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The Impact of Dietary Sugars and Saturated Fats on Body and Liver Fat in a Healthcare Worker Population

Sophia Eugenia Martinez-Vazquez [1,*,†], Ashuin Kammar-Garcia [2,†], Carlos Moctezuma-Velazquez [3], Javier Mancilla-Galindo [4], Ignacio García-Juárez [1] and Luis Uscanga-Domínguez [1]

[1] Department of Gastroenterology. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico.
[2] Dirección de Investigación. Instituto Nacional de Geriatría, Mexico City, Mexico.
[3] Department of Medicine. Division of gastroenterology. University of Alberta. Edmonton, Canada.
[4] Institute for Risk Assessment Sciences. Utrecht University, Utrecht, Netherlands.
† These authors contributed equally to the study and thus share first authorship.
* Correspondence: author: Sophia Eugenia Martínez- Vázquez (sophia.martinezv@incmnsz.mx). Department of Gastroenterology. Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Vasco de Quiroga 15, Col. Belisario Domínguez Sección XVI, Alcaldía Tlalpan, Mexico City 14080, Mexico.

Abstract: Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a prevalent disease influenced by dietary factors. While high sugar and fat consumption are associated with weight gain, their specific impact on liver fat accumulation remains unclear. This study aimed to evaluate the relationship between sugar and saturated fat intake with liver and body fat composition. **Methods**: A cross-sectional study was conducted from September 2021 to February 2023 in workers from a tertiary care center in Mexico City. Anthropometric measurements, body composition (bioelectrical impedance analysis and skinfold assessment), and liver fat (vibration-controlled transient elastography) were measured. Dietary intake was assessed with a 24-hour recall questionnaire and analyzed with specialized software. Linear and logistic regression models were fitted to study the relationship between nutrient intake and liver/body fat. **Results**: A total of 534 healthcare workers (median age: 41.5 years, 61.4% female) were included. Hepatic steatosis was present in 42.5% of participants. Higher carbohydrate intake was associated with increased liver fat (β=0.23, 95% CI: 0.02-0.45), with each additional 15g of carbohydrates increasing the risk of steatosis by 5% (OR=1.053, 95% CI: 1.006-1.102). Fat and sugar intake were associated with higher body fat but not liver fat. **Conclusion**: Carbohydrate intake was linked to liver fat accumulation, whereas fat and sugar intake were primarily associated with body fat. Tailored dietary recommendations could be informed by these findings. Prospective dietary assessment methods and a nutritional geometry approach could be applied in future studies.

Keywords: liver steatosis; body fat distribution; nutrient intakes; saturated fatty acids; dietary sugars

1. Introduction

Nonalcoholic fatty liver disease (NAFLD), now termed metabolic dysfunction-associated steatotic liver disease (MASLD), is a common global health problem affecting 25-35% of the population on average (1). It has been described that diet and sedentary lifestyle influence the development of the disease, with diets high in sugars and fats being important risk factors (2–10).

The high consumption of energy-dense foods, especially sugars and fats, is associated with body weight gain. In a systematic review of longitudinal studies in adolescents and early adulthood, a fast food diet pattern led to an excess odds of 23% (OR=1.23; 1.02-1.49) of annual BMI gain of 0.08 kg/m^2 (11). On the contrary, the consumption of dietary fiber in compliance with current recommendations

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(10-13 g/1000 kcals) (12) was associated with an avoidance of 0.44 kg/m^2 BMI gain. So far, it has not been defined whether the type of sugars or fats influences the deposition of fat in different body segments. Thus, the mechanisms by which body fat contributes to the development of metabolic liver disease is not established, although one study found that abdominal fat deposition is associated with greater metabolic imbalance (13). The objective of this study was to study the relationship between sugar and saturated fat consumption with liver and body fat.

2. Methods

Analytical cross-sectional study conducted from September 2021 to February 2023 in the outpatient clinic of the Department of Gastroenterology, Salvador Zubirán National Institute of Medical Sciences and Nutrition, a tertiary care center in Mexico City.

2.1. Population

Institutional staff were invited through an internal call. Eligible participants were adults over 18 years of both sexes without a previous diagnosis of fatty liver disease or alcohol use disorder. Respondents were scheduled for an intake interview where informed consent for participation in the study was obtained. Non-inclusion criteria were a diagnosis of cancer, heart disease, liver cirrhosis, hyperthyroidism, hypothyroidism, autoimmune diseases, bariatric or cosmetic surgery, metal prostheses, kidney failure, motor disability, amputations of limbs, use of medications that modify body composition (steroids, antipsychotics, antidepressants), and an alcohol consumption above 20 and 30 grams per day for women and men, respectively (14). Incomplete anthropometric assessments or vibration-controlled elastography were excluded from main analyses, but authorized 24-hour reminders from all participants were included.

2.2. Demographic, Clinical, and Biochemical Data

A questionnaire was applied to obtain the demographic characteristics of the study population. Self-reported questions enquired about comorbidities: diabetes mellitus, arterial hypertension, acute myocardial infarction, rheumatoid arthritis, dyslipidemia, hypothyroidism, insulin resistance, and tobacco use. Anthropometric measurements were obtained with a SECA model 274 stadiometer (Germany) for height (precision ±2 mm), and a scale with bioelectrical impedance (mBCA514), for weight (±100 g). Body mass index (BMI) was calculated as the ratio of kg and squared meters (kg/m^2) and categorized into universal BMI classes (<16.5, severely underweight; 16.5-18.5 underweight; 18.5-24.9 normal weight; 25-29.9, overweight; 30–34.9, class I obesity; 35-39.9, class II; and >40, class II obesity) (15). All participants had blood samples taken for blood cytometry, blood chemistry, liver enzymes, lipid profile, C-reactive protein (CRP), and insulin to calculate the homeostatic model to assess insulin resistance (HOMA-IR), which were analyzed with Beckman Coulter equipment (hematological DxH 1061 and series AU5800 for blood chemistry).

2.3. Body Composition Assessment

Multi-frequency bioelectrical impedance (BIA) analyses (11 frequencies) were performed using the SECA mBCA514 equipment. The data obtained from the BIA were: total fat mass in kg and percentage, and visceral fat in liters (L).

All measurements were made with the anthropometric method validated by the International Society for the Advancement of Kinathropometry and with standardized personnel using the Habitch technique. A Slim Guide caliper was used or body fold measurements (bicipital, tricipital, suprailiac and subscapular skinfold) and a Lufkin metal tape model W606PM for arm and waist circumferences. Body fat percentage was estimated from the sum of the skinfolds with the Durnin and Womersley formula (16).

2.4. Liver Fat Assessment

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The degree of steatosis and liver fibrosis was assessed by vibration-controlled transient elastography (Fibroscan® 502; Echosens, Paris, France) performed by trained physicians. The cut-off point used to determine steatosis was a controlled attenuation parameter (TAP) ≥275 dB/m (17) in accordance with European guidelines. Fasting of at least 3 hours was requested before measurements. Assessments that had 10 valid measurements and an IQR/med ≤30% were included.

2.5. Dietary Assessment

A 24-hour multi-step reminder (***supplementary material 1***) was used for dietary assessment. The analysis of the main nutrients, their types, and micronutrients was carried out with the Food Processor software v11.11. The analysis included quantification in grams of total sugars, added sugars, fructose and saturated fats, in addition to other types of nutrients. In total, 17 types of sugars, 5 types of fats, and total protein were quantified without distinction of their origin. It was estimated in terms of the amount of Kcal they represented for each nutrient, and as a percentage of average total energy. The 31 micronutrients were expressed per day in the corresponding and universal dietary unit of measurement.

2.6. *Sample Size*

The sample size was calculated as the difference between two proportions, considering an overall prevalence of fatty liver of 25.2% (18) and an effect size of 11% associated with the consumption of sugary drinks on liver fat (10). For this calculation, a confidence level of 95% and a statistical power of 80% were assumed. The minimum sample size needed to detect significant differences was 550 subjects. Calculations were performed using G*Power software, version 3.1.9.7.

2.7. Statistical Analysis

Descriptive data are presented as median and interquartile range (Q1-Q3) for quantitative variables and frequency and percentage for qualitative variables. The comparison of macro and micronutrient intake was made using the Mann-Whitney U test.

To determine the relationship between the intake of nutrients and liver and body fat (CAP, body mass index, body fat and waist circumference), different linear regression models were created for Kcal and each nutrient (carbohydrates, protein, fat, saturated fat, total sugars, added sugar and fructose, each one in grams and in percentage of energy). For each model the adjustment was made for age (quantitative), sex, BMI (quantitative), waist circumference (quantitative) and total Kcal (quantitative). The results were presented as the regression coefficient (β) with 95% confidence interval (95%CI). The variance inflation factor (VIF) was calculated to determine the presence of collinearity in the multivariable models, defined as a value greater than 10.

The three evaluation methods recommended in nutritional epidemiology were used to determine the degree of association (19) between Kcal, nutrient intake and hepatic steatosis (>275 dB/m): 1) degree of association between nutrient intake (quantitative) and hepatic steatosis; 2) nutrient intake distributed in quartiles, with quartile 1 as reference; and 3) the association between the total sugar consumption ≥10% (model 1) and saturated fat ≥7% (model 2) with hepatic steatosis. Both logistic regression models were adjusted for age (quantitative), sex, BMI (quantitative), waist circumference, and total kilocalories (quantitative).

The results of the models are presented as regression coefficient (β), standard error, Odds Ratio (OR), 95%CI of the OR and p-value. The assumptions of the models were evaluated by residual analysis. A value of $p< 0.05$ was considered as statistical significance. All analyses were performed using SPSS v21 software.

2.8. Ethical Procedures

Participants received and signed an informed consent form. In the event of not completing all evaluations, authorization was requested to include any completed evaluations according to the

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order of the studies. This study was approved by the institutional research ethics committee with registration number GAS-3794. The study was conducted under compliance with the Declaration of Helsinki, whilst maintaining anonymity, privacy and will of participants.

3. Results

A total to 583 eligible adults consented to participate in the study, of which 49 were not included for analysis as elastography was not available. Of the 534 participants, 61.4% (n=328) were female, and the median age was 41.5 (IQR: 29.0-52.0). The most frequently reported comorbidities were smoking (7.68%) and hypertension (3.98%); 30.7% (n=164) of the participants had normal weight (BMI 18-24.9), 41.9% (n=224) were overweight, 20.6% (n=110) were obese class 1, 4.9% were class 2 (n=26), and 0.9% (n=5) were class 3. A total of 493 participants had complete dietary analysis data. The flow of participants is depicted in Figure 1.

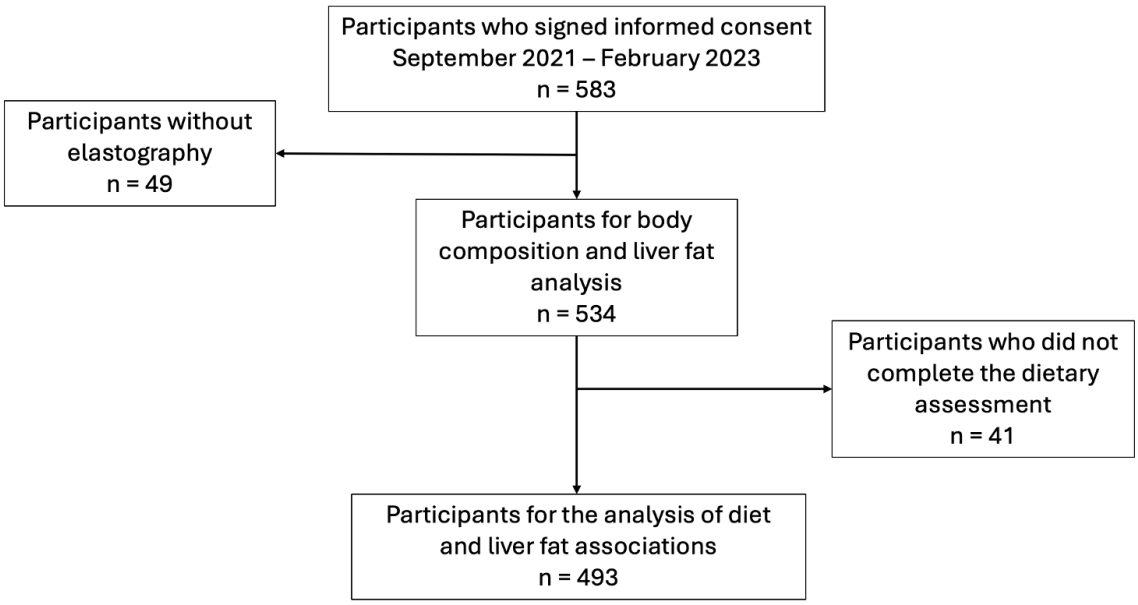

Figure 1. Flowchart of the participants.

Table 1 presents demographic data, body composition, and results of the most relevant laboratory studies of people with and without hepatic steatosis. People with steatosis had older age and more metabolic alterations and anthropometric indices than those without steatosis. In both groups, the frequency of acute myocardial infarction and arthritis was low (≤ 1 case due to comorbidity, per group). There were no differences between groups in urea nitrogen, urea, non-HDL cholesterol, liver function tests, or blood count.

Table 1. Clinical, body composition, and laboratory data among participants with and without hepatic steatosis.

	Total Sample (n=534)	No steatosis (n=307)	With steatosis (n=227)
Age (years)	41.5 (29.0-52.0)	36.0 (27.0-51.0)	45.0 (34.0-53.0)
Sex, n (%)			
Women	328 (61.4)	199 (64.8)	129 (56.8)
Smoking, n (%)	41 (7.68)	18 (5.86)	23 (10.13)
Comorbidities, n (%)			

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Diabetes	9 (1.69)	3 (0.98)	6 (2.64)
Hypertension	21 (3.93)	9 (2.93)	12 (5.29)
Dyslipidemia	3 (0.56)	0 (0.00)	3 (1.32)
Hypothyroidism	9 (1.69)	5 (1.63)	4 (1.76)
Insulin resistance	4 (0.75)	1 (0.33)	3 (1.32)
Body Composition			
Weight (kg)	70.5 (60.6-80.7)	64.2 (56.7-74.5)	77.8 (69.4-86.4)
BMI	27.1 (23.8-30.3)	25.1 (22.3-27.8)	29.4 (27.1-32.8)
Fat mass (kg)	25.1 (18.7-31.5)	20.8 (15.5-27-4)	29.6 (25.2-35.8)
Fat mass (%)	35.6 (29.0-42.2)	32.9 (26.2-38.7)	39.7 (32.6-44.7)
Visceral fat (L)	2.50 (1.80-3.50)	2.00 (1.50-2.70)	3.20 (2.48-4.20)
Waist circumference (m)	0.89 (0.80-0.99)	0.84 (0.76-0.92)	0.96 (0.89-1.04)
Bicipital skinfold (mm)	10.0 (7.0-14.0)	8.0 (6.0-12.0)	12.0 (9.0-17.0)
Triceps skinfold (mm)	17.0 (12.0-22.0)	15.0 (12.0-19.0)	20.0 (14.0-25.0)
Subscapular skinfold (mm)	23.0 (17.0-30.0)	19.0 (15.0-25.0)	28.0 (23.0-33.0)
Suprailiac skinfold (mm)	24.0 (17.0-30.0)	20.0 (15.0-27.0)	28.0 (24.0-35.0)
Biochemical data			
HOMA IR	1.58 (1.04-2.62)	1.25 (0.81-1.78)	2.45 (1.55-3.85)
Glucose (mg/dL)	89.0 (84.0-96.0)	87.0 (82.0-92.0)	93.0 (88.0-101.0)
Creatinine (mg/dL)	0.76 (0.66-0.89)	0.74 (0.66-0.87)	0.77 (0.66-0.92)
Cholesterol (mg/dL)	180.0 (155.0-206.0)	177.0 (155.0-202.0)	185.0 (156.0-217.0)
Low-density cholesterol (LDL-c; mg/dL)	110.0 (89.0-129.0)	105.0 (86.0-124.0)	116.0 (92.5-134.5)
LDL-c, Martin's method (mg/dL)	107.0 (86.0-126.0)	104.0 (83.0-122.0)	112.0 (88.0-133.0)
High-density cholesterol (HDL-c; mg/dL)	47.0 (40.0-56.0)	50.0 (43.0-59.0)	43.0 (37.0-51.0)
Triglycerides (mg/dL)	124.0 (90.0-174.0)	106.0 (79.0-142.0)	163.0 (114.5-233.0)
Total bilirubin (mg/dL)	0.64 (0.49-0.85)	0.64 (0.49-0.85)	0.63 (0.51-0.83)
Alanine aminotransferase (ALT; U/L)	21.6 (15.4-32.2)	18.1 (13.9-26.0)	26.5 (18.0-40.7)
Aspartate aminotransferase (AST; U/L)	19.6 (17.0-24.8)	19.1 (16.9-23.7)	21.4 (17.2-26.7)

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Gammaglutamyl transferase (U/L)	21.4 (15.1-35.3)	17.9 (13.1-26.7)	27.9 (19.1-42.6)
Alkaline phosphatase (U/L)	74.0 (61.5-88.0)	71.0 (58.0-85.0)	78.0 (66.0-92.0)
Albumin (g/dL)	4.42 (4.23-4.62)	4.46 (4.25-4.64)	4.38 (4.22-4.59)
Ultra-sensitive C-reactive protein (mg/dL)	0.15 (0.08-0.31)	0.13 (0.06-0.24)	0.20 (0.11-0.41)
Insulin (μIU/mL)	7.19 (4.95-11.38)	5.72 (3.96-8.15)	10.43 (6.91-16.02)
Platelets (10^3/μL)	249.0 (213.0-289.5)	251.0 (214.0-290.0)	242.5 (211.0-287.2)

Data presented as frequency and percentage (%) or as median and interquartile range (Q1-Q3).

The median CAP of the total sample was 263 dB/m (IQR: 211-304), while the median Kpa was 4.2 (IQR: 3.4-5.3). Of the 534 participants, 227 (42.5%, 95% CI: 38.3-46.7) had hepatic steatosis with a median CAP of 310 (IQR: 292-335) and Kpa 4.6 (IQR: 3.7-5.8). Of the 307 (57.5%) who did not have steatosis, the median CAP was 193 dB/m (IQR: 221-250) with Kpa of 3.3 (IQR: 4.0-4.8).

Table 2 presents the results of the comparisons of the intake of main nutrients and micronutrients between subjects with and without hepatic steatosis. Intake of energy, protein, total fat, and saturated fat was higher in people without hepatic steatosis. Although carbohydrate consumption was similar between both groups, monosaccharide consumption, particularly fructose, was higher in subjects without steatosis. An adjustment was made between the main nutrients by the kilocalories of consumption, thus obtaining the percentage of energy consumed in the form of each nutrient. It was observed that after this adjustment, there were no differences in consumption between the study groups. Regarding micronutrients, no differences were observed between those with and without hepatic steatosis.

Table 2. Comparison of energy and nutrient intake between participants with and without fatty liver collected with 24-hour recall.

	Total Sample (n=493)	No steatosis (n=282)	With steatosis (n=211)	P value
Kilocalories	1278.6 (1021.5-1533.9)	1326.2 (1050.7-1557.7)	1188.7 (991.8-1499.0)	0.009
Nutrients				
Carbohydrates (gr)	143.6 (67.5-211.7)	145.1 (53.6-216.96)	142.9 (78.0-208.5)	0.959
Protein (gr)	55.2 (44.7-69.0)	56.4 (46.5-70.5)	53.1 (40.8-65.8)	0.019
Fat (gr)	43.1 (30.6-75.2)	45.2 (32.5-86.6)	41.3 (28.8-62.3)	0.008
Kilocalories per carbohydrate	574.5 (270.1-846.7)	580.2 (214.4-867.8)	571.7 (312.2-834.1)	0.959
Carbohydrates (%)	53.7 (28.2-60.0)	51.8 (13.6-60.0)	55.4 (41.2-60.0)	0.253
Kilocalories from protein	221.0 (179.0-276.2)	225.9 (186.2-281.9)	212.3 (163.5-263.4)	0.019
Protein (%)	16.6 (15.2-18.9)	16.9 (15.4-19.4)	16.5 (15.0-18.6)	0.241
Kilocalories from fat	387.7 (275.7-676.8)	407.3 (293.1-780.1)	372.0 (259.2-560.9	0.008
Fats (%)	26.8 (23.2-49.6)	27.8 (23.2-70.5)	26.8 (23.1-39.4)	0.435
Saturated fat (gr)	16.8 (11.6-28.6)	17.7 (12.1-32.1)	15.8 (10.9-24.9)	0.022
Kilocalories per saturated fat	151.4 (104.3-257.9)	159.4 (109.1-288.7)	142.4 (98.4-224.9)	0.022
Saturated Fat (%)	10.6 (9.05-18.35)	10.6 (9.11-25.98)	10.5 (9.05-14.82)	0.587

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Monounsaturated fat (g)	3.91 (1.56-7.47)	3.98 (1.62-7.56)	3.50 (1.41-7.24)	0.516
Polyunsaturated fat (gr)	1.76 (0.81-3.36)	2.02 (0.90-3.39)	1.60 (0.78-3.34)	0.232
Trans fats (gr)	0.00 (0.00-0.12)	0.00 (0.00-0.08)	0.00 (0.00-0.13)	0.250
Types of carbohydrates				
Available CH (g)	134.2 (57.8-197.5)	134.3 (47.5-202.4)	134.2 (68.4-190.0)	0.886
Total sugars (g)	28.0 (17.98-55.39)	28.4 (18.61-52.03)	28.0 (16.68-55.99)	0.352
Added sugar (g)	7.53 (3.39-20.14)	7.53 (3.52-21.04)	7.67 (3.28-19.36)	0.649
Total sugars (%)	8.57 (6.43-14.73)	8.57 (6.43-14.64)	8.57 (6.43-16.14)	0.728
Added sugars (%)	2.44 (1.30-5.65)	2.43 (1.30-5.71)	2.49 (1.25-5.63)	0.845
Monosaccharides (gr)	7.80 (3.69-12.40)	8.44 (4.40-12.72)	6.76 (2.33-12.00)	0.037
Galactose (gr)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.794
Glucose (gr)	2.91 (1.52-4.71)	3.06 (1.99-4.67)	2.51 (0.99-4.72)	0.049
Fructose (gr)	4.86 (1.97-8.78)	5.42 (2.28-8.78)	4.35 (0.96-7.69)	0.017
Fructose (%)	1.56 (0.75-2.45)	1.65 (0.77-2.46)	1.50 (0.42-2.45)	0.266
Disaccharides (gr)	2.28 (1.18-3.75)	2.37 (1.41-3.75)	2.11 (0.71-3.62)	0.127
Lactose (gr)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.834
Maltose (gr)	0.00 (0.00-0.01)	0.00 (0.00-0.01)	0.00 (0.00-0.01)	0.404
Other HC (gr)	75.8 (15.3-142.7)	68.7 (15.1-143.3)	78.9 (15.6-138.6)	0.871
Net HC (gr)	134.4 (63.1-197.5)	134.4 (47.9-202.0)	134.4 (68.4-190.0)	0.938
Non-digestible HC (gr)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.00 (0.00-0.00)	0.833
Dietary fiber (gr)	8.57 (4.96-13.36)	8.56 (4.97-13.72)	8.57 (4.95-13.16)	0.807
Starch (gr)	0.00 (0.00-1.84)	0.00 (0.00-1.84)	0.00 (0.00-1.83)	0.418
Micronutrients				
Cholesterol (mg)	132.5 (83.4-217.4)	141.7 (94.1-221.1)	130.2 (78.8-205.9)	0.063
Folate (µg)	106.9 (58.7-157.1)	108.4 (59.4-159.3)	104.9 (54.9-156.9)	0.458
Folic acid (µg)	13.9 (0.00-62.3)	7.90 (0.00-66.1)	14.28 (0.00-60.1)	0.478
Vitamin B1 (mg)	0.48 (0.25-0.71)	0.49 (0.27-0.77)	0.45 (0.24-0.70)	0.158
Vitamin B2 (mg)	0.88 (0.58-1.28)	0.92 (0.58-1.33)	0.84 (0.57-1.26)	0.519
Vitamin B3 (mg)	12.0 (8.26-19.04)	12.4 (8.46-19.43)	11.6 (8.17-18.67)	0.468
Pantothenic acid (mg)	0.57 (0.25-1.02)	0.57 (0.19-1.02)	0.57 (0.27-1.05)	0.505
Vitamin B6 (mg)	0.85 (0.50-1.34)	0.92 (0.50-1.40)	0.79 (0.50-1.27)	0.158
Vitamin B12 (µg)	1.12 (0.41-2.37)	1.12 (0.37-2.45)	1.07 (0.41-2.35)	0.746
Vitamin C (mg)	42.3 (13.7-96.4)	40.1 (14.6-96.9)	42.9 (11.6-96.4)	0.952
Vitamin D (µg)	0.18 (0.00-0.79)	0.19 (0.00-0.74)	0.18 (0.00-0.82)	0.490
Vitamin E (mg)	0.48 (0.23-1.02)	0.50 (0.23-1.11)	0.47 (0.22-0.95)	0.521
Vitamin K (µg)	14.7 (5.03-51.57)	14.6 (5.19-52.50)	15.0 (4.62-49.98)	0.483
Biotin (µg)	3.59 (0.50-7.27)	3.68 (1.33-7.27)	3.49 (0.36-7.27)	0.401
Vitamin A (IU)	426.9 (135.4-856.3)	413.5 (124.9-863.7)	440.1 (155.9-849.0)	0.691
Calcium (mg)	916.6 (517.7-1311.1)	875.1 (453.7-1299.9)	958.4 (572.5-1320.7)	0.152
Copper (mg)	0.34 (0.21-0.53)	0.35 (0.21-0.54)	0.32 (0.21-0.51)	0.517
Iron (mg)	12.68 (7.52-18.20)	12.89 (7.35-18.56)	12.56 (8.04-17.71)	0.929
Magnesium (mg)	109.5 (62.3-163.3)	114.2 (62.6-173.3)	104.2 (61.6-157.5)	0.574
Boron (µg)	155.1 (0.00-529.19)	248.4 (0.00-529.19)	122.1 (0.00-529.19)	0.102
Chlorine (mg)	62.0 (0.0-150.0)	84.7 (0.0-150.0)	46.8 (0.0-150.0)	0.193
Chromium (µg)	1.39 (0.00-2.32)	1.39 (0.00-2.54)	1.05 (0.00-2.32)	0.380
Fluoride (mg)	0.00 (0.00-0.01)	0.00 (0.00-0.01)	0.00 (0.00-0.01)	0.841
Iodine (µg)	1.13 (0.15-33.51)	1.42 (0.15-36.74)	0.88 (0.12-19.79)	0.147
Manganese (mg)	0.32 (0.10-0.58)	0.32 (0.09-0.58)	0.32 (0.10-0.58)	0.657

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Molybdenum (µg)	0.00 (0.00-1.26)	0.00 (0.00-1.29)	0.00 (0.00-1.22)	0.833
Phosphorus (mg)	490.8 (305.7-724.6)	499.5 (300.4-772.5)	486.1 (309.1-684.1)	0.669
Potassium (mg)	1404.2 (845.2-1957.7)	1424.5 (807.9-1986.6)	1373.7 (888.0-1953.6)	0.833
Selenium (µg)	45.58 (30.59-70.16)	46.53 (30.36-72.71)	44.96 (30.59-68.29)	0.562
Sodium (mg)	2332.6 (1597.9-3033.6)	2335.7 (1589.2-3146.7)	2289.8 (1610.1-2968.2)	0.578
Zinc (mg)	3.73 (1.76-8.89)	4.33 (1.66-9.27)	3.47 (1.87-8.22)	0.496

Data presented as median and interquartile range (Q3-Q1). Comparisons were made using the Mann-Whitney U test. HC: carbohydrates.

Table 3 presents the results of multivariable linear regression models to determine the relationship between nutrient intake and the amount of liver fat, determined by CAP, body fat, visceral fat, and waist circumference. It was observed that as the percentage of carbohydrate consumption increases, the CAP increases and therefore the amount of liver fat (β=0.23, 95%CI: 0.02 to 0.45), in the same way it was found that the increase in the percentage of fat consumption is related to lower values of CAP (β=-0.22, 95%CI: -0.44 to -0.006). On the other hand, the consumption of Kcal, total fat, total sugars, added sugar and percentage of fat consumption is related to the increase in the percentage of body fat, while lower intakes of total carbohydrates (g), total protein (g), and percentages of carbohydrates are related to an increase in body fat. Waist circumference had a weak relationship with Kcal intake, and visceral fat was not associated with any nutrient intake.

Table 3. Multivariable linear regression models on the relationship of dietary intake with liver fat and body fat.

	CAP Model		**Fat model**		**Model Waist**		**Visceral fat model**	
Nutrient	B (95% CI)	p-value	B (95% CI)	p-value	B (95% CI)	p-value	B (95% CI)	p-value
Kilocalories‡	-0.006 (-0.016 to 0.004)	0.204	0.001 (0.044-0.000)	0.002	-0.001 (-0.002-0.000)	0.042	0.000 (0.000-0.000)	0.567
Carbohydrates (gr)*	0.060 (-0.001 to 0.121)	0.053	-0.002 (0.372- -0.007)	0.003	0.001 (-0.006-0.007)	0.862	0.000 (0.000-0.001)	0.589
Protein (gr)*	-0.082 (-0.434 to 0.271)	0.649	-0.009 (0.541- -0.039)	0.020	0.002 (-0.034-0.038)	0.900	-0.001 (-0.005-0.003)	0.682
Fat (gr)*	-0.129 (-0.266 to 0.007)	0.063	0.006 (0.318- -0.006)	0.017	-0.001 (-0.015-0.013)	0.845	0.000 (-0.002-0.001)	0.639
Saturated fat (gr)*	-0.325 (-0.706-0.056)	0.094	0.015 (0.347- -0.017)	0.047	-0.006 (-0.045-0.033)	0.747	-0.001 (-0.005-0.003)	0.701
Total sugars (gr)*	0.054 (-0.087-0.195)	0.453	0.005 (0.395- -0.007)	0.017	-0.009 (-0.023-0.006)	0.237	0.000 (-0.001-0.002)	0.812
Added sugar (gr)*	0.098 (-0.190-0.386)	0.506	0.018 (0.148- -0.006)	0.042	-0.012 (-0.041-0.017)	0.425	0.000 (-0.003-0.003)	0.834
Fructose (gr)*	-0.129 (-0.920-0.661)	0.748	0.026 (0.448- -0.041)	0.092	-0.052 (-0.132-0.029)	0.210	-0.001 (-0.009-0.008)	0.884
Protein (%)*	-0.285 (-1.336-0.766)	0.594	-0.019 (0.679- -0.106)	0.069	0.006 (-0.101-0.113)	0.912	-0.001 (-0.012-0.010)	0.862
Carbohydrates (%)*	0.234 (0.019-0.449)	0.033	-0.013 (0.155- -0.031)	0.005	0.000 (-0.022-0.022)	0.977	0.001 (-0.001-0.004)	0.247
Fat (%)*	-0.220 (-0.435-0.006)	0.044	0.014 (0.133- -0.004)	0.032	0.000 (-0.022-0.022)	0.995	-0.001 (-0.04-0.001)	0.263
Saturated Fat (%)*	-0.557 (-1.145-0.031)	0.063	0.037 (0.145- -0.013)	0.086	-0.003 (-0.064-0.057)	0.910	-0.003 (-0.010-0.003)	0.329
Total sugars (%)*	0.165 (-0.293-0.622)	0.480	0.009 (0.651- -0.029)	0.047	-0.028 (-0.075-0.018)	0.236	0.000 (-0.005-0.005)	0.954
Added sugar (%)*	0.211 (-0.649-1.072)	0.630	0.034 (0.355- -0.038)	0.106	-0.030 (-0.118-0.058)	0.503	-0.001 (-0.011-0.008)	0.791
Fructose (%)*	-0.719 (-3.298-1.859)	0.584	0.049 (0.654- -0.167)	0.266	-0.158 (-0.422-0.106)	0.240	-0.005 (-0.033-0.023)	0.725

‡: Models adjusted for age, sex, BMI, waist circumference. *: Models adjusted for age, sex, BMI, waist circumference and total Kcal.

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Table 4 presents the results of logistic regression models for determining the association of nutrient intake with hepatic steatosis. It was observed that carbohydrate intake was associated with a higher probability of having fatty liver: each gram of consumption was associated with a 0.3% increase in the probability of having fatty liver (β=0.003, p=0.03). We performed a regression model adjusting the variable of carbohydrate consumption per 15 grams, showing that the probability of having fatty liver increases by 5% (β=0.51, OR=1.053, 95%CI:1.006-1.102, p=0.03) for every 15 grams.

Table 4. Multivariable logistic regression models on the relationship between dietary intake and the presence of hepatic steatosis.

Nutrients	B	Standard Error	OR (95%CI)	P value
Kilocalories‡	0.000	0.000	1.000 (0.999-1.000)	0.183
Carbohydrates (gr)*	0.003	0.002	1.003 (1.000-1.006)	0.028
Protein (gr)*	-0.013	0.009	0.988 (0.970-1.005)	0.165
Fat (gr)*	-0.007	0.003	0.993 (0.986-1.000)	0.048
Saturated fat (gr)*	-0.017	0.010	0.983 (0.965-1.002)	0.072
Total sugars (gr)*	0.005	0.003	1.005 (0.998-1.011)	0.187
Added sugar (gr)*	0.004	0.007	1.004 (0.990-1.018)	0.555
Fructose (gr)*	-0.004	0.019	0.996 (0.960-1.035)	0.851
Protein (%)*	-0.032	0.026	0.969 (0.920-1.019)	0.222
Carbohydrates (%)*	0.011	0.005	1.011 (1.000-1.021)	0.050
Fat (%)*	-0.009	0.005	0.991 (0.980-1.001)	0.087
Saturated Fat (%)*	-0.023	0.015	0.977 (0.949-1.005)	0.110
Total sugars (%)*	0.013	0.011	1.013 (0.991-1.035)	0.245
Added sugar (%)*	0.004	0.021	1.004 (0.965-1.046)	0.835
Fructose (%)*	-0.015	0.062	0.985 (0.872-1.112)	0.802

‡: Models adjusted for age, sex, BMI, waist circumference. *: Models adjusted for age, sex, BMI, waist circumference and total Kcal.

The supplementary table 1 presents the results of the logistic regression models, considering the distributions of nutritional intake by quartiles. No association was observed between higher intake and increased risk of hepatic steatosis. For the analysis of this section, participants were classified as those who had consumption within the recommendations on free sugars <10% (20) and saturated fat consumption <7% (20).

Consumption of sugars ≥10% (n=217, 44%, OR=1.04, 95%CI: 0.67-1.60, p=0.86) or saturated fats ≥7% (n=451, 91.5%, OR=0.92, 95%CI: 0.43-1.96, p=0.83) was not associated with the presence of hepatic steatosis in quartile analyses.

Finally, the frequency of subjects with combined intakes of fats, total sugars, added sugars, and fructose was explored by considering those within quartile 4 of each of the nutrients as high consumption. The frequency of combined high consumption was low for all combinations: fats with total sugars (n=7, 1.3%), fats with added sugars (n=6, 1.1%) and fat with fructose (n=20, 3.7%).

4. Discussion

The objective of this study was to analyze the association of the consumption of nutrients, sugars, and saturated fat with liver and body fat in the general population. The results of our study showed that only carbohydrates were related to liver fat, and that, for each 15-gram serving of

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carbohydrates, the probability of developing hepatic steatosis increases by 5%. Furthermore, the highest consumption of energy from fats and sugars was associated with body fats, without having an effect on waist circumference or visceral fat. These findings give insights into how diet can influence body fat accumulation but do not conclude its effect on fat accumulation at the central level.

The WHO (20) recommends that the usual diet should contain less than 10% free sugars, as well as the least amount of saturated fats. The National Cholesterol Education Program (21) also suggests that the non-atherogenic diet contains less than 7% saturated fat; however, in this cross-sectional study, we found no association with hepatic steatosis that could be attributed to consumption above the aforementioned values, reason why specific nutritional recommendations should be established both to prevent and to treat the disease.

One of the limitations of this study was that a 24-hour reminder (R24) was applied on a single occasion to obtain dietary intakes. The usual dietary intake can be estimated more accurately through repeated R24. The fact that people with hepatic steatosis had lower caloric intake despite having a higher frequency of metabolic comorbidities and higher body weight suggests that the dietary estimate was not as accurate or that the respondents were aware of their metabolic comorbidity and had made lifestyle changes to correct it. This is a limitation of all cross-sectional studies where it is not possible to establish causality.

Another limitation is that we could not establish a consumption pattern with respect to schedules. It is thought that a possible factor that allows the reversal of lipid accumulation in the blood and liver is intermittent fasting (22) especially with high-fat diets. Analysis of the diet with the aforementioned software did not provide the amount of fats such as n-3, n-6 fatty acid, polyphenols, or carotenoids in the diet, which have also been noted as metabolic protective factors (23). Regarding dietary fiber, our results showed that the consumption of this nutrient is below the minimal intake recommendation, which limited the possibility of evaluating associations with outcomes despite the fact that its protective role has been described.

The inconsistent associations in the different studies that evaluate the main nutrients and micronutrients with hepatic steatosis may be due to the fact that aspects such as the radius of the nutrients, their origin, or the consumption pattern of each individual are not taken into account. In addition to this, there are most likely additional factors, with the microbiota, genetics and epigenetics (as in the case of fats), that modulate this association (23). Dietary analysis in such disease has taught us that isolated nutrients should not be interpreted causally, as it is difficult to evaluate the effect of nutrition if the interactions between the different components of nutrition are not taken into account. Therefore, future studies could evaluate the association between diet and steatosis from the point of view of nutritional geometry (24). Noteworthy, the latter has not allowed for the elucidation of the optimal balance between nutrients, foods, diets, appetite regulation, food matrices and homeostatic physiology despite proposing a multilevel analysis based on an axis such as the prioritization of protein (24). One conclusion of studies that have used nutritional geometry is that nutrient balance affects the relationship between food, energy intake, and various physiological functions in a variety of ways (23).

In this study, the relationship between nutrient consumption and liver and body fat was sought and not the relationship between types or groups of foods, nor between dietary patterns with liver or body fat, which can be very relevant in liver fat accumulation.

Another limitation is that the diagnosis of hepatic steatosis was based on the CAP, which, although more sensitive than ultrasound, is not very sensitive for mild degrees of steatosis, in addition to the fact that the cut-off points to define steatosis vary in the literature.

Once more than 6 kcals/day (25 kJ/day) from carbohydrates and 2 kcals/day (10 kJ/day) from protein are cumulatively ingested during the aging process is likely one of the most sensible explanations regarding the development of metabolic liver disease, reflecting that the nutrient ratio is just as important as the energy content (23). This is consistent with the controversy that exists about the consumption of sugary drinks (soft drinks in particular) as possible risk or protective factors. However, it has been described that the consumption of fructose from these products from 4 g/kg/day

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for one week increases liver fat content since it behaves as an inducer of lipogenesis and intestinal endotoxins (22).

Generalizability of the study findings could be limited to populations with similar characteristics to workers in the healthcare sector of Mexico, who are known to be young on average, low physical activity levels, with low fiber intake and excessive processed meats and sweetened beverages consumption, undergoing transition towards higher dietary fats, carbohydrates, and sodium (25). The application of multiple non-inclusion criteria in this study may further limit the generalizability of findings to other populations.

5. Conclusion

Consumption of free sugars and saturated fats was associated with body fat but not liver fat. The diet high in energy, fat and added sugars, but low in carbohydrates (including fiber) and protein, was found to be associated with body fat. To explain the possible relationship of diet with liver fat, it is necessary to consider a greater registration of the diet prospectively, as well as to analyze it with a focus on nutritional geometry.

Supplementary Materials: The following supporting information is available as supplementary material. Table S1: Multivariable logistic regression models on the relationship of dietary intake quartiles with the presence of hepatic steatosis. File 1: 24- hour recall format.

Author Contributions: SEMV: Conceptualization, Investigation, Methodology, Project administration, Supervision, Writing, Original Draft, Review and Editing, Funding adquisition, Visualization. AKG: Conceptualization, Methodology, Data curation, Formal Analysis, Supervision, Writing, Original Draft, Review and editing, Visualization. CMV: Methodology, Investigation, Supervision, Writing, Original Draft, Review and editing, Visualization. JMG: Methodology, Formal Analysis, Writing, Original Draft, Review and Editing, Visualization. IGJ: Methodology, Supervision, Review and editing, Visualization. LUD: Conceptualization, Supervision, Review and editing, Funding adquisition, Project administration, Visualization.

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Data availability statement: The data presented in this study are available on request from the corresponding author. The data are not publicly because they contain information that could compromise the privacy of research participants.

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