Nikparast *et al. Nutrition Journal* (2025) 24:75
https://doi.org/10.1186/s12937-025-01142-4

Nutrition Journal

RESEARCH Open Access

The association between total, animal, and plant protein intake and metabolic dysfunction-associated fatty liver disease in overweight and obese children and adolescents

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Abstract

Background Dietary protein plays a crucial role in the growth and development of children and adolescents. However, recent evidence has shown inconsistent findings regarding the impact of dietary protein sources on health outcomes. This study aimed to investigate the association between total, animal, and plant protein intake and the odds of metabolic dysfunction-associated fatty liver disease (MAFLD) in overweight and obese children and adolescents.

Methods This cross-sectional study included 505 participants (52.9% males) aged 6–18 years, with a body mass index (BMI)-for-age z-score ≥ 1 based on WHO standards. MAFLD diagnosis followed established consensus definitions. Dietary intake of total, animal, and plant protein was assessed using a validated 147-item food frequency questionnaire. Adjusted logistic regression models were used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for MAFLD across protein intake and subtype quartiles.

Results The participants had a mean age of 10.0 ± 2.3 years and a mean BMI-for-age z-score of 2.70 ± 1.01. Higher animal protein intake was significantly associated with an increased likelihood of MAFLD (highest vs. lowest quartile OR: 2.31; 95% CI: 1.01–5.30). Conversely, higher plant protein intake was significantly associated with reduced odds of MAFLD (highest vs. lowest quartile OR:0.48;95% CI:0.23–0.96). No significant relationship was found between total protein intake and MAFLD odds.

Conclusions Our findings highlight the significance of dietary protein source in the odds of MAFLD among overweight and obese children and adolescents. Further studies are warranted to confirm these findings and explore the underlying mechanisms.

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Keywords Dietary protein, Children, Adolescent, Obesity, MAFLD, Metabolic dysfunction associated fatty liver disease

Introduction

The global pediatric obesity epidemic has escalated to alarming levels, posing significant long-term health risks [1]. As of 2023, approximately one in five children worldwide is overweight or obese, increasing their susceptibility to various metabolic disorders, including cardiovascular disease, diabetes, and non-communicable liver diseases [2, 3]. Among the health challenges associated with excessive weight, nonalcoholic fatty liver disease (NAFLD) has emerged as a rapidly escalating issue in children and adolescents, with the highest prevalence observed in those classified as overweight or obese [4]. Recent epidemiological studies estimate that 3–12% of the general pediatric population and up to 40–50% of overweight or obese children have NAFLD [5, 6].

A recent international expert consensus recommended replacing NAFLD with metabolic dysfunction-associated fatty liver disease (MAFLD) [7, 8]. MAFLD is distinguished from NAFLD by the presence of hepatic steatosis in conjunction with metabolic dysfunction, which typically manifests as overweight or obesity and insulin resistance (IR) [7]. This innovative approach is designed to advance the understanding and management of fatty liver disease in light of the escalating obesity epidemic [8]. MAFLD has increasingly emerged as a significant contributor to metabolic syndrome and its related components, including obesity, type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, as well as liver-related diseases such as hepatocellular carcinoma and cirrhosis [9, 10]. While the precise causes of MAFLD remain incompletely understood, it is apparent that hyperinsulinemia and IR are significant contributors to the development of hepatic steatosis and the associated metabolic disturbances [11]. MAFLD presently lacks approved pharmacological treatments, emphasizing the importance of adopting lifestyle modifications as the primary strategy for managing this condition effectively [7, 8]. In this context, implementing healthy lifestyle factors, encompassing well-balanced dietary patterns and consistent physical activity, is paramount in the effective management of MAFLD. A comprehensive understanding of these influences can lead to significant improvements in the prevention and progression of MAFLD and associated metabolic disorders, making it essential to prioritize this research in our efforts to combat these increasingly prevalent conditions [12].

Dietary protein is a crucial macronutrient for growth, metabolic regulation, and muscle maintenance in children and adolescents [13]. However, recent evidence increasingly suggests that the source of protein intake—whether from animal or plant origins—may exert a distinct role in the development of obesity risk and metabolic health in children and adolescents [14, 15]. It seems that there was probable evidence for the association between higher animal protein intake and an increased risk of obesity among children and adolescents up to the age of 18 years, while the consumption of plant protein does not appear to have a significant impact on the risk of developing obesity [14, 15]. In this specific context, there is a limited number of studies that have examined the role of dietary protein intake and its sources on the risk of developing obesity-related disorders among children and adolescents [16, 17]. In 2018, a prospective study on Canadian children and adolescents revealed that an increased dietary protein intake was associated with a non-significant 27% reduction in the risk of developing an unhealthy obesity phenotype [17]. Furthermore, a recent cross-sectional study has indicated that obese adolescents who consumed higher amounts of total, animal, and plant-based proteins exhibited a significantly reduced risk of developing an unhealthy obese phenotype [16]. Current literature indicates a significant gap in research concerning the association between dietary protein intake and the risk of obesity-related disorders, particularly in the context of MAFLD. To address this gap, this study aimed to evaluate the relationship between total, animal, and plant protein intake and the odds of MAFLD among overweight and obese Iranian children and adolescents.

Method

This cross-sectional study was conducted between September 2023 and July 2024 as part of an obesity registry program for Iranian children and adolescents [18]. The sample size for this study was determined based on a prior investigation that assessed the relationship between dietary protein intake and metabolic health status among Iranian adolescents [16]. Using G*Power software, the sample size was calculated with the following parameters: alpha error probability of 0.05, power of 0.80, a mean dietary protein intake of 14.3 ± 2.0 g/day, and an effect size of 1.2 [16], resulting in a required sample size of 505 participants. The participants were randomly selected from individuals referred to the Gastroenterology, Hepatology, and Endocrinology outpatient clinics at Children's Hospital Medical Center in Tehran. Participants were included based on the following criteria: individuals aged 7 to 18 years classified as overweight or obese, defined by a body mass index-for-age (BMI-for-age) Z-score of 1 or greater, in accordance with World Health Organization guidelines [19]. Participants were excluded if they met the following criteria: (1) medical conditions such

as renal or other liver diseases (e.g., Wilson's disease, autoimmune liver disease, hemochromatosis, and viral infections), thyroid disorders, or malignancies; (2) use of hepatotoxic or steatogenic medications (e.g., valproate, amiodarone), weight-loss drugs, appetite suppressants; (3) dietary modifications in the past year due to illness or weight-loss interventions; and (4) incomplete responses to fewer than 35 items on the food frequency questionnaire (FFQ) or under- or over-reporting of dietary intake. Under- and over-reporting were identified by comparing reported energy intake with estimated energy requirements, following Institute of Medicine guidelines, and excluding deviations beyond ± 2 standard deviations [20]. The study adhered to the principles of the Helsinki Declaration and received ethical approval from the National Nutrition and Food Technology Research Institute's ethics committee (IR.SBMU.NNFTRI.REC.1402.015). Informed written consent was obtained from parents or legal guardians, and children also provided their assent.

Measurements

Qualified pediatric nutritionists conducted anthropometric assessments using standardized protocols. Body weight was measured using a calibrated Seca scale (Seca, Hamburg, Germany) with 100-gram precision, while height was recorded to the nearest 0.5 cm using a measuring tape. BMI was calculated as weight (kg) divided by height squared (m^2), with BMI-for-age Z-scores determined using internationally accepted growth charts [19]. Waist circumference (WC) was measured using a non-elastic tape, positioned midway between the iliac crest and the lowest rib, accurate to 0.5 cm. Pubertal status was evaluated by a pediatric endocrinologist using the Marshall and Tanner criteria, categorizing participants into prepubertal and pubertal groups based on breast and genital development stages [21, 22]. Physical activity was assessed via the Persian-translated Modifiable Activity Questionnaire (MAQ), which calculates metabolic equivalent task (MET) hours per week and has demonstrated high reliability (97%) and moderate validity (49%) in adolescents [23]. Blood pressure was measured manually on the right arm after a 15-minute rest using a mercury sphygmomanometer with an appropriately sized cuff. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were determined using the Korotkoff sound technique, with average values calculated from two measurements taken one minute apart.

Blood samples were collected between 7:00 and 9:00 AM subsequent to an overnight fasting period of 12 to 14 h. Samples were centrifuged within 30–45 min and analyzed on the same day. Fasting blood sugar (FBS) and triglycerides (TG) were measured using enzymatic colorimetric methods. Total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C) were assessed via cholesterol esterase and phosphotungstic acid methods, respectively, while low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation [24]. Liver function markers, including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), were quantified using enzymatic photometry, and gamma-glutamyl transferase (GGT) was measured via enzymatic colorimetric methods. These assessments were performed with commercial kits procured from Delta Darman Inc. (Tehran, Iran) and processed using a Selectra 2 auto-analyzer (Vital Scientific, Spankeren, The Netherlands). Fasting serum insulin levels were quantified via the electrochemiluminescence immunoassay (ECLIA) technique, employing Roche Diagnostics kits alongside the Roche/Hitachi Cobas e-411 analyzer (Roche Diagnostics, GmbH, Mannheim, Germany). Insulin resistance was estimated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated with the formula: HOMA-IR = [fasting glucose (mmol/L) × fasting insulin (μU/L)] / 22.5. All biochemical analyses exhibited intra- and inter-assay coefficients of variation below 5.3%.

Dietary intake assessment

Dietary data were collected using a validated 147-item semi-quantitative FFQ [25, 26]. Trained nutritionists interviewed participants or their parents/guardians to determine food consumption frequency and portion sizes over the past year. If participants had difficulty responding, their mothers were consulted. Portion sizes were primarily based on U.S. Department of Agriculture (USDA) serving sizes (e.g., one slice of bread, one medium apple, or one cup of dairy). When USDA data were unavailable, household measures were used (e.g., one tablespoon of beans, one chicken leg, or varying rice portion sizes) and converted into grams and servings. Nutrient composition was primarily derived from the USDA Food Composition Tables (FCT) due to limitations in the Iranian FCT, which lacks comprehensive data on raw food nutrient profiles. However, the Iranian FCT was referenced for traditional food items absent in the USDA FCT, such as Kashk.

Total dietary protein intake (g/day) was calculated as the sum of plant and animal protein intake. Plant protein intake was determined by aggregating protein content from plant-based foods (e.g., vegetables, grains, nuts, legumes), while animal protein intake was derived from protein contributions from animal-based foods (e.g., meats, dairy, eggs, fish).

Assessment of MAFLD

MAFLD was defined based on the presence of hepatic steatosis, assessed following an 8 to 12-hour fasting period using high-resolution B-mode ultrasonography performed by a trained radiologist. The examination was

conducted using a Samsung Medison SonoAce R3 ultrasound machine with a 7.5–10 MHz linear transducer. In accordance with the international expert consensus statement, participants met the MAFLD criteria if they had hepatic steatosis and a body BMI-for-age Z-score ≥ 1, as classified by WHO growth standards [8].

Statistical analysis

Descriptive statistics were used to summarize participants' demographic and clinical data, stratified by MAFLD status. Data normality was assessed using histogram plots and the Kolmogorov–Smirnov test. Continuous variables were presented as mean ± SD or median (interquartile range), while categorical variables were expressed as percentages. Comparisons were conducted using independent t-tests, Mann–Whitney U tests, or Chi-square tests as appropriate. Logistic regression models were applied to evaluate associations between dietary total, animal and plant protein intake and the odds of MAFLD, adjusting for confounders in three models: (1) unadjusted, (2) adjusted for age and sex, and (3) further adjusted for BMI for-age Z-score, puberty status, TG, HOMA-IR, PA, dietary energy intake, total dietary fiber (gram/1000 kcal), saturated fatty acid intake (% of energy), and total protein intake (% of energy); when the animal or plant-based protein intake considered as an exposure variable)). We conducted a separate analysis of the characteristics and dietary intakes of study participants based on age groups: children (ages 6–11 years) and adolescents (ages 12–18 years), to ensure a thorough understanding of developmental differences in metabolic profiles and dietary requirements. All statistical analyses were conducted utilizing SPSS software (version 26; SPSS Inc., Chicago, IL). A p-value of less than 0.05 was established as the threshold for statistical significance.

Results

Of the 548 overweight and obese children and adolescents initially enrolled, 31 participants were excluded due to incomplete anthropometric, dietary, biochemical, or ultrasound data, and an additional 12 were removed for implausible dietary reporting (over- or under-reporting). Consequently, the final analytical sample comprised 505 participants.

Table 1 presents the general characteristics of the study population, categorized into healthy individuals and those with MAFLD. The overall prevalence of MAFLD was 38.8%. The sample was predominantly male (52.9%), with 23.4% classified as prepubescent. The mean ± standard deviation (SD) for age and BMI-for-age Z-score were 10.0 ± 2.3 years and 2.87 ± 0.98, respectively. Participants with MAFLD exhibited significantly higher values for age, anthropometric indices, insulin, HOMA-IR, TG, and liver enzymes alongside lower HDL levels than healthy individuals (P-values < 0.05). However, no significant differences were observed between groups regarding gender, pubertal status, PA, passive smoker, FBS, Chol, LDL, SBP, and DBP (P-values > 0.05). In terms of macronutrient intake, no significant differences were noted between groups in total energy, carbohydrate, fat, or total protein intake. However, individuals with MAFLD had significantly higher animal protein intake and lower plant protein intake compared to their healthy counterparts (p-values < 0.05).

Table 2 presents participants' demographic characteristics stratified by quartiles of total, animal-based, and plant-based protein intake. Higher total protein intake was associated with a significant increase in the proportion of males, as well as greater height, PA levels, and FBS ($p < 0.05$). Increasing quartiles of animal protein intake were significantly associated with a higher proportion of males and greater exposure to passive smoking ($p < 0.05$). Conversely, higher quartiles of plant protein intake were linked to an increased proportion of males but a significant decrease in HDL-C levels (p-values < 0.05).

Table 3 outlines participants' macro- and micronutrient intakes across quartiles of total, animal, and plant protein consumption. Increasing total protein intake was significantly associated with higher intake of plant protein, animal protein, sodium, calcium, zinc, magnesium, and iron, while total fat, polyunsaturated fatty acids, carbohydrates, and dietary fiber decreased (p-values < 0.05). Regarding animal protein intake, higher quartiles were linked to increased consumption of total protein, total fat, saturated fatty acids, calcium, and zinc, with concomitant reductions in plant protein, polyunsaturated fatty acids, carbohydrates, iron, and dietary fiber (p-values < 0.05). Conversely, increasing plant protein intake was associated with higher consumption of total protein, carbohydrates, sodium, zinc, magnesium, iron, and dietary fiber, alongside lower intake of animal protein, total fat, saturated fatty acids, monounsaturated fatty acids, and calcium (p-values < 0.05).

Supplementary Table S1 represents the general characteristics of children according to MAFLD status. Children with MAFLD had significantly higher weight, height, BMI, BMI-for-age z-score, WC, ALT, and GGT, and lower HDL-C compared to non-MAFLD children (p-values < 0.05). Animal protein intake was significantly higher in children with MAFLD, while plant protein and total protein intake did not differ significantly (Supplementary Table S1).

Supplementary Tables S2 and S3 demonstrate the general characteristics and dietary intakes of children across quartiles of dietary total, animal, and plant-based protein intake. Across quartiles of dietary protein intake, children in the highest quartile of animal protein intake had significantly lower BMI, fasting insulin, and HOMA-IR

Table 1 Characteristics of the study participants according to metabolic dysfunction associated fatty liver disease status

	Total sample (*N* = 505)	Without MAFLD (*N* = 309)	MAFLD (*N* = 196)	*P*-value
Demographic data				
Age (years)	10.0 ± 2.3	9.7 ± 2.1	10.5 ± 2.5	< 0.01
Gender (Males, %)	52.9	52.1	54.1	0.66
Puberty (Prepubertal, %)	23.4	23.2	23.5	0.96
Weight (Kg)	49.6 ± 15.0	46.2 ± 12.3	55.1 ± 17.2	< 0.01
Height (cm)	142.2 ± 12.3	140.0 ± 11.4	145.5 ± 12.9	< 0.01
Body mass index (Kg/M^2)	24.0 ± 3.8	23.1 ± 3.1	24.4 ± 4.4	< 0.01
BMI for age z-score	2.87 ± 0.98	2.72 ± 0.80	2.99 ± 0.72	0.02
Waist circumference (cm)	83.5 ± 10.6	80.9 ± 9.4	87.6 ± 10.9	< 0.01
Passive smoker (%)	23.8	30.4	23.9	0.13
Physical activity (MET/hour/week)	8.6 (3.0-20.4)	8.9 (2.6–20.4)	7.5 (3.7–20.4)	0.89
Systolic blood pressure (mmHg)	105.0 (97.5–116.0)	105.0 (95.0-115.0)	105.0 (100.0-120.0)	0.26
Diastolic blood pressure (mmHg)	65.0 (60.0–75.0)	65.0 (60.0–75.0)	65.0 (60.0–70.0)	0.84
Biochemical data				
Fasting serum insulin (mU/mL)	16.4 ± 8.6	15.5 ± 7.9	17.8 ± 9.6	0.01
Fasting blood sugar (mg/dl)	91.1 ± 8.7	90.6 ± 8.9	91.9 ± 8.5	0.11
HOMA-IR	3.73 ± 2.16	3.5 ± 1.9	4.1 ± 2.3	0.01
Triglyceride (mg/dl)	109.5 (81.0-151.5)	110.0 (83.0-152.0)	168.0 (91.0-122.0)	0.01
Total cholesterol (mg/dl)	171.0 ± 55.9	172.3 ± 66.8	169.2 ± 31.8	0.54
HDL (mg/dl)	47.1 ± 11.7	49.0 ± 11.6	44.0 ± 11.3	< 0.01
LDL-C (mg/dl)	98.0 ± 25.5	97.3 ± 24.4	99.1 ± 27.2	0.43
Alanine aminotransferase (U/L)	16.0 (11.0–22.0)	15.0 (11.0–19.0)	18.0 (13.0–32.0)	0.01
Aspartate amino transferase (U/L)	23.0 (17.0–29.0)	22.0 (16.0–28.0)	25.0 (17.0–32.0)	0.01
Gamma-glutamyl transferase (U/L)	17.0 (15.0–21.0)	16.9 (14.0–19.0)	19.0 (16.0–24.0)	0.01
Dietary intake				
Energy (Kcal/day)	3046.3 ± 956.3	3069.9 ± 998.5	3009.1 ± 887.0	0.48
Carbohydrate (% of energy)	56.0 ± 6.2	56.1 ± 5.8	56.0 ± 3.7	0.89
Fat (% of energy)	33.1 ± 5.8	31.0 ± 5.5	31.1 ± 3.6	0.76
Total protein (% of energy)	13.4 ± 2.2	13.3 ± 2.1	13.6 ± 2.3	0.10
Animal protein (% of energy)	7.0 ± 2.5	6.7 ± 2.4	7.5 ± 2.6	0.01
Plant protein (% of energy)	6.4 ± 1.4	6.5 ± 1.5	6.2 ± 1.3	0.04

Significant p-values are highlighted in bold

Abbreviations: HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; HDL, high-density lipoprotein; LDL, Low-density lipoprotein

compared to the lowest quartile (p-values < 0.05). Higher quartiles of plant protein intake were associated with increased fiber, magnesium, and iron intake (p-values < 0.05).

Supplementary Table S4 shows the general characteristics of adolescents according to MAFLD status. Adolescents with MAFLD had significantly higher age, weight, BMI, BMI-for-age z-score, WC, TG, ALT, AST, GGT, and lower HDL-C compared to those without MAFLD (p-values < 0.05). Animal protein intake was significantly higher in the MAFLD group, while plant protein intake did not differ.

Supplementary Tables S5 and S6 illustrate the general characteristics and dietary intakes of children across quartiles of dietary total, animal, and plant-based protein intake. Higher quartiles of animal protein intake were associated with increased WC, and greater intake SFA, and calcium (p-values < 0.05). Plant protein intake was associated with significantly greater intake of fiber, iron, and magnesium, and lower intake of SFA, and calcium (p-values < 0.05).

Table 4 illustrates the association between dietary total, animal-based, and plant-based protein intake and the odds of MAFLD across quartiles. Total protein intake was not significantly associated with MAFLD in crude or adjusted models. However, animal protein intake demonstrated a positive association with MAFLD odds. In the crude model, participants in the highest quartile had significantly increased odds of MAFLD (OR = 1.88, 95% CI: 1.13–3.14) relative to those in the lowest quartile. This association remained significant after adjusting for age and sex (OR = 1.91, 95% CI: 1.13–3.22; *p*-trend = 0.01) and persisted in the fully adjusted Model (OR = 2.31, 95% CI: 1.01–5.30; *p*-trend = 0.05).

Conversely, plant protein intake exhibited an inverse association with MAFLD odds. While no significant relationship was observed in the crude model or age- and sex-adjusted model, the fully adjusted model revealed a

Table 2 General characteristics of study participants across quartile of dietary total, animal, and plant-based protein intake

	Total protein intake		Animal-based protein intake		Plant-based protein intake	
	Q1	Q4	Q1	Q4	Q1	Q4
Demographic data						
Age (years)	9.7 ± 2.1	9.7 ± 2.4	9.9 ± 1.9	9.9 ± 2.3	9.6 ± 2.2	10.1 ± 2.1
Gender (Males, %)	39.2	55.9*	38.2	55.0*	61.1	56.9*
Puberty (Prepubertal, %)	26.7	23.5	29.3	23.3	27.8	26.8
Weight (Kg)	48.8 ± 14.4	50.1 ± 16.6	49.8 ± 12.1	49.8 ± 15.3	48.9 ± 14.8	50.9 ± 15.9
Height (cm)	140.4 ± 11.5	142.1 ± 12.2*	142.0 ± 10.2	142.4 ± 12.3	141.7 ± 13.0	143.0 ± 11.7
Body mass index (Kg/M^2)	24.1 ± 3.9	24.1 ± 4.3	24.4 ± 3.9	24.0 ± 4.1	23.8 ± 4.0	24.3 ± 3.9
BMI for age z-score	2.93 ± 1.02	2.89 ± 1.05	2.90 ± 1.10	2.90 ± 1.00	29.98 ± 1.10	2.93 ± 0.99
Waist circumference (cm)	82.5 ± 9.8	84.1 ± 12.0	83.2 ± 9.2	83.7 ± 11.1	82.9 ± 11.0	83.9 ± 10.6
Passive smoker (%)	33.3	22.6	38.7	23.1*	25.4	22.7
Physical activity (MET/hour/week)	8.7 (3.7–14.6)	13.1 (3.9–23.7)*	5.4 (2.6–15.0)	12.2 (3.7–21.5)	9.6 (3.7–21.5)	8.9 (3.7–20.2)
Systolic blood pressure (mmHg)	100 (90–115)	105 (97–120)	100 (95–111)	105 (95–120)	104 (95–115)	105 (100–115)
Diastolic blood pressure (mmHg)	65 (60–75)	65 (60–70)	70 (60–79)	63 (60–70)	63 (60–70)	65 (60–75)
Biochemical data						
Fasting serum insulin (mU/mL)	17.5 ± 8.3	15.6 ± 7.9	18.2 ± 7.3	15.9 ± 8.3	15.1 ± 7.4	17.0 ± 8.5
Fasting blood sugar (mg/dl)	91.1 ± 8.8	93.0 ± 9.3*	91.9 ± 8.6	90.7 ± 7.6	91.4 ± 7.6	90.8 ± 8.7
HOMA-IR	4.10 ± 2.20	3.51 ± 1.94	4.16 ± 1.74	3.58 ± 1.95	3.42 ± 1.80	3.80 ± 2.00
Triglyceride (mg/dl)	124.5 (91.0-161.0)	79.0 (138.0-107.0)	109.0 (85.0-152.0)	127.0 (910 – 165.0)	105.0 (88.5-132.5)	89.0 (127.0-166.0)
Total cholesterol (mg/dl)	167.1 ± 29.5	171.1 ± 33.6	167.9 ± 31.6	168.9 ± 30.5	168.7 ± 27.0	167.7 ± 32.1
HDL (mg/dl)	46.0 ± 11.4	47.2 ± 11.1	45.2 ± 11.1	48.4 ± 11.0	48.4 ± 12.3	45.0 ± 10.6*
LDL-C (mg/dl)	97.7 ± 24.6	100.1 ± 29.4	98.5 ± 27.4	97.7 ± 24.7	99.4 ± 22.2	98.2 ± 29.4
Alanine aminotransferase (U/L)	16.0 (11.0-22.7)	16.0 (11.8–22.0)	17.0 (11.0–23.0)	16.0 (12.0–24.0)	15.0 (11.0–22.0)	15.0 (11.0–22.0)
Aspartate amino transferase (U/L)	15.0 (21.0-27.5)	23.0 (17.0–29.0)	22.0 (15.0–19.0)	23.0 (17.0–29.0)	17.0 (25.0–32.0)	17.0 (24.0–30.0)
Gamma-glutamyl transferase (U/L)	17.0 (14.0–21.0)	17.5 (15.0-21.2)	18.0 (15.0–21.0)	17.0 (15.0–22.0)	15.0 (17.0–21.0)	15.0 (17.0–21.0)

*P-value < 0.05

Obtained from ANOVA for continuous variables and chi-square test for categorical variables

Abbreviations: HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; HDL, high-density lipoprotein; LDL, Low-density lipoprotein

Table 3 Dietary intakes of study participants across quartile of dietary total, animal, and plant-based protein intake

	Total protein intake		Animal-based protein intake		Plant-based protein intake	
	Q1	Q4	Q1	Q4	Q1	Q4
Energy (Kcal)	3134 ± 1004	2995 ± 967	3210 ± 1043	3005 ± 948*	3049 ± 907	3109 ± 999
Total protein (% of energy)	10.7 ± 0.9	16.3 ± 1.5*	11.4 ± 1.6	15.9 ± 1.9*	13.6 ± 2.5	14.0 ± 2.1*
Plant-based protein (% of energy)	6.18 ± 1.06	6.50 ± 1.74*	7.3 ± 1.6	5.4 ± 1.2*	4.7 ± 0.54	8.3 ± 1.0*
Animal-based protein (% of energy)	4.59 ± 1.24	9.81 ± 2.46*	4.1 ± 0.86	7.6 ± 0.52*	8.9 ± 2.7	5.7 ± 2.1*
Fat (% of energy)	34.6 ± 6.6	32.4 ± 5.2*	32.7 ± 7.1	34.5 ± 5.3*	36.3 ± 5.1	30.1 ± 5.8*
SFA (% of energy)	10.1 ± 2.5	10.8 ± 2.8	9.3 ± 2.4	11.9 ± 2.7*	12.2 ± 2.4	9.1 ± 2.4*
MUFA (% of energy)	11.0 ± 2.9	10.5 ± 2.0	10.8 ± 2.9	11.1 ± 2.0	11.6 ± 2.3	9.7 ± 2.2*
PUFA (% of energy)	7.6 ± 2.3	6.4 ± 1.5*	7.4 ± 2.3	6.6 ± 1.6*	6.9 ± 2.1	6.7 ± 1.8
Carbohydrate (% of energy)	56.8 ± 6.6	54.4 ± 6.3*	58.4 ± 6.7	52.4 ± 6.1*	52.1 ± 5.1	59.4 ± 5.9*
Sodium (mg/1000 kcal)	1357 ± 414	1461 ± 372*	1456 ± 419	1424 ± 347	1403 ± 483	1552 ± 399*
Calcium (mg/1000 kcal)	381 ± 88	554 ± 150*	373 ± 70	575 ± 142*	529 ± 145	443 ± 126*
Zinc (mg/1000 kcal)	3.8 ± 0.5	5.4 ± 0.7*	4.2 ± 1.0	5.1 ± 0.7*	4.5 ± 0.7	4.7 ± 0.8*
Magnesium (mg/1000 kcal)	136 ± 20	175 ± 28*	156 ± 38	159 ± 25	140 ± 19	183 ± 31*
Iron (mg/1000 kcal)	6.6 ± 1.0	7.0 ± 1.3*	7.3 ± 1.3	6.3 ± 1.2*	5.7 ± 1.2	8.4 ± 1.2*
Fiber (g/1000 kcal)	18.2 ± 6.4	16.0 ± 5.3*	19.4 ± 6.4	14.1 ± 4.5*	12.9 ± 3.8	20.6 ± 5.3*

*P-value < 0.05

Obtained from One-way ANOVA

Abbreviations: HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; HDL, high-density lipoprotein; LDL, Low-density lipoprotein

Table 4 Multi-variable adjusted odds ratio (95% CI) of metabolic dysfunction-associated fatty liver disease across quartile of dietary total, plant, and animal protein intake

	Quartiles (percent of energy intake)				
	Q1	**Q2**	**Q3**	**Q4**	**P-trend***
Total protein					
Median intake	11.08	112.60	13.94	15.73	
Case/total	43/120	44/126	49/127	55/125	
Crude	1.00 (Ref)	0.86 (0.51–1.43)	1.00 (0.60–1.67)	1.26 (0.76–2.08)	0.29
Model 1[a]	1.00 (Ref)	0.85 (0.50–1.44)	0.91 (0.54–1.53)	1.16 (0.74–2.08)	0.37
Model 2[b]	1.00 (Ref)	1.14 (0.59–2.20)	0.98 (0.41 (2.35)	1.15 (0.34–3.82)	0.92
Animal-based protein					
Median intake	4.50	6.00	7.48	9.84	
Case/total	42/128	43/126	52/128	59/123	
Crude	1.00 (Ref)	1.06 (0.63–1.78)	1.40 (0.84–2.33)	**1.88 (1.13–3.14)**	**0.01**
Model 1[a]	1.00 (Ref)	0.99 (0.58–1.68)	1.42 (0.84–2.39)	**1.91 (1.13–3.22)**	**0.01**
Model 2[b]	1.00 (Ref)	1.30 (0.69–2.45)	1.55 (0.79–3.03)	**2.31 (1.01–5.30)**	**0.01**
Plant-based protein					
Median intake	4.90	5.83	6.75	7.93	
Case/total	55/126	47/127	48/125	46/125	
Crude	1.00 (Ref)	0.75 (0.45–1.25)	0.80 (0.48–1.33)	0.75 (0.45–1.24)	0.32
Model 1[a]	1.00 (Ref)	0.70 (0.41–1.17)	0.73 (0.43–1.23)	0.68 (0.41–1.51)	0.19
Model 2[b]	1.00 (Ref)	0.70 (0.39–1.27)	0.63 (0.32–1.23)	**0.48 (0.23–0.96)**	**0.04**

Obtained by Logistic regression analysis

* P-trend was obtained using a quartile of dietary exposure as an ordinal variable in the model.

Significant p-values are highlighted in bold

[a] Model 1 adjusted for age and sex

[b] Model 2 additionally adjusted for BMI for-age Z-score, puberty status, TG, HOMA-IR, PA, dietary energy intake, dietary fiber (g/1000 kcal), saturated fatty acid intake (% of energy), and total protein intake (% of energy) (when animal or plant-based protein intake is considered as an exposure variable)

protective effect at higher levels of plant protein intake. Specifically, participants in the highest quartile of plant protein consumption had significantly lower odds of MAFLD compared to those in the lowest quartile (OR = 0.48, 95% CI: 0.23–0.96; *p*-trend = 0.04).

Discussion

This study represents the first evaluation of the association between dietary protein intake and the odds of MAFLD among overweight and obese children and adolescents. We found that higher total protein intake was associated with a 40% increase in the odds of MAFLD, although this association did not reach statistical significance. However, increased animal protein intake was significantly associated with a 2.31-fold higher likelihood of MAFLD, whereas greater plant protein intake was significantly associated with a 53% decrease in the odds of developing MAFLD.

A review of the literature on protein intake and its potential link to NAFLD indicates that the majority of studies have been conducted in adult populations, with no data available of children and adolescents. In 2020, a cross-sectional study among overweight and obese adults showed that individuals who derive more than 17.3% of their daily caloric intake from protein have 5.09 times greater odds of NAFLD than those with lower intake [27]. In 2017, a cross-sectional study involving 1,128 Dutch adults found that higher total protein intake and protein intake from animal sources were linked to a 25% and 27% increased odds of developing NAFLD, respectively [28]; while higher plant protein intake was significantly associated with a 19% reduction in the odds of developing NAFLD [28].

Contrary to our findings, a recent case-control study among Iranian adults revealed that higher total protein intake was associated with a 74% reduced risk of NAFLD, while increased animal protein intake corresponded to a 2.80-fold higher risk; no significant association was observed for plant protein intake [29]. Furthermore, a recent cross-sectional study investigated the link between total protein intake, as well as protein consumption from plant and animal sources, and the likelihood of developing an unhealthy obesity phenotype among Iranian overweight and obese adolescents [16]. This study found that greater total, animal, and plant protein intake was linked to 68%, 80%, and 70% lower odds of an unhealthy obesity phenotype, respectively [16]. These discrepancies across studies may be attributed to variations in study design, sample size, age groups, and clinical outcomes, underscoring the need for further research on dietary protein intake and MAFLD risk, particularly in pediatric populations.

The findings of our study underscore the role of various dietary protein sources on the odds of developing MAFLD. In this context, several review studies evaluated the association between the consumption of protein-rich food groups and the development of NAFLD, yielding a range of conflicting results. A recent systematic review and meta-analysis assessed the association between food groups intake and the risk of NAFLD [30]. The findings of this study demonstrated that an increased intake of red meat was associated with a higher risk of NAFLD, whereas a greater consumption of nuts was linked to a reduced risk of NAFLD. Conversely, no significant association was identified between the intake of plant-based foods, dairy products, eggs, or fish, and the likelihood of developing NAFLD [30]. In 2024, a systematic review and meta-analysis indicated that a greater intake of red meat, including both processed and unprocessed varieties, was significantly associated with an elevated risk of NAFLD [31]. The results of the dose-response analysis from this study indicate a direct linear correlation between increased consumption of processed red meat and the elevated risk of NAFLD. Specifically, for every 25 g increase in the intake of processed red meat, the risk of developing NAFLD rises by 11.1%.

Furthermore, a systematic review and meta-analysis conducted in 2023 reveal that increased consumption of dairy products was \ associated with a 3% reduction in the risk of developing NAFLD [32]. In this context, when analyzing all animal protein sources collectively, increasing consumption of red meat and its products may counterbalance other animal protein sources' declining or neutral contributions. This trend can elevate the risk of developing NAFLD due to a heightened overall animal protein intake. On the other hand, research indicates that the food groups comprising plant proteins may be significantly or insignificantly associated with a decreased risk of developing NAFLD [30]. When all plant-based food groups contributing to protein intake are assessed collectively, they demonstrate a synergistic effect that can further reduce the risk of NAFLD. Therefore, future studies should analyze protein intake from both animal and plant sources, focusing on the specific contributions of each food group to overall protein quality. This is particularly important when considering the nutritional needs of children and adolescents.

Several mechanisms may explain the observed associations between protein intake and MAFLD. Higher animal protein intake has been linked to increased insulin levels [33] and insulin-like growth factor-1 in children and adolescents [34]. Insulin-like growth factor-1 is essential for the growth and development of children and adolescents [34]. Additionally, it contributes to the proliferation and differentiation of adipocytes, highlighting its significance in growth processes and metabolic regulation [34]. On the contrary, the protein's amino acid profile is of significant importance and should be carefully examined [35]. Branched-chain amino acids, primarily from animal products, have been associated with an elevated risk of obesity and related complications, including insulin resistance, T2DM, and NAFLD [35, 36]. This potential link may arise from their role in stimulating increased insulin secretion and insulin-like growth factor-1 [35].

On the other hand, research indicates that higher plant protein intake was associated with reduced serum levels of insulin-like growth factor-1 and elevated levels of insulin-like growth factor-1 binding protein [37]. Emerging evidence also suggests that persistent organic pollutants (POPs) in animal-derived foods may promote obesity and metabolic dysregulation by enhancing energy extraction from these foods and disrupting the metabolism of macronutrients [38, 39]. These changes occur due to alterations in the gut microbiota and can ultimately lead to an increase in fat mass [39]. Our findings further indicate that increased animal protein intake is associated with higher saturated fat intake and lower polyunsaturated fat and dietary fiber intake—nutritional patterns linked to liver fibrosis progression and NAFLD risk [40]. In contrast, plant protein intake appears to reduce saturated fat consumption while increasing dietary fiber intake, which may confer hepatoprotective effects [40].

While plant-based protein intake appears beneficial, our findings suggest it may also be associated with lower dietary calcium intake, raising concerns about adequate bone development in children and adolescents [41, 42]. The findings of our study also suggest that an increase in dietary sodium intake, associated with heightened protein consumption from plant sources, may lead to elevated calcium excretion [42]. Recent research indicates the need for calcium supplementation or the fortification of food products, such as flour and beverages, to enhance calcium intake in response to the increased consumption of plant proteins [42]. Furthermore, there are significant concerns associated with plant-based foods, particularly regarding their alignment with human nutritional requirements for protein [43]. These foods often contain essential amino acids in smaller quantities than necessary, leading to the presence of limiting amino acids that may restrict overall protein effectiveness [43]. Addressing these issues is crucial to ensure that plant-based diets can adequately fulfil the nutritional needs of individuals. In this context, nutritionists advocate for intentionally pairing different plant-based protein sources. This approach is essential for creating a comprehensive nutritional profile that includes all the amino acids required for proper function and overall well-being [38]. Therefore, it is advisable to conduct further research to examine the relationship between dietary protein intake from various sources and the risk of obesity and

associated complications such as MAFLD. Such studies should particularly consider the adequacy of micronutrient intake, especially among children and adolescents.

Strength and limitation

This study possesses several notable strengths that enhance its scientific rigor and relevance. To the best of our knowledge, it is the first to examine the association between dietary protein intake and the odds of MAFLD in the pediatric population. Using validated and reliable questionnaires to assess dietary intake and physical activity ensures the robustness of the data. The presence of mothers during face-to-face interviews likely improved the accuracy of dietary recall and intake quantification among child participants. Furthermore, all dietary and anthropometric assessments were conducted by skilled pediatric dietitians, reducing the likelihood of data collection errors. Nevertheless, certain limitations should be acknowledged. The study's cross-sectional design restricts the ability to infer causality between dietary protein intake and MAFLD risk. While a validated FFQ was employed to estimate nutritional intake, some measurement errors remain possible. Additionally, despite adjustments for confounding variables, the potential for residual confounding due to unmeasured or unidentified factors cannot be entirely excluded.

Conclusion

This study emphasizes the varying effects of different dietary protein sources on the likelihood of developing MAFLD. While total protein intake showed no significant association with MAFLD, higher animal-based protein consumption was linked to an increased risk, whereas plant-based protein intake exhibited a protective effect. These findings highlight the importance of protein quality and composition in metabolic health. Future research should explore the underlying mechanisms driving these associations and consider interventional studies to assess the efficacy of plant-based protein-rich diets in MAFLD prevention.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12937-025-01142-4.

Supplementary Material 1

Acknowledgements
The authors express their appreciation to the participants of the study for their enthusiastic support and to the staff of the involved hospitals for their valuable help. This study is taken from the Obesity registry program in children at Tehran University of Medical Sciences (IR.TUMS.CHMC. REC.1401.016). We are thankful to Dr. Mohammad Hassan Sohouli and Dr. Afshin Ostovar, head of the obesity registry at Tehran University of Medical Sciences.

Author contributions
Overall, GA and PR, supervised the project and approved the final version of the manuscript to be submitted. GA designed the research; PD assessed the non-alcoholic fatty liver disease; KF and MAF gathered data; AN and MHS analyzed and interpreted the data; AN drafted the initial manuscript; and GA critically revised the manuscript. All authors approved the final version of the manuscript submitted for publication.

Funding statement
This study was supported in part by a Grant (NO:43014285-1) from the Shahid Beheshti University of Medical Sciences.

Data availability
The datasets analyzed in the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
National nutrition and Food Technology Research Institute (NNFTRI) ethics committee approved the study protocol (IR.SBMU.NNFTRI.REC.1402.015). All participants provided written informed consent and were informed about the study. All procedures performed in studies involving human participants adhered to the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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Received: 24 February 2025 / Accepted: 28 April 2025
Published online: 10 May 2025

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