Turkish Archives of Pediatrics

ORIGINAL ARTICLE
DOI: 10.5152/TurkArchPediatr.2024.24016

Investigation of Trace Element Levels and Toxic Metals in Obese Children: A Single-Center Experience

Sultan Kaba[1], Sinan Kılıç[2]

[1]Department of Pediatric Endocrinology, Faculty of Medicine, Okan University, İstanbul, Türkiye
[2]Department of Pediatric Surgery, Gebze Yuzyil Hospital, Kocaeli, Türkiye

What is already known on this topic?

- *Obesity is a health problem that threatens public health and is the most important cause of morbidity and mortality.*
- *It has been reported that some trace elements are found to be high in different diseases.*

What this study adds on this topic?

- *Lead (Pb) level was statistically higher in obese patients.*
- *Levels of Mn and Fe may be linked to glucose intolerance.*

ABSTRACT

Objective: The complete role of factors contributing to the pathogenesis of childhood obesity remains to be fully elucidated. Limited research has addressed trace elements in the context of child obesity. Our objective was to assess trace element and lead (Pb), copper (Cu) (are toxic metal) levels in both healthy and obese children, and to investigate the potential correlations between these elements and obesity-related anthropometric measurements, lipid profiles, as well as insulin and glucose levels.

Materials and Methods: Furnace atomic absorption spectrophotometry was employed to measure the concentrations of trace elements in the serum. Additionally, fasting glucose, insulin, and lipid levels were determined in obese children (body mass index ≥ 95th percentile for age and sex), along with 50 healthy children. Only the obesity group underwent an oral glucose tolerance test (OGTT).

Results: Significantly reduced levels of Fe, Mg, Zn, and Co were observed in obese children, whereas Cu, Pb, and Mn levels were elevated ($P < .001$, $P < .001$, $P = .002$, $P = .008$, $P < .001$, $P = .001$, $P = .007$, respectively). Significant positive correlations were found between the 2-hour glucose level in OGTT and Mn ($P = .013$), as well as between peak insulin and insulin levels at the 30th and 60th minutes, and Fe ($P = .001$, $P = .025$, $P = .001$).

Conclusion: This study indicates that an imbalance in trace element levels and the accumulation of Pb may be associated with obesity, while levels of Mn and Fe may be linked to glucose intolerance.

Keywords: Trace elements, toxic metal, child, obesity

INTRODUCTION

Childhood obesity is a common crisis in developed countries, leading to numerous medical complications that elevate the risk of morbidity and premature mortality.[1] Childhood and adolescent obesity is a complex issue, presenting challenges for researchers and healthcare professionals.[2] The global prevalence of obesity, especially among children, is rapidly increasing. Currently, obesity has become a global pandemic, impacting a minimum of 250 million people, which accounts for approximately 7% of the global population.[3] Moreover, the number of overweight people surpasses this figure by at least 2-3 times. This serious condition is associated with increased rates of morbidity and mortality, encompassing cardiovascular conditions, type II diabetes mellitus, and respiratory and orthopedic disorders.[2,3]

Analytics advancements have greatly increased our knowledge of trace elements in the human body's tissues and fluids. Maintaining normal serum trace element levels is associated with a consistently healthy diet.[4] Different studies have shown that being overweight and obese is linked to physical inactivity and unhealthy eating habits.[5-8] Therefore, considering

Corresponding author:
Sinan Kılıç
✉ dr.sinankilic@yahoo.com
Received: January 23, 2024
Revision Requested: February 23, 2024
Last Revision Received: May 7, 2024
Accepted: May 11, 2024
Publication Date: July 1, 2024

Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

Cite this article as: Kaba S, Kılıç S. Investigation of trace element levels and toxic metals in obese children: A single-center experience. *Turk Arch Pediatr.* 2024;59(4):390-396.

that obesity is closely linked to an unhealthy diet, the influence of trace element levels in obese children is not very surprising. As a result, there is a gradual increase in research studies concerning the status of trace elements in both children and adults with obesity.[9-11] Since deficiencies in specific minerals and trace elements within the diet could potentially increase the absorption of harmful metals by certain tissues, it is crucial to examine the levels of lead (Pb), iron (Fe), copper (Cu), zinc (Zn), cobalt (Co), manganese (Mn), and magnesium (Mg) collectively.[12] However, there are very few studies measuring Pb, Fe, Cu, Zn, Co, Mg, and Mn levels together in obese children and analyzing the variations in glucose and insulin levels through an oral glucose tolerance test (OGTT) comparison. Moreover, understanding the relationship between trace element status and metabolic health in obese children is crucial for developing targeted interventions and preventive strategies to mitigate the burden of obesity-related diseases.

Currently, no studies have examined the relationship between obesity and Pb among people.

In this study, our aim is to assess the levels of five trace elements and 2 toxic metals in both obese and healthy children. While the link between obesity and factors like endocrine function, genetics, and diet has been extensively studied, there is still a lack of understanding regarding the influence of environmental factors such as heavy metals on obesity development. Therefore, we examined the blood levels of 7 crucial elements and heavy metals: Fe, Mn, Mg, Cu, Zn, Co, and Pb in obese children. Our aim was to investigate whether there is a connection between obesity and the levels of heavy metals and essential elements in the blood.

Additionally, we aimed to analyze the relationship between trace element levels and indicators of cardiovascular risk, as well as the correlation of total and low-density lipoprotein (LDL) cholesterol, insulin, and glucose levels during basal and OGTT stages.

MATERIALS AND METHODS

In the Department of Pediatric Endocrinology at Van Yüzüncü Yıl University Faculty of Medicine, we meticulously planned and executed this prospective observational study. Approval from the Ethics Committee of Van Yüzüncü Yıl University Faculty of Medicine was secured on January 30, 2014, under issue number 08. Throughout the study, strict adherence to the principles outlined in the Helsinki Declaration was maintained to uphold ethical integrity and participant welfare. Verbal and written informed consent was obtained from the patients' parents who agreed to take part in the study.

Study Design and Participants

The study group was composed of patients who applied to the Pediatric Endocrinology Clinic of Van Yüzüncü Yıl University Faculty of Medicine between January 20, 2014, and July 20, 2014, due to concerns about excess weight. The control group consisted of children who came for regular check-ups at the General Pediatrics Clinic in the same period. The study age range for both groups was 6-17 years.

Among patients diagnosed with obesity, patients with chronic diseases, taking medications, showing syndromic obesity characteristics, short stature, and symptoms suggestive of Cushing's syndrome were excluded from the study. As a result, patients with endocrine-related obesity were excluded from the study. Only patients with exogenous obesity were included.

Initially, 60 patients were included, but later, 10 patients were excluded from the study due to problems such as high thyroid-stimulating hormone (TSH) levels in biochemical and complete blood count, high transaminase levels, anemia, thrombocytopenia, and leukopenia.

Height measurements were taken using a wall-mounted stadiometer while children stood naturally. Body mass was measured with a precision weighing scale accurate to 100 grams, with children wearing only undergarments. Body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in meters. Waist circumference was measured at the narrowest point between the hips and ribs, and hip circumference was measured at the widest point of the hips. All measurements were taken and recorded by the same skilled observer.

Waist circumference/hip ratio, or waist-to-hip ratio, is a measurement used to assess the distribution of body fat. It is calculated by dividing the circumference of the waist by the circumference of the hips. This ratio provides an indication of how fat is distributed around the body, with higher values suggesting more fat around the waist compared to the hips.

Waist circumference/height ratio, or waist-to-height ratio, is a measurement used to assess central obesity and overall health risk. It is calculated by dividing the circumference of the waist by the height of the patient. This ratio provides insight into the distribution of body fat relative to overall height, with higher values indicating greater central obesity and potentially increased risk of metabolic complications such as diabetes and cardiovascular disease.

In both groups, we assessed triglyceride levels, total cholesterol, LDL, high-density lipoprotein (HDL), alanine aminotransferase, aspartate aminotransferase, cortisol, TSH, free triiodothyronine (fT3), free thyroxine (fT4), and glycated hemoglobin (HbA1c).

This analysis was performed using fasting venous blood samples procured during the early hours of the day. Blood sample was collected at 8:30 AM after a 12-hour fast. Serum specimens for trace element quantification were diligently preserved and stored at a frigid temperature of −40°C.

Then, an OGTT was performed on each obese patient who would be able to continue the research study. Before the OGTT, a carbohydrate-rich diet (at least 60% of daily calories or 300 g/day of carbohydrates) or a diet without any restrictions, along with normal activity for at least 3 days, was applied. The test was initiated following a 12-hour fasting period in the morning. Within five minutes after the basal blood sample, a glucose-containing solution (25%-30%) at a dose of 1.75 grams per kilogram (up to a maximum of 75 grams) was administered for the measurement of glucose and insulin. Blood samples were obtained every half-hour, at the 30-minute, 60-minute, 90-minute, and 120-minute marks.

Diagnostic Procedure

Obesity was diagnosed based on a BMI equal to or greater than the 95th percentile, determined using BMI percentile curves provided by the Centers for Disease Control and Prevention (CDC). A group of healthy children was carefully selected for comparison, ensuring they matched the age and sex of the obese group and fell within normal weight parameters. In this study, obesity diagnosis was determined by having a measurement at or above the 95th percentile, as per the criteria established by the World Health Organization.[13]

Blood Samples

Following a fasting duration of 12 hours, morning blood samples were extracted and immediately chilled on ice at a temperature of 4°C. In the subsequent steps, the sera were meticulously separated through centrifugation at 3000 revolutions per minute for a duration of 10 minutes. To facilitate subsequent analyses, these serum samples were then preserved at a temperature of −20°C.

Biochemical Analyses

In the Biochemistry Laboratory, we conducted the analysis of the serum lipid profile using the Bayer Opera Autoanalyzer along with the Biotrol kits. To quantify HgbA1c, we employed the liquid chromatographic high-performance liquid chromatography method (HP Agilent 1200 series equipment in conjunction with the Chrome Systems kit from Germany).

For this purpose, we introduced a mixture of HNO_3 and H_2O_2 (2 : 1) in a volume of 2 mL to the serum samples weighing 0.7 grams. This mixture was then subjected to a water bath set at 70°C for a span of 30 minutes, with intermittent stirring. Afterward, we introduced 1 mL of the identical acid mixture, moving the resulting blend into a Teflon vessel designed for the microwave oven. The vessel was securely sealed, and the solution underwent microwave irradiation for 3 minutes at 450 W. This microwave treatment was replicated using an extra 0.5 mL of the identical acid mixture. Following a 5-minute cooling period, we incorporated 2.0 mL of a 0.1 mol/L HNO_3 solution and shifted the solution into a tube of Pyrex.

The clear solution obtained after centrifugation was used to determine Zn, Co, Mn, Fe, Pb, and Mg levels. These measurements were carried out using the atomic absorption spectrophotometer technique. (UNICAM-929 spectrophotometer: Manufactured by Unicam Ltd, York Street, Cambridge, United Kingdom)

Statistical Analysis

Statistical analysis involved the utilization of the Statistical Package for the Social Sciences version 22.0 software (IBM Corp., Armonk, NY, USA). To perform an evaluation of the data, distribution analysis was carried out to distinguish between parametric and non-parametric traits. The Kolmogorov–Smirnov test was used to determine the distributional properties of the data. Parametric traits were assessed using tests such as the independent samples test. Proportional comparisons between groups were accomplished using Pearson's chi-square test. Statistical significance was defined as $P < .05$.

We used the Spearman correlation coefficient to assess the degree of correlation (Spearman correlation: <0.25 very weak relationship; 0.26-0.49 weak relationship; 0.50-0.69 medium relationship; 0.70-0.89 high relationship; 0.90-1.0 very high relationship).

RESULTS

Demographic and Clinical Characteristics

The study encompassed a total of 50 obese participants (comprising 26 girls and 24 boys) and 50 non-obese control subjects (comprising 23 girls and 27 boys). The obesity group showed no significant differences in terms of age, sex, or pubertal stage compared to the normal-weight children (Table 1).

Compared to the controls, body weight, BMI standard deviation, and waist-to-height ratio were notably higher in children with obesity (respectively $P < .001$, $P < .001$, $P = .001$). Fe, Co, Mg, and Zn levels were notably lower among obese children, while Mn, Cu, and Pb levels were higher. The laboratory values and clinical features of both groups are shown in Table 1.

Statistical Results

No significant correlation was determined between trace elements and obesity anthropometrics (body weight, height, BMI, waist/height ratio, waist/hip ratio), as well as no significant correlation between fasting lipid levels (Tables 2 and 3).

There was a weak relationship between HbA1c and Mn ($r = 0.141$, $P = .017$). There is a positive correlation between insulin and peak insulin levels at the 30th and 60th minutes and Fe ($P = .001$, $P = .025$, $P = .001$). Relationships among each trace element and insulin, glucose values in basal, and OGTT are shown in Table 3.

Table 1. Comparison of Anthropometric Measurements and Trace Elements of Obese and Those of the Control Group

	Obese Mean ± SD	Control Mean ± SD	*P*
Chronological age (years)	11.6 ± 3.0	10.8 ± 3.1	.842*
Sex (girl/boy)	26/24	23/27	.548**
Height age (years)	11.8 ± 3.7	12.8 ± 2.8	.850*
Body weight (kg)	66.18 ± 21.49	41.28 ± 18.17	<.001*
BMI SD	2.13 ± 0.47	1.45 ± 0.20	<.001*
Waist/height ratio	0.65 ± 0.07	0.58 ± 0.04	.001*
Waist/hip ratio	0.95 ± 0.08	0.92 ± 0.06	.01*
Fe (µg/dL)	0.57 ± 0.91	2.11 ± 1.45	<.001*
Mn (µg/dL)	0.66 ± 0.160	0.3310 ± 0.19	<.001*
Mg (µg/dL)	12.05 ± 6.13	22.33 ± 13.82	<.001*
Cu (µg/dL)	2.04 ± 1.38	1.16 ± 0.79	.002*
Zn (µg/dL)	2.43 ± 1.78	3.73 ± 2.45	.008*
Co (µg/dL)	0.03 ± 0.08	0.42 ± 0.73	.001*
Pb (µg/dL)	0.64 ± 0.62	0.34 ± 0.37	.007*
Glucose (mg/dL)	82.5 ± 7.3	63 ± 9.5	.002*
Insulin (mU/L)	19.5 ± 4.6	7.6 ± 2.4	<.001*
Total cholesterol (mg/dL)	139.7 ± 34.8	99.8 ± 25.9	.001*
LDL cholesterol (mg/dL)	87.0 ± 40.1	68.7 ± 19.4	.001*

P-value is significant when less than .05.
BMI, body mass index; SD, standard deviation.
*Independent samples test.
**Pearson's chi-square test.

Table 2. Correlations Between Trace Elements and Anthropometrics in Obese Patients

		Fe	Mn	Mg	Cu	Zn	Co	Pb
Body weight	*r*	0.299	0.040	0.053	0.197	−0.123	−0.001	−0.014
	P	.130	.839	.785	.306	.527	.995	.944
BMI	*r*	−0.349	−0.138	−0.137	0.029	−0.183	−0.133	−0.173
	P	.074	.474	.479	.880	.343	.490	.370
Waist/height ratio	*r*	−0.015	−0.143	0.100	0.250	−0.067	−0.156	−0.081
	P	.957	.570	.693	.316	.792	.538	.748
Waist/hip ratio	*r*	−0.081	−0.028	−0.166	0.422	0.006	−0.007	−0.032
	P	.813	.928	.587	.151	.985	.983	.916

P-value is significant when less than .05.
BMI, body mass index.

Table 3. The Correlation of Total Cholesterol, Low-Density Lipoprotein Cholesterol, Insulin, and Glucose Levels in Basal and Oral Glucose Tolerance Test

		Fe	Mn	Mg	Cu	Zn	Co	Pb
Glu.								
Glu. 0 min.	*r*	0.177	0.300	0.011	0.139	0.124	−0.077	0.238
	P	.368	.107	.953	.464	.512	.686	.205
Glu. 120 min.	*r*	0.954	0.200	0.446	0.234	0.667	0.763	0.960
	P	.023	.432	.056	.256	.213	.290	.186
Hba1c (%)	*r*	0.142	0.141	−0.249	−0.117	−0.112	−0.068	0.076
	P	.910	.017	.767	.172	.259	.120	.326
Ins. 0 min.	*r*	0.370	0.185	−0.025	−0.064	−0.094	−0.231	−0.246
	P	.053	.328	.896	.737	.620	.220	.190
Ins 30 min.	*r*	0.584**	0.206	−0.152	0.040	0.016	−0.027	−0.193
	P	.001	.275	.422	.832	.933	.886	.307
Ins. 60 min.	*r*	0.422*	0.248	−0.077	0.109	−0.054	−0.062	−0.229
	P	.025	.186	.688	.567	.775	.743	.224
Ins. 90 min.	*r*	0.100	0.355	0.111	0.320	0.080	−0.120	−0.234
	P	.611	.055	.559	.085	.676	.527	.214
Ins. 120 min.	*r*	0.223	0.370	−0.041	0.356	0.194	0.356	0.274
	P	.273	.052	.836	.063	.323	.063	.158
Ins. peak.	*r*	0.587**	0.227	−0.135	0.099	0.015	−0.029	0.250
	P	.001	.227	.477	.602	.939	.880	.183
Ins ave.	*r*	0.298	−0.110	−0.324	−0.018	−0.281	0.082	0.261
	P	.216	.646	.163	.941	.230	.731	.266
Total Chol. mg/dL)	*r*	−0.232	−0.071	−0.269	−0.063	−0.204	0.004	0.050
	P	.253	.718	.167	.751	.297	.982	.799
LDL Chol. (mg/dL)	*r*	0.069	−0.044	−0.169	0.037	−0.113	0.057	−0.106
	P	.743	.828	.400	.856	.574	.779	.599

P-value is significant when less than .05.
Chol., cholesterol; Glu., glucose (mg/dL); Ins. ave., insulin average (mU/L); Ins. peak., insulin peak value (mU/L); Min., minute.

DISCUSSION

In the present study, Fe, Mn, Mg, Cu, Zn, Co, and Pb levels were measured in blood samples from obese children and healthy children. Recent studies emphasize the relationship of obesity with micronutrients.[14] Various studies have shown an association between Fe deficiency and obesity.[15] Numerous factors have been associated with reduced Fe levels in patients with obesity. Initially, there was an inclination to attribute compromised plasma Fe concentrations to diets deficient in iron.[16] Nonetheless, a cross-sectional randomized clinical trial, accompanied by a seven-day dietary recall, underscored that hypoferremia defies straightforward explanations through variations in intake of heme or non-heme iron, and other dietary factors that impact Fe accessibility, such as vitamin C content and dietary calcium.[16,17] In more recent times, the elucidation of this phenomenon, often termed the "obesity paradox," has been anchored in the prevailing chronic low-level inflammatory milieu that accompanies obesity; this environment curtails Fe absorption and augments reticulo-endothelial iron sequestration, culminating in the manifestation of anemia characteristic of chronic illnesses.[17,18] Our investigation reaffirms the discernibly reduced serum iron levels within obese children in comparison to their non-obese counterparts ($P < .001$). Recent research suggests that there may be a negative correlation between iron levels and measures of obesity and

LDL cholesterol, but this correlation is not statistically significant.[19] More studies are needed to clarify that iron deficiency in obesity may not be caused solely by nutritional factors. The association between iron deficiency and obesity suggests that inflammation may be adiposity related. Studies on human and animal models have observed that iron is involved in the regulation of insulin and glucose.[20,21] While some studies have determined a negative correlation between Fe and glucose and insulin,[22] there was a positive correlation in our study group. Recent investigations and comprehensive meta-analyses concerning the impacts of Zn supplementation among patients with diabetes have unveiled noteworthy outcomes. These studies highlight that Zn supplementation leads to a reduction in total cholesterol, blood glucose, and LDL cholesterols, concurrently enhancing glycemic regulation as evidenced by a decline in HbA1c levels. Furthermore, the advantages of Zn supplementation extend to ameliorating insulin resistance and addressing key elements of the metabolic syndrome, even within the context of pre-pubertal obese children.[23] In our group of obese children, the considerably low level of Zn compared to healthy controls is consistent with previous studies.

Previous studies have shown that Cu levels are higher in obese children.[11,21] It is known that Cu concentration in erythrocytes is lower than in plasma in healthy children.[24] Erythrocytes maintain a more stable Cu content, while plasma levels can be affected by dietary factors and circadian rhythms.[25,26] The higher levels of Cu found in obese children compared to healthy children may indicate that Cu, as a component of antioxidant enzymes, could indirectly influence fatty acid oxidation and adenosine triphosphate production in mitochondria. However, this association is weak and should be supported by further research.[27]

Studies have revealed that the deficiency of Mn is linked to impaired glucose tolerance and the degradation of serum HDL.[28] The number of studies examining the relationship between Mn and obesity in humans is very limited, and the results are contradictory. In line with our study, although the Mn level is determined to be higher (statistically significant) than in the controls in Turkish obese children and Korean obese adults,[29,30] there are also studies reporting lower Mn levels in obese children.[22] In the study examining Mn levels in whole blood, plasma, and urine samples, while Mn levels in whole blood are lower in obese boys, they were found to be equal in girls. While Mn levels in plasma were low in both boy and girl obese groups, Mn levels in urine samples were higher in both sexes in the obese group.[11]

Although it has been shown in diabetic rats that Co has a positive effect on glucose metabolism and acts as an insulinomimetic,[27,28] human studies examining the influence of Co concentration on obesity are quite limited. Cobalt levels were shown to be low in obese adults and obese Turkish children.[11,28] In our study, it was demonstrated that there was a decrease in serum Co concentration in patients with obesity when compared to the control group.

Lead stands out as a preeminent environmental hazard due to its formidable toxicity, implicated in a multitude of adverse physiological ramifications. It is asserted that it may be associated with pathogenic factors such as oxidative stress, toxicity, interference with neurotransmitters, and effects on the hematopoietic system.[30,31] Mice subjected to Pb exposure spanning from conception through the weaning stage exhibited enduring alterations in adulthood. These modifications encompass escalated food consumption and body weight, along with shifts in energy expenditure, physical activity, glucose tolerance, and insulin responsiveness.[32,33] Studies examining the effect of Pb on obesity are extremely limited. In our study population, Pb levels were significantly higher in the obese group, even though the study on Turkish children did not find it statistically significant.[28] Although not statistically significant, the negative correlation between anthropometrics and Pb levels suggests that Pb exposure may be associated with obesity development.[34]

The assertion has been put forth that diets lacking in Mg are indicative of a propensity for inflammation. Furthermore, the impact of Mg seems to be mediated by its potential to elevate adiponectin secretion.[35] Hypomagnesemia has been determined in obese children.[36,37] Mg levels are significantly lower than in the controls in our patient group. Although not statistically significant, the negative correlations with anthropometrics, insulin, HbA1c, cholesterol, and HDL cholesterol, when evaluated together with the results of previous studies, suggest that Mg deficiency can have an effect on obesity and obesity complications.

The study's findings highlight a potential association between obesity and altered levels of trace elements, alongside a heightened accumulation of Pb. Additionally, our results indicate a potential involvement of Mn and Fe levels in the context of glucose intolerance.

There are several limitations in this current study. First, our study population consists only of obese children in the early stages of obesity (as our patients with the metabolic syndrome were very few in number, they were excluded from the study), and the total number of patients is low. The lack of a statistically significant correlation between many of the trace elements (excluding Fe and Mn) and basal insulin and glucose levels, as well as levels in OGTT, may be due to this limiting aspect of our study. Another limitation of our study is that we could not evaluate the data on nutritional habits on a sufficient scale.

In addition to these limitations, it is important to note the potential impact of diagnostic limited diversity within the study population, which may affect the generalizability of the findings. Furthermore, the exclusion of patients with metabolic syndrome, albeit due to their low representation, could introduce bias and limit the comprehensive understanding of the relationship between trace elements and metabolic parameters.

The findings of this study underscore a potential link between obesity and disrupted trace element levels, along with an increased presence of Pb accumulation. Furthermore, our results suggest that Mn and Fe levels might play a role in the context of glucose intolerance.

In light of the global epidemic of obesity, there is a pressing need for further research on this issue. A more extensive investigation into the relationship between obesity and heavy

metals is essential to mitigate the associated morbidity and mortality risks. Expanding studies in this area will contribute to a better understanding of the complex dynamics involved and facilitate the development of effective strategies for minimizing the health consequences of obesity.

Ethics Committee Approval: The study was approved by the Ethics Committee of Yüzüncü Yıl University Medical Faculty Dursun Odabaş Medical Center (approval number: B.30.2.YYU.0.01.00.00/39, date: January 30, 2014).

Informed Consent: Verbal and written informed consent was obtained from the patients' parents who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept and Design, Data Collection, Analysis and Interpretation of Results – S. Kaba: Draft Manuscript Preparation – S. Kılıç.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: This study received no funding.

REFERENCES

1. Kumar S, Kelly AS. Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc*. 2017;92(2):251-265. [CrossRef]
2. Salman H, Gürsoy Koca T, Dereci S, Akçam M. Comparison of body composition and body mass index in the determination of obesity in schoolchildren. *Turk Arch Pediatr*. 2022;57(5):506-510. [CrossRef]
3. Greydanus DE, Agana M, Kamboj MK, et al. Pediatric obesity: current concepts. *Dis Mon*. 2018;64(4):98-156. [CrossRef]
4. Turconi G, Minoia C, Ronchi A, et al. Dietary exposure estimates of twenty-one trace elements from a total diet study carried out in Pavia, Northern Italy. *Br J Nutr*. 2008;14:1-9.
5. Osredkar J, Geršak ŽM, Karas Kuželički N, et al. Association of Zn and Cu levels in cord blood and maternal milk with pregnancy outcomes among the Slovenian population. *Nutrients*. 2022;14(21):4667. [CrossRef]
6. Miller S, Shirazipour CH, Holmes AF, Salvy SJ, de la Haye K. Diet and physical activity behaviors of families receiving maternal and child health services: the perspective of the home visitor. *J Healthy Eat Act Living*. 2022;2(1):9-22. [CrossRef]
7. Chien YH, Lin CY, Hsu SY, et al. Effects of nonnutritive sweeteners on body composition changes during pubertal growth. *Nutrients*. 2023;15(10):2319. [CrossRef]
8. Nittari G, Scuri S, Petrelli F, Pirillo I, di Luca NM, Grappasonni I. Fighting obesity in children from European World Health Organization member states. Epidemiological data, medical-social aspects, and prevention programs. *Clin Ter*. 2019;170(3):e223-e230. [CrossRef]
9. Washi SA, Ageib MB. Poor diet quality and food habits are related to impaired nutritional status in 13- to 18-year-old adolescents in Jeddah. *Nutr Res*. 2010;30(8):527-534. [CrossRef]
10. Cayir A, Doneray H, Kurt N, et al. Thyroid functions and trace elements in pediatric patients with exogenous obesity. *Biol Trace Elem Res*. 2014;157(2):95-100. [CrossRef]
11. Özdoğan E, Özdoğan O, Altunoğlu EG, Köksal AR. Relationship of blood lipid levels with HbA1c and obesity in patients with type 2 diabetes mellitus. *Med Bull Sisli Etfal*. 2015;49(4):248-254. [CrossRef]
12. Błażewicz A, Klatka M, Astel A, Partyka M, Kocjan R. Differences in trace metal concentrations (Co, Cu, Fe, Mn, Zn, Cd, and Ni) in whole blood, plasma, and urine of obese and nonobese children. *Biol Trace Elem Res*. 2013;155(2):190-200. [CrossRef]
13. World Health Organization. *WHO European Regional Obesity Report 2022*, Geneva.
14. Yanagisawa H. Zinc deficiency and clinical practice--validity of zinc preparations. *Yakugaku Zasshi*. 2008;128(3):333-339. [CrossRef]
15. García OP, Ronquillo D, del Carmen Caamaño M, et al. Zinc, iron and vitamins A, C and e are associated with obesity, inflammation, lipid profile and insulin resistance in Mexican school-aged children. *Nutrients*. 2013;5(12):5012-5030. [CrossRef]
16. Moayeri H, Bidad K, Zadhoush S, Gholami N, Anari S. Increasing prevalence of iron deficiency in overweight and obese children and adolescents (Tehran Adolescent Obesity Study). *Eur J Pediatr*. 2006;165(11):813-814. [CrossRef]
17. Küçük Ö, Biçer S, Uğraş M. Evaluation of the association between obesity and short stature among children living in Yozgat city. *Med Bull Sisli Etfal Hosp*. 2012;46(4):203-207.
18. Greenberg AS, Obin MS. Obesity and the role of adipose tissue in inflammation and metabolism. *Am J Clin Nutr*. 2006;83(2):461S-465S. [CrossRef]
19. Weiss G, Goodnough LT. Anemia of chronic disease. *N Engl J Med*. 2005;352(10):1011-1023. [CrossRef]
20. Gibson RS, Bailey KB, Williams S, et al. Tissue iron deficiency and adiposity-related inflammation in disadvantaged preschoolers from NE Brazil. *Eur J Clin Nutr*. 2014;68(8):887-891. [CrossRef]
21. Suliburska J, Bogdański P, Pupek-Musialik D, Krejpcio Z. Dietary intake and serum and hair concentrations of minerals and their relationship with serum lipids and glucose levels in hypertensive and obese patients with insulin resistance. *Biol Trace Elem Res*. 2011;139(2):137-150. [CrossRef]
22. Biswas S, Tapryal N, Mukherjee R, Kumar R, Mukhopadhyay CK. Insulin promotes iron uptake in human hepatic cell by regulating transferrin receptor-1 transcription mediated by hypoxia inducible factor-1. *Biochim Biophys Acta*. 2013;1832(2):293-301. [CrossRef]
23. Azab SFA, Saleh SH, Elsaeed WF, Elshafie MA, Sherief LM, Esh AMH. Serum trace elements in obese Egyptian children: a case-control study. *Ital J Pediatr*. 2014;40:20. [CrossRef]
24. Jayawardena R, Ranasinghe P, Galappatthy P, Malkanthi R, Constantine G, Katulanda P. Effects of zinc supplementation on diabetes mellitus: a systematic review and meta-analysis. *Diabetol Metab Syndr*. 2012;4(1):13. [CrossRef]
25. Hatano S, Nishi Y, Usui T. Copper levels in plasma and erythrocytes in healthy Japanese children and adults. *Am J Clin Nutr*. 1982;35(1):120-126. [CrossRef]
26. Vitoux D, Arnaud J, Chappuis P. Are cooper, zinc, selenium in erythrocytes valuable biological indexes of nutrition and pathology? *J Trace Elem Med Biol*. 1999;13(3):113-128. [CrossRef]
27. Clouet P, Henninger C, Bézard J. Study of some factors controlling fatty acid oxidation in liver mitochondria of obese Zucker rats. *Biochem J*. 1986;239(1):103-108. [CrossRef]
28. Grochowski C, Blicharska E, Baj J, et al. Serum iron, magnesium, Copper, and Manganese Levels in Alcoholism: a Systematic Review. *Molecules*. 2019;24(7):1361. [CrossRef]
29. Tascilar ME, Ozgen IT, Abaci A, Serdar M, Aykut O. Trace elements in obese Turkish children. *Biol Trace Elem Res*. 2011;143(1):188-195. [CrossRef]
30. Kim HN, Song SW. Concentrations of chromium, selenium, and Copper in the hair of viscerally obese adults are associated with insulin resistance. *Biol Trace Elem Res*. 2014;158(2):152-157. [CrossRef]
31. Nomura Y, Okamoto SI, Sakamoto M, Feng Z, Nakamura T. Effect of cobalt on the liver glycogen content in the streptozotocin-induced diabetic rats. *Mol Cell Biochem*. 2005;277(1-2):127-130. [CrossRef]

32. Li N, Hou YH, Ma DD, Jing WX, Dahms HU, Wang L. Lead accumulation, oxidative damage and histopathological alteration in testes and accessory glands of freshwater crab, Sinopotamon henanense, induced by acute lead exposure. *Ecotoxicol Environ Saf*. 2015;117:20-27. [CrossRef]
33. Andrade VM, Mateus ML, Batoréu MC, Aschner M, Marreilha dos Santos AP. Lead, arsenic, and manganese metal mixture exposures: focus on biomarkers of effect. *Biol Trace Elem Res*. 2015;166(1):13-23. [CrossRef]
34. Hassan SAU, Ahmed I, Nasrullah A, et al. Comparison of serum magnesium levels in overweight and obese children and normal weight children. *Cureus*. 2017;9(8):e1607. [CrossRef]
35. Faulk C, Barks A, Sánchez BN, et al. Perinatal lead (Pb) exposure results in sex-specific effects on food intake, fat, weight, and insulin response across the murine life-course. *PLoS One*. 2014;9(8):e104273. [CrossRef]
36. López-Alarcón M, Perichart-Perera O, Flores-Huerta S, et al. Excessive refined carbohydrates and scarce micronutrients intakes increase inflammatory mediators and insulin resistance in prepubertal and pubertal obese children independently of obesity. *Mediators Inflammm*. 2014;2014:849031. [CrossRef]
37. Alzahrani BA, Badri ZA, Aljuhani JA, Alshamrani RM, Ahmed ME, Jari Alshumrani M. A comparison of magnesium levels in obese versus normal-weight children. *Cureus*. 2023;15(8):e44053. [CrossRef]