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The association of fructose and fiber consumption and physical activity with non-alcoholic fatty liver disease in children and adolescents: a cross-sectional study

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Abstract

Background Non-alcoholic fatty liver disease (NAFLD) is emerging as the most prevalent liver disease in overweight and obese children. While no cure exists, dietary and lifestyle modifications have been shown to improve the condition. This study investigates the relationship between fructose and fiber consumption, physical activity, and NAFLD in children.

Methods A cross-sectional study was conducted on 378 overweight and obese children aged 6–13 years. NAFLD diagnosis was confirmed via ultrasound, and dietary intake was assessed using a 147-item food frequency questionnaire (FFQ). Physical activity was evaluated using the Modifiable Activity Questionnaire (MAQ). Multivariable logistic regression models were applied to determine the associations.

Results After excluding 53 participants due to incomplete data, 325 were included in the final analysis. The mean age was 9.2 ± 1.7 years, and 35% had NAFLD. No significant association was found between fructose intake and NAFLD (OR: 0.67, 95% CI: 0.35–1.29, $P = 0.221$). However, higher intake of legume fiber (OR: 0.48, 95% CI: 0.26–0.90, $P = 0.03$) and nut fiber (OR: 0.52, 95% CI: 0.28–0.95, $P = 0.04$) was significantly associated with a reduced risk of NAFLD. Physical activity showed a trend towards reduced NAFLD risk but was not statistically significant after adjustments (OR: 0.53, 95% CI: 0.22–1.04, $P = 0.07$).

Conclusions While fructose intake was not significantly linked to NAFLD in this population, fiber from legumes and nuts appeared protective. Further prospective studies are needed to confirm these findings and clarify the role of physical activity in NAFLD prevention.

Keywords Fructose, Fiber, NAFLD, Non-alcoholic fatty liver disease, Children, Physical activity

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is a growing health concern among children and adolescents, characterized by excessive fat accumulation in the liver in the absence of significant alcohol consumption [1]. The prevalence of NAFLD in pediatric populations has risen alarmingly in recent years, with estimates ranging from 3 to 10% in the general pediatric population and up to 70–80% in obese children [2]. This surge in prevalence parallels the global increase in childhood obesity and sedentary lifestyles, making NAFLD the most common chronic liver disease in children and adolescents in developed countries [3]. The implications of NAFLD extend far beyond liver health, as it is associated with numerous complications that can significantly affect a child's quality of life and long-term health outcomes. These complications include an increased risk of type 2 diabetes, cardiovascular disease, and progression to more severe liver conditions such as non-alcoholic steatohepatitis (NASH), cirrhosis, and even hepatocellular carcinoma [4]. Moreover, NAFLD in childhood often persists into adulthood, contributing to the growing burden of liver disease in the general population [5]. The economic impact of NAFLD is substantial, with estimates suggesting annual direct medical costs of $103 billion in the United States alone [6]. This financial burden is expected to increase as the prevalence of NAFLD continues to rise, highlighting the urgent need for effective prevention and management strategies.

In recent years, dietary factors and physical activity have emerged as key modifiable risk factors in the development and progression of NAFLD. Particularly, the consumption of fructose, a sugar commonly found in sweetened beverages and processed foods, has been implicated in the pathogenesis of NAFLD [7]. Fructose consumption has quintupled over the past century and more than doubled in the last 30 years [8]. Fructose metabolism in the liver can lead to increased lipogenesis and insulin resistance, both of which are central to the development of fatty liver [9]. Conversely, dietary fiber has been associated with potential protective effects against NAFLD, possibly through its role in modulating gut microbiota and reducing systemic inflammation [10]. Physical activity, another crucial lifestyle factor, has been shown to have beneficial effects on liver health, independent of weight loss. Regular exercise can improve insulin sensitivity, reduce hepatic fat content, and enhance overall metabolic health [11].

Several studies have investigated the effect of these factors on NAFLD risk. For instance, a longitudinal study demonstrated a strong association between increased fructose consumption and the development of NAFLD in children [12]. Similarly, one study found that low dietary fiber intake was independently associated with NAFLD severity in a pediatric [13]. Regarding physical activity, a systematic review concluded that higher levels of physical activity were consistently associated with lower risk of NAFLD in children and adolescents [14]. While some research has found higher fructose intake in adults with NAFLD compared to controls, other studies have suggested a potential protective effect of fructose against fatty liver disease [15, 16]. These contradictory findings highlight a significant gap in our understanding, especially in pediatric populations where research is particularly scarce.

Therefore, this study aims to investigate the associations between fructose intake, fiber consumption, physical activity, and NAFLD in children and adolescents. We hypothesize that higher fructose intake and lower fiber consumption will be associated with an increased risk of NAFLD, while higher levels of physical activity will be associated with a decreased risk. By examining these relationships in a pediatric population, we aim to contribute valuable insights to inform dietary recommendations and lifestyle interventions for the prevention and management of NAFLD in children and adolescents.

Method and materials

Study design

This cross-sectional study was conducted on 378 obese and overweight children and adolescents, 6–13 years old. Participants were divided into two groups (NAFLD and control). NAFLD was diagnosed based on the American Association for the Study of Liver Diseases (AASLD). Individuals were included in the study based on the following criteria: Children and adolescents 6–13 years old and Overweight or obese (BMI Z score greater than 1 standard deviation). The subjects with an inherited liver disease, or alternative causes such as Wilson disease, or those who had heart disease or hepatitis or used liver drugs such as valproate and amiodarone were excluded. The parents or legal guardians of the individuals signed an informed written consent. The Research Institute has approved this study for Endocrine Science / Shahid Beheshti University of Medical Science (IR.TUMS.VCR.REC.1397.333). In this study, to start the sampling process, we first contacted the General Directorate of Education of Tehran. After explaining the objectives of the study and obtaining the necessary permits, the department introduced three areas in Tehran for conducting the present study. We then selected the schools by using simple random sampling with a table of random numbers, ensuring all schools in the aforementioned areas had an equal chance of being selected.

After the random selection of schools, we proceeded with the random selection of students. It should be noted that this random selection was among overweight or obese children and adolescents in the first and

second elementary years (6–13 years old) in the schools of the above-mentioned areas. After examining the health records in the selected schools, students whose body mass index was in the range of overweight or obesity (according to the World Health Organization standard, body mass index z-score > 1) were selected using a random number table.

Dietary assessment

Participants' food intake was assessed using a valid and reliable food frequency questionnaire (FFQ) of 147 items [17], which measured the frequency of their children's food consumption in the previous 12 months. The questionnaire validity and details about its design and food items were previously reported [18]. Each of the 147 items was declared by consumption for the day, week, month, and year. The size of the food portion was reported in natural units (plates, slices). Daily intake of foods, energy, fructose, and different types of fiber has been converted to grams. The actual weight (grams) of each participant's nutrient diet was determined using the USDA food composition table (RAW 2015) and for traditional Iranian food, we used the Iranian food composition Table [19].

Physical activity assessment

Physical activity was assessed using the Modifiable Activity Questionnaire (MAQ), which has been validated for use in children and adolescents [20]. The MAQ collects information on leisure-time and occupational physical activities performed over the past year [21]. Participants were asked to report activities they engaged in at least 10 times during the past year, specifying the frequency and duration of each activity [22].

For each reported activity, the average number of hours per week was calculated. Metabolic equivalent (MET) values were assigned to each activity based on the Compendium of Physical Activities [23]. The product of the MET value, average duration, and frequency of each activity was summed to estimate total weekly energy expenditure in MET-hours per week [24].

Physical activity levels were categorized as low (< 600 MET-minutes/week), moderate (600–3000 MET-minutes/week), or high (> 3000 MET-minutes/week) based on WHO guidelines [25]. Additionally, asking participants to report average daily time spent watching television, using computers, or playing video games, assessed sedentary behavior [26].

To ensure accuracy, trained interviewers administered the MAQ, and visual aids were used to help participants estimate time spent on various activities [27]. The reliability and validity of the MAQ in pediatric populations have been previously established, with test-retest correlations ranging from 0.74 to 0.92 [28].

Laboratory assessment

After fasting for 10–12 h overnight, a 5 cc blood sample was taken from all study subjects between 7 and 9 AM. To separate the serum, the samples were centrifuged at room temperature at 3000 revolutions per minute for 10 min. The separated serum was aliquoted into 1 mL microtubes to measure the desired biochemical factors and stored at -80 °C until testing.

Insulin was measured by electrochemiluminescence immunoassay using a Roche Diagnostics kit. Glucose was measured by the enzymatic method using glucose oxidase. Total cholesterol was measured by the enzymatic method using cholesterol esterase and cholesterol oxidase. HDL-C was measured after deposition of lipoproteins containing apo-B by phosphotungstic acid, using the same enzymatic method as total cholesterol. Triglycerides were measured enzymatically using glycerol phosphate oxidase. Laboratory kits made by Pars Azmoun company were used to measure glucose, total cholesterol, HDL-C, and triglycerides.

LDL-C in serum samples with triglyceride values equal to or less than 400 mg/dL was calculated using the Friedewald formula as follows:

LDL-C = Total Cholesterol - (HDL-C + Triglyceride/5).

The external and internal sensitivity (CV) of all biochemical measurements were less than 2% [29].

Anthropometry assessment

Trained healthcare professionals who underwent standardized training to ensure consistency and accuracy performed all anthropometric measurements. Weight was measured to the nearest 100 g using a calibrated scale (Seca, Hamburg, Germany), with participants wearing light clothing and no shoes. Height was measured barefoot to the nearest 0.5 cm using a standard stadiometer. Waist and hip circumferences were measured to the nearest 0.5 cm using a non-elastic tape measure, with waist circumference measured around the belly button and hip circumference at the widest point of the hips. All circumference measurements were taken over light clothing without applying pressure to the body surface. Body mass index (BMI) was calculated as weight in kilograms divided by height squared in meters. Trained pediatricians according to the criteria established by Marshall and Tanner (1970) for both girls and boys assessed pubertal staging. Certified nurse is following a standardized protocol conducted blood pressure measurements. Systolic and diastolic blood pressures were measured twice in the right arm using a calibrated mercury sphygmomanometer (certified by the National Institute of Standards and Industrial Research) after a minimum 5-minute resting period, with a 15-minute interval between readings. The average of the two readings was used for analysis. To ensure reliability, all measuring instruments were

calibrated regularly according to manufacturers' guidelines. Inter-observer and intra-observer variability were assessed periodically throughout the study to maintain measurement quality. All personnel involved in data collection underwent regular refresher training sessions to maintain consistency in measurement techniques.

Fatty liver assessment

Fatty liver disease was evaluated by ultrasound. In this study, the liver was examined through ultrasound after at least 6 h of fasting. Ultrasound of both the back and right front parts of the liver was taken using the standard abdominal transducer Samsung WS 80 A. A healthy liver is uniform and has the same or longer echo than the kidney and shorter than the spleen and the assessment and classification of fatty liver condition is typically performed by analyzing various ultrasound characteristics. These characteristics encompass the brightness level of the liver, the contrast distinction between the liver and kidney, the visual appearance of blood vessels within the liver, the texture of the liver tissue, and the clarity of the diaphragm region. Trained and certified professionals such as radiologists, sonographers, or specialized technicians typically perform ultrasound examinations, especially for diagnostic purposes like assessing fatty liver disease. These operators undergo formal education and training programs to learn how to properly operate ultrasound equipment and interpret the images. To ensure accurate and reliable results, ultrasound operators should follow standardized protocols and guidelines for conducting the examinations, optimizing image quality, and interpreting the findings [30].

Statistical analysis

To evaluate the normality of the distribution of the variables, the Kolmogorov-Smirnov test was used and a histogram was drawn. Demographic, anthropometric, clinical, biochemical, laboratory, food, and nutrient intake characteristics of the participants for normally distributed data were reported according to tertiles of fructose and fiber consumption (including leguminous fiber, cereal fiber, vegetable fiber, fruit fiber, nut fiber, and total fiber) as mean ± standard deviation or median (interquartile range 25–75) respectively. Non-normally distributed variables and qualitative variables were expressed as a percentage. ANOVA test (for normally distributed variables) or Kruskal-Wallis test (for non-normally distributed quantitative variables) and Chi-squared test (for qualitative variables) were used based on the fiber and fructose consumption. In addition, T-test (for quantitative variables with normal distribution) or Mann-Whitney test (for quantitative variables with non-normal distribution) and Chi-square test (for qualitative variables with normal distribution) respectively to compare variables based on the degree of suffering or no suffering from fatty liver. The determination of the relationship between fructose and dietary fiber intake as a quantitative variable with fatty liver status was examined by logistic regression. To investigate the relationship between fatty liver disease and dietary fructose intake, the following three models were used: Model 1, Crude model, Model 2: Multivariable adjusted based on gender, maturity status, body mass index z-score and total daily energy intake from the diet, and Model 3: Multivariable adjusted based on the gender, maturity status, z-score of body mass index, and total daily energy intake from diet, leisure time sports activity, and triglyceride. To examine the relationship between fatty liver disease and dietary fiber intake, the following three models were used: Model 1, Crude model, Model 2: Multivariable adjusted based on gender, maturity status, body mass index z-score and total daily energy intake from the diet, and Model 3: Multivariable adjusted based on the gender, maturity status, z-score of body mass index and total daily energy intake from diet, and triglyceride. Since there is no threshold for fructose and fiber consumption in the population of children and adolescents, in this study we decided to convert the food consumption of these two variables into tertiles (the first tertile is considered as a reference) using logistic regression. Additionally, odds ratios of increased fiber and fructose intake with 95% confidence intervals based on fatty liver disease and absence of fatty liver disease were calculated using logistic regression. All analyses were performed by SPSS version 20 (SPSS, Chicago, IL, USA) and a significance level of 0.05 was considered.

Results

Out of 378 overweight or obese children and adolescents, 51 participants were excluded from the study due to the following reasons: individuals with inadequate FFQ ($n=12$), Under-reporting or over-reporting ($n=16$), absence of biochemical data ($n=2$), and did not have an abdominal ultrasound ($n=21$) and finally, 325 individuals were included in the study.

The mean age of participants was 9.2 ± 1.7 and 51% were female and 82% were in pubertal status.

The mean BMI was 23.1 ± 3.1 and 35% had NAFLD. General characteristics of participants are shown in Table 1. As presented in Table 2, no significant differences were observed in age ($p=0.094$), sex ($p=1.033$), and pubertal status ($p=0.743$) between the NAFLD and non-NAFLAD groups. However, BMI Z-score was significantly higher in NAFLD group (NAFLD: 2.7 ± 0.7 vs. non-NAFLD: 2.5 ± 0.6; $P=0.003$). In addition, NAFLD group had a significantly higher waist circumference than the healthy group (p value<0.001). The mean FBS (92.1±9.3; $p=0.01$) and HOMA-IR (3.1 (2.5–5.1); $p<0.001$) were significantly higher in NAFLD group. HDL was

Table 1 Anthropometric, laboratory and nutrition intake data in obese and overweight patients and controls

Variable	Percentile /Mean ± SD(or median)
Age (y)	9.2 ± 1.7
Sex (girl)	165(51)
Puberty (%)	268(83)
BMI(kg/m^2)	23.1 ± 3.1
Z score BMI	2.5 ± 0.7
Waist circumference (cm)	80.2 ± 9.0
Physical activity (MET-h/week)	8.9 (2.3–24)
SBP (mmHg)	103.0(95.0-114.0)
DBP (mmHg)	63.0(60.0–70.0)
Laboratory:	
TG (mg/dl)	105.0(75.0-137.7)
HDL (mg/dl)	49.9 ± 11.8
LDL (mg/dl)	97.7 ± 25.4
FBS (mg/dl)	90.6 ± 9.3
TC (mg/dl)	171.7 ± 33.6
HOMA-IR	2.7(1.8–4.3)
Fatty liver (yes)	113(35)
Nutrition intake:	
Total energy (kcal)	2996.0 ± 1022.9
Carbohydrate (% energy)	56.1 ± 5.9
Protein (% energy)	13.4 ± 2.2
Fat (% energy)	33.0 ± 5.5
PUFA (% energy)	6.6 ± 1.9
MUFA (% energy)	10.5 ± 2.2
SFA (% energy)	10.3 ± 2.5
Fructose (gr/1000 kcal)	8.1 ± 3.2
Legumes fiber (gr/1000 kcal)	9.0 ± 1.2
Grain fiber (gr/1000 kcal)	3.3 ± 1.8
Vegetable fiber (gr/1000 kcal)	1.3 ± 0.8
Fruit fiber (gr/1000 kcal)	3.2 ± 1.7
Nut fiber (gr/1000 kcal)	0.2 ± 0.4
Total fiber (gr/1000 kcal)	9.7 ± 2.9

Results are expressed as mean ± SD for parametric data and median and interquartile range (IQR) for nonparametric data. Qualitative variables were presented as frequencies and percentages. BMI: body mass index SBP: Systolic blood pressure; DPB: Diastolic blood pressure; TG: triglycerides; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein cholesterol; TC: total cholesterol SFA: Saturated fatty acid, MUFA: Monounsaturated fatty acid, PUFA: Polyunsaturated fatty acid

significantly lower in NAFLD group than the healthy group (NAFLD: 47.6±11.2 vs. non-NAFLD: 51.1±12.0; P=0.01). There was no statistically significant difference between two groups in nutritional intakes, but Legume fiber consumption was significantly higher in NAFLD group (p=0.023) (Table 2).

Crude and adjusted multivariable odds ratios and 95% CIs for NAFLD based on dietary fiber, fruit, and vegetables are presented in Table 3. After multivariable adjustment in model 2 (OR: 1.0, 95%, CI: 0.35–1.29, P=0.221), and multivariable adjustment in model3 (OR: 0.67, 95%, CI: 0.35–1.29, P=0.221), there were no significant differences between fructose consumption and NAFLD. In addition, there was no significant relation between fructose and NAFLD in the crude model (OR: 1.06, 95%, CI: 0.61–1.84, P=0.70) (Table 3).

In comparison with first tertile of vegetable fiber, the risk of NAFLD was 47% lower in individuals in the third tertile (OR: 0.53, 95%, CI: 0.29–0.99). A significant relation was indicated between legume fiber (P=0.03) and nut fiber (P=0.04) with NAFLD risk. The risk of NAFLD was 24% lower in participants in the third legume fiber group and 48% lower in participants in the third nut fiber group (OR:0.48,95%, CI:0.26–0.90; OR:0.52,95%, CI:0.28–0.95 respectively) (Table 3).

The results of this study indicate that higher levels of physical activity, as measured by MAQ, might be associated with a reduced risk of NAFLD in overweight and obese children and adolescents. In the crude model, the odds of having NAFLD were lower in the second and third tertiles of physical activity compared to the first tertile, though the results were not statistically significant.

Table 2 Anthropometric, laboratory and nutrition intake data in obese patients and controls

Variable	NAFLD *n* = 113 Percentile /Mean ± SD(or median)	NON- NAFLD *n* = 212 Percentile /Mean ± SD(or median)	*P* Value
Age (y)	9.4 ± 1.8	9.0 ± 1.6	0.094
Sex (girl)	47	53	1.033
Puberty (%)	85	81	0.743
BMI(kg/m^2)	24.2 ± 3.5	22.5 ± 2.7	< 0.001
Z score BMI	2.7 ± 0.7	2.5 ± 0.6	0.003
Waist circumference (cm)	83.8 ± 9.7	78.3 ± 8.1	< 0.001
Physical activity (MET-h/week)	8.9 (2.8–24.7)	8.6 (2.1–23.5)	0.55
SBP (mmHg)	105.0(99.0-110.0)	103.0(90.0-115.0)	0.703
DBP (mmHg)	60.0(60.0–70.0)	63.0(60.0–70.0)	0.376
Laboratory			
TG (mg/dl)	108(82–148)	102(75–135)	0.114
HDL (mg/dl)	47.6 ± 11.2	51.1 ± 12.0	0.010
LDL (mg/dl)	100.4 ± 26.9	96.3 ± 9.3	0.174
FBS (mg/dl)	92.1 ± 9.3	89.9 ± 9.3	0.044
TC (mg/dl)	172.2 ± 30.5	171.4 ± 35.2	0.840
HOMA-IR	3.1(2.5–5.1)	2.4(1.6–3.8)	< 0.001
Nutrition intake			
Total energy (kcal)	3082.3 ± 1141.1	2950.0 ± 953.6	0.267
Carbohydrate (% energy)	55.8 ± 6.4	56.3 ± 5.5	0.406
Protein (% energy)	13.6 ± 2.3	13.4 ± 2.2	0.396
Fat (% energy)	33.4 ± 6.0	32.8 ± 5.2	0.387
PUFA (% energy)	6.9 ± 2.1	6.9 ± 1.8	0.991
MUFA (% energy)	10.5 ± 2.5	10.4 ± 2.1	0.661
Saturated fat (% energy)	10.5 ± 2.7	10.3 ± 2.3	0.515
Fructose (gr/1000 kcal)	9.7 ± 1.3	8.1 ± 3.3	0.529
Legumes fiber (gr/1000 kcal)	1.0 ± 0.7	1.2 ± 1.0	0.023
Grain fiber (gr/1000 kcal)	3.4 ± 1.6	3.2 ± 1.9	0.228
Vegetable fiber (gr/1000 kcal)	1.2 ± 0.8	1.3 ± 0.8	0.289
Fruit fiber (gr/1000 kcal)	3.0 ± 1.5	3.3 ± 1.8	0.109
Nut fiber (gr/1000 kcal)	0.1 ± 0.2	0.2 ± 0.4	0.113
Total fiber (gr/1000 kcal)	9.5 ± 2.6	9.9 ± 3.0	0.194

Results are expressed as mean ± SD for parametric data and median and interquartile range (IQR) for nonparametric data. Qualitative variables were presented as percentages

After adjusting for potential confounders (e.g., gender, BMI z-score, energy intake, and triglyceride levels), the association between physical activity and NAFLD risk remained non-significant, but the odds ratios continued to suggest a possible protective effect, with those in the highest tertile of physical activity having approximately 47% lower odds of developing NAFLD compared to the least active group (OR: 0.53, 95% CI: 0.22–1.04, $P=0.07$).

Discussion

This cross-sectional study provides valuable insights into the association between fructose intake, fiber consumption, and non-alcoholic fatty liver disease (NAFLD) in overweight and obese children and adolescents. While we hypothesized that higher fructose intake would be associated with increased NAFLD risk, our findings did not support a significant relationship between fructose consumption and NAFLD. In contrast, we observed that higher legume and nut fiber intake was significantly associated with a reduced risk of NAFLD, suggesting a potential protective role of specific types of fiber in liver health. While physical activity did not show a robust association with NAFLD after adjusting for confounders, the observed trends suggest it could still play a supportive role in overall metabolic health.

Studies in both pediatric and adult populations have reported a positive correlation between high fructose consumption and increased liver fat [1, 7]. For instance, Abdelmalek et al. found that fructose intake was associated with increased hepatic fibrosis in patients with NAFLD, further supporting the role of fructose in exacerbating liver disease [31].

However, our study did not observe a significant association between fructose intake and NAFLD. This finding is consistent with a recent study, which found no significant association between fructose intake and liver

Table 3 Crude and multivariable-adjusted odds ratios and 95% CI for NAFLD according to fructose, and fiber type's tertile

NAFLD				*P trend
	Tertile 1	Tertile 2	Tertile 3	
	Fructose consumption			
Crude	1	1.06(0.61–1.84)	0.91(0.52–1.60)	0.70
Model 1	1	0.90(0.50–1.60)	0.67(0.35–1.29)	0.21
Model 2	1	0.90(0.50–1.61)	0.67(0.35–1.29)	0.22
	Legumes fiber consumption			
Crude	1	0.87(0.50–1.50)	0.60(0.34–1.06)	0.07
Model 1	1	0.78(0.44–1.38)	0.51(0.28–0.95)	0.03
Model 2	1	0.76(0.43–1.34)	0.48(0.26–0.90)	0.02
	Grain fiber consumption			
Crude	1	1.33(0.75–2.37)	1.73(0.98–3.05)	0.06
Model 1	1	1.27(0.70–2.32)	1.65(0.86–3.17)	0.13
Model 2	1	1.25(0.69–2.29)	1.62(0.84–3.13)	0.15
	Vegetables fiber consumption			
Crude	1	0.67(0.38–1.17)	0.67(0.38–1.17)	0.20
Model 1	1	0.66(0.37–1.16)	0.55(0.30–1.02)	0.06
Model 2	1	0.66(0.37–1.17)	0.53(0.29–0.99)	0.05
	Fruit fiber consumption			
Crude	1	1.01(0.58–1.77)	0.84(0.47–1.47)	0.49
Model 1	1	0.88(0.50–1.57)	0.67(0.36–1.24)	0.19
Model 2	1	0.89(0.50–1.58)	0.67(0.36–1.26)	0.20
	Nut fiber consumption			
Crude	1	0.83(0.48–1.45)	0.63(0.35–1.10)	0.11
Model 1	1	0.71(0.40–1.26)	0.51(0.28–0.93)	0.03
Model 2	1	0.72(0.41–1.28)	0.52(0.28–0.95)	0.04
	Total fiber consumption			
Crude	1	0.97(0.56–1.70)	0.99(0.56–1.73)	0.97
Model 1	1	0.80(0.44–1.45)	0.72(0.36–1.43)	0.36
Model 2	1	0.78(0.43–1.41)	0.68(0.34–1.38)	0.31
		Physical activity (MET-h/week)		
Crude	1	0.9(0.55–1.47)	0.65 (0.33–1.22)	0.18
Model 1	1	0.86(0.48–1.18)	0.62(0.28–1.16)	0.11
Model 2	1	0.79(0.41–1.10)	0.53(0.22–1.04)	0.07

Mode1: Adjusted for sex, puberty, body mass index Z score and energy intake

Model 2: Adjusted for sex, puberty, body mass index Z score, energy intake, and triglycerides

*P-values from logistic regression

fat content in a cohort of overweight children [32]. One possible explanation for this discrepancy is the source of fructose in our study population. While many studies focus on fructose from sugary drinks and processed foods, the primary source of fructose in our cohort may have been fruits, which contain fiber, vitamins, and antioxidants that may mitigate the adverse effects of fructose on liver health [33]. Further research is needed to distinguish between the effects of fructose from natural sources and that from processed foods and sugary beverages. A recent meta-analysis demonstrated that higher dietary fiber intake, particularly soluble fiber, was associated with a reduced risk of NAFLD across multiple populations [34].

Moreover, our findings are consistent with the work of Farrell et al., who found that higher legume and nut consumption was associated with improved liver health outcomes in individuals with NAFLD [35]. The beneficial effects of legume and nut fibers may be due to their higher content of fermentable fibers, which are broken down by gut bacteria into short-chain fatty acids, with known anti-inflammatory and metabolic benefits [36]. The reduced glycemic load of these foods may also help prevent spikes in blood glucose and insulin levels, which are key drivers of NAFLD development. Interestingly, while legume and nut fiber were significantly associated with reduced NAFLD risk in our study, total fiber intake was not. This suggests that the specific types of fiber consumed may be more important than total fiber intake in influencing NAFLD risk, a notion supported by recent research, reported that the source of dietary fiber, rather

than the total amount, was critical in modulating liver fat accumulation and metabolic health [37].

A recent systematic review examined the effects of physical activity on NAFLD and found that both aerobic and resistance exercise were associated with reduced liver fat independent of changes in body weight [38]. Similarly, a cohort study by Zhang et al. reported that higher levels of physical activity were inversely associated with the risk of NAFLD in children and adolescents, further supporting the role of exercise in mitigating liver fat accumulation [39]. The lack of statistical significance in our study may be due to the relatively small sample size or the cross-sectional design, which limits the ability to assess the long-term effects of physical activity on liver health.

Our study's findings suggest that legume and nut fiber intake is significantly associated with a reduced risk of NAFLD, which may be explained by several mechanisms. Dietary fibers, particularly fermentable fibers found in legumes and nuts, are metabolized by gut microbiota into short-chain fatty acids (SCFAs), such as butyrate, acetate, and propionate, which have been shown to exert anti-inflammatory and insulin-sensitizing effects [40, 41]. These SCFAs can reduce liver fat accumulation by improving hepatic insulin sensitivity and decreasing lipogenesis [42]. Additionally, fermentable fibers may enhance the gut barrier function, reducing the translocation of endotoxins into the bloodstream, which is a known trigger of systemic inflammation and NAFLD progression [43]. Moreover, fibers can modulate glucose metabolism by slowing down the absorption of carbohydrates, thereby preventing postprandial spikes in insulin and glucose levels, which are key factors in the development of insulin resistance and fatty liver [44].

This study presents several strengths. First, it focuses on a pediatric population, which is crucial given the rising prevalence of NAFLD among children and adolescents. Additionally, the use of a validated 147-item FFQ and MAQ ensures a comprehensive and reliable assessment of dietary intake and physical activity levels. The thorough collection of anthropometric, biochemical, and clinical data, along with the use of standardized protocols for ultrasound-based NAFLD diagnosis, enhances the study's methodological rigor. Furthermore, adjusting for multiple confounders, such as BMI z-score, gender, and energy intake, strengthens the validity of the findings.

However, the study also has several limitations. As a cross-sectional design, it cannot establish causality between fructose or fiber intake and NAFLD. The reliance on self-reported dietary and physical activity data may introduce recall bias or inaccuracies in reporting. Additionally, the study population is limited to overweight and obese children from a specific geographic area, which may reduce the generalizability of the findings to other populations. Finally, while the study adjusted for several confounders, residual confounding from unmeasured variables, such as genetic factors or other lifestyle behaviors could still affect the results.

In conclusion, the results showed no significant link between fructose consumption and NAFLD. However, higher intakes of fiber from legumes and nuts were associated with a reduced risk of NAFLD. Although the association between physical activity and NAFLD risk was not statistically significant, the trends suggest a potential protective effect of higher activity levels on liver health. The findings emphasize the importance of focusing on the quality of diet, particularly increasing fiber intake from legumes and nuts, and encouraging physical activity as part of strategies to prevent or manage NAFLD in pediatric populations. Longitudinal or interventional studies are needed to confirm these associations and explore long-term effects.

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Author contributions
HI and PM are the guarantors; CA and ME wrote the manuscript, SS and AT collected the data. GA and EY interpreted the data, provided professional comments, and PD and AA revised the manuscript.

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Data availability
The datasets generated and/or analyzed during the current study are not publicly available, but are available from the corresponding author at reasonable request.

Declarations

Ethics approval and consent to participate
The Research Institute has approved this study for Endocrine Science / Shahid Beheshti University of Medical Science (IR.TUMS.VCR.REC.1397.333). The parents or legal guardians of the individuals signed an informed written consent.

Consent for publication
This manuscript does not contain data from any individual person and there fore this is not applicable.

Conflict of interest
All the authors declared that they have no conflicts of interest.

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