Original article

Assessment of the role of resistin in the metabolic disorders in prepubescent obese children

Evaluación de la función de la resistina en los trastornos metabólicos en niños obesos prepúberes

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ABSTRACT

Introduction: Obesity is one of common causes of the formation of insulin resistance, which triggers a cascade of pathological reactions and the formation of metabolic disorders.

Objective: to evaluate the role of resistin in the metabolic disorders in obese children.

Methods: A cross-sectional, one-stage study was conducted of 38 children at the age of 8-10 years old, 3 groups were identified: 1st – children with obesity (n=16), 2nd –with overweight, 3rd – with normal body weight (n=14). Was assessment of serum levels of resistin, 25(OH)D, parathyroid hormone, calcium, phosphorus, alanine

aminotransferase, aspartate aminotransferase, cholesterol, low-density lipoproteins, triglycerides, glucose, insulin, and calculated the index of insulin resistance. The degree of interrelationships was assessed using the Spearman correlation (r). The criterion x2 was used to determine the relationship between two categorical variables.

Results: The serum resistin concentration increased with increasing Body Mass Index ($r=0.9$, $p<0.05$). The median of serum resistin concentration in children with vitamin D deficiency was the highest ($p<0.05$). In obese children a significant positive correlation was found between the level of resistin and the index of insulin resistance, glucose, cholesterol, triglycerides, and the average number of diagnoses per patient; a strong and moderate negative relationship was established between the level of resistin and serum concentrations of P, Mg, Ca, alkaline phosphatase, and iron.

Conclusions: With increasing serum concentrations of resistin, changes in lipid and carbohydrate metabolism significantly increase, and the content of calcium, phosphorus, alkaline phosphatase, and serum iron decreases, which contributes to the formation of diseases associated with obesity.

Keywords: resistin; children; obesity; micronutrients.

RESUMEN

Introducción: La obesidad es una de las causas comunes de resistencia a la insulina, la cual desencadena una cascada de reacciones patológicas y la formación de trastornos metabólicos.

Objetivo: Evaluar la función de la resistina en los trastornos metabólicos en niños obesos.

Métodos: Se realizó un estudio transversal de una sola etapa con 38 niños de entre ocho y diez años. Se identificaron tres grupos: 1: niños con obesidad (n = 16); 2: niños con sobrepeso; 3: niños con peso normal (n = 14). Se evaluaron los niveles séricos de resistina, 25(OH)D, hormona paratiroidea, calcio, fósforo, alanina aminotransferasa (ALT), aspartato aminotransferasa (AST), colesterol,

lipoproteínas de baja densidad (LDL), triglicéridos, glucosa e insulina, y se calculó el índice de resistencia a la insulina. El grado de interrelaciones se evaluó mediante la correlación de Spearman (*r*). Se utilizó la prueba χ^2 para determinar la relación entre dos variables categóricas.

Resultados: La concentración sérica de resistina aumentó con el incremento del índice de masa corporal (r = 0,9; p < 0,05). La mediana de la concentración sérica de resistina en niños con deficiencia de vitamina D fue la más alta (p < 0,05). En niños obesos se encontró una correlación positiva significativa entre el nivel de resistina y el índice de resistencia a la insulina, la glucosa, el colesterol, los triglicéridos y el número promedio de diagnósticos por paciente. Se estableció una correlación negativa fuerte y moderada entre el nivel de resistina y las concentraciones séricas de fósforo (P), magnesio (Mg), calcio (Ca), fosfatasa alcalina y hierro.

Conclusiones: Con el aumento de las concentraciones séricas de resistina, se incrementan significativamente las alteraciones en el metabolismo de lípidos y carbohidratos, y disminuyó el contenido de calcio, fósforo, fosfatasa alcalina y hierro sérico, lo que contribuye al desarrollo de enfermedades asociadas a la obesidad.

Palabras clave: resistina; niños; obesidad; micronutrientes.

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Introduction

Obesity is a heterogeneous group of diseases associated with excessive accumulation of adipose tissue in the body.[1] It is obesity that is one of the most common causes of the formation of insulin resistance (IR), which is a pathophysiological phenomenon that triggers a cascade of pathological reactions and the formation of a complex of disorders and diseases.

Adipose tissue is an endocrine organ whose cells (adipocytes) produce more than 600 different types of hormone–like substances - adipokines. With excessive accumulation of adipose tissue, a change in the profile of adipokines is observed, which causes a violation of metabolic processes in various organs and tissues. Adipokines are involved in a variety of functions, including modulation of energy and appetite, lipid and glucose metabolism, inflammation, etc.[2,3]

The resistin is the one of least studied of all the adipokines. To date, there are not many studies concerning the assessment of the serum resistin concentration in the child and adolescent population, and they are contradictory. Thus, in a study by *Rubin et al*,[4] back in 2008, it was found that an increase in BMI leads to an increase in serum resistin concentration and *Abdelghaffar et al*,[5] in 2010, the relationship between the serum resistin concentration and insulin resistance in obese children was confirmed. However, some researchers did not identify differences in the serum resistin concentration in children with normal body weight and obesity, and also did not confirm the relationship of resistin and IR.[6]

Ordóñez-Díaz et al,[7] considered that children born prematurely have a higher serum resistin concentration in primary school age compared with full-term peers, and explained these differences by adipose tissue dysfunction in premature infants in the neonatal period. *Tariq et al*,[8] found a significant negative correlation between serum resistin concentration and Vitamin D (VD) status in overweight and obese children. At the same time, the authors argue that the effect of resistin on bone tissue and its relationship with vitamin D may be multidirectional, and the mechanisms underlying them are not fully known. *Machura et al*,[9] when assessing the serum resistin content in children with asthma, a higher serum resistin concentration was revealed compared with healthy peers. At the same time, an increase in this indicator in children with asthma was directly correlated with the amount of adipose tissue. A link was also found between the serum resistin concentration and chronic kidney disease in children. Thus, in the work of *Nehus et al*,[10] it was revealed that with a decrease in the glomerular filtration rate, the serum resistin concentration increases.

The mechanisms of the relationship between serum resistin concentration and BMI, insulin resistance, and the frequency of comorbid pathology continue to be studied. The study of this problem in children of prepubescent age is especially relevant, since the prerequisites for the formation of most chronic diseases of adults are laid already in childhood. And understanding the pathogenetic mechanisms of their implementation is the basis for successful prevention of these conditions.

The aim of the study was to assess the serum resistin concentration in children depending on BMI, as well as its role in the formation of metabolic disorders in obese children.

Methods

The study (cross-sectional, one-stage) was conducted on a sample of 38 children with different body weight. Among the examined children there were 17 boys (45.0%) and 21 girls (55.0%) aged from 8 to 10 years old (the average age was 9,55±0,69 years) permanently residing in the city of Ryazan. All children were of the Caucasian race.

The research was carried out on the bases of the Ryazan Regional Child Hospital named after N.V. Dmitrieva" (chief physician - PhD Lebedeva I.N.), the Central Research Laboratory of the Ryazan State Medical University (head - PhD, Associate Professor Nikiforov A.A.).

Criteria for inclusion in the study: age 8-10 years old; satisfactory condition at the time of the study; absence at the time of inclusion in the study of acute or exacerbation of chronic diseases, chronic diseases of the kidneys, liver, gastrointestinal tract; signed informed consent of the patient's parent to participate in the study.

Exclusion criteria: children with organic pathology or genetic syndromes; the presence of obesity due to genetic syndromes (Prader-Willi, Down and others),

endocrine diseases (hypothyroidism, hypercriticism and others) or the hypothalamic-pituitary injuries.

All stages of the study did not contradict the legislation of the Russian Federation and were approved by the local ethics committee of the Ryazan State Medical University (Protocol No. 11 of 06/11/19). Anthropometric measurements were carried out during a medical examination by trained medical professionals in accordance with a standardized protocol developed by World Health Organization (WHO)[11] The physical development of children was assessed using the WHO AnthroPlus program (2009).[12] The following indicators were calculated: the ratio of body weight to age (Weight-for-Age Z-score, WAZ), body mass index to age (BMI-for-Age Z-score, BAZ). The interpretation of the obtained Z-scores values was carried out according to the criteria: malnutrition - at <-2 SDS, low nutrition from -2 to -1 SDS, norm – from -1 to +1 SDS, overweight – at SDS from +1 to +2, obesity - at SDS > +2.[13]

According to anthropometry data, 3 groups were formed: 1 group consisted of obese children (n=16, 8 girls, 8 boys), 2 groups – (n=8, 3 girls, 5 boys) overweight, 3 groups - (n=14, 10 girls, 4 boys) people with normal body weight. The median (Me) of BMI was 19.50 [17.30; 25.30]. The characteristics of the groups are presented in table 1.

All children had serum resistin concentration, serum levels of 25(OH)D, parathyroid hormone (PTH), glucose, insulin, triglycerides (TG), alanine aminotransferase (ALT), aspartate aminotransferase (AST), low density lipoproteins (LDL), cholesterol (HC), calcium (Ca), phosphorus (P), iron (Fe). Blood sampling was carried out on an empty stomach, from the ulnar vein, by a procedural nurse in a manipulation room on the basis of the Ryazan Regional Child Hospital named after N.V. Dmitrieva. The normal value was taken to be the serum resistin concentration in the range of 2.5-14.7 ng/ml, calcium – 2.30-2.75 mmol/l, phosphorus – 1.1-2.0 mmol/l, ALT – up to 40 U/l, AST – up to 40 U/l, LDL – up to 3.4 mmol/l, TG – 0.34-1.48 mmol/l, HC – 2.8-5.5 mmol/l, glucose – 3.4-6.1 mmol/l, serum iron – 9-26 mmol/l. The HOMA-IR was calculated using the formula: (immunoreactive fasting insulin (IRI) x blood glucose per fasting)/22.5 (normally below 3,2 units).[14]

The STATISTICA 12 software package was used for statistical analysis. Continuous variables were presented as medians with an interquartile range (25-75 percentiles), categorical variables – as a percentage. The degree of interrelationships was assessed using the Spearman correlation (*r*). The criterion x2 was used to determine the relationship between two categorical variables. The differences were considered statistically significant at $p<0.05$.

Table 1 - Characteristics of the group examined children

Indicator	Group 1 - children with obesity (n=16)	Group 2 - children with overweight (n=8)	Group 3 - children with normal body weight (n=14)	p_{1-2}	p_{1-3}	p_{2-3}
Age: years, M±SD	9.6±0.6	9.9±0.4	9.3±0.8	0.735	0.378	0.194
Female, n (%)	8 (50.0)	3 (37.5)	10 (71.4)	0.886	0.412	0.270
Male sex, n (%)	8 (50.0)	5 (62.5)	4 (28.6)	0.886	0.412	0.270
Median BMI, kg/m2 [25%; 75%]	25.6 [24.7;27.9]	19.3 [18.7;19.8]	17.1 [15.8;17.8]	0.001	0.000	0.000
Median SDS BMI [25%; 75%]	+2.9 [+2.5;+3.0]	+1.0 [+1.0;+1.3]	+0.2 [-0.4;+0.6]	0.001	0.000	0.000
Comparison of Mean Values Using ANOVA Statistical Significance of Differences Between Two Relative Scores Using the Chi-Square Test Comparison of the median score using the Kruskal-Wallis test for independent samples						

Results

The serum resistin concentration was within the reference values in all examined groups (table 2). However, the median of serum resistin concentration in obese children was 1,2 times higher than in overweight ($p=0.000$) and in 1.7 times higher compared with children with normal BMI ($p=0.000$). Moreover, with an increase in BMI, the serum resistin concentration significantly increased ($r=0.9$, $p<0.05$).

Table 2 - Median of the serum resistin concentration depending on the Z-score of BMI

Median [25%; 75%]	Group 1 (n=16)	Group 2 (n=8)	Group 3 (n=14)	$p_{k-w\ 1-2}$	p_{k-w1-3}	p_{k-w2-3}
Resistin, ng/ml	6.7 [6.3; 7.1]	5.4 [5.3; 5.6]	3.9 [3.2; 4.4]	0.000	0.000	0.000

The serum resistin concentration was 6.7 mmol/l in obese girls, which is 1.4 ng/ml higher than in girls and boys of group 2 (p=0.056) and 2.8 ng/ml higher compared with peers of group 3 (p=0.000).

Estimating the median of the serum resistin concentration depending on serum 25(OH)D concentration we found that this indicator was highest in children with VD deficiency and amounted to 6.2 ng/ml, compared with that in children with insufficient VD-status and its normal serum 25(OH)D concentration (5.9 ng/ml (p=0.410) and 4.1 ng/ml (p=0.000), respectively).

The median of serum resistin concentration in children with overweight and VD deficiency was 2 times higher than in children with normal body weight and VD deficiency (p=0.000), and 1.7 times higher compared with children with normal BMI and sufficient VD-status provision (p=0.055) (figure). Based on the data obtained, it can be said that the increase in the serum resistin concentration is more influenced by BMI than by hypovitaminosis D.

In obese girls, the serum resistin concentration was 6.7 mmol/l, which is 1.4 ng/ml higher than in girls and boys of group 2 (p=0.056) and 2.8 ng/ml higher compared with peers of group 3 (p=0.000).

Estimating the median of the serum resistin concentration depending on VD-status we found that this indicator in children with VD deficiency was the highest and amounted to 6.2 ng/ml, compared with that in children with insufficient and normal VD-status –5.9 ng/ml (p=0.410) and 4.1 ng/ml (p=0.000), respectively.

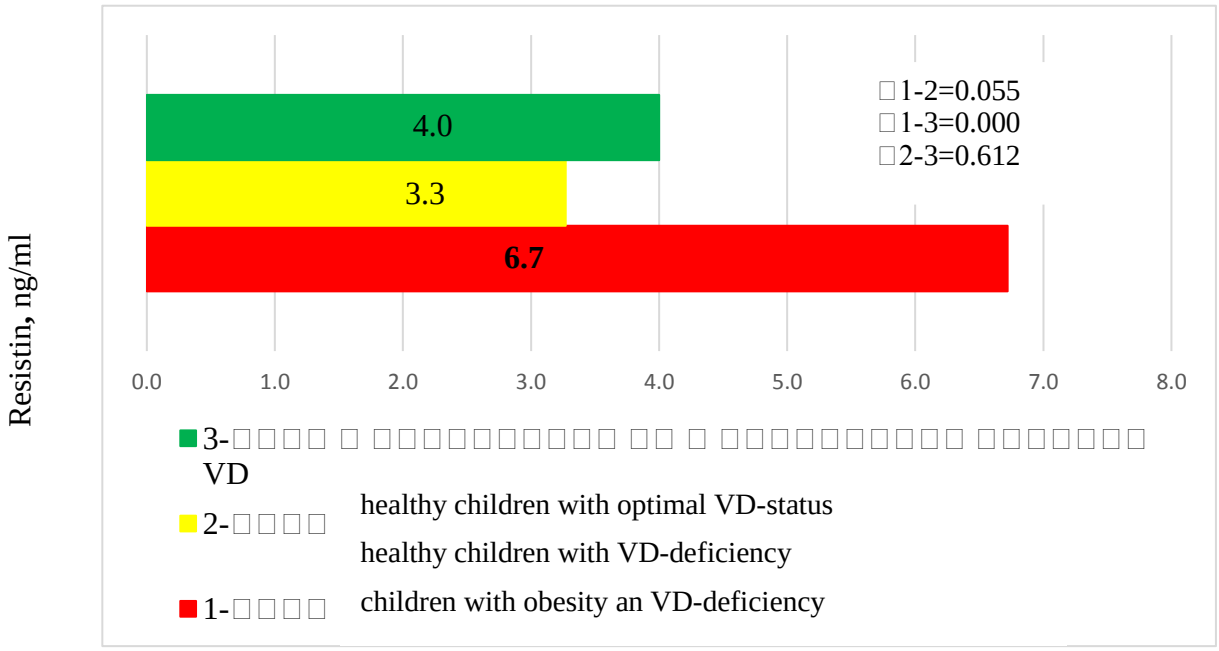

Figure - Median of serum resistin concentration depending on BMI and VD-status.

When assessing the correlations between the serum resistin concentration and indicators of mineral, lipid, carbohydrate metabolism, iron levels and the average number of diagnoses per one child, we found that already at primary school age there is a direct correlation of strong intensity between the level of resistin and BMI, ALT, TG, LDL, glucose, insulin, HOMA index-IR has an average number of diagnoses per person (table 3).

Table 3 - Spearman's correlation coefficients between of the serum resistin concentration, serum 25(OH)D concentration, indicators of mineral, lipid, carbohydrate metabolism, serum iron concentration and the number of diagnoses

Indicator	Serum resistin concentration	
	r	*p*
z-score BMI/age	0.95	<0.05
25(OH)D, ng/ml	-0.61	<0.05
PTH, pg/ml	0.24	≥0.05
AP, U/l	-0.66	<0.05

Ca, mmol/l	-0.63	<0.05
P, mmol/l	-0.70	<0.05
Na, mmol/l	0.32	≥0.05
K, mmol/l	-0.73	≥0.05
Mg, mmol/l	-0.74	<0.05
Fe, μmol/l	-0.73	<0.05
ALT, U/l	0.79	<0.05
AST, U/l	0.64	<0.05
Cholesterol, mmol/l	0.44	<0.05
LDL, mmol/l	0.87	<0.05
TG, mmol/l	0.77	<0.05
Glucose, mmol/l	0.85	<0.05
Insulin, μIU/ml	0.76	<0.05
HOMA-IR	0.84	<0.05
Number of diagnoses, pieces	0.75	<0.05

Leyend: r - is the correlation coefficient between the serum resistin concentration, the number of diagnoses, z-score BMI/age, indicators of mineral, lipid, carbohydrate metabolism.

A direct correlation of moderate intensity was found between the serum resistin concentration and the level of AST, HC, and a moderate negative correlation with the indicators of mineral metabolism (25(OH)D, alkaline phosphatase, Ca, P, Mg) and serum iron concentration.

However, when conducting a comparative assessment of the above indicators and the serum resistin concentration depending on BMI, it was found that only in the group of obese children, the serum resistin concentration had a positive correlation with the NOMA-IR, glucose, cholesterol, TG, LDL and the average number of diagnoses per one child; a negative relationship of strong and moderate intensity was revealed. There was no correlation between the serum resistin concentration and P, Mg, Ca, alkaline phosphatase and iron (table 4). There was no correlation between the serum resistin concentration and the serum 25(OH)D concentration in any group.

Thus, with an increase in BMI, the serum resistance concentration increases significantly. With an increase in the serum resistin concentration, the level of

cholesterol, TG, LDL, glucose increases statistically significantly, the HOMA-IR increases, as well as the average number of diagnoses per child. At the same time, there is a decrease in serum levels of calcium, phosphorus, alkaline phosphatase and iron.

Table 4 - Spearman's correlation coefficients between the of serum resistin concentration, serum 25(OH)D concentration, indicators of mineral, lipid, carbohydrate metabolism, serum iron concentration and the number of diagnoses depending on BMI

Indicators	Serum resistin concentration (ng/ml)					
	Group 1 (n=16)		Group 2 (n=8)		Group 3 (n=14)	
	r	*p*	*r*	*p*	*r*	*p*
25(OH)D, ng/ml	-0.46	≥0.05	0.02	≥0.05	0.31	≥0.05
PTH, pg/ml	0.33	≥0.05	-0.57	≥0.05	-0.14	≥0.05
AP, U/l	-0.69	<0.05	-0.24	≥0.05	-0.26	≥0.05
Ca, mmol/l	-0.73	<0.05	0.07	≥0.05	0.40	≥0.05
P, mmol/l	-0.79	<0.05	-0.11	≥0.05	0.06	≥0.05
Na, mmol/l	0.01	≥0.05	0.17	≥0.05	0.36	≥0.05
K, mmol/l	-0.45	≥0.05	-0.02	≥0.05	0.65	≥0.05
Mg, mmol/l	-0.86	<0.05	-0.73	<0.05	-0.27	≥0.05
Fe, μmol/l	-0.71	<0.05	0.60	≥0.05	-0.40	≥0.05
ALT, U/l	-0.02	≥0.05	0.28	≥0.05	-0.51	≥0.05
AST, U/l	-0.18	≥0.05	0.45	≥0.05	-0.36	≥0.05
Cholesterol, mmol/l	0.75	<0.05	0.25	≥0.05	-0.09	≥0.05
LDL, mmol/l	0.65	<0.05	-0.26	≥0.05	0.16	≥0.05
TG, mmol/l	0.57	<0.05	-0.26	≥0.05	0.25	≥0.05
Glucose, mmol/l	0.77	<0.05	0.24	≥0.05	0.39	≥0.05
Insulin, μIU/ml	0.24	≥0.05	0.02	≥0.05	0.31	≥0.05
HOMA-IR	0.94	<0.05	0.04	≥0.05	0.34	≥0.05
Number of diagnoses, pieces	0.55	<0.05	-0.38	≥0.05	0.51	≥0.05

Discussion

The results of the study of the effect of increasing the serum resistin concentration on the formation of comorbid pathology showed that the value of the HOMA-IR, which plays a role in the development of cardiometabolic diseases, as well as indicators of lipid, calcium-phosphorus metabolism and iron metabolism, were directly related to resistin only in obese children. Currently, the role of this adipokine in the development of insulin resistance and the formation of comorbid pathology is actively discussed, but the results of such studies are contradictory. Our data are consistent with *Farkhondeh et al*,[15] which revealed the presence of a direct high-intensity correlation between serum resistin concentration and the HOMA-IR in obese children.

Currently, it remains controversial whether hypovitaminosis D is a risk factor for impaired glucose homeostasis in children and whether it is associated with the formation of insulin resistance. Thus, *Mohamed et al*,[16] revealed an increase in the serum resistin concentration, which positively correlated with the index of glycated hemoglobin and had an inverse correlation with the serum 25(OH)D concentration in children with type 1 diabetes. The authors have suggested the involvement of VD in the regulation of resistin synthesis. In study, there was no correlation between these indicators in children with different BMI, which may be due to higher levels of serum 25(OH)D concentration in patient groups or the predominant effects of obesity parameters. The data obtained in this investigation are comparable with the conclusions of other researchers.[17,18]

In the group of obese children, a significant negative correlation was found between the serum resistin concentration and indicators of calcium-phosphorus metabolism. This is probably due to the participation of resistin in bone remodeling processes, as well as its significant role in the induction of osteolysis.[19] However, the results obtained require further study.

The results of the study demonstrated the presence of an inverse correlation between the serum resistin concentration and serum iron concentration, which may be due to the participation of resistin in the activation of inflammatory reactions with

further induction of hepcidin synthesis.[20] However, the mechanisms of such interaction require additional research.

In conclusion, with an increase in serum resistin concentration, the serum levels of cholesterol, TG, glucose, HOMA-IR, the number of diagnoses per person significantly increase, while the serum concentrations of calcium, phosphorus, alkaline phosphatase and serum iron decrease, which contribute to the formation of diseases, associated with obesity.

References bibliography

1. Belykh NA, Blokhova EE. Obesity and micronutrient disbalance in children. Science of the young (Eruditio Juvenium). 2019;7(3):429-38 (in Russ)] DOI: https://doi.org/10.23888/HMJ201973429-438

2. Fasshauer M, Blüher M. Adipokines in health and disease. Trends Pharm Sci 2015;36: 461-70.

3. Yakimovich IY, Borodin DA, Podrezov IK, Vasil`ev VN, Kotlovskij MY, *et al*. Influence of physical activities on morphometric parameters of the mesenteric and subcutaneous tissue of rats with obesity, high-fat diet induced. I.P. Pavlov Russian Medical Biological Herald. 2015;23(2): 41-9 (in Russ) DOI: https://doi.org/10.17816/PAVLOVJ2015241-49

4. Rubin DA, McMurray RG, Harrell JS, Hackney AC, Thorpe DE, Haqq AM. The association between insulin resistance and cytokines in adolescents: the role of weight status and exercise. Metabolism. 2008;57:683-90. DOI: https://doi.org/10.1016/j.metabol.2008.01.005

5. Abdelghaffar S, Hafez MH, Shaaban FA, Abou Esmail LA, Salama SI, Rashed RG. Resistin and obesity-associated insulin resistance in children. J Am Sci. 2010;6:256-66. DOI: https://doi.org/10.1016/S1043-2760(01)00522-7

6. Reinehr T, Roth CL, Menke T, Andler W. Resistin concentrations before and after weight loss in obese children. Int J Obes (Lond). 2006;30:297-301. DOI: https://doi.org/10.1038/sj.ijo.0803116

7. Ordóñez-Díaz M, Gil-Campos M, Flores-Rojas K, Muñoz-Villanueva M, Aguilera-García C, Torre-Aguilar M, *et al*. Plasma Adipokines Profile in Prepubertal Children with a History of Prematurity or Extrauterine Growth Restriction Nutrients. Nutrients. 2020;12(4):1201. DOI: https://doi.org/10.3390/nu12041201

8. Tariq S, Tariq S, Khaliq S, Baig M, Murad MA, Lone KP. Association Between Vitamin D and Resistin in Postmenopausal Females With Altered Bone Health. Frontiers in Endocrinology. 2021;11:615440. DOI: https://doi.org/10.3389/fendo.2020.615440

9. Machura E, Szczepanska M, Ziora K, Ziora D, Swietochowska E, Barc-Czarnecka MB, *et al*. Evaluation of Adipokines: Apelin, Visfatin, and Resistin in Children with Atopic Dermatitis Hindawi Publishing Corporation. Mediators of Inflammation. 2013;2013:1-8. DOI: http://dx.doi.org/10.1155/2013/760691

10. Nehus E, Furth S, Warady B, Mitsnefes M. Correlates of Resistin in Children with Chronic Kidney Disease: The Chronic Kidney Disease in Children Cohort. The Journal of Pediatrics. 2012;161(2):276-80. DOI: https://doi.org/10.1016/j.jpeds.2012.01.055

11. World Health Organization Regional Office for Europe: Copenhagen, Denmark. WHO European Childhood Obesity Surveillance Initiative. Protocol. 2016. [accessed 01/02/2022]. Available from: http://www.euro.who.int/__data/assets/pdf_file/0018/333900/COSI protocolen.pdf?ua=1

12. World Health Organization. AnthroPlus for Personal Computers Manual: Software for Assessing Growth of the World's Children and Adolescents; World Health Organization: Geneva, Switzerland; 2009. [access 01/02/2022]. Available from: http://www.who.int/entity/growthref/tools/who_anthroplus_manual.pdf

13. Peterkova VA, Nagaeva EV, Shiryaeva TYo. Assessment of physical development of children and adolescents. Methodological recommendations. Moscow. 2017;1-98. (in Russ). [accessed 10/03/2025]. Available from: https://m.eruditor.one/file/3841609/

14. Peterkova VA, Bezlepkina OB, Kuraeva TL, Laptev DN, Zil'berman LI, Eremina IA, *et al*. Type 2 diabetes mellitus in children. Clinical recommendations. 2020;1-56. (in Russ). [accessed 10/03/2025]. Available from: https://www.endocrincentr.ru/sites/default/files/specialists/science/clinic-recomendations/saharnyy_diabet_2_tipa_deti.pdf

15. Farkhondeh T, Llorens S, Pourbagher-Shahri AM, Talebi M, Shakibaei M, Samarghandian S. An Overview of the Role of Adipokines in Cardiometabolic Diseases. Molecules. 2020;25:5218. DOI: https://doi.org/10.3390/molecules25215218

16. Mohamed M, Tamer A, Alshaymaa A, Huda Marzouk. Serum adipokines and vitamin D levels in patients with type 1 diabetes mellitus. Arch Med Sci. 2017;13(4):738-44. DOI: https://doi.org/10.5114/aoms.2016.60680

17. Lamendola CA, Ariel D, Feldman D, Reaven GM. Relations between obesity, insulin resistance, and 25-hydroxyvitamin D. Am J Clin Nutrition. 2012;95:1055-9.

18. Heras J, Rajakumar K, Lee S, Bacha F, Holick MF, Arslanian SA. 25-Hydroxyvitamin D in obese youth across the spectrum of glucose tolerance from normal to prediabetes to type 2 diabetes. Diabetes Care. 2013;36:2048-53.

19. Thommesen L, Stunes AK, Monjo M, Grøsvik K, Tamburstuen MV, Kjøbli E, et al. Expression and regulation of resistin in osteoblasts and osteoclasts indicate a role in bone metabolism. J Cell Biochem. 2006;99(3):824-34. DOI: https://doi.org/10.1002/jcb.20915

20. Aigner E, Feldman A, Datz C. Obesity as an Emerging Risk Factor for Iron Deficiency. Nutrients. 2014;6:3587-600. DOI: https://doi.org/10.3390/nu6093587

Conflict of interests

The authors declare that there is not conflict of interests.

Authors' contributions

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