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Artículo Original

Association of liver enzymes and lipid profile with body fat distribution in Mexican university students

Edith Valbuena-Gregorio[1, 2], Marco Antonio López-Mata[1], Francisco Javier Olivas-Aguirre[3], Adriana Alejandra Márquez-Ibarra[1], Brianda Joanna Armenta-Guirado[1], Andrea Arreguín Coronado[4].

Abstract: Association of liver enzymes and lipid profile with body fat distribution in Mexican university students. Introduction: Evidence suggests that the type of adipose tissue distribution contributes to a higher risk of developing cardiovascular diseases. It has been observed that visceral adipose tissue is highly lipolytic, and lipid distribution is primarily directed towards the liver, resulting in an excessive flow of fatty acids that can trigger lipid alterations and a high risk of hepatic steatosis. **Objective:** This study aimed to find the association of liver enzymes and lipid profile with body fat distribution in Mexico university students. **Materials and methods:** Cross-sectional, descriptive, and correlational study with a sample of university students aged 18 to 26 years. Anthropometric measurements, a 24-hour dietary recall (R24h), Pittsburgh sleep quality index, body fat determination, DXA-derived visceral adipose tissue (cm^2) and subcutaneous adipose tissue (g), and blood samples were collected to determine lipid profile (low-density lipoprotein [LDL], high-density lipoprotein [HDL], very low-density lipoprotein [VLDL], triglycerides [TG], total cholesterol [TC]) and liver enzymes (aspartate aminotransferase [AST], alanine aminotransferase [ALT], and gamma-glutamyl transferase [γ-GT]). **Results:** We found a positive association between the type of fat distribution and LDL, VLDL, TC, TG, ALT, and γ-GT, indicating that higher visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), and body mass index (BMI) increase the concentrations of these biochemical parameters. In contrast, HDL showed a negative association, decreasing with an increase in VAT, SAT, and BMI. **Conclusions:** Body fat distribution (VAT and SAT) influences the lipid (LDL, VLDL, TC, TG, and HDL) and liver (ALT and γ-GT) levels of university students, which may indicate metabolic alterations. **Arch Latinoam Nutr 2025; 75(3):163-174.**

Keywords: adipose cell, visceral fat, subcutaneous fat, DXA, cardiovascular disease, young adult.

Resumen: Asociación de las enzimas hepáticas y el perfil lipídico con la distribución de la grasa corporal en estudiantes universitarios mexicanos. Introducción: La evidencia sugiere que el tipo de distribución del tejido adiposo contribuye a un mayor riesgo de desarrollar enfermedades cardiovasculares. Se ha observado que el tejido adiposo visceral es altamente lipolítico, y la distribución de lípidos se dirige principalmente hacia el hígado, lo que resulta en un flujo excesivo de ácidos grasos que puede desencadenar alteraciones lipídicas y un alto riesgo de esteatosis hepática. Objetivos: Este estudio tuvo como objetivo determinar la asociación entre las enzimas hepáticas y el perfil lipídico con la distribución de grasa corporal en estudiantes universitarios de México. **Materiales y métodos:** Estudio transversal, descriptivo y correlacional con una muestra de estudiantes universitarios de 18 a 26 años. Se realizaron mediciones antropométricas, un recordatorio dietético de 24 horas (R24h), índice de calidad del sueño de Pittsburgh, determinación de grasa corporal, tejido adiposo visceral (cm^2) y tejido adiposo subcutáneo (g) derivados de DXA, y se tomó muestra de sangre para determinar el perfil lipídico (lipoproteínas de baja densidad [LDL], lipoproteínas de alta densidad [HDL], lipoproteínas de muy baja densidad [VLDL], triglicéridos [TG], colesterol total [CT]) y enzimas hepáticas (aspartato aminotransferasa [AST], alanina aminotransferasa [ALT] y gamma-glutamil transferasa [γ-GT]). **Resultados:** Encontramos una asociación positiva entre el tipo de distribución de la grasa y LDL, VLDL, TC, TG, ALT y γ-GT, indicando que un mayor tejido adiposo visceral (TAV), tejido adiposo subcutáneo (TAS) e índice de masa corporal (IMC), aumentan las concentraciones de los parámetros bioquímicos. En cambio, la HDL mostró una asociación negativa, disminuyendo con el aumento del TAV, el TAS e IMC. **Conclusiones:** La distribución de la grasa corporal (TAV y TAS) influye en los niveles lipídicos (LDL, VLDL, TC, TG y HDL) y enzimas hepáticas (ALT y γ-GT) de los estudiantes universitarios, lo que puede indicar alteraciones metabólicas.. **Arch Latinoam Nutr 2025; 75(3): 163-174.**

Palabras clave: célula adiposa, grasa visceral, grasa subcutánea, DXA, enfermedad cardiovascular, adulto joven.

Introduction

Overweight and obesity are abnormal or excessive fat accumulation, posing a health risk (1). The accumulation, distribution, storage, and differences in the mobilization of adipose tissue

[1]Department of Health Sciences, University of Sonora, Campus Cajeme. [2]Department of Health and Nutrition, International Iberoamerican University. [3]CONAHCyT National Laboratory of Body Composition and Energetic Metabolism (LaNCoCoME). Autonomous University of Baja California. [4]Faculty of Nursing and Nutrition, Autonomous University of San Luis Potosí. Corresponding author: Andrea Arreguín Coronado, e-mail: andrea.arreguin@uaslp.mx

play a significant role in the development of cardiovascular diseases (2,3). Although vascular diseases typically manifest their signs and symptoms during adulthood, some of these issues may originate from early life stages (4). University students are not exempt from this problem, as they face increased responsibilities and autonomy, including independence in decision-making. These decisions encompass not only academic matters but also dietary choices. Furthermore, changes in lifestyle, such as the number of hours of sleep, type of physical activity, and food consumption, negatively impact their body composition, potentially leading to overweight or obesity (5).

Adipose tissue is considered the organ with the greatest plasticity, as it can modify its size in response to environmental factors such as age, physical activity, diet (6), sleep quality, and even genetic factors (7). The roles of adipose tissue include fat storage, which is influenced by two processes: cellular hypertrophy, referring to the increase in the volume of pre-existing adipose cells, and hyperplasia, referring to the formation of new adipocytes (8). Several compartments related to the storage of fat are recognized in the body, including the distribution of subcutaneous adipose tissue (SAT), which is subdivided into the superficial layer of SAT (SATs) and a deeper layer (SATp). Another compartment is the gluteofemoral fat, distributed in the lower part of the body. These two compartments are the most abundant in the body (approximately 80%). Additionally, visceral adipose tissue (VAT, composed of omental and mesenteric fat) can represent 10% of total fat in women and up to 25% in men (9). VAT has been reported to be highly lipolytic, with the liver as its primary target organ. Excessive stimulation in the release of lipids by VAT can cause an excessive flow of non-esterified fatty acids, which may lead to hypertriglyceridemia due to the overproduction of very low-density lipoproteins (VLDL) and a subsequent increase in low-density lipoproteins (LDL), predisposing to the metabolic dysfunction-associated steatotic liver disease (MASLD) (10). MASLD can cause cellular damage, often associated with elevated levels of intracellular liver enzymes such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (γ-GT) (11), and increased mortality related to cardiovascular diseases. In contrast, subcutaneous adipose tissue has been associated with a protective effect against the development of cardiometabolic diseases (12).

Previous studies agree that the distribution of fat and dysfunction of adipose tissue are related to the development of obesity and associated diseases such as insulin resistance, cardiovascular diseases, diabetes, and certain types of cancer (13). In this regard, university students in northwestern Mexico represent a high-risk group, as it has been reported that 32.6% of their total energy intake derives from processed foods (PF) and ultra-processed foods (UPF) (14). Specifically, the northern region of Mexico has a higher energy intake from these sources compared to the central and southern regions of the country, in addition to being the region with the highest prevalence of overweight and obesity in adults (15). Therefore, this study aimed to find the association of liver enzymes and lipid profile with body fat distribution in Mexico university students.

Materials and methods

Study Design

This cross-sectional, descriptive, and correlational study included university students enrolled at the University of Sonora, aged between 18 and 26 years. The objective was to evaluate the association between liver enzymes and lipid profile with body fat distribution. The study period was from October 2023 to November 2024. A total of 227 participants were recruited; after applying elimination criteria (incomplete records, incomplete anthropometric measurements, or outliers), the total sample was 219 participants. Exclusion criteria were as follows: undergoing medical treatment, having any body metal implants, consumption of diuretics, engagement in strenuous exercise the day before body composition evaluation, adherence to a prior dietary regime, pregnancy, lactation, and chronic diseases such as type 1 or type 2 diabetes, hypertension, MASLD, hypertriglyceridemia, and previously diagnosed hyperlipidemia.

Clinical Data and Laboratory Tests

All participants were scheduled for blood sample collection following a 12-hour fasting period. Blood samples were taken from the antecubital region of the arm. The obtained samples were centrifuged at 3500 rpm for 15 minutes to separate the serum. All serum samples were stored in cryovials in a freezer at -20°C until analysis. Biochemical analyses were conducted using a semi-automated clinical chemistry analyzer (SPIN-LAB, photometer-quartz-iodide lamp) manufactured in the Netherlands. All results were obtained using normal and pathological controls from Spinreact®.

Lipid Profile

Lipid profile parameters were determined following the Spinreact® kit protocols: triglycerides [glycerol phosphate dehydrogenase-peroxidase method], total cholesterol [cholesterol oxidase-peroxidase method], and high-density lipoprotein cholesterol (HDLc) [direct enzymatic method]. Very low-density lipoprotein (VLDL) was estimated using the equation: VLDL = triglycerides (mg/dL)/5, and low-density lipoprotein (LDL) using the Friedewald equation: [LDL = total cholesterol - HDL - VLDL]. Lipid biomarkers were reported in mg/dL and were analyzed as continuous variables.

Liver Enzymes

The liver enzymes determined were alanine aminotransferase (ALT) [NADH. Kinetic UV method], aspartate aminotransferase (AST) [NADH. Kinetic UV method], and gamma-glutamyltransferase (γ-GT) [carboxylated substrate-Kinetic method] using Spinreact kits. Liver enzymes biomarkers are reported in U/L and were analyzed as continuous variables.

Body Fat Distribution

Body fat distribution was assessed using dual-energy X-ray absorptiometry (DXA) equipment (PRIMUS model, OsteoSys Software Version 3.1, Osteosys Co., Ltd., Guro-gu, Seoul, Korea). DXA allowed for the measurement of visceral adipose tissue (VAT) by area (cm^2) and subcutaneous adipose tissue (SAT) mass (g). VAT and SAT were determined by in a central abdominal ROI by positioning the upper pelvic marker above the iliac crests. The lateral pelvic markers were adjusted by the edge so that it crossed the femoral necks. The leg ROI was defined to isolate one lower limb, the android ROI was set to delimit the abdominal region, and the gynoid ROI for the gluteal-femoral region. The cutoff points for ratio VAT/SAT were based on those used by Fujioka et al (16). For the SAT, no cutoff points associated with health benefits have been established (17). The DXA scan also provided the fat mass index, the fat mass (kg) ratio to area (m^2). Classification was based on the cutoff points proposed by Messina et al (18).

Dietary Assessment

A 24-hour recall (R24h) was used to obtain detailed information regarding the foods and beverages consumed the previous day (type, quantity, preparation method, etc.), with the technique described by Sanjur employed for this purpose (19). The SMAE 5th edition database was used to obtain the energy and nutrient intake of the participants (20).

Anthropometry

Weight and height were measured for all participants using a Seca scale model 284 with a capacity of 300 kg (± 50 g margin of error) and a stadiometer 30-220 cm (± 2 mm margin of error), following standardized techniques (21).

Pittsburgh Sleep Quality Index (PSQI)

The PSQI, validated in Spanish for the Mexican population by Jiménez-Genchi et al., was used. The PSQI evaluates sleep conditions over the past month. The cutoff points proposed were those suggested by Jiménez-Genchi et al (22).

International Physical Activity Questionnaire (IPAQ)

The short version of the IPAQ consisted of seven questions regarding the frequency, duration, and intensity of physical activity (moderate and intense) performed in the last seven days and walking and sitting time on a workday. Data obtained were classified according to the scoring described by Mantilla et al (23).

Statistical Analysis

The total population of university students was 1400, which was the basis for calculating the sample size using the G Power program (2010-2020 Heinrich-Heine-Universität Düsseldorf)

(24), through an a priori analysis with Fisher's exact test for two tails for the independent variable HDL, while the rest were calculated with one tail. The probability of H1 was determined using a previously reported ratio by Mahmoud et al. (25) of 0.22, an alpha error of 0.05, and a beta error of 0.95, with a power of 95.1%, resulting in a sample size of 219 subjects. Descriptive tests assessed data normality using skewness, kurtosis, and the Shapiro-Wilk test. For mean comparisons, t-test and analysis of variance (ANOVA) or their non-parametric alternatives (Wilcoxon and Kruskal-Wallis, respectively) were used; the Bonferroni post hoc test was considered to determine the group evoking the difference. Categorical variables were analyzed using contrast tests (Chi-square). Implausible energy values (n=3) (<500 kcal and >6500 kcal) were eliminated and replaced by the population's mean energy (1844.14 kcal).

The association between lipid and hepatic profiles with body fat distribution in university students was evaluated using multiple linear regression. A crude model and an adjusted model for confounding variables: age (years), sex (male or female), physical activity (METs), energy (kcal/day), and sleep quality (points), identified considering the literature and directed acyclic diagrams (26) (Supplementary Figures 1 and 2). Independent models were created for each response variable. All analyses were performed using STATA version 17 (StataCorp. 2021. Stata Statistical Software: Release 17. College Station, TX: StataCorp LLC), setting a statistical significance level at α=0.05.

Ethical Statements

The study was conducted following the Helsinki Declaration of 1975, as revised in 2000. The study protocol was approved by the Ethics Committee of International Iberomerican University (CR-270). Everyone provided written informed consent before the laboratory test.

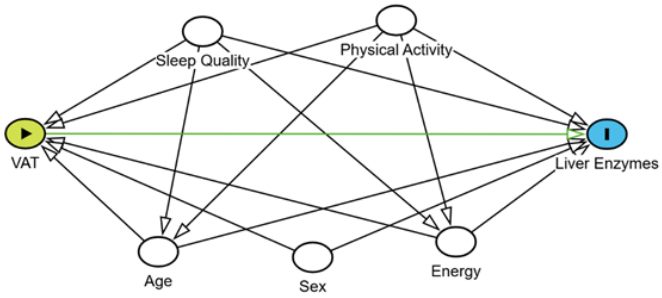

Figure 1. Directed Acyclic Graph (DAG) of the association between body fat distribution and liver enzymes.

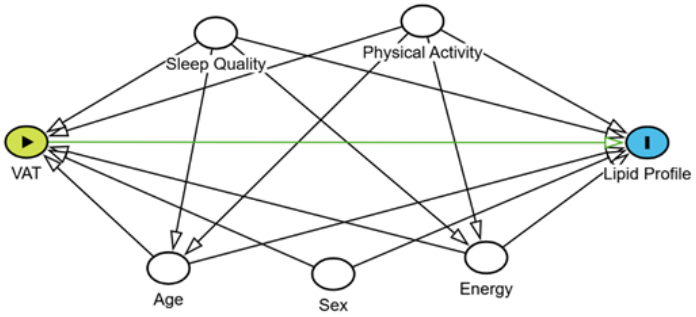

Figure 2. Directed Acyclic Graph (DAG) of the association between body fat distribution and lipid profile.

Results

A total of 219 university students were evaluated, 72.60% were females. The mean age was 20.15 ± 1.59 years for males and 20.47 ± 2.10 years for females. Daily energy intake was higher in males compared to females (2237.18 ± 974.96 kcal/day vs. 1695.21 ± 723.34 kcal/day; p < 0.000). The values of SAT mass vs. VAT area showed differences by sex. SAT mass was similar between males and females (419.33 ± 73.21 g vs. 404.06 ± 61.21 g; p = 0.1207), whereas the VAT area was significantly lower in males compared to females (76.46 ± 76.67 cm^2 vs. 107.11 ± 66.44 cm^2; p = 0.0039). Similarly, the fat mass index (FMI)

presented significant differences between both groups (males: 4.87 ± 3.32 kg/m², females: 6.86 ± 3.39 kg/m²; $p < 0.001$). Detailed population characteristics are presented in Table 1.

A sex-stratified analysis of body fat distribution by FMI in relation to lipid profile, liver enzymes, VAT, and SAT is shown in Table 2 y 3. In females (Table 2), there was a significant increase ($p < 0.05$) in the values of VLDL, HDL, triglycerides (TG), and γ-GT among overweight females compared to the underweight group. In males (Table 3), a significant increase ($p < 0.05$) in LDL and total cholesterol (TC) concentrations was observed in the obesity group compared to the overweight group. A significant difference ($p<0.05$) was observed with higher LDL and TC levels in men with normal FMI compared to the overweight group. Regarding ALT, a significant increase ($p < 0.05$) was found in the obesity group compared to the underweight and overweight groups. For γ-GT, a significant increase ($p < 0.05$) was observed in the obesity group compared to the underweight and normal weight groups. Significant differences in the amount of adipose tissue ($p < 0.05$) were found between all groups for both VAT and SAT in females and males, showing an increase in these deposits as individuals moved from normal weight to obesity.

Regarding the association between lipid profile and liver enzymes with body fat distribution, a positive and statistically significant association was observed between VAT and SAT with the lipid profile (LDL, VLDL, TC, and TG), suggesting that higher VAT and SAT increase the concentrations of these lipoproteins. For HDL levels, the association was inversely significant with VAT and SAT, indicating that lower HDL concentrations correlate with higher VAT and SAT volume and mass,

Tabla 1. Characteristics of the population (n= 219)

Variables	Females n=159	Males n=60	*p*-value
	Mean ± SD	Mean ± SD	
Age (years)	20.47 ± 2.10	20.15 ± 1.59	0.2756
Physical activity (METs)	1630.31 ± 2108.88	2208.02 ± 2767.72	0.0998
Sleep quality (points)	7.44 ± 2.75	7.30 ± 2.45	0.7181
BMI (kg/m²)	25.03 ± 5.02	26.54 ± 4.93	0.0470
Energy (kcal/day)	1695.21 ± 723.34	2237.18± 974.96	0.0000
BMI (%)			0.1610
Underweight	6.92	1.67	
Normal	49.69	40.00	
Overweight	28.93	40.00	
Obesity	14.47	18.33	
SAT mass (g)	404.06 ± 61.21	419.33 ± 73.21	0.1207
VAT area (cm²)	107.11 ± 66.44	76.46 ± 76.67	0.0039
Ratio VAT/SAT	1.20 ± 0.62	0.73 ± 0.66	0.0000
Classification of the VAT/SAT Ratio (%)			0.0000
Visceral Fat (≥ 0.4)*	89.94	61.67	
Subcutaneous Fat (< 0.4)*	10.06	38.33	
FMI (kg/m²)	6.86 ± 3.39	4.87 ± 3.32	0.0001

DE: standard deviation; MET: metabolic equivalent; BMI: body mass index; FMI: fat mass index; SAT: subcutaneous adipose tissue; VAT: visceral adipose tissue; FMI: fat mass index. *Cut-off point according to Fujioka et al. (16) Classification of the VAT/SAT ratio. *p*-value defined as alpha < 0.05.

Tabla 2. Body Fat Distribution According to FMI by Lipid Profile, Liver Enzymes, Visceral and Subcutaneous Adipose Tissue in Female (n=159)

	Underweight n= 51	Normal n= 73	Overweight n= 26	Obesity n= 9	*p*-value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Lipid profile					
LDL (mg/dL)	77.24 ± 30.22[a]	74.21 ± 28.51[a]	80.30 ± 25.50[a]	100.93 ± 27.56[a]	0.0681
VLDL (mg/dL)	13.38 ± 5.56[a]	16.87 ± 10.21[ab]	20.92 ± 11.36[b]	22.40 ± 14.59[ab]	0.0029
HDL (mg/dL)	69.82 ± 19.59[a]	64.88 ± 16.86[ab]	58.07 ± 14.60[b]	58.00 ± 10.85[ab]	0.0240
TC (mg/dL)	160.45 ± 35.01[a]	155.97 ± 32.87[a]	159.30 ± 25.66[a]	181.33 ± 31.16[a]	0.1782
TG (mg/dL)	67.01± 27.76[a]	84.36 ± 50.97[ab]	104.73 ± 56.70[b]	112.00 ± 72.65[ab]	0.0028
Liver enzymes					
AST (U/L)	18.33 ± 11.24[a]	18.50 ± 6.96[a]	18.11 ± 6.45[a]	19.77 ± 3.89[a]	0.9640
ALT (U/L)	9.58 ± 4.35[a]	11.06 ± 6.84[a]	12.15 ± 4.96[a]	10.22 ± 4.26[a]	0.2698
γ-GT (U/L)	10.84 ± 3.41[a]	12.71 ± 7.56[ab]	19.50 ± 26.64[b]	17.77 ± 5.60[ab]	0.0187
VAT / SAT					
VAT area (cm^2)	39.17 ± 22.68[a]	108.98 ± 26.18[b]	187.73 ± 29.23[c]	244.00 ± 58.91[d]	0.0000
SAT mass (g)	352.01 ± 27.90[a]	399.97 ± 37.21[b]	473.96 ± 33.40[c]	530.22 ± 44.52[d]	0.0000

FMI: fat mass index; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; HDL: high-density lipoprotein; TC: total cholesterol; TG: triglycerides; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; γ-GT: γ-Gamma glutamyltransferase; VAT: Visceral adipose tissue; SAT: subcutaneous adipose tissue. SD: standard deviation. [a - d] Different literals per column indicate significant difference with Bonferroni post hoc test. *p* value defined as alpha < 0.05.

Tabla 3. Body Fat Distribution According to FMI by Lipid Profile, Liver Enzymes, Visceral and Subcutaneous Adipose Tissue in Male (n=60)

	Underweight n= 18	Normal n= 26	Overweight n= 9	Obesity n= 7	*p*-value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Lipid profile					
LDL (mg/dL)	94.4 ± 52.43[ab]	93.61 ± 33.99[ab]	66.42 ± 32.57[a]	139.45 ± 21.54[b]	0.0063
VLDL (mg/dL)	15.93 ± 7.90[a]	17.01 ± 7.99[a]	17.15 ± 7.86[a]	22.74 ± 13.22[a]	0.3659
HDL (mg/dL)	56.94 ± 17.70[a]	55.30 ± 13.86[a]	56.88 ± 18.81[a]	49.57 ± 9.81[a]	0.7432
TC (mg/dL)	167.27 ± 48.60[ab]	165.92 ± 30.78[ab]	140.44 ± 47.51[a]	211.85 ± 29.67[b]	0.0078
TG (mg/dL)	79.66 ± 39.53[a]	84.96 ± 40.01[a]	85.77 ± 39.30[a]	113.57 ± 66.26[a]	0.3697
Liver enzymes					
AST (U/L)	16.72 ± 6.56[a]	20.03 ± 8.84[a]	18.44 ± 6.93[a]	23.14 ± 7.71[a]	0.2765
ALT (U/L)	10.55 ± 4.23[a]	15.07 ± 10.69[ab]	10.77 ± 3.92[a]	24.71 ± 12.56[b]	0.0038
γ–GT (U/L)	14.72 ± 4.79[a]	15.03 ± 5.82[a]	17.66 ± 7.10[ab]	26.14 ± 9.51[b]	0.0007
VAT / SAT					
VAT area (cm^2)	15.50 ± 37.77[a]	60.53 ± 30.17[b]	122.00 ± 30.17[c]	233.85 ± 63.90[d]	0.0000
SAT mass (g)	347.77 ± 40.18[a]	415.65 ± 23.19[b]	467.11 ± 27.89[c]	555.57 ± 65.98[d]	0.0000

FMI: fat mass index; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; HDL: high-density lipoprotein; TC: total cholesterol; TG: triglycerides; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; γ-GT: γ-Gamma glutamyltransferase; VAT: Visceral adipose tissue; SAT: subcutaneous adipose tissue. SD: standard deviation. [a - d] Different literals per column indicate significant difference with Bonferroni post hoc test. P value defined as alpha < 0.05.

Tabla 4. Association Models between VAT, SAT, and FMI with Lipid Profile and Liver Enzymes respectively (n=219)

VAT (cm^2)				
	Undajusted Model		Adjusted Model	
	β (IC 95%)	*p*-value	β (IC 95%)	*p*-value
Lipid profile				
LDL (mg/dL)	0.26 (-0.01, 0.53)	0.061	0.34 (0.07, 0.61)	0.011
VLDL (mg/dL)	2.12 (1.17, 3.07)	0.000	1.91 (0.97, 2.85)	0.000
HDL (mg/dL)	-0.61 (-1.15, -0.08)	0.024	-0.77 (-1.30, -0.25)	0.004
TC (mg/dL)	0.24 (-0.01, 0.50)	0.070	0.25 (0.00, 0.50)	0.045
TG (mg/dL)	0.42 (0.23, 0.61)	0.000	0.38 (0.19, 0.57)	0.000
Liver enzymes				
AST (U/L)	0.81 (-0.32, 1.96)	0.161	0.77 (-0.31, 1.87)	0.163
ALT (U/L)	1.71 (0.41, 3.02)	0.010	2.00 (0.73, 3.28)	0.002
γ-GT (U/L)	1.28 (0.46, 2.10)	0.002	1.29 (0.49, 2.08)	0.002
SAT (g)				
	Undajusted Model		Adjusted model	
	β (IC 95%)	*p*-value	β (IC 95%)	*p*-value
Lipid profile				
LDL (mg/dL)	0.42 (0.17, 0.67)	0.001	0.37 (0.12, 0.625)	0.003
VLDL (mg/dL)	2.12 (1.25, 2.98)	0.000	1.80 (0.93, 2.67)	0.000
HDL (mg/dL)	-0.93 (-1.41, -0.45)	0.000	-0.80 (-1.29, -0.31)	0.001
TC (mg/dL)	0.31 (0.07, 0.55)	0.010	0.26 (0.03, 0.50)	0.024
TG (mg/dL)	0.42 (0.25, 0.59)	0.000	0.36 (0.18, 0.53)	0.000
Liver enzymes				
AST (U/L)	0.71 (-0.34, 1.76)	0.184	0.55 (-0.46, 1.57)	0.285
ALT (U/L)	2.27 (1.09, 3.45)	0.000	1.90 (0.71, 3.09)	0.002
γ-GT (U/L)	1.55 (0.81, 2.29)	0.000	1.37 (0.63, 2.10)	0.000
FMI (kg/m^2)				
	Undajusted Model		Adjusted model	
	β (IC 95%)	*p*-value	β (IC 95%)	*p*-value
Lipid profile				
LDL (mg/dL)	0.01 (-0.00, 0.02)	0.098	0.016 (0.00, 0.03)	0.011
VLDL (mg/dL)	0.09 (0.52, 0.14)	0.000	0.09 (0.04, 0.13)	0.000
HDL (mg/dL)	-0.02 (-0.05, -0.00)	0.045	-0.03 (-0.06, -0.01)	0.004
TC (mg/dL)	0.01 (-0.00, 0.02)	0.094	0.01 (0.00, 0.02)	0.049
TG (mg/dL)	0.02 (0.01, 0.02)	0.000	0.01 (0.00, 0.02)	0.000
Liver enzymes				
AST (U/L)	0.03 (-0.01, 0.09)	0.195	0.03 (-0.01, 0.09)	0.178
ALT (U/L)	0.07 (0.01, 0.14)	0.020	0.09 (0.03, 0.16)	0.002
γ-GT (U/L)	0.05 (0.01, 0.09)	0.006	0.06 (0.02, 0.09)	0.002

VAT: Visceral adipose tissue; SAT: subcutaneous adipose tissue; FMI: fat mass index; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; HDL: high-density lipoprotein; TC: total cholesterol; TG: triglycerides; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; α-GT: α-Gamma glutamyltransferase. P-value defined as alpha < 0.05

respectively. The same trend was observed for VAT and SAT with liver enzymes ALT and γ-GT; an increase in body composition parameters (VAT and SAT) was associated with alterations in these liver enzymes. FMI was also positively associated with liver enzymes and lipoproteins, indicating an increase in biochemical concentrations when VAT and SAT increased, except for HDL, where the association was negative, meaning HDL decreased when FMI increased (Table 4).

Discussion

Our study showed higher VAT levels in women compared with men, while the literature indicates that men tend to have higher VAT than women. However, national data from Mexico reported that women have a higher prevalence of abdominal adiposity than men (88.4% and 72.1%, respectively). It has also been reported that adults aged 20 - 29 years have a 62.1% prevalence of abdominal adiposity, and by region, Northern Mexico (which includes the state of Sonora) ranks second, with a high prevalence of this adiposity (83.5%) compared with other regions of the country, except for Mexico City, which shows the highest prevalence (88%) (15). In this regard, another study reported a subgroup of adults with a normal BMI but metabolic abnormalities called metabolically unhealthy normal weight in which 71.6% of women and 56.5% of men had elevated visceral adipose tissue (VAT); the same study also reported that 26.42% of women and 5.54% of men had central obesity (27). Similarly, in Mexican adults aged 20 - 64 years, women have been reported to engage in less physical activity than men (28), consistent with what we observed in our study population. These findings may offer a possible explanation for why men exhibited lower visceral adipose tissue (VAT) than women.

The findings of this study indicate a positive association between body fat distribution, as assessed by dual-energy X-ray absorptiometry (DXA), and biochemical concentrations of liver enzymes and lipid profiles. Furthermore, a negative association

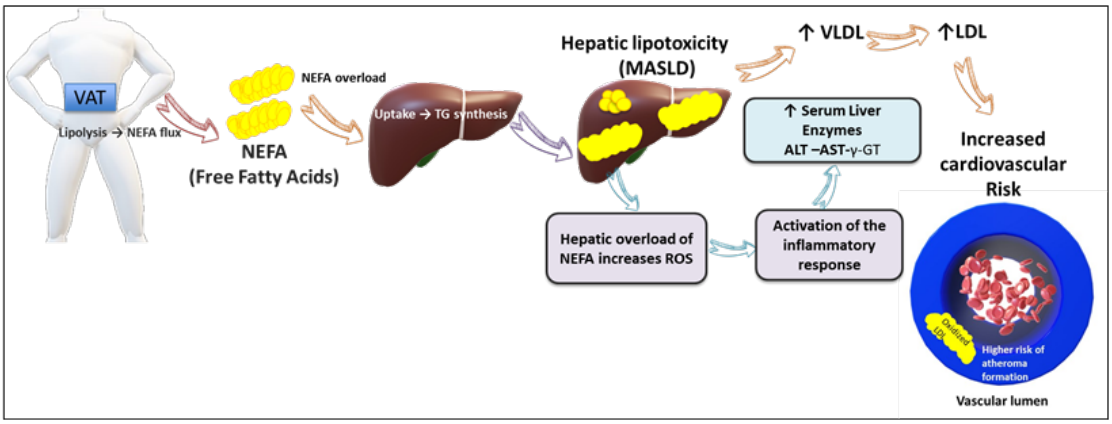

Figure 3. Schematic representation of the pathway linking VAT to dyslipidemia, hepatic dysfunction, inflammatory processes, and vascular risk.

was observed between visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) with high-density lipoprotein (HDL) concentrations. These associations were significant for most liver enzymes, except for aspartate aminotransferase (AST) (Figure 3).

Our study demonstrates that body fat distribution is related to changes in lipid biochemical parameters among university students, suggesting that an increase in body fat alters lipid concentrations, increasing the levels of lipoproteins (LDL and VLDL), TG, and TC. Previous studies align with our findings, establishing that an increase in visceral adiposity leads to elevated lipid biochemical profiles (29, 30). A descriptive, cross-sectional, prospective study determined the relationship between the degree of MASLD and quantitative lipid profile values in overweight and obese patients, reporting increased levels of TC, LDL, and TG from the early stages of fatty liver disease. Also, identified that elevated TG and LDL were indicative parameters of fatty liver disease; these are necessary to consider in diagnosis. They concluded that greater obesity and steatosis also increase LDL and decrease HDL, and significant lipid profile changes can occur even with overweight alone (31). Although hepatic steatosis was not evaluated in the patients in this study, biochemical parameters were used as risk indicators, which can be utilized for more specific studies and to prevent potential pathologies at an early age (university students).

Furthermore, we found a relationship between body fat distribution and alterations in liver enzyme parameters among university students. This indicates that an increase in visceral fat results in elevated plasma levels of liver enzymes. A previous study conducted on apparently healthy and preclinical Japanese subjects found that subcutaneous fat was not associated with significant changes in liver enzymes. Instead, visceral fat accumulation was positively associated with increased enzyme levels (32), which concurs with our study. Similarly, Kotronen et al. studied 356 American adults aged 18-70 years, reporting a significant correlation of SAT with TG (p < 0.01 females; p < 0.001 males), ALT (p < 0.05 females; P < 0.001 males) and AST (P < 0.001 males) and a negative relationship with HDL (p < 0.001 both sexes) and VAT with TG (p < 0.001 both sexes), ALT (P < 0.001 both sexes) and AST (p < 0.05 females; p < 0.001 males) and a negative relationship with HDL (p < 0.001 both sexes). These authors suggested that the association of VAT and liver enzymes might be an important indicator of increased liver fat in individuals with abdominal obesity (33). Another study by Liu et al. (34), evaluating 2986 participants (1581 with hepatic steatosis) over 18 years old, found that subjects with steatosis had higher levels of ALT, AST, TG, TC, and VAT area (p<0.001 for all) compared to those without steatosis. The association of VAT with ALT was also observed in the present study. Similarly, Mukherjee et al. evaluated 135 apparently healthy university students from Bangladesh regarding the presence of hepatic steatosis with liver enzymes and other variables, finding a positive relationship between ALT and γ-GT and hepatic steatosis, highlighting γ-GT elevation as a better predictor of fatty liver severity (regardless

of etiopathogenesis) than ALT (35). Another study related to Chinese adults observed an increase in VAT associated with elevated γ-GT values (β = 0.796; P = 0.043), but not with ALT, and no significant association with other liver enzymes (36). This contrasts with our study findings. It appears that γ-GT elevation might be closely related to the progression towards developing fatty liver, and some authors have noted that this enzyme might play an important role in the formation and progression of metabolic dysfunction-associated steatotic liver disease (MASLD) (37).

FMI has proven to be a useful tool for evaluating body composition in subjects, eliminating the difference between fat-free mass and body fat associated with height. It is also useful for identifying individuals with excess muscle mass but without excess body fat (38). In this study, a significant association was found between FMI and lipid profile (LDL - $p < 0.011$; VLDL - $p < 0.000$; HDL - $p < 0.004$; TC - $p < 0.049$; TG - $p < 0.000$), as well as with liver enzymes (ALT – $p < 0.002$ and γ-GT – $p < 0.002$). These results align with those reported by Salinas et al. in young Mexican adults (18-25 years), who reported that an increase in FMI significantly correlated with high TG levels in both males and females (r = 0.293, $p < 0.0001$ females; r = 0.332, $p< 0.0001$ males). They also indicated that females with obesity and males with higher fat mass tend to present a higher risk of cardiovascular risk factors. The researchers confirmed that increased adiposity has an opposite association with metabolism, thereby increasing the risk of developing metabolic disorders (39).

There are biological mechanisms related to fat distribution, where different fat deposits regulate lipid metabolism differently, suggesting that metabolic dysfunction could be more associated with fat distribution (lipoproteins) (40). Additionally, visceral obesity is recognized to be associated with cardiovascular diseases (CVD) and alterations in lipid and carbohydrate metabolism. Akiyama et al. observed that patients diagnosed with increased abdominal obesity (ICT ≥ 0.5) tend to present significantly higher levels of TG, VLDL, and all VLDL subclasses (41). It has also been documented that VAT is highly lipolytic, directing lipid distribution primarily towards the liver, producing an excessive flow of non-esterified fatty acids, which can cause overproduction of VLDL, potentially leading to hypertriglyceridemia and indirectly increasing LDL levels through VLDL conversion, as well as increasing the risk of fatty liver disease (42). This accumulation of fat in the liver can also be associated with elevated liver enzymes above the normal interval (43). Denova-Gutiérrez et al. mentioned that obesity, metabolic syndrome, and insulin resistance promote increased serum levels of AST, ALT, and γ-GT (44). SAT represents the most voluminous structure of the body, and its lipolysis could affect blood lipid levels, with adipocytes in SAT potentially playing a significant role in the pathogenesis of certain diseases (45).

Our study has some limitations. Regarding the sample, it was convenience-based and non-probabilistic, comprising Health Sciences students, which may indicate a higher health awareness or lifestyle choices influenced by their academic training. Additionally, the cross-sectional design precludes the ability to establish causality, and the possibility of reverse causality cannot be ruled out. To mitigate this, participants provided a 24-hour dietary recall to reflect their usual consumption and family history. Selection bias (46) is likely due to the non-probabilistic nature of the sample, as students who opted to participate might have been more health-conscious and engaged in better self-care practices compared to those who did not participate, thereby limiting the generalizability of the findings to the broader population. Furthermore, non-differential measurement error may be inherent (47) in dietary information questionnaires, assessments of sleep quality, and evaluations of physical activity, as these tools rely on retrospective self-reporting and memory, which are subject to bias despite their continued use and validation in research. Likewise, the use of a single 24-hour dietary recall, instead of the recommended two or more recalls to capture habitual intake, may introduce recall bias and intra-individual variability, particularly for nutrients with high day-to-day variation (e.g., DHA and HEPA), since the lack of repeated measures limits the adjustment for within-person variability. Nevertheless, to mitigate this potential bias, the mean energy intake in our sample was estimated at 1823.36 kcal, which is comparable to the population mean reported by ENSANUT 2016 (1908.0 kcal) (48), supporting

the validity of our group estimates and their consistency with nationally representative data. In the analysis of fat distribution by sex, overweight men were showed lower LDL and TC values compared to the normal group. This inconsistency could be explained by the sample size (overweight n=9 and normal n=26), which may have limited representativeness. It has also been reported that small samples can limit the ability to draw meaningful conclusions and generalize findings (49). Therefore, these results should be interpreted with caution, and further research with larger sample size should be conducted. Similarly, the obesity group had a smaller sample size (n=7), the magnitude of the effect was stronger in this group. This could be explained by the fact that this group had a higher amount of fat mass, which is associated with metabolic alterations (30), and the effect on LDL and TC levels was more consistent.

Our study has several important strengths. The primary one is the use of DXA, a reference standard considered one of the gold standards (50 – 52) for measuring body composition and fat distribution. It has high precision and reliability (r2=0.996) and low measurement variability (coefficient of variation [CV] less than 4%). It can estimate abdominal fat and quantify fat mass with a CV of 2%, and it has a good correlation for muscle mass compared to other methods (53). Another strength to consider is the biochemical evaluation of the lipid profile and liver enzymes using standardized methods, providing a detailed metabolic profile evaluation (54, 55). To mitigate unassessed confounding variables, multiple regression models were performed, and sample size calculation inferred greater statistical reliability.

Conclusions

The findings of this study indicate that body fat distribution (VAT and SAT) is associated with lipid levels (LDL, VLDL, TC, TG, and HDL) and certain liver enzymes (ALT and γ-GT) in university students. These may indicate the onset of metabolic alterations. Additionally, we demonstrated that FMI is a simple indicator that can function as a predictor of lipid profile and liver enzyme alterations, potentially aiding in the prevention of diseases related to excess and the type of body fat accumulation.

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Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article..

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