to determine if the association of LSM and liver fibrosis with CVH was consistent across different dimensions of the population. Table IV demonstrated that CVH and LSM have a strong non-positive connection for all ages, genders, races, education levels, marital status, and PIR, with no statistically significant differences across strata. Similarly, a substantial inverse relationship existed between CVH and the incidence of liver fibrosis in all subgroups (Table V). Furthermore, when comparing participants under 60 to those over 60, we found a stronger negative correlation between CVH and the incidence of liver fibrosis (p for interaction = 0.022).

DISCUSSION

For a cross-sectional investigation, we used the adult population of the United States from the NHANES database. We discovered a strong negative connection between the incidence of liver fibrosis and CVH/CVH sub-scores (along with nicotine exposure, sleep health, and blood lipid scores). The high CVH group showed the strongest negative correlation with liver fibrosis in the fully adjusted model. Notably, after accomplishing stratified analyses, we found a stronger correlation between CVH and the incidence of hepatic fibrosis in U.S. people below the age of 60.

To our knowledge, this is the first study to investigate the link between LE8 and liver fibrosis. The original CVH criteria, LS7, have yet to be well-researched for their relationship to liver fibrosis**.** A

cohort study from China, which comprised a middle-aged and older population without fatty liver at baseline, found that intermediate and ideal LS7 levels were substantially and inversely related to nonalcoholic fatty liver fibrosis (12). In another cohort research of Korean people, greater LS7 scores were associated with a lower risk of hepatic fibrosis and non-alcoholic fatty liver disease (NAFLD) (13). In an NHANES research, the LS7 score was adversely linked with the likelihood of advanced liver fibrosis and cirrhosis (14). The results above supported our findings that the overall CVH score was adversely connected with liver fibrosis, and the connection was more significant with higher scores. Compared to LS7, LE8 follows the perception and definition of cardiovascular health-related indicators in the contemporary scenario, breaking the constraints of prior quantitative indicators to better capture intra-individual variability and inter-population variances (8). In addition to the total CVH score, we further evaluated the relationship between eight health metrics and hepatic fibrosis, discovering that higher levels of BP, blood glucose, BMI, and lower diet and PA scores were risk factors for the development of advanced hepatic fibrosis, which agreed with the results of earlier research (22,23). Interestingly, the association of diet and PA scores with liver fibrosis was revealed in the high-scoring group, showing that stronger standards of health practices are required to regress hepatic fibrosis. Furthermore, there was no statistically significant correlation seen between blood lipids and hepatic fibrosis, contradicting previous research (24). Our study did not distinguish between cirrhosis and hepatic fibrosis, and non-high-density lipoprotein cholesterol was an indirect index of cholesterol minus HDL, which may explain the lack of correlation between the two. Previous research has found a relationship between sleep health,

nicotine exposure, and liver fibrosis (25,26). Still, the link was insignificant in this study, most likely because both data sources were questionnaires with memory bias. In the subgroup analysis, it is significant to take into account that the relationship between CVH and liver fibrosis changed with age. Liver fibrosis and CVH had a more substantial negative correlation in young and middle-aged individuals, showing that there may be greater benefit from initiating health management at a younger age.

The connection between LE8 and liver fibrosis has not been elucidated. Yet, the CVH subcomponents are all metabolically connected, and there is evidence that metabolic problems contribute to the development of chronic liver disease into liver fibrosis. Two large retrospective cohort studies found that metabolic abnormalities were independently related to worse outcomes in hepatitis B, even in the presence of viral suppression and the absence of cirrhosis. In contrast, Hepatic steatosis alone did not increase the probability of negative consequences, nor did the absence of metabolic abnormalities (27,28). Hepatitis C patients were more likely to have simultaneous hepatic lipid deposition, leading to lower efficacy of antiviral medication, increased hepatic fibrosis, and an increased risk of metabolic syndrome and cardiovascular disease with hepatitis C virus infection (29,30). A cohort study of 863 individuals with NAFLD found a substantial connection between liver fibrosis and visceral adiposity, blood glucose, and lipids (31). Age, BMI, blood glucose, and alcoholic liver fibrosis all demonstrated independent positive relationships (32). At the same time, cardiovascular disease and liver fibrosis have a bidirectional link. A meta-analysis of 36 longitudinal studies found a significantly increased risk of fatal and nonfatal adverse cardiovascular events in the fibrotic phase of liver fibrosis

compared to the nonfibrotic population, and the correlation was demonstrated in two prospective cohort studies with 898 and 10,422 participants, respectively (6,33,34). A Framingham Heart Study community-based cohort research with 3,276 adult participants used VCTE as the method for determining liver fibrosis and LSM = 8.2 kPa as the threshold for liver fibrosis; they discovered that liver fibrosis and cardiovascular disease risk variables (obesity, metabolic syndrome, hypertension, diabetes mellitus, and HDL) were substantially linked with one other, even after controlling for the variable (22). It has been reported that low-grade systemic inflammation stimulates the release of several cytokines (e.g., interleukin-1, interleukin-6, interleukin-8, tumor necrosis factor-α), oxidative stress, and changes in gut microbiology (trimethylamine N-oxide), all of which may be implicated in the development of CVD (35,36). Similarly, pro-inflammatory factor release, reactive oxygen species generation, and alterations in intestinal flora can activate hepatic stellate cells, leading to hepatic fibrosis (37). Based on these findings, it is understandable that there is a link between LE8 as a screening and preventative tool for cardiovascular disease and liver fibrosis. Lower CVH scores should alert people to the danger of liver fibrosis. In contrast, higher CVH targets are expected to minimize or even reverse liver fibrosis and reduce the global public health burden.

This study has several flaws: First, the NHANES questionnaire was used to assess health behaviors, and the results might need to be corrected owing to memory bias. Second, VCTE has received widespread attention and is used as a noninvasive method for detecting the extent of liver tissue damage (38-40). However, liver biopsy remains the only gold standard for testing the degree of progression of liver disease. The rate of correct VCTE judgment is

lower than that of liver biopsy. There may be misclassification during the screening process using VCTE, which may affect the accuracy of the results. Third, even though we incorporated important factors to tweak the model, some confounders may still need to be protected and interfere with the results. Fourth, a portion of our study inclusion population belongs to Hispanics, who typically have a higher-than-average prevalence of hepatic steatosis. Despite our subgroup analyses, differences in incidence may still introduce bias in the results. Therefore, the general applicability of the study needs to be further enhanced in the future through multicenter international collaborations, broader subject recruitment, and more in-depth stratified analyses. In the end, though our sample size was large enough to represent the adult population in the United States, based on cross-sectional characteristics, further prospective research is required to confirm the causal link between LE8 and liver fibrosis.

CONCLUSIONS

Our research revealed a strong inverse association between liver fibrosis and LE8 and its subcomponents, particularly in young and middle-aged individuals under the age of 60. Our results suggest that maintaining good CVH levels may be of public health importance in reducing the prevalence of liver stiffness and cirrhosis.

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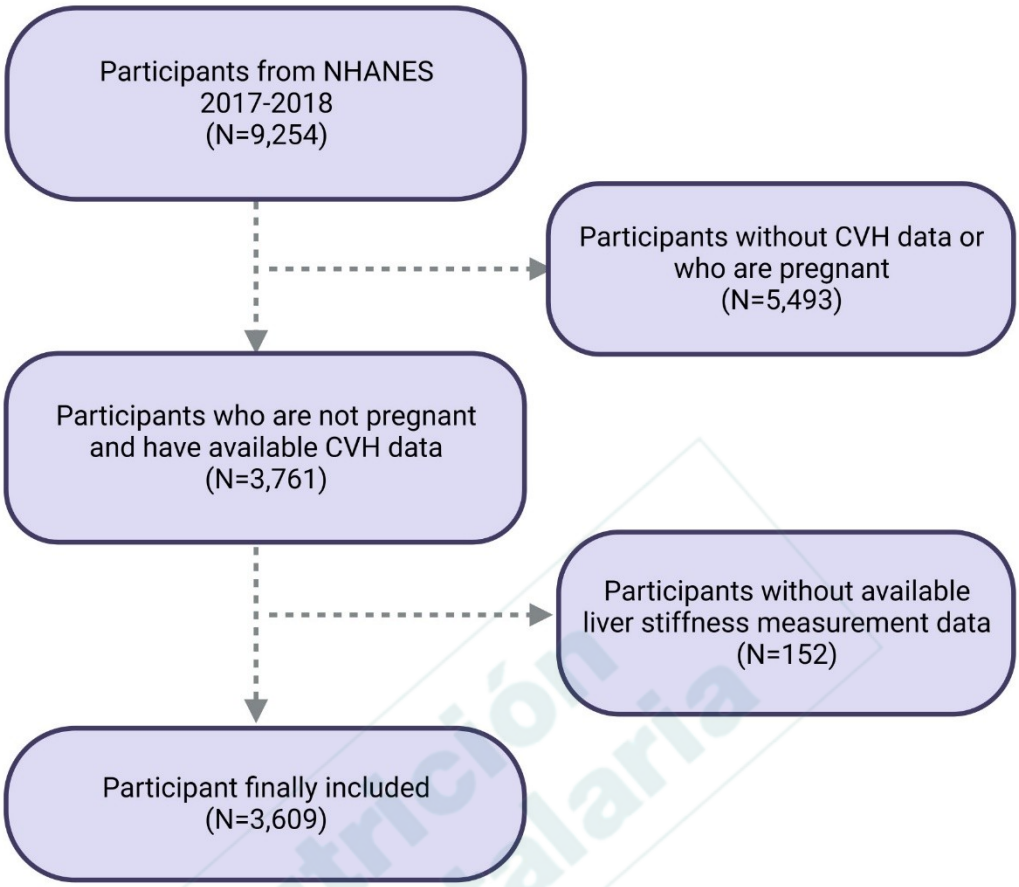

Figure 1. Flow chart of participants selection. NHANES: National Health and Nutrition Examination Survey; CVH: cardiovascular health.

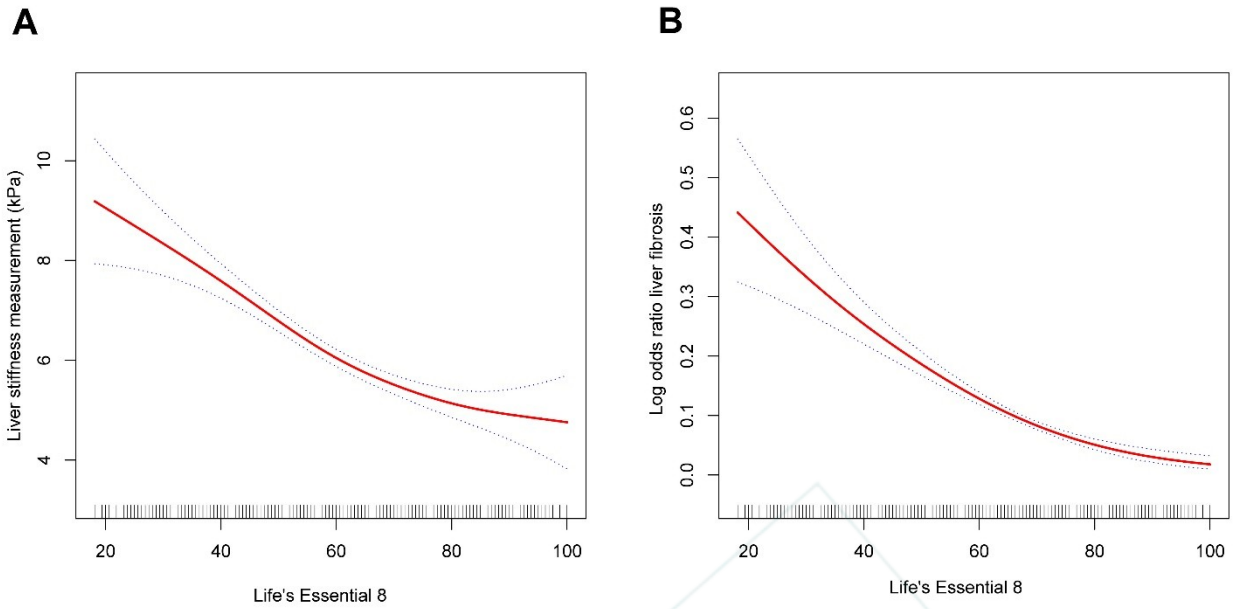

Figure 2. The solid red line represents the smooth curve fit between variables. Blue bands represent the 95 % confidence interval from the fit. A. Life's Essential 8 and liver stiffness measurement. B. Life's Essential 8 and liver fibrosis.

Table I. Baseline characteristics of participants with different CVH levels estimated from the LE8 score

Characteristics	**Low (LE8 < 50)**	**Moderate (LE8 50–79)**	**High (LE8 ≥ 80)**	***p*-value**
No. of participants in sample	807	2277	525	
Age, yrs (SD)	55.49 ± 14.88	51.32 ± 17.39	41.42 ± 16.52	< 0.001
PIR	2.20 ± 1.48	2.61 ± 1.61	3.23 ± 1.62	< 0.001
Gender, n (%)				< 0.001
Male	438 (54.28)	1140 (50.07)	208 (39.62)	
Female	369 (45.72)	1137 (49.93)	317 (60.38)	
Race/Ethnicity, n (%)				< 0.001
Non-Hispanic White	106 (13.14)	302 (13.26)	66 (12.57)	
Non-Hispanic Black	57 (7.06)	214 (9.40)	50 (9.52)	
Mexican American	351 (43.49)	831 (36.50)	179 (34.10)	
Other Hispanic	204 (25.28)	522 (22.92)	66 (12.57)	
Others	89 (11.03)	408 (17.92)	164 (31.24)	
Education level, n (%)				< 0.001
Less than high school	191 (23.67)	396 (17.39)	36 (6.86)	

High school	245 (30.36)	541 (23.76)	82 (15.62)	
More than high school	371 (45.97)	1340 (58.85)	407 (77.52)	
Marital status, n (%)				0.098
Married/living with partner	463 (57.37)	1370 (60.17)	332 (63.24)	
Others	344 (42.63)	907 (39.83)	193 (36.76)	
AHA LE8 score (SD)				
Mean total CVH score	41.65 ± 6.60	63.71 ± 8.16	86.71 ± 5.23	< 0.001
Mean DASH diet score	24.75 ± 26.51	43.03 ± 31.72	65.67 ± 29.88	< 0.001
Mean physical activity score	9.64 ± 27.05	45.67 ± 46.97	90.82 ± 24.06	< 0.001
Mean nicotine exposure score	47.38 ± 39.70	70.19 ± 36.94	92.02 ± 18.07	< 0.001
Mean sleep health score	69.38 ± 30.76	83.76 ± 23.56	92.38 ± 15.23	< 0.001
Mean body mass index score	32.41 ± 29.19	58.65 ± 32.53	84.44 ± 22.01	< 0.001
Mean blood lipids score	50.78 ± 29.36	68.13 ± 28.93	86.55 ± 21.82	< 0.001
Mean blood glucose score	58.84 ± 29.77	79.24 ± 26.24	95.54 ± 13.49	< 0.001
Mean blood pressure score	40.04 ± 28.72	60.98 ± 32.30	86.25 ± 22.15	< 0.001
LSM, kPa (SD)	7.61 ± 8.47	5.88 ± 4.71	4.77 ± 2.93	< 0.001
Liver fibrosis, *n* (%)	172 (21.31)	248 (10.89)	11 (2.10)	< 0.001

Mean (SD) for continuous variables: the *p* value was calculated by the weighted linear regression model. Percentages for categorical variables: the *p* value was calculated by the weighted chi-square test. Cardiovascular

health (CVH) is categorized into 3 grades, low: LE8 score < 50, medium: 50 ≤ LE8 score < 80, high: LE8 score ≥ 80. AHA: American Heart Association; LE8: Life's Essential 8; CVH: cardiovascular health; DASH: Dietary Approaches to Stop Hypertension; PIR: the ratio of family income to poverty; LSM: liver stiffness measurement.

Table II. Linear regression model between Total/Subclass CVH score and LSM

LSM, kPa	**Model 1 [β (95 % CI)]**	**Model 2 [β (95 % CI)]**	**Model 3 [β (95 % CI)]**
Total CVH score	-0.06 (-0.08, -0.05)‡	-0.06 (-0.07, -0.05)‡	-0.06 (-0.07, -0.04)‡
CVH categories			
Low (LE8 < 50)	0 (reference)	0 (reference)	0 (reference)
Moderate (50 ≤ LE8 < 80)	-1.73 (-2.18, -1.28)‡	-1.62 (-2.07, -1.17)‡	-1.60 (-2.06, -1.15)‡
High (LE8 ≥ 80)	-2.84 (-3.46, -2.23)‡	-2.47 (-3.12, -1.83)‡	-2.37 (-3.03, -1.70)‡
Subclass CVH scores			
DASH diet score	-0.01 (-0.01, -0.00)*	-0.01 (-0.01, -0.00)†	-0.01 (-0.01, -0.00)*
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	0.00 (-0.47, 0.47)	-0.02 (-0.49, 0.45)	0.03 (-0.44, 0.50)
High (80-100)	-0.51 (-0.94, -0.08)*	-0.62 (-1.07, -0.18)†	-0.52 (-0.97, -0.07)*
Physical activity score	-0.01 (-0.01, -0.00)‡	-0.01 (-0.01, -0.00)‡	-0.01 (-0.01, -0.00)†
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	-0.05 (-1.03, 0.92)	0.10 (-0.87, 1.07)	0.12 (-0.86, 1.09)
High (80-100)	-0.91 (-1.29, -0.53)‡	-0.81 (-1.19, -0.43)‡	-0.74 (-1.14, -0.34)‡
Tobacco exposure score	-0.00 (-0.01, 0.00)	-0.00 (-0.01, 0.00)	0.00 (-0.00, 0.01)
Low (0-49)	0 (reference)	0 (reference)	0 (reference)

Moderate (50-79)	0.70 (0.15, 1.25)*	0.41 (-0.17, 0.98)	0.56 (-0.02, 1.14)
High (80-100)	-0.25 (-0.69, 0.18)	-0.09 (-0.54, 0.36)	0.15 (-0.32, 0.62)
Sleep health score	-0.00 (-0.01, 0.01)	0.00 (-0.01, 0.01)	0.00 (-0.00, 0.01)
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	0.12 (-0.52, 0.75)	0.14 (-0.49, 0.77)	0.24 (-0.40, 0.87)
High (80-100)	-0.08 (-0.57, 0.41)	0.01 (-0.48, 0.50)	0.12 (-0.37, 0.62)
Body mass index score	-0.03 (-0.04, -0.03)‡	-0.03 (-0.04, -0.03)‡	-0.03 (-0.04, -0.03)‡
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	-1.70 (-2.13, -1.28)‡	-1.89 (-2.32, -1.46)‡	-1.86 (-2.29, -1.42)‡
High (80-100)	-2.26 (-2.72, -1.81)‡	-2.21 (-2.67, -1.74)‡	-2.18 (-2.65, -1.72)‡
Blood lipid score	-0.01 (-0.01, -0.00)*	-0.00 (-0.01, 0.00)	-0.00 (-0.01, 0.00)
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	-0.25 (-0.76, 0.27)	-0.06 (-0.58, 0.46)	-0.02 (-0.53, 0.50)
High (80-100)	-0.34 (-0.77, 0.09)	-0.18 (-0.61, 0.25)	-0.15 (-0.59, 0.28)
Blood glucose score	-0.03 (-0.04, -0.02)‡	-0.03 (-0.03, -0.02)‡	-0.03 (-0.03, -0.02)‡
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	-1.61 (-2.18, -1.04)‡	-1.57 (-2.14, -0.99)‡	-1.58 (-2.15, -1.00)‡
High (80-100)	-2.46 (-2.98, -1.93)‡	-2.30 (-2.87, -1.74)‡	-2.30 (-2.86, -1.73)‡

Blood pressure score	-0.02 (-0.03, -0.01)‡	-0.02 (-0.02, -0.01) ‡	-0.02 (-0.02, -0.01)‡
Low (0-49)	0 (reference)	0 (reference)	0 (reference)
Moderate (50-79)	-0.82 (-1.27, -0.36)‡	-0.70 (-1.18, -0.22)†	-0.67 (-1.14, -0.19)†
High (80-100)	-1.64 (-2.09, -1.18)‡	-1.31 (-1.82, -0.80)‡	-1.24 (-1.76, -0.73)‡

Data are presented as β, 95 % CI (confidence intervals), and *p*-value. *$p < 0.05$, †$p < 0.01$, ‡$p < 0.001$. Model 1 was unadjusted for covariates; Model 2 adjusted for including age, sex, and race; and Model augmented Model 2 integrating education level, family income-to-poverty ratio, and cardiovascular health; LSM: liver stiffness measurement; DASH: Dietary Approaches to Stop Hypertension.

Table III. Logistic regression model between total/subclass CVH score and liver fibrosis

Liver fibrosis (≥ 8.0 kPa)	**Model 1 [OR (95 % CI)]**	**Model 2 [OR (95 % CI)]**	**Model 3 [OR (95 % CI)]**
Total CVH score	0.96 (0.95, 0.96)‡	0.96 (0.95, 0.96)‡	0.96 (0.95, 0.97)‡
CVH categories			
Low (LE8 < 50)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50 ≤ LE8 < 80)	0.45 (0.36, 0.56)‡	0.48 (0.38, 0.59)‡	0.47 (0.38, 0.59)‡
High (LE8 ≥ 80)	0.08 (0.04, 0.15)‡	0.10 (0.05, 0.19)‡	0.10 (0.05, 0.19)‡
Subclass CVH scores			
DASH diet score	1.00 (0.99, 1.00)†	0.99 (0.99, 1.00)‡	0.99 (0.99, 1.00)†
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	1.17 (0.92, 1.49)	1.13 (0.88, 1.44)	1.13 (0.88, 1.45)
High (80-100)	0.74 (0.57, 0.95)*	0.65 (0.50, 0.84)†	0.67 (0.52, 0.87)†
Physical activity score	0.99 (0.99, 1.00)‡	1.00 (0.99, 1.00)‡	1.00 (0.99, 1.00)‡
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.90 (0.54, 1.50)	1.02 (0.61, 1.71)	1.04 (0.62, 1.75)
High (80-100)	0.58 (0.46, 0.72)‡	0.64 (0.51, 0.80)‡	0.65 (0.52, 0.82)‡
Tobacco exposure score	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)

Moderate (50-79)	1.56 (1.17, 2.06)†	1.19 (0.88, 1.60)	1.24 (0.92, 1.68)
High (80-100)	0.95 (0.74, 1.21)	0.98 (0.76, 1.26)	1.05 (0.81, 1.38)
Sleep health score	1.00 (0.99, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.98 (0.70, 1.37)	1.02 (0.73, 1.43)	1.04 (0.74, 1.47)
High (80-100)	0.86 (0.66, 1.11)	0.89 (0.69, 1.16)	0.93 (0.71, 1.21)
Body mass index score	0.98 (0.97, 0.98)‡	0.97 (0.97, 0.98)‡	0.97 (0.97, 0.98)‡
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.27 (0.21, 0.36)‡	0.23 (0.18, 0.31)‡	0.23 (0.18, 0.31)‡
High (80-100)	0.23 (0.17, 0.31)‡	0.23 (0.16, 0.31)‡	0.23 (0.16, 0.31)‡
Blood lipid score	1.00 (0.99, 1.00)	1.00 (1.00, 1.00)	1.00 (1.00, 1.00)
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.84 (0.63, 1.11)	0.94 (0.71, 1.25)	0.96 (0.72, 1.27)
High (80-100)	0.88 (0.70, 1.10)	0.95 (0.75, 1.20)	0.96 (0.76, 1.22)
Blood glucose score	0.98 (0.98, 0.98)‡	0.98 (0.98, 0.99)‡	0.98 (0.98, 0.99)‡
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.48 (0.37, 0.62)‡	0.50 (0.38, 0.65)‡	0.49 (0.38, 0.64)‡
High (80-100)	0.22 (0.17, 0.29)‡	0.26 (0.19, 0.34)‡	0.26 (0.19, 0.34)‡

Blood pressure score	0.99 (0.98, 0.99)‡	0.99 (0.99, 0.99)‡	0.99 (0.99, 0.99)‡
Low (0-49)	1 (Reference)	1 (Reference)	1 (Reference)
Moderate (50-79)	0.58 (0.46, 0.73)‡	0.65 (0.51, 0.82)‡	0.65 (0.51, 0.83)‡
High (80-100)	0.31 (0.24, 0.40)‡	0.40 (0.30, 0.53)‡	0.41 (0.30, 0.55)‡

Data are presented as odds ratios, 95% CI (confidence intervals), and *p*-value. *$p < 0.05$, †$p < 0.01$, ‡$p < 0.001$. Model 1 was unadjusted for covariates; Model 2 enhanced Model 1 by including age, sex, and ethnicity; and Model 3 further augmented Model 2 by integrating education level, family income-to-poverty ratio, marital status, and CVH: cardiovascular health; LSM: liver stiffness measurement; DASH: Dietary Approaches to Stop Hypertension.

Table IV. Subgroup analysis of the association between CVH and LSM

Subgroup	**LSM [β (95 % CI)]**	***p* for interaction**
Gender		0.210
Male	-0.07 (-0.08, -0.05)	
Female	-0.05 (-0.07, -0.03)	
Age		0.100
< 60 years	-0.07 (-0.08, -0.05)	
≥ 60 years	-0.04 (-0.07, -0.02)	
Race/ethnicity		0.681
Non-Hispanic White	-0.05 (-0.08, -0.01)	
Non-Hispanic Black	-0.08 (-0.12, -0.04)	
Mexican American	-0.06 (-0.08, -0.04)	
Other Hispanic	-0.05 (-0.08, -0.02)	
Others	-0.05 (-0.08, -0.02)	
Education level		0.526
Less than high school	-0.06 (-0.09, -0.03)	
High school	-0.05 (-0.07, -0.02)	
More than high school	-0.06 (-0.08, -0.05)	
Marital status		0.426
Married/living with partner	-0.06 (-0.08, -0.05)	
Others	-0.05 (-0.07, -0.03)	
Poverty ratio		0.363
< 1.3	-0.05 (-0.07, -0.02)	
1.3-3.5	-0.07 (-0.09, -0.05)	

> 3.5	-0.06 (-0.08, -0.04)	

Gender subgroup: adjusting age, race, education level, marital status, and poverty ratio.

Age subgroup: adjusting gender, race, education level, marital status, and poverty ratio.

Race/ethnicity subgroup: adjusting age, gender, education level, marital status, and poverty ratio. Education level subgroup: adjusting age, gender, race, marital status, and poverty ratio. Marital status subgroup: adjusting age, gender, race, education level, and poverty ratio. Poverty ratio subgroup: adjusting age, gender, race, education level, and marital status. CVH: cardiovascular health; LSM: liver stiffness measurement; CI: confidence interval.

Table V: Subgroup analysis of the association between CVH and liver fibrosis

Subgroup	**Liver fibrosis [OR (95 % CI)]**	***p* for interaction**
Gender		*0.993*
Male	0.96 (0.95, 0.97)	
Female	0.96 (0.95, 0.97)	
Age		*0.022*
< 60 years	0.95 (0.94, 0.96)	
≥ 60 years	0.97 (0.96, 0.98)	
Race/ethnicity		*0.542*
Non-Hispanic White	0.95 (0.93, 0.97)	
Non-Hispanic Black	0.95 (0.92, 0.97)	
Mexican American	0.96 (0.95, 0.97)	
Other Hispanic	0.97 (0.95, 0.98)	
Others	0.96 (0.94, 0.98)	
Education level		*0.419*
Less than high school	0.96 (0.94, 0.97)	
High school	0.97 (0.95, 0.98)	
More than high school	0.95 (0.94, 0.96)	
Marital status		*0.137*
Married/living with partner	0.95 (0.94, 0.96)	
Others	0.96 (0.95, 0.98)	
Poverty ratio		*0.739*

< 1.3	0.96 (0.95, 0.97)	
1.3-3.5	0.96 (0.95, 0.97)	
> 3.5	0.95 (0.94, 0.97)	

Gender subgroup: adjusting age, race, education level, marital status, and poverty ratio.

Age subgroup: adjusting gender, race, education level, marital status, and poverty ratio.

Race/ethnicity subgroup: adjusting age, gender, education level, marital status, and poverty ratio. Education level subgroup: adjusting age, gender, race, marital status, and poverty ratio. Marital status subgroup: adjusting age, gender, race, education level, and poverty ratio. Poverty ratio subgroup: adjusting age, gender, race, education level, and marital status. CVH: cardiovascular health; OR: odds ratio; CI: confidence interval.