PEDIATRICS

ASSESSMENT OF THE PARAMETERS OF THE FATTY ACID SPECTRUM OF BLOOD SERUM IN RELATION TO HORMONAL STATUS IN OBESE ADOLESCENTS

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RESUME

Rationale. *The development and implementation of new high-tech mass spectrometric diagnostic methods into laboratory practice has determined the possibility of a global analysis of the human lipidome, in particular, for a detailed study of its fatty acid component and assessment of the role of individual free fatty acids (FFA) in the pathogenesis of obesity and associated diseases.*

Objective. *To identify the features of changes in the parameters of the fatty acid spectrum of blood serum and establish their relationship to hormonal status indicators in adolescents with obesity.*

Materials and methods. *A total of 27 adolescents aged 10–18 years with obesity (SDS BMI 2.0-3.9) were examined. The control group consisted of 27 adolescents with normal weight with comparable characteristics by gender and age. SDS BMI was calculated using the WHO Anthroplus calculator. The concentration of hormones and peptides in the blood serum was measured by ELISA. The mobile fatty acid pool of blood serum was assessed by chromatography-mass spectrometry on an Agilent 7000B detector.*

Results. *In adolescents with obesity of 1–3 degrees, elevated levels of insulin and C-peptide, decreased concentration of GLP-2 and fatty acid imbalance (decreased proportion of GLA, DGLA, DPA, DHA, AA and increased content of ALA, OA, POA, BA, MA, PA, MAA), as well as a low risk index for the development of a subintimal inflammatory reaction are recorded in the blood. In the group with obesity, direct and negative correlations were established between the content of individual hormones and fatty acids, which were absent between the corresponding parameters in the group of healthy individuals.*

Conclusion. *The established endocrine-metabolic changes in adolescents with obesity are pathogenetic factors of a complex of compensatory-adaptive reactions accompanying low-intensity inflammation.*

Key words: *adolescents, obesity, fatty acids, insulin, leptin, glucagon-like peptide 2*

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ОЦЕНКА ПАРАМЕТРОВ ЖИРНОКИСЛОТНОГО СПЕКТРА СЫВОРОТКИ КРОВИ ВО ВЗАИМОСВЯЗИ С ПОКАЗАТЕЛЯМИ ГОРМОНАЛЬНОГО СТАТУСА У ПОДРОСТКОВ С ОЖИРЕНИЕМ

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РЕЗЮМЕ

Обоснование. *Развитие и внедрение в лабораторную практику новых высокотехнологичных масс-спектрометрических методов диагностики определило возможность глобального анализа липидома человека, в частности, для детального изучения его жирнокислотной составляющей и оценки роли отдельных свободных жирных кислот (СЖК) в патогенезе ожирения и ассоциированных с ним заболеваний.*

Цель. *Выявить особенности изменений параметров жирнокислотного спектра сыворотки крови и установить их взаимосвязь с показателями гормонального статуса у подростков с ожирением.*

Материалы и методы. *Обследовано 27 подростков в возрасте 10–18 лет с ожирением (SDS ИМТ 2,0–3,9). Группу контроля составили 27 подростков с нормальным весом с сопоставимыми характеристиками по полу и возрасту. Расчет SDS ИМТ проводился с использованием калькулятора ВОЗ «Anthroplus». Концентрацию гормонов и пептидов в сыворотке крови осуществляли методом ИФА. Мобильный жирнокислотный пул сыворотки крови оценивали методом хромато-масс-спектрометрии на детекторе Agilent 7000B.*

Результаты. *У подростков с ожирением 1–3 степеней в крови регистрируются повышенные уровни инсулина и С-пептида, снижение концентрации GLP-2 и жирнокислотный дисбаланс (снижение доли GLA, DGLA, DPA, DHA, AA и повышение содержания ALA, OA, POA, BA, MA, PA, MAA), а также низкий индекс риска развития субинтимальной воспалительной реакции. В группе с ожирением установлены прямые и отрицательные корреляции между содержанием отдельных гормонов и жирных кислот, отсутствующие между соответствующими параметрами в группе здоровых лиц.*

Заключение. *Установленные эндокринно-метаболические изменения у подростков с ожирением являются патогенетическими факторами комплекса компенсаторно-приспособительных реакций, сопровождающих низкоинтенсивное воспаление.*

Ключевые слова: *подростки, ожирение, жирные кислоты, инсулин, лептин, глюкагонподобный пептид 2*

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BACKGROUND

The steady increase in childhood obesity during puberty is a prominent feature of its current epidemiology. The primary causes of excessive body weight include stress-induced overeating, poor nutrition, physical inactivity, hormonal changes during adolescence, and other factors. Obesity during adolescence poses a higher risk than in adulthood, as it can lead to the early onset of a wide range of diseases and pathological conditions. Neurohumoral factors originating from local visceral fat depots are the primary drivers of the systemic and organ-specific disorders that constitute metabolic syndrome [1].

Accurate diagnosis of visceral obesity remains a challenging task. Most current methods, including anthropometric measurements (body mass index, waist circumference, and waist-to-hip ratio) and adipose tissue imaging techniques (ultrasound, magnetic resonance imaging, and computed tomography), lack sufficient accuracy for diagnosing visceral obesity, particularly regarding perivascular fat depots [2]. While these methods serve as effective and accessible screening tools for identifying overweight and obese individuals, they do not always reliably stratify patients into high-risk groups for cardiovascular disease and other complications associated with excess visceral fat accumulation. Furthermore, the growing number of "metabolically healthy" obese individuals underscores the need for standardized identification criteria, as anthropometric indicators alone are insufficient for diagnosis [3].

Recently, there has been a surge of interest in the field of metabolomics, specifically in the area of lipid metabolism, known as lipidomics. The advancement and implementation of novel high-tech mass spectrometry techniques in laboratory settings have made it feasible to conduct a comprehensive, high-resolution analysis of the human lipid profile, particularly its fatty acid component. The analysis of serum free fatty acid (FFA) concentrations has emerged as a promising new biomarker for lipid metabolism, including in the context of obesity [4]. Investigating the mechanisms by which individual FFAs and an imbalanced fatty acid composition contribute to the development of obesity and related metabolic disorders is a crucial area of contemporary research [5]. Alterations in fatty acid profiles, especially when combined with hormonal status parameters, may represent a novel potential biomarker for assessing the risk of metabolic syndrome, cardiovascular disease, rheumatologic conditions, infectious diseases, and other complications associated with visceral obesity. These changes could ultimately form the basis for developing highly accessible, non-invasive laboratory methods for diagnosing and monitoring visceral obesity and its associated conditions.

THE AIM OF THE STUDY

To identify specific changes in the serum fatty acid profile and to establish their correlation with hormonal status in adolescents with obesity.

METHODS

Study design. This single-center, observational, cross-sectional comparative study involved 27 adolescents (16 girls and 11 boys) aged 10 to 18 years, at pubertal stages 2–4 (according to Tanner) with grade 1–3 obesity (body mass index standard deviation score (SDS BMI) within 2.0–3.9). All adolescents were combined into a single (main) group. To assess hormonal status and anthropometric parameters, the participants were divided by sex into two subgroups within the main group. The control group comprised 27 adolescents (17 girls and 10 boys) with normal weight, at pubertal stages 2–4 (according to Tanner) and with statistically comparable age characteristics. The subjects were enrolled in the study using a consecutive sampling approach. Group allocation was performed according to the predefined inclusion and exclusion criteria.

Eligibility criteria. The inclusion criteria for the study were: age 10 to 18 years; a diagnosis of exogenous-constitutional obesity grades 1–3; and written informed consent from the participants or their legal representatives (for participants under the age of 15). The exclusion criteria for the main study group were: age under 10 or over 18 years; absence of a diagnosis of exogenous-constitutional obesity grades 1–3; presence of syndromic or monogenic forms of obesity; diabetes mellitus types 1 or 2; acute or chronic somatic conditions; and diseases requiring the use of antibacterial or hormonal medications. Additionally, individuals with laboratory-confirmed helminthic invasion and those who had taken antibacterial medications in the three months prior to the study were excluded.

The study was conducted at the Children's Clinic of Siberian State Medical University and the Professor Medical Center (Tomsk, Russia), where adolescents with and without obesity were recruited. Healthy school-aged children from the Perspektiva School in Tomsk were also invited to participate.

Study duration. Recruitment for the obesity group took place between September 2023 and October 2024 during patient visits to the healthcare facility.

Materials and methods. The SDS BMI was calculated using the World Health Organization's AnthroPlus software, taking into account the sex, age, and anthropometric measurements (weight and height) of each participant. Height was measured using an MSK-233 vertical stadiometer (accuracy of 0.1 cm), and weight was measured using an electronic scale (accuracy of 0.1 kg).

The study material was venous blood serum, collected once from the cubital vein of each participant under fasting conditions.

Hormone and peptide concentrations in venous serum were measured using enzyme-linked immunosorbent assay (ELISA) on a Uniplan analyzer (Pikon, Russia). Leptin concentration was measured using an Active Human Leptin ELISA Kit (DSL-10-23100, Diagnostic Systems Laboratories Inc., USA). The levels of other hormones and peptides (insulin, C-peptide, glucagon, glucagon-like

peptide 1 and 2, irisin, and resistin) were measured in serum using ELISA reagents from Claudio Clone Corporation (USA).

The mobile fatty acid profile of blood serum was analyzed using validated gas chromatography with mass-selective detection on an Agilent 7000B system (Agilent Technologies Inc., USA) [6]. The results were reported as relative percentages.

Ethical approval. The study was approved by the ethics committee of Siberian State Medical University (protocol No. 8459/2, dated October 28, 2020).

Statistical analysis. Statistical analysis was conducted using standard statistical methods and IBM SPSS Statistics software. The Shapiro-Wilk test was used to test the hypothesis of a normal distribution. For samples with non-normal distributions, the non-parametric Mann – Whitney U-test was used to assess statistical significance between groups. Results are presented as medians and interquartile ranges (Me [Q25; Q75]). Correlations between quantitative variables were determined using Spearman's rank correlation coefficient. Statistical significance was set at $p < 0.05$ for all comparisons.

RESULTS

The characteristics of the study participants, stratified by sex, age, and anthropometric parameters, are presented in Table 1. The median age of subjects in the main group was 14.8 [13.15; 16.15] years, and in the control group, 16.50 [13.45; 17.75] years. The study groups were comparable in terms of sex and age distribution. No statistically significant differences were observed between the parameters of male and female adolescents within each study group.

An analysis of hormone levels in adolescents with obesity revealed increased serum insulin and C-peptide levels, as well as decreased glucagon-like peptide 2 levels, compared to the control group (Table 2). No sex-specific differences in these hormone levels were found within either group; therefore, subsequent analyses of fatty acid profiles were performed on pooled data from both sexes.

In adolescents with grade 1–3 obesity, the serum mobile fatty acid pool was altered relative to controls, characterized by an increase in the relative percentages of saturated (SFA) and monounsaturated (MUFA) fatty acids and a decrease in the proportion of polyunsaturated fatty acids (PUFAs) (Fig. 1).

Detailed analysis of the serum fatty acid profile in obese adolescents revealed an imbalance within the PUFA pool. Specifically, there was a significant decrease in the concentrations of GLA, DGLA, DPA, DHA, and AA, alongside an approximately 1.4-fold increase in the concentration of ALA, compared to controls (Table 3).

An imbalance was also observed within the MUFA pool, reflected by increased levels of OA and POA

TABLE 1

AGE-SEX AND ANTHROPOMETRIC CHARACTERISTICS OF THE EXAMINED ADOLESCENTS IN THE MAIN AND CONTROL GROUPS, ME [Q1; Q3]

Parameters	Control group, *n*=27 Girls, *n*=17 Boys, *n*=10	Main group, *n*=27 Girls, *n*=16 Boys, *n*=11	*p*
Age, years			
All adolescents	16.50 [13.45; 17.75]	14.80 [13.15; 16.15]	0.139
Girls	15.30 [11.75; 16.10]	14.35 [12.00; 15.60]	0.183
Boys	16.30 [13.35; 17.20]	15.50 [14.37; 17.40]	0.122
Weight, kg			
All adolescents	53.30 [49.30; 56.70]	86.80 [65.35; 95.75]	<0.001*
Girls	53.70 [50.10; 56.80]	87.35 [70.35; 96.25]	<0.001*
Boys	54.05 [49.93; 56.08]	90.00 [60.85; 98.10]	<0.001*
Height, cm			
All adolescents	162.00 [156.50; 168.00]	164.00 [154.25; 172.20]	0.395
Girls	159.00 [155.00; 164.00]	162.00 [156.37; 168.50]	0.378
Boys	162.00 [156.50; 168.00]	169.00 [152.00; 174.50]	0.449
SDS BMI, c.u.			
All adolescents	-0.08 [-0.79; 0.50]	2.60 [2.40; 2.95]	<0.001*
Girls	0.27 [-0.21; 0.65]	2.55 [2.40; 2.95]	<0.001*
Boys	-0.80 [-0.70; 0.40]	2.72 [2.51; 2.81]	<0.001*

Note. Me – median, Q1, Q3 – lower and upper quartiles; BMI – body mass index; BMI SDS – body mass index standard deviation score; *p* – significance level for differences between the main and control groups (Mann – Whitney U-test); * – statistically significant differences between groups.

and decreased levels of mid-chain MUFAs in adolescents with obesity compared to their healthy peers (Table 3).

The serum SFA pool was similarly altered in the obesity group, with a significant increase in the proportions of BA, MA, PA, and MAA relative to the control group (Table 3).

Assessment of fatty acid-derived indices revealed a reduction in the AA/EPA ratio, a risk index for subintimal inflammation, in adolescents with obesity compared to those without obesity (Table 3).

Correlation analysis was performed for all parameters. While a number of expected correlations were observed between individual fatty acid levels and hormonal status in both groups, several correlations were identified exclusively in the obese cohort.

In obese adolescents, statistically significant positive correlations were observed between serum levels of adrenic acid (ADA) and resistin (r = 0.584; p = 0.0019; 95% CI [0.262; 0.789]), nervonic acid (NA) and glucagon (r = 0.531; *p* = 0.0045; 95% CI [0.189; 0.758]), and palmitoleic acid (POA) and irisin (r = 0.513; *p* = 0.0018; 95% CI [0.165; 0.747]). Negative correlations were recorded between ADA and irisin (r = –0.520; *p* = 0.0321; 95% CI [-0.751; -0.174]), ALA and resistin (r = –0.544; *p* = 0.0305; 95% CI [-0.765; -0.206]), NA and C-peptide (r = –0.572; *p* = 0.0452; 95% CI [-0.782; -0.245]), and POA and leptin (r = –0.575; *p* = 0.0042; 95% CI [-0.783; -0.249]). These

TABLE 2

SERUM HORMONE CONCENTRATIONS IN OBESE ADOLESCENT CHILDREN WITH NORMAL BODY WEIGHT, ME [Q1; Q3]

Parameters	Control group, *n*=27 Girls, *n*=17 Boys, *n*=10	Main group, *n*=27 Girls, *n*=16 Boys, *n*=11	*p*
Insulin, μU/ml			
All adolescents	8.40 [5.70; 10.55]	22.60 [17.65; 26.25]	0.011*
Girls	8.45 [6.43; 11.10]	23.10 [19.90; 32.03]	0.023*
Boys	7.30 [5.23; 9.58]	21.25 [15.40; 25.80]	0.012*
C-peptide, ng/ml			
All adolescents	2.00 [1.40; 2.40]	2.60 [1.90; 3.10]	0.022*
Girls	2.00 [1.43; 2.38]	2.60 [1.90; 2.80]	0.031*
Boys	2.10 [1.50; 2.40]	2.55 [2.12; 3.52]	0.016*
Glucagon, pg/ml			
All adolescents	188.9 [160.00; 224.70]	170.60 [160.40; 176.00]	0.315
Girls	186.7 [173.80; 234.10]	163.00 [156.65; 193.00]	0.188
Boys	160.0 [152.20; 208.40]	176.60 [168.10; 186.00]	0.240
GLP-1, pg/ml			
All adolescents	24.47 [23.17; 29.37]	22.48 [20.54; 29.47]	0.710
Girls	24.51 [22.62; 29.37]	22.64 [20.27; 29.58]	0.655
Boys	24.47 [23.61; 30.43]	27.43 [20.38; 29.68]	0.789
GLP-2, pg/ml			
All adolescents	506.20 [358.70; 667.00]	114.60 [102.95; 168.80]	<0.001*
Girls	478.00 [295.15; 546.15]	111.65 [103.50; 288.00]	<0.001*
Boys	546.00 [487.45; 684.55]	128.60 [100.12; 235.68]	<0.001*
Irisin, pg/ml			
All adolescents	7.13 [7.13; 7.44]	7.33 [6.98; 7.65]	0.188
Girls	7.13 [7.13; 7.34]	7.30 [7.22; 7.65]	0.270
Boys	7.29 [6.89; 7.44]	7.33 [6.96; 7.49]	0.145
Leptin, ng/ml			
All adolescents	10.00 [7.18; 12.58]	7.84 [3.99; 9.98]	0.083
Girls	10.44 [8.37; 12.91]	6.46 [4.12; 9.38]	0.079
Boys	9.20 [6.68; 11.97]	8.41 [6.78; 10.48]	0.141
Resistin, ng/ml			
All adolescents	12.17 [7.22; 18.49]	14.72 [9.97; 21.03]	0.290
Girls	10.38 [6.92; 18.98]	14.54 [9.44; 21.21]	0.360
Boys	11.51 [7.71; 16.14]	16.53 [12.32; 20.34]	0.494

Note. Me – median, Q1, Q3 – lower and upper quartiles; GLP – glucagon-like peptide; *p* – significance level for differences between groups (Mann – Whitney U-test); * – statistically significant differences between groups.

correlations were not observed in the control group of healthy individuals.

DISCUSSION

Hyperinsulinemia is a well-established pathogenetic factor in obesity, with insulin exerting a pronounced anabolic effect on all types of metabolism. While its primary function is to regulate blood glucose levels, insulin also plays an active role in lipid metabolism. It stimulates lipid synthesis in the liver and adipose tissue, directly induces glucose entry into fat cells, and promotes the hydrolysis of triacylglycerols associated with blood lipoproteins, facilitating fatty acid entry into adipocytes. This process contributes to glycerophosphate formation, leading to increased adipose tissue mass [7]. Furthermore, insulin suppresses cAMP-mediated lipolysis by inhibiting hormone-dependent intracellular lipoprotein lipase [8]. C-peptide, a marker of insulin secretion, varies in response to fluctuations in endogenous insulin levels [9]. Therefore, the elevated C-peptide levels observed in obese adolescents with concurrent hyperinsulinemia were an expected finding.

Conversely, glucagon, a counter-regulatory hormone to insulin, stimulates hepatic glucose release to maintain glucose homeostasis and also promotes fat breakdown in adipose tissue [8]. Glucagon secretion is directly related to blood glucose levels, decreasing as glucose increases. In the present study, no statistically significant differences in glucagon levels were observed between obese and non-obese adolescents, although a downward trend was noted in the obese group.

In obese adolescents with elevated insulin levels, we observed low levels of glucagon-like peptide 2 (GLP-2). GLP-2 is produced by central nervous system neurons and intestinal L-cells and exerts multiple effects, including intestinal trophic actions (mucosal proliferation and improved barrier function), increased mesenteric blood flow, reduced bone loss, and neuroprotection [10]. GLP-2 receptors are present in the gastrointestinal tract, liver, adipose tissue, and central nervous system. While GLP-2 does not affect appetite or food intake in humans, its intestinal trophic action is significant, particularly in the context of obesity. Obesity is associated with impaired intestinal barrier function, leading to increased translocation of pro-inflammatory luminal contents into the bloodstream, which negatively impacts the development of obesity-related conditions. Moreover, GLP-2 has a positive effect on normalizing glucose levels in obese individuals [10]. The reduced GLP-2 levels observed in our overweight cohort may thus contribute to heightened systemic inflammation and, consequently, to the development of complications and comorbidity.

Considering the observed changes in the serum fatty acid profile in conjunction with hormonal alterations, the pattern of reduced total PUFAs and increased SFAs and MUFAs is consistent with the metabolic

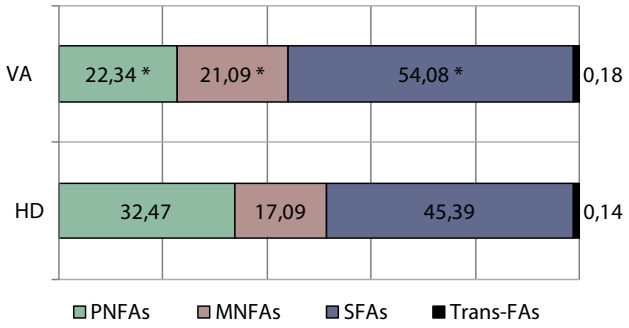

FIG. 1.
*Percentage ratio of pools of polyunsaturated (PN), monounsaturated (MN), saturated fatty acids (SFAs) and their trans-isomers (trans-FAs) in serum in obese adolescents (VA) and normal body weight (HD). Note: * – significance level of differences $p < 0.05$ (Mann – Whitney U test).*

consequences of poor dietary habits, physical inactivity, and stress, which are prevalent in modern adolescents during puberty [11]. This is further supported by the specific fatty acid imbalances identified in the obese group: a reduction in omega-3 (DPA, DHA) and omega-6 (GLA, DGLA, AA) PUFAs, alongside excessive amounts of OA and POA (MUFAs) and BA, MA, PA, and MAA (SFAs). These findings are broadly consistent with existing scientific literature. However, as studies in school-aged children have shown, many children with a normal BMI may exhibit dietary deficiencies similar to those seen in obese children without subsequent weight gain [8]. Notably, our findings indicate that obese adolescents did not exhibit a deficiency in the essential PUFAs (LA, ALA); indeed, ALA concentrations exceeded those in the control group.

Chronic low-grade inflammation, in combination with increased release of fatty acids into the bloodstream and ectopic fat accumulation, is now recognized as a significant factor in the pathogenesis of obesity [12]. Markers of low-grade inflammation include increased levels of certain adipokines (primarily leptin and resistin), as well as elevated concentrations of classic inflammatory markers such as C-reactive protein (CRP) and pro-inflammatory cytokines (IL-6, TNFα, MCP1) [13].

Leptin, primarily produced by adipocytes, serves as a key regulator of body fat mass. As a mediator between adipose tissue and the hypothalamic-pituitary system, it reduces appetite and food intake by signaling to the brain. Numerous studies have shown that blood leptin levels increase with higher body weight, linked to the development of leptin resistance [14]. However, in our study, we did not find an increase in leptin levels in obese adolescents compared to normal-weight controls; in fact, a downward trend was observed. Additionally, a negative correlation was found between leptin and POA levels in the main study group. While this finding may seem counterintuitive regarding leptin's role in appetite and weight regulation,

TABLE 3

FATTY ACID CONTENT AS A PERCENTAGE OF THE TOTAL MOBILE POOL IN THE SERUM OF OBESE ADOLESCENTS WITH NORMAL BODY WEIGHT, ME [Q1; Q3]

Fatty acids in blood serum, %	Control group, *n*=27	Main group, *n*=27	*p*
Polyunsaturated fatty acids			
Alpha-linolenic acid (ALA 18:3n3)	0.18 [0.15; 0.23]	0.26 [0.22; 0.29]	0.029*
Eicosapentaenoic acid (EPA 20:5n3)	0.10 [0.04; 0.14]	0.07 [0.06; 0.08]	0.921
Docosahexaenoic acid (DHA 22:6n3)	1.74 [0.65; 2.21]	1.01 [0.91; 1.25]	0.038*
Docosapentaenoic acid (DPA 22:5n3)	0.40 [0.34; 0.78]	0.22 [0.21; 0.27]	<0.001*
Arachidonic acid (AA 20:4n6)	8.48 [7.81; 9.69]	3.90 [3.58; 4.17]	<0.001*
Gamma-linolenic acid (GLA 18:3n6)	0.22 [0.14; 14.42]	0.11 [0.07; 0.15]	0.017*
Dihomo-gamma-linolenic acid (DGLA 20:3n6)	1.01 [0.89; 1.37]	0.67 [0.50; 0.84]	0.031*
Linoleic acid (LA 18:2n6)	20.40 [16.23; 23.73]	16.50 [15.36; 17.72]	0.107
Adrenic acid (ADA 22:4n6)	0.95 [0.56; 1.28]	1.05 [0.52; 1.48]	0.064
Monounsaturated fatty acids			
Nervonic acid (NA 24:1n9)	1.79 [1.68; 1.99]	1.82 [1.75; 1.94]	0.811
Oleic acid (OA 18:1n9)	14.10 [12.74; 16.41]	17.23 [16.41; 18.72]	0.038*
Erucic acid (ERA 22:1n9)	0.00 [0.00; 0.00]	0.00 [0.00; 0.00]	1.000
Mead acid (20:3n9), %	0.07 [0.04; 0.24]	0.04 [0.03; 0.05]	0.033*
Myristoleic acid (MOA 14:1n5)	0.08 [0.06; 0.09]	0.08 [0.05; 0.10]	0.960
Palmitoleic acid (POA 16:1n7)	0.83 [0.67; 1.02]	1.14 [0.92; 1.32]	0.027*
Saturated fatty acids, including those containing an odd number of carbon atoms			
Arachidic acid (ANA 20:0)	0.35 [0.31; 0.38]	0.37 [0.35; 0.40]	0.097
Behenic acid (BA 22:0)	1.23 [1.14; 1.31]	1.34 [1.24; 1.48]	0.010*
Decanoic acid (DA 10:0)	0.02 [0.01; 0.02]	0.02 [0.02; 0.03]	0.076
Lauric acid (LAA 12:0)	0.04 [0.02; 0.05]	0.07 [0.02; 0.11]	0.371
Lignoceric acid (LCA 24:0)	2.55 [2.14; 2.65]	2.52 [2.37; 2.75]	0.064
Myristic acid (MA 14:0)	0.48 [0.32; 0.78]	0.82 [0.59; 1.06]	0.032*
Palmitic acid (PA 16:0)	24.75 [21.86; 28.12]	31.69 [29.24; 32.52]	<0.001*
Stearic acid (SA 18:0)	14.78 [13.76; 15.25]	16.56 [15.06; 16.98]	0.055

TABLE 3 (*continued*)

Pentadecanoic acid (PDA 15:0)	0.24 [0.19; 0.33]	0.28 [0.23; 0.32]	0.703
Margaric acid (MAA 17:0)	0.33 [0.29; 0.38]	0.44 [0.38; 0.46]	0.008*
Heptadecenoic acid (GDA 17:1n7)	0.07 [0.06; 0.08]	0.08 [0.07; 0.10]	0.710
Heneicosanoic acid (GEA 21:0)	0.02 [0.02; 0.03]	0.01 [0.01; 0.03]	0.512
Tricosanoic acid (TA 23:0)	0.25 [0.21; 0.30]	0.34 [0.26; 0.40]	0.098
	Trans fatty acids		
Linoelaidic acid (LELA 18:2ct)	0.10 [0.08; 0.13]	0.08 [0.06; 0.19]	0.412
Elaidic acid (ELA 18:1n9t)	0.03 [0.03; 0.05]	0.05 [0.05; 0.07]	0.066
	Fatty acid indices		
LA/DGLA	22.05 [18.21; 29.03]	25.34 [21.92; 31.73]	0.112
ω6/ω3	11.98 [10.56; 13.67]	13.98 [11.43; 15.92]	0.295
AA/EPA:(%AA/%EPA)	81.45 [63.12; 168.60]	51.48 [46.81; 65.79]	0.004*

Note. LA/DGLA – omega-6 desaturase activity index; ω6/ω3 – omega-6 to omega-3 fatty acid ratio; AA/EPA:(%AA/%EPA) – index of subintimal inflammatory response (risk of cardiovascular complications/level of body's protective reserve); *p* – significance level for differences between groups (Mann-Whitney U-test); * – statistically significant differences between groups.

an alternative interpretation considers leptin's significant role in the pathogenesis of inflammation and its capacity to regulate immune homeostasis within and beyond adipose tissue.

Leptin's effects influence virtually all cellular components of the immune system. *In vitro*, leptin stimulates the proliferation of circulating human monocytes, enhances the expression of activation markers (CD25, CD38, CD69, CD71, HLA-DR, CD11b, and CD11c), and stimulates neutrophil chemotaxis and oxygen radical release [15]. On eosinophils, leptin promotes the expression of adhesion molecules ICAM-1 and CD18 and induces chemotaxis and secretion of inflammatory cytokines (IL-1β, IL-6). Its effects on basophilic granulocytes include stimulation of migration, degranulation, and pro-inflammatory cytokine synthesis. Low leptin levels have been linked to a shift in basophil and mast cell potential towards anti-inflammatory activity, promoting M2-type macrophage polarization and subsequent IL-10 secretion. Leptin is also implicated in the proliferation, differentiation, and activation of natural killer (NK) cells, enhancing their cytotoxic activity by upregulating IL-2, IL-12, and perforin gene expression. Consequently, leptin plays an active role in the initiation and perpetuation of inflammation.

Regarding POA, this common MUFA plays an important role in metabolism. It is a component of triacylglycerols found in all body tissues and is biosynthesized from PA via stearoyl-CoA desaturase-1. Our study observed elevated concentrations of both PA and POA in obese subjects. While pro-inflammatory effects have been described for PA, consistent with metabolic inflammation in obesity, anti-inflammatory properties have been confirmed for POA. Adding POA to LPS-stimulated macrophage cultures reduced the production of IL-1β, IL-6, and TNFα, decreased expression of NFκB, MyD88, caspase-1, and TLR4 [16]. Furthermore, POA has been shown to enhance adipocyte and hepatocyte sensitivity to insulin and, in combination with oleic acid, to beneficially affect lipid metabolism and reduce inflammation [17]. The inverse correlation between leptin and POA levels established in our study suggests a potential role for POA in mitigating the pro-inflammatory effects of leptin in obese adolescents, possibly by regulating its concentration. This mechanism warrants further investigation, taking into account sex, developmental stage, and, in female participants, menarche status and menstrual cycle phase.

Additional correlations of interest involve the hormone resistin and the fatty acids ALA and ADA. In our study, resistin concentrations in obese adolescents were not significantly different from controls. However, a negative correlation was found between resistin and ALA levels (which were elevated in the study group),

and a positive correlation between resistin and ADA levels.

Originally identified as a molecule involved in insulin resistance, resistin is now recognized as an inflammatory regulator, promoting a pro-inflammatory state both *in vitro* and *in vivo* [18]. Produced by adipocytes, monocytes/macrophages, myocytes, cardiomyocytes, and hepatocytes, resistin influences a wide range of cell types through autocrine, paracrine, and endocrine mechanisms. It increases the reactivity of macrophages, mononuclear leukocytes, and endothelial cells, with NF-κB-mediated secretion of TNFα, IL-6, IL-12, and MCP1 demonstrated in response to recombinant human resistin [18]. Resistin levels positively correlate with common inflammatory biomarkers such as CRP, TNFα, and IL-6 in various diseases, and may reflect disease severity [19]. Thus, resistin is considered a pro-inflammatory hormone.

ALA is an essential PUFA and precursor to other omega-3 PUFAs, although its conversion to EPA is limited, and further conversion to DPA and DHA is minimal (no more than 4–8 %). ALA is generally recognized for its beneficial, anti-inflammatory effects. Studies have shown that ALA treatment can reduce fat accumulation in adipocytes, improve glucose homeostasis, regulate lipid metabolism, and reduce insulin resistance [20]. It reduces TNFα levels and inhibits the expression of nitric oxide synthase, cyclooxygenase-2, and TNFα by inhibiting the NF-κB and MAPK pathways. A novel immunomodulatory role for ALA has recently been identified through the formation of oxylipins with pronounced anti-inflammatory properties [21]. ALA supplementation leads to the formation of oxylipins such as 9-HOTrE and 13-HOTrE, which in murine models significantly reduced reactive oxygen species production and inflammatory cytokine expression (IL-1β, TNFα) while increasing IL-10 secretion by macrophages [21]. In human adipocytes, these oxylipins reduced triacylglycerol accumulation and decreased MCP-1 and TNFα production [22]. Therefore, the increased concentration of ALA in the bloodstream of obese adolescents may represent a compensatory mechanism to help maintain PUFA balance and reduce adipose tissue inflammation. This interpretation is indirectly supported by the negative correlation between resistin levels and ALA concentrations in the context of DPA and DHA deficiency.

ADA is a 22-carbon PUFA widely distributed in the adrenal glands, liver, brain, kidneys, and vascular walls, playing a role in regulating inflammation [23]. Studies have shown that ADA contributes to inflammation in the liver and coronary arteries, as well as triacylglycerol accumulation in fibroblasts [23]. Conversely, ADA can function as an epigenetic regulator of TNFα secretion, increasing its methylation and thereby reducing TNFα-mediated inflammation [24]. The positive correlation between ADA levels and resistin concentrations established in this study suggests a possible role for this fatty acid in regulating hormone levels, potentially facilitating its release into the blood [25]. Additionally, we found a negative correlation between ADA and irisin levels, the latter being within control values in obese adolescents.

Irisin is a thermogenic myokine involved in regulating lipolysis and is also a biomarker for metabolic syndrome in pre-pubertal children [26]. Irisin has been demonstrated to reduce concentrations of pro-inflammatory cytokines (TNFα, IL-1β, MIP1α, MIP1β) and increase anti-inflammatory cytokines in the blood, adipose tissue, endothelial cells, and cardiomyocytes. It reduces LDL and triacylglycerol levels, ameliorates endothelial dysfunction, vascular inflammation, and insulin resistance, and exerts anti-inflammatory effects on the heart, liver, lungs, and intestines [27]. Although we did not observe changes in serum irisin levels in obese adolescents, its active role in reducing the inflammatory response in obesity is suggested by its negative correlation with ADA and its positive correlation with POA.

The negative correlation between NA levels and C-peptide concentrations (a marker of endogenous insulin secretion), coupled with the positive correlation between NA and glucagon, may indicate specific negative effects of insulin/C-peptide on nervous tissue in obese individuals. NA is an omega-9 MUFA crucial for myelin biosynthesis in nerve cells, and its synthesis is a rate-limiting step in myelin sheath lipid homeostasis [28]. An NA-enriched diet has been shown to reduce weight gain, improve memory and learning, and reduce inflammation in mice [29]. While insulin has been associated with neuroprotection, its role remains ambiguous. The correlations identified with NA highlight the need for further study into the mechanisms influencing nervous tissue in obesity.

A comprehensive analysis of changes in the fatty acid spectrum in obese adolescents suggests that many of the observed alterations represent a complex of protective and adaptive responses to obesity and metabolic inflammation. The synthesis pathways for omega-3 and omega-6 PUFAs are similar, both utilizing the same desaturase/elongase enzymes and competing for them. Research indicates that PUFAs from these pathways often exert antagonistic effects, particularly through their eicosanoid derivatives. Generally, EPA-derived eicosanoids have anti-inflammatory or less inflammatory effects compared to those synthesized from AA. Despite sufficient levels of the omega-6 precursor LA, obese adolescents in our study exhibited low levels of GLA, DGLA, and AA. The LA/DGLA ratio (an index of delta-6 desaturase activity), reflecting the conversion of LA to DGLA and endogenous omega-6 synthesis, did not differ significantly from normal values. Meanwhile, EPA concentrations were within normal ranges, suggesting either adequate dietary intake or efficient synthesis from the excess ALA precursor. Eicosanoids synthesized from EPA are likely to exert a more anti-inflammatory effect. The deficiency of other omega-3 fatty acids (DPA and DHA) may be attributed to dietary deficit or reduced synthesis from EPA. For instance, obesity

has been associated with reduced delta-5-desaturase activity, required for DPA production. Furthermore, delta-6-desaturase exhibits a stronger affinity for ALA than for tetracosapentaenoic omega-3 PUFA, a precursor in DHA synthesis [30].

This hypothesis is supported by the lower index of subintimal inflammatory response (AA/EPA ratio) observed in the obese group, which indicates a favorable balance of pro- and anti-inflammatory eicosanoids.

Study limitations. The limitations of this study include its single-center design and relatively small sample size. Additionally, the study did not account for menarche status or menstrual cycle phase in girls, ethnic background, or disease duration in adolescents with obesity.

CONCLUSION

A comprehensive analysis of the observed changes in hormonal levels and blood fatty acid profiles in adolescents with obesity suggests that these alterations represent elements of protective, compensatory, and adaptive responses to low-grade inflammation associated with excessive visceral adipose tissue accumulation. The correlations between specific hormones and fatty acids identified exclusively in the obese cohort warrant further investigation and offer promising avenues for the discovery of novel diagnostic and prognostic biomarkers.

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Conflicts of interest

The authors declare no conflicts of interest.

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