

Association Between Weight Status, Weight Loss Behaviours, and Liver Fibrosis Among Adults With Metabolic Dysfunction-Associated Steatotic Liver Disease and Radiological Findings

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Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is strongly linked to excess adiposity, but the relationship between current weight status, reported weight-loss behaviours, and liver fibrosis is less clear at the population level. This study examined these associations using vibration-controlled transient elastography (VCTE) in U.S. adults with MASLD.

Methods: A cross-sectional analysis of NHANES 2017-March 2020 pre-pandemic data was conducted. The primary MASLD cohort included 1,910 adults with complete VCTE, controlled attenuation parameter (CAP) ≥ 285 dB/m, at least one cardiometabolic risk factor, no detected active hepatitis B or C, and alcohol intake below the MASLD threshold. Survey-weighted regression models evaluated BMI category, past-year weight-loss attempts, recent weight change, and specific weight-loss methods in relation to significant fibrosis (liver stiffness measurement [LSM] ≥ 8 kPa).

Results: Significant fibrosis affected 17.8% of the weighted cohort and increased from 4.4% among participants with BMI < 25 kg/m² to 22.3% among those with obesity. In the fully adjusted model, obesity was strongly associated with significant fibrosis (adjusted odds ratio [aOR] 6.66, 95% CI 2.24-19.79), whereas overweight and a past-year weight-loss attempt were not. Obesity was also associated with advanced fibrosis and higher continuous LSM. Neither losing $\geq 5\%$ of body weight nor any individual weight-loss method remained significantly associated with fibrosis after adjustment and false-discovery-rate correction.

Conclusion: Among U.S. adults with MASLD, current obesity showed a strong and consistent relationship with VCTE-defined fibrosis, while self-reported weight-loss attempts and individual strategies did not independently identify lower fibrosis burden in this cross-sectional setting. Longitudinal studies are needed to distinguish attempted from sustained, achieved weight loss and its effect on fibrosis progression.

Keywords: MASLD; obesity; weight loss; liver fibrosis; transient elastography; NHANES

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Introduction

The framework of nonalcoholic fatty liver disease (NAFLD) has recently been updated to metabolic dysfunction-associated steatotic liver disease (MASLD) in individuals with limited alcohol consumption and cardiometabolic risk factors (Rinella et al., 2023b). This terminology places metabolic dysfunction at the centre of case definition and reflects the close relationships among hepatic steatosis, obesity, dysglycaemia, hypertension, and dyslipidaemia (European Association for the Study of the Liver et al., 2024; Marchesini et al., 2001; Rinella

et al., 2023b). Steatotic liver disease affects a substantial proportion of adults worldwide and is an increasingly important cause of chronic liver disease, cirrhosis, hepatocellular carcinoma, and extrahepatic morbidity (Brunt et al., 2015; Byrne & Targher, 2015; Powell et al., 2021; Riazi et al., 2022; Sayiner et al., 2016; Younossi et al., 2016, 2018).

Steatosis alone does not determine prognosis. Liver-related complications and mortality are much more strongly associated with fibrosis severity, and risk rises

progressively as fibrosis advances (Angulo et al., 2015; Dulai et al., 2017; Ekstedt et al., 2015; Hagstrom et al., 2017; Sanyal et al., 2021; Taylor et al., 2020). Although adverse outcomes can also occur in non-obese disease, clinically important fibrosis remains closely linked to metabolic risk (Leung et al., 2017). These findings highlight the need to identify factors associated with fibrosis among individuals who already meet MASLD criteria.

Weight management is therefore central to MASLD care, but current body weight, achieved weight change, and reported weight-loss behaviour are distinct exposures. Lifestyle trials and evidence syntheses show that sustained weight reduction can improve steatosis and steatohepatitis, with larger losses offering the greatest prospect of fibrosis improvement (Hallsworth et al., 2011; Koutoukidis et al., 2019; Promrat et al., 2010; Romero-Gomez et al., 2017; Vilar-Gomez et al., 2015; Zelber-Sagi et al., 2017). This evidence underpins current clinical guidance (Chalasani et al., 2018; Rinella et al., 2023a). However, in population surveys, many adults report attempting weight loss without achieving or sustaining clinically meaningful loss and often use several strategies concurrently (Han et al., 2019; Mokari-Yamchi & Rosenkranz, 2024). An observed association between a weight-loss attempt and fibrosis may therefore reflect greater baseline obesity or metabolic disease rather than the effect of the attempt itself.

These relationships can be studied noninvasively using vibration-controlled transient elastography (VCTE). The controlled attenuation parameter (CAP) estimates hepatic steatosis, whereas liver stiffness measurement (LSM) indicates fibrosis risk (Cao et al., 2022; Eddowes et al., 2019; Karlas et al., 2017; Oeda et al., 2020; Pu et al., 2019; Wong et al., 2010). VCTE does not reproduce biopsy staging exactly, and its performance varies with the population, probe, and threshold; nevertheless, it is well suited to population-level risk assessment (Castera et al., 2019; Newsome et al., 2020). During 2017–March 2020, NHANES collected VCTE data, creating a nationally representative platform in which anthropometry, weight history, behaviours, metabolic factors, and liver imaging can be examined together (Ciardullo & Perseghin, 2021; Nouredin et al., 2022; Unalp-Arida & Ruhl, 2025; Zhang et al., 2021).

Despite this evidence, few population-based studies have examined measured weight status, recent weight change, reported weight-loss attempts, and specific weight-loss methods together in an imaging-defined MASLD population. Distinguishing achieved weight change from an intention or attempt to lose weight is important because the latter may be strongly confounded by baseline adiposity, diabetes, and clinical advice.

There is a strong link between obesity and the metabolic changes associated with MASLD. Insulin resistance, increased adipose-tissue lipolysis, oxidative stress, inflammatory signalling, lipotoxicity, and hepatic

stellate-cell activation connect excess adiposity with steatosis and fibrogenesis (Buzzetti et al., 2016; Friedman et al., 2018; Lomonaco et al., 2011, 2012; Polyzos et al., 2019; Schwabe et al., 2020, 2021). Longitudinal studies have shown that baseline obesity and subsequent weight gain are associated with fibrosis progression, indicating that weight status may signal risk beyond the presence of steatosis alone (Kim et al., 2019). In U.S. NHANES data, weight gain has also been linked to fatty liver and fibrosis measured by FibroScan (Wijarnpreecha et al., 2022), suggesting that weight change may add information beyond a single BMI measurement.

Complementary evidence comes from intervention studies. Among adults with steatohepatitis, histological improvement follows a dose-response pattern, with the greatest benefit among those who achieve larger and sustained weight loss (Promrat et al., 2010; Vilar-Gomez et al., 2015). Systematic evidence also supports structured weight-loss programmes, dietary change, and physical activity for improving liver fat and disease activity, although evidence for durable fibrosis regression is less certain (Koutoukidis et al., 2019; Romero-Gomez et al., 2017). Current European and American guidance accordingly identifies weight loss, physical activity, and dietary improvement as fundamental components of management (Chalasani et al., 2018; European Association for the Study of the Liver et al., 2024; Rinella et al., 2023a). These findings establish the clinical relevance of successful weight loss but do not imply that a self-reported intention or attempt has the same biological meaning as achieved, maintained loss.

Review of population-level weight-loss behaviour studies reveals significant variation in approaches to weight loss among individuals. Eating less, exercise, and changes of food choice were reported frequently and commercial products, pills, skipping meals, and other potentially unsafe practices were reported by a smaller proportion of people in analyses of NHANES (Han et al., 2019; Mokari-Yamchi & Rosenkranz, 2024). The social and clinical patterning of behaviour choice is also observed. Those who are obese or have more metabolic disease may be more likely to be advised to lose weight; they may even try multiple strategies simultaneously. This results in confounding by indication in cross-sectional data – an individual who has started a weight loss program might have a higher number of underlying diseases prior to the initiation of weight loss. Thus, interpretation of weight-loss behaviours should consider current adiposity level and metabolic factors associated with weight loss, and refrain from causal attributions.

The answer may be fibrosis as it is a good predictor of steatotic liver disease. Meta analytic studies have shown that fibrosis stage is gradually correlated with worsening clinical outcomes (Dulai et al., 2017; Taylor et al., 2020). VCTE is a population level approach to non-biopsy fibrosis estimation that is feasible. Validation studies

have demonstrated that LSM offers clinically valuable diagnostic accuracy for the presence of clinically relevant fibrosis while the CAP offers a quantitative assessment of steatosis; however, test performance and optimal thresholds differ across population and spectrum of disease (Eddowes et al., 2019). This variation in thresholds is important when using epidemiological data to identify cohorts of MASLD, and is useful for sensitivity analyses using different CAP definitions. Previous NHANES studies have established the value of VCTE for estimating the population burden and correlates of steatotic liver disease and fibrosis (Ciardullo & Perseghin, 2021; Noureddin et al., 2022; Unalp-Arida & Ruhl, 2025; Zhang et al., 2021). Less attention has been given to measured weight status, recent weight change, reported weight-loss attempts, and specific weight-loss methods within one imaging-defined MASLD population. Examining these characteristics together separates the direct question of how adiposity relates to fibrosis from the more complex question of how weight-management behaviours are distributed across the disease spectrum.

Purpose of the Study

The purpose of this study was to clarify whether current weight status, recent weight change, and reported weight-loss behaviours show distinct relationships with VCTE-defined fibrosis among U.S. adults with MASLD.

Aim and Objectives

The study aimed to examine the association of weight status and past-year weight-loss behaviours with liver fibrosis. The objectives were to (1) describe fibrosis across BMI categories; (2) test whether a past-year weight-loss attempt and a loss of at least 5% of body weight were associated with fibrosis; (3) describe commonly reported weight-loss methods and evaluate their adjusted associations with fibrosis; and (4) assess whether the primary findings were robust to alternative CAP thresholds for steatosis.

Research Questions

The study addressed three questions: How does fibrosis burden vary across BMI categories? Are a reported weight-loss attempt or recent loss of at least 5% independently associated with fibrosis after accounting for current adiposity and clinical covariates? Do individual weight-loss methods show independent associations with fibrosis among participants attempting weight loss?

It was hypothesised that weight status would show a stronger association with fibrosis than reported weight-loss attempts or individual strategies and that greater adiposity would be associated with a greater fibrosis burden.

Methodology

Study Design

A cross-sectional secondary analysis was performed using the NHANES 2017-March 2020 pre-pandemic data release.

Data Source and Data Collection

NHANES uses a stratified, multistage probability design to represent the civilian, noninstitutionalised population of the United States. Data are collected through household interviews and standardised physical examinations and laboratory assessments conducted in mobile examination centres (National Center for Health Statistics, 2020). For the present analysis, demographic, body-measure, weight-history, liver-ultrasound elastography, blood-pressure, diabetes, lipid, biochemistry, smoking, alcohol, and physical-activity components were linked using the unique participant identifier (SEQN).

Study Population

Participants were eligible if they were aged 18 years or older and had a complete FibroScan/VCTE examination.

MASLD Definition

The primary MASLD cohort required CAP ≥ 285 dB/m, at least one cardiometabolic risk factor, no detected active hepatitis B or C, and alcohol consumption < 210 g/week in men and < 140 g/week in women (Rinella et al., 2023b). Cardiometabolic risk factors comprised overweight or central adiposity, dysglycaemia or diabetes, elevated blood pressure or antihypertensive treatment, elevated triglycerides or lipid-lowering treatment, and low HDL cholesterol or lipid-lowering treatment (Rinella et al., 2023b). Because CAP thresholds vary across populations and validation studies, sensitivity cohorts used CAP ≥ 274 dB/m and CAP ≥ 248 dB/m while retaining the same metabolic, viral-hepatitis, and alcohol criteria (Eddowes et al., 2019).

Data Collection Instruments and Measures

Anthropometric and Weight-History Measures

NHANES measured BMI based on examination weight and height. Participants were clustered as BMI < 25 kg/m², Overweight (BMI 25.0-29.9 kg/m²) and Obese (BMI ≥ 30 kg/m²), the single underweight participant was grouped with BMI < 25 . Further categorization of obesity was done for descriptive visualization as obesity class I (BMI 30.0-34.9), obesity class II (BMI 35.0-39.9), and obesity class III (BMI ≥ 40.0 kg/m²). Past year weight loss attempt was determined using the NHANES weight-history question "Have you at any time made an effort to lose weight in the past 12 months?" The percentage weight change was assessed using current measured weight and self-reported weight one year prior, with a loss of 5% or more considered as a secondary exposure. Method-specific indicators were investigated for common behaviors like eating less, exercising, drinking more water, cutting down on sugar/fast food, consuming more fruit/vegetables, and cutting back on carbs, which were examined among those who reported a weight loss attempt (Han et al., 2019; Mokari-Yamchi & Rosenkranz, 2024).

VCTE Liver Measures

The main result was liver fibrosis, which was taken to be a VCTE liver stiffness ≥ 8 kPa. Descriptive cirrhosis range stiffness (LSM ≥ 15 kPa), advanced fibrosis (LSM ≥ 12 kPa) and continuous LSM were secondary outcomes. Continuous LSM was log transformed due to right skew. CAP was used as a radiological measure of hepatic steatosis, and summarized by weight-status group. These thresholds were used as epidemiological operational definitions of the staging instead of as biopsy equivalents as VCTE cut points differ between validation studies (Eddowes et al., 2019).

Covariates

Covariates were chosen a priori for modelling due to potential associations with body weight, weight-management behaviours, and fibrosis. Core adjustment variables included age, sex, race/ethnicity, smoking and self-reported diabetes. Weight status and past year weight loss attempt were modelled concurrently in the primary model to avoid the behavioural estimate being a marker of higher BMI. Continuous BMI was also added to the method-specific models among weight-loss attempters. Education and poverty-income ratio were described but not included in the primary model as the latter had appreciable missingness and the goal was to keep the primary model stable and interpretable.

Statistical Analysis

The NHANES mobile examination center weight, sampling stratum and primary sampling unit were included in all population estimates. Taylor-series linearization was used for the estimation of standard errors and 95% confidence intervals (25 design degrees of freedom in the primary MASLD cohort). The approximately symmetric continuous variables were summarized as weighted means, weighted medians and interquartile ranges for CAP and LSM, and as survey-weighted percentages for categorical variables. Participant-selection process documented prior to the outcome modelling.

Survey-weighted logistic regression was used to calculate the primary association with significant fibrosis. Model 1 incorporated weight status, past year weight-loss attempt, age, sex and race/ethnicity. Model 2 also comprised of smoking status, diabetes and was used as the principal adjusted model. Survey weighted logistic regression models were used for secondary models to assess advanced fibrosis, and survey-weighted linear regression models were used for the log transformed LSM. Percentages differences were calculated by exponentiated coefficients of LSM model. Recent weight change was evaluated with both a binary indicator of $\geq 5\%$ weight loss and with percentage weight change scaled per 5 percentage-point increase, adjusted for current BMI, and the core covariates.

Only those who indicated that they were attempting weight loss had specific weight-loss methods evaluated. To lessen the possibility of collinearity between

behaviours that were often used together, each was separately fitted into an adjusted survey-weighted logistic model. These were adjusted for BMI, age, sex, race/ethnicity, smoking and diabetes. Only behaviours reported by at least 30 individuals were included in method-specific analyses and Benjamini-Hochberg false-discovery-rate procedure was used to correct for tested methods. Sensitivity analyses were conducted in CAP ≥ 274 dB/m and ≥ 248 dB/m cohorts in MASLD, and repeated the main analyses. Two-sided $P < 0.05$ was used for the prespecified primary analysis and method-specific findings were interpreted using false-discovery-rate adjusted q values. Analyses were performed in Python 3.13.5 using pandas 2.2.3, NumPy 2.3.5, statsmodels 0.14.6, SciPy 1.17.0, and Matplotlib 3.10.8.

Ethical Considerations

NHANES protocols were approved by the National Center for Health Statistics Ethics Review Board. The 2017-2018 cycle operated under Protocol #2018-01 from October 26, 2017, with continuation of Protocol #2011-17 before that date; the 2019-2020 cycle continued Protocol #2018-01 (National Center for Health Statistics, 2026). All participants provided documented informed consent. The present study used publicly available, de-identified data and involved no direct participant contact.

Results

Study Population and Weighted Characteristics

The merged NHANES files contained 15,560 participants, of whom 9,693 were adults. A complete FibroScan examination was available for 7,768 adults; 2,802 had CAP ≥ 285 dB/m, 2,785 also had at least one cardiometabolic risk factor, and 2,753 had no detected active hepatitis B or C. After applying the alcohol criterion, 1,910 participants comprised the primary MASLD cohort. Figure 1 summarizes the selection process.

Figure 1. Participant selection for the primary MASLD cohort.

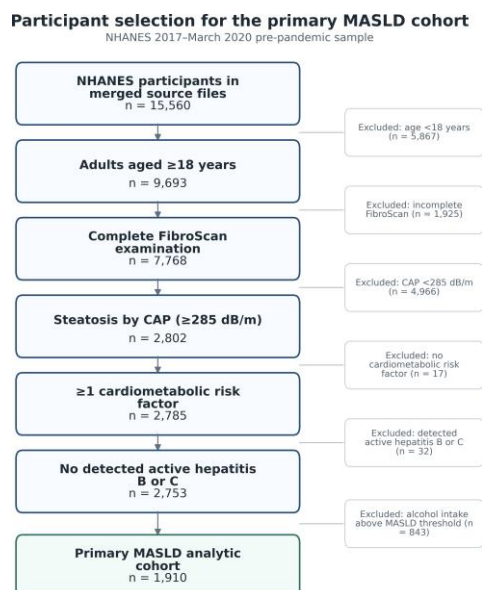


Table 1 summarises the survey-weighted characteristics of the cohort overall and by broad weight-status category. The weighted mean age was 49.1 years (SE 0.8), 57.2% were men, and the weighted mean BMI was 34.2 kg/m². Approximately 70.7% of the weighted MASLD population had obesity, 25.0% had overweight, and the remaining 4.3% had BMI <25 kg/m². Among participants with available weight-history information, 58.6% reported trying to lose weight during the previous year and 18.1% had lost at least 5% of their reported body weight. The weighted prevalence of significant fibrosis was 17.8% (95% CI, 14.4%–21.1%), advanced fibrosis 7.1% (95% CI, 5.2%–8.9%), and cirrhosis-range stiffness 3.8% (95% CI, 2.4%–5.2%).

Table 1. Survey-weighted characteristics of adults with MASLD, overall and by weight status

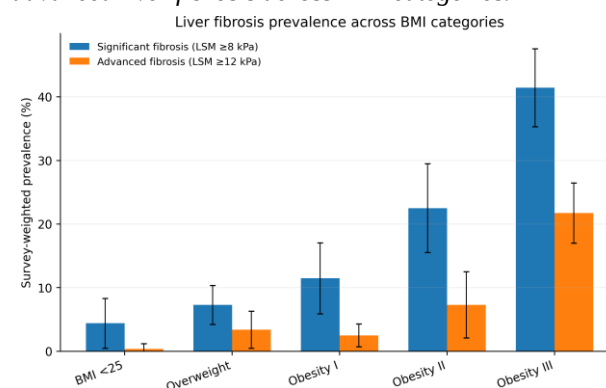
Characteristic	Overall	BMI <25	Overweight	Obesity
Unweighted n	1,910	94	502	1,307
Age, years	49.1 (0.8)	58.0 (2.0)	52.7 (1.2)	47.2 (0.8)
Male, %	57.2% (1.9)	51.3% (7.5)	62.1% (2.3)	55.8% (2.5)
BMI, kg/m ²	34.2 (0.3)	23.5 (0.2)	27.7 (0.1)	37.2 (0.2)
Waist circumference, cm	112.1 (0.7)	89.7 (0.6)	98.6 (0.4)	118.3 (0.6)
Diabetes, %	18.7% (1.7)	23.2% (8.4)	15.6% (1.8)	19.5% (1.9)

Characteristic	Overall	BMI <25	Overweight	Obesity
Current smoking, %	12.3% (1.0)	17.4% (7.3)	13.6% (1.8)	11.6% (1.0)
Tried to lose weight, %	58.6% (1.8)	21.5% (5.1)	49.3% (3.7)	65.2% (2.2)
Lost ≥5% body weight, %	18.1% (1.3)	16.0% (6.3)	17.4% (2.4)	18.5% (1.4)
CAP, dB/m, median [IQR]	323.0 [302.0–353.0]	302.7 [296.0–318.0]	308.0 [296.0–331.8]	331.0 [307.0–362.0]
LSM, kPa, median [IQR]	5.5 [4.5–7.0]	4.7 [4.1–5.3]	4.9 [4.1–6.1]	5.9 [4.8–7.7]
Significant fibrosis, %	17.8% (1.6)	4.4% (1.9)	7.3% (1.5)	22.3% (2.3)
Advanced fibrosis, %	7.1% (0.9)	0.4% (0.4)	3.4% (1.4)	8.8% (1.0)

Note. Values are survey-weighted mean (SE), percentage (SE), or median [IQR] unless otherwise indicated. CAP = controlled attenuation parameter; LSM = liver stiffness measurement; MASLD = metabolic dysfunction-associated steatotic liver disease.

A clear gradient was present across the more detailed BMI categories. Survey-weighted significant fibrosis prevalence increased from 4.4% among participants with BMI <25 kg/m² to 7.3% in overweight, 11.5% in obesity class I, 22.5% in class II, and 41.4% in class III. Advanced fibrosis increased from 0.4% in the BMI <25 group to 21.7% in obesity class III. These estimates and their 95% confidence intervals are shown in Figure 2.

Figure 2. Survey-weighted prevalence of significant and advanced liver fibrosis across BMI categories.



Note. Error bars represent 95% confidence intervals from Taylor-series linearization. LSM = liver stiffness measurement.

Primary and Secondary Associations With Fibrosis

In the primary survey-weighted logistic model, obesity showed a strong association with significant fibrosis,

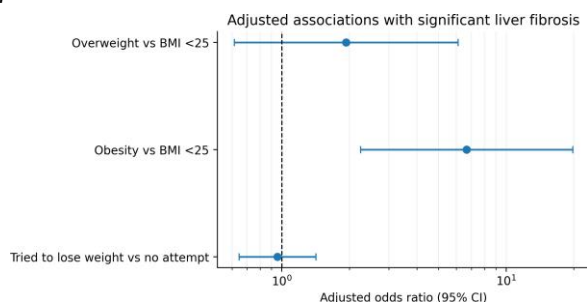
whereas overweight and a reported past-year weight-loss attempt did not. Adjustment for smoking and diabetes changed the estimates very little: compared with BMI <25 kg/m², obesity was associated with an adjusted odds ratio (aOR) of 6.66 (95% CI, 2.24-19.79; P=0.001), while the aOR for overweight was 1.93 (95% CI, 0.61-6.09; P=0.247). The adjusted association for trying to lose weight was close to null (aOR, 0.96; 95% CI, 0.64-1.42; P=0.815). Table 2 presents both sequential models, and Figure 3 displays the fully adjusted estimates for the prespecified exposures.

Table 2. Survey-weighted logistic regression for significant liver fibrosis (LSM ≥ 8 kPa)

Exposure	Model 1 aOR (95% CI)	P value	Model 2 aOR (95% CI)	P value
Overweight vs BMI <25	1.80 (0.59- 5.56)	0.290	1.93 (0.61- 6.09)	0.247
Obesity vs BMI <25	6.66 (2.31- 19.18)	0.001	6.66 (2.24- 19.79)	0.001
Tried to lose weight vs no attempt	0.97 (0.66- 1.42)	0.860	0.96 (0.64- 1.42)	0.815

Note. Model 1 adjusted for age, sex, and race/ethnicity, with weight status and weight-loss attempt entered together. Model 2 additionally adjusted for smoking status and diabetes. Smoking and diabetes were included as adjustment covariates; their coefficients were not displayed as separate rows. Complete-case N = 1,589; 273 participants had LSM ≥ 8 kPa. aOR = adjusted odds ratio; CI = confidence interval.

Figure 3. Fully adjusted associations of weight status and past-year weight-loss attempt with significant fibrosis.



Note. Estimates are from Model 2 in Table 2. The x-axis is logarithmic.

Secondary analyses were consistent with a greater fibrosis burden in participants with obesity. Obesity was associated with advanced fibrosis (aOR, 23.17; 95% CI, 2.44-219.87; P=0.008), although the wide interval

reflected the small number of advanced-fibrosis events in the BMI <25 reference group. In the continuous LSM model, obesity was associated with 36.2% higher adjusted LSM (95% CI, 19.1%-55.8%; P<0.001), while the estimate for overweight was smaller and did not reach conventional significance. Neither losing at least 5% of body weight during the preceding year nor a 5-percentage-point increment in recent weight change was significantly associated with significant fibrosis after adjustment for current BMI and covariates (Table 3).

Table 3. Secondary survey-weighted analyses of fibrosis and recent weight change

Outcome	Exposure	Adjusted effect	95% CI	P value
Advanced fibrosis (LSM ≥ 12 kPa)	Overweight vs BMI <25	9.21	0.80-105.34	0.072
Advanced fibrosis (LSM ≥ 12 kPa)	Obesity vs BMI <25	23.17	2.44-219.87	0.008
Advanced fibrosis (LSM ≥ 12 kPa)	Tried to lose weight	1.56	0.88-2.75	0.119
Log LSM	Overweight vs BMI <25	10.8%	-0.6% to 23.5%	0.063
Log LSM	Obesity vs BMI <25	36.2%	19.1% to 55.8%	<0.001
Log LSM	Tried to lose weight	1.8%	-4.6% to 8.7%	0.569
Significant fibrosis	Lost $\geq 5\%$ body weight	0.75	0.44-1.28	0.280
Significant fibrosis	Weight change per +5 percentage points	1.03	0.96-1.11	0.376

Note. Advanced-fibrosis models adjusted for weight status, weight-loss attempt, age, sex, race/ethnicity, and diabetes. Log-LSM models additionally adjusted for smoking; effects are presented as percentage differences in LSM. Recent weight-change models adjusted for

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current BMI, age, sex, race/ethnicity, smoking, and diabetes.

Weight-Loss Methods Among Participants Attempting Weight Loss

Among participants who reported trying to lose weight, the most common analysed strategies were eating less, drinking more water, exercising, increasing fruit/vegetable/salad intake, and reducing junk or fast food. In separate adjusted models, no individual strategy remained statistically significant after false-discovery-rate correction. Eating fewer carbohydrates had a nominal association with higher odds of significant fibrosis (aOR, 1.93; 95% CI, 1.02-3.66; P=0.043), but the FDR-adjusted q value was 0.340. The remaining q values were also above 0.05, indicating that the method-specific findings should be treated as exploratory rather than evidence of independent behavioural effects (Table 4 and Figure 4).

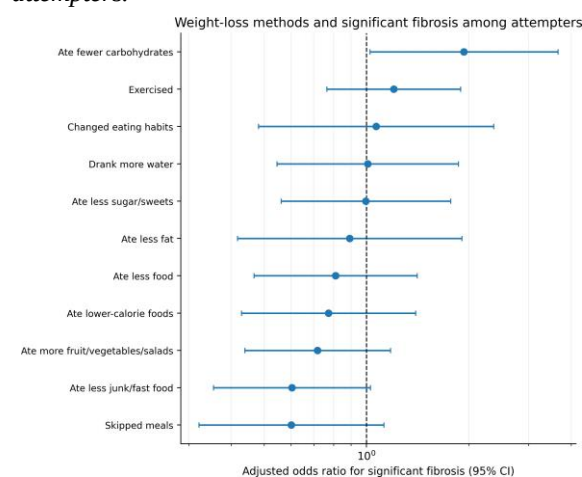
Table 4. Weight-loss methods and significant fibrosis among past-year weight-loss attempters

Weight-loss method	Weighted use, %	aOR (95% CI)	P value	FDR q
Ate less food	63.2%	0.81 (0.47 - 1.41)	0.441	0.693
Drank more water	58.9%	1.01 (0.55 - 1.86)	0.979	0.987
Exercised	58.3%	1.20 (0.76 - 1.89)	0.410	0.693
Ate more fruit/vegetables/salads	54.1%	0.72 (0.44 - 1.18)	0.179	0.491
Ate less junk/fast food	52.0%	0.60 (0.35 - 1.03)	0.062	0.340
Ate less sugar/sweets	46.1%	1.00 (0.56 - 1.77)	0.987	0.987
Changed eating habits	40.6%	1.07 (0.48 - 2.37)	0.869	0.987

Weight-loss method	Weighted use, %	aOR (95% CI)	P value	FDR q
Ate fewer carbohydrates	37.1%	1.93 (1.02 - 3.66)	0.043	0.340
Ate lower-calorie foods	36.6%	0.77 (0.43 - 1.39)	0.377	0.693
Ate less fat	33.0%	0.89 (0.42 - 1.91)	0.761	0.987
Skipped meals	25.3%	0.60 (0.32 - 1.12)	0.106	0.390

Note. Each method was fitted in a separate survey-weighted model adjusted for current BMI, age, sex, race/ethnicity, smoking status, and diabetes. FDR q values use the Benjamini-Hochberg procedure across the 11 method-specific tests.

Figure 4. Adjusted associations of individual weight-loss methods with significant fibrosis among weight-loss attempters.



Note. Each point represents a separate adjusted model. The x-axis is logarithmic; the dashed line indicates an odds ratio of 1.0. No association remained significant after FDR correction.

Sensitivity Analyses

The direction of the obesity association was preserved when the steatosis threshold was changed, but its magnitude depended on the CAP definition. The adjusted obesity aOR was 7.75 (95% CI, 2.88-20.85; P<0.001) in the CAP ≥ 274 dB/m cohort (n=2,258) and 2.24 (95% CI, 0.70-7.15; P=0.166) in the broader CAP ≥ 248 dB/m

cohort (n=3,011), compared with 6.66 in the primary CAP ≥ 285 dB/m cohort. In contrast, the adjusted estimate for past-year weight-loss attempt remained close to the null across all three definitions (aORs 0.96, 0.96, and 0.95, respectively). These analyses indicate that the behavioural result was stable, while the strength and precision of the obesity-fibrosis association varied with the steatosis threshold used to define the MASLD population.

For clearer interpretation of the radiological context, Figure 5 presents representative ultrasonographic and unenhanced CT appearances of normal and steatotic liver. These images are illustrative examples from Lee (2017), not images from NHANES participants; the study outcomes were derived from VCTE-based CAP and LSM measurements.

Ultrasonography



Unenhanced computed tomography

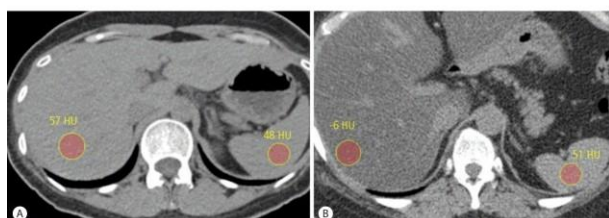


Figure 5. Representative radiological appearances of normal liver and hepatic steatosis.

Note. Top row: ultrasonography of normal liver (left) and hepatic steatosis (right). Bottom row: unenhanced CT of normal liver (left) and fatty liver (right). Adapted from Lee (2017), *Clinical and Molecular Hepatology*, 23(4), 290–301, <https://doi.org/10.3350/cmh.2017.0042>, under the Creative Commons Attribution-NonCommercial 3.0 License.

Discussion

The current study evaluated the weight status, weight loss behaviors and VCTE fibrosis among U.S. adults who met criteria for MASLD at the national level. The one thing that did not vary was the high correlation between obesity and fibrosis. The prevalence of significant fibrosis was steadily higher by BMI level and obesity was significantly associated with the presence of significant fibrosis even when adjusted for age, sex, race/ethnicity, smoking, diabetes and weight-loss attempt. Here a similar trend was observed for the advanced fibrosis and continuous LSM, where the result was not dependent on a single binary threshold. In contrast, only reporting that a weight loss attempt occurred in the past year was not independently associated with lower fibrosis and none of

the individual weight-loss methods were strongly associated after adjusting for multiple measures and performing multivariable analyses.

The obesity result is consistent with longitudinal, clinical, and population evidence linking greater adiposity with steatotic liver disease severity and fibrotic progression (Kim et al., 2019; Polyzos et al., 2019; Powell et al., 2021). In the prospective cohort reported by Kim et al. (2019), higher baseline BMI and subsequent weight gain were independently associated with progression of a noninvasive fibrosis marker, whereas weight loss was associated with reduced progression risk. U.S. population studies using liver elastography similarly identify obesity and diabetes as important correlates of steatotic liver disease and fibrosis (Ciardullo & Perseghin, 2021; Nouredin et al., 2022; Unalp-Arida & Ruhl, 2025; Zhang et al., 2021). The present analysis extends that evidence within a defined MASLD population and shows a clear increase in VCTE-defined fibrosis across BMI categories. The obesity estimate was large, but the confidence interval was wide, particularly for advanced fibrosis, because few participants with BMI <25 kg/m² had advanced-fibrosis events. The estimate should therefore be interpreted primarily as strong directional evidence rather than as a precise measure of effect size. Several biological pathways support this association. Visceral and dysfunctional adipose tissue increases delivery of free fatty acids to the liver and promotes insulin resistance, inflammatory signalling, oxidative stress, mitochondrial dysfunction, and lipotoxic injury (Buzzetti et al., 2016; Friedman et al., 2018; Lomonaco et al., 2012; Marchesini et al., 2001; Polyzos et al., 2019). Persistent hepatocellular injury and macrophage-stellate-cell crosstalk can activate hepatic stellate cells and increase extracellular-matrix deposition, providing a mechanistic link between obesity and fibrosis progression (Schwabe et al., 2020, 2021). Current MASLD guidance therefore prioritises cardiometabolic risk reduction and durable weight management (European Association for the Study of the Liver et al., 2024; Rinella et al., 2023a). The higher CAP and LSM values observed among participants with obesity are consistent with these biological and population data.

There was no independent association between a past-year weight-loss attempt and fibrosis. A reported attempt captures intention or self-reported action, not the magnitude, duration, or maintenance of achieved weight loss. Confounding by indication and reverse causation are plausible because participants with more severe obesity, diabetes, or prior clinical advice may be more likely to attempt weight loss. NHANES studies show that weight-loss attempts are common and that adults frequently combine eating less, exercise, food-choice changes, and other strategies (Han et al., 2019; Mokari-Yamchi & Rosenkranz, 2024). A binary measure can therefore combine participants with markedly different adherence, duration, achieved loss, and metabolic

response. The null association should not be interpreted as evidence that effective weight management lacks benefit for MASLD (Chalasani et al., 2018; European Association for the Study of the Liver et al., 2024; Rinella et al., 2023a).

This distinction explains why the present findings do not contradict intervention evidence. Biopsy-based studies show a dose-response relationship between achieved weight loss and histological improvement, with the greatest NASH resolution and fibrosis regression among participants achieving larger losses (Promrat et al., 2010; Vilar-Gomez et al., 2015). A broader meta-analysis likewise found that weight-loss interventions improve liver enzymes, steatosis, and disease activity, although short-term trial evidence for fibrosis change was less certain (Koutoukidis et al., 2019). The key differences are temporality and exposure definition: intervention studies follow treatment over time and quantify achieved loss, whereas this cross-sectional analysis measured current fibrosis near the time participants reported behaviour and recalled prior weight. Participants may have begun trying to lose weight after weight gain, a diagnosis, or metabolic deterioration. The data cannot establish whether the behaviour preceded fibrosis.

Similarly careful interpretation should be given to the secondary analyses, using actual recent weight change. There was no significant association between weight loss $\geq 5\%$ and reduced odds of significant fibrosis nor was there an association with the continuous measure of weight change. Modest weight loss isn't necessarily biologically harmful, as these estimates demonstrate. Instead, they may be partly due to the difficulty of recalling a 1 year weight history, the lack of uniformity in the intent to lose weight and the lack of information on the timing of the weight change to the onset of fibrosis. Fibrosis, too, evolves more slowly than body weight – a recently slimmed down individual may still have stiffness due to metabolic exposure over the previous years. On the other hand, in the case of chronic diseases, an unintentional weight loss may make this association less apparent, if the intentionality and trajectory of the weight loss are not properly measured. For temporal effects, longitudinal studies (e.g. Kim et al., 2019) that measure weight once and then evaluate fibrosis risk thereafter, are more appropriate.

None of the individual weight-loss strategies was associated with fibrosis after false-discovery-rate correction. The nominal positive estimate for eating fewer carbohydrates should not be interpreted as evidence of harm. The survey did not capture the amount, quality, duration, or adherence associated with carbohydrate restriction, and similar measurement limitations apply to the other methods. Evidence from structured interventions indicates that dietary improvement and exercise can reduce liver fat and improve metabolic outcomes, but their effects depend on intervention intensity and adherence (Hallsworth et al.,

2011; Romero-Gomez et al., 2017; Zelber-Sagi et al., 2017). Moreover, participants could report several correlated strategies, which made multiplicity correction important. In this cross-sectional setting, obesity discriminated current fibrosis burden more clearly than the particular self-reported strategy selected during the previous year.

The sensitivity analyses reinforced the main qualitative finding. The obesity association remained in the same direction at lower CAP cut-offs, although its magnitude and precision changed. Alternative CAP definitions include participants with different degrees of steatosis and therefore alter the clinical profile of the MASLD cohort. Diagnostic studies and meta-analyses have documented variation in CAP and LSM performance by population, probe, BMI, and threshold (Cao et al., 2022; Eddowes et al., 2019; Karlas et al., 2017; Oeda et al., 2020; Pu et al., 2019; Wong et al., 2010). Several CAP cut-points have also been used in NHANES analyses (Ciardullo & Perseghin, 2021; Unalp-Arida & Ruhl, 2025; Zhang et al., 2021). Demonstrating that the direction of the primary conclusion did not depend on CAP ≥ 285 dB/m supports robustness while underscoring the need for transparent threshold reporting.

Strengths

This study has a number of merits. NHANES has a large probability sample and allows to make estimates that adjust for the complex survey design of the U.S. survey. Fibrosis was evaluated by VCTE and not by liver diagnosis based on a questionnaire and both dichotomous fibrosis status and continuous LSM were evaluated. The exposures weight status, weight-loss attempt, actual recent weight change, and individual strategies were analysed separately, thus preventing the assumption of interchangeability of these conceptually different exposures. Additionally, clinically relevant covariates were added to the study, multiple testing was accounted for in the analyses by method, and alternate definitions of steatosis were tested.

Limitations

Several limitations should be considered. First, the cross-sectional design does not permit conclusions about temporal direction or causality. Second, weight history, weight-loss attempts and methods, smoking status, and diabetes history included self-reported measures and were therefore susceptible to recall or classification error. Third, VCTE is a validated noninvasive tool but is not equivalent to liver biopsy, and LSM can be influenced by factors other than fibrosis. Fourth, CAP and LSM cut-points vary across studies; sensitivity analyses examined alternative steatosis thresholds, but no threshold provides perfect classification. Fifth, the BMI <25 kg/m² reference group was small and contained few advanced-fibrosis events, producing wide confidence intervals. Sixth, complete-case regression may introduce selection bias if missingness was related to both exposures and outcomes. Finally, the data could not fully characterise

the intensity, duration, adherence, or long-term maintenance of each weight-loss strategy and could not always distinguish intentional from illness-related weight loss.

Recommendations

Clinical assessment should distinguish a reported intention to lose weight from objectively achieved and maintained weight loss. Weight-management programmes for adults with MASLD should monitor weight trajectory, metabolic response, and fibrosis risk rather than treating a past-year attempt as evidence of effective intervention. Future longitudinal studies should include repeated measurements of weight, body composition, adherence, and liver stiffness or histology to identify which sustained weight-loss trajectories and behavioural changes are associated with fibrosis regression.

Conclusion

Obesity was strongly and consistently associated with higher VCTE-defined liver fibrosis in this nationally representative cross-sectional study of U.S. adults with MASLD. Significant and advanced fibrosis increased progressively across BMI categories, and the obesity association persisted after adjustment for demographic and clinical factors. In contrast, recent $\geq 5\%$ weight loss, a past-year weight-loss attempt, and individual self-reported methods were not independently associated with lower fibrosis burden after adjustment and multiple-testing correction. These results do not imply that weight reduction is ineffective for MASLD; they show that a recently reported attempt is an inadequate substitute for objectively measured, sustained weight loss. The findings reinforce the importance of obesity and cardiometabolic risk in assessing fibrosis burden and support longitudinal research linking durable weight loss with subsequent liver stiffness and clinically relevant fibrosis outcomes.

Declarations

Ethics Approval and Consent to Participate

NHANES was approved by the National Center for Health Statistics Ethics Review Board under Protocols #2011-17 and #2018-01, and all participants provided documented informed consent. This secondary analysis used publicly available, de-identified data and required no direct participant contact.

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Conflicts of Interest

The authors declare no competing interests.

Data Availability

The NHANES 2017-March 2020 pre-pandemic data and documentation are publicly available from the National Center for Health Statistics website.

References

- Angulo, P., Kleiner, D. E., Dam-Larsen, S., Adams, L. A., Bjornsson, E. S., Charatchoenwithaya, P., Mills, P. R., Keach, J. C., Lafferty, H. D., Stahler, A., Haflidadottir, S., & Bendtsen, F. (2015). Liver fibrosis, but no other histologic features, is associated with long-term outcomes of patients with nonalcoholic fatty liver disease. *Gastroenterology*, 149(2), 389-397.e10. <https://doi.org/10.1053/j.gastro.2015.04.043>
- Brunt, E. M., Wong, V. W.-S., Nobili, V., Day, C. P., Sookoian, S., Maher, J. J., Bugianesi, E., Sirlin, C. B., Neuschwander-Tetri, B. A., & Rinella, M. E. (2015). Nonalcoholic fatty liver disease. *Nature Reviews Disease Primers*, 1, 15080. <https://doi.org/10.1038/nrdp.2015.80>
- Buzzetti, E., Pinzani, M., & Tsochatzis, E. A. (2016). The multiple-hit pathogenesis of non-alcoholic fatty liver disease. *Metabolism*, 65(8), 1038-1048. <https://doi.org/10.1016/j.metabol.2015.12.012>
- Byrne, C. D., & Targher, G. (2015). NAFLD: A multisystem disease. *Journal of Hepatology*, 62(1 Suppl), S47-S64. <https://doi.org/10.1016/j.jhep.2014.12.012>
- Cao, Y.-T., Xiang, L.-L., Qi, F., Zhang, Y.-J., Chen, Y., & Zhou, X.-Q. (2022). Accuracy of controlled attenuation parameter and liver stiffness measurement for assessing steatosis and fibrosis in non-alcoholic fatty liver disease: A systematic review and meta-analysis. *EClinicalMedicine*, 51, 101547. <https://doi.org/10.1016/j.eclinm.2022.101547>
- Castera, L., Friedrich-Rust, M., & Loomba, R. (2019). Noninvasive assessment of liver disease in patients with nonalcoholic fatty liver disease. *Gastroenterology*, 156(5), 1264-1281.e4. <https://doi.org/10.1053/j.gastro.2018.12.036>
- Chalasani, N., Younossi, Z., Lavine, J. E., Charlton, M., Cusi, K., Rinella, M., Harrison, S. A., Brunt, E. M., & Sanyal, A. J. (2018). The diagnosis and management of nonalcoholic fatty liver disease: Practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*, 67(1), 328-357. <https://doi.org/10.1002/hep.29367>
- Ciardullo, S., & Perseghin, G. (2021). Prevalence of NAFLD, MAFLD and associated advanced fibrosis in the contemporary United States population. *Liver International*, 41(6), 1290-1293. <https://doi.org/10.1111/liv.14828>
- Dulai, P. S., Singh, S., Patel, J., Soni, M., Prokop, L. J., Younossi, Z., Sebastiani, G., Ekstedt, M., Hagstrom, H., Nasr, P., Stal, P., Wong, V. W.-S., Kechagias, S., Hultcrantz, R., & Loomba, R. (2017). Increased risk of mortality by fibrosis stage in nonalcoholic fatty liver disease: Systematic review and meta-analysis. *Hepatology*, 65(5), 1557-1565. <https://doi.org/10.1002/hep.29085>
- Eddowes, P. J., Sasso, M., Allison, M., Tsochatzis, E., Anstee, Q. M., Sheridan, D., Guha, I. N., Cobbold, J. F., Deeks, J. J., Paradis, V., Bedossa, P., & Newsome, P. N. (2019). Accuracy of FibroScan controlled attenuation parameter and liver stiffness measurement in assessing steatosis and fibrosis in patients with nonalcoholic fatty liver disease. *Gastroenterology*, 156(6), 1717-1730. <https://doi.org/10.1053/j.gastro.2019.01.042>
- Ekstedt, M., Hagstrom, H., Nasr, P., Fredrikson, M., Stal, P., Kechagias, S., & Hultcrantz, R. (2015). Fibrosis stage is the strongest predictor for disease-specific mortality in NAFLD after up to 33 years of follow-up. *Hepatology*, 61(5), 1547-1554. <https://doi.org/10.1002/hep.27368>
- European Association for the Study of the Liver, European Association for the Study of Diabetes, & European Association for the Study of Obesity. (2024). EASL-EASD-EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). *Journal of Hepatology*, 81(3), 492-542. <https://doi.org/10.1016/j.jhep.2024.04.031>
- Friedman, S. L., Neuschwander-Tetri, B. A., Rinella, M., & Sanyal, A. J. (2018). Mechanisms of NAFLD development and therapeutic strategies. *Nature Medicine*, 24(7), 908-922. <https://doi.org/10.1038/s41591-018-0104-9>
- Hagstrom, H., Nasr, P., Ekstedt, M., Hammar, U., Stal, P., Hultcrantz, R., & Kechagias, S. (2017). Fibrosis stage but not NASH predicts mortality and time to development of severe liver disease in biopsy-proven NAFLD. *Journal of Hepatology*, 67(6), 1265-1273. <https://doi.org/10.1016/j.jhep.2017.07.027>
- Hallsworth, K., Fattakhova, G., Hollingsworth, K. G., Thoma, C., Moore, S., Taylor, R., Day, C. P., & Trenell, M. I. (2011). Resistance exercise reduces liver fat and its mediators in non-alcoholic fatty liver disease independent of weight loss. *Gut*, 60(9), 1278-1283. <https://doi.org/10.1136/gut.2011.242073>
- Han, L., You, D., Zeng, F., Feng, X., Astell-Burt, T., Duan, S., & Qi, L. (2019). Trends in self-perceived weight status, weight loss attempts, and weight loss strategies among adults in the United States, 1999-2016. *JAMA Network Open*, 2(11), e1915219. <https://doi.org/10.1001/jamanetworkopen.2019.15219>
- Karlas, T., Petroff, D., Sasso, M., Fan, J.-G., Mi, Y.-Q., de Ledinghen, V., Kumar, M., Lupsor-Platon, M., Han, K.-H., Cardoso, A. C., Ferraioli, G., Chan, W.-K., Wong, V. W.-S., Myers, R. P.,

- Chayama, K., Friedrich-Rust, M., Beaugrand, M., Shen, F., Hiriart, J.-B., ... Wiegand, J. (2017). Individual patient data meta-analysis of controlled attenuation parameter technology for assessing steatosis. *Journal of Hepatology*, 66(5), 1022-1030. <https://doi.org/10.1016/j.jhep.2016.12.022>
- Kim, Y., Chang, Y., Cho, Y. K., Ahn, J., Shin, H., & Ryu, S. (2019). Obesity and weight gain are associated with progression of fibrosis in patients with nonalcoholic fatty liver disease. *Clinical Gastroenterology and Hepatology*, 17(3), 543-550.e2. <https://doi.org/10.1016/j.cgh.2018.07.006>
- Koutoukidis, D. A., Astbury, N. M., Tudor, K. E., Morris, E., Henry, J. A., Noreik, M., Jebb, S. A., & Aveyard, P. (2019). Association of weight loss interventions with changes in biomarkers of nonalcoholic fatty liver disease: A systematic review and meta-analysis. *JAMA Internal Medicine*, 179(9), 1262-1271. <https://doi.org/10.1001/jamainternmed.2019.2248>
- Lee, D. H. (2017). Imaging evaluation of non-alcoholic fatty liver disease: Focused on quantification. *Clinical and Molecular Hepatology*, 23(4), 290-301. <https://doi.org/10.3350/cmh.2017.0042>
- Leung, J. C.-F., Loong, T. C.-W., Wei, J. L., Wong, G. L.-H., Chan, A. W.-H., Choi, P. C.-L., Shu, S. S.-T., Chim, A. M.-L., Chan, H. L.-Y., & Wong, V. W.-S. (2017). Histological severity and clinical outcomes of nonalcoholic fatty liver disease in nonobese patients. *Hepatology*, 65(1), 54-64. <https://doi.org/10.1002/hep.28697>
- Lomonaco, R., Ortiz-Lopez, C., Orsak, B., Finch, J., Webb, A., Bril, F., Loudon, C., Tio, F., & Cusi, K. (2011). Role of ethnicity in overweight and obese patients with nonalcoholic steatohepatitis. *Hepatology*, 54(3), 837-845. <https://doi.org/10.1002/hep.24483>
- Lomonaco, R., Ortiz-Lopez, C., Orsak, B., Webb, A., Hardies, J., Darland, C., Finch, J., Gastaldelli, A., Harrison, S., Tio, F., & Cusi, K. (2012). Effect of adipose tissue insulin resistance on metabolic parameters and liver histology in obese patients with nonalcoholic fatty liver disease. *Hepatology*, 55(5), 1389-1397. <https://doi.org/10.1002/hep.25539>
- Marchesini, G., Brizi, M., Bianchi, G., Tomassetti, S., Bugianesi, E., Lenzi, M., McCullough, A. J., Natale, S., Forlani, G., & Melchionda, N. (2001). Nonalcoholic fatty liver disease: A feature of the metabolic syndrome. *Diabetes*, 50(8), 1844-1850. <https://doi.org/10.2337/diabetes.50.8.1844>
- Mokari-Yamchi, A., & Rosenkranz, R. R. (2024). Demographic correlates of weight-loss strategies in US adults: Cross-sectional analysis of NHANES data 2017-2020. *Preventive Medicine Reports*, 46, 102873. <https://doi.org/10.1016/j.pmedr.2024.102873>
- National Center for Health Statistics. (2020). National Health and Nutrition Examination Survey: 2017-March 2020 pre-pandemic data files and documentation. Centers for Disease Control and Prevention.
- National Center for Health Statistics. (2026). Ethics Review Board approval. Centers for Disease Control and Prevention. <https://www.cdc.gov/nchs/nhanes/about/erb.html>
- Newsome, P. N., Sasso, M., Deeks, J. J., Paredes, A., Boursier, J., Chan, W.-K., Yilmaz, Y., Czernichow, S., Zheng, M.-H., Wong, V. W.-S., Allison, M., Tsochatzis, E., Anstee, Q. M., Sheridan, D. A., Eddowes, P. J., Guha, I. N., Cobbald, J. F., Paradis, V., Bedossa, P., ... Harrison, S. A. (2020). FibroScan-AST score for the non-invasive identification of patients with non-alcoholic steatohepatitis with significant activity and fibrosis: A prospective derivation and global validation study. *The Lancet Gastroenterology & Hepatology*, 5(4), 362-373. [https://doi.org/10.1016/S2468-1253\(19\)30383-8](https://doi.org/10.1016/S2468-1253(19)30383-8)
- Noureddin, M., Ntanios, F., Malhotra, D., Hoover, K., Emir, B., McLeod, E., & Alkhouiri, N. (2022). Predicting NAFLD prevalence in the United States using National Health and Nutrition Examination Survey 2017-2018 transient elastography data and application of machine learning. *Hepatology Communications*, 6(7), 1537-1548. <https://doi.org/10.1002/hep4.1935>
- Oeda, S., Takahashi, H., Imajo, K., Seko, Y., Ogawa, Y., Moriguchi, M., Yoneda, M., Anzai, K., Aishima, S., Kage, M., Itoh, Y., Nakajima, A., & Eguchi, Y. (2020). Accuracy of liver stiffness measurement and controlled attenuation parameter using FibroScan M/XL probes to diagnose liver fibrosis and steatosis in patients with nonalcoholic fatty liver disease: A multicenter prospective study. *Journal of Gastroenterology*, 55(4), 428-440. <https://doi.org/10.1007/s00535-019-01635-0>
- Polyzos, S. A., Kountouras, J., & Mantzoros, C. S. (2019). Obesity and nonalcoholic fatty liver disease: From pathophysiology to therapeutics. *Metabolism*, 92, 82-97. <https://doi.org/10.1016/j.metabol.2018.11.014>
- Powell, E. E., Wong, V. W.-S., & Rinella, M. (2021). Non-alcoholic fatty liver disease. *The Lancet*, 397(10290), 2212-2224.

- [https://doi.org/10.1016/S0140-6736\(20\)32511-3](https://doi.org/10.1016/S0140-6736(20)32511-3)
- Promrat, K., Kleiner, D. E., Niemeier, H. M., Jackvony, E., Kearns, M., Wands, J. R., Fava, J. L., & Wing, R. R. (2010). Randomized controlled trial testing the effects of weight loss on nonalcoholic steatohepatitis. *Hepatology*, 51(1), 121-129. <https://doi.org/10.1002/hep.23276>
- Pu, K., Wang, Y., Bai, S., Wei, H., Zhou, Y., Fan, J., & Qiao, L. (2019). Diagnostic accuracy of controlled attenuation parameter as a non-invasive test for steatosis in suspected non-alcoholic fatty liver disease: A systematic review and meta-analysis. *BMC Gastroenterology*, 19, 51. <https://doi.org/10.1186/s12876-019-0961-9>
- Riazi, K., Azhari, H., Charette, J. H., Underwood, F. E., King, J. A., Afshar, E. E., Swain, M. G., Congly, S. E., Kaplan, G. G., & Shaheen, A.-A. (2022). The prevalence and incidence of NAFLD worldwide: A systematic review and meta-analysis. *The Lancet Gastroenterology & Hepatology*, 7(9), 851-861. [https://doi.org/10.1016/S2468-1253\(22\)00165-0](https://doi.org/10.1016/S2468-1253(22)00165-0)
- Rinella, M. E., Neuschwander-Tetri, B. A., Siddiqui, M. S., Abdelmalek, M. F., Caldwell, S., Barb, D., Kleiner, D. E., & Loomba, R. (2023a). AASLD practice guidance on the clinical assessment and management of nonalcoholic fatty liver disease. *Hepatology*, 77(5), 1797-1835. <https://doi.org/10.1097/HEP.0000000000000323>
- Rinella, M. E., Lazarus, J. V., Ratziu, V., Francque, S. M., Sanyal, A. J., Kanwal, F., Romero, D., Abdelmalek, M. F., Anstee, Q. M., Arab, J. P., Arrese, M., Bataller, R., Beuers, U., Boursier, J., Bugianesi, E., Byrne, C. D., Castro Narro, G. E., Chowdhury, A., Cortez-Pinto, H., Newsome, P. N. (2023b). A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *Hepatology*, 78(6), 1966-1986. <https://doi.org/10.1097/HEP.0000000000000520>
- Romero-Gomez, M., Zelber-Sagi, S., & Trenell, M. (2017). Treatment of NAFLD with diet, physical activity and exercise. *Journal of Hepatology*, 67(4), 829-846. <https://doi.org/10.1016/j.jhep.2017.05.016>
- Sanyal, A. J., Van Natta, M. L., Clark, J., Neuschwander-Tetri, B. A., Diehl, A., Dasarathy, S., Loomba, R., Chalasani, N., Kowdley, K., Hameed, B., Wilson, L. A., Yates, K. P., Belt, P., Lazo, M., Kleiner, D. E., Behling, C., & Tonascia, J. (2021). Prospective study of outcomes in adults with nonalcoholic fatty liver disease. *New England Journal of Medicine*, 385(17), 1559-1569. <https://doi.org/10.1056/NEJMoa2029349>
- Sayiner, M., Koenig, A., Henry, L., & Younossi, Z. M. (2016). Epidemiology of nonalcoholic fatty liver disease and nonalcoholic steatohepatitis in the United States and the rest of the world. *Clinics in Liver Disease*, 20(2), 205-214. <https://doi.org/10.1016/j.cld.2015.10.001>
- Schwabe, R. F., Tabas, I., & Pajvani, U. B. (2020). Mechanisms of fibrosis development in nonalcoholic steatohepatitis. *Gastroenterology*, 158(7), 1913-1928. <https://doi.org/10.1053/j.gastro.2019.11.311>
- Schwabe, R. F., Tabas, I., & Pajvani, U. B. (2021). Molecular and cellular mechanisms of liver fibrosis and its regression. *Nature Reviews Gastroenterology & Hepatology*, 18, 151-166. <https://doi.org/10.1038/s41575-020-00372-7>
- Taylor, R. S., Taylor, R. J., Bayliss, S., Hagstrom, H., Nasr, P., Schattenberg, J. M., Ishigami, M., Toyoda, H., Wong, V. W.-S., Peleg, N., Shlomain, A., Sebastiani, G., Seko, Y., Bhala, N., Younossi, Z. M., Anstee, Q. M., McPherson, S., & Newsome, P. N. (2020). Association between fibrosis stage and outcomes of patients with nonalcoholic fatty liver disease: A systematic review and meta-analysis. *Gastroenterology*, 158(6), 1611-1625.e12. <https://doi.org/10.1053/j.gastro.2020.01.043>
- Unalp-Arida, A., & Ruhl, C. E. (2025). Prevalence of metabolic dysfunction-associated steatotic liver disease and fibrosis defined by liver elastography in the United States using National Health and Nutrition Examination Survey 2017-March 2020 and August 2021-August 2023 data. *Hepatology*, 82(5), 1256-1273. <https://doi.org/10.1097/HEP.0000000000000121>
- Vilar-Gomez, E., Martinez-Perez, Y., Calzadilla-Bertot, L., Torres-Gonzalez, A., Gra-Oramas, B., Gonzalez-Fabian, L., Friedman, S. L., Diago, M., & Romero-Gomez, M. (2015). Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. *Gastroenterology*, 149(2), 367-378.e5. <https://doi.org/10.1053/j.gastro.2015.04.005>
- Wong, V. W.-S., Vergniol, J., Wong, G. L.-H., Foucher, J., Chan, H. L.-Y., Le Bail, B., Choi, P. C.-L., Kowo, M., Chan, A. W.-H., Merrouche, W., Sung, J. J.-Y., & de Ledinghen, V. (2010). Diagnosis of fibrosis and cirrhosis using liver stiffness measurement in nonalcoholic fatty liver disease. *Hepatology*, 51(2), 454-462. <https://doi.org/10.1002/hep.23312>
- Wijarnpreecha, K., Aby, E. S., Ahmed, A., & Kim, D. (2022). The association of weight gain with nonalcoholic fatty liver disease and fibrosis

- detected by FibroScan in the United States. *Annals of Gastroenterology*, 35(2), 194-202. <https://doi.org/10.20524/aog.2022.0687>
- Younossi, Z. M., Anstee, Q. M., Marietti, M., Hardy, T., Henry, L., Eslam, M., George, J., & Bugianesi, E. (2018). Global burden of NAFLD and NASH: Trends, predictions, risk factors and prevention. *Nature Reviews Gastroenterology & Hepatology*, 15(1), 11-20. <https://doi.org/10.1038/nrgastro.2017.109>
- Younossi, Z. M., Koenig, A. B., Abdelatif, D., Fazel, Y., Henry, L., & Wymer, M. (2016). Global epidemiology of nonalcoholic fatty liver disease: Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*, 64(1), 73-84. <https://doi.org/10.1002/hep.28431>
- Zelber-Sagi, S., Salomone, F., & Mlynarsky, L. (2017). The Mediterranean dietary pattern as the diet of choice for non-alcoholic fatty liver disease: Evidence and plausible mechanisms. *Liver International*, 37(7), 936-949. <https://doi.org/10.1111/liv.13435>
- Zhang, X., Heredia, N. I., Balakrishnan, M., & Thrift, A. P. (2021). Prevalence and factors associated with NAFLD detected by vibration controlled transient elastography among US adults: Results from NHANES 2017-2018. *PLOS ONE*, 16(6), e0252164. <https://doi.org/10.1371/journal.pone.0252164>