Diabetes, Metabolic Syndrome and Obesity

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Predictive Value of the second-Trimester Triglyceride-Glucose Index and Its Derived Indices for Macrosomia in Gestational Diabetes Mellitus

Yiting Chen*, Shuyu Li*, Lanlan Xiang, Yitian Zhu, Yu Zeng, Yajun Chen, Tianying Zhong

Department of Clinical Laboratory, Women's Hospital of Nanjing Medical University, Nanjing Women and Children's Healthcare Hospital, Nanjing, Jiangsu Province, 210003, People's Republic of China

*These authors contributed equally to this work

Correspondence: Yajun Chen; Tianying Zhong, Department of Clinical Laboratory, Women's Hospital of Nanjing Medical University, Nanjing Women and Children's Healthcare Hospital, Nanjing, Jiangsu Province, 210003, People's Republic of China, Email yajunchen@njmu.edu.cn; zhongtianying@njmu.edu.cn

Video abstract

Point your SmartPhone at the code above. If you have a QR code reader the video abstract will appear. Or use: https://youtu.be/rqVLzW2OI0w

Background: Macrosomia is a major adverse outcome associated with gestational diabetes mellitus (GDM), primarily driven by insulin resistance (IR). The triglyceride-glucose (TyG) index is a well-established surrogate marker for insulin resistance; however, its predictive value when combined with obesity indicators remains incompletely defined. This study aimed to compare the predictive performance of the TyG index and its derived indices during the second trimester for the risk of macrosomia in GDM.

Methods: The TyG index, TyG body mass index (TyG-BMI), TyG waist circumference (TyG-WC), and TyG waist-to-height ratio (TyG-WHtR) were assessed at 24–28 weeks of gestation. Their associations with macrosomia were analyzed using binary logistic regression and restricted cubic spline (RCS) analysis. The predictive performance of these indices was compared using receiver operating characteristic (ROC) curves, integrated discrimination improvement (IDI), and net reclassification improvement (NRI). The associations between TyG and its derived indices and the risk of delivering a macrosomic infant were further explored across different subgroups.

Results: Logistic regression revealed that GDM women in the highest tertile had a 3.458- and 3.718-fold higher risks of delivering macrosomia compared to the lowest tertile for TyG-BMI and TyG-WC, respectively. RCS analysis showed a dose-response relationship (all P for overall < 0.001). Compared with the conventional TyG index, which had an area under the curve (AUC) of 0.596, both TyG-BMI (AUC = 0.682) and TyG-WC (AUC = 0.681) demonstrated significantly better performance ($P < 0.01$); furthermore, their superiority was confirmed by significant IDI and NRI values ($P < 0.01$). The association was more pronounced in primiparous women aged < 35 years ($P < 0.05$).

Conclusion: Elevated second-trimester TyG index and its derived indices are associated with an increased risk of macrosomia in GDM women. TyG-BMI and TyG-WC outperformed the conventional TyG index and may serve as valuable supplementary markers for early risk stratification in GDM.

Keywords: gestational diabetes mellitus, insulin resistance, macrosomia, predictive model, triglyceride-glucose index

Introduction

Gestational diabetes mellitus (GDM) is characterized by glucose intolerance initially identified during pregnancy,[1] affecting 9.3% to 25.5% of pregnancies worldwide.[2] The prevalence in China reaches approximately 14.8% and continues to rise each year. Macrosomia, defined as birth weight ≥4000 g.[3] represents a frequent complication of GDM and correlates strongly with various adverse maternal and neonatal outcomes. Maternal complications often include obstetric trauma and postpartum hemorrhage, while neonates are predisposed to neonatal hypoglycemia and an elevated risk of developing metabolic disorders in adulthood.[4,5] Therefore, identifying effective predictive markers for early detection of macrosomia holds profound clinical significance in improving both short and long term adverse outcomes for mothers and infants. Although routine ultrasound

https://doi.org/10.2147/DMSO.S577687
Received: 2 November 2025
Accepted: 10 April 2026
Published: 23 April 2026

examination at 24–28 weeks of gestation is essential for structural screening, its sensitivity for predicting macrosomia is limited. This limitation arises because macrosomia typically manifests as accelerated growth in the late third trimester, fetal biometric measurements often remain within the normal range at this earlier stage, and operator-dependent weight estimation formulas are inherently inaccurate. Consequently, the predictive value of a single second-trimester ultrasound is limited, highlighting the need for supplementary biochemical markers to improve early risk stratification.[6]

Insulin resistance (IR), a central pathological mechanism in glucose and lipid dysregulation, significantly contributes to the development of GDM.[7–9] Recent studies confirm that the triglycerides-glucose (TyG) index correlates strongly with both the hyperinsulinemic-euglycemic clamp (the gold-standard measure of insulin resistance) and the homeostasis model assessment of insulin resistance (HOMA-IR).[10,11] It is also closely related to diabetes,[12] cardiovascular disease,[13,14] hypertension and other diseases. These findings establish the TyG index as a practical and economical biomarker for assessing insulin resistance.[15–17] Although the TyG index is currently being used more and more in pregnant populations,[18–21] more research is needed to determine its specificity and sensitivity in GDM patients, who have noticeable IR characteristics.[22] Body mass index (BMI) and gestational weight gain (GWG) are established independent risk factors for macrosomia. Anthropometric measures alone, however, may inadequately reflect underlying metabolic dysregulation, including insulin resistance. This limitation underscores the need for composite indices. Recent studies suggest that derivative indices like TyG-body mass index (TyG-BMI) and TyG-waist circumference (TyG-WC) could improve the predictive accuracy of the TyG index.[23–27] However, their ability to predict macrosomia in GDM pregnancies remains uncertain. This retrospective study aims to evaluate the predictive value of the TyG index and its derivatives for macrosomia in GDM pregnancies. The findings may provide a clinical basis for early identification of high-risk GDM individuals and mitigation of macrosomia risk.

Materials and Methods

Study Participants

Pregnant women with GDM who underwent regular prenatal examinations and delivered at Nanjing Women and Children's Healthcare Hospital between January 2017 and June 2022 were enrolled. The inclusion criteria were as follows: (1) Singleton pregnancy with delivery between 37 and 42 weeks of gestation; (2) Age 18–45 years; (3) Diagnosis of GDM confirmed by a 75g oral glucose tolerance test (OGTT) at 24–28 weeks of gestation, in accordance with the diagnostic criteria;[28] a diagnosis of GDM was confirmed if one or more of the following plasma glucose thresholds were met or exceeded: fasting plasma glucose (FPG) ≥ 5.1 mmol/L, 1-hour plasma glucose ≥ 10.0 mmol/L, or 2-hour plasma glucose≥ 8.5 mmol/L.(4) Complete clinical data available. The exclusion criteria included: (1) Pre-existing type 1 or type 2 diabetes mellitus, diagnosis of diabetes mellitus before pregnancy, or lipid-lowering medication use prior to pregnancy; (2) Coexisting pregnancy complications such as gestational hypertension; (3) Presence of autoimmune diseases, cardiovascular diseases, severe hepatic or renal dysfunction or malignancy; (4) Requirement of pharmacotherapy for other chronic conditions; and (5) Fetal congenital anomalies, chromosomal abnormalities, or inherited metabolic disorders. A total of 5,502 GDM patients aged 18–45 years (mean ± SD: 29.93 ± 3.64) were included. This study was approved by the Ethics Committee of Nanjing Maternity and Child Health Hospital (Approval No.: 2022KY-068). (Figure 1) details the flow chart for the selection of participants.

Research Method

Data Collection and Measurements

The relevant clinical data of the study subjects were collected through the electronic medical record information system, including age, height, pre-pregnancy body weight, gravidity, parity, family history of diabetes, present and past medical history, gestational weeks at delivery, pregnancy outcomes, and neonatal birth weight. Neonates with a birth weight ≥4000 g were defined as macrosomia.[3] During the second-trimester (24–28 weeks of gestation), obstetric examinations were performed on the pregnant women. Standardized measurements were obtained for height, body weight, waist circumference (WC), diastolic blood pressure, and systolic blood pressure. Body mass index (BMI) and waist-to-height ratio (WHtR) were subsequently calculated.

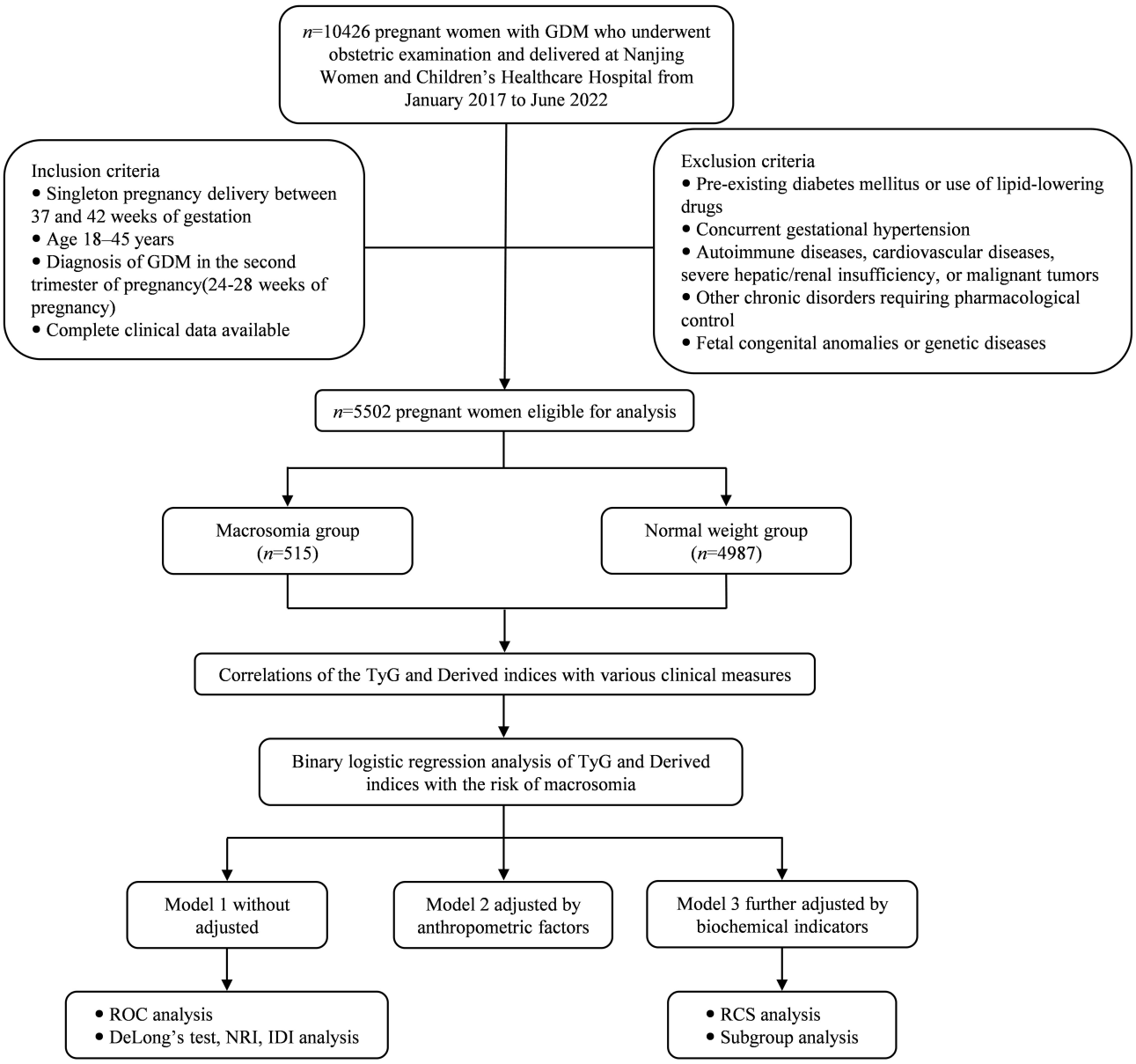

Figure 1 Study population selection and data analysis flowchart.
Abbreviations: GDM, gestational diabetes mellitus; TyG, Triglycerides-glucose index; ROC, receiver operating characteristic; RCS, restricted cubic spline; NRI, net reclassification improvement; IDI, integrated discrimination improvement.

Biochemical Index Measurement

Fasted for over 10 hours during the second-trimester (24–28 weeks of gestation), and 5 mL of venous blood was collected from the antecubital vein the following morning under fasting conditions. Fasting plasma glucose (FPG), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and other biochemical parameters were measured using a fully automated biochemical analyzer (AU5800, Beckman Coulter). Glycated hemoglobin (HbA1c) was determined using a fully automated HbA1c analyzer (HA-8180, ARKRAY Inc). The TyG index and its derived indices were subsequently calculated.

Specific calculation formulas:

$$\mathrm{BMI} = \text{body weight}(\mathrm{kg})/\text{height}^2\left(\mathrm{m}^2\right)$$

https://doi.org/10.2147/DMSO.S577687

$$\mathrm{WHtR} = \mathrm{WC(cm)}/\mathrm{height(cm)}$$

$$\mathrm{TyG} = \mathrm{Ln}[\mathrm{TG(mg/dL)} \times \text{fasting glucose(mg/dL)}/2]$$

$$\mathrm{TyG-BMI} = \mathrm{TyG} \times \mathrm{BMI}$$

$$\mathrm{TyG-WC} = \mathrm{TyG} \times \mathrm{WC}$$

$$\mathrm{TyG-WHtR} = \mathrm{TyG} \times \mathrm{WHtR}$$

Grouping

Based on the tertile cutoff values of TyG, TyG-BMI, TyG-WC, and TyG-WHtR, participants were categorized into tertile 1 (T1), tertile 2 (T2), and tertile 3 (T3) groups, respectively. For trend analysis the first tertile served as the reference: TyG index (tertile 1, ≤ 8.849, tertile 2, 8.805–9.181; tertile 3, ≥ 9.181) TyG-BMI (tertile 1, ≤ 206.942, tertile 2, 206.942–236.037; tertile 3, ≥ 236.037) TyG-WC (tertile 1, ≤ 777.697, tertile 2, 777.697–845.598, tertile 3, ≥ 845.598) TyG-WHtR (tertile 1, ≤ 4.791, tertile 2, 4.791–5.221, tertile 3, ≥ 5.221).

Covariates

In the multivariate adjusted analysis, several demographic factors known to potentially influence the association between the TyG index and its derivatives and macrosomia were included as covariates, namely maternal age, gravidity (1, 2, or ≥ 3 times), parity (1 or ≥ 2 times), systolic blood pressure (SBP), and diastolic blood pressure (DBP). Additionally, adjustments were made for the following biochemical parameters as potential confounders: HbA1c, HDL-C, and LDL-C.

Statistical Analyses

Statistical analysis was performed using IBM SPSS 27.0. The normality of continuous variables was assessed by the Kolmogorov–Smirnov test. Normally distributed measurement data were expressed as mean ± standard deviation ($\bar{x} \pm s$) and normally distributed continuous variables were analyzed using one-way ANOVA for intergroup comparisons, while categorical data were presented as n (%) and analyzed by χ^2 test. Patients were stratified into tertiles (T1-T3) based on TyG index and its derived indices. Continuous variables were compared using ANOVA or Kruskal–Wallis *H*-test, whereas categorical variables were analyzed by chi-square test. Spearman correlation analysis was conducted using the Correlation Plot module in Origin 2025 to examine the relationships between TyG and its derived indices with other parameters. Three multivariate-adjusted logistic regression models were developed to identify factors influencing macrosomia delivery in GDM patients. To further assess potential nonlinear associations between the TyG index and its derived indices with macrosomia, we performed restricted cubic spline (RCS) analysis, adjusting for all covariates included in Model 3. Receiver operating characteristic (ROC) curves were plotted, and the area under the curve (AUC) was calculated to evaluate the diagnostic performance of TyG and its derived indices for predicting macrosomia in GDM pregnancies. DeLong's test along with net reclassification improvement (NRI) and integrated discrimination improvement (IDI), was employed to compare the diagnostic efficacy among TyG, TyG-BMI, TyG-WC, and TyG-WHtR. Subgroup analyses were performed by stratifying participants based on age (<35 or ≥35 years), parity (1 or ≥2), to assess the influence of these variables. Interaction effects across subgroups were examined using likelihood ratio tests. Statistical analyses were conducted using R software (version 4.2.0). All tests were two-sided, with $P < 0.05$ considered statistically significant.

Results

Baseline Characteristics

All pregnant women with GDM were stratified into tertiles based on their TyG index values (Table 1), T1 group (TyG ≤ 8.848, n=1838), T2 group (TyG 8.849–9.180, n=1830), and T3 group (TyG ≥ 9.180, n=1834).A progressive increase was observed across T1, T2, and T3 groups in maternal age, waist circumference (WC), body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), FPG, HbA1c, TG, infant weight and the proportion of macrosomia ($P < 0.001$). Conversely,

Table 1 Baseline Characteristics of Participants According to Tertiles of the TyG Index [$\bar{x} \pm s$, n(%)]

Characteristic	Overall (n=5502)	TyG Index Tertiles			*P* value
		T1 (n=1838)	T2 (n=1830)	T3 (n=1834)	
Maternal characteristics					
Age (years)	29.93±3.64	29.48±3.56	29.88±3.60	30.44±3.71	<0.001
GWG (kg)	6.22±3.37	6.19±3.18	6.25±3.41	6.21±3.52	0.848
Gravidity					<0.001
1	2657(48.30)	1010(54.95)	880(48.09)	767(41.82)	
2	1668(30.32)	511(27.80)	557(30.44)	600(32.72)	
≥3	1177(21.39)	317(17.25)	393(21.48)	467(25.46)	
Parity					<0.001
0	3842(69.80)	1385(75.35)	1278(69.84)	1179(64.29)	
≥1	1660(30.20)	453(24.65)	552(30.16)	655(35.71)	
Clinical data at Second-trimester					
WC (cm)	90.48±7.14	88.13±6.44	90.45±6.91	92.88±7.25	<0.001
BMI (kg/m^2)	24.78±3.22	23.63±2.90	24.76±3.14	25.96±3.17	<0.001
SBP (mmHg)	112.46±11.26	110.40±10.97	112.28±11.11	114.69±11.28	<0.001
DBP (mmHg)	69.87±8.34	68.40±8.12	69.76±8.24	71.45±8.37	<0.001
FPG (mmol/L)	4.84±0.52	4.65±0.42	4.82±0.42	5.06±0.60	<0.001
HbA1c (%)	5.11±0.30	5.03±0.25	5.11±0.28	5.20±0.35	<0.001
LDL-C (mmol/L)	3.01±0.73	2.93±0.66	3.10±0.72	3.01±0.78	<0.001
HDL-C (mmol/L)	2.32±0.44	2.49±0.42	2.34±0.42	2.12±0.40	<0.001
TG (mmol/L)	2.34±0.95	1.53±0.27	2.17±0.27	3.31±0.98	<0.001
TC (mmol/L)	5.92±0.99	5.82±0.88	6.01±0.99	5.93±1.07	<0.001
ALT (U/L)	19.63±15.93	22.10±16.52	19.69±17.86	17.10±12.54	<0.001
AST (U/L)	18.79±8.37	20.12±7.94	18.55±8.79	17.69±8.18	<0.001
Neonatal characteristics					
Infant weight (kg)	3.46±0.39	3.41±0.36	3.46±0.39	3.52±0.41	<0.001
Gestational age (weeks)	39.50±0.96	39.57±0.94	39.53±0.95	39.39±0.97	<0.001
Macrosomia	515(9.40)	110(6.00)	180(9.83)	225(12.27)	<0.001
Infant sex					0.278
Boy	2860(52.00)	932(50.71)	970(53.01)	958(52.24)	
Girl	2642(48.00)	906(49.29)	860(46.99)	876(47.76)	

Abbreviations: T, tertiles; TyG, Triglycerides-glucose index GWG, Gestational weight gain; WC, waist circumference; BMI, body mass index; SBP, systolic blood pressure; DBP, Diastolic blood pressure; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; LDL-C, low-density lipoprotein; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; TC, total cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GDM, gestational diabetes mellitus.

gestational age at delivery, HDL-C, ALT, and AST levels demonstrated a sequential decline ($P < 0.001$). No significant differences were detected among TyG index tertiles for gestational weight gain (GWG) or infant sex. Similar trends were stratified by TyG-BMI,TyG-WC, and TyG-WHtR tertiles.

Spearman Correlation Analysis of the TyG Index and Related Parameters with Other Variables

As shown in (Figure 2), Spearman correlation analysis revealed that the TyG index was positively correlated with LDL-C, FPG, HbA1c, TG, TC, and infant weight ($P < 0.05$), while exhibiting a negative correlation with HDL-C ($P < 0.001$). The TyG-derived indices (TyG-BMI, TyG-WC, TyG-WHtR) demonstrated significant positive correlations with GWG,

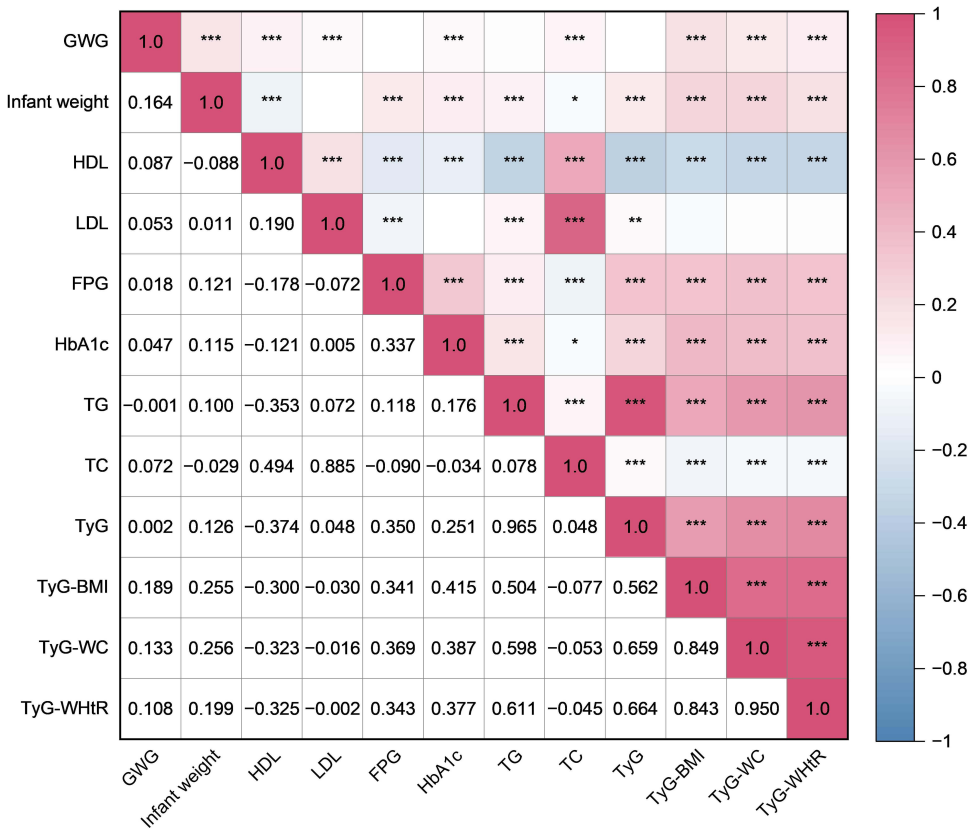

Figure 2 Spearman correlation analysis of the correlation between TyG index related indices and other indicators.
Notes: *$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$.
Abbreviations: GWG, Gestational weight gain; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin; TG, triglycerides; TC, total cholesterol; BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; TyG, Triglycerides-glucose index.

FPG, HbA1c, TG, and infant weight ($P < 0.001$), whereas they were negatively correlated with HDL-C and TC ($P < 0.05$).

Comparative Predictive Value of the TyG Index and Its Derived Indices for Macrosomia in Pregnant Women with GDM

Using macrosomia delivery by GDM mothers as the dependent variable, the TyG index and its derived indices were incorporated into the model according to their respective tertiles (T1, T2, T3), with T1 as the reference group (Figure 3). Logistic regression analysis was performed, and the results demonstrated that after adjusting for relevant confounding factors (age, gravidity, parity, SBP, DBP, HbA1c, HDL-C, LDL-C), the OR (95% CI) for macrosomia delivery in the T3 groups of TyG index, TyG-BMI, TyG-WC, and TyG-WHtR were 1.769 (1.278–2.450), 3.458 (2.427–4.926), 3.718 (2.603–5.312), and 2.279 (1.641–3.164) times higher (all $P < 0.01$), respectively, compared to the T1 group.

Restricted cubic spline analysis showed a dose–response relationship between the TyG index and its derived indices and macrosomia after adjusting for multiple covariates (all P for overall < 0.001; Figure 4). The incidence of macrosomia increased linearly with rising TyG-BMI and TyG-WHtR (all P for Nonlinear > 0.05). The association between TyG and macrosomia risk followed an approximate inverted "U"-shaped curve (P for Nonlinear < 0.05). TyG < 9.0, the risk of macrosomia rose with increasing TyG levels; above this threshold, the risk plateaued and declined slightly. In contrast, TyG-WC exhibited a "J"-shaped relationship with macrosomia risk (P for Nonlinear < 0.05). Once TyG-WC exceeded 900, further increases were associated with a sharp rise in macrosomia incidence.

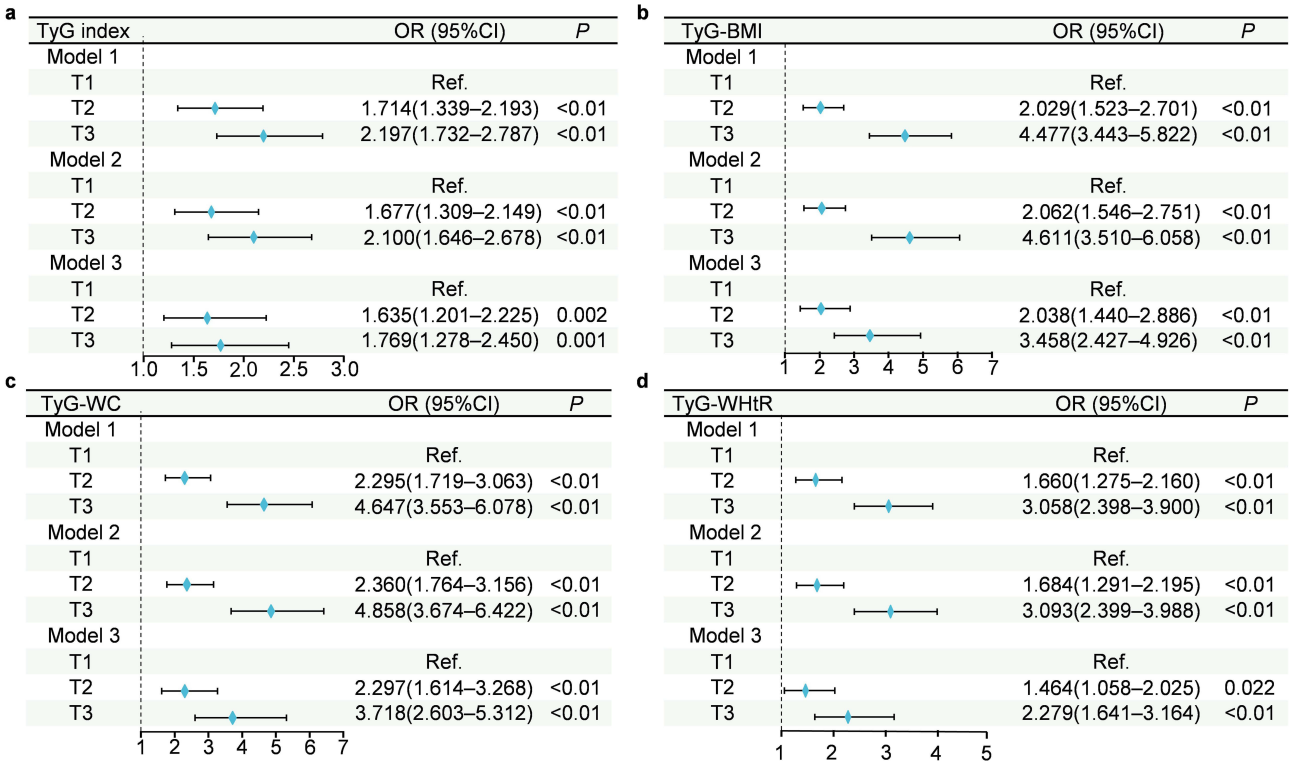

Figure 3 Forest plots show the Logistic regression analysis of factors influencing macrosomia in GDM pregnancies. (**a**) TyG index; (**b**) TyG-BMI; (**c**) TyG-WC; (**d**) TyG-WHtR. Model 1: Unadjusted; Model 2: Adjusted age, gravidity, parity, SBP, DBP; Model 3: Adjusted age, gravidity, parity, SBP, DBP, HbA1C, HDL-C, LDL-C.
Abbreviations: T, tertiles; Ref, Reference group; SBP, systolic blood pressure; DBP, diastolic blood pressure, HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein; HbA1c, glycated hemoglobin. BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; TyG, Triglycerides-glucose index; GDM, gestational diabetes mellitus.

Model Prediction Performance

Compare the discriminative ability of the TyG index and its derived indices for predicting macrosomia in pregnant women with GDM (Table 2 and Figure 5). The AUC (95% CI) values for TyG, TyG-BMI, TyG-WC, and TyG-WHtR were 0.596 (0.572–0.621), 0.682 (0.658–0.706), 0.681 (0.658–0.705), and 0.651 (0.626–0.675), respectively. The corresponding sensitivities were 86.2%, 61.0%, 63.3%, and 61.2%, while the specificities were 28.2%, 66.3%, 63.0%, and 61.0%. The optimal cutoff values were 8.79, 233.05, 832.03, and 5.11, respectively. DeLong's test revealed that the AUC of TyG-BMI and TyG-WC were significantly higher than that of TyG ($P < 0.05$), whereas no statistically significant difference was observed between TyG-BMI and TyG-WC ($P = 0.881$).

Regarding the NRI and IDI, TyG-BMI and TyG-WC exhibited slightly superior performance compared to the other indices. The IDI values were 0.030 and 0.029, while the NRI values were 0.443 and 0.450, ($P < 0.01$). These findings indicate that TyG-BMI and TyG-WC serve as more effective predictive factors with slightly better discriminative ability than TyG and TyG-WHtR. However, no statistically significant difference in predictive ability was observed between TyG-BMI and TyG-WC ($P > 0.05$).

We further evaluated whether the composite indices TyG-BMI and TyG-WC possessed higher predictive value than the individual indices BMI and WC. The AUC (95% CI) for BMI and WC alone were 0.677 (0.652–0.701) and 0.674 (0.650–0.698), respectively. Although these values were slightly lower than those of TyG-BMI and TyG-WC, the differences did not reach statistical significance (BMI vs TyG-BMI, $P = 0.103$; WC vs TyG-WC, $P = 0.203$). However, comparisons using the NRI and IDI demonstrated that TyG-BMI and TyG-WC, which incorporate the TyG index, outperformed BMI and WC alone. The NRI values were 0.219 and 0.190 ($P < 0.01$), and the IDI values were 0.014 and 0.016 ($P < 0.01$).

https://doi.org/10.2147/DMSO.S577687

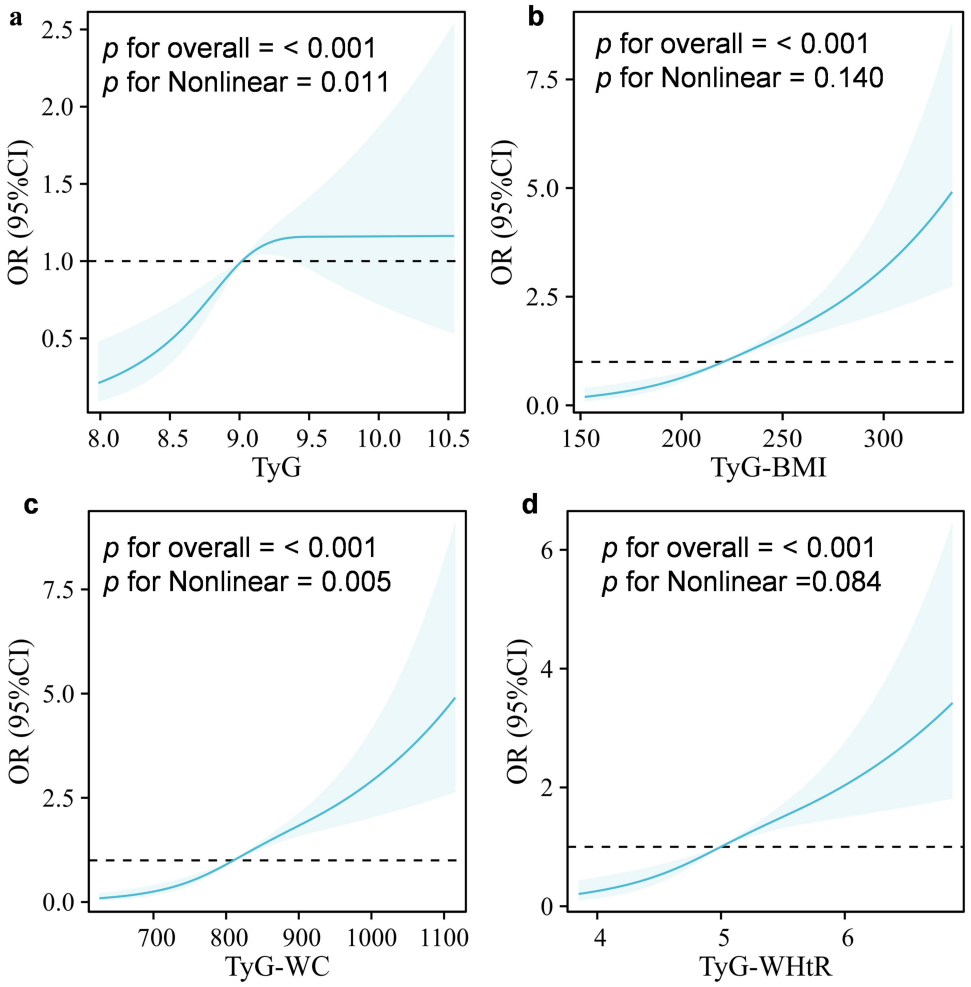

Figure 4 Dose-responsive relationship of the TyG index and its derived indices with the risk of macrosomia. (**a**) TyG; (**b**) TyG-BMI; (**c**) TyG-WC; (**d**) TyG-WHtR; Models were adjusted for Model 3.

Abbreviations: OR, odds ratio; CI, confidence interval; BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; TyG, Triglycerides-glucose index; GDM, gestational diabetes mellitus.

Subgroup Analysis

To further investigate the association of TyG, TyG-BMI, TyG-WC, and TyG-WHtR with macrosomia in women with GDM, subgroup analyses were performed based on maternal age and parity (Figure 6 and Table 3). After adjusting for confounding factors in Model 3, no significant interaction was observed between subgroup variables (age, parity) and the association of TyG and its derived indices with the risk of macrosomia in GDM pregnancies (*P* for interaction >0.05). All

Table 2 Comparative Analysis of TyG and Its Derived Indices in Predicting Macrosomia in GDM

Index	AUC (95% CI)	Cut-off Value	Sensitivity	Specificity	Youden Index
BMI	0.677(0.652–0.701)	25.509	61.0	65.0	0.260
WC	0.674(0.650–0.698)	89.5	75.0	52.3	0.247
TyG	0.596(0.572–0.621)	8.79	86.2	28.2	0.144
TyG-BMI	0.682(0.658–0.706)	233.05	61.0	66.3	0.272
TyG-WC	0.681(0.658–0.705)	832.03	63.3	63.0	0.261
TyG-WHtR	0.651(0.626–0.675)	5.11	61.2	61.0	0.221

(*Continued*)

https://doi.org/10.2147/DMSO.S577687

Table 2 (Continued).

Comparison	TyG vs TyG-BMI		TyG vs TyG-WC		TyG vs TyG-WHtR	
	Difference	***P***	**Difference**	***P***	**Difference**	***P***
AUC	−0.086	<0.01	−0.085	<0.01	−0.055	<0.01
NRI	0.443	<0.01	0.450	0.029	0.343	<0.01
IDI	0.030	<0.01	0.029	<0.01	0.017	<0.01
Comparison	**TyG-BMI vs TyG-WC**		**TyG-BMI vs TyG-WHtR**		**TyG-WC vs TyG-WHtR**	
	Difference	***P***	**Difference**	***P***	**Difference**	***P***
AUC	0.001	0.881	0.031	<0.01	0.030	<0.01
NRI	−0.012	0.801	−0.377	<0.01	−0.516	<0.01
IDI	−0.001	0.693	−0.013	<0.01	−0.012	<0.01
Comparison	**BMI vs TyG-BMI**		**WC vs TyG-WC**			
	Difference	***P***	**Difference**	***P***		
AUC	−0.004	0.103	−0.007	0.203		
NRI	0.219	<0.01	0.190	<0.01		
IDI	0.014	<0.01	0.016	<0.01		

Abbreviations: AUC, area under the curve; CI, confidence interval; IDI, integrated discrimination improvement; NRI, net reclassification improvement; TyG, Triglycerides-glucose index; BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; GDM, gestational diabetes mellitus.

four indices exhibited a positive correlation with macrosomia risk. Notably, TyG-BMI and TyG-WC demonstrated significant correlations in both age and parity subgroups, whereas TyG and TyG-WHtR showed no significant association in the parity ≥1 subgroup and the age ≥35 subgroup, respectively.

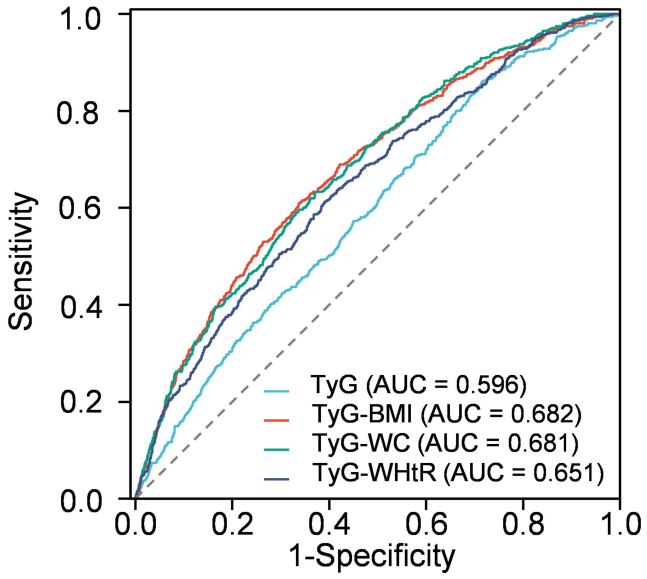

Figure 5 ROC curve analysis of four indexes for macrosomia delivery in GDM Pregnancies.
Abbreviations: BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; TyG, Triglycerides-glucose index; GDM, gestational diabetes mellitus.

https://doi.org/10.2147/DMSO.S577687

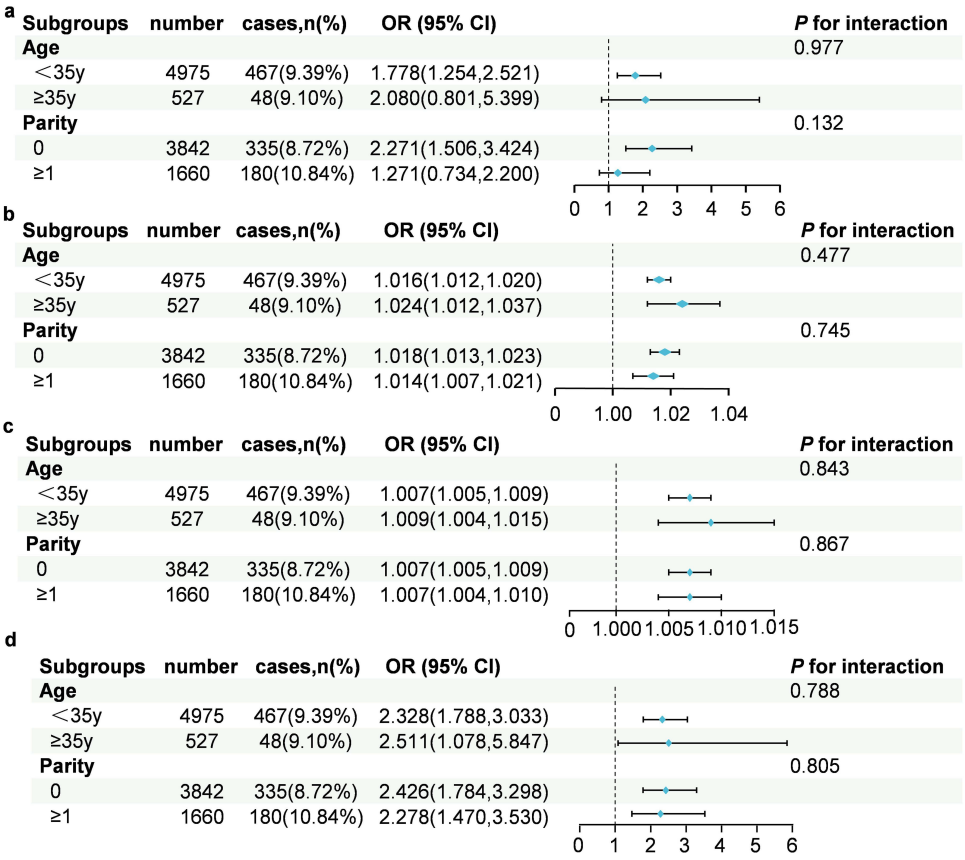

Figure 6 Subgroup analysis of the association between TyG and its derived indices for macrosomia in GDM pregnancies. (**a**) TyG; (**b**) TyG-BMI; (**c**) TyG-WC; (**d**) TyG-WHtR, Models were adjusted for Model 3.

Abbreviations: BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; TyG, Triglycerides-glucose index; GDM, gestational diabetes mellitus.

Discussion

This study analyzed clinical data from women with singleton pregnancies who received routine prenatal care and delivered at the Women's Hospital of Nanjing Medical University, Nanjing Women and Children's Healthcare Hospital. It examined the association between the TyG index and its derived indices (TyG-BMI, TyG-WC, TyG-WHtR) measured during the second trimester and the risk of macrosomia in women with GDM. These findings may support earlier identification and intervention in high-risk populations. The results showed that the probability of delivering a macrosomic infant increased with higher TyG

Table 3 Subgroup Analysis for the Association of the TyG and Its Derived Indices Tertiles for Macrosomia in GDM Pregnancies

Subgroups	Group	Number	Cases, n (%)	OR (95%CI)	*P* for Interaction
TyG					
Age					0.977
< 35 y	T1	1713	103(6.00%)	Ref.	
	T2	1661	164(9.90%)	1.612(1.167–2.227)	
	T3	1601	200(12.50%)	1.718(1.219–2.421)	
≥ 35 y	T1	125	7(5.60%)	Ref.	
	T2	169	16(10.70%)	1.945(0.665–5.688)	
	T3	233	25(10.70%)	2.397(0.82–7.002)	

(*Continued*)

Table 3 (Continued).

Subgroups	Group	Number	Cases, n (%)	OR (95%CI)	*P* for Interaction
Parity					0.132
1	T1	1385	78(5.60%)	Ref.	
	T2	1278	113(8.80%)	1.687(1.152–2.471)	
	T3	1179	144(12.20%)	2.156(1.444–3.22)	
≥ 2	T1	453	32(7.10%)	Ref.	
	T2	552	67(12.10%)	1.512(0.893–2.560)	
	T3	655	81(12.40%)	1.24(0.713–2.157)	
TyG-BMI					
Age					0.478
< 35 y	T1	1717	73(4,30%)	Ref.	
	T2	1649	136(8.20%)	1.983(1.385–2.839)	
	T3	1609	258(16.00%)	3.124(2.160–4.517)	
≥ 35 y	T1	117	2(1.70%)	Ref.	
	T2	185	10(5.40%)	3.32(0.703–15.671)	
	T3	225	36(16.00%)	8.861(1.948–40.308)	
Parity					0.414
1	T1	1361	49(3.6%)	Ref.	
	T2	1280	98(7.70%)	2.766(1.768–4.326)	
	T3	1201	188(15.70%)	4.77(3.004–7.575)	
≥ 2	T1	473	26(5.50%)	Ref.	
	T2	554	48(8.70%)	1.199(0.681–2.109)	
	T3	633	106(16.70%)	2.051(1.180–3.562)	
TyG-WC					
Age					0.843
< 35 y	T1	1740	68(3.90%)	Ref.	
	T2	1667	142(8.50%)	2.315(1.604–3.340)	
	T3	1568	257(16.40%)	3.708(2.553–5.386)	
≥ 35 y	T1	35	3(3.20%)	Ref.	
	T2	166	13(7.80%)	2.006(0.53–7.591)	
	T3	266	32(12.00%)	3.139(0.869–11.344)	
Parity					0.867
1	T1	1384	50(3.60%)	Ref.	
	T2	1303	106(8.10%)	2.775(1.791–4.300)	
	T3	1155	179(15.50%)	4.597(2.933–7.204)	
≥ 2	T1	451	21(4.70%)	Ref.	
	T2	530	49(9.20%)	1.576(0.863–2.877)	
	T3	679	110(16.20%)	2.488(1.387–4.463)	
TyG-WHtR					
Age					0.788
< 35 y	T1	1745	91(5.20%)	Ref.	
	T2	1672	141(8.40%)	1.495(1.064–2.100)	
	T3	1558	235(15.10%)	2.346(1.661–3.312)	

(*Continued*)

https://doi.org/10.2147/DMSO.S577687

Table 3 (Continued).

Subgroups	Group	Number	Cases, n (%)	OR (95%CI)	*P* for Interaction
≥ 35 y	T1	89	5(5.60%)	Ref.	
	T2	162	13(8.00%)	0.999(0.324–3.083)	
	T3	276	30(10.90%)	1.373(0.467–4.035)	
Parity					0.805
1	T1	1392	69(5.00%)	Ref.	
	T2	1306	108(8.30%)	1.672(1.127–2.481)	
	T3	1144	158(13.80%)	2.547(1.694–3.831)	
≥ 2	T1	442	27(6.10%)	Ref.	
	T2	528	46(8.70%)	1.098(0.615–1.959)	
	T3	690	107(15.50%)	1.830(1.055–3.173)	

Notes: Models were adjusted for Model 3.
Abbreviations: TyG, Triglycerides-glucose index; BMI, Body mass index; WC, Waist circumference; WHtR, Waist-to-height ratio; OR, odds ratio; Ref, Reference group; GDM, gestational diabetes mellitus.

index values. Spearman correlation analysis revealed that TyG and its derived indices were positively correlated with TG, TC, and LDL-C but negatively correlated with HDL-C ($P < 0.01$). After adjusting for potential confounding factors, the highest tertiles of TyG, TyG-BMI, TyG-WC, and TyG-WHtR were associated with 1.769-fold, 3.458-fold, 3.718-fold, and 2.279-fold increased risks of macrosomia, respectively, compared with the lowest tertiles (all $P < 0.05$). It indicates that TyG and its derived indicators are independent risk factors for macrosomia in women with GDM.

The TyG index, a metabolic status evaluation metric based on glycemic and lipid parameters, was initially introduced in 2008 as a novel surrogate marker for assessing IR.[29] With advancing understanding of GDM, studies have demonstrated that IR induced by GDM plays a significant role in macrosomia.[30] Research has revealed that the TyG index exhibits diagnostic performance comparable to the OGTT for GDM between 24–28 weeks of gestation.[31] Ning Ma et al[32] confirmed the predictive value of first-trimester TyG index for GDM and its significant correlation with macrosomia. Retrospective analyses have identified pre-pregnancy TyG index as an independent risk factor for macrosomia in GDM patients, demonstrating its combined predictive value with GWG for macrosomia.[33] The underlying mechanism may involve elevated TG and blood glucose during the second-trimester activating serine-threonine kinases that inhibit insulin signaling pathways, thereby exacerbating IR.[34] IR can induce fetal β-cell hyperplasia and accelerate the maturation of the stimulus-secretion coupling mechanism in β-cells. Consequently, the fetus produces excessive insulin in response to hyperglycemia caused by increased placental glucose transfer. As insulin acts as a growth hormone, this leads to enhanced fetal growth and adiposity. The combined effects of hyperglycemia and hyperinsulinemia promote increased fetal fat and protein storage, ultimately resulting in macrosomia.[34]

The results of this study demonstrated that the TyG index during the second-trimester of pregnancy predicted macrosomia in women with GDM with an AUC of 0.596. The optimal cut-off value was 8.79, yielding a sensitivity of 86.3% and specificity of 28.2%. A retrospective cohort study conducted at two tertiary hospitals reported a similar AUC of 0.599 for TyG in predicting macrosomia,[35] which is consistent with our findings. The RCS analysis confirmed these results. Although TyG and its related indices were correlated (all P for overall <0.001), the RCS curve for TyG demonstrated a nonlinear, approximately inverted U-shaped relationship with macrosomia. This phenomenon may result from the combined effects of pathophysiological mechanisms and clinical interventions. The severe glucolipotoxicity associated with an extremely high TyG index may lead to vascular endothelial injury and placental insufficiency,[36] thereby limiting nutrient transport. Furthermore, competitive risks such as the saturation of placental transporters or preterm birth may also restrict further fetal weight gain under extreme metabolic conditions.[37,38] Clinically, a high TyG index often indicates severe hyperglycemia, prompting earlier and more intensive clinical management. However, when glycemic targets are not met, the initiation of insulin therapy effectively curbs excessive fetal growth. This finding suggests that for such high-risk pregnant women, relying solely on routine gestational weight management is often insufficient, and the TyG index can serve as a key tool to guide the timely initiation of intensive metabolic management. The high sensitivity but low specificity of the TyG index suggests that

using a single parameter to predict macrosomia in GDM patients has certain limitations. This may be attributed to the multifactorial nature of GDM,[39] with numerous factors influencing fetal macrosomia, including maternal age, gravidity, parity, and excessive GWG.[35] Therefore, relying solely on a single indicator to predict macrosomia in GDM patients has inherent limitations.

Previous studies have confirmed that maternal body mass index (BMI) is an independent risk factor for macrosomia. Maternal obesity during pregnancy is not only closely associated with hyperinsulinemia and dyslipidemia, but also correlated with impaired endothelial function and increased inflammation.[40,41] A meta-analysis comprising 23 studies (including a total of 17,711 women) demonstrated a significant association between maternal HbA1c levels and macrosomia.[42] A prospective cohort study further revealed that elevated maternal serum lipid levels remained an independent risk factor for macrosomia even after adjusting for maternal blood glucose levels and pre-pregnancy BMI.

Therefore, further analysis was conducted to evaluate the predictive capabilities of TyG-derived indices incorporating obesity-related parameters (TyG-BMI, TyG-WC, and TyG-WHtR) for macrosomia in women with GDM, with their predictive performances compared using DeLong's test. The results demonstrated that TyG-BMI, TyG-WC, and TyG-WHtR yielded AUC values of 0.682, 0.681, and 0.651, respectively, for predicting macrosomia in GDM patients, all significantly superior to the TyG index alone ($P < 0.05$). Notably, TyG-BMI and TyG-WC showed comparable discriminative power, suggesting their equivalent utility in GDM screening ($P = 0.881$). However, compared to BMI alone (AUC = 0.677) or WC alone (AUC = 0.674), the improvement in AUC was modest and not statistically significant ($P > 0.05$). Among these composite indices, BMI and WC appear to be the primary drivers of predictive performance. NRI and IDI analyses further confirmed that TyG-BMI and TyG-WC significantly improved risk reclassification and discriminative ability (all $P < 0.01$). These results suggest that the incorporation of obesity parameters (BMI or WC) provides a more comprehensive assessment of the synergistic effect between metabolic abnormalities and adiposity accumulation, which is consistent with previous studies demonstrating that TyG combined with other risk factors enhances predictive performance.[43]

The underlying mechanism may involve excessive free fatty acids, reactive oxygen species (ROS), and proinflammatory cytokines released by adipose tissue in overweight or obese individuals, which exacerbate insulin resistance, stimulate fetal hyperinsulinemia, promote excessive fetal growth, and consequently increase macrosomia risk. As composite indices derived from TG, FPG, and anthropometric parameters, TyG-BMI and TyG-WC integrate glycolipid metabolic risk factors into clinically actionable metrics. Rather than replacing traditional BMI assessment, these indices serve as complementary tools that enable early screening and lifestyle modification to reduce macrosomia risk in GDM pregnancies, thereby providing valuable guidance for perinatal care.

Furthermore, our subgroup analysis demonstrated that the associations of TyG-BMI and TyG-WC with macrosomia in GDM pregnancies remained consistent across different age and parity groups. However, TyG and TyG-WHtR showed no significant correlation in the age ≥35 years group, and TyG exhibited no significant association in the parity ≥1 group. This may be attributed to the decline in metabolic function among advanced-age pregnant women, who are exposed to additional risk factors beyond IR or obesity. Additionally, altered insulin sensitivity in multiparous women may render single indicators (eg. TyG) less reliable. In contrast, TyG-BMI and TyG-WC, which incorporate body size parameters, exhibit more stable predictive performance.

The profound significance of this study lies in its value for optimizing risk stratification using readily accessible and low-cost indicators. Current clinical practice primarily focuses on metrics such as BMI, blood glucose, and GWG. Incorporating composite indicators like the TyG index and its derivatives for synergistic assessment can capture underlying metabolic risks. These indicators can be calculated during the OGTT at 24–28 weeks of gestation, thereby aiding in the early collaborative identification of high-risk pregnant women and facilitating early, stratified, and precise management.

This study has several limitations. First, dietary influences on blood glucose and lipid levels were not evaluated. Second, the analysis relied solely on second-trimester single-point data, omitting longitudinal tracking throughout pregnancy. Third, for women with GDM, we could not determine whether insulin therapy weakened the observed associations between the TyG index, its derived indices, and macrosomia incidence.

Future studies should integrate nutritional assessments and monitor key indicators longitudinally. Incorporating additional clinical examination data would enable a more comprehensive assessment of TyG and its derived indices in predicting macrosomia risk among GDM patients.

Conclusion

In summary, elevated TyG index and its derived indices during the second-trimester are associated with an increased risk of macrosomia in women with GDM, all demonstrating predictive value. Notably, compared to the TyG index alone, the TyG-BMI and TyG-WC demonstrated better predictive performance. These composite indices can serve as effective screening adjuncts to better identify high-risk pregnancies, particularly by capturing the synergistic risk of obesity and insulin resistance.

Data Sharing Statement

The data underlying this article will be shared on reasonable request to the corresponding author.

Ethics Approval and Consent to Participate

This study followed the Declaration of Helsinki on medical protocols and ethics. Given that this study was a retrospective study and the data did not include any personally identifiable information, the study applied for the exemption from informed consent, which was approved by the Medical Ethics Committee of Nanjing Women and Children's Healthcare Hospital (Approval No. 2022KY-068).

Acknowledgments

We thank all the investigators and subjects who participated in this project.

Author Contributions

Yiting Chen and Shuyu Li should be considered as co-first authors.
Yajun Chen and Tianying Zhong should be considered as co-corresponding authors.
Yiting Chen: Investigation, Formal analysis, Data curation, Conceptualization, Writing – original draft.
Shuyu Li: Investigation, Formal analysis, Data curation, Conceptualization, Writing – original draft.
Lanlan Xiang: Investigation, Data curation, Writing – review and editing.
Yitian Zhu: Investigation, Data curation, Writing – review and editing.
Yu Zeng: Funding acquisition, Formal analysis, Conceptualization, Writing – review and editing.
Yajun Chen: Supervision, Funding acquisition, Conceptualization, Resources, Writing – review and editing.
Tianying Zhong: Supervision, Funding acquisition, Conceptualization, Resources, Writing – review and editing.

All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

Nanjing Medical Science and Technique Development Foundation, No. YKK23151; the Opening Foundation of Key Laboratory, No. JSHD202313; Yingke Xinchuang Research Foundation of Jiangsu Blood Transfusion Association, No. JSYK2024006; Open Project of the State Key Laboratory of Reproductive Medicine of Nanjing Medical University, No. SKLRM-K202107.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Baker AM, Haeri S, Camargo CA, Stuebe AM, Boggess KA. First-trimester maternal vitamin D status and risk for gestational diabetes (GDM) a nested case-control study. *Diabetes Metab Res Rev*. 2012;28(2):164–168. doi:10.1002/dmrr.1282
2. Lyu X, Wang S, Zhong J, et al. Gut microbiome interacts with pregnancy hormone metabolites in gestational diabetes mellitus. *Front Microbiol*. 2023;14:1175065. doi:10.3389/fmicb.2023.1175065
3. American College of Obstetricians and Gynecologists. Macrosomia: ACOG Practice Bulletin, Number 216. *Obstet Gynecol*. 2020;135(1):e18–e35. doi:10.1097/AOG.0000000000003606

4. Sweeting A, Wong J, Murphy HR, Ross GP. A clinical update on gestational diabetes mellitus. *Endocr Rev*. 2022;43(5):763–793. doi:10.1210/endrev/bnac003
5. Chukwuemeka S, Chivese T, Gopinath A, Obikeze K. Adverse pregnancy outcomes in gestational diabetes mellitus: a systematic review and meta-analysis protocol. *BMJ Open*. 2024;14(5):e058625. doi:10.1136/bmjopen-2021-058625
6. Ewington LJ, Hugh O, Butler E, Quenby S, Gardosi J. Accuracy of antenatal ultrasound in predicting large-for-gestational-age babies: population-based cohort study. *Am J Obstet Gynecol*. 2025;232(2):210.e1–210.e10. doi:10.1016/j.ajog.2024.04.052
7. Zhou K, Xiao S, Cao S, Zhao C, Zhang M, Fu Y. Improvement of glucolipid metabolism and oxidative stress via modulating PI3K/Akt pathway in insulin resistance HepG2 cells by chickpea flavonoids. *Food Chem X*. 2024;23:101630. doi:10.1016/j.fochx.2024.101630
8. Sharma AK, Singh S, Singh H, et al. Deep insight of the pathophysiology of gestational diabetes mellitus. *Cells*. 2022;11(17):2672. doi:10.3390/cells11172672
9. Xiang LL, Feng J, Li SY, et al. Predictive ability of lipid indices for large-for-gestational-age infants in pregnant females with gestational diabetes mellitus. *World J Diabetes*. 2025;16(7):104306. doi:10.4239/wjd.v16.i7.104306
10. Park SY, Gautier JF, Chon S. Assessment of insulin secretion and insulin resistance in human. *Diabetes Metab J*. 2021;45(5):641–654. doi:10.4093/dmj.2021.0220
11. Li T, Zhang H, Wu Q, Guo S, Hu W. Association between triglyceride glycemic index and gout in US adults. *J Health Popul Nutr*. 2024;43(1):115. doi:10.1186/s41043-024-00613-4
12. Pan Y, Zhong S, Zhou K, et al. Association between diabetes complications and the triglyceride-glucose index in hospitalized patients with type 2 diabetes. *J Diabetes Res*. 2021;2021:8757996. doi:10.1155/2021/8757996
13. Sergi D, Spaggiari R, Dalla Nora E, et al. HOMA-IR and TyG index differ for their relationship with dietary, anthropometric, inflammatory factors and capacity to predict cardiovascular risk. *Diabet Res Clin Pract*. 2025;222:112103. doi:10.1016/j.diabres.2025.112103
14. Mansoori A, Allahyari M, Mirvahabi MS, et al. Predictive properties of novel anthropometric and biochemical indexes for prediction of cardiovascular risk. *Diabetol Metab Syndr*. 2024;16(1):304. doi:10.1186/s13098-024-01516-4
15. Zhao L, Zheng L, Wang R, et al. Association between triglyceride glucose combined with body mass index and hypertension in the NHANES 2017 to 2020. *Sci Rep*. 2025;15(1):9092. doi:10.1038/s41598-025-93723-w
16. Tahapary DL, Pratisthita LB, Fitri NA, et al. Challenges in the diagnosis of insulin resistance: focusing on the role of HOMA-IR and Tryglyceride/glucose index. *Diabetes Metab Syndr*. 2022;16(8):102581. doi:10.1016/j.dsx.2022.102581
17. Zhu YT, Xiang LL, Chen YJ, Zhong TY, Wang JJ, Zeng Y. Developing and validating a predictive model of delivering large-for-gestational-age infants among women with gestational diabetes mellitus. *World J Diabetes*. 2024;15(6):1242–1253. doi:10.4239/wjd.v15.i6.1242
18. Zeng Y, Yin L, Yin X, Zhao D. Association of triglyceride-glucose index levels with gestational diabetes mellitus in the US pregnant women: a cross-sectional study. *Front Endocrinol*. 2023;14:1241372. doi:10.3389/fendo.2023.1241372
19. Mo Z, Cao C, Han Y, Hu H, He Y, Zuo X. Relationships between triglyceride-glucose index and incident gestational diabetes mellitus: a prospective cohort study of a Korean population using publicly available data. *Front Public Health*. 2024;12:1294588. doi:10.3389/fpubh.2024.1294588
20. Kim JA, Kim J, Roh E, et al. Triglyceride and glucose index and the risk of gestational diabetes mellitus: a nationwide population-based cohort study. *Diabet Res Clin Pract*. 2021;171:108533. doi:10.1016/j.diabres.2020.108533
21. Sánchez-García A, Rodríguez-Gutiérrez R, Saldívar-Rodríguez D, et al. Early triglyceride and glucose index as a risk marker for gestational diabetes mellitus. *Int J Gynaecol Obstet*. 2020;151(1):117–123. doi:10.1002/ijgo.13311
22. Pan Y, Zou S, Xu Y, et al. Is there any association between early trimester triglyceride-glucose index and incidence of hypertensive disorder of pregnancy and adverse pregnancy outcomes? *Front Endocrinol*. 2023;14:1093991. doi:10.3389/fendo.2023.1093991
23. Cui C, Qi Y, Song J, et al. Comparison of triglyceride glucose index and modified triglyceride glucose indices in prediction of cardiovascular diseases in middle aged and older Chinese adults. *Cardiovasc Diabetol*. 2024;23(1):185. doi:10.1186/s12933-024-02278-z
24. Yang Y, Wang H, He Y, He X, Yao Z, Yang H. Analysis of the association between TG/HDL, TyG, TyG-BMI, and ZJU indices with metabolic dysfunction-associated fatty liver disease in patients with type 2 diabetes. *DMSO*. 2025;18:3797–3812. doi:10.2147/DMSO.S549457
25. Ni W, Jiang R, Xu D, et al. Association between insulin resistance indices and outcomes in patients with heart failure with preserved ejection fraction. *Cardiovasc Diabetol*. 2025;24(1):32. doi:10.1186/s12933-025-02595-x
26. Rokicka D, Hudzik B, Wróbel M, et al. The prognostic impact of insulin resistance surrogates in patients with acute myocardial infarction with and without type 2 diabetes. *Cardiovasc Diabetol*. 2024;23(1):147. doi:10.1186/s12933-024-02240-z
27. Tian J, Cao Y, Zhang W, et al. The potential of insulin resistance indices to predict non-alcoholic fatty liver disease in patients with type 2 diabetes. *BMC Endocr Disord*. 2024;24(1):261. doi:10.1186/s12902-024-01794-z
28. Todi S, Sagili H, Kamalanathan SK. Comparison of criteria of International Association of Diabetes and Pregnancy Study Groups (IADPSG) with National Institute for Health and Care Excellence (NICE) for diagnosis of gestational diabetes mellitus. *Arch Gynecol Obstet*. 2020;302(1):47–52. doi:10.1007/s00404-020-05564-9
29. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metab Syndr Relat Disord*. 2008;6(4):299–304. doi:10.1089/met.2008.0034
30. Yuan Y, Zhu Q, Yao X, Shi Z, Wen J. Maternal circulating metabolic biomarkers and their prediction performance for gestational diabetes mellitus related macrosomia. *BMC Pregnancy Childbirth*. 2023;23(1):113. doi:10.1186/s12884-023-05440-9
31. Sánchez-García A, Rodríguez-Gutiérrez R, Saldívar-Rodríguez D, et al. Diagnostic accuracy of the triglyceride-glucose index for gestational diabetes screening: a practical approach. *Gynecol Endocrinol*. 2020;36(12):1112–1115. doi:10.1080/09513590.2020.1742687
32. Ma N, Bai L, Lu Q. First-trimester triglyceride-glucose index and triglyceride/high-density lipoprotein cholesterol are predictors of gestational diabetes mellitus among the four surrogate biomarkers of insulin resistance. *Diabetes Metab Syndr Obes*. 2024;17:1575–1583. doi:10.2147/DMSO.S454826
33. Liu PJ, Liu Y, Ma L, et al. The predictive ability of two triglyceride-associated indices for gestational diabetes mellitus and large for gestational age infant among chinese pregnancies: a preliminary cohort study. *Diabetes Metab Syndr Obes*. 2020;13:2025–2035. doi:10.2147/DMSO.S251846
34. Kc K, Shakya S, Zhang H. Gestational diabetes mellitus and macrosomia: a literature review. *Ann Nutr Metab*. 2015;66(Suppl 2):14–20. doi:10.1159/000371628

35. Bai X, Zhu Q, Wang W, et al. Second-trimester triglyceride-glucose index to predict adverse outcomes in women with gestational diabetes mellitus: a retrospective multicenter cohort study. *J Diabetes Investig*. 2024;15(10):1489–1499. doi:10.1111/jdi.14269
36. Yalcin SE, Nerez N, Yalcin Y, Cetinkaya BD. First-trimester atherogenic index of plasma and triglyceride–glucose indices in pregestational diabetes mellitus: associations with adverse pregnancy outcomes. *BMC Pregnancy Childbirth*. 2025;25:1351. doi:10.1186/s12884-025-08491-2
37. Song S, Luo Q, Zhong X, Huang M, Zhu J. An elevated triglyceride-glucose index in the first-trimester predicts adverse pregnancy outcomes: a retrospective cohort study. *Arch Gynecol Obstetrics*. 2025;311(3):915. doi:10.1007/s00404-025-07973-0
38. Cui J, Jiang H, Huang F, et al. First-trimester triglyceride-glucose index and birth weight: a retrospective cohort mediation analysis of preterm birth and gestational complications. *BMC Pregnancy Childbirth*. 2025;25(1):765. doi:10.1186/s12884-025-07885-6
39. Xiang LL, Chen C, Wang QY, Zhu YT, Chen YJ, Zeng Y. Impact of inflammatory factors, hemoglobin A1c, and platelet parameters in gestational diabetes mellitus. *Arch Gynecol Obstet*. 2023;307(2):439–446. doi:10.1007/s00404-022-06528-x
40. Scheidl TB, Brightwell AL, Easson SH, Thompson JA. Maternal obesity and programming of metabolic syndrome in the offspring: searching for mechanisms in the adipocyte progenitor pool. *BMC Med*. 2023;21(1):50. doi:10.1186/s12916-023-02730-z
41. Denizli M, Capitano ML, Kua KL. Maternal obesity and the impact of associated early-life inflammation on long-term health of offspring. *Front Cell Infect Microbiol*. 2022;12:940937. doi:10.3389/fcimb.2022.940937
42. Mou SS, Gillies C, Hu J, et al. Association between HbA1c levels and fetal macrosomia and large for gestational age babies in women with gestational diabetes mellitus: a systematic review and meta-analysis of 17,711 Women. *J Clin Med*. 2023;12(11):3852. doi:10.3390/jcm12113852
43. Wang X, Liu J, Cheng Z, Zhong Y, Chen X, Song W. Triglyceride glucose-body mass index and the risk of diabetes: a general population-based cohort study. *Lipids Health Dis*. 2021;20(1):99. doi:10.1186/s12944-021-01532-7