Turkish Journal of Health Science and Life

Exploring The Impact of Dietary and Metabolic Risk Factors on Non-Alcoholic Fatty Liver Disease (NAFLD) : A Cross-Sectional Study

İzel Aycan Orakçı [a*], İlyas Tuncer [b], Fatma Esra Güneş [c]

[a] Department of Nutrition and Dietetics, Bucak School of Health, Burdur Mehmet Akif Ersoy University, 15300 Bucak, Burdur, Türkiye
[b] Division of Gastroenterology, Department of Internal Medicine, School of Medicine, Istanbul Medeniyet University, Istanbul, Türkiye
[c] Department of Nutrition and Dietetics, Istanbul Medeniyet University, Faculty of Health Sciences, Istanbul, Türkiye

ARTICLE INFO

RESEARCH ARTICLE

Article history:
Received: 17 April 2025
Accepted: 10 June 2025
Available : 30 August 2025

[a] https://orcid.org/0000-0003-1199-3194
[b] https://orcid.org/0000-0001-8866-9734
[c] https://orcid.org/0000-0003-1693-6375

*Correspondence: İzel Aycan Orakçı
Department of Nutrition and Dietetics, Bucak School of Health, Burdur Mehmet Akif Ersoy University, 15300 Bucak, Burdur, Türkiye
e-mail: dyt.aycanbasoglu@hotmail.com

Turkish Journal of Health Science and Life
2025, Vol.8, No.2, 29-44.
DOI: https://doi.org/tjhsl.1677733

ABSTRACT

Objectives: This study aimed to investigate the associations between the severity of non-alcoholic fatty liver disease (NAFLD) and a range of dietary, anthropometric, and biochemical parameters in adults with clinically diagnosed NAFLD.

Materials and Methods: A total of 114 adult NAFLD patients were included in this cross-sectional study. Dietary intake was assessed using three 24-hour recalls. Participants were stratified by steatosis grade (Grade 1–3) based on ultrasonographic evaluation. Anthropometric measurements and biochemical markers were analyzed across groups. Ordinal logistic regression and ANOVA were used to evaluate group differences and associations.

Results: Dietary cholesterol intake was significantly higher in the moderate steatosis group ($p=0.026$). Waist circumference, body fat percentage, and muscle mass were significantly associated with steatosis severity. However, no significant differences were observed among steatosis grades in energy, macronutrients, fiber, or polyunsaturated fatty acids (PUFA) intake. Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), fasting glucose, and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) levels were elevated in more advanced steatosis groups ($p<0.05$), while lipid parameters such as low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol and triglycerides (TG) showed no significant differences.

Conclusion: Findings suggest that metabolic and anthropometric parameters—rather than general dietary intake—are more strongly associated with the severity of NAFLD. Early monitoring of central adiposity, insulin resistance, and hepatic enzymes may improve risk stratification and support timely interventions.

Key Words: *Anthropometric measurements, Dietary intake, Hepatic enzymes, Metabolic risk factors, NAFLD*

1. INTRODUCTION

The global prevalence of non-alcoholic fatty liver disease (NAFLD) is estimated to be approximately 30% (1-3). NAFLD is characterized by excessive hepatic fat accumulation defined by the presence of steatosis in more than 5% of hepatocytes in individuals who consume little or no alcohol and encompasses a clinical spectrum ranging from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma (4, 5).

The pathogenesis of NAFLD involves complex interactions between multiple factors. Insulin resistance, hyperinsulinemia, dyslipidemia, genetic predisposition, and lifestyle choices, including diet and sedentary behavior contribute to lipid accumulation in the liver (6, 7). In particular, high intakes of energy, sugar, saturated fats, trans fats, and cholesterol have been associated with worsening hepatic steatosis, whereas low intake of n-3 polyunsaturated fatty acids (PUFAs) and dietary fiber has been linked to disease progression (8-12).

Increased visceral adiposity in obesity leads to higher

concentrations of circulating free fatty acids (FFAs), promoting fat deposition in the liver. Consequently, NAFLD is more prevalent among individuals with central obesity (5, 13). Anthropometric indices such as body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), and visceral adiposity index (VAI) have been shown to be significant predictors of NAFLD (14-16). Similarly, various biochemical markers, including triglycerides, HDL cholesterol, liver enzymes (ALT and AST), glucose, total protein, and HOMA-IR, are useful indicators of liver function and overall metabolic health in NAFLD patients (17, 18).

While NAFLD has been widely studied, the combined impact of dietary intake and metabolic indicators on steatosis severity remains insufficiently understood. To address this, the present study investigates the relationships between liver steatosis and dietary composition, anthropometric measures, and biochemical parameters in adults diagnosed with NAFLD. The findings are likely to contribute to identifying potential dietary and metabolic predictors of hepatic steatosis severity, thereby supporting early interventions and treatment approaches.

2. MATERIALS AND METHODS

2.1. Study Design and Subjects

This observational, cross-sectional analytical study was conducted between February 2022 and December 2024 at the Gastroenterology Department of Göztepe Prof. Dr. Süleyman Yalçın City Hospital in Istanbul, Turkey. A total of 114 adults diagnosed with non-alcoholic fatty liver disease (NAFLD) were included in the study. Individuals aged 18 to 65 years with ultrasonography-confirmed NAFLD and alcohol consumption below 30 g/day for men and 20 g/day for women were eligible for inclusion. Exclusion criteria included the presence of hepatitis B or C, cirrhosis, Wilson's disease, autoimmune liver diseases, hypothyroidism, cancer, inflammatory bowel diseases (Crohn's disease and ulcerative colitis), celiac disease, cystic fibrosis, hereditary disorders, irritable bowel syndrome, kidney diseases, eating disorders, allergic conditions, rheumatic diseases, AIDS/HIV, Parkinson's disease, epilepsy, a history of bariatric surgery, pregnancy or lactation, recent surgery or bypass operation within the past three months, and the use of experimental drugs, insulin, or antibiotics.

The degree of hepatic steatosis in all participants was assessed via conventional ultrasonography. Demographic information was collected through a structured questionnaire. Additionally, dietary intake records were obtained, anthropometric measurements were conducted, and biochemical laboratory parameters were analyzed. Ethical approval for the study was granted by the Ethics Committee of the Marmara University Faculty of Medicine (approval date: October 8, 2021; No: 09.2021.1109).

2.2. Noninvasive Quantification of Liver Steatosis

Conventional ultrasonography (CUS) examinations were performed by a board-certified radiologist with expertise in abdominal imaging, who was blinded to the participants' clinical data. These images were subsequently reviewed by the same radiologist for CUS scoring. CUS scoring of liver steatosis was performed qualitatively on a 3-point ordinal scale adapted from Ballestri et al. and Paige et al.'s research (19, 20). Hepatic steatosis was categorized as follows: Grade 1 – mild/intermediate steatosis (CUS score 1), Grade 2 – moderate steatosis (CUS score 2), and Grade 3 – severe steatosis (CUS score 3).

2.3. Anthropometric and Laboratory Measurements

Anthropometric parameters were measured twice. If a second measurement was not close enough to the first measurement, a third measurement was taken. A body composition analyzer based on bioelectrical impedance analysis (BIA) was used to determine body weight and body fat percentage. The measurements were conducted in accordance with key BIA protocols, including a minimum fasting period of 4 hours prior to the assessment, avoidance

of vigorous physical activity for 24–48 hours before the test, and exclusion of measurements during the menstrual period (21). Height measurements were obtained using a stadiometer while participants stood upright in a straight position. The movable headpiece was gently adjusted to rest on the top of the participant's head, and the measurement was recorded to the nearest 1 mm. Body mass index (BMI) was calculated using the formula BMI = weight / height^2 (kg/m^2), reflecting the distribution of body weight relative to height. Waist and hip circumferences were measured using a non-elastic measuring tape. For waist circumference (WC), the participant stood upright with a relaxed abdomen, arms at the sides, and feet together. The tape was placed horizontally around the abdomen at the midpoint between the lowest rib and the iliac crest, and the measurement was taken at the end of a normal expiration. BMI, waist circumference (WC), and waist-to-hip ratio (WHR) were interpreted according to the classification criteria established by the World Health Organization (22). BMI was categorized as follows: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), obesity class I (30.0-34.9 kg/m^2), obesity class II (35.0-39.9 kg/m^2) and obesity class III (>40.0 kg/m^2). Waist circumference was evaluated as an indicator of abdominal obesity, with increased risk defined as ≥94 cm in men and ≥80 cm in women, and substantially increased risk as ≥102 cm in men and ≥88 cm in women. For waist-to-hip ratio, values above 0.90 for men and 0.85 for women were considered indicative of increased cardiometabolic risk (22).

The Visceral Adiposity Index (VAI) was computed using sex-specific formulas (23).

For males: [WC / (39.68 + 1.88 × BMI)] × (TG / 1.03) × (1.31 / HDL-c);

For females: [WC / (36.58 + 1.89 × BMI)] × (TG / 0.81) × (1.52 / HDL-c).

In these equations, waist circumference (WC) was measured in centimeters, while triglyceride (TG) and high-density lipoprotein cholesterol (HDL-c) concentrations were expressed in mmol/L.

Peripheral venous blood samples were collected after fasting overnight. The biochemical indicators included the following: fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), serum fasting insulin and, hemoglobin A1C (HbA1C) and homeostasis model assessment of insulin resistance (HOMA-IR).

2.4. Dietary Nutrients Intake Assessment

Dietary intake data were collected through three 24-hour dietary recalls (24-HDRs), including two weekdays and one weekend day, to assess usual intake and account for day-to-day variability. All interviews were conducted face-to-face by a trained dietitian using a structured and standardized protocol to minimize recall bias. Reported food and beverage items were portioned using visual aids and household measurement tools, including a validated photo atlas (24). Nutrient and energy intakes were analyzed using BeBiS (Nutrition Information System) version 9.0, the Turkish adaptation of the Ebispro software (Stuttgart, Germany).

2.5. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics (version 22, IBM Corp., Armonk, NY, USA). Continuous variables were presented as means and standard deviations (SD), while categorical variables were reported as frequencies and percentages. A two-tailed p-value of <0.05 was considered statistically significant for all analyses.

To assess group differences in demographic, anthropometric, biochemical, and dietary variables across hepatic steatosis grades (Grade 1–3), one-way ANOVA was used for normally distributed variables. When normality assumptions were not met, the Kruskal–Wallis H test was applied. Chi-square tests

were conducted to compare categorical variables. To identify independent predictors of hepatic steatosis severity, ordinal logistic regression analysis was performed. A fully adjusted model controlling for age, body mass index (BMI), and total energy intake was selected as the final model. The cumulative odds ordinal logistic regression model assumes that the relationship between each pair of outcome groups is the same.

The general model can be written as:

$$\text{logit}[P(Y \leq j)] = \alpha\_j - (\beta_1 X_1 + \beta_2 X_2 + \ldots + \beta_k X_k)$$

where j is the category of the ordinal dependent variable (steatosis grade), α_j is the threshold for category j, and β_k represents the coefficients of the independent variables X_k.

All available parameters were initially considered for inclusion in the regression models. However, due to statistical constraints such as low variance, insufficient number of valid observations, or multicollinearity leading to model instability, several variables were excluded from the final analyses.

3. RESULT AND DISCUSSION

3.1. Results

Table 1 presents the demographic and anthropometric characteristics of the study population (N = 114). The majority of participants were female (68.4%), with a mean age of 47.0 ± 12.3 years. The average height and body weight were 163.6 ± 9.9 cm and 88.3 ± 17.3 kg, respectively . The mean BMI was 32.9 ± 5.1 kg/m^2, indicating that the majority of individuals were overweight or obese. Participants were primarily classified as Obesity Class I (44.7%), followed by Class II (23.7%) and Class III (5.2%), with only 2.7% falling within the normal weight range. Visceral adiposity index (VAI) was 0.9 ± 0.4. The mean body fat percentage was 41.0 ± 8.8%, and the average muscle mass was 29.1 ± 7.6 kg. Waist and hip circumferences were 104.7 ± 12 cm and 114.9 ± 12.9 cm, respectively, with a mean waist/hip ratio of 0.9 ± 0.1. Based on waist circumference, 80.7% of participants were categorized as having a substantially increased risk of metabolic complications. Similarly, based on waist/hip ratio, 81.6% were classified as having substantially increased metabolic risk. Regarding liver steatosis, nearly half of the participants (48.2%) were classified as Grade 1, while 35.1% were Grade 2 and 16.7% were Grade 3.

Table 2 compares the demographic, anthropometric, biochemical, and dietary intake characteristics of participants across the three grades of hepatic steatosis (Grade 1–3). Sex distribution differed significantly across groups (p = 0.018), with a higher proportion of females in Grade 1 and more males in Grade 2.

Although mean BMI, body weight, and waist circumference increased from Grade 1 to Grade 3, the differences did not reach statistical significance. Notably, body fat percentage was significantly lower in the moderate steatosis group compared to the mild and severe groups (p = 0.017), suggesting possible variability in fat distribution among grades. Liver enzyme levels demonstrated a progressive and statistically significant increase with steatosis severity. AST, ALT, and GGT levels were significantly higher in Grades 2 and 3 compared to Grade 1 ($p < 0.05$ for all pairwise comparisons). Fasting blood glucose (FBG), insulin, HOMA-IR, and HbA1c values also rose with increasing GRADE, showing statistically significant elevations particularly in Grade 3.

In terms of dietary intake, dietary cholesterol intake was significantly lower in Grade 3 compared to Grades 1 and 2 (p = 0.026), while other macronutrients and micronutrients showed no significant differences. Total energy intake, macronutrient composition (carbohydrate, protein, and fat), dietary fiber, and vitamin/mineral intakes were similar across grades.

In order to investigate potential sex-specific differences in the severity of hepatic steatosis, subgroup analyses were performed separately for female and male participants. Among female

Table 1. Characteristics and Anthropometric Variables of Participants

Characteristics	**n=114**
Sex (n(%))	
Female	78 (68.4%)
Male	36 (31.6%)
Age (year)	47 ± 2.3
Height (cm)	163.6 ± 9.9
Body weight (kg)	88.3 ± 7.3
BMI (kg/m²)	32.9 ± 5.1
BMI Classification (N(%))	
Normal weight	3 (2.7%)
Overweight	27 (23.7%)
Obesity class I	51 (44.7%)
Obesity class II	27 (23.7%)
Obesity class III	6 (5.2%)
VAI	0.9 ± 0.4
Body fat (kg) ǂ	36.1 ± 10.5
Body fat (%)ǂ	41 ± 8.8
Mass muscle (kg)	29.1 ± 7.6
Waist circumference (cm)	104.7 ± 12
Hip circumference (cm)	114.9 ± 12.9
Waist/Hip ratio	0.9 ± 0.1
Waist circumference classification for risk of metabolic complications	
Normal	4 (3.5%)
Increased	18 (15.8%)
Substantially increased	92 (80.7%)
Waist/hip ratio classification for risk of metabolic complications (N(%))	
Normal	21 (18.4%)
Substantially increased	62 (81.6%)
Degrees of fatty liver (N(%))	
Grade 1 (Mild/Intermediate)	55 (48.2%)
Grade 2 (Moderate)	40 (35.1%)
Grade 3 (Severe)	19 (16.7%)

Abbreviations: BMI: body mass index; VAI: Visceral adiposity index.

All values are shown as mean± standard deviation (SD) for continuous variable and categorical variables are numbers (the percentage) of participants

Table 2 Comparison of Demographic and Anthropometric Measurements, Dietary Intake and Clinical Parameters of Participants According to Degrees of Fatty Liver

a. Demographic and Anthropometric Parameters

Characteristics	Grade 1 (n=55)	Grade 2 (n=40)	Grade 3 (n=19)	*p*
Sex (row-%)				
Female	46 (59.6%)[a]	21 (26.9%)[b]	11 (13.5%)	0.018*
Male	9 (25.0%)[a]	19 (54.2%)[b]	8 (20.8%)	
Age (year)	45.8 ± 11.6	47.7 ±11.7	50.3 ±15.7	> 0.05
Height (cm)	161.2 ± 7.7	166.8 ± 12.1	163.9 ± 9.9	> 0.05
BMI (kg/m²)	32.0 ± 3.8	32.9 ± 7.0	35.6 ± 1.9	> 0.05
Body weight (kg)	83.4 ± 12.4	91.7 ± 22.8	95.9 ± 3.2	> 0.05
Body fat (kg)	35.3 ± 7.6	34.6 ± 14.5	41.6 ± 5.7	> 0.05
Body fat (%)	37.0 ± 10.4 [a]	43.0 ± 7.2 [b]	43.9 ± 6.5 [a b]	0.017*
Mass muscle (kg)	32.0 ± 8.7	26.5 ± 6.0	30.6 ± 6.8	> 0.05
Waist circumfe-	100.5 ± 9.4	106.9 ± 14.9	112.9 ± 6.0	> 0.05
Hip circumference	113.9 ± 10.4 [a]	114.2 ± 17.6 [b]	118.8 ± 6.9 [b]	> 0.05
Waist/Hip ratio	0.88 ± 0.06[a]	0.94 ± 0.07[b]	0.91 ± 0.07[b]	Grade 1 b/w 2 p: 0.006* Grade 1 b/w 3 p:0.009*
VAI	0.8 ± 0.3	0.9 ± 0.5	0.8 ± 0.5	> 0.05

b. Clinical Parameters

Parameters	Grade 1 (n=55)	Grade 2 (n=40)	Grade 3 (n=19)	*p*
AST (U/L)	19.9 ± 9.5[a]	27.8 ± 15.1[b]	38.4 ± 15.6[b]	Grade 1 b/w 2 p: 0.012* Grade 1 b/w 3 p:0.000*
ALT(U/L)	24.3 ± 21.4 [a]	39.7 ± 33.1 [b]	52.5 ± 35.4 [b]	Grade 1 b/w 2 p: 0.004* Grade 1 b/w 3 p:0.001*
ALP (U/L)	80 ± 19.9	78 ± 19.8	75.3 ± 11.8	> 0.05
GGT (U/L)	27.5 ± 36.7 [a]	33.6 ± 19.6 [b]	42.8 ± 23.7 [b]	Grade 1 b/w 2 p: 0.014* Grade 1 b/w 3 p:0.012*
LDL-C (mg/dL)	134.1 ± 45.9	124.1 ± 41.3	111.8 ± 38.9	> 0.05
HDL-C (mg/dL)	52.6 ± 12.4	47.9 ± 18.2	53 ± 33.8	> 0.05
TC (mg/dL)	214.6 ± 51.4	203.1 ± 50.1	196.2 ± 66.2	> 0.05
TG (mg/dL)	153.9 ± 73.4	156.6 ± 70.3	139.4 ± 84.2	> 0.05
FBG (mg/dL)	93.5 ± 12.8[a]	101.7 ± 19.5	109.9 ± 17.6[b]	0.013*
Insulin (mU/L)	13.7 ± 8.4 [a]	15.7 ± 8	20.1 ± 6.4 [b]	0.034*
Homa-IR	3.1 ± 1.7 [a]	4.2 ± 2.7	5.2 ± 1.8 [b]	0.047*
Hb A1c (IFCC) (mmol/mol)	38.8 ± 4.0	39.4 ± 7	43 ± 4.7	0.026*

c. Dietary Nutrient Intakes

Nutrients	Grade 1 (n=55)	Grade 2 (n=40)	Grade 3 (n=19)	*p*
Total Energy Intake (kcal)	2.018.9±602.7	2.122.9±662.8	1.956.8 ± 496.2	> 0.05
Carbohydrate Intake (%)	42.1±9.9	40.5±8.0	47.4±7.2	> 0.05
Carbohydrate Intake ()	205.6±68.7	209.6±71.4	226.3±65.6	> 0.05
Protein Intake (%)	16.0 ± 4.1	17.1 ± 4.2	15.5 ± 3.3	> 0.05
Protein Intake (g)	78.9 ± 29.3	88.0 ± 30.0	75.2 ± 28.3	> 0.05
Fat Intake (%)	41.6 ± 9.3	42.1 ± 9.4	37.0 ± 6.8	> 0.05
Fat Intake (g)	95.3 ± 41.8	101.0 ± 42.9	81.0 ± 23.3	> 0.05
Dietary fiber (g)	23.9 ± 8.0	23.0 ± 8.6	24.6 ± 9.4	> 0.05
Soluble dietary fiber (g)	7.2 ± 2.5	6.7 ± 2.3	7.5 ± 2.9	> 0.05
Insoluble dietary fiber(g)	16.1 ± 5.9	15.6 ± 6.6	16.2 ± 7.2	> 0.05
SFA (g)	37.9 ± 17.6	40.2 ± 17.6	31.9 ± 10.5	> 0.05
Dietary cholesterol (mg)	290.4 ± 160.8 [a]	487.6 ± 233.6 [b]	412.4 ± 213.5	0.026*
PUFA (g)	16.2 ± 8.3	15.6 ± 8.8	14.8 ± 5.3	> 0.05
EPA (g)	0.7 ± 0.3	0.7 ± 0.4	0.6 ± 0.2	> 0.05
DHA (g)	0.2 ± 0.1	0.2 ± 0.2	0.2± 0.1	> 0.05
Fructose (g)	12.4 ± 5.3	13.9 ± 10.0	10.8± 7.1	> 0.05
Alcohol (g)	0.9 ± 2.9	0.7 ± 3.0	0.03± 0.0	> 0.05
Sodium (mg)	4.017.7 ± 1.618.4	4.362.6 ± 1.662.0	4.750.5 ± 1.949.6	> 0.05
Potassium (mg)	2.804.3 ± 1.014.3	2.730.1 ± 749.4	2.404.8 ± 850.2	> 0.05
Calcium (mg)	838.0±322.5	841.2 ± 314.2	860.8 ± 365.2	> 0.05
Magnesium (mg)	319.4±103.4	321.6 ± 114.8	290.8 ± 94.8	> 0.05
Phosphorus (mg)	1.298.5±409.4	1.382.2±409.7	1.300.2 ± 340.5	> 0.05
Iron (mg)	11.6±3.3	12.3±3.8	10.0 ± 2.7	> 0.05
Zinc(mg)	12.1±4.4	13.4±4.9	10.3 ± 3.2	> 0.05
Vitamin A/Retinol (µg)	1.891.4 ± 1.577.5	1.478.7 ± 609.0	1.082.5 ± 518.2	> 0.05
Vitamin E (mg)	14.9 ± 8.0	13.4 ± 8.4	13.6 ± 7.8	> 0.05
Vitamin B1/ Thiamin (mg)	1.1 ± 0.4	1.1 ± 0.4	0.9 ± 0.2	> 0.05
Vitamin B2/ Riboflavin (mg)	1.6 ± 0.5	1.7 ± 0.4	1.3 ± 0.3	> 0.05
Vitamin B3/Niasin (mg)	17.1 ± 9.4	19.0 ± 11.6	16.0 ±10.0	> 0.05
Vitamin B6/ Pyridoxine (mg)	1.4 ± 0.5	1.5 ± 0.4	1.3 ± 0.5	> 0.05
Vitamin B9/Folate (µg)	358.6 ± 128.8	347.5 ± 118.2	329.7 ± 129.5	> 0.05
Vitamin C	113.6 ± 76.1	105.1 ± 72.4	109.9 ± 70.1	> 0.05
Vitamin B12/ Cobalamin (µg)	5.7 ± 4.1	5.4 ± 3.5	3.8 ± 1.7	> 0.05
Vitamin D (µg)	4.1 ± 3.1	3.6 ± 2.8	3.3 ± 2.3	> 0.05
Biotin (µg)	54.0 ± 23.1	54.0 ± 22.8	42.0 ± 16.5	> 0.05

Abbreviations: BMI: body mass index; VAI: Visceral adiposity index; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase; LDL-C: low-density lipoprotein-cholesterol; HDL-C: high-density lipoprotein cholesterol; TC: total cholesterol; TG: triglycerides; FBG: fasting blood glucose; HOMA-IR. homeostasis model assessment of insulin resistance; HbA1c: glycated hemoglobin; PUFA: polyunsaturated fatty acids; SFA: saturated fatty acids; EPA: eicosapentaenoic acid; DHA: docosahexaenoic acid.

All values are shown as mean ± standard deviation (SD) for continuous variables and categorical variables are numbers (the percentage) of participants. One-way ANOVA test and chi-square tests were separately used to compare differences in continuous variables and categorical variables. If not normally distributed after transformation, Kruskal Wallis-H Test was conducted.

Statistically significant differences ($p < 0.05$) were identified among values with different letters in the same row.

** $p < 0.05$; b/w: between.*

participants, a statistically significant difference was observed in waist circumference (WC) across steatosis grades. Specifically, WC was significantly lower in the Grade 1 group (101.2 ± 10.7 cm) compared to the Grade 3 group (114.7 ± 13.1 cm) (p = 0.003). visceral adiposity index score was significantly lower in the Grade 1 group (0.86 ± 0.05 cm) compared to the Grade 2 group (0.92 ± 0.06 cm) and to the Grade 3 group (0.93 ± 0.06 cm) (p = 0.044; p= 0.021). The significant differences in both WC and VAI suggest that central adiposity may contribute to hepatic fat accumulation in women.

Among male participants, significant differences were identified in aspartate aminotransferase (AST) and triglyceride (TG) levels between steatosis grades. Serum AST levels were higher in Grade 3 (40.0 ± 9.8 U/L) compared to Grade 1 (26.1 ± 9.7 U/L) (p = 0.033), and TG levels were elevated in Grade 3 (239.1 ± 130.3 mg/dL) compared to Grade 1 (149.7 ± 67.1 mg/dL) (p = 0.043), indicating increased hepatic injury and dyslipidemia in more advanced stages. In addition, the percentage of dietary carbohydrate intake differed significantly between Grade 2 (46.3 ± 6.6%) and Grade 3 (54.8 ± 3.6%) groups in men (p = 0.040), suggesting that higher dietary carbohydrate contribution may be associated with more severe steatosis in males.

Ordinal logistic regression was performed to determine independent predictors of hepatic steatosis severity, using a fully adjusted model that accounted for age, BMI, and total energy intake. Although multiple models were tested with varying levels of adjustment, only the fully adjusted model is presented in Table 3. Other models yielded similar trends but did not reach statistical significance.

According to the fully adjusted model, body weight (p = 0.022), muscle mass (p = 0.014), waist circumference (p = 0.003), AST (p < 0.001), ALT (p = 0.006), glucose (p = 0.005) and HOMA-IR (p = 0.018) were found to be significantly associated with steatosis grade. Among the dietary variables, none — including energy, carbohydrate, protein, fat, fiber, saturated fat, dietary cholesterol, or PUFA — showed a statistically significant association with (p > 0.05 for all). The results of odds ratios are shown in Table 3. We observe that fasting blood glucose (OR = 1.039, 95% CI: 1.011–1.066), HOMA-IR (OR = 1.328, 95% CI: 1.049–1.682), waist circumference (OR = 1.062, 95% CI: 1.020–1.104) and muscle mass (OR = 1.073, 95% CI: 1.015–1.135) are significantly related to the steatosis grade categories.

3.2. Discussion

This study aimed to evaluate the associations between disease severity and various dietary, anthropometric, and metabolic parameters in individuals diagnosed with Non-Alcoholic Fatty Liver Disease (NAFLD). Specifically, our objective was to investigate how these variables relate to the severity of hepatic steatosis.

Our findings indicate that while dietary composition—except for cholesterol—may not directly influence steatosis severity, anthropometric markers such as central adiposity and muscle mass, along with metabolic indicators like insulin resistance and liver enzymes, are significantly associated with disease progression.

3.2.1. Dietary Factors

Among the dietary variables assessed, no statistically significant differences were observed between steatosis grades for total energy intake, carbohydrate, protein, fat, dietary fiber, fructose, or polyunsaturated fatty acids (PUFA). These results suggest that these dietary components may not have had a measurable impact on the severity of liver fat accumulation in our study population.

Both the quantitative (e.g., total energy intake) and qualitative (e.g., type and source of macronutrients) aspects of the diet are believed to play critical roles in the development and progression of NAFLD (25). Excessive energy intake is known to increase hepatic triglyceride synthesis and accumulation (25, 26). Carbohydrate intake is closely linked to de novo lipogenesis (DNL), and diets high in carbohydrates, fructose, and low in protein have been shown to exacerbate hepatic lipid accumulation (27, 28).

Table 3. Ordinal Logistic Regression Results Predicting Hepatic Steatosis Grade (N = 114)

Variables	Value	Std. Error	Z	OR	95% CI (2.5%)	95% CI (97.5%)	*p*
Dietary nutrient intakes							
Carbohydrate (g)	0.0025	0.0032	0.77	1.003	0.996	1.009	> 0.05
Carbohydrate (%)	0.0229	0.0247	0.93	1.023	0.975	1.074	> 0.05
Dietary fiber (g)	-0.0004	0.0261	-0.01	1.0	0.95	1.052	> 0.05
Soluble dietary fiber (g)	-0.0126	0.0871	-0.14	0.987	0.833	1.171	> 0.05
Insoluble dietary fiber(g)	0.0041	0.0348	0.12	0.996	0.93	1.066	> 0.05
Fructose (g)	0.0022	0.0272	0.08	0.998	0.946	1.052	> 0.05
Demographic and anthropometric parameters							
Height (cm)	-0.0339	0.0212	-1.6	1.034	0.992	1.078	> 0.05
Age (year)	0.0192	0.0186	1.03	1.019	0.983	1.057	> 0.05
Body weight (kg)	0.0282	0.0123	2.28	1.029	1.004	1.054	0.022*
BMI (kg/m2)	0.0681	0.0404	1.69	1.07	0.989	1.159	> 0.05
Body fat (kg)	0.021	0.0192	1.09	1.021	0.984	1.06	> 0.05
Body fat (%)	0.0239	0.0239	1.0	0.976	0.932	1.023	> 0.05
Mass muscle (kg)	-0.0708	0.0287	-2.47	1.073	1.015	1.135	0.014*
Waist circumference (cm)	0.0598	0.0202	2.96	1.062	1.02	1.104	0.003*
Hip circumference (cm)	0.0129	0.016	0.81	1.013	0.982	1.045	> 0.05
Clinical parameters							
AST (U/L)	0.0654	0.0179	3.65	1.068	1.031	1.106	0.000*
ALT(U/L)	0.0234	0.0084	2.77	1.024	1.007	1.041	0.006*
ALP (U/L)	0.0082	0.0118	0.7	0.992	0.969	1.015	> 0.05
GGT (U/L)	0.0141	0.0107	1.32	1.014	0.993	1.036	> 0.05
LDL-C (mg/dL)	0.0082	0.0054	1.52	0.992	0.981	1.002	> 0.05
HDL-C (mg/dL)	0.0062	0.0137	0.45	0.994	0.967	1.021	> 0.05
TC (mg/dL)	0.0052	0.0046	1.13	0.995	0.986	1.004	> 0.05
TG (mg/dL)	0.0012	0.0032	0.36	0.999	0.993	1.005	> 0.05
FBG (mg/dL)	0.0378	0.0135	2.79	1.039	1.011	1.066	0.005*
Insulin (mU/L)	0.0594	0.0317	1.87	1.061	0.997	1.129	> 0.05
Homa-IR	0.284	0.1204	2.36	1.328	1.049	1.682	0.018*
Hb A1c (IFCC) (mmol/ mol)	0.061	0.0477	1.28	1.063	0.968	1.167	> 0.05

Abbreviations: BMI: body mass index; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase; LDL-C: low-density lipoprotein-cholesterol; HDL-C: high-density lipoprotein cholesterol; TC: total cholesterol; TG: triglycerides; FBG: fasting blood glucose; HOMA-IR. homeostasis model assessment of insulin resistance; HbA1c: glycated hemoglobin;

Ordinal logistic regression analysis was conducted using hepatic steatosis grade (Grade 1–3) as the dependent variable. The model was fully adjusted for age, body mass index (BMI), and total energy intake. The cumulative odds ordinal logistic regression model assumes proportional odds across outcome categories. The table includes only those predictors for which valid and interpretable regression models could be fitted.

** (p < 0.05). OR: Odds Ratio; CI: Confidence Interval*

Additionally, low fiber intake and deficiencies in certain micronutrients, such as vitamin D, niacin, and copper, have also been associated with NAFLD progression (29-32).

The effects of dietary fats on NAFLD vary based on the type of fatty acids consumed. Excess saturated fatty acid (SFA) intake has been shown to enhance adipose tissue lipolysis, thereby increasing the influx of free fatty acids to the liver (34). In contrast, PUFAs—particularly n-3 PUFAs—are known for their anti-inflammatory and anti-steatogenic properties (33, 34). Xie et al. (2021) reported a non-linear association between PUFA intake and NAFLD risk, where moderate intake (18.8–29.3 g/d) appeared protective, while very high or very low levels were not associated with increased risk (35).

Our comparison across steatosis grades (Grades 1–3) within NAFLD patients revealed the highest cholesterol intake in the moderate steatosis group . Excessive dietary cholesterol has been identified as a key contributor to hepatic lipid accumulation and liver injury (36). In a recent animal study, Gao et al. (2023) demonstrated that high cholesterol intake accelerates the progression from NAFLD to non-alcoholic steatohepatitis (NASH) and induces marked hepatic inflammation (37). This paradoxical finding highlights an unexpected pattern that warrants further investigation.

The fact that energy intake was found to be similar between the groups in our study may indicate that general energy consumption showed a similar distribution among our participants and that energy intake alone is not a determinant among different severities of NAFLD. Another potential explanation as to why we did not observe any significant main effects of dietary factors except dietary cholesterol intake may be related to the considerable underreporting of daily dietary intake among our study participants.

Although this study used a stringent dietary assessment protocol, including three 24-hour recalls, underreporting may have influenced the observed associations with hepatic steatosis. Underreporting can be of particular concern with self-reported data, especially among individuals with obesity (38). In our study, 97% of NAFLD patients were in the obese/ overweight category.

3.2.2. Anthropometric Measurement

In the present study, several anthropometric parameters, including waist circumference (WC), waist-to-hip ratio (WHR), body mass index (BMI), muscle mass, body weight, and body fat percentage, were significantly associated with the severity of non-alcoholic fatty liver disease (NAFLD). Visceral adipose tissue is known to have high lipolytic activity, releasing free fatty acids directly into the portal circulation and thereby promoting hepatic triglyceride accumulation and insulin resistance (13). WC, widely recognized as a proxy for visceral adiposity, has been consistently linked to NAFLD onset and progression (39). For instance, a population -based study among Korean adults reported that higher WC quartiles were significantly associated with increased prevalence of NAFLD, insulin resistance, and elevated alanine aminotransferase (ALT) levels (40). Longitudinal evidence further supports that increases in WC raise NAFLD incidence, whereas reductions in WC are protective (41). Notably, even independent of abdominal obesity classification, an upward trend in WC has been shown to be positively associated with NAFLD risk (39).

Although BMI did not differ significantly across steatosis grades in our study, this may be explained by the high prevalence of overweight or obesity in our sample. Nonetheless, the relationship between BMI and NAFLD is well-documented in the literature, with higher BMI categories linked to markedly increased NAFLD risk—up to 5-9-fold among obese individuals compared to those of normal weight (42). In addition, BMI appears to mediate the relationship between pro-inflammatory dietary patterns and NAFLD, suggesting that dietary modulation may affect liver fat accumulation via its impact on body weight (43). Weight gain over time has also been shown to significantly contribute to the development

of NAFLD, underscoring the importance of long-term weight management strategies (44).

It is important to note, however, that BMI may not adequately reflect visceral fat accumulation. Alternative anthropometric markers such as WC and WHR have been proposed as better predictors of NAFLD risk in this context (14, 45). Indeed, Mendelian randomization studies support a potential causal relationship between increased WHR and NAFLD development, highlighting the importance of fat distribution rather than overall adiposity (45, 46).

Another emerging area of interest is the role of skeletal muscle mass in NAFLD. Lower muscle mass has been associated not only with NAFLD but also with liver fibrosis. As skeletal muscle is the primary site for insulin-mediated glucose uptake, greater muscle mass contributes to enhanced insulin sensitivity and reduced hepatic lipid accumulation (47). Consistent with these findings, our results showed that individuals with higher muscle mass tended to have lower steatosis severity.

In addition, we observed that body fat percentage varied significantly across steatosis grades, with participants in Grade 1 exhibiting significantly lower body fat compared to Grade 2, and lower—though not significantly—than Grade 3. These findings suggest that body fat accumulation—and perhaps more importantly, its distribution—plays a pivotal role in the early pathogenesis and progression of hepatic steatosis. A growing body of research supports the central role of visceral adiposity in NAFLD development (48, 49). Visceral fat depots not only release free fatty acids but also secrete a host of proinflammatory cytokines (e.g., TNF-α, IL-6) and adipokines (e.g., leptin, resistin), which aggravate hepatic insulin resistance and promote inflammatory cascades that drive disease progression (48). Even modest increases in body fat percentage are likely to contribute to severity of NAFLD, particularly when adiposity is concentrated in visceral compartments.

3.2.3. Biochemical parameters

In our study, serum levels of AST and ALT were significantly associated with severity of NAFLD, whereas GGT and ALP did not show a significant relationship. Hepatic transaminases—particularly ALT—are well-established biomarkers of liver injury, with ALT generally considered to be more liver-specific than AST in the context of NAFLD (50). Multiple studies have consistently reported elevated levels of liver enzymes, especially ALT and AST, in patients with NAFLD when compared to control groups (51-53). Similar to our study, in studies comparing liver enzymes according to liver steatosis levels with ultrasound, a significant relationship was found between AST and ALT levels and the severity of liver steatosis (52, 54).

We observed a significant positive association between both HOMA-IR and fasting serum glucose (FSG) levels and the severity of NAFLD. Insulin resistance plays a central role in the pathogenesis of NAFLD by stimulating lipolysis in adipose tissue, thereby increasing the release of free fatty acids (FFAs) into the circulation (55). Studies have shown that NAFLD patients often exhibit elevated HOMA-IR values, indicating increased insulin resistance (IR) (56-58). According to the results of a recent meta-analysis study NAFLD patients showed markedly higher HOMA-IR and FSG levels compared to healthy controls (59). These results support the hypothesis that insulin resistance is a key contributor to hepatic steatosis.

In our study, we did not observe statistically significant associations between any serum lipid parameters and the severity of hepatic steatosis. The relationship between serum lipid parameters and non-alcoholic fatty liver disease (NAFLD) remains inconclusive in the literature . While several studies have reported that dyslipidemia—characterized by elevated triglyceride (TG) and low-density lipoprotein cholesterol (LDL-C) levels and reduced high-density lipoprotein cholesterol (HDL-C) is associated with NAFLD (60, 61). Shousha et al. (2020), acknowledged the potential utility of certain biochemical markers for identifying early-stage NAFLD (53). However, some studies have challenged this association, especially

when stratifying patients by steatosis grade. For instance, Huang et al. (2023) found that conventional lipid markers such as TG, LDL-C, HDL-C did not significantly differentiate between severity of NAFLD (62). Cuenza et al. (2017) reported a significant association between TG levels and steatosis grade but found no relationship with total cholesterol (TC), HDL-C, or LDL-C (63). Conversely, Mahaling et al. (2013) observed that increasing severity of NAFLD correlated with higher levels of TC and LDL-C and lower HDL-C concentrations, yet no significant association was found with TG levels (64). This absence of a significant association with cholesterol likely reflects the complex regulation of hepatic lipids, in which insulin resistance—by disrupting pathways of lipid uptake, synthesis, degradation, and secretion—serves as the key driver of NAFLD (65).

3.2.4. Sex Differences

NAFLD exhibits marked sexual dimorphism, with significant differences in prevalence and severity between men and women. The disease is generally more prevalent among men (66, 67). These disparities are attributed to a combination of biological factors, such as chromosomal structure and sex hormone levels, as well as sociocultural influences that shape lifestyle behaviors. Sex hormones play a central role in the development and progression of NAFLD. Estrogen is believed to exert a protective effect, whereas androgens are associated with increased hepatic lipid accumulation and liver injury (66, 68). In our study, sex distribution significantly differed across steatosis grades, with a higher proportion of females in the mild steatosis group (Grade 1) and more males in the moderate group (Grade 2). This finding corroborates prior evidence suggesting that men may be more prone to developing moderate-to-severe forms of NAFLD, while women tend to present with milder stages.

Moreover, we observed that the percentage of dietary carbohydrate intake was significantly higher in Grade 3 males compared to Grade 2. In contrast, no significant associations were found between dietary variables and steatosis severity in the female subgroup. As previously discussed, macronutrient composition may influence NAFLD severity, particularly through its role in hepatic fat accumulation and metabolic regulation. This may reflect previously reported trends of higher dietary awareness and health consciousness among women (69).

In our sex-stratified analysis, several key differences emerged in the associations between steatosis severity and anthropometric, biochemical, and dietary variables. In the female subgroup, WC and VAI were significantly higher in participants with Grade 3 steatosis, suggesting that central and visceral adiposity may play a more prominent role in disease progression among women. In contrast, no significant differences in anthropometric parameters were observed across steatosis grades in the male subgroup. These findings indicate a possible sex-specific relationship between fat distribution and NAFLD severity. Supporting this, a previous study reported that female NAFLD patients had significantly higher levels of visceral adiposity—reflected by body fat percentage, WC, and WHR—compared to healthy controls (70).

In the male subgroup, AST and TG levels were significantly higher in participants with Grade 3 steatosis. A recent cohort study similarly demonstrated that elevated TG and LDL levels were significantly associated with advanced liver fibrosis in male patients, further highlighting sex-specific differences in lipid metabolism and liver disease severity (71). These sex-specific patterns underscore the importance of integrating gender-sensitive strategies in NAFLD risk assessment and clinical management.

3.2.5. Strengths and Limitations

This study presents several strengths. First, it is among the relatively few studies to assess the associations between hepatic steatosis severity and a broad range of dietary, anthropometric, and metabolic parameters specifically within a clinically diagnosed NAFLD population. The use of three 24-hour dietary recalls enhanced the reliability of dietary

intake assessments, and stratification by steatosis grade allowed for detailed comparisons across disease severity levels. Furthermore, sex-stratified analyses provided valuable insights into potential sex -based differences in clinical and metabolic profiles, contributing to a more nuanced understanding of NAFLD pathophysiology.

Anthropometric measures beyond BMI—such as waist circumference, visceral adiposity index, and body composition—were considered in depth, offering a comprehensive view of fat distribution and its relevance to disease progression. Additionally, the use of ordinal logistic regression allowed for the identification of independent predictors while adjusting for key confounders.

However, certain limitations should be acknowledged. The cross-sectional design prevents any causal inference between the studied variables and NAFLD severity. The absence of imaging-based quantification methods such as magnetic resonance imaging (MRI) or magnetic resonance spectroscopy (MRS) may limit the precision in grading hepatic steatosis. Moreover, the reliance on self-reported dietary intake introduces the possibility of underreporting, particularly among overweight and obese individuals. Lastly, although the sex-stratified subgroup analyses revealed interesting trends, the relatively small sample size within each sex and steatosis grade subgroup may limit the generalizability of these findings and reduce statistical power.

Future studies should employ longitudinal designs with objective imaging modalities (e.g., MRI/MRS) and biochemical or biomarker-based dietary assessments, incorporate larger and more diverse sex-stratified cohorts, and explore mechanistic pathways—such as genetic factors (e.g., PNPLA3 variants) and gut-liver axis interactions—to better elucidate causal relationships and interindividual variability in NAFLD progression.

4. CONCLUSION

This study investigated the associations between steatosis severity and a comprehensive set of dietary, anthropometric, and metabolic factors in adults with clinically diagnosed NAFLD. Our findings indicate that metabolic and anthropometric indicators—particularly waist circumference, muscle mass, insulin resistance, and liver enzyme levels—are more strongly associated with disease severity than general dietary intake.

Although most dietary components did not differ significantly across steatosis grades, higher dietary cholesterol intake in the moderate steatosis group suggests that cholesterol may play a role in disease progression. Furthermore, sex-specific subgroup analyses revealed distinct patterns in the associations between steatosis severity and clinical parameters. Central and visceral adiposity emerged as more relevant in women, whereas men exhibited greater biochemical alterations and dietary carbohydrate intake differences in advanced steatosis stages.

Taken together, these results underscore the importance of incorporating metabolic markers, body composition, and sex-specific factors into routine NAFLD evaluation to enhance early risk detection and inform individualized management strategies.

Acknowledgements: The authors thank the participants for their collaboration and [Göztepe Prof. Dr. Süleyman Yalçın City Hospital] for support during data collection.

Financial Support: This research received no grant from any funding agency/sector.

Conflicts of Interest: The authors declared that there is no conflict of interest.

Ethical Statement: This study was approved by the Ethics Committee of the Marmara University Faculty of Medicine (approval date: October 8, 2021; No: 09.2021.1109).

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