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Body Weight and Insulin Resistance Indicators Among Children

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Background: A child's body weight is one of the factors that can contribute to the development of insulin resistance. The aim of the study was to determine the relationship between body weight and selected indicators of insulin resistance in children.

Material/Methods: A cross-sectional study was conducted at the University Children's Hospital in Lublin between 2023 and 2024, involving a group of 99 children aged 8 to 14 years. Anthropometric measurements (body weight and height) were taken, clinical data (sex, age, BMI, classification of obesity), and biochemical indicators (glucose and insulin levels) were analyzed. Insulin resistance was identified using the Homeostatic Model Assessment of Insulin Resistance index and the Quantitative Insulin Sensitivity Check Index.

Results: Insulin resistance was diagnosed in 43.43% of children. Mean insulin values were higher in girls (15.99; median, 12.10) than in boys (11.02; median, 7.86). Individuals with insulin resistance had a higher body mass index (P=0.001), and insulin resistance was more common in children with overweight than in those with normal weight or low weight.

Conclusions: These results confirm that many children and adolescents with overweight have insulin resistance. This condition is associated with an increased risk of developing metabolic disorders and may lead to serious long-term health consequences. An early diagnosis allows the implementation of preventive measures, particularly lifestyle modifications, to reduce the progression of metabolic abnormalities.

Keywords: Body Mass Index • Body Weight • Child • Insulin Resistance

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Introduction

Insulin resistance is characterized by a reduced sensitivity of cells to insulin, which impairs the hormone's ability to perform its physiological functions [1,2]. Consequently, this leads to an increase in blood glucose levels and a compensatory increase in insulin production by pancreatic β cells and the subsequent development of hyperinsulinemia [1].

When pancreatic β cells secrete more insulin than the body requires, the excess insulin binds to cell membranes, which impairs glucose uptake into the cells and causes glucose to remain in the bloodstream [2]. Insulin resistance and chronic exposure to excess glucose and high levels of free fatty acids ultimately leads to the damage of pancreatic β cells, impairing their ability to synthesize and secrete insulin. This results in β-cell apoptosis and the onset of type 2 diabetes [2].

Adipose tissue functions as a key endocrine organ that secretes numerous biologically active substances, such as leptin, adiponectin, tumor necrosis factor α, and monocyte chemoattractant protein 1, collectively referred to as adipocytokines [3]. As body weight increases in the context of obesity, the size of adipocytes increases as well. Enlarged adipocytes and adipose tissue release free fatty acids, reactive oxygen species, and pro-inflammatory cytokines. With expanding adipose tissue, adipocyte hypertrophy occurs due to insufficient oxygen supply [4]. Macrophages surrounding dead adipocytes form a crown-like structure to remove the dead cells. In this way, lipids from dead adipocytes are taken up by macrophages, which disrupts their normal activity, sustaining local inflammation and metabolic disorders [5].

Chronic overproduction of free fatty acids and excessive lipid accumulation during obesity and overnutrition lead to lipotoxicity in insulin-sensitive tissues, including the skeletal muscle, liver, pancreas, and heart [5]. Insulin resistance arises not only from increased adipose mass but also from inflammation driven by ectopic lipid deposition. Ectopic lipids generate toxic intermediates, such as ceramides and diacylglycerols, which disrupt insulin signaling by activating protein kinase C, c-Jun N-terminal kinase, and the inhibitor of nuclear factor kappa B kinase complex, inducing oxidative stress, endoplasmic reticulum stress, and apoptosis [6]. Ceramides play a central role in lipotoxic insulin resistance, thereby impairing glucose uptake and lipid metabolism. Notably, insulin resistance itself promotes hepatic de novo lipogenesis [7], creating a feed-forward cycle that exacerbates ceramide accumulation and metabolic dysfunction [5].

The incidence of insulin resistance among children has risen markedly in recent years [8,9]. This increase is associated with multiple factors, including higher rates of obesity in children and adolescents, lifestyle changes, and reduced physical activity driven by sedentary behaviors such as prolonged television viewing and excessive use of social media [10,11].

Insulin resistance is a critical precursor to various metabolic disorders. Early recognition and intervention are essential to mitigate the risk of progression to more serious complications [8], including hypertension, hyperlipidemia, cardiovascular disease, and type 2 diabetes [9-11]. Insulin resistance in childhood is associated with cardiometabolic risk [12], which increases the likelihood of developing dyslipidemia [13]. Accurate and early detection of insulin resistance is equally important to delay or prevent the onset of prediabetes and type 2 diabetes [14].

In this study, we aimed to investigate the associations between children's body weight and 2 selected indicators of insulin resistance, the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index and the Quantitative Insulin Sensitivity Check Index (QUICKI), stratified by age group and sex.

Material and Methods

Study design

The research project was based on a cross-sectional clinical observational study conducted at a single center between 2023 and 2024, involving the analysis of children's medical records, assessment of biochemical indicators and anthropometric measurements. Laboratory test results were collected from patients, including fasting glucose and insulin measurements. The records were analyzed using a convenience sample. The research concept received a positive opinion from the Bioethics Committee (No. KE-0254/14/01/2023) of Medical University of Lublin. Anthropometric measurements, such as body weight and height, were taken using an electronic column scale with a height gauge. Body mass index (BMI) was calculated using the formula: BMI=body weight (kg)/height $(m)^2$.

Based on BMI percentile values, 4 subgroups of patients were identified: (1) patients with low body weight (3^{rd} to 15^{th} percentile); (2) patients with normal body weight (15^{th} to 85^{th} percentile); (3) patients with overweight (85^{th} to 97^{th} percentile); and (4) patients with obesity (above the 97^{th} percentile). Based on the BMI, analysis, 19 children had low body weight, 50 had normal weight, 28 had overweight, and 2 had obesity.

The inclusion criteria were children aged 8 to 14 years and hospitalized children. The exclusion criteria were children with underweight, active inflammation in the body, secondary hypertension, renal defects, renal hypertension, previous cardiac surgery, previous organ transplantation, aortic valve defects, bicuspid aortic valve defects, female sex with Turner

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syndrome, May-Thurner syndrome, genetic defects, cardiomyopathy, long-term steroid therapy, current oncological treatment, current treatment with beta-blockers, current treatment for hyperthyroidism or hypothyroidism, diabetes, treatment with growth hormone, attention-deficit/hyperactivity disorder, cerebral palsy, myopathies, psychomotor retardation, cardiac arrhythmias visible on routine ECG, second-degree or higher heart block, pacemakers, psychomotor disorders, autism, depression or anxiety, lack of fluency in Polish, residence in an orphanage, and post-traumatic stress disorder.

During the study, circumstances arose that affected the selection process. For instance, some participants withdrew, some developed inflammation and were ineligible, and in some cases, samples could not be collected due to patient discharge.

Percentile Charts Used

The OLA and OLAF percentile charts were used to assess the physical development of patients. These charts are tools used to assess the physical development of children and adolescents aged 3 to 18 in Poland [15]. The OLAF calculator was also used to calculate accurate percentiles for height, weight, and BMI for children aged 6 to 18 years [15]. This calculator was developed based on data obtained from the nationwide epidemiological study PL0080 OLAF, representative of the national population, conducted and coordinated throughout the Poland by the Department of Public Health of the Children's Memorial Health Institute [15].

Biological Material

Blood samples were drawn from the children's elbow veins in the morning, during vein puncture for diagnostic testing, and after obtaining a written consent from the parents. Due to the planned fasting glucose measurement, blood samples were taken from the children after a 12-hour overnight fasting.

Glucose levels were measured using the spectrophotometric method with Cobas Pro device. Insulin levels were taken using the Elecsys Insulin test from Roche using the ECLIA method using a Cobas device. The laboratory responsible met the requirements of ISO 9001: 2015 in the field of medical laboratory diagnostic services.

Because of the physiological insulin resistance observed during puberty, interpreting the HOMA-IR index in children and adolescents is not straightforward [16]. In pediatric populations, HOMA-IR values are adjusted for age and sex, as insulin resistance is a normal component of growth and maturation and varies according to developmental stage. It is assumed that insulin resistance in children is indicated by: HOMA-IR value greater than 2.67 in boys and greater than 2.22 in girls before puberty [17,18]. During puberty, which is generally considered to occur between the ages of 12.5 and 15 years for boys and 10.5 and 13 years for girls [18], the normal HOMA-IR is higher and can reach values of greater than 5.22 in boys and greater than 3.82 in girls [17,18]. The HOMA-IR cut-offs used in the present study were derived from research conducted by Kurtoglu et al [17] and have been applied in multiple international pediatric studies for classifying insulin resistance [19-21].

The HOMA-IR index was calculated using the following formula: HOMA=(fasting insulin (μIU/mL)×fasting glucose (mg/dL))/405 [22]. For the HOMA-IR index, higher values indicate greater insulin resistance. The QUICKI was calculated using the following formula: QUICKI=1/(log(fasting insulin μIU/mL)+ log(fasting glucose mg/dL)) [23]. For the QUICKI index, lower values indicate greater insulin resistance, with insulin resistance commonly defined as a QUICKI value below 0.34 [22].

Statistical Analysis

The dependence of the qualitative variables was assessed using the chi-squared test. The normality of the variable distributions in the studied groups was calculated using the Shapiro-Wilk test. The Mann-Whitney test was used to compare the results between the 2 groups. The Pearson correlation coefficient was used to assess the linear dependence of quantitative variables. Univariate and multivariate logistic regression analysis was used to evaluate the expected relationships. Variable relationships were visualized using scatterplots with locally estimated scatterplot smoothing curves. A significance level of $P<0.05$ indicated statistically significant differences or relationships. The analyses were performed using Statistica 9.1 (StatSoft, Poland) and PQStat 1.8.2.

Data on lifestyle behaviors, such as diet, physical activity levels, and time spent sitting, were not included in the statistical analyses of this study.

Results

Characteristics of the Research Group

The study involved 61 girls (61.62%) and 38 boys (38.38%) aged 8 to 14 years. Of the girls, 70.49% had slim or normal weight, and 29.51% had overweight or obesity. In contrast, 68.42% of the boys had slim or normal weight, and 31.58% had overweight or obesity. The characteristics of the study participants are presented in **Table 1**.

Table 2 shows the comparison of biochemical parameters and BMI between children with slim and normal weight (group A) and those with overweight and obesity (group B). The results

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Table 1. Characteristics of the study participants according to sociodemographic indicators and insulin resistance indicators (n=99).

		n	%	Mean	SD	Min	Q1	Median	Q3	Max
Sex	Girls	61	61.62							
	Boys	38	38.38							
Age				11.81	2.09	7.99	9.97	11.75	13.81	14.98
Body weight	Percentile			55.04	32.14	0.80	22.00	59.50	83.50	99.20
	Z-score			0.16	1.14	-2.40	-0.77	0.24	0.97	2.40
Growth	Percentile			50.57	30.72	0.30	20.90	46.50	80.10	99.90
	Z-score			0.02	1.09	-2.76	-0.81	-0.09	0.84	3.11
BMI	Thin	19	19.19							
	Normal weight	50	50.51							
	Overweight	28	28.28							
	Obesity	2	2.02							
	Percentile			55.70	33.14	3.70	21.60	62.20	87.60	98.10
	Z-score			0.20	1.11	-1.79	-0.79	0.31	1.16	2.07
Glucose	Below 70	6	6.06							
	70-99	90	90.91	84.71	11.06	49.00	81.00	85.00	89.00	131.00
	Above 99	3	3.03							
Insulin	Below 2.60	5	5.05							
	2.60-24.90	83	83.84	14.08	15.38	0.42	5.99	10.10	16.30	121.00
	Above 24.90	11	11.11							
HOMA-IR	Norm	56	56.57							
	Insulin resistance	43	43.43							
	Girls			3.61	4.89	0.12	1.54	2.61	3.67	34.96
	Boys			2.53	3.06	0.05	1.04	1.67	2.81	17.89
QUICKI	Norm	55	55.56	0.35	0.07	0.26	0.32	0.34	0.37	0.76
	Insulin resistance	44	44.44							

SD, standard deviation; Min, minimum value; Q1, lower quartile; Q3, upper quartile; Max, maximum value; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index; QUICKI, Quantitative Insulin Sensitivity Check Index.

showed differences between insulin levels and BMI (P=0.006) between the HOMA-IR index and BMI (P=0.003 for boys) and between the QUICKI and BMI (P=0.008).

Table 3 summarizes the comparison of the results between patients with and without insulin resistance, classified according to the HOMA-IR and QUICKI indices. The Mann-Whitney test was used for group comparisons.

Table 4 shows the correlations between BMI and insulin resistance as assessed by the HOMA-IR and QUICKI indices. Pearson correlation coefficients were used to determine the strength and direction of these associations. BMI showed a significant correlation with higher HOMA-IR values and lower QUICKI values, indicating reduced insulin sensitivity. BMI also showed a significant correlation with fasting insulin concentrations, whereas no significant correlation was observed between BMI and fasting glucose levels.

Table 5 shows the relationship between insulin resistance, sex, and BMI categories. The chi-squared analysis revealed significant differences across sex and BMI categories for both the

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Table 2. Comparison of glucose, insulin, HOMA-IR, and QUICKI measurements based on BMI levels.

Analysed variable	Group	n	M	SD	Min	Q1	Me	Q3	Max	Group comparison
Glucose	A	69	84.23	11.00	49.00	79.00	85.00	88.00	131.00	Z=-0.351 *P*=0.726
	B	30	85.80	11.31	54.00	81.00	85.00	90.00	117.00	
Insulin	A	69	11.51	9.70	0.42	5.16	8.25	15.10	55.30	Z=-2.730 *P*=0.006
	B	30	19.99	22.96	1.23	9.86	13.50	19.80	121.00	
HOMA-IR (girls)	A	43	2.69	1.91	0.12	1.16	2.19	3.47	8.79	Z=-1.255 *P*=0.210
	B	18	5.82	8.26	0.16	2.09	2.83	6.08	34.96	
HOMA-IR (boys)	A	26	2.35	3.56	0.05	1.01	1.47	1.88	17.89	Z=-2.984 *P*=0.003
	B	12	2.93	1.55	0.58	1.79	3.09	4.19	5.16	
QUICKI	A	69	0.36	0.08	0.26	0.32	0.35	0.38	0.76	Z=2.639 *P*=0.008
	B	30	0.33	0.05	0.26	0.30	0.33	0.34	0.55	

A: thinness and normal body weight, B: overweight and obesity. SD, standard deviation; Min, minimum value; Q1, lower quartile; Q3, upper quartile; Max, maximum value; BMI, body mass index; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index; QUICKI, Quantitative Insulin Sensitivity Check Index. Z, Mann-Whitney test; *P*, test probability.

HOMA-IR and QUICKI indices. Statistical significance was observed for both sexes (HOMA-IR: *P*=0.007; QUICKI: *P*=0.014) and BMI (HOMA-IR: *P*=0.008; QUICKI: *P*=0.003).

Table 6 shows the results of univariable and multivariable logistic regression analyses for the likelihood of insulin resistance, assessed using the HOMA-IR. In both models, female sex and overweight and obesity were significantly associated with higher odds of insulin resistance. In the univariable model, girls had an odds ratio (OR) of 3.30 (95% CI, 1.37-7.96; *P*=0.008) compared with boys, and children with overweight and obesity had an OR of 3.24 (95% CI, 1.33-7.91; *P*=0.010) compared with children with thinness and normal weight. In the multivariable model, adjusting for both factors simultaneously, the associations remained significant, with an OR of 3.81 (95% CI, 1.49-9.73; *P*=0.005) for girls and 3.78 (95% CI, 1.45-9.84; *P*=0.006) for children with overweight and obesity. These results indicate that female sex and excess body weight are independent predictors of insulin resistance in the studied pediatric population.

Table 7 shows the distribution of insulin resistance, assessed by HOMA-IR, according to sex and BMI categories. The chi-square analysis demonstrated statistically significant differences across sex and BMI categories (chi-square, 16.315; *P*=0.00098), highlighting that overweight and obesity are strongly associated with insulin resistance in both girls and boys, with girls showing a slightly higher prevalence in the overweight obesity group compared with boys.

Relationship between the analyzed variables is further illustrated in **Figure 1** using a scatterplot with both a fitted linear regression line and a locally estimated scatterplot smoothing curve for visualization.

Discussion

Studies indicate that insulin resistance occurs not only in adults but also in children and adolescents; however, its prevalence in pediatric populations remains insufficiently characterized and varies widely across studies [24]. In the present study, insulin resistance was common in a population of children aged 8 to 14 years, affecting approximately 43% to 44% of participants, regardless of whether HOMA-IR or QUICKI indices were used. A systematic review by van der Aa et al summarized population-based studies on the epidemiology of insulin resistance in children and adolescents from 13 countries across Europe, Asia, North America, and South America, reporting prevalence rates ranging from 3.1% to 44% [25]. Similarly, Ling et al reported that one-third (33%) of 13-year-old adolescents in Malaysia with overweight and obesity met the HOMA-IR criteria for insulin resistance [26]. In a Polish study conducted by Skoczeń et al, reduced insulin sensitivity was found in 38.3% of children with obesity aged 10 to 16 years (n=133) [27]. Differences in reported prevalence may be attributable to variations in the study design, age range, pubertal status, and diagnostic criteria used to define insulin resistance.

In our study, higher BMI was significantly associated with elevated insulin concentrations, higher HOMA-IR values, and

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Table 3. Comparison of body weight measurements and BMI in patients without insulin resistance (group A) and patients with insulin resistance (group B) determined on the basis of the HOMA-IR and QUICKI indices.

Analysed variable	Group	n	M	SD	Min	Q1	Me	Q3	Max	Group comparison
HOMA-IR										
Body weight [percentile]	A	56	46.69	31.78	0.80	19.05	46.30	74.85	97.10	Z=-3.000 *P*=0.003
	B	43	65.92	29.56	2.10	41.40	72.90	92.90	99.20	
Body weight [z-score]	A	56	-0.15	1.12	-2.40	-0.88	-0.09	0.67	1.90	Z=-2.997 *P*=0.003
	B	43	0.56	1.04	-2.04	-0.22	0.60	1.47	2.40	
BMI [percentile]	A	56	46.47	33.48	3.70	13.00	42.70	79.40	98.10	Z=-3.477 *P*=0.001
	B	43	67.72	28.87	4.40	45.90	78.30	94.20	98.10	
BMI [z-score]	A	56	-0.13	1.10	-1.79	-1.13	-0.18	0.82	2.07	Z=-3.491 *P*<0.000
	B	43	0.63	0.98	-1.71	-0.10	0.78	1.57	2.07	
QUICKI										
Body weight [percentile]	A	55	45.86	31.45	0.80	18.60	45.00	73.90	97.10	Z=-3.232 *P*=0.001
	B	44	66.53	29.49	2.10	41.90	73.25	92.80	99.20	
Body weight [z-score]	A	55	-0.18	1.11	-2.40	-0.89	-0.13	0.64	1.90	Z=-3.229 *P*=0.001
	B	44	0.58	1.03	-2.04	-0.20	0.62	1.46	2.40	
BMI [percentile]	A	55	45.55	33.07	3.70	12.70	42.30	78.80	98.10	Z=-3.799 *P*<0.001
	B	44	68.38	28.87	4.40	47.25	79.65	94.20	98.10	
BMI [z-score]	A	55	-0.16	1.08	-1.79	-1.14	-0.20	0.80	2.07	Z=-3.813 *P*<0.001
	B	44	0.66	0.98	-1.71	-0.07	0.83	1.57	2.07	

A: no insulin resistance, B: insulin resistance. SD, standard deviation; Min, minimum value; Q1, lower quartile; Q3, upper quartile; Max, maximum value; BMI, body mass index; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index; QUICKI, Quantitative Insulin Sensitivity Check Index. Z, Mann-Whitney test; *P,* test probability.

Table 4. Correlation between glucose, insulin, HOMA-IR and QUICKI indices and BMI in the study participants.

		Glucose	Insulin	HOMA-IR	QUICKI
BMI [percentile]	r	0.168	0.232	0.211	-0.192
	P	0.097	0.021	0.036	0.057
BMI [z-score]	r	0.175	0.253	0.229	-0.209
	P	0.083	0.012	0.023	0.037

HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index; QUICKI, Quantitative Insulin Sensitivity Check Index. R, Pearson's r correlation coefficient; *P,* test probability.

lower QUICKI values, indicating reduced insulin sensitivity in children with excess body weight. This finding is consistent with several previous reports. Niu et al pointed to a correlation analysis revealing a close relationship between the HOMA-IR index and BMI [28], similar to the studies by Nur Zati Iwani et al [29], Daneshzad et al [30] and Romualdo et al [31].

The stage of puberty should be considered a potential unmeasured confounding factor when analyzing the relationship between BMI and markers of insulin resistance. The variables used in the analysis, such as age and sex, can only approximate the stage of sexual development and do not directly measure it.

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Table 5. Relationship between insulin resistance and sex and BMI levels of the patients studied.

			HOMA-IR		χ^2 *P*
			Norm (n=56)	Insulin resistance (n=43)	
Sex	Girls	n	28	33	χ^2=7.356 *P*=0.007
		%	45.90%	54.10%	
	Boys	n	28	10	
		%	73.68%	26.32%	
BMI level	Thinness and normal weight	n	45	24	χ^2=6.937 *P*=0.008
		%	65.22%	34.78%	
	Overweight and obesity	n	11	19	
		%	36.67%	63.33%	
			QUICKI		χ^2 *P*
			Norm (n=55)	Insulin resistance (n=44)	
Sex	Girls	n	28	33	χ^2=5.999 *P*=0.014
		%	45.90%	54.10%	
	Boys	n	27	11	
		%	71.05%	28.95%	
BMI level	Thinness and normal weight	n	45	24	χ^2=8.609 *P*=0.003
		%	65.22%	34.78%	
	Overweight and obesity	n	10	20	
		%	33.33%	66.67%	

HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index; QUICKI, Quantitative Insulin Sensitivity Check Index; BMI, body mass index. χ^2, chi-square test result; *P*, test probability.

Table 6. Logistic regression analysis results for the occurrence of insulin resistance (determined based on HOMA-IR).

	Univariable logistic regression model			Multivariable logistic regression model		
	P	OR	95% CI	*P*	OR	95% CI
Girls (reference: boys)	0.008	3.300	1.368-7.958	0.005	3.807	1.4-0-9.730
Overweight and obesity (reference: thinness and normal weight)	0.010	3.239	1.326-7.907	0.006	3.778	1.451-9.837

HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index. *P*, test probability; OR, odds ratio.

Consistently, children with overweight and obesity were significantly more likely to exhibit features of insulin resistance than their peers with low or normal weight. Hyperinsulinemia resulting from insulin resistance is common in children and adolescents with obesity [32]. Children with overweight and obesity present higher rates of insulin resistance compared with children with normal weight [25]. This association is likely related to the increased adiposity, as excess body fat is physiologically resistant to insulin [23]. However, insulin resistance can also occur in children with normal weight, as demonstrated in the present study, suggesting that elevated body weight is not the sole determinant of impaired insulin sensitivity [23,33].

Insulin sensitivity changes throughout childhood and adolescence. Adolescence is a time of significant and dynamic changes, including alterations to metabolic and hormonal regulation, modification of adipose tissue and its distribution, and processing of increased insulin resistance [34-36]. During puberty, the secretion of sex hormones leads to a significant decrease in insulin sensitivity, accompanied by compensatory increases in insulin secretion [37]. Insulin sensitivity appears to be highest before the onset of puberty, reaches its lowest level in the middle of puberty, and approaches pre-pubertal levels by the end of puberty [34,35]. In healthy adolescents, insulin sensitivity generally normalizes after puberty [38]. However, some studies

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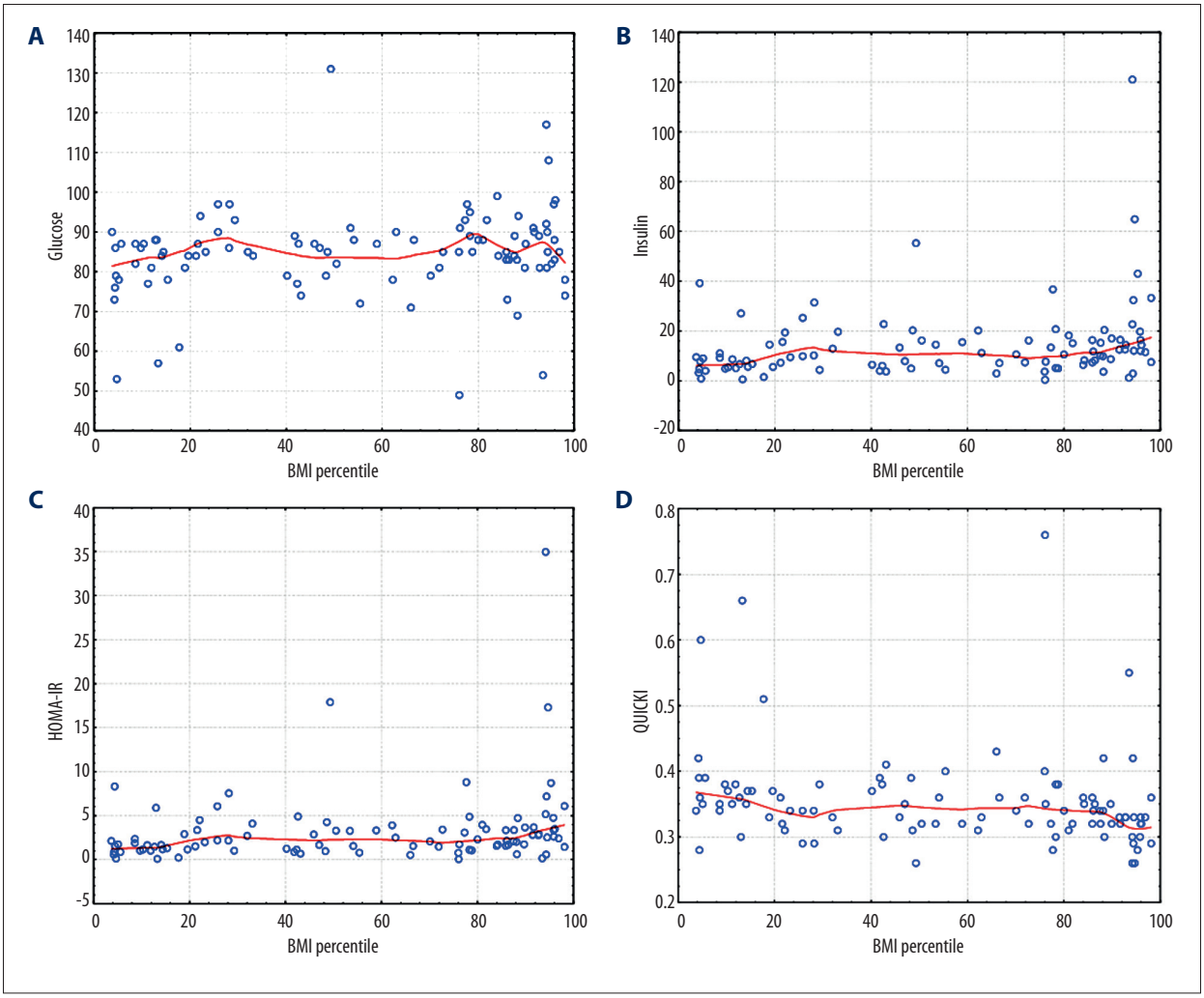
Table 7. Relationship between insulin resistance and sex and BMI levels of the study participants.

			HOMA-IR		χ^2 P
			Norm (n=56)	Insulin resistance (n=43)	
Sex/BMI	Girls, thinness and normal weight	n	22	21	χ^2=16.315 P=0.00098
		%	51.16%	48.84%	
	Girls, overweight and obesity	n	6	12	
		%	33.33%	66.67%	
	Boys, thinness and normal weight	n	23	3	
		%	88.46%	11.54%	
	Boys, overweight and obesity	n	5	7	
		%	41.67%	58.33%	

BMI, body mass index; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance index. χ^2, chi-square test result; *P*, test probability.



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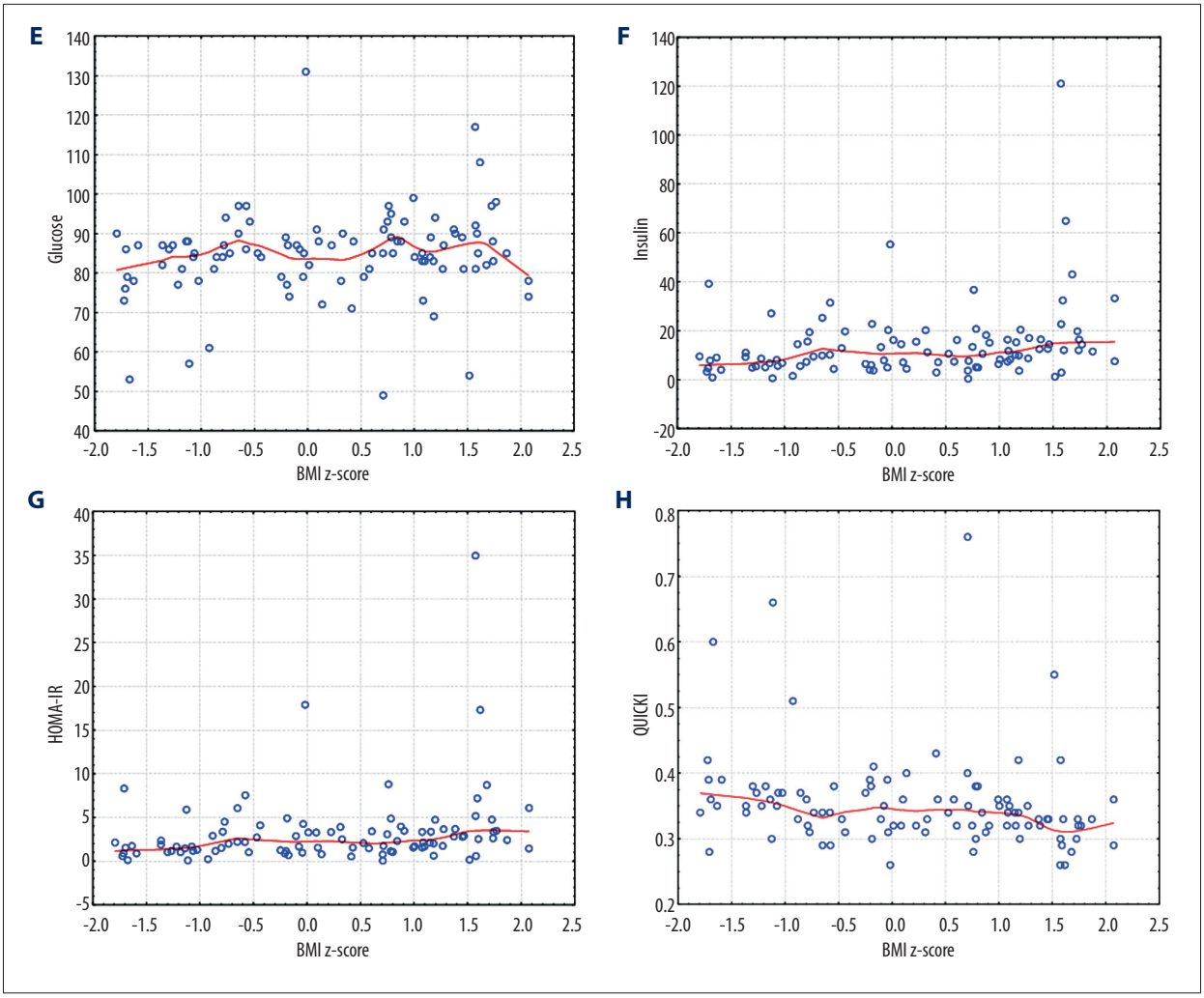

Figure 1. Relationship between the analyzed variables. Scatterplots with locally estimated scatterplot smoothing curves. Panels **A-D** present the relationships between BMI percentile and glucose (**A**), insulin (**B**), Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index (**C**), and the Quantitative Insulin Sensitivity Check Index (QUICKI) (**D**). Panels **E-H** present the corresponding relationships with BMI z-score; glucose (**E**), insulin (**F**), Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index (**G**), and the Quantitative Insulin Sensitivity Check Index (QUICKI) (**H**).

suggest that adolescents with obesity may not completely recover their insulin sensitivity, potentially leading to impaired pancreatic β-cell function [38]. Pubertal status is therefore a key determinant of insulin sensitivity in pediatric populations. In the present study, direct assessment of pubertal stage (eg, Tanner staging) was not available; thus, age and sex were used as proxy variables. This limitation should be considered when interpreting the observed associations between BMI and insulin resistance, as pubertal maturation may partially contribute to the metabolic differences observed between individuals.

Associations between BMI and insulin resistance have been observed in girls and boys, suggesting potential sex-related differences in metabolic profiles during school age and adolescence. In the present study, higher mean insulin concentrations were observed in girls (15.99; median, 12.10) than in boys (11.02; median, 7.86). However, it is notable that sex did not significantly differentiate the BMI z-score (P=0.974). This finding indicates that factors other than body mass alone may contribute to the observed differences in insulin levels.

Sex-related variations in insulin sensitivity during childhood and adolescence have been reported previously and are often attributed to hormonal changes accompanying pubertal development. Some evidence suggests that alterations in estrogen may influence insulin sensitivity [39]. Estrogen deficiency or impaired signaling is associated with insulin resistance and metabolic homeostasis dysregulation [40]. Estrogen may have a beneficial effect on insulin sensitivity through various processes, such as regulating central and peripheral glucose

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homeostasis, body composition, metabolism of adipose tissue, or influence on pro-inflammatory markers [40,41]. Consistent with our findings, Ling et al reported significantly higher HOMA-IR values in girls than in boys [26]. Moreover, a systematic review by van der Aa et al showed that in more than half of the studies reporting sex-specific results, girls were more vulnerable to insulin resistance than boys [25].

However, given the lack of direct assessment of pubertal stage in the present study, the observed sex-related differences should be interpreted with caution, as differences in pubertal timing and hormonal status may partially explain the higher insulin levels observed in girls.

Notably, fasting glucose levels in the present study were not significantly correlated with BMI, while strong associations were observed between BMI and insulin-based indices, suggesting that early metabolic disturbances in children may develop despite normoglycemia.

In our study, we compared the HOMA-IR and QUICKI indices to evaluate their consistency in determining insulin resistance. The simplicity of both the HOMA and QUICKI models lies in the fact that they consider fasting insulin levels and plasma glucose levels [42]. Based on the HOMA-IR index, insulin resistance was identified in 43 patients (43.43%), whereas according to the QUICKI, it was identified in 44 patients (44.44%). These differences are minor and may be due to the fact that the HOMA-IR index interpretation considered the sex and age of the study participants, with particular attention paid to the period of puberty.

Limitations of the Study

There are several important limitations of this study that should be acknowledged. First, the observational, cross-sectional design precludes drawing conclusions about the causal relationship between body weight and insulin resistance. While cross-sectional studies provide valuable population-specific pediatric data, they cannot capture the temporal dynamics of insulin resistance development over time, as longitudinal studies would. Additionally, the absence of direct pubertal stage assessment (eg, Tanner staging) is a notable limitation, given that puberty profoundly influences insulin sensitivity and metabolic parameters. To better elucidate the determinants of pediatric insulin resistance, future studies should incorporate longitudinal designs and more precise measures of body composition and pubertal staging.

Furthermore, the BMI, which is used as a general indicator of adiposity, does not accurately reflect body composition or distinguish between visceral and subcutaneous fat. This limits the precision with which the relationship between fat distribution and metabolic risk can be assessed. Additionally, the small number of children with overweight and obesity in the study sample restricted the possibility of performing more advanced statistical analyses and limits the generalizability of the findings to a broader pediatric population.

Furthermore, national percentile charts (OLA and OLAF) were used to accurately classify physical development within the Polish pediatric population, which can limit direct international comparisons. However, our primary objective was to ensure accurate classification of body weight and physical development within the studied Polish pediatric population. Local growth references are recommended for population-based studies, as they better reflect regional anthropometric characteristics and reduce the risk of misclassification that can occur when international standards are applied.

Although the HOMA-IR cut-offs applied in this study serve as a useful reference, their diagnostic performance can differ across populations, and individual risk estimates should be interpreted with caution. Additionally, the lack of lifestyle data is a significant limitation of the study that can influence the interpretation of the observed associations.

Future Research Directions

Future research on the development of insulin resistance and overweight in children should focus on the following: (1) a longitudinal study design to monitor glucose and insulin changes over time; (2) precise metabolic measurements, including inflammatory markers, advanced body composition assessments, and detailed evaluations of insulin sensitivity to elucidate underlying mechanisms; (3) hormonal and pubertal evaluation to examine hormonal profiles, epigenetic influences, and standardized assessment of pubertal stage (eg, Tanner staging), as puberty significantly affects insulin sensitivity and may contribute to sex-specific differences; and (4) the validation of diagnostic tools, because although HOMA-IR and QUICKI showed high concordance in the present analysis, formal assessment of their diagnostic agreement and clinical implications in pediatric populations remains important.

Conclusions

This cross-sectional study demonstrated that higher BMI was significantly associated with insulin resistance in children and adolescents, consistent with findings from previous research. Although the study design does not allow for causal inference, the findings highlight the importance of early metabolic screening in pediatric populations with excess body weight. The results also underscore the need for longitudinal studies to better understand the development of insulin resistance over

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time. Finally, the comparable performance of HOMA-IR and QUICKI supports their usefulness as practical screening tools for identifying insulin resistance in children and adolescents.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Courtney CH, Olefsky JM. Insulin resistance. In: Mechanisms of insulin action: Medical Intelligence Unit. Springer New York; 2007;185-209
2. Druet C, Tubiana-Rufi N, Chevenne D, et al. Characterization of insulin secretion and resistance in type 2 diabetes of adolescents. J Clin Endocrinol Metab. 2006;91(2):401-4
3. Suganami T, Nishida J, Ogawa Y. A paracrine loop between adipocytes and macrophages aggravates inflammatory changes. Arterioscler Thromb Vasc Biol. 2005;25(10):2062-68
4. Arner E, Westermark PO, Spalding KL, et al. Adipocyte turnover: Relevance to human adipose tissue morphology. Diabetes. 2010;59(1):105-9
5. Ahmed B, Sultana R, Greene MW. Adipose tissue and insulin resistance in obese. Biomed Pharmacother. 2021;137:111315
6. Vázquez-Jiménez JG, Roura-Guiberna A, Jiménez-Mena LR, Olivares-Reyes JA. Role of free fatty acids on insulin resistance. Gac Med Mex. 2023;153(7):M18000092
7. Smith GI, Shankaran M, Yoshino M, et al. Insulin resistance drives hepatic de novo lipogenesis in nonalcoholic fatty liver disease. J Clin Invest. 2020;130(3):1453-60
8. Huang X, Yi K, Jia L, et al. Development and validation of an insulin resistance prediction model in children and adolescents using machine learning algorithms. Transl Pediatr. 2025;14(3):452-62
9. Lukic I, Savic N, Simic M, et al. Risk sssessment and determination of factors that cause the development of hyperinsulinemia in school-age adolescents. Medicina (Lithuania). 2022;58(1):9
10. Cho J, Hong H, Park S, et al. Insulin resistance and its association with metabolic syndrome in Korean children. Biomed Res Int. 2017;2017:8728017
11. Mat SHC, Yaacob NM, Hussain S. Rate of weight gain and its association with Homeostatic Model Assessment-Insulin Resistance (HOMA-IR) among obese children attending Paediatric Endocrine Clinic, Hospital Universiti Sains Malaysia. J ASEAN Fed Endocr Soc. 2021;36:149-55
12. Levy-Marchal C, Arslanian S, Cutfield W, et al. Insulin resistance in children: Consensus, perspective, and future directions. J Clin Endocrinol Metab. 2010;95(12):5189-98
13. Lentferink YE, Elst MAJ, Knibbe CAJ, Van Der Vorst MMJ. Predictors of insulin resistance in children versus adolescents with obesity. J Obes. 2017;2017:3793868
14. Groselj U, Kafol J, Sikonja J, et al. Enhanced oral glucose tolerance test for early detection of insulin resistance and metabolic complications in children with obesity. Am J Prev Cardiol. 2025;23:101016
15. Różdżyńska-Świątkowska A, Kułaga Z, Grajda A, et al. [Height, weight and body mass index references for growth and nutritional status assessment in children and adolescents 3-18 year of age]. Standardy Medyczne Pediatria. 2013;10(1):379 [in Polish]
16. Peplies J, Jiménez-Pavón D, Savva SC, et al. Percentiles of fasting serum insulin, glucose, HbA1c and HOMA-IR in pre-pubertal normal weight European children from the Idefics cohort. Int J Obes. 2014;38:S39-S47
17. Kurtoğlu S, Hatipoğlu N, Mazıcıoğlu M, et al. Insulin resistance in obese children and adolescents: HOMA-IR cut-off levels in the prepubertal and pubertal periods – Original article. J Clin Res Pediatr Endocrinol. 2010;2(3):100-6
18. Goluch-Koniuszy Z, Salmanowicz M. [The importance of nutrition in physiological insulin resistance in adolescents undergoing pubertal growth spurt]. Bromat Chem Toksykol. – L. 2017;1:8-16 [in Polish]
19. Aleman MN, Luciardi MC, Albornoz ER, Bazán MC, Abregú AV. Relationship between inflammatory biomarkers and insulin resistance in excess-weight Latin children. Clin Exp Pediatr. 2024;67(1):37-45
20. Valle MD, Laatikainen T, Potinkara H, et al. Girls and boys have a different cardiometabolic response to obesity treatment. Front Endocrinol (Lausanne). 2018;9:579
21. Lee GY, Lim JH, Joung H, Yoon D. Association between ultraprocessed food consumption and metabolic disorders in children and adolescents with obesity. Nutrients. 2024;16(20):3524
22. Stawerska R, Łupińska A, Bieniek E, Lewiński A. [Insulin resistance and prediabetes—diagnosis and treatment.] Lekarz POZ. 2021;7(5):358-65 [in Polish]
23. Tagi VM, Giannini C, Chiarelli F. Insulin resistance in children. Front Endocrinol (Lausanne). 2019;10:342
24. Krawczyk M, Rumińska M, Czerwonogrodzka-Senczyna A, Pyrżak B. Insulin resistance in children and adolescents – aetiology, complications and diagnostic methods. Pediatria i Medycyna Rodzinna. 2021;17(4):303-9
25. van der Aa MP, Fazeli Farsani S, Knibbe CAJ, et al. Population-based studies on the epidemiology of insulin resistance in children. J Diabetes Res. 2015;2015:362375
26. Ling JCY, Mohamed MNA, Jalaludin MY, et al. Determinants of high fasting insulin and insulin resistance among overweight/obese adolescents. Sci Rep. 2016;6:36270
27. Skoczeń M, Radwański K, Chrzanowska J, Zubkiewicz-Kucharska A. [The occurrence of carbohydrate and lipid metabolism disorders in children with simple obesity depending on age and degree of obesity.] Endocrinol Ped. 2014;4(49):27-36 [in Polish]
28. Niu Y, Tang Q, Zhao X, et al. Obesity-induced insulin resistance is mediated by high uric acid in obese children and adolescents. Front Endocrinol (Lausanne). 2021;12:773820
29. Nur Zati Iwani AK, Jalaludin MY, Roslan FA, et al. Cardiometabolic risk factors among children who are affected by overweight, obesity and severe obesity. Front Public Health. 2023;11:1097675
30. Daneshzad E, Rostami S, Aghamahdi F, et al. Association of cardiometabolic risk factors with insulin resistance in overweight and obese children. BMC Endocr Disord. 2022;22(1):320
31. Romualdo MCDS, De Nóbrega FJ, Escrivão MAMS. Insulin resistance in obese children and adolescents. J Pediatr (Rio J). 2014;90:600-7
32. Luo Y, Luo D, Li M, Tang B. Insulin resistance in pediatric obesity: From mechanisms to treatment strategies. Pediatr Diabetes. 2024;2024:2298306
33. Sinaiko AR, Caprio S. Insulin resistance. J Pediatr. 2012;161(1):11-15
34. Kelly LA, Lane CJ, Weigensberg MJ, et al. Pubertal changes of insulin sensitivity, acute insulin response, and β-cell function in overweight Latino youth. J Pediatr. 2011;158(3):442-46
35. Ball GDC, Huang TTK, Gower BA, et al. Longitudinal changes in insulin sensitivity, insulin secretion, and β-cell function during puberty. J Pediatr. 2006;148(1):16-22
36. Marwitz SE, Gaines MV, Brady SM, et al. Cross-sectional and longitudinal examination of insulin sensitivity and secretion across puberty among non-hispanic black and white children. Endocrinol Metab (Seoul). 2020;35(4):847-57
37. Yan W, Wu S, Liu Q, et al. The link between obesity and insulin resistance among children: Effects of key metabolites. J Diabetes. 2023;15(12):1020-28
38. Kelsey MM, Zeitler PS. Insulin resistance of puberty. Curr Diab Rep. 2016;16(7):64
39. Suba Z. Interplay between insulin resistance and estrogen deficiency as coactivators in carcinogenesis. Pathol Oncol Res. 2012;18(2):123-33

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40. Yan H, Yang W, Zhou F, et al. Estrogen improves insulin sensitivity and suppresses gluconeogenesis via the transcription factor Foxo1. Diabetes. 2019;68(2):291-304
41. Geer EB, Shen W. Gender differences in insulin resistance, body composition, and energy balance. Gend Med. 2009;6(Suppl. 1):60-75
42. Farkas GJ, Gordon PS, Trewick N, et al. Comparison of various indices in identifying insulin resistance and diabetes in chronic spinal cord injury. J Clin Med. 2021;10(23):5591

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