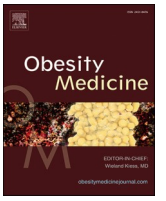
Obesity Medicine 62 (2026) 100728



Contents lists available at ScienceDirect

Obesity Medicine

journal homepage: www.elsevier.com/locate/obmed



Waist circumference associates with ultrasound-defined steatosis grade in NAFLD patients characterized by uniformly suboptimal diet

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ARTICLE INFO

Keywords:
Non alcoholic fatty liver
Waist circumference
Dietary inflammatory index
Plant based diet
Diet quality

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) clinical phenotypes involve complex interactions between adiposity and diet. A particularly underexplored question is whether dietary quality differs across steatosis severity grades in clinically complex, high-risk cohorts with prevalent T2DM. We hypothesized that specific dietary patterns would be associated with disease severity, independent of anthropometric indices.

Methods: In this cross-sectional study, 160 adults with confirmed NAFLD were evaluated using structured questionnaires, anthropometric measurements, and a 108-item food frequency questionnaire. Steatosis severity (Grades 1–3) was determined by ultrasonography. Healthy Eating Index-2015 (HEI-2015), Dietary Inflammatory Index (DII), Potential Renal Acid Load (PRAL), and Plant-Based Diet Index (PDI) were computed. Hierarchical binary logistic regression was performed using complete-case analysis ($n = 122$), adjusting for sex, type 2 diabetes, and total energy intake.

Results: Body weight, BMI, and waist circumference increased significantly across steatosis grades ($p < 0.001$). No significant differences were observed in HEI-2015, DII, PRAL, or PDI across groups ($p > 0.05$), and none showed independent associations with steatosis grade in adjusted models. Dietary quality was uniformly suboptimal across all severity grades. Waist circumference was consistently associated with higher ultrasound-assigned steatosis grades across all models in this selected cohort (OR: 1.10, 95% CI: 1.05–1.16, $p < 0.001$), accounting for 27% of the variance explained by the base logistic model.

Conclusion: A key finding is that dietary quality was uniformly suboptimal across all steatosis severity grades, including the mildest stage, supporting consideration of nutritional assessment and intervention from early disease stages regardless of disease severity. In this highly selected clinical cohort with a high burden of metabolic comorbidities, waist circumference demonstrated a consistent independent association with ultrasound-defined steatosis grade. These findings should not be generalized beyond this specific metabolic phenotype.

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https://doi.org/10.1016/j.obmed.2026.100728
Received 11 May 2026; Received in revised form 29 June 2026; Accepted 1 July 2026
Available online 3 July 2026
2451-8476/© 2026 Published by Elsevier Ltd.

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most prevalent chronic liver disorder worldwide, affecting approximately 38% of the global population (Younossi et al., 2023). The pathogenesis of NAFLD is driven by a multiple-parallel hits phenomenon, where insulin resistance, lipotoxicity, and chronic low-grade inflammation synergistically promote hepatic lipid accumulation and subsequent fibrotic progression (Zhu et al., 2021). Central to this pathogenic cascade is abdominal obesity, which leads to adipose tissue dysfunction and an augmented flux of free fatty acids (FFAs) via the portal vein into the liver (Xiang et al., 2024). This hepatic lipid overload induces mitochondrial dysfunction and endoplasmic reticulum stress, while the concurrent release of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), triggers systemic low-grade inflammation that activates Kupffer and hepatic stellate cells, driving the progression from simple steatosis to steatohepatitis and fibrotic phenotypes (Sheikh et al., 2025).

Dietary habits play a pivotal role as environmental modulators in this process. A Western dietary pattern, characterized by high intakes of fructose, saturated fats, and ultra-processed foods, directly stimulates hepatic *de novo* lipogenesis and exacerbates central adiposity (Hassani Zadeh et al., 2021). Furthermore, this dietary pattern promotes gut microbiota dysbiosis and increased intestinal permeability, facilitating the translocation of endotoxins (e.g., lipopolysaccharides) into the liver and compounding hepatic inflammation (Hamamah et al., 2024). Conversely, adherence to fiber-rich, antioxidant-dense dietary patterns, such as the Mediterranean diet, is generally considered protective. These diets, rich in mono- and polyunsaturated fatty acids and polyphenols, attenuate oxidative stress, improve insulin sensitivity, and restore the microbiome-gut-liver axis, effectively mitigating steatotic changes (Bakhshimoghaddam et al., 2024; Del Bo et al., 2023). An overview of these multifactorial dietary influences and their pathophysiological mechanisms in NAFLD is illustrated in Fig. 1.

To quantitatively evaluate these dietary influences, various scoring systems have been established as clinical determinants of NAFLD severity. The Healthy Eating Index (HEI-2015) reflects overall diet quality, showing an inverse association with hepatic fat accumulation and NAFLD phenotypes (Wang et al., 2025). The Plant-based Diet Index (PDI) further differentiates dietary impacts; while healthful plant-based diets foster microbial diversity and hepatic protection, unhealthful plant-based diets rich in refined carbohydrates stimulate lipogenesis and increase disease risk (Miryan et al., 2025; Peña-Jorquera et al., 2023). Additionally, the Dietary Inflammatory Index (DII) quantifies the pro-inflammatory load of the diet, which is positively correlated with disease severity, mitochondrial dysfunction, and fibrogenesis (Petermann-Rocha et al., 2023). Similarly, the Potential Renal Acid Load (PRAL), driven predominantly by animal proteins and refined grains, induces systemic low-grade metabolic acidosis, disrupts insulin signaling pathways, and exacerbates cellular metabolic dysfunction (Homayounfar et al., 2026).

Despite the well-documented influence of nutrition, a critical knowledge gap remains regarding the relative contribution of diet quality versus central obesity in relation to disease severity. While numerous epidemiological studies have examined dietary trends and indices, it is still not fully clear how overall dietary patterns or their inflammatory potential perform hierarchically when competing with potent metabolic drivers like central obesity. It remains unclear whether dietary risk factors are independently associated with different stages of steatosis, or whether central adiposity indices show more consistent independent associations. Therefore, the primary objective of this study was to evaluate the independent associations of diverse dietary indices (HEI-2015, DII, PRAL, PDI) with NAFLD severity grades using a hierarchical approach, particularly within a clinical sample characterized by a high burden of metabolic comorbidities. Based on current literature, we tested the hypothesis that higher diet quality, lower dietary inflammatory potential, and higher adherence to plant-based diets would be associated with lower steatosis severity, independent of body mass index (BMI) and waist circumference, thereby clarifying the timing and necessity of clinical nutritional interventions in patients with established metabolic risks.

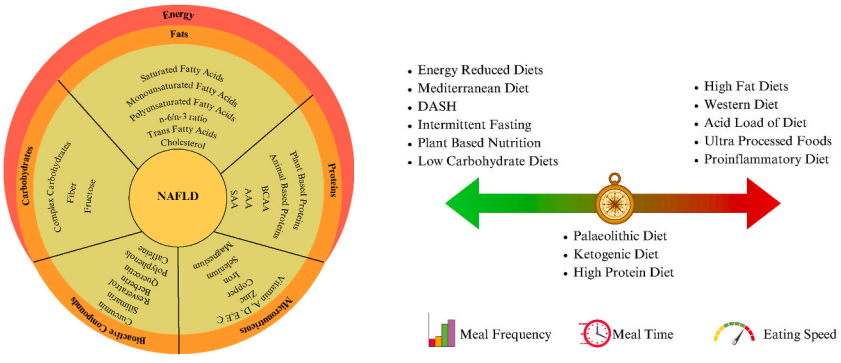


Fig. 1. Conceptual framework illustrating the multifactorial relationship between dietary components and NAFLD pathogenesis. While pro-inflammatory factors and excessive caloric intake promote hepatic steatosis, antioxidants and healthy dietary patterns offer protective mechanisms.

2. Methods

2.1. Study design and participants

This cross-sectional study was conducted between April 2022 and April 2024 at the Akdeniz University Hospital Endocrinology Clinic. During this period, a total of 203 patients were initially screened. Following the application of strict inclusion and exclusion criteria, 160 adult patients with a confirmed diagnosis of NAFLD were ultimately included in the final analysis (Fig. 2). Regarding the terminology, we acknowledge the recent global consensus proposing the transition from 'Non-Alcoholic Fatty Liver Disease (NAFLD)' to 'Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)' (Rinella and Sookoian, 2024). However, since this study was designed and conducted during a period when NAFLD was the universally accepted nomenclature, we have maintained the use of 'NAFLD' throughout this manuscript to ensure consistency with the diagnostic criteria applied at the time. Nevertheless, our findings on metabolic and anthropometric risk factors remain fully applicable to the MASLD framework.

The protocol received formal approval from Akdeniz University Faculty of Medicine Clinical Research Ethics Committee (KAEK-168). Prior to enrollment, informed consent was obtained from every participant.

Sample size estimation was performed via G*Power software. Referencing prior study (Gürsoy, 2023), an effect size of d = 0.3 was applied. To achieve 80% statistical power with an alpha error of 0.05 using an F-test across three groups, the calculation mandated a minimum of 111 subjects.

Ultrasonographic imaging was performed by a single, experienced endocrinologist who was fully blinded to the patients' dietary intakes, meaning there was no inter-observer variability, though intra-observer reproducibility was not formally assessed. All examinations were conducted using a high-resolution clinical ultrasound system (Mindray Resona I9, Shenzhen, China) equipped with a 1.2–6.0 MHz SC6-1s convex transducer. Participants were stratified into three distinct cohorts based on standard semi-quantitative

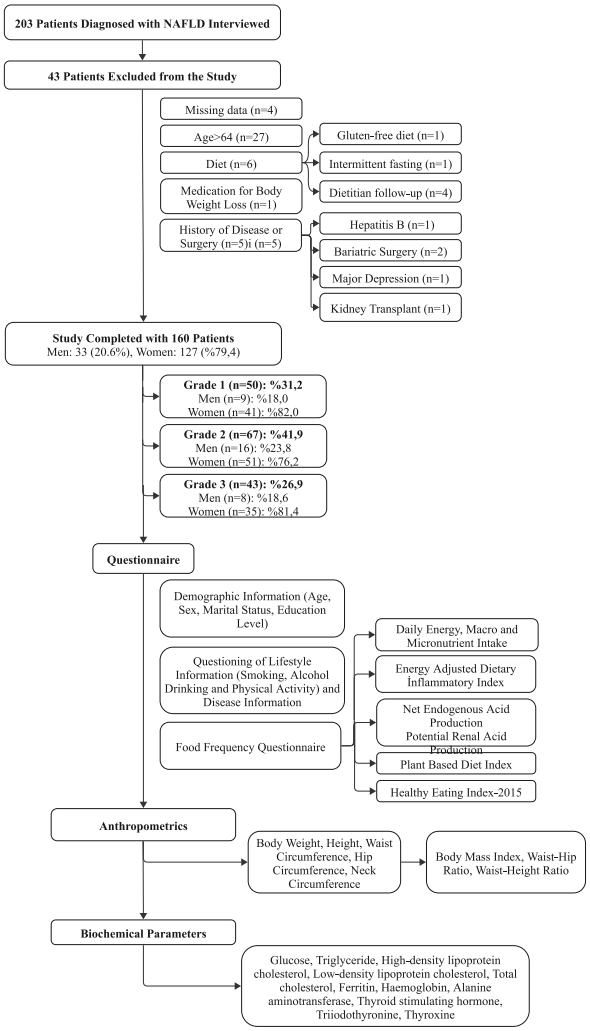


Fig. 2. Flowchart of the study design, illustrating the participant selection process, exclusion criteria, and final grouping based on steatosis severity.

criteria: Grade 1 (mild; slight diffuse increase in hepatic echogenicity with normal visualization of intrahepatic vessel walls and diaphragm), Grade 2 (moderate; moderate diffuse increase in hepatic echogenicity with partial blurring of intrahepatic vessels and diaphragm), and Grade 3 (severe; marked increase in echogenicity with poor or no visualization of intrahepatic vessels, diaphragm, and posterior right lobe of the liver).

2.2. Data collection

Data collection was carried out entirely in person during a single clinical visit at the hospital, ensuring all measurements and tools were administered simultaneously. A structured, face-to-face questionnaire was conducted by trained researchers to gather comprehensive data on socioeconomic status (age, sex, marital status, education, and occupation), lifestyle habits (smoking status and alcohol consumption), and self-reported medical history. Information regarding co-morbidities, including Type 2 Diabetes Mellitus (T2DM), hypertension, goiter, and Hashimoto's thyroiditis, was initially collected via this questionnaire and subsequently cross-verified with patients' official electronic medical records and active prescription data to ensure accuracy.

Following the interview, all participants underwent anthropometric measurements in person on the same day, adhering to standardized clinical protocols. Dietary assessment tools (the 24-h dietary recall and the 108-item FFQ) and physical activity levels via the Short Form of the International Physical Activity Questionnaire (IPAQ-SF) (Craig et al., 2003) were also completed in person via one-on-one interviews during the same visit to eliminate self-reporting discrepancies and maximize data completeness.

From this data, we calculated various dietary metrics, including the Energy-Adjusted Dietary Inflammatory Index (E-DII) (Shivappa et al., 2014), dietary acid load (Frassetto et al., 1998; Remer and Manz, 1994, 1995), Plant-Based Diet Indices (PDI, HPDI, UPDI) (Satija et al., 2016), and the Healthy Eating Index-2015 (HEI-2015) (Krebs-Smith et al., 2018) to determine diet quality. Detailed calculations of diet-related parameters are provided in the supplementary materials. Anthropometric data, comprising weight, height, and circumferences of the waist, hip, and neck, were recorded adhering to standard protocols (Ben-Noun et al., 2001; Lee and Nieman, 2010). Specifically, waist and hip measurements were available for 122 patients, while neck circumference was measured in 95 individuals. Body Mass Index was computed and categorized based on the World Health Organization (WHO) guideline: normal weight (18.50–24.99 kg/m^2), overweight (25.00–29.99 kg/m^2), obese ($\geq$30.00 kg/m^2), and severely obese ($\geq$40.00 kg/m^2). Additionally, waist-to-hip and waist-to-height ratios were calculated. Metabolic risk thresholds for waist circumference were defined as >102 cm for men and >88 cm for women (WHO, 2011), while a waist-to-height ratio of $\geq$0.5 was considered indicative of elevated metabolic risk (Ashwell and Hsieh, 2005).

2.3. Statistical analyses

Data analysis employed a dual-platform approach utilizing SPSS Statistics (Version 23.0, IBM Corp, Armonk, NY) for descriptive statistics and group comparisons, and R Software (Version 4.3.2, R Foundation for Statistical Computing) for advanced regression modeling and data visualization.

Distribution normality was assessed via the Kolmogorov-Smirnov test and visual inspection of histograms. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR), while categorical variables were presented as frequencies (n) and percentages (%). To evaluate differences across NAFLD severity grades, One-way ANOVA or the Kruskal-Wallis H test was applied based on distribution characteristics. For non-parametric data, post-hoc pairwise comparisons between specific grades were performed using the Wilcoxon rank-sum test. Categorical associations were tested using the Chi-square test. Where applicable, the Bonferroni correction was implemented to adjust for multiple post-hoc comparisons. Box plots were generated using the ggplot2 and ggsignif packages in R.

To identify independent factors associated with disease severity, a hierarchical binary logistic regression analysis was conducted using the gtsummary, car, and DescTools packages in R. The outcome variable was dichotomized into 'Mild' (Grade 1) and 'Moderate-to-Severe' (Grades 2-3) steatosis. This dichotomization strategy was strictly employed to optimize statistical power, ensure model stability by achieving an adequate event-per-variable ratio in the logistic regression, and minimize potential minor misclassification bias between adjacent higher grades (Grade 2 vs. Grade 3) that commonly occurs in conventional ultrasonography. This grouping approach aligns with established epidemiological protocols where moderate and severe hepatic steatosis are combined to represent clinically significant metabolic fatty liver burden.

To evaluate the robustness of our binary outcome dichotomization (Grade 1 vs. Grades 2–3) and assess consistency across operational cutoffs, a rigorous sensitivity analysis protocol was implemented. First, ordinal logistic regression models were constructed across all three independent steatosis levels (Grades 1, 2, and 3) to preserve the original ordinal variance, and the proportional odds assumption was formally verified using the Brant test. Waist circumference consistently retained its independent association with steatosis grade across all individual dietary models ($p < 0.001$), while the Brant test verified that the parallel regression assumption was fully satisfied ($p = 0.09$). Second, alternative binary logistic regression iterations were evaluated by restricting boundaries, specifically, by completely excluding Grade 2 participants to isolate extreme phenotypes (Grade 1 vs. Grade 3) and by shifting the operational threshold to evaluate severe steatosis (Grades 1–2 vs. Grade 3), adjusting for identical baseline covariates. In this extreme phenotype verification, waist circumference remained the only stable independent correlate (Estimate = 0.104, SE = 0.027, $p < 0.001$). Finally, shifting the dichotomization threshold to evaluate associations with steatosis alone (Grades 1–2 vs. Grade 3) yielded identical stability for waist circumference across the full sample of 122 participants (Estimate = 0.070, SE = 0.021, $p < 0.001$), while all dietary quality indices remained statistically non-significant throughout all sensitivity scenarios. These sensitivity analyses demonstrate robustness to operational outcome dichotomization choices; however, they do not validate the ultrasound-based outcome

measure itself, and this fundamental limitation is discussed in the Limitations section.

The analysis was structured in two stages to isolate the effects of metabolic versus dietary factors. Initially, Model 1 was constructed including core anthropometric and clinical risk factors (waist circumference, sex, T2DM presence, and total energy intake) to establish the baseline metabolic risk. The hierarchical logistic regression models were constructed using a complete-case analysis approach, restricting the multivariate entry exclusively to participants with fully recorded waist circumference measurements (n = 122). To systematically assess potential selection bias introduced by this approach, the baseline clinical, anthropometric, and dietary characteristics of the excluded participants were rigorously compared against the analyzed sub-population. As detailed in Supplementary Table 1, no statistically significant differences were observed between the two groups across any of the evaluated parameters, including age, sex distribution, BMI, Type 2 diabetes prevalence, or baseline dietary indices, confirming that the missing data occurred completely at random. Subsequently, a representative selection strategy was applied to the dietary indices. Based on preliminary correlation analyses which revealed high collinearity among indices measuring similar constructs, one representative index was selected for each major nutritional dimension: HEI-2015 for overall diet quality, DII for inflammatory potential, PRAL for dietary acid load, and PDI for plant-based dietary adherence. These indices were added individually to the Model 1 to evaluate their incremental contribution to the statistical model for higher steatosis grades. Model fit and explanatory power were evaluated using the Hosmer-Lemeshow test and Nagelkerke's R^2, respectively. Multicollinearity among predictors was screened via Variance Inflation Factors (VIF), with values < 2.5 indicating robust model stability. Because multiple individual dietary indices were tested across separate sequential regression configurations, these models were considered exploratory in nature, and no formal alpha inflation correction for multiple testing was applied to the secondary dietary entries. A two-tailed p-value of <0.05 was considered statistically significant for all analyses.

Table 1
Baseline sociodemographic characteristics of patients according to NAFLD severity.

Sociodemographic Characteristics	Grade 1 (n = 50)		Grade 2 (n = 67)		Grade 3 (n = 43)		χ2	Total		p
	n	%	n	%	n	%				
Sex										
Men	9	18,0	16	23,8	8	18,6	0.011	33	20.6	0.917
Women	41	82,0	51	76,2	35	81,4		127	79.4	
Marital Status										
Married	37	74,0	52	77,6	31	72,1	0.938	120	75,0	0.626
Single	13	26,0	15	22,4	12	27,9		40	25,0	
Education Level										
Illiterate	-	-	4	6,0	1	2,3	9.176*	5	3,1	0.308*
Primary School Graduate	14	28,0	18	26,9	11	25,6		43	26,9	
Middle School Graduate	4	8,0	6	8,9	4	9,3		14	8,8	
High School Graduate	10	20,0	14	20,9	16	37,2		40	25,0	
University Graduate	22	44,0	25	37,3	11	25,6		58	36,2	
Occupation										
Worker	5	10,0	10	14,9	6	13,9	12.006*	21	13,2	0,738*
Retired	7	14,0	12	17,9	4	9,3		23	14,4	
Self-employed	6	12,0	13	19,4	9	20,9		28	17,5	
Homemaker	21	42,0	22	32,8	18	41,8		61	38,1	
Civil Servant	9	18,0	9	13,5	4	9,4		22	13,7	
Student	2	4,0	1	1,5	2	4,7		5	3,1	
Morbidities										
T2DM	36	72,0	58	86,5	36	83,7	-	130	81,2	-
Hashimoto's Thyroiditis	11	22,0	18	26,8	3	6,9		32	20,0	
Hypertension	8	16,0	11	16,4	8	18,6		27	16,8	
Goiter	11	22,0	7	10,4	8	18,6		26	16,2	
Smoking Status										
Yes	15	30,0	23	34,3	10	23,3	2156*	48	30,0	0,728*
No	31	62,0	39	58,2	28	65,1		98	61,2	
Quit	4	8,0	5	7,5	5	11,6		14	8,8	
Alcohol Consumption										
Yes	4	8,0	8	11,9	7	16,3	1405	19	11,9	0,504
No	46	92,0	59	88,1	36	83,7		141	88,1	
Physical Activity Level										
Inactive	36	72,0	46	68,7	28	65,1	1829*	110	68,8	0,795*
Minimally Active	13	26,0	19	28,3	12	27,9		44	27,5	
Very Active	1	2,0	2	3,0	3	7,0		6	3,7	

Data are presented as frequencies (n) and percentages (%). Statistical significance was evaluated using the Pearson Chi-square test or Fisher's exact test where applicable. A two-tailed p-value of <0.05 was considered statistically significant. Abbreviations: n = number of patients; T2DM = Type 2 Diabetes Mellitus. *Fisher's exact test.

3. Results

3.1. Sociodemographic and clinical characteristics

A total of 160 patients (20.6% men, 79.4% women) were included in the study. The study population was distributed into Grade 1 (31.2%), Grade 2 (41.9%), and Grade 3 (26.9%) steatosis. There were no significant differences in sociodemographic characteristics such as sex, marital status, or educational level among the steatosis grades ($p > 0.05$). The overall prevalence of T2DM in our sample was high at 81.2% (Table 1).

3.2. Anthropometric and biochemical parameters across steatosis grades

A significant relationship was identified between NAFLD severity and key anthropometric indicators. Specifically, body weight,

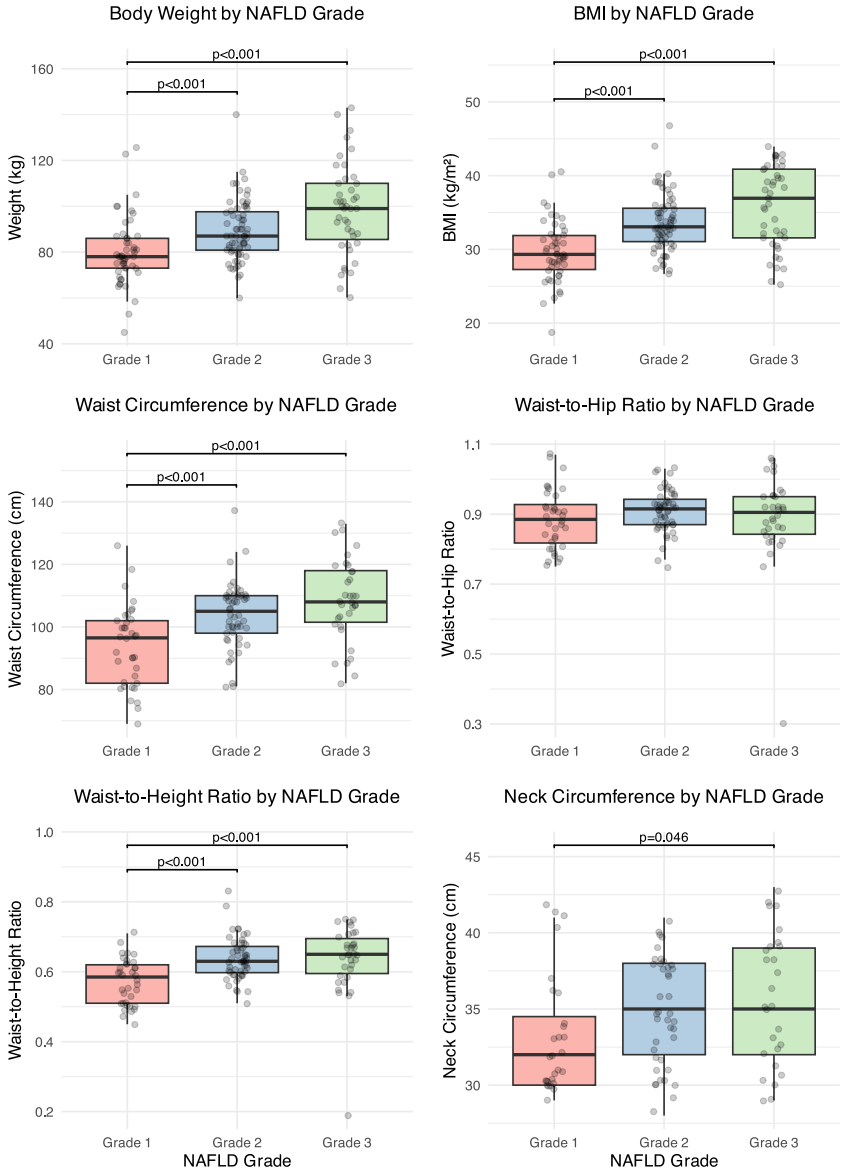


Fig. 3. Distribution of Anthropometric Parameters by NAFLD Grades. The panels display: (A) Body Weight, (B) Body Mass Index (BMI), (C) Waist Circumference, (D) Waist-to-Hip Ratio, (E) Waist-to-Height Ratio, and (F) Neck Circumference. In each plot, the central band denotes the median, while the box boundaries encompass the interquartile range (IQR). Whiskers represent the 1.5×IQR limit, with individual observations superimposed as jittered points. Pairwise comparisons revealing statistically significant differences (p-values) were calculated using the Wilcoxon rank-sum test. A two-tailed p-value of <0.05 was considered statistically significant for all comparisons.

BMI, waist circumference, and waist-to-height ratio were markedly elevated in patients with higher steatosis grades ($p < 0.001$ for all). Neck circumference also increased with disease severity ($p = 0.046$) (Fig. 3).

In contrast to the anthropometric findings, most biochemical parameters, including fasting glucose, triglycerides, low-density lipoprotein cholesterol (LDL-C), total cholesterol, and alanine aminotransferase (ALT), did not differ significantly across the severity grades. Hemoglobin was the only parameter showing a significant variation ($p = 0.014$). (Detailed results in Supplementary Table 2).

3.3. Dietary intake and nutrient analysis

There were no statistically significant differences in total energy intake or the intake of carbohydrates and protein across the three groups. Total fat intake showed a non-significant increasing trend with disease severity ($p = 0.065$), while saturated fatty acid intake was significantly lower in patients with higher steatosis grades ($p = 0.025$). The intake of most micronutrients was also comparable across groups, except for phosphorus ($p = 0.030$). (Detailed nutrient analysis in Supplementary Tables 3 and 4).

Analysis of food group consumption revealed a significant difference only in the intake of legumes and nuts, which was significantly lower in patients with severe steatosis compared to other groups ($p < 0.001$). (Consumption data for other food groups are available in Supplementary Table 5).

3.4. Evaluation of dietary indices

The assessment of total HEI-2015 scores revealed no significant disparity among the groups ($p = 0.509$). Notably, this reflects a consistently suboptimal diet quality across the entire study cohort. (Detailed HEI-2015 component scores are available in Supplementary Table 6). Furthermore, indices measuring the inflammatory potential and acid load of the diet, including the DII, PRAL, NEAP, and DAL, showed no significant variation among the NAFLD severity groups. Similarly, PDI scores did not differ by steatosis severity (Supplementary Table 7).

3.5. Factors independently associated with steatosis grade

To identify factors independently associated with disease severity and isolate the effects of dietary composition from metabolic factors, a hierarchical logistic regression analysis was performed (Table 2). In Model 1, which included core clinical risk factors, waist circumference was identified as a consistent independent factor associated with higher ultrasound-defined steatosis grade. After adjusting for sex, T2DM, and total energy intake, every 1 cm increase in waist circumference was associated with a 10% increase in the odds of having moderate-to-severe steatosis (OR: 1.10, 95% CI: 1.05–1.16, $p < 0.001$). The base model explained 27% of the variance in disease severity (Nagelkerke $R^2 = 0.270$).

In the subsequent steps (Models 2–5), dietary indices were added individually to the base model. None of the dietary indices yielded statistical significance ($p > 0.05$ for all). Furthermore, the addition of these dietary variables resulted in negligible improvement in the model's explanatory power (change in $R^2 < 0.015$). This indicates that within this specific clinical dataset, waist circumference demonstrated a consistent independent association with higher steatosis grades, whereas dietary indices assessed here did not show independent associations after metabolic factors were controlled. This pattern should be interpreted in the context of limitations of

Table 2
Hierarchical logistic regression analysis of anthropometric and dietary risk factors for higher steatosis grades (n = 122).

Variable	Odds Ratio (OR)	95% CI	P-Value	Model R^2	VIF
MODEL 1: Base Model (Anthropometric & Clinical Risk Factors)					
Waist Circumference (cm)	1.10	1.05, 1.16	**<0.001**	0.270	1.35
Sex (Female)				0.270	1.35
0	—	—		0.270	
1	2.54	0.695, 9.15	0.150	0.270	
Type 2 Diabetes (Yes)				0.270	1.05
0	—	—		0.270	
1	1.21	0.352, 3.93	0.757	0.270	
Total Energy Intake (kcal)	1.00	0.999, 1.00	0.721	0.270	1.05
MODEL 2: Base Model + Diet Quality (HEI-2015)					
HEI-2015	1.02	0.981, 1.07	0.288	0.281	1.06
MODEL 3: Base Model + Inflammation (DII)					
DII	1.03	0.493, 2.12	0.936	0.270	1.03
MODEL 4: Base Model + Acid Load (PRAL)					
PRAL	0.979	0.925, 1.03	0.454	0.275	1.69
MODEL 5: Base Model + Plant-Based Index (PDI)					
PDI	1.02	0.944, 1.11	0.581	0.273	1.02

Abbreviations: CI = Confidence Interval, OR = Odds Ratio.
Model R^2: Nagelkerke R-Squared. VIF: Variance Inflation Factor. Model 1 serves as the baseline reference model; subsequent models (Models 2–5) represent the independent addition of each specific dietary index to Model 1. A two-tailed p-value of <0.05 was considered statistically significant; p-values demonstrating statistical significance are highlighted in bold font. Abbreviations: CI = Confidence Interval; DII = Dietary Inflammatory Index; HEI-2015 = Healthy Eating Index-2015; OR = Odds Ratio; PDI = Plant-Based Diet Index; PRAL = Potential Renal Acid Load.

both the dietary assessment method and the ultrasound outcome measure.

4. Discussion

A key finding of this study is the marked disparity between anthropometric and dietary profiles across the spectrum of NAFLD severity. While anthropometric risk factors -particularly measures of central obesity-progressively worsened with increasing steatosis, the dietary profile, including total energy intake, macro- and micronutrient consumption, and scores from various dietary indices, remained uniformly poor and largely undifferentiated across all groups. This suggests that while the dietary indices assessed were not independently associated with higher steatosis grades in this dataset using these specific dietary assessment methods, central adiposity indices remained consistently associated with ultrasound-defined steatosis.

4.1. The role of central adiposity in disease severity

Our data confirm that the progression of NAFLD is mirrored by a significant rise in adiposity. We identified a robust graded association where BMI, waist circumference, and waist-to-height ratio increased progressively from Grade 1 to Grade 3. This aligns with extensive research identifying obesity as a primary engine of NAFLD pathogenesis (Le et al., 2022).

A notable finding from the hierarchical regression analysis (Model 1) was the independent association of waist circumference with higher steatosis grades, which persisted after adjustment for sex, diabetes, and energy intake (OR: 1.10, $p < 0.001$). Our findings are consistent with studies showing that increased waist circumference is associated with an increased risk of NAFLD severity (De et al., 2025). Consistent with prior evidence, a meta-analysis reported that abdominal obesity defined by waist circumference was more strongly associated with NAFLD risk than BMI-defined obesity in population-level analyses (Pang, 2015), a pattern that appears to extend to ultrasound-defined severity in the present clinically selected cohort. This supports the concept that visceral adipose tissue is a metabolically active organ that drains directly into the portal circulation, exposing the liver to pro-inflammatory cytokines (Sakers et al., 2022). Consequently, our results are consistent with existing evidence suggesting that waist circumference may offer clinically meaningful information alongside BMI for risk characterization in NAFLD, particularly in high-risk metabolic populations.

4.2. The dietary profile: Why nutrients were not associated with severity?

In our hierarchical analysis (Models 2–5), dietary indices (HEI-2015, DII, PRAL, PDI) did not show independent associations with higher steatosis grades. This null finding must be interpreted cautiously, as it may partially reflect measurement imprecision inherent to a non-validated FFQ rather than a definitive lack of biological relationship. Nevertheless, the descriptive observation that dietary quality and inflammatory scores were uniformly poor across all grades indicates a generalized nutritional vulnerability in this study population. While suboptimal dietary habits did not statistically stratify disease severity in our specific models, their consistent presence across the entire study population suggests that it may be prudent to consider comprehensive nutritional evaluation and lifestyle modification even at early stages of clinical management.

Biologically, a high-quality diet, rich in fiber, antioxidants, and unsaturated fats, is theoretically expected to protect the liver by reducing systemic inflammation and oxidative stress (Alahmari, 2024). Yet, our findings of a universally poor diet quality align with Xu et al. (2024), who also reported generally low diet quality in this patient population.

Furthermore, while some studies report an inverse relationship between diet quality and NAFLD severity, this relationship is predominantly observed in individuals of normal body weight (Huang et al., 2024). The protective effect of a high-quality diet is significantly attenuated in patients with T2DM and central obesity (Heredia et al., 2022; Yoo et al., 2020). Given that our sample reflects a highly selected, third-tier referral clinical pattern with an overwhelming T2DM prevalence of 81.2%, these baseline metabolic complexities likely masked the subtle independent associations of dietary variations. In such a high-risk metabolic phenotype, advanced insulin resistance and potential pharmacological treatments significantly modulate hepatic lipid pathways. Consequently, the lack of statistical significance for dietary indices within this specific sample should not be generalized to a broader, healthier NAFLD population, but rather interpreted as a reflection of a saturated metabolic profile where underlying disease severity and physiological disruption override the cross-sectional influence of current dietary quality.

Regarding plant-based diets, while hPDI is shown to have protective effects in some cohorts (Li et al., 2023), other findings show no relationship between PDI and NAFLD (Fallah et al., 2024). Similarly, although pro-inflammatory diets are linked to severe NAFLD risk (Petermann-Rocha et al., 2023), this relationship is often non-linear and significantly attenuated after adjusting for BMI and T2DM (Li et al., 2023). Future studies using more comprehensive tools, such as the Oxidative Balance Score, may offer stronger explanatory value (Liu et al., 2023).

Based on all these findings, the absence of a statistically detectable relationship between NAFLD severity and dietary indices in this dataset is likely multifactorial. First, in contrast to the dynamic changes in anthropometry, the dietary profile of our sample was remarkably static across severity grades. Second, the overwhelming prevalence of obesity and T2DM makes the additional graded impact of diet difficult to detect. Finally, NAFLD severity is associated with a complex interplay of lifestyle and genetic factors beyond diet alone.

4.3. Limitations

Several limitations must be considered when interpreting our findings. Primarily, the cross-sectional nature of the study design

inherently restricts the determination of causality, as both exposure and outcome were measured simultaneously. Consequently, the potential for reverse causality cannot be entirely ruled out, as individuals aware of their NAFLD status might have recently modified their dietary or lifestyle behaviors in response to prior clinical counseling, potentially obscuring long-term nutritional patterns. To elucidate the temporal impact of dietary risks on NAFLD progression, large-scale prospective longitudinal studies are warranted. Additionally, the statistical power to capture subtle nutritional variations might have been constrained by the relatively modest sample size. Second, dietary data were collected using a non-validated FFQ designed by the research team, introducing a risk of information bias and measurement error in exposure classification. Although this limitation was partially mitigated by employing two independent 24-h dietary recalls to cross-verify nutritional macro-patterns, the lack of precision in capturing long-term micro-nutrient variations may have limited our ability to identify subtle, graded linear associations between dietary indices and steatosis severity. Furthermore, the assessment of NAFLD severity relied on conventional semi-quantitative ultrasonography rather than validated quantitative modalities such as controlled attenuation parameter (CAP), MRI-proton density fat fraction (MRI-PDFF), or liver biopsy. Although imaging was performed by a single experienced endocrinologist blinded to dietary intake, a single-operator design eliminates inter-observer variability only because no second observer was present; it does not eliminate intra-observer bias or provide a measure of intra-observer reproducibility. No formal intra-observer repeatability assessment was conducted, and the ultrasound findings were not validated against any quantitative reference standard. All imaging was performed using a Mindray Resona I9 ultrasound system equipped with a 1.2–6.0 MHz SC6-1s convex transducer. Associations reported in this study should be understood as pertaining specifically to ultrasound-defined steatosis grade, and this represents a major limitation of the present work.

Crucially, our study population reflects a highly selected clinical sample with an exceptional burden of metabolic comorbidities, particularly T2DM (81.2%). The lack of granular data regarding disease duration, precise long-term glycemic control metrics (e.g., serial HbA1c), and the concurrent use of metabolic medications represents a substantial unmeasured constraint. Concomitant therapies, including GLP-1 receptor agonists, SGLT2 inhibitors, insulin, statins, and anti-obesity agents, exert profound independent effects on body weight, visceral adiposity, hepatic lipid accumulation, and dietary behaviors. Because these pharmacological confounding variables were not adjusted for in the regression models, our results must be interpreted strictly within the boundaries of this specific metabolic phenotype, limiting generalizability to the broader, non-diabetic NAFLD spectrum.

Finally, certain biological and physiological parameters, such as direct biochemical markers of insulin resistance and sex-specific menopausal status, were not evaluated. The study is also constrained by the absence of a healthy control group, which limits our ability to quantify the true extent of these dietary and anthropometric deficits relative to a non-diseased population. Future research accounting for these unassessed physiological, pharmacological, and clinical variables is essential to comprehensively map out the nutritional epidemiology of NAFLD.

4.4. Conclusions

In conclusion, while the strong association between waist circumference and steatosis severity aligns with expected anthropometric trends, our findings reveal that dietary quality is uniformly poor across all NAFLD grades, even in the earliest mild stages. The lack of independent associations between the evaluated dietary indices and disease severity should be strictly interpreted within the context of the non-validated dietary assessment tools used, the ultrasound-based outcome measure, and the metabolic complexity of this clinical sample, rather than concluding that dietary composition lacks biological importance. This sub-optimal nutritional baseline is particularly concerning for patients with co-existing metabolic conditions such as obesity and T2DM, where the intersection of a high-risk diet and metabolic disease creates a highly vulnerable physiological state from the outset. Therefore, clinical management in this selected cohort could benefit from incorporating early nutritional assessment alongside strategies targeting central obesity. However, given the cross-sectional design, the aforementioned dietary assessment constraints, and the ultrasound-based outcome measure, these observations should be regarded as hypothesis-generating.

Ethical approval

This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol received formal ethical approval from the Akdeniz University Clinical Research Ethics Committee (KAEK-168). Prior to enrollment, written informed consent was obtained from all participants.

Consent for publication

Not applicable. This study does not contain any individual person's data in any form (including individual details, images, or videos). All data presented are aggregated group-level findings.

Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly, but are available from the corresponding author on reasonable request, subject to institutional data-sharing policy.

Declaration of generative AI and AI-assisted technologies

During the preparation of this work the author(s) used Google Gemini in order to assist with English translation and stylistic refinement. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Travel and participation support for the presentation of preliminary results at the ESPEN Congress was provided by The Scientific and Technological Research Council of Turkey (TÜBİTAK) [Grant Program 2224-A]. The funding agency had no role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript.

CRediT authorship contribution statement

Caner Özyildirim: Conceptualization, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft. **Mustafa Aydemir:** Data curation, Resources, Validation, Writing – review & editing. **Nurcan Yabanci Ayhan:** Conceptualization, Methodology, Supervision, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

N/A.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.obmed.2026.100728.

References

Alahmari, L.A., 2024. Dietary fiber influence on overall health, with an emphasis on CVD, diabetes, obesity, colon cancer, and inflammation. Front. Nutr. 11, 1510564.
Ashwell, M., Hsieh, S.D., 2005. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. Int. J. Food Sci. Nutr. 56 (5), 303–307.
Bakhshimoghaddam, F., Baez, D., Dolatkhah, N., et al., 2024. Which dietary patterns fend off nonalcoholic fatty liver disease? A systematic review of observational and interventional studies. BMC Nutrition 10 (1), 153.
Ben-Noun, L., Sohar, E., Laor, A., 2001. Neck circumference as a simple screening measure for identifying overweight and Obese patients. Obes. Res. 9 (8), 470–477.
Craig, C.L., Marshall, A.L., Sjöström, et al., 2003. International physical activity questionnaire: 12-Country reliability and validity. Med. Sci. Sports Exerc. 35 (8), 1381–1395.
De, A., Bhagat, N., Mehta, M., et al., 2025. Central obesity is an independent determinant of advanced fibrosis in lean patients with nonalcoholic Fatty liver disease. J. Clin. Exp. Hepatol. 15 (1), 102400.
Del, Bo' C., Perna, S., Allehdan, S., et al., 2023. Does the Mediterranean Diet have any effect on lipid profile, central obesity and liver enzymes in non-alcoholic fatty liver disease (NAFLD) subjects? A systematic review and meta-analysis of randomized control trials. Nutrients 15 (10), 2250.
Fallah, A., Abdolazimi, H., Darabi, Z., et al., 2024. The association between score of plant-based diet and non-alcoholic fatty liver disease in adults. Clinical Nutrition ESPEN 61, 407–412.
Frassetto, L.A., Todd, K.M., Morris, R.C., et al., 1998. Estimation of net endogenous noncarbonic acid production in humans from diet potassium and protein contents. Am. J. Clin. Nutr. 68 (3), 576–583.
Gürsoy, N., 2023. Evaluation of Daily Dietary Fiber Consumption of Individuals with Non-alcoholic Fatty Liver [Master Thesis. Bahcesehir University İstanbul.
Hamamah, S., Iatcu, O.C., Covasa, M., 2024. Dietary influences on gut Microbiota and their role in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Nutrients 17 (1), 143.
Hassani Zadeh, S., Mansoori, A., Hosseinzadeh, M., 2021. Relationship between dietary patterns and non-alcoholic fatty liver disease: a systematic review and meta-analysis. J. Gastroenterol. Hepatol. 36 (6), 1470–1478.
Heredia, N.I., Zhang, X., Balakrishnan, M., et al., 2022. Physical activity and diet quality in relation to non-alcoholic fatty liver disease: a cross-sectional study in a representative sample of U.S. adults using NHANES 2017–2018. Prev. Med. 154, 106903.
Homayounfar, N., Rivaz, R., Sedghi, Z., et al., 2026. The association between dietary acid load and non-alcoholic fatty liver disease. Food Sci. Nutr. 14 (5), e71597.
Huang, X., Gan, D., Fan, Y., et al., 2024. The associations between healthy eating patterns and risk of metabolic dysfunction-associated steatotic liver disease: a case–control study. Nutrients 16 (12), 1956.
Krebs-Smith, S.M., Pannucci, T.E., Subar, A.F., et al., 2018. Update of the Healthy Eating Index: HEI-2015. J. Acad. Nutr. Diet. 118 (9), 1591–1602.
Le, M.H., Yeo, Y.H., Zou, B., et al., 2022. Forecasted 2040 global prevalence of nonalcoholic fatty liver disease using hierarchical bayesian approach. Clin. Mol. Hepatol. 28 (4), 841–850.
Lee, R.D., Nieman, D.C., 2010. Nutritional Assessment, fifth ed. McGraw-Hill Incorporation, New York. New York.

Li, R., Li, M., Bidulescu, A., et al., 2023. Diets with higher anti-inflammatory potential associated with lower risk of development of nonalcoholic Fatty liver disease and early-stage of fibrosis among US adults. Dig. Dis. Sci. 68 (10), 4009–4021.
Liu, Xuna, Wang, Y., Liu, Xuyan, et al., 2023. Higher oxidative balance scores are associated with lower nonalcoholic fatty liver disease and not with fibrosis in US adults. Nutr. Metabol. Cardiovasc. Dis. 33 (12), 2488–2496.
Miryan, M., Azizi, A., Pasdar, Y., et al., 2025. Adherence to plant based diets reduce the risk of hepatic fibrosis in nonalcoholic fatty liver disease. Sci. Rep. 15 (1), 17403.
Pang, Q., 2015. Central obesity and nonalcoholic fatty liver disease risk after adjusting for body mass index. World J. Gastroenterol. 21 (5), 1650.
Peña-Jorquera, H., Cid-Jofré, V., Landaeta-Díaz, L., et al., 2023. Plant-Based nutrition: exploring health benefits for atherosclerosis, chronic diseases, and Metabolic Syndrome—A comprehensive review. Nutrients 15 (14), 3244.
Petermann-Rocha, F., Wirth, M.D., Boonpor, J., et al., 2023. Associations between an inflammatory diet index and severe non-alcoholic fatty liver disease: a prospective study of 171,544 UK Biobank participants. BMC Med. 21 (1), 123.
Remer, T., Manz, F., 1994. Estimation of the renal net acid excretion by adults consuming diets containing variable amounts of protein. Am. J. Clin. Nutr. 59 (6), 1356–1361.
Remer, T., Manz, F., 1995. Potential renal acid load of foods and its influence on urine pH. J. Am. Diet. Assoc. 95 (7), 791–797.
Rinella, M.E., Sookoian, S., 2024. From NAFLD to MASLD: updated naming and diagnosis criteria for fatty liver disease. JLR (J. Lipid Res.) 65 (1), 100485.
Sakers, A., De Siqueira, M.K., Seale, P., et al., 2022. Adipose-tissue plasticity in health and disease. Cell 185 (3), 419–446.
Satija, A., Bhupathiraju, S.N., Rimm, E.B., et al., 2016. Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: results from three prospective cohort studies. PLOS Medicine 13 (6), e1002039.
Sheikh, M.Y., Younus, M.F., Shergill, A., et al., 2025. Diet and lifestyle interventions in metabolic dysfunction-associated fatty liver disease: a comprehensive review. Int. J. Mol. Sci. 26 (19), 9625.
Shivappa, N., Steck, S.E., Hurley, T.G., et al., 2014. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 17 (8), 1689–1696.
Wang, Q., Chen, R., Chen, S., et al., 2025. Exploring the association between dietary indices and metabolic dysfunction-associated steatotic liver disease: mediation analysis and evidence from NHANES. PLoS One 20 (4), e0321251.
World Health Organization. Obesity and overweight [Internet]. [cited 2026 Jan 9]; Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
World Health Organization, 2011. Waist circumference and waist-hip ratio: report of a WHO expert consultation [Internet]. [cited 2026 Jan 9]; Available from: https://www.who.int/publications/i/item/9789241501491.
Xiang, M., Tian, X., Wang, H., et al., 2024. Inappropriate diet exacerbates metabolic dysfunction-associated steatotic liver disease via abdominal obesity. Nutrients 16 (23), 4208.
Xu, M., Zhan, Y., Gao, G., et al., 2024. Associations of five dietary indices with metabolic dysfunction-associated steatotic liver disease and liver fibrosis among the United States population. Front. Nutr. 11, 1446694.
Yoo, E.R., Kim, D., Vazquez-Montesino, L.M., et al., 2020. Diet quality and its association with nonalcoholic fatty liver disease and all-cause and cause-specific mortality. Liver Int. 40 (4), 815–824.
Younossi, Z.M., Golabi, P., Paik, J.M., et al., 2023. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. Hepatology 77 (4), 1335–1347.
Zhu, B., Chan, S.-L., Li, J., et al., 2021. Non-alcoholic steatohepatitis pathogenesis, diagnosis, and treatment. Front. Cardiovasc. Med. 8, 742382.