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Association between cardiovascular health and metabolic dysfunction-associated steatotic liver disease: a nationwide crosssectional study

Lian-Zhen Huang^{1†}, Ze-Bin Ni^{1,2†}, Wei-Feng Huang¹, Li-Ping Sheng¹, Yan-Qing Wang^{3*†} and Jin-Yan Zhang^{1,2*†}

Abstract

Purpose Evidence concerning the effect of cardiovascular health (CVH) on the risk of metabolic dysfunctionalassociated steatotic liver disease (MASLD) is scarce. This study aimed to investigate the association between CVH and MASLD.

Methods 5680 adults aged ≥ 20 years from the National Health and Nutrition Examination Survey 2017-March 2020 were included. Life's essential 8 (LE8) was applied to assess CVH. Weighted binary logistic regression was employed to calculate the odds ratio (OR) and 95% confidence interval (CI) to investigate the association of CVH with MASLD. Restricted cubic spline (RCS) was conducted to explore the dose-response association between LE8 and its subscales scores with MASLD.

Results Among 5680 participants, 724, 3901, and 1055 had low, moderate, and high CVH levels, respectively, with a MASLD diagnosis prevalence of 36.83%. In the fully adjusted logistic regression model, ORs for MASLD were 0.50 (95% CI, 0.37–0.69) for participants with moderate CVH and 0.21 (95% CI, 0.13–0.34) for those with high CVH, when compared to those with low CVH (P < 0.001 for trend). OR for MASLD was 0.68 (95% CI, 0.61–0.77) for each 10-point increase in LE8 score. RCS model demonstrated a non-linear dose-response relationship between LE8 score and health factors score with MASLD, while a linear relationship was found between health behaviors score and MASLD. Subgroup analysis showed a consistent negative correlation between LE8 score and MASLD, and sensitivity analysis validated the reliability of these findings.

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Conclusions Higher LE8 score was associated with a lower risk of MASLD. Encouraging adherence to optimal CVH levels may help mitigate the burden of MASLD.

Keywords Cardiovascular health, NHANES, Life's essential 8, MASLD

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD), a newly proposed term to replace nonalcoholic fatty liver disease (NAFLD) [1], represents a progressive spectrum of conditions, including simple steatosis, metabolic dysfunction-associated steatohepatitis (MASH), liver fibrosis, and potentially cirrhosis and hepatocellular carcinoma (HCC) [2, 3]. Over the past several decades, both the incidence and prevalence of MASLD have dramatically increased, emerging as a significant health issue globally. According to recent studies, more than 30% of the adult population worldwide are affected by MASLD [4-7]. MASLD is considered a hepatic manifestation of a constellation of diseases related to systemic metabolic dysfunction, including hypertension, insulin resistance, diabetes, hyperlipidemia, and obesity, all of which are acknowledged risk factors for cardiovascular disease (CVD) [8–11]. A growing body of evidence suggests a relationship between the presence of MASLD and a higher risk of CVD [12–14]. Although the Food and Drug Administration (FDA) has recently approved resmetirom as the only drug for MASH treatment [15], practicing and maintaining a healthy lifestyle such as dietary adjustments, weight control, and increased physical activity remains crucial for effective management [16-22].

In 2022, the American Heart Association (AHA) introduced a new quantification algorithm for cardiovascular health (CVH), known as the "Life's Essential 8 (LE8)" score, building upon the previously established "Life's Simple 7 (LS7)" score [23]. The LE8 score updated the definitions and scoring of the previous 7 components (diet, physical activity, nicotine exposure, body mass index [BMI], blood lipids, blood glucose, and blood pressure) and added a sleep health component. Compared to LS7, the LE8 score is assessed on a scale of 0 to 100, making it more easily understandable, improving the quantification of individual CVH, and increasing sensitivity to measuring CVH changes over time at both individual and population levels [24]. Since its release, LE8 score has not only been utilized in CVD prevention and management but have also shown promise in assessing non-CVD conditions such as osteoporosis, cognitive function, chronic kidney disease, and kidney stones [25-28]. Given substantial evidence indicating that MASLD shares many risk factors with CVD and is closely related to CVD, LE8 may serve as a promising tool for assessing MASLD risk [29, 30]. It is imperative to urgently evaluate the comprehensive effects of introducing and applying the LE8 concept on the burden of MASLD within the general population. However, there is currently limited research investigating the relationship between CVH and MASLD. Therefore, this study aims to assess the association between CVH, as measured by LE8 score, and MASLD, utilizing the latest available National Health and Nutrition Examination Survey (NHANES) data.

Materials and methods

Study design and participants

NHANES, an ongoing series of nationally representative surveys conducted biennially, is dedicated to monitoring the nutritional and health status of the non-institutionalized US civilian population. Utilizing a sophisticated probability multi-stage sampling design, NHANES ensures the accuracy of estimates. Due to the COVID-19 pandemic, survey operations were halted in March 2020. Consequently, data collected from 2019 to March 2020 was merged with that from the 2017–2018 cycle, yielding the NHANES 2017-2020 pre-pandemic dataset. Ethics Review Committee of the National Center for Health Statistics approved the original survey protocol, and written informed consent was obtained from all participants. Given the use of publicly available and de-identified NHANES datasets, the current analysis does not necessitate the approval and informed consent of the Institutional Review Board. This study follows the reporting guidelines of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) [31].

We opted for the NHANES cycle from 2017 to March 2020 due to its exclusive availability of data on liver ultrasound vibration-controlled transient elastography (VCTE). The detailed study flowchart is depicted in Fig. 1. Among the initial 15 560 participants, we excluded individuals based on the following criteria: (1) under 20 years old (n = 6328), (2) missing controlled attenuation parameter (CAP) values from hepatic VCTE assessments (n = 689), (3) ineligible (n = 333), not done (n = 222), or partial (n = 593) VCTE examinations, (4) excessive alcohol consumption, defined as more than three standard alcoholic drinks daily for males or two for females (n = 224), (5) missing data on CVH metrics (n = 1418), and (6) missing partial covariates data (n=73), which included 58 missing aspartate aminotransferase (AST) values, 8 on thyroid disease history, 5 on education levels, and 2 on marital status. Ultimately, 5680 participants were included in the final analysis.



Fig. 1 Flowchart of participant selection. NHANES, National Health and Nutrition Examination Survey; VCTE, vibration controlled transient elastography; CAP, controlled attenuation parameter; CVH, cardiovascular health; AST, aspartate aminotransferase; MASLD, metabolic dysfunction-associated steatotic liver disease

Assessment of CVH

LE8 score was applied to assess CVH, comprising four health behaviors (diet, physical activity, nicotine exposure, and sleep) as well as 4 health factors (BMI, blood pressure, blood glucose, and non-high-density lipoprotein [non-HDL] cholesterol) [23, 24]. Health behaviors scores were derived from questionnaire responses. Diet indicators were assessed using the Healthy Eating Index (HEI) 2015 [32], and its scoring criteria were showed in Supplementary Table S1. BMI and blood pressure scores originated from physical examination measurements, and blood glucose and non-HDL scores were based on laboratory analyses of blood samples. The method for computing LE8 score was documented in previous literature, with details provided in Supplementary Table S2. By averaging the scores of the 8 metrics, the overall CVH score was calculated. Similarly, scores for health behaviors and health factors were determined using relevant metrics. Scores ranged from 0 to 100, with higher scores indicating better health. In accordance with the guidelines set forth by the AHA, overall CVH, healthy behaviors, and health factors were classified into three categories: low (0–49 points), moderate (50–79 points), and high (80–100 points).

Definition of MASLD

The Fibro Scan[®] model 502 V2 Touch device was utilized for VCTE examinations at the mobile examination center to evaluate the CAP values and liver stiffness. An examination was considered complete if the fasting time was at least 3 h, there were 10 or more valid stiffness measurements, and the interquartile range/median of liver stiffness was 30% or less. We identified hepatic steatosis using a median CAP of at least 285 dB/m based on previous studies (80% sensitivity and 77% specificity for detecting 5% steatosis) [33, 34]. MASLD was defined as the presence of hepatic steatosis, at least one of the five cardiometabolic risk factors as recommended in the recent consensus statement [1], and the absence of excessive alcohol consumption (≥ 2 drinks for women and ≥ 3 drinks for men).

Assessment of covariates

Covariates in this study included age, gender (male/ female), race/ethnicity (Hispanic, non-Hispanic White, non-Hispanic Black, and other), education level (high school or less, some college or associates degree, and college graduate or above), marital status (married/living with partner, widowed/divorced/separated, and never married), poverty income ratio (PIR), alanine aminotransferase (ALT), AST, gamma-glutamyl transferase (GGT), obesity (yes/no), history of thyroid disease (yes/ no), and sleep apnea (yes/no). Obesity was defined as BMI \ge 30 kg/m² [35]. Recent research has indicated a correlation between obstructive sleep apnea and the occurrence and progression of NAFLD [36]. Considering the widespread prevalence of sleep apnea in CVD patients and the significant role of sleep health in CVH assessment [23, 37], this study included sleep apnea as a covariate. The diagnosis of sleep apnea was based on the sleep questionnaire SLQ040 "In the past 12 months, how often did you snort, gasp, or stop breathing while you were asleep?". The presence of sleep apnea was considered when the responses were rarely (1-2 nights per week), occasionally (3-4 nights per week), or frequently (5 or more nights per week).

Statistical analysis

To ensure the representativeness of the entire nation, this study considered the intricate sampling design of NHANES by utilizing sample weights in all analyses. Continuous variables were reported as weighted means and standard errors (SE), while categorical variables were presented as weighted percentages along with their respective 95% confidence intervals (CIs). Baseline characteristics of study participants were compared using unadjusted linear regression for continuous variables and Rao-Scott chi-square tests for categorical variables. Additionally, we calculated the age-standardized prevalence estimates and their corresponding 95% CIs for each CVH category.

Weighted binary logistic regressions were utilized to calculate the odds ratio (OR) and 95% CI to investigate the associations of CVH using the LE8 score with MASLD. Our study applied three models. Model 1 did not adjust for any potential confounders. Model 2 made adjustments for age, gender, and race/ethnicity. Model 3 further adjusted for education level, marital status, PIR, ALT, AST, GGT, obesity, history of thyroid disease and sleep apnea.

Restricted cubic spline (RCS) analysis with 3 knots (at the 5th, 50th, and 95th percentiles) was conducted to explore the nonlinear relationships between LE8 and its subscale scores with MASLD after adjusting for variables in model 3. The likelihood ratio test was utilized to evaluate nonlinearity. Subsequently, subgroup and interaction analyses were carried out based on gender, age strata (20–39 years, 40–59 years, and \geq 60 years), race/ethnicity, education level, marital status, PIR (low income: PIR < 1.30, middle income: PIR \geq 1.30, < 3.50, and high income: PIR \geq 3.50), and obesity.

We also undertook three sensitivity analyses to confirm the robustness of our results. Firstly, we reanalyzed the data by excluding participants who self-reported a history of CVD, encompassing heart attack, angina, coronary heart disease, or stroke (n = 559). Secondly, we employed multivariate multiple imputation with chained equations to impute missing values. Missing data for both sleep apnea and PIR were imputed. Finally, repeated analysis was conducted by using a median CAP value of 263dB/m or more as the definition of hepatic steatosis (90% sensitivity) [38]. All statistical analyses were conducted using R version 4.2.1 software (R Foundation for Statistical Computing, Vienna, Austria). A 2-sided *P*-value less than 0.05 was deemed statistically significant.

Results

Baseline characteristics of the study population

The baseline characteristics of the 5680 participants aged 20 years or older are summarized in Table 1. The weighted mean (SE) age was 48.03 (0.64) years with 2881 females (weighted, 50.80%) and 2799 males (weighted, 49.20%). The weighted mean (SE) LE8 score for the study population was 68.25 (0.44), with the score indicating low, moderate, and high CVH at 42.11 (0.33), 65.76 (0.24) and 86.95 (0.28), respectively. Participants with low CVH were generally older, male, and had lower income levels compared to those with high CVH. They were also more likely to be widowed, divorced, separated, or never married, and exhibited a higher prevalence of thyroid disease, sleep apnea, and obesity. A total of 2130 participants (weighted, 36.83%) were diagnosed with MASLD. The non-MASLD group had higher LE8 scores.

LE8 score and MASLD

Figure 2 presents the age-adjusted prevalence of MASLD, with a significantly lower prevalence observed in the high CVH group (12.0%, 95% CI, 10.1–14.2%) than in the moderate (40.8%, 95% CI, 39.3–42.4%) and low (62.0%, 95%CI, 58.4–65.5%) CVH groups. In Model 3 (Table 2),

Characteristic	Nb	Overall CVH	Low CVH	Moderate CVH	High CVH	<i>P</i> value
		(<i>n</i> = 5680)	(n=724)	(n=3901)	(<i>n</i> = 1055)	
Age, mean (SE), y	/	48.03 (0.64)	55.14 (0.93)	49.36 (0.7)	41.02 (0.85)	< 0.001
Age, y						
20–39	1767	36.01 (32.95–39.19)	17.17 (12.86–22.56)	32.66 (29.36–36.15)	54.11 (48.99–59.14)	< 0.001
40–59	1907	34.83 (32.40-37.33)	40.51 (34.34–46.99)	36.37 (33.68–39.13)	27.79 (23.61–32.40)	
≥60	2006	29.16 (25.83–32.73)	42.32 (35.11–49.87)	30.96 (27.19–35.02)	18.10 (14.57–22.27)	
Gender						
male	2799	49.20 (46.94–51.46)	43.14 (36.93–49.57)	53.84 (50.87–56.79)	38.04 (33.43–42.88)	< 0.001
Female	2881	50.80 (48.54–53.06)	56.86 (50.43-63.07)	46.16 (43.21–49.13)	61.96 (57.12-66.57)	
Race and ethnicity		,	,			
Hispanic	1259	15.37 (12.63–18.59)	14.39 (11.05–18.52)	15.27 (12.49–18.53)	16.11 (12.63–20.34)	< 0.001
Non-Hispanic White	2065	65.23 (60.31–69.84)	59.78 (51.52-67.52)	65.71 (60.61–70.47)	66.16 (59.84–71.96)	
Non-Hispanic Black	1432	10.11 (7.73–13.12)	16.77 (12.30-22.44)	10.37 (7.83–13.61)	6.47 (4.94-8.44)	
Others ^c	924	9.29 (7.59–11.33)	9.07 (5.88–13.74)	8.66 (7.17–10.42)	11.25 (8.13–15.37)	
Poverty income ratio				,		
<13	1344	1762 (1582-1958)	29.03 (22.60-36.43)	17 27 (15 54–19 16)	13 67 (11 44–16 26)	< 0.001
>13<35	1971	33 51 (30 60-36 55)	43.62 (37.93-49.48)	34.12 (30.69-37.73)	27 34 (23 39-31 67)	0.001
> 3 5	1758	48 87 (45 17-52 59)	27 35(21 22-34 47)	48.60(44.09-53.14)	58.99 (54.55-63.29)	
	1750	10.07 (15.17 52.55)	27.55(21.22 51.17)	10.00(11.09 99.11)	50.55 (51.55 (53.25)	
High school or less	2262	35 87 (37 58_30 10)	53 98 (50 10-57 80)	38 79(37 63-73 10)	10 17 (16 75_21 85)	< 0.001
Some college or associates degree	107/	32 55 (29 34-35 93)	32.69 (29.41_36.15)	32.03(30.13_33.00)	25.08 (21.60_30.78)	< 0.001
College graduate or above	1724	32.00 (29.04-00.90)	12 22 (10 / 8 16 82)	20.10(24.83, 33.06)	54 85 (40 24 60 34)	
Marital status	1494	55.40 (29.50-57.00)	15.55 (10.46-10.62)	29.19(24.05-55.90)	54.05 (49.24-00.54)	
Married / Living with Dartpor	2256	62 11 (60 12 65 00)	EQ 14 (EQ 62 6E 20)	6412(61106704)	62 22 (56 71 67 44)	< 0.001
Widewed/Diversed/Separated	1240	19 10 (16 57 10 72)	20.14 (20.02-02.29)	10.06 (16.91 - 21.52)	11 00 (001 1201)	< 0.001
Never married	1004	18.00 (16.57 - 19.73)	27.72 (23.01-33.00)	19.00 (10.01-21.33)	26 70 (21 64 22 4F)	
	1004	10.00 (10.39-21.22)	14.14 (10.40-10.39)	10.01 (14.00-19.21)	20.70 (21.04-52.45)	
MASED	2120	26 02 (21 70 20 00)	67 20 (62 02 71 26)	A1 A7(20 15 A2 02)	0.00 (7.25 12.17)	< 0.001
yes	2150	50.65 (54.70-59.00)	07.20 (05.05-71.20)	41.47(39.13-43.03)	9.00 (7.55-15.17)	< 0.001
The world disease bistory	5550	05.17 (01.00-05.50)	52.72 (20.74-50.97)	50.55 (50.17-00.65)	90.12 (00.05-92.05)	
Inyrold disease flistory	675	1266 (11 47 120E)	22 10 (14 21 22 02)	11.04/10.26 12.62)	10.09 (9.02, 12.46)	0.002
yes	075	12.00 (11.47-15.95)	22.19 (14.21-52.95)	11.04 (10.20-13.02)	10.96 (6.92-15.40)	0.005
	5005	87.34 (80.05–88.53)	//.81 (0/.0/-85./9)	88.10 (80.38–89.74)	89.02(80.54-91.08)	
Obesity	2427	41 54 (20 46 44 60)	77 20 (71 (2, 02, 20)	47.02 (44.11 51.50)		< 0.001
yes	2427	41.54 (38.40-44.09)	77.39 (71.03-82.28)	47.83 (44.11-51.58)	7.37 (5.00-9.05)	< 0.001
	3253	58.40 (55.31-01.54)	22.01 (17.72-28.37)	52.17 (48.42-55.89)	92.03 (90.35-94.40)	
Sleep apnea	1250		22.00/26 40.20.17)	24.05 (22.02, 27.21)	1715(1227 2174)	.0.001
yes	1356	23.81 (22.29–25.41)	32.00 (26.40-38.17)	24.95 (22.82-27.21)	17.15(13.37-21.74)	< 0.001
	4040	76.19 (74.59-77.71)	68.00 (61.83-73.60)	/5.05 (/2./9-//.18)	82.85 (78.26-86.63)	.0.001
ALI, mean (SE), U/L	/	22.56 (0.26)	24.30 (0.83)	23.70 (0.34)	18.42 (0.30)	< 0.001
ASI, mean (SE), U/L	1	21.44 (0.16)	21.99 (0.64)	21.54 (0.14)	20.91 (0.40)	0.12
GGT, mean (SE), U/L	1	28.28 (0.59)	37.12 (1.88)	30.06 (0.67)	19.18 (0.58)	< 0.001
Poverty income ratio, mean (SE)	/	3.23 (0.06)	2.52 (0.15)	3.22 (0.06)	3.56 (0.07)	< 0.001
LE8 scores (out of 100 possible points)					/ >	
LE8 score, mean (SE)	/	68.25 (0.44)	42.11 (0.33)	65./6 (0.24)	86.95 (0.28)	< 0.001
HEI-2015 diet score, mean (SE)	/	39.02 (1.08)	21.19 (1.15)	35.01 (1.11)	58.65 (1.08)	< 0.001
Physical activity score, mean (SE)	/	/6.91 (0.76)	31.07 (3.59)	/6.94 (1.16)	96.66 (0.55)	< 0.001
Nicotine exposure score, mean (SE)	/	/5.31 (1.00)	46.85 (1.75)	/3.38 (1.27)	93.36 (0.88)	< 0.001
Sleep health score, mean (SE)	/	86.24 (0.6)	67.99 (2.26)	86.39 (0.63)	93.69 (0.64)	< 0.001
Body mass index score, mean (SE)	/	57.07 (1.01)	30.66 (1.71)	51.96 (1.21)	83.63 (0.79)	< 0.001
Blood lipids score, mean (SE)	/	67.03 (0.88)	45.11 (1.20)	64.06 (1.29)	85.30 (1.02)	< 0.001

Characteristic	N ^b	Overall CVH	Low CVH	Moderate CVH	High CVH	<i>P</i> value
		(n=5680)	(n=724)	(<i>n</i> =3901)	(<i>n</i> = 1055)	
Blood glucose score, mean (SE)	/	76.57 (0.51)	56.13 (1.26)	74.05 (0.69)	92.92 (0.78)	< 0.001
Blood pressure score, mean (SE)	/	67.85 (0.84)	37.88 (1.65)	64.28 (0.84)	91.40 (0.58)	< 0.001

NHANES, National Health and Nutrition Examination Survey; CVH, cardiovascular health; SE, standard error; MASLD, metabolic dysfunction-associated steatotic liver disease; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; LE8, Life's Essential 8; HEI, healthy eating index ^a Continuous variables are reported as weighted mean (standard error), while categorical variables are reported as weighted percentage with 95% confidence interval

^b Numbers of each stratum may not add up to the total population due to missing data

^c Others include Non-Hispanic Asian, other non-Hispanic, and multi-race individuals



Fig. 2 Age-adjusted prevalence of MASLD in different levels of CVH. Bar whiskers represent the 95% confidence level. MASLD, metabolic dysfunctionassociated steatotic liver disease; CVH, cardiovascular health

the ORs for MASLD were 0.50 (95% CI, 0.37–0.69) for participants with moderate CVH and 0.21 (95% CI, 0.13–0.34) for those with high CVH, when compared to those with low CVH (P<0.001 for trend). Furthermore, the OR for MASLD was 0.68 (95%CI, 0.61–0.77) for each 10-point increase in the LE8 score. Figure 3A exhibits a nonlinear relationship between LE8 score and MASLD (P=0.02 for nonlinearity), with the lowest threshold for a beneficial relationship observed at 67 points (estimated OR = 1).

Health behaviors and MASLD

After correction for age, individuals with high health behaviors (34.0%, 95% CI, 31.9–36.3%) were observed to exhibit a lower prevalence of MASLD compared to those with moderate (39.5%, 95% CI, 37.7–41.3%) or low (41.0%, 95% CI, 37.9–44.2%) health behaviors (Fig. 2). In the fully adjusted multivariate logistic regression model, compared with the low health behaviors group, ORs were 0.87 (95% CI 0.70–1.08) and 0.82 (95% CI, 0.53–1.26) in the moderate and high health behaviors

Model	Model 1 ^a		Model 2 ^b		Model 3 ^c	
	OR (95% CI)	Pvalue	OR (95% CI)	<i>P</i> value	OR (95% CI)	<i>P</i> value
LE8 score						
Per 10 points increase	0.52 (0.48–0.56)	< 0.001	0.51 (0.48–0.55)	< 0.001	0.68 (0.61–0.77)	< 0.001
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.34 (0.28-0.43)	< 0.001	0.32 (0.26-0.40)	< 0.001	0.50 (0.37-0.69)	0.001
High (80–100)	0.05 (0.04-0.08)	< 0.001	0.05 (0.04-0.08)	< 0.001	0.21 (0.13-0.34)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001
Health behaviors score						
Per 10 points increase	0.91 (0.86–0.95)	< 0.001	0.89 (0.84-0.94)	< 0.001	0.94 (0.87-1.03)	0.15
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.90 (0.77-1.05)	0.16	0.87 (0.74-1.02)	0.08	0.87 (0.70–1.08)	0.18
High (80–100)	0.65 (0.50–0.85)	0.003	0.60 (0.46–0.79)	< 0.001	0.82 (0.53–1.26)	0.31
P for trend		0.002		< 0.001		0.37
Health factors score						
Per 10 points increase	0.52 (0.50–0.55)	< 0.001	0.50 (0.47-0.53)	< 0.001	0.63 (0.58–0.69)	< 0.001
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.30 (0.24–0.37)	< 0.001	0.27 (0.22-0.34)	< 0.001	0.46 (0.34-0.62)	< 0.001
High (80–100)	0.04 (0.03–0.05)	< 0.001	0.03 (0.03-0.04)	< 0.001	0.13 (0.09–0.19)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001

Table 2	Association	of LE8 and	l its subscales	scores with	i MASLD
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LE8, Life's Essential 8; MASLD, metabolic dysfunction-associated steatotic liver disease; OR, odds ratio; CI, confidence interval

^a Crude model

^b Adjusted for age (as a continuous variable), gender, race/ethnicity

^c Additionally adjusted for education level, marital status, poverty income ratio (as a continuous variable), serum alanine aminotransferase, aspartate

aminotransferase, gamma-glutamyl transferase, obesity, thyroid disease history, and sleep apnea



Fig. 3 Dose–response relationships between LE8 score (**A**), health behaviors score (**B**), health factors score (**C**), and MASLD. ORs (solid lines) and 95% CIs (shaded areas) were adjusted for age (as a continuous variable), gender, race/ethnicity, education level, marital status, poverty income ratio (as a continuous variable), serum alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, obesity, thyroid disease history, and sleep apnea. Vertical dotted lines indicate the minimal threshold for the beneficial association with estimated OR = 1. LE8, Life's Essential 8; MASLD, metabolic dysfunction-associated steatotic liver disease; OR, odds ratio; CI, confidence interval

groups, respectively (P = 0.37 for trend). OR for MASLD was 0.94 (95% CI, 0.87–1.03) for each 10-point increase in health behavior score (Table 2). Analysis of the multivariate adjusted RCS revealed a linear dose–response relationship between health behaviors score and MASLD (P = 0.62 for nonlinearity, Fig. 3B). The minimum threshold for a favorable association was estimated to be 70 points (estimated OR = 1).

Health factors and MASLD

The age-adjusted prevalence of MASLD was significantly lower in participants with high health factors scores (7.7%, 95% CI, 6.5–9.2%) compared to those with moderate health factors scores (38.6%, 95% CI, 36.9–40.4%) and low health factors scores (64.2%, 95% CI, 61.6–66.8%) (Fig. 2). The fully adjusted multivariate logistic regression model demonstrated that, in comparison to the low health factors group, the ORs of MASLD were 0.46 (95% CI, 0.34–0.62) and 0.13 (95% CI, 0.09–0.19) in the moderate and high health factors groups, respectively (P<0.001 for trend). Moreover, for every 10-point increase in the health factors score, the OR associated with MASLD was 0.63 (95% CI, 0.58–0.69) (Table 2). Figure 3C shows a non-linear relationship between health factors score and MASLD (P=0.003 for nonlinearity). The lowest threshold for the beneficial relationship was 65 scores (estimated OR = 1).

Subgroup and sensitivity analyses

As shown in Fig. 4, the subgroup analysis results indicate a negative association between LE8 score and MASLD across all subgroups. There were no significant interactions detected between LE8 and variables including gender, age, race/ethnicity, education level, marital status, PIR, and obesity with MASLD (P<0.05 for interaction).

The results of sensitivity analyses are presented in Table 3. The associations between LE8 and its subscales scores with MASLD were not significantly altered after excluding participants with a history of CVD. In addition, the findings remained robust when the analysis was repeated with multiple imputations for missing covariates. Finally, we conducted sensitivity analysis using a median CAP of 263 dB/m as the cutoff value for defining hepatic steatosis, the results were still robust.

Discussion

In this analysis involving a nationally representative cohort of US adults, we have demonstrated a negative association between CVH defined by LE8 score and MASLD, and this observation remained consistent across various subgroup analyses (e.g., gender, age, race/ethnicity, education, income, marital status, and obesity) and sensitivity analyses. Overall, our findings highlight the significance of maintaining higher CVH as an essential approach to preventing MASLD.

Several studies have investigated the relationship between LS7 and NAFLD. A cross-sectional study conducted in China demonstrated an inverse correlation between the prevalence of NAFLD and LS7 quartiles [39]. A cohort study in Korea found that higher LS7 scores were associated with a decreased incidence of NAFLD and its regression [40]. Similarly, the US multiethnic study of atherosclerosis cohort also reported that higher LS7 levels correlated with a lower prevalence of NAFLD [41]. These findings are consistent with the negative association between CVH levels and NAFLD. However, LS7, as a precursor to LE8, may not fully reflect current health behaviors and practices due to limitations in metric quantification and sensitivity to individual variations, rendering it unsuitable for dose-response assessments [24]. Previous research has also explored the association

between LE8 and NAFLD. Wang et al. found that higher LE8 and its subscales scores were non-linearly correlated with NAFLD [42]. A prospective study involving 3266 adults without NAFLD indicated a declining trend in NAFLD risk with higher modified LE8 scores [43]. Furthermore, a UK Biobank analysis revealed a significant association between healthy lifestyle, elevated LE8 scores, and a lower risk of severe NAFLD, independent of genetic factors [44]. However, in light of the 2023 Delphi statement from three leading liver societies, NAFLD has been redefined as MASLD, which not only changes the terminology but also expands the definition to acknowledge the multifactorial metabolic drivers of fatty liver disease beyond the absence of alcohol intake [1]. This shift enhances our understanding and management of this prevalent liver condition. Nonetheless, it raises concerns about extrapolating past NAFLD research findings to the new MASLD definition. Investigating the correlation between CVH and MASLD is essential. To our knowledge, this study is the first to comprehensively investigate the relationship between the new CVH metric, LE8 score, and MASLD risk, thereby updating our understanding of CVH and MASLD.

In this study, we found that LE8 score and health factors score, but not health behaviors score, exhibited a negative correlation with MASLD. We speculate that this discrepancy may be attributed to several factors: (1) The four components of the health factors score-BMI, blood pressure, blood lipid profiles, and blood glucose levels—overlap with the definition of MASLD, which may directly impact patients' metabolic health status. (2) The influence of health behaviors on MASLD might exhibit a threshold effect or interaction. For instance, certain health behaviors (such as physical activity, sleep) may have a significant protective effect on MASLD within a certain quantity or frequency, but the effect may diminish or disappear below or above a critical point. (3) The assessment of diet, physical activity, nicotine exposure, and sleep health in health behaviors score may involve a degree of subjectivity, with participants potentially failing to accurately recall or honestly report their behaviors. Future studies could employ more objective assessment methods, such as exercise monitors, sleep quality measurement tools to more precisely evaluate the impact of these behaviors on MASLD. (4) The sample size and characteristics of the specific population may have influenced the significance of the results, necessitating a larger sample size to detect such relationships.

In our study, RCS analysis revealed a non-linear relationship between LE8 score and health factors score with MASLD. ORs significantly decreased in the lower range of scores and gradually stabilized in the higher range, exhibiting a saturation effect. In contrast, health behaviors score showed a linear relationship with MASLD,

Subgroups		OR (95% CI)	P for interaction
Overall	•	0.66 (0.59-0.73)	
Gender			0.09
Male	—	0.64 (0.53-0.79)	
Female	 i	0.71 (0.63-0.81)	
Age strata		, , ,	0.22
20-39	•	0.68 (0.54-0.86)	
40-59	⊢ ∎−−−1	0.65 (0.54-0.78)	
≥60	⊢_	0.71 (0.62-0.81)	
Race/Ethnicity		1 1 1	0.07
Hispanic	⊢ ∎−−−1	0.82 (0.72-0.94)	
Non-Hispanic White	⊢ ∎−−1	0.63 (0.54-0.74)	
Non-Hispanic Black	⊢ (0.73 (0.60-0.88)	
Others	I	0.75 (0.61-0.92)	
Education		1 1 1	0.39
High school or less	⊢ ∎−−1	0.70 (0.61-0.81)	
Some college or associates degree	⊢ ∎−−1	0.66 (0.57-0.77)	
College graduate or above	⊢ ∎−−−1	0.65 (0.52-0.82)	
Poverty income ratio			0.15
<1.3	H	0.82 (0.67-1.00)	
≥1.3,<3.5	⊢ ∎−−−1	0.71 (0.58-0.88)	
≥3.5	⊢ ∎−−−1	0.57 (0.48-0.69)	
Marital status		1 	0.47
Married/Living with Partner	⊢ ∎−−1	0.65 (0.57-0.75)	
Widowed/Divorced/Separated	⊢ ∎−−−1	0.74 (0.63-0.87)	
Never married	⊢ ∎−−−−1	0.78 (0.63-0.98)	
Obesity		- - 	0.59
Yes	⊢ ∎−−1	0.68 (0.59-0.78)	
No	⊢ ∎I	0.67 (0.56-0.81)	
٦ 0.	4	1	

Fig. 4 Subgroup analysis of association of LE8 score and MASLD. ORs were calculated as per 10 points increase in LE8 score. Each stratification was adjusted for age (as a continuous variable), gender, race/ethnicity, education level, marital status, poverty income ratio (as a continuous variable), serum alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, obesity, thyroid disease history, and sleep apnea. LE8, Life's Essential 8; MASLD, metabolic dysfunction-associated steatotic liver disease; OR, odds ratio; CI, confidence interval

with no saturation effect observed. These findings suggest that stricter health behaviors criteria may be more ideal.

Although the mechanisms between CVH and MASLD remain unclear, prior research has shown a significant

correlation between MASLD and various metabolic disorders, such as obesity, hyperglycemia, hypertension, or dyslipidemia [11]. These are also consistent components of the health factor indicators of LE8. The onset and progression of MASLD are influenced by multiple factors

Tab	le 3	Sensitivity	[,] analysis of	the association	of LE8 and its subso	cales scores with MASLD ^e
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	Excluding participants with CVD history		Multiple imputation	on ^b	CAP≥263 dB/m	
	OR (95% CI)	<i>P</i> value	OR (95% CI)	P value	OR (95% CI)	<i>P</i> value
LE8 score						
Per 10 points increase	0.69 (0.60-0.78)	< 0.001	0.68 (0.60–0.76)	< 0.001	0.66(0.59–0.73)	< 0.001
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.49 (0.37-0.66)	< 0.001	0.50 (0.37–0.67)	< 0.001	0.50(0.34-0.74)	0.004
High (80–100)	0.20 (0.12-0.33)	< 0.001	0.20 (0.13–0.32)	< 0.001	0.21(0.13-0.34)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001
Health behaviors score						
Per 10 points increase	0.95 (0.86-1.04)	0.21	0.93 (0.86–1.01)	0.07	0.92(0.84-1.00)	0.06
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.82 (0.65-1.05)	0.10	0.81 (0.65–0.99)	0.04	0.85(0.66-1.11)	0.19
High (80–100)	0.80 (0.49-1.31)	0.32	0.73 (0.49–1.10)	0.12	0.70(0.45-1.10)	0.10
P for trend		0.43		0.16		0.09
Health factors score						
Per 10 points increase	0.64 (0.58–0.70)	< 0.001	0.64 (0.60–0.69)	< 0.001	0.63(0.58–0.68)	< 0.001
Low (0–49)	Reference		Reference		Reference	
Moderate (50–79)	0.46 (0.33-0.64)	< 0.001	0.49 (0.38–0.63)	< 0.001	0.51(0.34-0.77)	0.01
High (80–100)	0.13 (0.08–0.20)	< 0.001	0.14 (0.10–0.20)	< 0.001	0.15 (0.10-0.22)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001

LE8, Life's Essential 8; MASLD, metabolic dysfunction-associated steatotic liver disease; CVD, cardiovascular disease; CAP, controlled

attenuation parameter; OR, odds ratio; CI, confidence interval

^a Adjusted for age (as a continuous variable), gender, race/ethnicity, education level, marital status, poverty income ratio (as a continuous variable), serum

alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, obesity, thyroid disease history, and sleep apnea

^b Missing data for both poverty income ratio and sleep apnea were imputed

Strengths and limitations

such as high-calorie diet, lack of physical exercise, modern lifestyle, the four health factors, and genetic predisposition. These factors in turn impact liver function and lipid accumulation, leading to various abnormalities such as insulin resistance, oxidative stress, endoplasmic reticulum stress, lipid toxicity, abnormal de novo lipogenesis, mitochondrial dysfunction, endothelial dysfunction, and disruption of gut microbiota [45–47].

Our study underscores the potential value of LE8 score in fostering interdisciplinary collaboration, particularly between hepatologists and cardiologists. By identifying health behaviors and health factors associated with increased CVD risk, LE8 score can function not only as a predictive tool for CVD [29], but also as a novel strategy for preventing MASLD. This interdisciplinary approach to screening and management will aid in achieving a comprehensive evaluation and intervention of patients' health status. Given the relationship between LE8 score and MASLD risk, we believe that integrating this score into routine health assessments can offer significant insights for early detection and intervention. Additionally, considering the well-recognized association between MASLD and CVD [30], our results further reinforce the significance of sustaining optimal CVH in the prevention of MASLD.

Our study has several notable strengths. First, we utilized the updated LE8 to reflect CVH and extensively analyzed the relationship between LE8 and its subscales scores with MASLD. Second, we used data from the nationally representative NHANES sample, which has a relatively large sample size, potentially providing a better reflection of the overall population. Third, we applied strict inclusion and exclusion criteria to ensure data quality and adjusted for some confounding factors. However, this study also has some limitations that need to be addressed. As this was a cross-sectional study, we could not establish a causal relationship between CVH and MASLD. Additionally, health behavior metrics such as diet, PA, smoking, and sleep status were all self-reported, which could lead to recall bias. Lastly, liver biopsy is regarded as the diagnostic gold standard for hepatic steatosis. Nevertheless, conducting liver biopsies on a large population is infeasible and impractical due to its well-known limitations. This study employed VTCE results as the diagnostic criterion for hepatic fat deposition, based on previous research suggesting that VTCE could be a suitable evaluation tool in large-scale epidemiological investigations **[48]**.

Conclusions

In this population-based cross-sectional study, we found a robust negative association between CVH and MASLD. Higher CVH levels were associated with a lower risk of MASLD. This correlation was also evident in health factors score. RCS analysis indicated a non-linear relationship between LE8 score and health factors score with MASLD. These findings highlight the importance of maintaining optimal CVH as a potential preventive measure against MASLD. Future research should focus on exploring the causal relationship between CVH and MASLD, as well as elucidating the underlying mechanisms.

Supplementary Information

The online version contains supplementary material available at https://doi.or g/10.1186/s41043-025-00745-1.

Supplementary Material 1

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Author contributions

Lian-Zhen Huang: Conceptualization, formal analysis, methodology, visualization, writing – original draft. Ze-Bin Ni: Conceptualization, formal analysis, methodology. Wei-Feng Huang: Formal analysis, writing – review and editing. Li-Ping Sheng: Formal analysis, methodology, visualization. Yan-Qing Wang: Conceptualization, formal analysis, methodology, writing – review and editing. Jin-Yan Zhang: Conceptualization, funding acquisition, supervision, writing – review and editing. All authors have approved the final draft of the manuscript.

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Data availability

Publicly available datasets were analyzed in this study. These data can be found on the NHANES website (https://www.cdc.gov/nchs/nhanes/index.ht m).

Declarations

Ethical approval and consent to participate

The NHANES protocols underwent review and approval by the National Center for Health Statistics institutional review board. All participants provided written informed consent at the time of participation. Ethical review and approval for this study were waived, as secondary analysis did not necessitate additional institutional review board approval.

Competing interests

The authors declare no competing interests.

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References

- 1. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. J Hepatol. 2023;79(6):1542–56.
- 2. Powell EE, Wong VW, Rinella M. Non-alcoholic fatty liver disease. Lancet. 2021;397(10290):2212–24.
- Takahashi Y, Dungubat E, Kusano H, Fukusato T. Pathology and pathogenesis of metabolic dysfunction-associated steatotic liver disease-associated hepatic tumors. Biomedicines. 2023;11(10):2761.
- Younossi ZM, Golabi P, Paik JM, et al. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. Hepatology. 2023;77(4):1335–47.
- Riazi K, Azhari H, Charette JH, et al. The prevalence and incidence of NAFLD worldwide: a systematic review and meta-analysis. Lancet Gastroenterol Hepatol. 2022;7(9):851–61.
- Miao L, Targher G, Byrne CD, et al. Current status and future trends of the global burden of MASLD. Trends Endocrinol Metab. 2024;35(8):697–707.
- Stefan N, Yki-Järvinen H, Neuschwander-Tetri BA. Metabolic dysfunctionassociated steatotic liver disease: heterogeneous pathomechanisms and effectiveness of metabolism-based treatment. Lancet Diabetes Endocrinol. 2024 Dec 13:S2213-8587(24)00318-8. https://doi.org/10.1016/S2213-8587(24) 00318-8. Online ahead of print.
- Driessen S, Francque SM, Anker SD, et al. Metabolic dysfunction-associated steatotic liver disease and the heart. Hepatol. 2023 Dec 25. https://doi.org/10. 1097/HEP.00000000000735. Online ahead of print.
- Zhou F, Zhou J, Wang W, et al. Unexpected rapid increase in the burden of NAFLD in China from 2008 to 2018: a systematic review and meta-analysis. Hepatology. 2019;70(4):1119–33.
- 10. Younossi ZM, Henry L. Understanding the burden of nonalcoholic fatty liver disease: time for action. Diabetes Spectr. 2024;37(1):9–19.
- Semmler G, Balcar L, Wernly S, et al. Insulin resistance and central obesity determine hepatic steatosis and explain cardiovascular risk in steatotic liver disease. Front Endocrinol (Lausanne). 2023;14:1244405.
- 12. Targher G, Byrne CD, Tilg H. MASLD: a systemic metabolic disorder with cardiovascular and malignant complications. Gut. 2024;73(4):691–702.
- Lee HH, Lee HA, Kim EJ, et al. Metabolic dysfunction-associated steatotic liver disease and risk of cardiovascular disease. Gut. 2024;73(3):533–40.
- 14. Moon JH, Jeong S, Jang H, et al. Metabolic dysfunction-associated steatotic liver disease increases the risk of incident cardiovascular disease: a nation-wide cohort study. EClinicalMedicine. 2023;28:65:102292.
- 15. Keam SJ. Resmetirom: first approval drugs. 2024;84(6):729–35.
- Huang X, Gan D, Fan Y, et al. The associations between healthy eating patterns and risk of metabolic dysfunction-associated steatotic liver disease: a case-control study. Nutrients. 2024;16(12):1956.
- Dobbie LJ, Burgess J, Hamid A, et al. Effect of a low-calorie dietary intervention on liver health and body weight in adults with metabolic-dysfunction associated steatotic liver disease (MASLD) and overweight/obesity: a systematic review and meta-analysis. Nutrients. 2024;16(7):1030.
- Orliacq J, Pérez-Cornago A, Parry SA, et al. Associations between types and sources of dietary carbohydrates and liver fat: a UK Biobank study. BMC Med. 2023;21(1):444.
- Zhao Y, He Y, Zhang L, et al. Effect of CVAI on the incidence of MASLD compared to BMI in populations with different body types: a prospective cohort study in China. Nutr Metab Cardiovasc Dis. 2024;34(2):307–16.
- Cuthbertson DJ, Keating SE, Pugh CJA, et al. Exercise improves surrogate measures of liver histological response in metabolic dysfunction-associated steatotic liver disease. Liver Int. 2024;44(9):2368–81.
- Harris SJ, Smith N, Hummer B, et al. Exercise training improves serum biomarkers of liver fibroinflammation in patients with metabolic dysfunctionassociated steatohepatitis. Liver Int. 2024;44(2):532–40.
- 22. Zhang S, Huo Z, Borné Y, et al. Adherence to a healthy lifestyle including sleep and sedentary behaviors and risk of metabolic dysfunction-associated steatotic liver disease in Chinese adults. Prev Med. 2024;184:107971.
- Lloyd-Jones DM, Allen NB, Anderson C, et al. Life's essential 8: updating and enhancing the American Heart Association's construct of Cardiovascular Health: a Presidential Advisory from the American Heart Association. Circulation. 2022;146:e18–43.
- 24. Lloyd-Jones DM, Ning H, Labarthe D, et al. Status of cardiovascular health in US adults and children using the American heart association's new life's

essential 8 metrics: prevalence estimates from the National Health and Nutrition Examination Survey (NHANES), 2013 through 2018. Circulation. 2022;146(11):822–35.

- Tang Y, Dong W, Shen J, et al. Life's essential 8 and osteoporosis in adults aged 50 years or older: data from the National Health and Nutrition Examination Survey. Arch Osteoporos. 2024;19(1):13.
- Liang K, Zhang X. Association between life's essential 8 and cognitive function: insights from NHANES 2011–2014. Front Aging Neurosci. 2024;16:1386498.
- Ruan YX, Wu MX, Gao JW, et al. AHA life's essential 8 and new-onset CKD: a prospective cohort study from the UK Biobank. Clin Exp Nephrol. 2024;28(4):325–36.
- Du YZ, Guo B, Hu HJ, et al. Association between kidney stones and life's essential 8: a population-based study. World J Urol. 2024;42(1):274.
- Sebastian SA, Shah Y, Paul H, Arsene C. Life's Essential 8 and the risk of cardiovascular disease: a systematic review and meta-analysis. Eur J Prev Cardiol. 2024 Aug 22:zwae280. https://doi.org/10.1093/eurjpc/zwae280. Online ahead of print.
- Zheng H, Sechi LA, Navarese EP, et al. Metabolic dysfunction-associated steatotic liver disease and cardiovascular risk: a comprehensive review. Cardiovasc Diabetol. 2024;28(1):346.
- von Elm E, Altman DG, Egger M, et al. The strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7.
- 32. Reedy J, Lerman JL, Krebs-Smith SM, et al. Evaluation of the healthy eating Index-2015. J Acad Nutr Diet. 2018;118(9):1622–33.
- Siddiqui MS, Vuppalanchi R, Van Natta ML, et al. Vibration-controlled transient elastography to assess fibrosis and steatosis in patients with nonalcoholic fatty liver disease. Clin Gastroenterol Hepatol. 2019;17(1):156–e632.
- Kalligeros M, Vassilopoulos A, Vassilopoulos S, et al. Prevalence of steatotic liver disease (MASLD, MetALD, and ALD) in the United States: NHANES 2017–2020. Clin Gastroenterol Hepatol. 2024;22(6):1330–e24.
- Flegal KM, Kruszon-Moran D, Carroll MD, et al. Trends in obesity among adults in the United States, 2005 to 2014. JAMA. 2016;7(21):2284–91.
- Mesarwi OA, Loomba R, Malhotra A. Obstructive sleep apnea, hypoxia, and nonalcoholic fatty liver disease. Am J Respir Crit Care Med. 2019;1(7):830–41.
- Yeghiazarians Y, Jneid H, Tietjens JR, et al. Obstructive sleep apnea and cardiovascular disease: a scientific statement from the American heart association. Circulation. 2021;144:e56–67.

- Kim D, Cholankeril G, Loomba R, et al. Prevalence of nonalcoholic fatty liver disease and hepatic fibrosis among US adults with prediabetes and diabetes, NHANES 2017–2018. J Gen Intern Med. 2022;37(1):261–3.
- Liu H, Yao Y, Wang Y, et al. Ideal cardiovascular health metrics and the risk of non-alcoholic fatty liver disease: a cross-sectional study in northern China. Liver Int. 2019;39(5):950–5.
- Jang EH, Chang Y, Ryu S, et al. Cardiovascular health metrics in the development and regression of nonalcoholic fatty liver disease: a cohort study. J Clin Med. 2019;8(5):610.
- Oni E, Ogunmoroti O, Allen N, et al. Life's simple 7 and nonalcoholic fatty liver disease: the multiethnic study of atherosclerosis. Am J Med. 2021;134(4):519–25.
- Wang L, Yi J, Guo X, et al. Associations between life's essential 8 and nonalcoholic fatty liver disease among US adults. J Transl Med. 2022;20(1):616.
- 43. Huang J, Xin Z, Cao Q, et al. Association between updated cardiovascular health construct and risks of non-alcoholic fatty liver disease. Nutr Metab Cardiovasc Dis. 2024;34(2):317–25.
- He P, Zhang Y, Ye Z, et al. A healthy lifestyle, Life's essential 8 scores and new-onset severe NAFLD: a prospective analysis in UK Biobank. Metabolism. 2023;146:155643.
- Soto A, Spongberg C, Martinino A, et al. Exploring the multifaceted landscape of MASLD: a comprehensive synthesis of recent studies, from pathophysiology to organoids and beyond. Biomedicines. 2024;12(2):397.
- Platek AE, Szymanska A. Metabolic dysfunction-associated steatotic liver disease as a cardiovascular risk factor. Clin Exp Hepatol. 2023;9(3):187–92.
- Sahu P, Chhabra P, Mehendale AM. A comprehensive review on non-alcoholic fatty liver disease. Cureus. 2023;15(12):e50159.
- Eddowes PJ, Sasso M, Allison M, et al. Accuracy of FibroScan controlled attenuation parameter and liver stiffness measurement in assessing steatosis and fibrosis in patients with nonalcoholic fatty liver disease. Gastroenterology. 2019;156(6):1717–30.

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