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Original Article

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Diagnostic Accuracy of the Triglyceride–Glucose Derived Indices in Detecting Metabolic Syndrome in Pediatric Patients

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Background: Given the challenges associated with direct insulin measurements, several surrogate markers combining both anthropometric data and laboratory tests (such as glucose and lipid levels) have been developed. This study aims to assess the diagnostic accuracy of the following indices: triglyceride-glucose (TyG) index, TyG index adjusted for body mass index (TyG-BMI), TyG index adjusted for waist circumference (TyG-WC), and TyG index adjusted for waist-to-height ratio (TyG-WHtR), for detecting metabolic syndrome (MetS) in the pediatric population, in comparison with conventional parameters.

Methods: We retrospectively analyzed data from 746 children and adolescents (372 female/374 male) categorized into groups based on BMI z-score. Insulin resistance (IR) surrogates were calculated as: homeostasis model assessment of insulin resistance (HOMA-IR), TyG, TyG-BMI, TyG-WC, and TyG-WHtR. MetS was defined by the presence of at least three of the following components: BMI z-score ≥2 standard deviation and/or WC/height ratio ≥0.5; fasting blood glucose >100 mg/dL and/or pathological HOMA-IR; dyslipidemia; and hypertension.

Results: The overall prevalence of MetS was 15.01% (112/746), occurring exclusively in children with obesity or overweight ($P=0.004$). The third quartile (Q3) was identified as the optimal cut-off point for the TyG indices (TyG, 8.1; TyG-BMI, 231.54; TyG-WC, 734.03; and TyG-WHtR, 4.73). The areas under the receiver operating characteristic curves (AUCs) of TyG, TyG-BMI, TyG-WC, and TyG-WHtR were 0.801 ($P<0.001$), 0.840 ($P<0.001$), 0.832 ($P<0.001$), and 0.816 ($P<0.001$) respectively, comparable to the AUC of HOMA-IR, which was 0.866 ($P<0.001$).

Conclusion: The TyG, TyG-BMI, TyG-WC, and TyG-WHtR indices demonstrated promising predictive value for MetS in children with overweight or obesity. These markers can effectively identify high-risk individuals and offer a cost-effective alternative to direct assessment of insulin sensitivity.

Key words: Triglyceride-glucose index, Metabolic syndrome, Pediatrics, Child, Insulin resistance

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INTRODUCTION

Metabolic syndrome (MetS) significantly increases the risk of complications such as type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVDs). Although its prevalence is increasing worldwide starting from early childhood, a universally accepted consensus on MetS diagnostic criteria is not available.[1,2]

MetS prevalence varies depending on age, gender, ethnic background, and the diagnostic criteria applied. A literature review by Reisinger et al.[3] reported a global prevalence of MetS in adolescents ranging from 0.3% to 26.4%, depending on the diagnostic criteria. Moreover, according to Noubiap et al.,[1] approximately 3% of children aged 6 to 12 years and 5% of adolescents aged 13 to 18 years were diagnosed with MetS worldwide in 2020. These data vary

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across countries and regions. Reported prevalence rates among children and adolescents include 3.1% to 5.4% in the United States, 9.5% in Chile, 1.1% to 7.6% in China, 7.6% in Iran, 3.8% in Spain, and 17% in Italy.[4] MetS prevalence seems to be higher in males than in females, and the risk is higher in children from lower socioeconomic backgrounds. A systematic review by Friend et al.,[5] which analyzed 85 studies on childhood obesity, found that the median prevalence of MetS was 3.3% in the general pediatric population, 11.9% in subjects with overweight, and 29.9% in subjects with obesity, demonstrating a correlation between obesity and MetS.

These findings underscore the urgent need to accurately identify risk factors, to enable the early prediction and prevention of MetS. Insulin resistance (IR) is widely recognized as one of the key risk factors for MetS,[6] as highlighted by both cross-sectional and prospective studies in children. Indeed, IR can also result from an insulin-resistant phenotype that develops during childhood, leading to the early onset of T2DM and hypertension and eventually increases the likelihood of cardiovascular complications. Cruz et al.[7] showed that decreased insulin sensitivity is related to MetS independent of body fatness. Furthermore, another study conducted on pediatric patients reported that children who later developed MetS had higher baseline insulin concentrations.[8] Therefore, early identification of IR in children has been proposed as a valid strategy for identifying those with a high risk of MetS.

The hyperinsulinemic-euglycemic clamp is the gold standard for diagnosing IR;[9] however, its use in epidemiological studies is limited by its invasiveness, cost, and complexity, particularly in pediatric populations. A widely accepted alternative is the homeostasis model assessment of insulin resistance (HOMA-IR), a highly specific and sensitive index that combines fasting blood glucose (FBG) and insulin levels to assess IR.[10] In accordance with this, in children, HOMA-IR model has been reported to be a stronger indicator of MetS than FBG alone.[11]

However, since serum insulin concentrations are not routinely measured in all laboratories, the practical utility of HOMA-IR can be limited.[12] Given the challenges of direct insulin measurement, several surrogate markers have been developed based on glucose concentrations, lipid profiles, and anthropometric parameters. These markers do not require serum insulin measurements and correlate better with the hyperinsulinemic-euglycemic clamp method compared to HOMA-IR.[12] Among these markers, triglyceride-glucose (TyG) index, TyG index adjusted for body mass index (TyG-BMI), TyG index adjusted for waist circumference (TyG-WC), and TyG index adjusted for waist-to-height ratio (TyG-WHtR) have been proposed for their simplicity and practicality.[13]

In adult populations, previous studies have shown associations of these indices with IR and T2DM,[14] MetS,[15] metabolic dysfunction-associated steatotic liver disease,[13] and coronary artery disease,[14] supporting their role as early markers of metabolic derangement.

The role of TyG as a surrogate for IR has also been described throughout the pediatric age range, particularly in children and adolescents with obesity, indicating that it is a valuable tool to identify MetS.[16,17] Furthermore, in adults, combining the TyG index with anthropometric parameters can increase the diagnostic accuracy for MetS and its related complications.[18] Conversely, the diagnostic performance of TyG-BMI, TyG-WC, and TyG-WHtR indices in detecting MetS in pediatric cases has been studied to a limited extent.[19,20]

This study aimed to evaluate the diagnostic accuracy of the TyG index and its anthropometric derivatives (TyG-BMI, TyG-WC, and TyG-WHtR) in comparison with traditional markers for identifying MetS in a pediatric cohort, with a focus on patients with overweight and obesity. These indices offer additional, straightforward, accessible, and cost-effective options for early detection of MetS.

METHODS

Patients

We retrospectively analyzed data from 746 children and adolescents (372 females and 374 males) referred to the outpatients clinics of Children's Hospital Vittore Buzzi (Milan, Italy), for auxological assessment and/or obesity, following referral by their general practitioner or community pediatrician between October 2020 and May 2024.

Patients were classified based on BMI z-score as obesity (BMI z-score ≥2 standard deviation [SD]), overweight (BMI z-score between 1 and 2 SD), and normal weight (BMI z-score between –1 and 1 SD). Inclusion criteria were age 4 to 18 years, any Tanner stage (1 to 5), and no history of acute and/or chronic illness. Exclusion criteria were secondary obesity, ongoing pharmacological

treatment, and acute and/or chronic illness. All participants underwent outpatient clinical evaluations and blood sampling for biochemical analyses.

The study protocol was approved by the Institutional Ethics Committee of Children's Hospital Vittore Buzzi (2020/ST/234 MI) and conducted in accordance with protocol, international Good Clinical Practice (GCP) guidelines, and current clinical research regulations. Written informed consent was obtained from all participants or their legal guardians after full disclosure of the study aims and procedures.

Clinical evaluation

All measurements were recorded using the metric system (cm/kg), following standardized protocols for anthropometric and clinical assessments. Measured parameters were weight, height, BMI, WC, and blood pressure (BP).

As previously described,[12] measurements were performed with subjects barefoot and wearing only underwear. Weight was measured with a scale accurate to ± 100 g, while height was measured with a wall-mounted stadiometer accurate to ± 1 mm. WC was measured with a non-elastic tape positioned horizontally between the lower margin of the last rib and the iliac crest while the patient was standing upright.

BMI was calculated using the formula weight/height^2 (kg/m^2) and expressed as BMI z-scores, according to World Health Organization (WHO) reference values.[21] WHtR was also calculated and used as an indicator of abdominal obesity.[22] Pubertal status was assessed during clinical examination using Tanner staging, with subjects classified as stage 1 (prepubertal), stages 2–3 (early puberty), or stages 4–5 (advanced puberty to adult).[23,24] BP was measured using a sphygmomanometer in accordance with established pediatric guidelines.[25]

Biochemical evaluation

Fasting morning venous blood samples were collected after at least 8 hours of fasting and analyzed for glucose, insulin, lipid profile, and IR markers. The lipid profile included total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), all analyzed with the Cobas 6000 analyzer series (Roche).

Dyslipidemia was defined as LDL-C > 130 mg/dL and/or TG ≥ 100 mg/dL (ages 0–9 years), TG ≥ 130 mg/dL (ages 10–19 years), and HDL-C < 40 mg/dL (females) or HDL-C < 50 mg/dL (males).[26] Glucose metabolism was assessed using concentrations of FBG, insulin, and glycosylated hemoglobin, also using the Cobas 6000 analyzer series.

IR was evaluated using the following surrogate indices:

HOMA-IR, calculated as fasting glucose (mmol/L) × fasting insulin (U/mL)/22.5.

Additional surrogate markers were

- TyG = log[TG (mg/dL) × glucose (mg/dL)/2]
- TyG-BMI = TyG index × BMI
- TyG-WC = [(TyG index) × WC (cm)]
- TyG-WHtR = (TyG index) × WHtR.

As there is no universally accepted definition for pediatric MetS, we adopted a previously established and reported[27,28] classification, defining MetS as the presence of at least three of the following criteria:

- BMI z-score ≥ 2 SD and/or WC/height ratio ≥ 0.5;
- FBG > 100 mg/dL and/or HOMA-IR ≥ 2.5 for prepubertal children or HOMA-IR ≥ 4 for pubertal/postpubertal subjects (corresponding to > 95th percentile, as reported by d'Annunzio et al.[29]);
- TG ≥ 100 mg/dL for ages 0–9 years; ≥ 130 mg/dL for ages 10–19 years;[26]
- HDL-C < 40 mg/dL (for females) or HDL-C < 50 mg/dL (for males);[26]
- Systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 85 mmHg.[30]

To define MetS, we chose not to use the International Diabetes Federation (IDF) definition. Instead, we applied an investigator-defined combination of existing criteria, selected for their relevance to pediatric populations and their ability to address the limitations of the IDF definition, particularly in younger age groups. Specifically, the use of both BMI z-scores and WHtR provided a standardized and age-independent method for identifying central adiposity. As an indicator of gluco-metabolic dysfunction, we examined pathological levels of FBG and/or IR, given that impaired fasting glucose is rare in childhood. IR is known to precede abnormalities in glucose levels and plays a crucial role in the progression from normal

to impaired glucose tolerance.[6,10] While the euglycemic-hyperinsulinemic clamp is considered the gold standard for assessing IR, its invasive and time-consuming nature limits its use in pediatric populations. Given the significant impact of pubertal transition on insulin secretion and sensitivity, we applied age-specific thresholds for TG and pubertal-stage-specific cut-offs for HOMA-IR to better reflect physiological changes during growth.

This strategy aimed to improve early detection of metabolic alterations and to account for developmental variability, enhancing the accuracy and relevance of our IR and MetS classification in children and adolescents.

Statistical analysis

Quantitative data were reported as mean ± SD or median with interquartile range (IQR), while qualitative data were presented as counts and percentages. Differences between patient groups for quantitative variables were analyzed using the one-way analysis of variance test for unpaired data with a significance level set at 95%. The significance of the area under the receiver operating characteristic (ROC) curve (AUC) was assessed using logistic regression. A $P < 0.005$ was considered statistically significant.

The distributions of values were analyzed and compared for each index using subdivision into quantiles, with particular emphasis on the third quartile (Q3), which was the cut-off value for subsequent calculations for the four indices: TyG, TyG-BMI, TyG-WC, and TyG-WHtR.

We emphasized Q3 because, compared to the mean, it is a more stable measure and less influenced by extreme values or outliers, which could otherwise introduce distortions and bias into the results. Additionally, Q3 provides a more representative measure of central tendency in potentially asymmetrical distributions. The Q3 is also preferred over the median, which simply divides the dataset into two equal parts; Q3 is more effective in identifying high-risk subgroups, isolating the upper 25% of patients. Furthermore, Q3 was for each parameter favored over an absolute clinical cut-off due to the absence of a universally accepted pediatric TyG threshold.

Within each group, patients diagnosed with MetS were identified, and a two-entry contingency table (confusion matrix) was constructed for each parameter. The qualitative variables healthy/sick and positive/negative test results were represented in columns and rows, respectively. Diagnostic performance was evaluated by calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).

To further validate diagnostic accuracy, ROC curves were generated for each index and compared with those of traditional MetS diagnostic parameters. Each point on the ROC curve represents a pair of values (sensitivity and 1-specificity) as the cut-off value varies within the IQR. Furthermore, through numerical integration of the trapezoids, the AUC was evaluated for each of the parameters as a further element of comparison. AUC values approaching 1 indicate greater diagnostic accuracy.

In addition, the Youden index was calculated for each parameter to determine the optimal cut-off point. The index provides a global, synthetic measure of the diagnostic quality of a test to support the representation of the ROC curves to identify the best cut-off. The Youden index in relation to the optimal threshold value corresponds to the maximum sum of sensitivity and specificity: $J = \mathrm{Max}_c(\mathrm{sensitivity}_c + \mathrm{specificity}_c - 1)$.

All statistical analyses were performed using Microsoft Excel, with additional specific extensions (e.g., the Solver add-in) and advanced data analysis features enabled through the Data Analysis ToolPak. To ensure accuracy, calculations were partially verified manually and cross-checked using R software (R Foundation for Statistical Computing).

RESULTS

Study pediatric population

The characteristics of children and adolescents are summarized based on BMI z-score category in Table 1. As expected, compared to subjects with normal weight, patients with overweight and obesity showed significantly higher BMI z-score ($P < 0.001$), WC ($P < 0.001$), WHtR ($P < 0.001$), FBG ($P = 0.0089$), HOMA-IR ($P < 0.001$), HDL-C ($P < 0.001$), TG ($P < 0.001$), SBP ($P < 0.001$), and DBP ($P = 0.0012$), TyG ($P < 0.001$), TyG-BMI ($P < 0.001$), TyG-WC ($P < 0.001$), and TyG-WHtR ($P < 0.001$).

Box plot representations of TyG, TyG-BMI, TyG-WC, and TyG-WHtR values are shown in Fig. 1A, alongside the distribution of index values and identification of outliers. Outliers are defined as values above the threshold of Q3+1.5 × IQR or below the threshold

Table 1. Mean metabolic indices according to BMI z-score category

Variable	Obesity (n=451)	Overweight (n=179)	Normal weight (n=116)	Total population (n=746)
Age (yr)*	11.47±2.56	12.30±2.18	11.36±2.48	11.65±2.48
Sex (male/female)	208/243	106/73	58/58	372/374
Weight (kg)*	65.98±20.34	55.75±13.06	38.98±11.15	59.33±20.10
Height (cm)*	150.84±14.56	154.13±12.43	146.47±15.92	150.95±14.48
BMI (kg/m^2)*	28.23±4.65	23.07±2.34	17.73±1.85	25.36±5.50
BMI z-score*	2.77±0.54	1.58±0.28	0.03±0.57	2.06±1.11
WHtR*	0.59±0.06	0.50±0.05	0.43±0.04	0.54±0.08
Fasting blood glucose (mg/dL)	79.11±10.03	78.11±10.08	75.96±9.14	78.38±9.96
HOMA-IR*	2.87±2.62	2.22±1.88	1.05±0.62	2.43±2.34
HDL-C (mg/dL)*	46.29±9.79	49.87±9.99	54.73±10.24	48.45±10.36
Triglycerides (mg/dL)*	80.89±41.08	66.52±31.06	52.49±17.45	73.00±37.54
Systolic blood pressure (mmHg)*	110.62±11.51	107.64±11.53	101.51±11.51	108.48±11.95
Diastolic blood pressure (mmHg)	67.89±8.54	67.06±7.96	64.66±8.47	67.18±8.46
TyG*	7.95±0.48	7.77±0.42	7.54±0.34	7.85±0.47
TyG-BMI*	225.04±42.29	179.47±22.59	133.68±14.92	199.79±49.01
TyG-WC*	703.94±115.42	604.81±85.76	474.67±62.44	644.49±131.91
TyG-WHtR*	4.63±0.69	3.92±0.47	3.24±0.32	4.24±0.81

Values are presented as mean±standard deviation.
*For all parameters marked with an asterisk, the *P*-values obtained from analysis of variance comparing the three groups were <0.001.
BMI, body mass index; WHtR, waist-to-height ratio; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride-glucose; TyG-BMI, TyG index adjusted for body mass index; TyG-WC, TyG index adjusted for waist circumference; TyG-WHtR, TyG index adjusted for waist-to-height ratio.

of Q1–1.5×IQR, where IQR is the IQR. Table 2 presents the distribution of metabolic indices stratified by quantile.

The overall prevalence of MetS in the cohort was 15.01% (112 of 746 patients). Specifically, MetS occurred in 100 children with obesity (89.29%) and in 12 overweight children (10.71%). No cases were observed among children with normal weight. A significant difference in MetS prevalence was also found between sexes, affecting 19.25% of boys and 10.75% of girls ($P=0.004$).

Diagnostic accuracy of TyG, TyG-BMI, TyG-WC, and TyG-WHtR indices

Table 3 presents the specificity, sensitivity, and AUCs of metabolic indices stratified by BMI z-score category (Supplementary Table 1). Table 4 reports the corresponding PPV and NPV.

The AUC values of TyG, TyG-BMI, TyG-WC, and TyG-WHtR in the assessment of MetS were 0.801 (95% confidence interval [CI], 0.760 to 0.841; $P<0.001$), 0.840 (95% CI, 0.803 to 0.877; $P<0.001$), 0.832 (95% CI, 0.793 to 0.870; $P<0.001$), and TyG-WHtR 0.816 (95% CI, 0.777 to 0.856; $P<0.001$), respectively. These values were comparable to that of HOMA-IR, which showed an AUC of 0.866 (95% CI, 0.831 to 0.901; $P<0.001$), as in Tables 3 and 5.

Although PPV and NPV are prevalence-dependent measures of diagnostic accuracy and are not typically subjected to classical significance testing, a comparison was conducted among three independent groups (obesity, overweight, and normal weight) using the chi-square test to compare proportions within subgroups. Regarding PPV, the following *P*-values were obtained: FBG ($P=0.646$); HOMA-IR ($P=0.479$); HDL-C ($P<0.001$); TG ($P=0.168$); SBP ($P<0.001$); DBP ($P=0.273$); TyG, TyG-BMI, TyG-WC, and TyG-WHtR (all $P<0.001$); BMI z-score (not computed); and WHtR ($P=0.061$). For NPV, the *P*-values were as follows: FBG, HOMA-IR, TG, SBP, DBP, TyG, TyG-BMI, TyG-WC, TyG-WHtR, and WHtR (all $P<0.001$); HDL-C ($P=0.029$); and BMI z-score ($P=0.018$).

The Youden indices of all parameters are shown in Table 5 and Supplementary Fig. 1. For parameters with both high AUC and a relatively large Youden index, we noted a good proximity of the chosen threshold value to the theoretical value that maximizes the function of sensitivity q(c) and specificity p(c). This is the cut-off point that most correctly identifies the distinction between healthy and

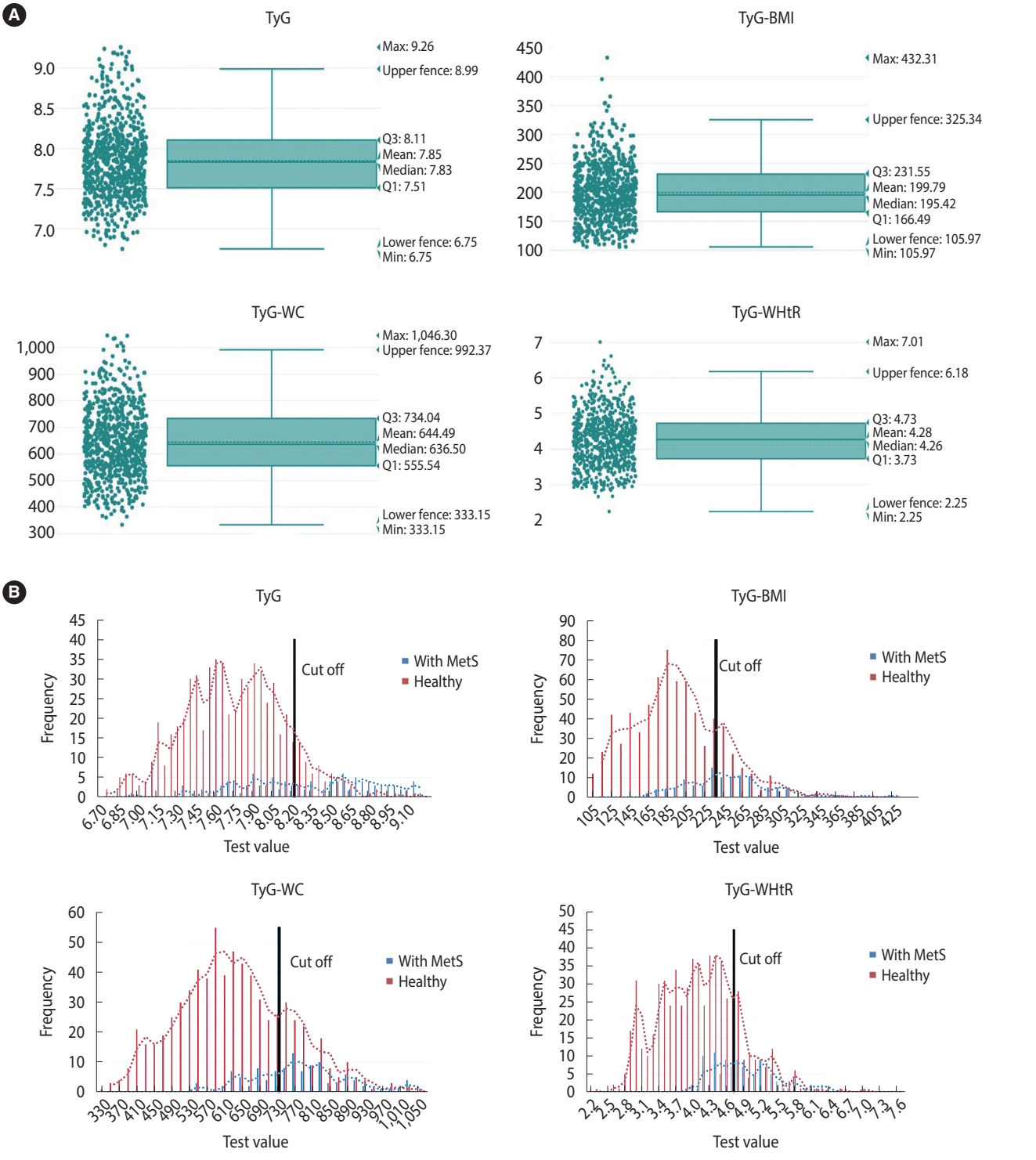

Figure 1. (A) Summary of triglyceride-glucose (TyG) index, TyG index adjusted for body mass index (TyG-BMI), TyG index adjusted for waist circumference (TyG-WC), and TyG index adjusted for waist-to-height ratio (TyG-WHtR) indices. (B) Frequency distribution curves of subjects with or without metabolic syndrome (MetS) according to the presence of pathological TyG, TyG-BMI, TyG-WC, and TyG-WHtR indices. Pathological values are noted in the graphs with vertical black lines: TyG (> Q3 = 8.013), TyG-BMI (> Q3 = 192.487), TyG-WC (> Q3 = 644.449), and TyG-WHtR (> Q3 = 4.217). To analyze whether there were significant differences between the two groups (with or without metabolic syndrome) in the prevalence of pathological values compared to metabolic parameters, a chi-square test was performed. *P*-values lower than 0.001 were obtained for all the indices represented in the figure (TyG, TyG-BMI, TyG-WC, and TyG-WHtR). Q, quartile.

Table 2. Quartile distribution of metabolic indices according to BMI z-score category

Variable	Quartile distribution	Total population	Normal weight	Over-weight	Obesity
WHtR	Minimum	0.294	0.294	0.386	0.455
	Q1	0.490	0.400	0.474	0.543
	Median	0.544	0.422	0.500	0.579
	Q3	0.594	0.459	0.534	0.619
	Maximum	0.833	0.560	0.686	0.833
Blood glucose (mg/dL)	Minimum	41	45	48	41
	Q1	73	71	72	73
	Median	79	77	78	80
	Q3	85	83	84.5	85
	Maximum	125	91	109	125
HOMA-IR	Minimum	0.000	0.222	0.257	0.000
	Q1	1.062	0.533	1.214	1.379
	Median	1.929	0.954	1.926	2.330
	Q3	2.927	1.460	2.673	3.604
	Maximum	31.795	2.848	18.230	31.795
HDL-C (mg/dL)	Minimum	22	34	30	22
	Q1	41	47.25	43	39
	Median	47	55	47	46
	Q3	55	60	57	52
	Maximum	83	83	83	79
Triglycerides (mg/dL)	Minimum	22	22	28	24
	Q1	48	42	45	53
	Median	63	48	60	71
	Q3	84	58	78.5	94
	Maximum	249	142	218	249
Systolic blood pressure (mmHg)	Minimum	80	80	80	80
	Q1	100	90	100	100
	Median	110	100	105	110
	Q3	120	110	115	120
	Maximum	150	140	144	150
Diastolic blood pressure (mmHg)	Minimum	40	40	50	45
	Q1	60	60	60	60
	Median	70	60	70	70
	Q3	70	70	70	70
	Maximum	90	80	85	90
TyG	Minimum	6.751	6.751	6.798	6.900
	Q1	7.509	7.317	7.468	7.615
	Median	7.825	7.572	7.732	7.934
	Q3	8.100	7.780	8.013	8.213
	Maximum	9.261	8.390	9.244	9.261
TyG-BMI	Minimum	105.971	105.971	126.794	139.036
	Q1	166.489	122.258	165.876	195.054
	Median	195.420	132.569	180.752	220.552
	Q3	231.545	143.668	192.387	249.085
	Maximum	432.311	176.589	241.972	432.311
TyG-WC	Minimum	333.154	333.154	387.483	444.547
	Q1	555.533	423.385	555.429	619.543
	Median	636.499	468.493	600.340	691.749
	Q3	734.036	520.036	644.449	780.488
	Maximum	1,046.298	625.680	989.141	1,046.298
TyG-WHtR	Minimum	2.248	2.248	2.845	3.397
	Q1	3.730	3.018	3.575	4.262
	Median	4.275	3.148	3.859	4.601
	Q3	4.730	3.425	4.217	4.992
	Maximum	7.011	4.095	6.341	7.011

BMI, body mass index; WHtR, waist-to-height ratio; Q, quartile; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride-glucose; TyG-BMI, TyG index adjusted for body mass index; TyG-WC, TyG index adjusted for waist circumference; TyG-WHtR, TyG index adjusted for waist-to-height ratio.

affected people. On the contrary, for parameters with lower AUC and Youden index, there is a strong discrepancy between the optimal cut-off and that adopted for the diagnostic test. Note that both AUC and Youden can theoretically have values in the range (0 to 1). For most parameters, the selected cut-off for identifying MetS closely aligned with the most discriminative threshold.

Fig. 1B displays the frequency distribution curves of subjects with or without MetS based on the presence of pathological values for TyG, TyG-BMI, TyG-WC, and TyG-WHtR.

DISCUSSION

Based on our findings, TyG indices combined with anthropometric measurements show a good accuracy in detecting MetS. These indices could be used as screening tools for identifying children and adolescents with obesity or overweight at higher risk of MetS.

MetS is a well-documented predisposing factor for non-communicable diseases such as T2DM and CVD.[2] Although MetS and its related complications are affected by numerous gene-environment interactions, IR is the primary risk factor for MetS. Therefore, early detection of IR is crucial to prevent this condition.[2,4]

The TyG index, derived from circulating TG and glucose concentrations, has emerged as a promising tool for IR assessment, outperforming HOMA-IR in predictive accuracy.[17] Compared to other

Table 3. Specificity and sensitivity of metabolic indices by category of BMI z-score

Parameter	Obesity		Overweight		Normal weight		Total population	
	Specificity	95% CI	Specificity	95% CI	Specificity	95% CI	Specificity	95% CI
Specificity								
Fasting blood glucose (mg/dL)	0.991	0.982–1.000	0.994	0.983–1.000	1.000	1.000	0.994	0.988–1.000
HOMA-IR	0.883	0.853–0.913	0.946	0.913–0.967	1.000	1.000	0.920	0.901–0.939
HDL-C (mg/dL)	0.647	0.603–0.691	0.743	0.679–0.783	0.836	0.769–0.903	0.707	0.674–0.740
Triglycerides (mg/dL)	0.952	0.932–0.972	0.976	0.954–0.990	0.991	0.974–1.000	0.967	0.954–0.980
Systolic blood pressure (mmHg)	0.983	0.971–0.995	0.970	0.945–0.986	0.966	0.933–0.999	0.976	0.965–0.987
Diastolic blood pressure (mmHg)	0.994	0.987–1.000	0.994	0.983–1.000	1.000	1.000	0.995	0.990–1.000
TyG	0.849	0.816–0.882	0.772	0.711–0.811	0.759	0.681–0.837	0.820	0.792–0.848
TyG-BMI	0.843	0.809–0.877	0.772	0.711–0.811	0.759	0.681–0.837	0.826	0.799–0.853
TyG-WC	0.835	0.801–0.869	0.784	0.724–0.822	0.750	0.671–0.829	0.831	0.804–0.858
TyG-WHtR	0.815	0.779–0.851	0.945	0.912–0.966	0.954	0.916–0.992	0.814	0.786–0.842
BMI z-score	0.000	0.000–0.000	1.000	1.000	1.000	1.000	0.446	0.410–0.482
WHtR	0.043	0.024–0.062	0.503	0.430–0.549	0.966	0.933–0.999	0.333	0.299–0.367
Sensitivity								
Fasting blood glucose (mg/dL)	0.030	0.014–0.046	0.000	0.000	0.000	0.000	0.027	0.015–0.039
HOMA-IR	0.690	0.647–0.733	0.667	0.598–0.736	0.000	0.000	0.679	0.645–0.713
HDL-C (mg/dL)	0.900	0.872–0.928	0.917	0.877–0.957	0.000	0.000	0.911	0.891–0.931
Triglycerides (mg/dL)	0.420	0.374–0.466	0.333	0.264–0.402	0.000	0.000	0.411	0.376–0.446
Systolic blood pressure (mmHg)	0.260	0.220–0.300	0.250	0.187–0.313	0.000	0.000	0.259	0.228–0.290
Diastolic blood pressure (mmHg)	0.070	0.046–0.094	0.000	0.000	0.000	0.000	0.063	0.046–0.080
TyG	0.590	0.545–0.635	0.583	0.511–0.655	0.000	0.000	0.643	0.609–0.677
TyG-BMI	0.570	0.524–0.616	0.583	0.511–0.655	0.000	0.000	0.679	0.645–0.713
TyG-WC	0.540	0.494–0.586	0.750	0.687–0.813	0.000	0.000	0.705	0.672–0.738
TyG-WHtR	0.470	0.424–0.516	0.089	0.047–0.131	0.000	0.000	0.607	0.572–0.642
BMI z-score	1.000	1.000–1.000	0.000	0.000	0.000	0.000	0.893	0.871–0.915
WHtR	0.970	0.954–0.986	1.000	1.000	0.000	0.000	0.973	0.961–0.985

BMI, body mass index; CI, confidence interval; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride-glucose; TyG-BMI, TyG index adjusted for body mass index; TyG-WC, TyG index adjusted for waist circumference; TyG-WHtR, TyG index adjusted for waist-to-height ratio.

biochemical markers of IR, such as the quantitative insulin sensitivity check index (QUICKI)[31] and Matsuda index,[32] the TyG index appears to have a slightly weaker predictive power. However, these latter indices require more complex measurements and are less feasible to perform in the pediatric population than the TyG index.

Considering its strong correlation with IR and ease of clinical application, the TyG index is particularly valuable. Several studies have demonstrated the usefulness of the TyG index for MetS screening in pediatric populations.[7,33,34] Our data support a discriminative capacity for detecting MetS (0.801; 95% CI, 0.760 to 0.841) that is similar to that of HOMA-IR (AUC, 0.866; 95% CI, 0.831 to 0.901).

In our population, the overall prevalence of MetS was 15.01%, consistent with other Italian studies,[4] with a higher prevalence in boys compared to girls.[3] Body-fat excess and distribution play critical roles in MetS risk.[35] BMI, WC, and WHtR are the most commonly used indices in clinical practice. Despite their limitations, especially in children, where they might not accurately distinguish between lean and fat mass, these indices are useful markers for metabolic and cardiovascular comorbidities.[4]

Although the relationship between obesity and MetS is not fully understood, as there are patients with obesity without metabolic disorders and patients with normal weight diagnosed with MetS,[36] in our population, MetS occurred exclusively in children with obesity and overweight, and no cases were reported among children with normal weight.

Considering the role of body-fat excess and distribution in the

Table 4. PPV and NPV of metabolic indices stratified by category of BMI z-score

Parameter	Obesity				Overweight				Normal weight				Total population			
	PPV	95% CI	NPV	95% CI	PPV	95% CI	NPV	95% CI	PPV	95% CI	NPV	95% CI	PPV	95% CI	NPV	95% CI
Fasting blood glucose (>100 mg/dL)	0.500	0.454–0.546	0.782	0.744–0.820	0.000	0.000	0.933	0.896–0.970	0.000	0.000	1.000	1.000	0.429	0.393–0.465	0.853	0.828–0.878
HOMA-IR (>2.5 if prepubertal stage and >4 if pubertal stage)	0.627	0.582–0.672	0.909	0.882–0.936	0.471	0.398–0.544	0.975	0.952–0.998	0.000	0.000	1.000	1.000	0.598	0.563–0.633	0.942	0.925–0.959
HDL-C (<50 mg/dL if male and <40 mg/dL if female)	0.421	0.375–0.467	0.958	0.939–0.977	0.204	0.145–0.263	0.992	0.979–1.005	0.000	0.000	1.000	1.000	0.354	0.320–0.388	0.978	0.967–0.989
Triglycerides (≥100 mg/dL if 0≤ age ≤9 and ≥130 mg/dL if 9≤age≤10)	0.712	0.670–0.754	0.852	0.819–0.885	0.500	0.427–0.573	0.953	0.922–0.984	0.000	0.000	1.000	1.000	0.687	0.654–0.720	0.903	0.882–0.924
Systolic blood pressure (≥130 mmHg)	0.813	0.777–0.849	0.823	0.788–0.858	0.375	0.304–0.446	0.947	0.914–0.980	0.000	0.000	1.000	1.000	0.659	0.625–0.693	0.882	0.859–0.905
Diastolic blood pressure (≥85 mmHg)	0.778	0.740–0.816	0.790	0.752–0.828	0.000	0.000	0.933	0.896–0.970	0.000	0.000	1.000	1.000	0.700	0.667–0.733	0.857	0.832–0.882
TyG (>Q3=8.013)	0.527	0.481–0.573	0.879	0.849–0.909	0.156	0.103–0.209	0.963	0.935–0.991	0.000	0.000	1.000	1.000	0.387	0.352–0.422	0.929	0.911–0.947
TyG-BMI (>Q3=192.487)	0.509	0.463–0.555	0.873	0.842–0.904	0.156	0.103–0.209	0.963	0.935–0.991	0.000	0.000	1.000	1.000	0.409	0.374–0.444	0.936	0.918–0.954
TyG-WC (>Q3=644.449)	0.482	0.436–0.528	0.864	0.832–0.896	0.200	0.141–0.259	0.978	0.957–0.999	0.000	0.000	1.000	1.000	0.425	0.390–0.460	0.941	0.924–0.958
TyG-WHtR (>Q3=4.217)	0.420	0.374–0.466	0.844	0.811–0.877	0.222	0.161–0.283	0.854	0.802–0.906	0.000	0.000	0.844	0.791–0.897	0.366	0.331–0.401	0.921	0.902–0.940
BMI Z-score (>Q3=2)	0.222	0.184–0.260	0.000	0.000	0.000	0.000	0.933	0.896–0.970	0.000	0.000	1.000	1.000	0.222	0.192–0.252	0.959	0.945–0.973
WHtR (≥0.5)	0.224	0.186–0.262	0.833	0.799–0.867	0.126	0.077–0.175	0.000	0.000	0.000	0.000	1.000	1.000	0.205	0.176–0.234	0.986	0.978–0.994

To calculate predictive value, the sample was divided into three groups (obesity, overweight, and normal weight) based on BMI z-score. Each group was evaluated separately and then analyzed collectively. For each group, contingency tables were created to analyze glycemia, HOMA, triglycerides, and others. In these tables, the test outcome (positive or negative based on the chosen cut-off) and the presence or absence of metabolic syndrome were treated as dichotomous variables. Using these tables, sensitivity, specificity, NPV, PPV, and their corresponding 95% CIs were calculated.

PPV, positive predictive value; NPV, negative predictive value; BMI, body mass index; CI, confidence interval; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride-glucose; TyG-BMI, TyG index adjusted for body mass index; TyG-WC, TyG index adjusted for waist circumference; TyG-WHtR, TyG index adjusted for waist-to-height ratio.

Table 5. Discriminative capacity expressed in terms of AUC and Youden index

	AUC	Youden index	Best cut-off	Chosen cut-off
Fasting blood glucose (mg/dL)	0.659	0.284	79	100
HOMA-IR	0.866	0.621	3	2.5/4
Triglycerides (mg/dL)	0.799	0.462	105	100/130
Systolic blood pressure (mmHg)	0.700	0.327	112	130
Diastolic blood pressure (mmHg)	0.628	0.228	64	85
TyG	0.801	0.468	8.2	8.2
TyG-BMI	0.840	0.523	213	231
TyG-WC	0.832	0.561	686	734
TyG-WHtR	0.777	0.505	4.2	4.73
WHtR	0.729	0.340	0.52	0.5
HDL-C females (mg/dL)	0.842	0.645	40	40
HDL-C males (mg/dL)	0.801	0.513	50	50

AUC, area under the receiver operating characteristic curve; HOMA-IR, homeostasis model assessment of insulin resistance; TyG, triglyceride-glucose; TyG-BMI, TyG index adjusted for body mass index; TyG-WC, TyG index adjusted for waist circumference; TyG-WHtR, TyG index adjusted for waist-to-height ratio; HDL-C, high-density lipoprotein cholesterol.

development of IR and MetS, it was hypothesized that incorporating BMI, WC, and WHtR into the TyG index would enhance its predictive ability. In adults, TyG-related parameters improved MetS identification and prediction.[16] Recently, Wei et al.[37] reported the clinical value of TyG-related indices in predicting the survival of patients with MetS.

Consistent with findings in adults, our data support the applicability of this hypothesis in the pediatric population, showing that combined TyG-related parameters, such as TyG-BMI (AUC, 0.840; 95% CI, 0.803 to 0.877) and TyG-WC (AUC, 0.832; 95% CI, 0.793 to 0.870), outperform the standalone TyG index in IR evaluation.

These indices showed diagnostic accuracy similar to that of HOMA-IR and were not inferior to other parameters typically included in MetS criteria. Their performance was further documented using the Youden test, which not only summarizes the discriminatory accuracy of a diagnostic test, but also offers a practical and optimal threshold for future diagnostic purposes. PPV and NPV are influenced by the prevalence of disease in the population being tested. In our model, a high NPV suggests that these indices are also reliable in ensuring that predicted negatives are correct, which is useful for determining the need for further diagnostic tests.

The TyG index is easy to calculate based on routine laboratory measurements and is a highly cost-effective test available in all clinical settings.[12] Integrating auxological parameters into this index enhances its diagnostic performance for IR screening without additional costs, producing a valuable tool for health policy makers.

Although several optimal cut-off values for the TyG index have been proposed,[38] we focused on the Q3 values (TyG, 8.1; TyG-BMI, 231.54; TyG-WC, 734.03; and TyG-WHtR, 4.73), as they provide a more robust and representative measure than the mean or median and are less sensitive to outliers and better suited to asymmetrical distributions. Such use also facilitates the identification of high-risk subgroups, especially in the absence of a standardized TyG cut-off for pediatric populations.

This study has several limitations. First, its retrospective design limits control over exposure factors, covariates, and potential confounders. Second, we did not account for dietary and exercise habits, family history, economic status, and life stress, which could introduce confounding biases and should be considered in future studies. Last, several studies have highlighted that TyG index cut-offs might vary across ethnic groups, reflecting genetic, dietary, and lifestyle factors that influence insulin sensitivity across populations. For instance, in Brazilian children aged 4 to 9 years, TyG cut-offs of 7.9 for boys and 8.1 for girls were associated with increased cardiometabolic risk, including overweight, central adiposity, and dyslipidemia.[39] Similarly, in Turkish children aged 9 to 10 years, the TyG index, with a cut-off of 8.3 in boys and 8.5 in girls, showed greater predictive accuracy for MetS than did HOMA-IR, with sex-specific thresholds and high AUC values.[40] Therefore, including multi-ethnic cohorts in future studies would be essential to establish more gen-

eralizable reference values and improve the applicability of TyG-related parameters in pediatric populations worldwide.

In conclusion, the TyG, TyG-BMI, TyG-WC, and TyG-WHtR indices demonstrate promising predictive values for identifying MetS in pediatric populations with overweight and obesity. Considering the high cost of insulin sensitivity testing, utilizing these TyG-derived indices as surrogate markers for assessing IR could potentially reduce expenses in clinical practice. These indices enable the identification of high-risk groups, facilitating early intervention to prevent T2DM, improve prognosis, and lower management costs.

SUPPLEMENTARY MATERIALS

Supplementary materials can be found online at https://doi.org/10.7570/jomes25002.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Study concept and design: VC and GZ; acquisition of data: LL, EC, VR, and VF; analysis and interpretation of data: LL; drafting of the manuscript: VC, LL, EC, VR, VF, and GZ; critical revision of the manuscript: VC, LL, EC, LS, VR, AC, MT, VF, and GZ; statistical analysis: LL; obtained funding: VC and GZ; administrative, technical, or material support: LL, EC, VR, and VF; and study supervision: VC and GZ.

REFERENCES

1. Noubiap JJ, Nansseu JR, Lontchi-Yimagou E, Nkeck JR, Nyaga UF, Ngouo AT, et al. Global, regional, and country estimates of metabolic syndrome burden in children and adolescents in 2020: a systematic review and modelling analysis. Lancet Child Adolesc Health 2022;6:158-70.
2. Codazzi V, Frontino G, Galimberti L, Giustina A, Petrelli A. Mechanisms and risk factors of metabolic syndrome in children and adolescents. Endocrine 2024;84:16-28.
3. Reisinger C, Nkeh-Chungag BN, Fredriksen PM, Goswami N. The prevalence of pediatric metabolic syndrome-a critical look on the discrepancies between definitions and its clinical importance. Int J Obes (Lond) 2021;45:12-24.
4. Brufani C, Fintini D, Giordano U, Tozzi AE, Barbetti F, Cappa M. Metabolic syndrome in Italian obese children and adolescents: stronger association with central fat depot than with insulin sensitivity and birth weight. Int J Hypertens 2011;2011:257168.
5. Friend A, Craig L, Turner S. The prevalence of metabolic syndrome in children: a systematic review of the literature. Metab Syndr Relat Disord 2013;11:71-80.
6. White MF. Insulin signaling in health and disease. Science 2003;302:1710-1.
7. Cruz ML, Weigensberg MJ, Huang TT, Ball G, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. J Clin Endocrinol Metab 2004;89:108-13.
8. Raitakari OT, Porkka KV, Rönnemaa T, Knip M, Uhari M, Akerblom HK, et al. The role of insulin in clustering of serum lipids and blood pressure in children and adolescents: the cardiovascular risk in young Finns study. Diabetologia 1995;38:1042-50.
9. Gastaldelli A. Measuring and estimating insulin resistance in clinical and research settings. Obesity (Silver Spring) 2022;30:1549-63.
10. Esteghamati A, Ashraf H, Khalilzadeh O, Zandieh A, Nakhjavani M, Rashidi A, et al. Optimal cut-off of homeostasis model assessment of insulin resistance (HOMA-IR) for the diagnosis of metabolic syndrome: third national surveillance of risk

factors of non-communicable diseases in Iran (SuRFNCD-2007). Nutr Metab (Lond) 2010;7:26.
11. Tagi VM, Samvelyan S, Chiarelli F. An update of the consensus statement on insulin resistance in children 2010. Front Endocrinol (Lausanne) 2022;13:1061524.
12. Di Bonito P, Di Sessa A, Licenziati MR, Corica D, Wasniewska MG, Morandi A, et al. Ability of triglyceride-glucose indices to predict metabolic dysfunction associated with steatotic liver disease in pediatric obesity. J Pediatr Gastroenterol Nutr 2025;81:1287-95.
13. Song S, Son DH, Baik SJ, Cho WJ, Lee YJ. Triglyceride glucose-waist circumference (TyG-WC) is a reliable marker to predict non-alcoholic fatty liver disease. Biomedicines 2022;10:2251.
14. Malek M, Khamseh ME, Chehrehgosha H, Nobarani S, Alaei-Shahmiri F. Triglyceride glucose-waist to height ratio: a novel and effective marker for identifying hepatic steatosis in individuals with type 2 diabetes mellitus. Endocrine 2021;74:538-45.
15. Yang C, Song Y, Wang X, Yang Y, Zhou Y, Wang D, et al. Association of hypertension with the triglyceride-glucose index and its associated indices in the Chinese population: a 6-year prospective cohort study. J Clin Hypertens (Greenwich) 2024;26:53-62.
16. Raimi TH, Dele-Ojo BF, Dada SA, Fadare JO, Ajayi DD, Ajayi EA, et al. Triglyceride-glucose index and related parameters predicted metabolic syndrome in Nigerians. Metab Syndr Relat Disord 2021;19:76-82.
17. Aslan Çin NN, Yardımcı H, Koç N, Uçaktürk SA, Akçil Ok M. Triglycerides/high-density lipoprotein cholesterol is a predictor similar to the triglyceride-glucose index for the diagnosis of metabolic syndrome using International Diabetes Federation criteria of insulin resistance in obese adolescents: a cross-sectional study. J Pediatr Endocrinol Metab 2020;33:777-84.
18. Bazyar H, Zare Javid A, Masoudi MR, Haidari F, Heidari Z, Hajializadeh S, et al. Assessing the predictive value of insulin resistance indices for metabolic syndrome risk in type 2 diabetes mellitus patients. Sci Rep 2024;14:8917.
19. Tang X, Zhang K, He R. Correction: the association of triglyceride-glucose and triglyceride-glucose related indices with the risk of heart disease in a national cohort study. Cardiovasc Diabetol 2025;24:174.
20. Mijangos-Trejo A, Gómez-Mendoza R, Ramos-Ostos MH, Castro-Narro G, Uribe M, Juárez-Hernández E, et al. Diagnostic accuracy of the triglyceride-glucose index (TyG), TyG body mass index, and TyG waist circumference index for liver steatosis detection. Diagnostics (Basel) 2024;14:762.
21. World Health Organization. Child growth standards [Internet]. WHO; 2025 [cited 2025 Dec 26]. Available from: https://www.who.int/tools/child-growth-standards
22. Leone A, Vizzuso S, Brambilla P, Mameli C, Ravella S, De Amicis R, et al. Evaluation of different adiposity indices and association with metabolic syndrome risk in obese children: is there a winner? Int J Mol Sci 2020;21:4083.
23. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970;45:13-23.
24. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969;44:291-303.
25. Muntner P, Shimbo D, Carey RM, Charleston JB, Gaillard T, Misra S, et al. Measurement of blood pressure in humans: a scientific statement from the American Heart Association. Hypertension 2019;73:e35-66.
26. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 2001;285:2486-97.
27. Calcaterra V, Biganzoli G, Ferraro S, Verduci E, Rossi V, Vizzuso S, et al. A multivariate analysis of "metabolic phenotype" patterns in children and adolescents with obesity for the early stratification of patients at risk of metabolic syndrome. J Clin Med 2022;11:1856.
28. Calcaterra V, Fabiano V, De Silvestri A, Colombo C, Tranfaglia V, Loiodice M, et al. The impact of vitamin D status on lipid profiles and atherogenic dyslipidemia markers in children and adolescents with obesity. Nutr Metab Cardiovasc Dis 2024;34:2596-605.
29. d'Annunzio G, Vanelli M, Pistorio A, Minuto N, Bergamino L, Iafusco D, et al. Insulin resistance and secretion indexes in healthy Italian children and adolescents: a multicentre study. Acta Biomed 2009;80:21-8.

30. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
31. Kalyoncu D, Kavrak Kursun M. Comparison of triglyceride glucose index and other insulin resistance indexes in children with overweight and obesity. BMC Endocr Disord 2025;25:96.
32. Malagón-Soriano VA, Ledezma-Forero AJ, Espinel-Pachon CF, Burgos-Cárdenas ÁJ, Garces MF, Ortega-Ramírez GE, et al. Surrogate indices of insulin resistance using the Matsuda index as reference in adult men-a computational approach. Front Endocrinol (Lausanne) 2024;15:1343641.
33. Wan Mahmud Sabri WM, Mohamed RZ, Yaacob NM, Hussain S. Prevalence of metabolic syndrome and its associated risk factors in pediatric obesity. J ASEAN Fed Endocr Soc 2022;37:24-30.
34. Sinaiko AR, Jacobs DR, Steinberger J, Moran A, Luepker R, Rocchini AP, et al. Insulin resistance syndrome in childhood: associations of the euglycemic insulin clamp and fasting insulin with fatness and other risk factors. J Pediatr 2001;139:700-7.
35. Guseman EH, Eisenmann JC, Laurson KR, Cook SR, Stratbucker W. Calculating a continuous metabolic syndrome score using nationally representative reference values. Acad Pediatr 2018;18:589-92.
36. Jakubiak GK, Osadnik K, Lejawa M, Osadnik T, Goławski M, Lewandowski P, et al. "Obesity and insulin resistance" is the component of the metabolic syndrome most strongly associated with oxidative stress. Antioxidants (Basel) 2021;11:79.
37. Wei X, Min Y, Song G, Ye X, Liu L. Association between triglyceride-glucose related indices with the all-cause and cause-specific mortality among the population with metabolic syndrome. Cardiovasc Diabetol 2024;23:134.
38. Brito ADM, Hermsdorff HH, Filgueiras MS, Suhett LG, Vieira-Ribeiro SA, Franceschini SDCC, et al. Predictive capacity of triglyceride-glucose (TyG) index for insulin resistance and cardiometabolic risk in children and adolescents: a systematic review. Crit Rev Food Sci Nutr 2021;61:2783-92.
39. Bessho S. Elections and COVID-19 benefit payments. J Asian Econ 2023;87:101630.
40. Košnář Z, Mercl F, Pierdonà L, Chane AD, Míchal P, Tlustoš P. Concentration of the main persistent organic pollutants in sewage sludge in relation to wastewater treatment plant parameters and sludge stabilisation. Environ Pollut 2023;333:122060.