

Association of the Planetary Health Diet 2.0 with risk of metabolic dysfunction-associated steatotic liver disease

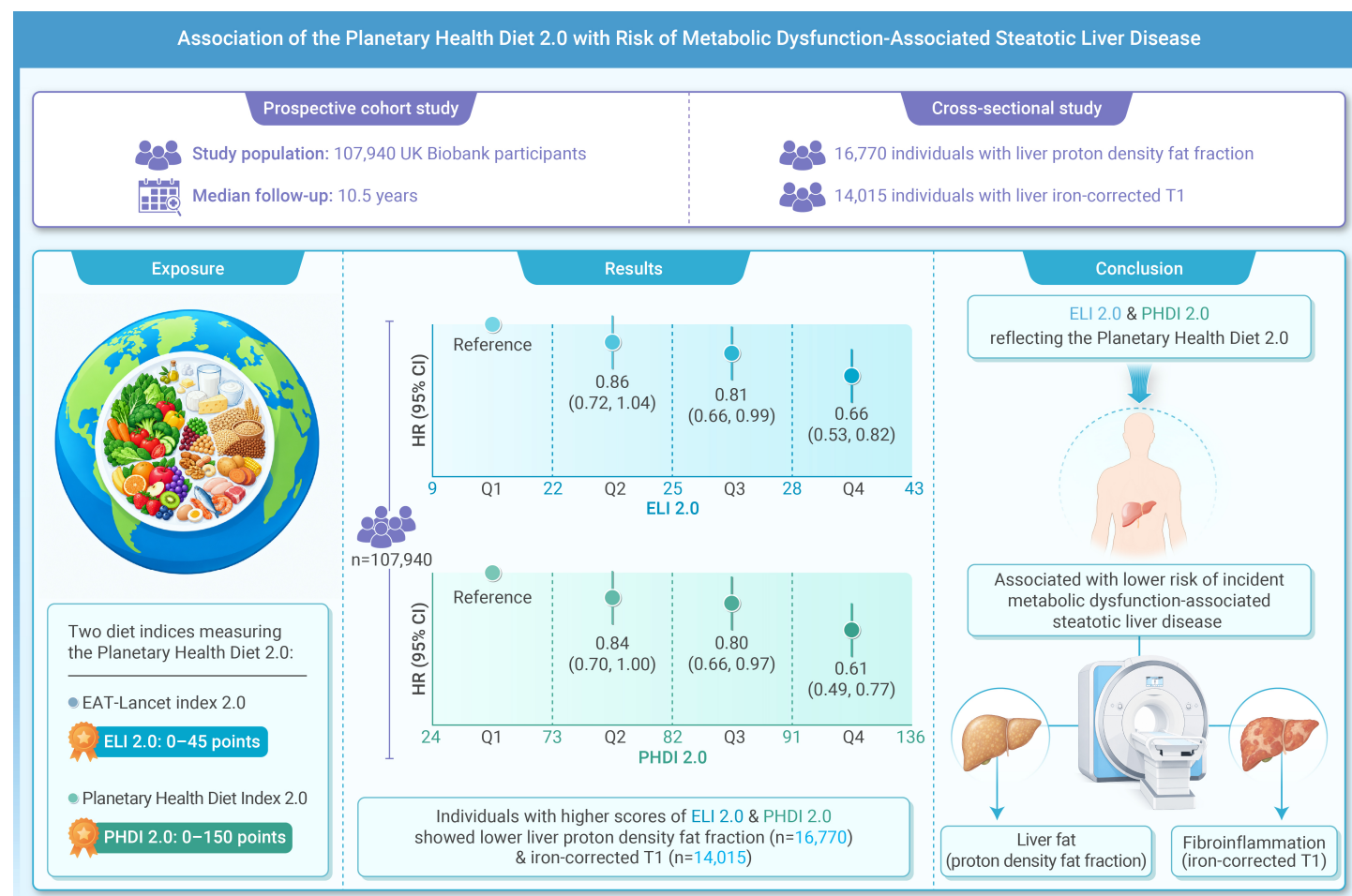
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GRAPHICAL ABSTRACT



PUBLIC SUMMARY

- Planetary Health Diet 2.0 is a sustainable dietary framework designed to promote human and planetary health.
- This diet emphasizes plant-based foods while limiting animal-source foods, saturated fat, sugar, and sodium.
- Adherence to this diet is related to lower risk of metabolic dysfunction-associated steatotic liver disease.
- Magnetic resonance imaging results support the hepatoprotective link of the Planetary Health Diet 2.0.

Association of the Planetary Health Diet 2.0 with risk of metabolic dysfunction-associated steatotic liver disease

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The second EAT-Lancet Commission in 2025 updated the Planetary Health Diet (PHD 2.0) to support the transition toward healthy, sustainable, and just food systems. However, little is known regarding its association with metabolic dysfunction-associated steatotic liver disease (MASLD). This prospective cohort study analyzed data from 107,940 participants who completed at least two 24-hour dietary assessments within the UK Biobank. Adherence to the PHD 2.0 was quantified using the EAT-Lancet index 2.0 (range: 0–45 points) and modified Planetary Health Diet Index (range: 0–150 points). Incident MASLD was identified through electronic health records. In addition, proton density fat fraction ($n = 16,770$) and iron-corrected T1 ($n = 14,015$) were assessed using magnetic resonance imaging. Associations were analyzed using Cox proportional hazards models and logistic models. Over a median follow-up of 10.5 years, 775 developed MASLD. The fully adjusted hazard ratios (95% confidence intervals) for incident MASLD comparing the highest with the lowest quartiles were 0.66 (0.53, 0.82) for the EAT-Lancet index 2.0 and 0.61 (0.49, 0.77) for the modified Planetary Health Diet Index, respectively (P for trend < 0.0001). Moreover, both diet indices showed inverse associations with hepatic steatosis defined by proton density fat fraction (odds ratios [95% confidence intervals]: 0.56 [0.50, 0.63] and 0.59 [0.53, 0.66] for comparing the extreme quartiles) and elevated iron-corrected T1 (odds ratios [95% confidence intervals]: 0.63 [0.49, 0.80] and 0.61 [0.48, 0.78]). In conclusion, greater adherence to the PHD 2.0 is associated with a decreased risk of MASLD as well as favorable magnetic resonance imaging biomarkers.

INTRODUCTION

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly referred to as non-alcoholic fatty liver disease, currently affects approximately 30% to 40% of the adult population worldwide.¹ Beyond liver-related events such as cirrhosis and hepatocellular carcinoma, MASLD substantially increases the risks of type 2 diabetes, chronic kidney disease, cardiovascular disease, and all-cause mortality.^{1–2} However, to date, there are only two drugs (resmetirom and semaglutide) approved specifically for the treatment of non-cirrhotic metabolic dysfunction-associated steatohepatitis with moderate-to-advanced fibrosis.¹ It is important to note that these medications offer limited efficacy, and their long-term safety profile remains unclear.³ Therefore, a key focus of reducing the global impact of MASLD is prevention.

Diet is a key modifiable risk factor in the prevention of MASLD.⁴ In addition, a healthy diet should consider the environmental impact because food production is a major contributor to global climate change.⁵ In this context, the EAT-Lancet Commission in 2019 proposed a healthy global reference diet, the Planetary Health Diet (PHD), to jointly promote human health and environmental sustainability.⁶ This diet emphasizes plant-based foods (e.g., whole grains, vegetables, fruits, nuts, and legumes), with modest intake of fish, poultry, dairy, and eggs, while limiting meat, added sugars, and saturated fats.⁶ Moreover, our previous studies have demonstrated that adherence to the PHD was associated with lower risks of MASLD,^{7–8} and cardiometabolic diseases.⁹ In October 2025, the EAT-Lancet Commission provided an expanded scientific foundation for the PHD, designed to support human and planetary health as well as just food systems.¹⁰ Compared with the original PHD,⁶ the updated version (PHD 2.0) refines food group-specific intake targets and includes sodium as a separate evaluation criterion.¹⁰

Moreover, accumulating evidence suggests that excessive sodium intake may contribute to the onset and progression of MASLD.^{11–12} To quantify adherence to the PHD 2.0, we recently developed the EAT-Lancet index 2.0 (ELI 2.0).¹³ However, the association between adherence to the ELI 2.0 and risk of MASLD remains unclear.

In the current study, we investigated the association between adherence to the PHD 2.0 and risk of incident MASLD in a population-based setting. Adherence to the PHD 2.0 was assessed using two different indices: the ELI 2.0 developed in our previous work,¹³ and a modified Planetary Health Diet Index (PHDI 2.0) based on the index reflecting the PHD 1.0 originally developed by Bui and colleagues.¹⁴ The original PHDI was based on the reported biological dose-response associations between each food component and risks of major chronic diseases,¹⁴ and our previous work has shown that this index had a stronger association with MASLD than the original ELI.⁸ In addition, the two indices enabled a comprehensive evaluation of adherence to the reference diet across different methodological frameworks. We hypothesized that individuals with diets most consistent with the PHD 2.0 would have a reduced risk of MASLD.

METHODS

Study population

The UK Biobank is a large-scale, population-based cohort comprising more than 500,000 participants aged 40–69 years at recruitment (2006–2010) from 22 assessment centers across the UK.¹⁵ At recruitment, participants completed standardized touchscreen questionnaires and underwent physical examinations. Between April 2009 and June 2012, a subset of the participants filled out up to five web-based 24-hour dietary assessments, using the Oxford WebQ tool. Ethical approval for the study protocol was granted by the North West Multicentre Research Ethics Committee (reference number: 11/NW/0382), and all participants provided written informed consent.

The primary prospective cohort analysis consisted of 107,940 participants after excluding those with fewer than two 24-hour dietary recalls (to represent usual dietary intake, $n = 375,672$), implausible total energy intake (males: < 800 or $> 4,200$ kcal/day; females: < 600 or $> 3,500$ kcal/day;^{14,16} $n = 675$), prevalent cardiovascular disease ($n = 5,546$), cancer ($n = 10,439$), liver diseases ($n = 1,930$), and those lost to follow-up ($n = 325$). For secondary cross-sectional analysis with liver proton density fat fraction (PDFF, $n = 16,770$) and iron-corrected T1 (cT1, $n = 14,015$), we further excluded individuals with missing respective data. The selection flow of the study sample is shown in Figure S1. The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline,¹⁷ and the STROBE-nut guideline.¹⁸

Assessment of the PHD

Daily intakes of food groups, nutrients, and total energy were estimated using the mean values of available repeated measurements from the validated Oxford WebQ online 24-hour dietary questionnaire.¹⁹ The calculation details have been published elsewhere.^{20–21} Different components for each diet index, as well as the criteria for scoring of the original diet indices reflecting adherence to the PHD 1.0, can be found in our previous publication.⁸ Consistent with our recent work,¹³ the ELI 2.0 was created based on 15 food components. Briefly, each component was graded from 0 to 3 points according to predefined target intakes and/or reference intervals, resulting in a total

score ranging from 0 to 45 points (Table S1). Compared with ELI 1.0, the ELI 2.0 incorporated updated intake targets, revised reference ranges, and structural modifications to the dietary framework, including the consolidation of red meat categories and the inclusion of sodium as a new component. Furthermore, we built the PHDI 2.0 based on the original PHDI,¹⁴ and the second EAT-Lancet Commission report.¹⁰ Compared with the original PHDI (score range: 0–140 points),¹⁴ the PHDI 2.0 incorporated sodium intake as an additional component, scored from 0 to 10 points based on intake levels: 0 points for ≥ 4 g/day, 10 points for ≤ 2 g/day, and proportional scores for intermediate intakes, totaling a score ranging from 0 to 150 points (Table S2). The main differences between the two indices reflecting the same dietary pattern were that the PHDI covered a broader range of points and was based on associations with diseases.

Ascertainment of liver disease and follow-up

Prevalent liver diseases were identified by self-reported codes and the International Classification of Diseases, Tenth Revision codes. Table S3 details the diagnostic codes. The primary outcome was the occurrence of incident MASLD, which was defined according to the International Classification of Diseases, Tenth Revision codes K76.0 and K75.8.²² The follow-up period commenced on the date of completion of the first 24-hour dietary assessment and extended until the first occurrence of MASLD diagnosis, death, or the censoring date (September 30, 2021 in England; February 28, 2018 in Wales; and July 31, 2021 in Scotland), whichever occurred earliest.

Assessment of liver magnetic resonance imaging

Between 2014 and 2020, a subsample underwent liver magnetic resonance imaging scans. Hepatic fat content was quantified using PDFF,²³ and fibroinflammation was assessed by cT1.²⁴ As secondary outcomes, hepatic steatosis was defined as PDFF $\geq 5\%$,²⁵ while elevated cT1 was defined as ≥ 800 ms.²⁴

Assessment of covariates

Information on age (continuous: years), sex (male or female), ethnicity (white or non-white), Townsend deprivation index (higher scores reflecting greater deprivation, continuous), and smoking status (current, former, or never) was collected using a standardized touchscreen questionnaire at recruitment. Alcohol intake (g/day) was derived from 24-hour dietary assessments.²⁰ Physical activity (continuous: metabolic equivalent hours/week) was assessed by the short-form International Physical Activity Questionnaire.²⁶ Height and weight were measured by trained staff to calculate body mass index (BMI, kg/m²). Hypertension was defined as self-reported, physician diagnosis, use of antihypertensive medication, or blood pressure $\geq 140/90$ mmHg. Diabetes was defined as self-reported, physician diagnosis, or insulin use. Hyperlipidemia was defined as self-reported high cholesterol or use of medication for cholesterol.

Statistical analysis

Normality of continuous variables was evaluated using the Kolmogorov-Smirnov test and quantile-quantile plots. Continuous variables were presented as medians (P25, P75) due to their non-normal distributions, while categorical variables were summarized as frequencies (percentages). Spearman correlation coefficient was calculated between ELI 2.0 and PHDI 2.0.

Cox proportional hazards regression models were utilized to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the associations of ELI 2.0 and PHDI 2.0 scores (modelled as quartiles and per 10% increment) with the risk of incident MASLD. Models were adjusted incrementally for covariates to minimize over-adjustment bias.²⁷ Model 1 was unadjusted. Model 2 was adjusted for age, sex, ethnicity, and Townsend deprivation index. Model 3 was additionally adjusted for smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, and BMI. The proportional hazards assumption was verified using Schoenfeld residuals for each model, with no violations detected (P values for global test > 0.05). Tests for linear trends were performed by assigning the median value of each quartile as a continuous variable in the regression models. To assess the potential dose-response associations between the two diet indices and

MASLD, restricted cubic splines were performed with four knots positioned at the 5th, 35th, 65th, and 95th percentiles of the exposures, with the median value as the reference point.²⁸ Missing values in covariates were handled using multiple imputation by chained equations. Furthermore, potential effect modification was assessed by including interaction terms between diet scores and covariates in the final models. For covariates with statistically significant interactions, stratified analyses were further performed.

To confirm the robustness of the findings, several sensitivity analyses were performed. First, a complete case analysis was conducted by excluding participants with missing covariate data. Second, to improve the estimation of habitual dietary intake, the analytic population was restricted to those who had completed at least three 24-hour dietary assessments. Third, to minimize the potential impact of reverse causality, participants diagnosed with incident MASLD within the first two years of follow-up were excluded. Fourth, E -values were calculated to quantify the potential influence on the observed associations.^{29–30} Finally, to test whether the inclusion of sodium in PHD 2.0 improved the prediction of incident MASLD compared with PHD 1.0, which does not include sodium, we computed the c-index differences between models with PHD 2.0 and those with PHD 1.0,^{31–32} as well as the net reclassification improvement index.³³

Logistic regression models were used to examine the associations of both diet indices (quartiles and per 10% increment) with odds of PDFF-defined hepatic steatosis and elevated cT1, adjusting for the same covariates as in the three Cox models above. In addition, considering the multi-year gap between dietary assessment (2009–2012) and magnetic resonance imaging scan (2014–2020), we further adjusted for the diet assessment-to-magnetic resonance imaging time interval in the final model.

Analyses were carried out using SAS software version 9.4 (SAS Institute Inc., Cary, NC, USA) and R version 4.4.2 (R Foundation for Statistical Computing). All statistical tests were two-sided, and the threshold for statistical significance was defined at an alpha level of 0.05.

RESULTS

Baseline characteristics of study participants

Among the 107,940 participants, the median (P25, P75) age was 57.0 (49.0, 62.0) years, and 43.8% were males. Overall, participants in the highest quartiles of both ELI 2.0 and PHDI 2.0 (Spearman correlation coefficient = 0.91, $P < 0.0001$) tended to be older, more likely to be female, and more physically active, but less likely to be current smokers than those in the lowest quartiles. They also tended to have a lower BMI, total energy intake, and alcohol intake, as well as a lower prevalence of hypertension, hyperlipidemia, and diabetes (Table 1).

Associations of ELI 2.0 and PHDI 2.0 with risk of incident MASLD

During a median follow-up of 10.5 (9.94, 10.6) years and 1,077,026 person-years, 775 incident MASLD cases were identified. Both ELI 2.0 and PHDI 2.0 were inversely associated with the risk of incident MASLD across three models (Table 2). In the fully adjusted models, the HRs (95% CIs) for MASLD risk across increasing quartiles of the ELI 2.0 were 1.00 (reference) for quartile 1, 0.86 (0.72, 1.04) for quartile 2, 0.81 (0.66, 0.99) for quartile 3, and 0.66 (0.53, 0.82) for quartile 4 (P for trend < 0.0001). The corresponding HRs (95% CIs) for the PHDI 2.0 were 1.00 (reference), 0.84 (0.70, 1.00), 0.80 (0.66, 0.97), and 0.61 (0.49, 0.77), with P for trend < 0.0001 . In addition, the mostly adjusted HRs (95% CIs) per 10% increment in diet scores for MASLD risk were 0.87 (0.82, 0.92) for ELI 2.0 and 0.85 (0.80, 0.91) for PHDI 2.0, respectively. Furthermore, the two diet scores showed linear and inverse associations with the risk of incident MASLD (P for overall < 0.001 and P for non-linearity ≥ 0.46 , Figure 1).

Effect modification and subgroup analyses

No significant effect modification by covariates was observed for the associations between diet indices and MASLD risk in the fully adjusted models, except for ethnicity, hypertension, and BMI (Table S4). Specifically, the inverse associations of ELI 2.0 and PHDI 2.0 with MASLD risk were more pronounced among white participants, individuals without hypertension, and those with BMI ≥ 25 kg/m² compared with their respective counterparts (Table S5).

Table 1. Baseline characteristics of the study participants according to quartiles of the Planetary Health Diet 2.0 (n = 107,940). ¹

Characteristics	Categories of the diet indices			
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
EAT-Lancet index 2.0				
Diet score	20.0 (18.0, 21.0)	23.0 (22.0, 24.0)	26.0 (25.0, 27.0)	30.0 (28.0, 31.0)
Number of participants	24,649	27,454	27,236	28,601
Age (years)	55.0 (48.0, 61.0)	57.0 (49.0, 62.0)	57.0 (50.0, 62.0)	57.0 (50.0, 62.0)
Sex (male)	14102 (57.2)	12876 (46.9)	10826 (39.7)	9438 (33.0)
Body mass index (kg/m ²)	26.8 (24.3, 29.9)	26.3 (23.8, 29.3)	25.8 (23.5, 28.8)	25.0 (22.8, 27.8)
Ethnicity (white)	23936 (97.1)	26617 (97.0)	26261 (96.4)	27246 (95.3)
Townsend deprivation index	-2.35 (-3.75, -0.04)	-2.44 (-3.81, -0.25)	-2.44 (-3.81, -0.19)	-2.24 (-3.70, 0.24)
Smoking status				
Current	2523 (10.2)	1927 (7.02)	1555 (5.71)	1300 (4.55)
Former	8305 (33.7)	9491 (34.6)	9531 (35.0)	10030 (35.1)
Never	13769 (55.9)	15983 (58.2)	16093 (59.1)	17210 (60.2)
Missing	52 (0.21)	53 (0.19)	57 (0.21)	61 (0.21)
Alcohol intake (g/day)	13.6 (0.05, 31.4)	12.4 (0.05, 27.6)	11.3 (0.04, 25.1)	8.58 (0.01, 21.0)
Physical activity (MET-hours/week)	24.6 (11.1, 50.0)	26.5 (12.6, 51.1)	29.0 (14.3, 53.8)	32.2 (16.5, 57.8)
Total energy intake (kcal/day)	2177.4 (1861.3, 2538.2)	2040.4 (1744.0, 2369.2)	1970.7 (1692.7, 2285.4)	1905.0 (1623.6, 2216.7)
Hypertension	12700 (51.5)	13816 (50.3)	13421 (49.3)	12952 (45.3)
Hyperlipidemia	3386 (13.7)	3604 (13.1)	3451 (12.7)	3235 (11.3)
Diabetes	834 (3.38)	941 (3.43)	909 (3.34)	888 (3.10)
Planetary Health Diet Index 2.0				
Diet score	66.5 (61.3, 70.2)	78.0 (75.7, 80.2)	86.7 (84.5, 88.9)	97.3 (94.0, 102.1)
Number of participants	26,985	26,985	26,985	26,985
Age (years)	55.0 (48.0, 61.0)	57.0 (49.0, 62.0)	57.0 (50.0, 62.0)	57.0 (50.0, 62.0)
Sex (male)	15730 (58.3)	12571 (46.6)	10585 (39.2)	8356 (31.0)
Body mass index (kg/m ²)	26.9 (24.4, 30.0)	26.2 (23.8, 29.3)	25.7 (23.4, 28.6)	24.9 (22.7, 27.7)
Ethnicity (white)	26134 (96.8)	26117 (96.8)	26039 (96.5)	25770 (95.5)
Townsend deprivation index	-2.35 (-3.75, -0.02)	-2.45 (-3.81, -0.22)	-2.43 (-3.80, -0.23)	-2.23 (-3.70, 0.23)
Smoking status				
Current	2835 (10.5)	1818 (6.74)	1434 (5.31)	1218 (4.51)
Former	9110 (33.8)	9347 (34.6)	9386 (34.8)	9514 (35.3)
Never	14981 (55.5)	15769 (58.4)	16109 (59.7)	16196 (60.0)
Missing	59 (0.22)	51 (0.19)	56 (0.21)	57 (0.21)
Alcohol intake (g/day)	13.3 (0.04, 31.7)	12.1 (0.04, 27.1)	11.2 (0.04, 24.6)	9.05 (0.02, 21.6)
Physical activity (MET-hours/week)	24.9 (11.0, 50.8)	26.9 (12.8, 51.1)	29.0 (14.4, 53.6)	32.2 (16.6, 57.6)
Total energy intake (kcal/day)	2128.8 (1815.5, 2483.3)	2031.2 (1735.0, 2360.8)	1985.4 (1700.6, 2308.1)	1918.7 (1640.6, 2236.2)
Hypertension	14132 (52.4)	13541 (50.2)	13094 (48.5)	12122 (44.9)
Hyperlipidemia	3763 (13.9)	3613 (13.4)	3367 (12.5)	2933 (10.9)
Diabetes	952 (3.53)	966 (3.58)	865 (3.21)	789 (2.92)

¹Continuous variables were expressed as medians (P25, P75) and categorical variables as numbers (percentages). MET denotes metabolic equivalent.

Table 2. Association between the Planetary Health Diet 2.0 and risk of incident metabolic dysfunction-associated steatotic liver disease (n = 107,940).¹

	Categories of the diet indices				P for trend ²	Per 10% increment
	Quartile 1	Quartile 2	Quartile 3	Quartile 4		
EAT-Lancet index 2.0						
Number of participants	24,649	27,454	27,236	28,601	-	-
Number of cases	244	210	181	140	-	-
Person-years	244,893	273,743	271,926	286,465	-	-
Incidence per 1000 person-years	1.00	0.77	0.67	0.49	-	-
Model 1	1.00 (reference)	0.77 (0.64, 0.92)	0.67 (0.55, 0.81)	0.49 (0.40, 0.60)	< 0.0001	0.80 (0.75, 0.84)
Model 2	1.00 (reference)	0.78 (0.64, 0.93)	0.67 (0.55, 0.82)	0.48 (0.39, 0.59)	< 0.0001	0.79 (0.75, 0.84)
Model 3	1.00 (reference)	0.86 (0.72, 1.04)	0.81 (0.66, 0.99)	0.66 (0.53, 0.82)	< 0.0001	0.87 (0.82, 0.92)
Planetary Health Diet Index 2.0						
Number of participants	26,985	26,985	26,985	26,985	-	-
Number of cases	272	203	177	123	-	-
Person-years	267,645	268,961	269,866	270,553	-	-
Incidence per 1000 person-years	1.02	0.75	0.66	0.45	-	-
Model 1	1.00 (reference)	0.74 (0.62, 0.89)	0.64 (0.53, 0.78)	0.44 (0.36, 0.55)	< 0.0001	0.77 (0.73, 0.82)
Model 2	1.00 (reference)	0.75 (0.62, 0.90)	0.64 (0.53, 0.78)	0.43 (0.35, 0.54)	< 0.0001	0.77 (0.72, 0.82)
Model 3	1.00 (reference)	0.84 (0.70, 1.00)	0.80 (0.66, 0.97)	0.61 (0.49, 0.77)	< 0.0001	0.85 (0.80, 0.91)

¹Values are hazard ratios (95% confidence intervals) unless otherwise indicated. ²*P* for trend was calculated by using the median of the diet index categories as an ordinal variable. Model 1 was unadjusted. Model 2 was adjusted for age, sex, ethnicity, and Townsend deprivation index. Model 3 was further adjusted for smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, and body mass index.

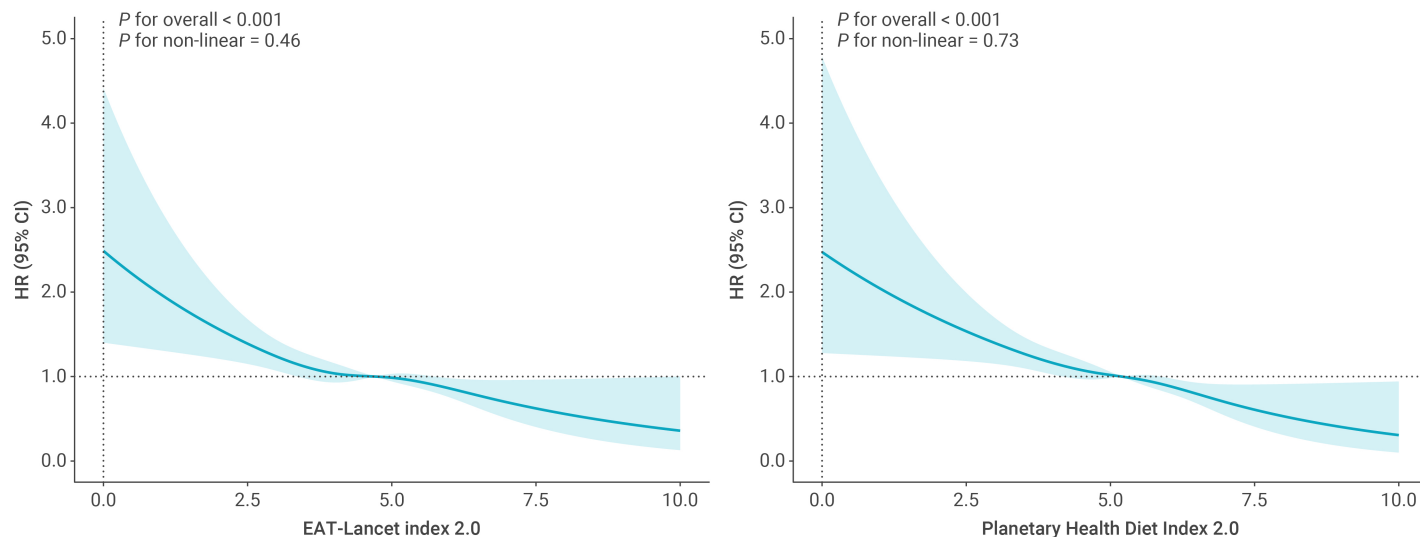


Figure 1. Dose-response association between the Planetary Health Diet 2.0 and risk of incident metabolic dysfunction-associated steatotic liver disease Cox proportional hazards model adjusted for age, sex, ethnicity, Townsend deprivation index, smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, and body mass index. Knots were placed at the 5th, 35th, 65th, and 95th percentiles of the diet indices, with the median values of the indices serving as the references. To facilitate the comparison of effect estimates across two diet index scores with different ranges, each score was scaled to a 0–10 range. Abbreviations: CI, confidence interval; HR, hazard ratio.

Sensitivity analyses

Similar inverse associations were observed when excluding participants with missing covariate data, excluding those with fewer than three 24-hour dietary assessments, or excluding incident MASLD cases that occurred within the first two years of follow-up (Table 3). In addition, the E-values of the fully adjusted HRs for comparing extreme quartiles were 2.40 for ELI 2.0 and 2.66 for PHDI 2.0, indicating that unmeasured confounders would need to be associated with both the diet indices and MASLD risk by a magnitude of at least 2.40- or 2.66-fold to explain away the observed associations. More-

over, the discrimination and reclassification were modestly improved when adding the PHD 2.0 compared with the PHD 1.0 to the base model for MASLD (Table 4).

Associations of ELI 2.0 and PHDI 2.0 with PDFF-defined hepatic steatosis and elevated cT1

The inverse associations of the PHD 2.0 with odds of PDFF-defined hepatic steatosis and elevated cT1 were observed across different models (Table 5). The fully adjusted odds ratios (95% CIs) of PDFF-defined hepatic steatosis for

Table 3. Sensitivity analyses for the association between the Planetary Health Diet 2.0 and risk of incident MASLD. ¹

	Categories of the diet indices				P for trend ²	Per 10% increment
	Quartile 1	Quartile 2	Quartile 3	Quartile 4		
Participants with missing covariate data were excluded from the analysis (n = 92,402)						
EAT-Lancet index 2.0						
Number of participants	20,963	23,368	23,320	24,751	-	-
Number of cases	209	173	141	122	-	-
Person-years	208,083	232,943	232,683	247,904	-	-
Incidence per 1000 person-years	1.00	0.74	0.61	0.49	-	-
Multivariable model	1.00 (reference)	0.83 (0.68, 1.02)	0.73 (0.59, 0.91)	0.65 (0.52, 0.83)	< 0.001	0.86 (0.81, 0.92)
Planetary Health Diet Index 2.0						
Number of participants	23,100	23,101	23,101	23,100	-	-
Number of cases	227	169	143	106	-	-
Person-years	228,989	230,064	230,971	231,588	-	-
Incidence per 1000 person-years	0.99	0.73	0.62	0.46	-	-
Multivariable model	1.00 (reference)	0.83 (0.68, 1.02)	0.77 (0.62, 0.95)	0.63 (0.50, 0.80)	< 0.001	0.85 (0.79, 0.91)
Participants with fewer than three 24-hour dietary assessments were excluded from the analysis (n = 67,097)						
EAT-Lancet index 2.0						
Number of participants	14,617	17,130	17,244	18,106	-	-
Number of cases	147	121	113	86	-	-
Person-years	146,511	172,088	173,541	182,795	-	-
Incidence per 1000 person-years	1.00	0.70	0.65	0.47	-	-
Multivariable model	1.00 (reference)	0.82 (0.64, 1.05)	0.83 (0.64, 1.07)	0.68 (0.52, 0.91)	0.01	0.89 (0.82, 0.96)
Planetary Health Diet Index 2.0						
Number of participants	16,774	16,774	16,775	16,774	-	-
Number of cases	163	133	92	79	-	-
Person-years	168,027	168,288	169,325	169,295	-	-
Incidence per 1000 person-years	0.97	0.79	0.54	0.47	-	-
Multivariable model	1.00 (reference)	0.95 (0.76, 1.20)	0.72 (0.56, 0.94)	0.71 (0.54, 0.94)	< 0.01	0.87 (0.80, 0.95)
Participants with incident MASLD within the first two years of follow-up were excluded from the analysis (n = 107,889)						
EAT-Lancet index 2.0						
Number of participants	24,629	27,441	27,224	28,595	-	-
Number of cases	224	197	169	134	-	-
Person-years	244,874	273,731	271,913	286,461	-	-
Incidence per 1000 person-years	0.91	0.72	0.62	0.47	-	-
Multivariable model	1.00 (reference)	0.88 (0.72, 1.06)	0.82 (0.67, 1.00)	0.68 (0.55, 0.86)	< 0.001	0.88 (0.83, 0.94)
Planetary Health Diet Index 2.0						
Number of participants	26,972	26,972	26,973	26,972	-	-
Number of cases	252	191	165	116	-	-
Person-years	267,694	268,942	269,849	270,492	-	-
Incidence per 1000 person-years	0.94	0.71	0.61	0.43	-	-
Multivariable model	1.00 (reference)	0.85 (0.70, 1.02)	0.80 (0.65, 0.98)	0.62 (0.49, 0.78)	< 0.0001	0.86 (0.80, 0.91)

Abbreviation: MASLD, metabolic dysfunction-associated steatotic liver disease. ¹ Values are hazard ratios (95% confidence intervals) unless otherwise indicated. ² P for trend was calculated by using the median of the diet index categories as an ordinal variable. Multivariable Cox proportional hazards models were adjusted for age, sex, ethnicity, Townsend deprivation index, smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, and body mass index.

Table 4. NRI for predicting incident MASLD risk in relation to the Planetary Health Diet indices.

Different survival models	Difference in c-statistic (95% CI)	P value	NRI (95% CI)	P value
Base model + EAT-Lancet index	0.0032 (0.0014, 0.0050)	< 0.001	0.0734 (0.0027, 0.1440)	0.04
Base model + EAT-Lancet index 2.0			0.0978 (0.0272, 0.1684)	< 0.01
Base model + Planetary Health Diet Index	0.0005 (0.0001, 0.0010)	0.02	0.1138 (0.0432, 0.1844)	< 0.01
Base model + Planetary Health Diet Index 2.0			0.1340 (0.0634, 0.2046)	< 0.001

Abbreviations: CI, confidence interval; MASLD, metabolic dysfunction-associated steatotic liver disease; NRI, net reclassification improvement. Base Cox proportional hazards models were adjusted for age, sex, ethnicity, Townsend deprivation index, smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, and body mass index.

Table 5. Associations of the Planetary Health Diet 2.0 with odds of PDFF-defined hepatic steatosis and elevated iron-corrected T1. ¹

	Categories of the diet indices				P for trend ²	Per 10% increment
	Quartile 1	Quartile 2	Quartile 3	Quartile 4		
PDFF-defined hepatic steatosis (n = 16,770)						
EAT-Lancet index 2.0						
Number of participants	3,681	4,250	4,260	4,579	-	-
Number of cases	1,259	1,169	985	789	-	-
Model 1	1.00 (reference)	0.73 (0.66, 0.80)	0.58 (0.52, 0.64)	0.40 (0.36, 0.44)	< 0.0001	0.76 (0.74, 0.79)
Model 2	1.00 (reference)	0.76 (0.69, 0.84)	0.62 (0.56, 0.69)	0.45 (0.41, 0.50)	< 0.0001	0.79 (0.77, 0.81)
Model 3	1.00 (reference)	0.83 (0.75, 0.93)	0.70 (0.63, 0.79)	0.56 (0.50, 0.63)	< 0.0001	0.84 (0.82, 0.87)
Planetary Health Diet Index 2.0						
Number of participants	4,192	4,193	4,193	4,192	-	-
Number of cases	1,434	1,150	892	726	-	-
Model 1	1.00 (reference)	0.73 (0.66, 0.80)	0.52 (0.47, 0.57)	0.40 (0.36, 0.45)	< 0.0001	0.73 (0.71, 0.76)
Model 2	1.00 (reference)	0.77 (0.70, 0.84)	0.57 (0.51, 0.63)	0.46 (0.41, 0.51)	< 0.0001	0.76 (0.74, 0.79)
Model 3	1.00 (reference)	0.84 (0.76, 0.93)	0.64 (0.58, 0.72)	0.59 (0.53, 0.66)	< 0.0001	0.83 (0.80, 0.86)
Elevated iron-corrected T1 (n = 14,015)						
EAT-Lancet index 2.0						
Number of participants	3,057	3,547	3,531	3,880	-	-
Number of cases	227	181	149	124	-	-
Model 1	1.00 (reference)	0.67 (0.55, 0.82)	0.55 (0.44, 0.68)	0.41 (0.33, 0.51)	< 0.0001	0.77 (0.73, 0.82)
Model 2	1.00 (reference)	0.71 (0.58, 0.87)	0.60 (0.48, 0.74)	0.46 (0.37, 0.58)	< 0.0001	0.80 (0.75, 0.85)
Model 3	1.00 (reference)	0.82 (0.66, 1.02)	0.71 (0.56, 0.89)	0.63 (0.49, 0.80)	< 0.0001	0.88 (0.82, 0.94)
Planetary Health Diet Index 2.0						
Number of participants	3,450	3,501	3,543	3,521	-	-
Number of cases	258	181	134	108	-	-
Model 1	1.00 (reference)	0.67 (0.55, 0.82)	0.49 (0.39, 0.60)	0.39 (0.31, 0.49)	< 0.0001	0.73 (0.68, 0.78)
Model 2	1.00 (reference)	0.71 (0.58, 0.87)	0.53 (0.43, 0.66)	0.44 (0.35, 0.56)	< 0.0001	0.76 (0.71, 0.81)
Model 3	1.00 (reference)	0.82 (0.67, 1.01)	0.65 (0.51, 0.81)	0.61 (0.48, 0.78)	< 0.0001	0.84 (0.78, 0.90)

Abbreviation: PDFF, proton density fat fraction. ¹ Values are odds ratios (95% confidence intervals) unless otherwise indicated. ² P for trend was calculated by using the median of the diet index categories as an ordinal variable. Model 1 was unadjusted. Model 2 was adjusted for age, sex, ethnicity, and Townsend deprivation index. Model 3 was further adjusted for smoking status, alcohol intake, physical activity, total energy intake, hypertension, hyperlipidemia, diabetes, body mass index, and the interval between dietary assessment and magnetic resonance imaging scan.

comparing the extreme quartiles were 0.56 (0.50, 0.63) for ELI 2.0 and 0.59 (0.53, 0.66) for PHDI 2.0, respectively (*P* for trend < 0.0001). The corresponding odds ratios (95% CIs) per 10% increase in diet scores were 0.84 (0.82, 0.87) for ELI 2.0 and 0.83 (0.80, 0.86) for PHDI 2.0. Likewise, fully adjusted odds ratios (95% CIs) per 10% increase in diet scores for elevated cT1 were 0.88 (0.82, 0.94) for ELI 2.0 and 0.84 (0.78, 0.90) for PHDI 2.0.

DISCUSSION

Principal findings

In this large prospective population-based cohort, we found that higher adherence to the PHD 2.0, quantified by the newly developed ELI 2.0 and PHDI 2.0, was significantly associated with a lower risk of incident MASLD. Such associations remained consistent across a series of sensitivity analyses. Moreover, these associations were supported by favorable liver magnetic resonance imaging markers. In addition, the two new indices showed modest improvements in predicting MASLD risk compared with their original versions.

Comparison with previous studies

To our knowledge, this is the first study to evaluate the association of the PHD 2.0 with MASLD. Previously, several epidemiological studies have investigated the association of the PHD with MASLD. For instance, our previous multi-cohort studies among Chinese adults and UK adults have shown the beneficial associations of the PHD with MASLD and its severity.⁷⁻⁸ In addition, previous research in the UK Biobank,³⁴⁻³⁵ the US National Health and Nutrition Examination Survey and the Global Dietary Database,³⁶⁻³⁷ as well as in Iran,³⁸⁻³⁹ all indicated an inverse association between the PHD and MASLD. Notably, these previous studies assessed adherence to the PHD according to the 2019 EAT-Lancet Commission report. By leveraging the ELI 2.0 and the PHDI 2.0, our current study advances previous findings by providing incremental evidence from a large prospective cohort, highlighting the potential role of the PHD 2.0 in MASLD prevention. Moreover, our evaluation of the associations of ELI 2.0 and PHDI 2.0 with odds of PDFF-defined hepatic steatosis and elevated cT1 provides novel evidence regarding the relationship between PHD 2.0 and liver magnetic resonance imaging markers, an area that remains insufficiently explored. Overall, our findings corroborate and extend previous observational evidence linking higher PHD adherence with a lower risk of liver disease, while imaging-based cross-sectional analyses provide complementary phenotypic evidence that was directionally consistent with the prospective findings.

The necessity of transitioning from the first-generation sustainable dietary metrics to the updated version 2.0 frameworks is primarily driven by their alignment with the revised dietary standards proposed by the 2025 EAT-Lancet Commission.^{10,13} Compared to their original predecessors, these updated indices incorporate revised intake targets and reference intervals for various food groups, while also refining the dietary assessment framework through structural modifications, including the consolidation of red meat categories and the introduction of a dedicated sodium intake dimension.¹³ The incorporation of sodium as an evaluation component is both biologically plausible and clinically relevant, given that excessive sodium intake represents a major contributor to the global burden of chronic diseases.⁴⁰ According to the Global Burden of Disease Study, high sodium intake accounts for approximately three million deaths worldwide annually, making it one of the leading modifiable dietary risk factors for mortality.⁴¹ Indeed, the models with ELI 2.0 or PHDI 2.0 showed modestly improved discrimination for MASLD risk compared with models incorporating their original versions reflecting the PHD 1.0. Therefore, incorporating the sodium dimension into ELI 2.0 and PHDI 2.0 enhances their capacity to capture the multidimensional characteristics of sustainable nutrition, providing a more comprehensive and refined approach for evaluating metabolic liver health at both population and individual levels.

Our findings highlight the unique advantages of the PHD 2.0 over conventional healthy dietary patterns in the prevention of MASLD. The consistent inverse associations between these diet indices and MASLD support the robustness of our findings, indicating that the observed hepatoprotective effects remain stable despite differences in index construction. On the other hand, the PHD 2.0 shares some overlap with several well-established healthy dietary patterns, such as the Mediterranean diet and the Dietary Approaches to Stop Hypertension diet. Nevertheless, important distinctions exist between the PHD 2.0 and these traditional dietary approaches. For example, a central feature of the PHD 2.0 is the importance of nuts and legumes (including beans, pulses, and soy) as major protein sources, with small amounts of red meat and dairy foods. In contrast, the Mediterranean diet places particular emphasis on olive oil as the primary culinary fat source and incorporates

moderate wine consumption, whereas the Dietary Approaches to Stop Hypertension diet was originally designed with a strong focus on sodium reduction for cardiovascular health.⁴² Another key advantage of the PHD 2.0 framework is its integrated consideration of human health, planetary sustainability, and just food systems. Unlike conventional dietary patterns, which are primarily developed to optimize individual health outcomes, the PHD 2.0 explicitly incorporates environmental considerations, addressing the interconnected challenges of chronic disease prevention and global climate change. Therefore, widespread adoption of this dietary pattern may provide complementary benefits for both population health and environmental sustainability.¹⁰ Nevertheless, the PHD 2.0 does not account for alcohol intake and may therefore not fully capture overall dietary behaviors.

The underlying mechanisms for the inverse association between the PHD 2.0 and MASLD may be explained by several aspects. First, adherence to the PHD 2.0 emphasizes a higher intake of plant-based foods, including whole grains, fruits, vegetables, legumes, and nuts, which are rich in dietary fiber, polyphenols, and micronutrients that may improve insulin sensitivity, reduce oxidative stress, and alleviate hepatic lipid accumulation.⁴³⁻⁴⁴ Second, the PHD 2.0 recommends limiting meats, refined grains, and sugars, thereby reducing excessive saturated fatty acid intake, *de novo* lipogenesis, chronic low-grade inflammation, and promoting iron homeostasis balance, which are key contributors to MASLD development.⁴⁵⁻⁴⁶ Third, sodium is the key new component of the PHD 2.0. Experimental evidence suggests that increased hepatic osmolality induced by high sodium intake may activate the aldose reductase-fructokinase pathway, thereby increasing endogenous fructose production and promoting hepatic triglyceride accumulation and fatty liver.⁴⁷ Finally, PHD 2.0 may beneficially modulate gut microbiota composition and function, promoting the production of short-chain fatty acids and maintaining intestinal barrier integrity, which could attenuate metabolic endotoxemia and hepatic inflammation.⁴⁸⁻⁴⁹

Interestingly, the inverse association between the PHD 2.0 and MASLD was stronger among white participants, individuals without hypertension, and those with overweight/obesity. The null association in non-white participants and individuals with BMI < 25 kg/m² may be partly explained by limited statistical power due to the relatively small number of MASLD events in these subgroups. In addition, the findings among non-white participants may raise the possibility that the applicability of the PHD 2.0 varies across populations with different cultural and dietary contexts. The stronger association among individuals with overweight/obesity may reflect their heightened metabolic vulnerability and greater responsiveness to dietary modification targeting insulin resistance, inflammation, and lipid metabolism. However, the association was generally consistent across hypertension strata, though with a significant interaction.

Strengths and limitations

The strengths of our study include its large sample size, prospective design, long-term follow-up, use of liver magnetic resonance imaging data, and careful adjustment for potential confounders. More importantly, this study is the first to evaluate the associations of the newly released PHD 2.0 with clinical and imaging indicators of liver health using the ELI 2.0 and PHDI 2.0.

However, several limitations should be acknowledged. First, dietary data were collected using 24-hour dietary assessments, which may not fully capture long-term dietary habits.⁵⁰ In particular, sodium intake estimated from such assessment is subject to measurement error. However, the validity of the dietary assessment tool has been demonstrated in the UK Biobank,¹⁹ and sensitivity analyses based on at least three measures produced similar results. Second, incident MASLD was identified using diagnostic codes, which may have led to an underestimation of incident cases. Such ascertainment bias would attenuate the observed associations towards the null because only more advanced stages of MASLD were identified. Nevertheless, the observed favorable associations were supported by liver magnetic resonance imaging data. Third, although we adjusted for a comprehensive range of covariates, residual confounding cannot be entirely ruled out. However, E-value analyses suggested that unmeasured confounders were less likely to negate the observed associations. Fourth, as with any observational study, causality cannot be inferred. Finally, the study

population was restricted to the UK Biobank participants, which may limit the generalizability of our findings to other populations with different genetic or ethnic backgrounds. Future studies in more diverse populations are warranted to confirm our results.

CONCLUSIONS

In conclusion, higher adherence to the PHD 2.0 was significantly associated with a lower risk of incident MASLD as well as favorable magnetic resonance imaging profiles among the general adult population. These findings provide incremental evidence linking the 2025 EAT-Lancet framework to liver health. Our results lend support to the importance of the newly released PHD 2.0 metrics in informing dietary approaches for liver disease prevention.

REFERENCES

- Tilg H, Petta S, Stefan N, et al. (2026). Metabolic dysfunction-associated steatotic liver disease in adults: A review. *JAMA* **335**:163–174. DOI:10.1001/jama.2025.19615
- Targher G, Byrne C. D. and Tilg H. (2024). MASLD: A systemic metabolic disorder with cardiovascular and malignant complications. *Gut* **73**:691–702. DOI:10.1136/gutjnl-2023-330595
- Li W, Alazawi W. and Loomba R. (2026). Current and emerging therapeutic landscape for metabolic dysfunction-associated steatohepatitis. *Lancet Gastroenterol. Hepatol.* **11**:150–162. DOI:10.1016/S2468-1253(25)00260-2
- Zeng X. F., Varady K. A., Wang X. D., et al. (2024). The role of dietary modification in the prevention and management of metabolic dysfunction-associated fatty liver disease: An international multidisciplinary expert consensus. *Metabolism* **161**:156028. DOI:10.1016/j.metabol.2024.156028
- Tilman D. and Clark M. (2014). Global diets link environmental sustainability and human health. *Nature* **515**:518–522. DOI:10.1038/nature13959
- Willett W., Rockstrom J., Loken B., et al. (2019). Food in the Anthropocene: The EAT-Lancet commission on healthy diets from sustainable food systems. *Lancet* **393**:447–492. DOI:10.1016/S0140-6736(18)31788-4
- Zhang S., Yan Y., Zeng X. F., et al. (2025). Associations of the EAT-Lancet reference diet with metabolic dysfunction-associated steatotic liver disease and its severity: A multicohort study. *Hepatology* **81**:1583–1594. DOI:10.1097/HEP.0000000000001039
- Zhang S., Zeng X. F., Borne Y., et al. (2025). Adherence to the EAT-Lancet reference diet and risk of type 2 diabetes, metabolic dysfunction-associated steatotic liver disease, and their co-occurrence. *Food Funct.* **16**:6773–6785. DOI:10.1039/d4fo05852f
- Stubbendorff A., Janzi S., Jukkola J., et al. (2025). Mini-review of the EAT-Lancet planetary health diet and its role in cardiometabolic disease prevention. *Metabolism* **172**:156373. DOI:10.1016/j.metabol.2025.156373
- Rockstrom J., Thilsted S. H., Willett W. C., et al. (2025). The EAT-Lancet Commission on healthy, sustainable, and just food systems. *Lancet* **406**:1625–1700. DOI:10.1016/S0140-6736(25)01201-2
- Shojaei-Zarghani S., Safarpour A. R., Fattahi M. R., et al. (2022). Sodium in relation with nonalcoholic fatty liver disease: A systematic review and meta-analysis of observational studies. *Food Sci. Nutr.* **10**:1579–1591. DOI:10.1002/fsn3.2781
- Zhang S., Huo Z., Borne Y., et al. (2025). Adding salt to foods and risk of metabolic dysfunction-associated steatotic liver disease and other chronic liver diseases. *Eur. J. Nutr.* **64**:224. DOI:10.1007/s00394-025-03745-3
- Stubbendorff A., Ericson U., Hallstrom E., et al. (2026). EAT-Lancet index 2.0: A dietary index measuring adherence to the Planetary Health Diet 2.0. *Lancet Planet Health* **10**:101469. DOI:10.1016/j.lanph.2026.101469
- Bui L. P., Pham T. T., Wang F., et al. (2024). Planetary Health Diet Index and risk of total and cause-specific mortality in three prospective cohorts. *Am. J. Clin. Nutr.* **120**:80–91. DOI:10.1016/j.ajcnut.2024.03.019
- Ollier W., Sprosen T. and Peakman T. (2005). UK Biobank: From concept to reality. *Pharmacogenomics* **6**:639–646. DOI:10.2217/14622416.6.6.639
- Xu X., Dong Y., Hu J., et al. (2026). Planetary health diet index and risk of irritable bowel syndrome: A prospective cohort study. *Eur. J. Nutr.* **65**:199. DOI:10.1007/s00394-026-04055-y
- Vandenbroucke J. P., von Elm E., Altman D. G., et al. (2014). Strengthening the Reporting of Observational Studies in Epidemiology (STROBE): Explanation and elaboration. *Int J Surg* **12**:1500–1524. DOI:10.1016/j.ijsu.2014.07.014
- Lachat C., Hawwash D., Ocke M. C., et al. (2016). Strengthening the Reporting of Observational Studies in Epidemiology-Nutritional Epidemiology (STROBE-nut): An Extension of the STROBE Statement. *PLoS Med.* **13**e1002036. DOI:10.1371/journal.pmed.1002036
- Greenwood D. C., Hardie L. J., Frost G. S., et al. (2019). Validation of the Oxford WebQ online 24-hour dietary questionnaire using biomarkers. *Am. J. Epidemiol.* **188**:1858–1867. DOI:10.1093/aje/kwz165
- Perez-Cornago A., Pollard Z., Young H., et al. (2021). Description of the updated nutrition calculation of the Oxford WebQ questionnaire and comparison with the previous version among 207,144 participants in UK Biobank. *Eur. J. Nutr.* **60**:4019–4030. DOI:10.1007/s00394-021-02558-4
- Piernas C., Perez-Cornago A., Gao M., et al. (2021). Describing a new food group classification system for UK Biobank: Analysis of food groups and sources of macro- and micronutrients in 208,200 participants. *Eur. J. Nutr.* **60**:2879–2890. DOI:10.1007/s00394-021-02535-x
- Hagstrom H., Adams L. A., Allen A. M., et al. (2021). Administrative coding in electronic health care record-based research of NAFLD: An expert panel consensus statement. *Hepatology* **74**:474–482. DOI:10.1002/hep.31726
- Wilman H. R., Kelly M., Garratt S., et al. (2017). Characterisation of liver fat in the UK Biobank cohort. *PLoS One* **12**e0172921. DOI:10.1371/journal.pone.0172921
- Jackson E., Dennis A., Alkhouri N., et al. (2025). Cardiac and liver impairment on multiorgan MRI and risk of major adverse cardiovascular and liver events. *Nat. Med.* **31**:2289–2296. DOI:10.1038/s41591-025-03654-2
- Caussy C., Alkharish M. H., Nguyen P., et al. (2018). Optimal threshold of controlled attenuation parameter with MRI-PDFF as the gold standard for the detection of hepatic steatosis. *Hepatology* **67**:1348–1359. DOI:10.1002/hep.29639
- Craig C. L., Marshall A. L., Sjostrom M., et al. (2003). International physical activity questionnaire: 12-country reliability and validity. *MED SCI SPORT EXER* **35**:1381–1395. DOI:10.1249/01.MSS.0000078924.61453.FB
- Schisterman E. F., Cole S. R. and Platt R. W. (2009). Overadjustment bias and unnecessary adjustment in epidemiologic studies. *Epidemiology* **20**:488–495. DOI:10.1097/EDE.0b013e3181a819a1
- Desquilbet L. and Mariotti F. (2010). Dose-response analyses using restricted cubic spline functions in public health research. *Stat. Med.* **29**:1037–1057. DOI:10.1002/sim.3841
- Mathur M. B., Ding P., Riddell C. A., et al. (2018). Website and R package for computing E-values. *Epidemiology* **29**:e45–e47. DOI:10.1097/EDE.0000000000000864
- VanderWeele T. J. and Ding P. (2017). Sensitivity analysis in observational research: Introducing the E-value. *Ann. Intern. Med.* **167**:268–274. DOI:10.7326/M16-2607
- Uno H., Cai T., Pencina M. J., et al. (2011). On the C-statistics for evaluating overall adequacy of risk prediction procedures with censored survival data. *Stat. Med.* **30**:1105–1117. DOI:10.1002/sim.4154
- Harrell F. E., Jr., Lee K. L. and Mark D. B. (1996). Multivariable prognostic models: Issues in developing models, evaluating assumptions and adequacy, and measuring and reducing errors. *Stat. Med.* **15**:361–387. DOI:10.1002/(SICI)1097-0258(19960229)15:4<361::AID-SIM168>3.0.CO;2-4
- Pencina M. J., D'Agostino R. B., Sr. and Steyerberg E. W. (2011). Extensions of net reclassification improvement calculations to measure usefulness of new biomarkers. *Stat. Med.* **30**:11–21. DOI:10.1002/sim.4085
- Wu S. Y., Bo Y. C., Li Z. Y., et al. (2025). EAT-Lancet diet and risk of metabolic dysfunction-associated steatotic liver disease and other liver chronic diseases: A large prospective cohort study in the UK Biobank. *Front. Nutr.* **12**:1589424. DOI:10.3389/fnut.2025.1589424
- Wu H., Wei J., Wang S., et al. (2024). Dietary pattern modifies the risk of MASLD through metabolomic signature. *JHEP Rep* **6**:101133. DOI:10.1016/j.jhepr.2024.101133
- Qiu X., Shen S., Jiang N., et al. (2025). Adherence to the planetary health diet index and metabolic dysfunction-associated steatotic liver disease: A cross-sectional study. *Front. Nutr.* **12**:1534604. DOI:10.3389/fnut.2025.1534604
- Liu Z. Q., Zhou B. G., Fang J. W., et al. (2025). Protective role of the planetary health diet index against metabolic dysfunction-associated steatotic liver disease: Global and individual evidence. *Front. Nutr.* **12**:1673662. DOI:10.3389/fnut.2025.1673662
- Doustmohammadian A., Esfandiyari S., Motamed N., et al. (2026). EAT-Lancet dietary pattern adherence and metabolic dysfunction-associated steatotic liver disease: A cross-sectional analysis of the Amol Cohort Study. *J Nutr* **156**:101433. DOI:10.1016/j.tjnut.2026.101433
- Kamrani F., Imannezhad M., Etesami E., et al. (2026). Higher adherence to the EAT-Lancet diet is associated with reduced risk of metabolic dysfunction-associated steatotic liver disease: a 5-year middle eastern cohort study. *Diabetol Metab Syndr* **18**:176. DOI:10.1186/s13098-026-02198-w
- Mozaffarian D., Fahimi S., Singh G. M., et al. (2014). Global sodium consumption and death from cardiovascular causes. *NEJM* **371**:624–634. DOI:10.1056/NEJMoa1304127
- Collaborators G. B. D. D. (2019). Health effects of dietary risks in 195 countries, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *Lancet* **393**:1958–1972. DOI:10.1016/S0140-6736(19)30041-8
- Hu F. B. (2024). Diet strategies for promoting healthy aging and longevity: An epidemiological perspective. *J. Intern. Med.* **295**:508–531. DOI:10.1111/joim.13728
- Jurek J. M., Zablocka-Slowinska K., Pieczynska J., et al. (2025). Systematic review of the effects of plant-based foods on metabolic outcomes in adults with MASLD and comorbidities such as obesity, metabolic syndrome, and type 2 diabetes. *Nutrients* **17**:3020. DOI:10.3390/nu17183020
- Ly Y., Rong S., Deng Y., et al. (2023). Plant-based diets, genetic predisposition and risk of non-alcoholic fatty liver disease. *BMC MED* **21**:351. DOI:10.1186/s12916-023-03028-w
- Carli F., Della Pepa G., Sabatini S., et al. (2024). Lipid metabolism in MASLD and MASH: From mechanism to the clinic. *JHEP Rep* **6**:101185. DOI:10.1016/j.jhepr.2024.101185

46. Zhou Y., Chen D., Zhu W., et al. (2025). Causal relationships between iron status and nonalcoholic fatty liver disease: Two-sample, multivariable, and two-step Mendelian randomization. *Int. J. Vitam. Nutr. Res.* **95**:26773. DOI:10.31083/IJVN26773
47. Lanaspa M. A., Kuwabara M., Andres-Hernando A., et al. (2018). High salt intake causes leptin resistance and obesity in mice by stimulating endogenous fructose production and metabolism. *PNAS*. **115**:3138–3143. DOI:10.1073/pnas.1713837115
48. Che Z., Xue W., Zhao X., et al. (2025). Regulatory role and biomarker potential of gut microbiota metabolites in the progression of metabolic dysfunction-associated steatotic liver disease to hepatocellular carcinoma. *CTG* **16**e00914. DOI:10.14309/ctg.0000000000000914
49. Bahitham W., Banoun Y., Aljahdali M., et al. (2025). "Trust your gut": Exploring the connection between gut microbiome dysbiosis and the advancement of metabolic associated steatosis liver disease (MASLD)/metabolic associated steatohepatitis (MASH): A systematic review of animal and human studies. *Front. Nutr.* **12**:1637071. DOI:10.3389/fnut.2025.1637071
50. Zhang J., Geng T., Wang Y., et al. (2026). Challenges, advances and future directions in nutritional epidemiology. *Innov.Nutri.* **1**:100009. DOI:10.59717/j.xinn-nutri.2026.100009

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AUTHOR CONTRIBUTIONS

T.H. and S.Z. conceived and designed the research. S.Z. analyzed the data. S.Z. and X.X. wrote the manuscript. X.X., A.S., Y.B., E.S., T.H., and S.Z. contributed to the interpretation of results and revision of the manuscript. All authors contributed to and approved the manuscript.

DECLARATION OF INTERESTS

The authors declare no competing interests.

ETHICAL STATEMENT AND PATIENT CONSENT

Ethical approval for the study protocol was granted by the North West Multicentre Research Ethics Committee (reference number: 11/NW/0382), and all participants provided written informed consent.

DATA AND CODE AVAILABILITY

The UK Biobank data can be obtained upon application from <https://www.ukbiobank.ac.uk/use-our-data/apply-for-access/>. Analytic codes are available from the corresponding author upon reasonable request.

SUPPLEMENTAL INFORMATION

It can be found online at <https://doi.org/10.59717/j.xinn-nutri.2026.100041>