Section: Biochemistry

Original Research Article

CLINICAL SIGNIFICANCE OF THE TRIGLYCERIDE-GLUCOSE INDEX IN IDENTIFYING INSULIN RESISTANCE AMONG NON-DIABETIC INDIVIDUALS

Arshiya Masood Osmani[1], *Sufia Liaquat*[2]

[1]*Associate Professor, Department of Biochemistry, Dr. V.R.K. Women's Medical College, Teaching Hospital and Research Centre, Aziznagar, Hyderabad, Telangana, India,*
[2]*Duty Medical Officer, Glenfield Mallareddy Brain and Heart Hospital, Nampally, Hyderabad, Telangana, India,*

Received *: 06/10/2026*
Received in revised form *: 04/11/2026*
Accepted *: 17/11/2026*

Corresponding Author:
Dr. Arshiya Masood Osmani,
Associate Professor, Department of Biochemistry, Dr. V.R.K. Women's Medical College, Teaching Hospital and Research Centre, Aziznagar, Hyderabad, Telangana, India..
Email: arshiya1346@gmail.com

DOI: 10.70034/ijmedph.2026.4.385

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 2565-2568

ABSTRACT

Background: Insulin resistance underlies metabolic syndrome and type 2 diabetes mellitus, often developing years before blood glucose reaches diabetic thresholds. This study evaluates whether the triglyceride-glucose (TyG) index can serve as a practical, low-cost surrogate marker for identifying insulin resistance among non-diabetic adults.

Materials and Methods: We conducted an observational cross-sectional study of 100 non-diabetic adults at Dr. V.R.K. Women's Medical College, Teaching Hospital and Research Centre. Demographic profiles and lifestyle habits were documented using a structured questionnaire. Following an overnight fast, venous blood samples were assayed for plasma glucose, serum triglycerides, and fasting insulin. Data were analyzed using SPSS.

Results: The cohort recorded a mean TyG index of 8.55 ± 0.44 and a mean HOMA-IR of 2.01 ± 1.33, with 24% of participants meeting the criterion for insulin resistance (HOMA-IR ≥ 2.5). The TyG index correlated moderately with HOMA-IR ($r = 0.48$, $p < 0.001$) and yielded an area under the ROC curve (AUC) of 0.72 (95% CI: 0.60–0.85). At an optimal cutoff of 8.47, sensitivity reached 87.5% while specificity was 52.6%.

Conclusion: Owing to its high sensitivity and negative predictive value, the TyG index works well as a first-line screening tool to rule out insulin resistance in non-diabetic patients. However, its modest specificity means elevated scores require confirmatory testing before establishing a clinical diagnosis.

Keywords: Triglyceride-glucose index; Insulin resistance; HOMA-IR; Non-diabetic adults; Metabolic screening.

INTRODUCTION

Insulin resistance (IR) stems from chronic low-grade metabolic inflammation and cellular stress across insulin-sensitive tissues.[1] Because IR precedes overt hyperglycemia by several years, detecting it early in non-diabetic individuals offers a critical window for preventive intervention.

Although the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) remains a standard research tool, calculating it requires fasting serum insulin assays. In routine Indian healthcare settings, fasting insulin testing adds substantial cost, lacks uniform standardization across laboratories, and is rarely available at primary care clinics.[2] Clinicians therefore need simpler screening markers derived from routine biochemical panels.

The triglyceride-glucose (TyG) index—computed directly from fasting plasma glucose and serum triglyceride values—has emerged as a practical alternative.[3] Hepatic insulin resistance impairs both insulin-mediated glucose suppression and very-low-density lipoprotein (VLDL) triglyceride clearance, linking lipid and glucose dysregulation within a single ratio.[4] While recent studies highlight the diagnostic value of the TyG index across cardiometabolic conditions.[5] diagnostic cutoffs vary markedly across ethnic groups.

We conducted this study to evaluate the clinical utility of the TyG index in detecting insulin resistance among non-diabetic adults at Dr. V.R.K. Women's Medical College, Teaching Hospital and Research Centre, Hyderabad.

The Objectives of this Study are:

1. Establish the distribution of TyG index values in our non-diabetic hospital cohort.
2. Evaluate the correlation and diagnostic accuracy of the TyG index against HOMA-IR.
3. Examine how demographic traits and self-reported lifestyle habits relate to metabolic risk in this population.

MATERIALS AND METHODS

Study Design and Population: This observational cross-sectional study took place over six months from January to June 2026 in the Department of Biochemistry at Dr. V.R.K. Women's Medical College, Teaching Hospital and Research Centre, Aziznagar, Hyderabad. Using non-probability convenience sampling, we recruited 100 non-diabetic adults aged 18 years and older. Patients with previously diagnosed diabetes mellitus, severe acute or chronic systemic illness, or active use of glucose-lowering or lipid-modifying drugs were excluded.

Ethical Considerations: Prior approval was obtained from the Institutional Ethics Committee (IEC) of Dr. V.R.K. Women's Medical College. Written informed consent was collected from every participant after explaining the study purpose, blood draw procedures, and their right to withdraw at any point without affecting their medical care.

Data Collection and Biochemical Assays: Using a structured, interviewer-administered questionnaire, we recorded age, family history of diabetes, dietary habits (intake of fatty/processed foods and fruits/vegetables), physical activity levels, 30-minute exercise frequency, sleep quality, and perceived chronic stress. Participants also noted whether a physician had previously informed them of elevated blood glucose or abnormal lipid levels.

After an overnight fast of 8 to 12 hours, venous blood samples were collected under aseptic precautions. Fasting plasma glucose (FPG) and serum triglycerides (TG) were measured on an automated clinical chemistry analyzer using standard enzymatic methods, and fasting serum insulin was estimated via chemiluminescence immunoassay.

Surrogate indices were calculated using standard formulas:

TyG Index = ln [(Fasting Triglycerides [mg/dL] × Fasting Plasma Glucose [mg/dL]) / 2]

HOMA-IR = (Fasting Insulin [μU/mL] × Fasting Plasma Glucose [mg/dL]) / 405

Subjects with a HOMA-IR score ≥ 2.5 were classified as insulin-resistant.

Statistical Analysis: Data were coded and analyzed in SPSS. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were summarized as frequencies and percentages. Pearson's correlation coefficient (r) assessed linear relationships among the TyG index, HOMA-IR, and coded lifestyle scores. Independent-samples t-tests compared continuous variables between groups, and Chi-square (χ^2) tests evaluated associations between categorical variables. Receiver Operating Characteristic (ROC) curve analysis determined the area under the curve (AUC), optimal Youden cutoff, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of the TyG index. Statistical significance was set at $p < 0.05$ (two-tailed).

RESULTS

Cohort Characteristics: Among the 100 non-diabetic adults enrolled, 33% were aged 18–25 years and 24% were aged 26–35 years, placing 57% of the study sample under 36 years of age [Figure 1]. Moderate physical activity was reported by 40% of participants, whereas 26% engaged in high or very high physical activity. Frequent intake of fatty or processed foods was reported by 28%, and poor or very poor sleep quality was noted by 28%. Notably, 73% reported having been previously informed of borderline or elevated blood glucose or lipid levels, 22% had normal prior screening results, and 5% had never been screened previously [Figure 2].

Biochemical Findings and Diagnostic Performance of the TyG Index: Across the cohort, the mean TyG index was 8.55 ± 0.44 and the mean HOMA-IR was 2.01 ± 1.33. Using the HOMA-IR ≥ 2.5 threshold, 24% of participants were classified as insulin-resistant.

The TyG index showed a statistically significant, moderate positive correlation with HOMA-IR ($r = 0.48$, $p < 0.001$). Mean TyG values were significantly higher in the insulin-resistant group than in the insulin-sensitive group (8.81 vs. 8.47; Cohen's d = 0.82). The coefficient of determination ($r^2 = 0.23$) indicated that the TyG index accounted for 23% of the total variance in HOMA-IR.

ROC curve analysis demonstrated moderate diagnostic discrimination for the TyG index, with an AUC of 0.72 (95% CI: 0.60–0.85). At the optimal Youden cutoff of 8.47, the TyG index yielded a sensitivity of 87.5%, specificity of 52.6%, PPV of 37.0%, and NPV of 93.0% [Table 1].

Table 1: Summary of Biochemical Outcomes and Diagnostic Accuracy of the TyG Index (N = 100)

Parameter	Value / Outcome
Mean TyG Index (Overall Cohort)	8.55 ± 0.44
Mean HOMA-IR (Overall Cohort)	2.01 ± 1.33
Prevalence of Insulin Resistance (HOMA-IR ≥ 2.5)	24.0% (n = 24)
Mean TyG Index: Insulin-Resistant vs. Non-Insulin-Resistant	8.81 vs. 8.47 (Cohen's d = 0.82)
Correlation with HOMA-IR	r = 0.48, r^2 = 0.23 (p < 0.001)
ROC Area Under the Curve (AUC)	0.72 (95% CI: 0.60–0.85)
Diagnostic Performance at Youden Cutoff (8.47)	Sensitivity: 87.5% | Specificity: 52.6% | PPV: 37.0% | NPV: 93.0%

Lifestyle and Demographic Associations: Comparing participants who had previously been informed of abnormal glucose or lipid levels (n = 73) with those who had not (n = 22; excluding 5 unscreened subjects), independent-samples t-tests showed significant differences in fatty/processed food intake ($t(93) = -2.128$, $p = 0.036$), 30-minute exercise frequency ($t(93) = -2.048$, $p = 0.043$), prolonged stress ($t(93) = -2.119$, $p = 0.037$), and sleep patterns ($t(93) = -2.586$, $p = 0.011$; Cohen's $d = -0.629$). Age ($p = 0.068$), overall physical activity ($p = 0.235$), and fruit/vegetable consumption ($p = 0.169$) showed no significant group differences.

Pearson's correlation analysis revealed weak negative correlations between prior abnormal metabolic history and fatty/processed food intake ($r = -0.216$, $p = 0.036$), 30-minute exercise frequency ($r = -0.208$, $p = 0.043$), prolonged stress ($r = -0.215$, $p = 0.037$), and sleep patterns ($r = -0.259$, $p = 0.011$) (Figure 3). Chi-square tests confirmed significant associations between prior metabolic abnormalities and age group ($\chi^2 = 9.163$, $df = 3$, $p = 0.027$, Cramer's $V = 0.311$), fatty/processed food intake ($\chi^2 = 6.651$, $df = 2$, $p = 0.036$, $V = 0.265$), prolonged stress ($\chi^2 = 7.223$, $df = 2$, $p = 0.027$, $V = 0.276$), and family history of diabetes ($\chi^2 = 6.813$, $df = 1$, $p = 0.009$, $V = 0.268$).

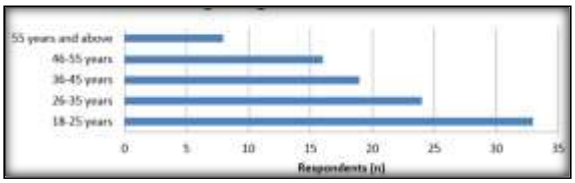

Figure 1: Age Distribution of Study Participants

This figure illustrates the age breakdown of the 100 study participants, showing that 57% of the cohort fell within the young adult age range of 18 to 35 years. The largest single group comprised individuals aged 18–25 years (33%).

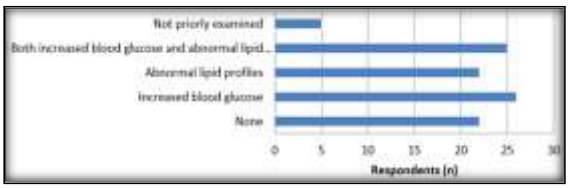

Figure 2: Prior Awareness of Raised Glucose or Lipid Levels

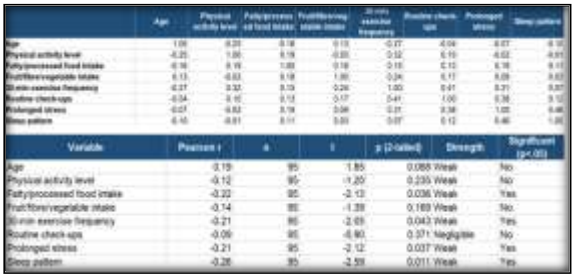

Figure 3: Pearson's Correlation Matrix of Lifestyle Traits and Metabolic Risk Indicators

[Figure 2] summarizes self-reported prior screening awareness among respondents regarding blood glucose and lipid abnormalities. A significant majority (73%) reported having been previously informed of elevated glucose or lipid parameters.

[Figure 3] presents the correlation matrix and statistical values evaluating relationships between lifestyle variables and metabolic risk indicators. It highlights weak yet statistically significant negative correlations linking dietary habits, exercise frequency, stress, and sleep patterns with prior metabolic findings.

DISCUSSION

Detecting insulin resistance early in non-diabetic adults is crucial for preventing progression to type 2 diabetes. Although 57% of our study participants were under 36 years of age, nearly one in four (24%) already exhibited biochemical insulin resistance (HOMA-IR $\geq$ 2.5). This finding highlights the substantial burden of subclinical metabolic dysfunction among young Indian adults.

Our results confirmed a moderate positive correlation between the TyG index and HOMA-IR ($r = 0.48$, $p < 0.001$), alongside significantly higher TyG scores among insulin-resistant subjects (8.81 vs. 8.47). These observations mirror findings from the ELSA-Brasil study by Lelis et al,[6] which noted moderate agreement between the two indices while emphasizing that they capture distinct physiological facets of insulin resistance. Whereas HOMA-IR primarily reflects hepatic insulin-mediated glucose regulation, the TyG index captures triglyceride-driven lipotoxicity. Because the TyG equation excludes circulating insulin, accounting for 23% of HOMA-IR variance ($r^2 = 0.23$) is biologically consistent. Combining the TyG index with anthropometric parameters such as body mass index (TyG-BMI) or waist circumference can further improve predictive accuracy.[7]

With an AUC of 0.72, high sensitivity (87.5%), and strong negative predictive value (93.0%) at a cutoff of 8.47, the TyG index functions effectively as a rule-out screening test rather than a standalone diagnostic marker. Notably, our optimal threshold of 8.47 is lower than cutoffs established in European populations, such as $\geq$ 8.741 in a Polish cohort reported by Aliyu et al,[8] and aligns closely with population-specific thresholds proposed in South Indian cohorts.[3] This discrepancy reflects the well-recognized propensity of South Asian populations to develop visceral adiposity and insulin resistance at lower lipid and body-mass thresholds, underscoring the importance of regional reference cutoffs.

Significant associations linking prior metabolic abnormalities with age, family history of diabetes, processed food intake, and chronic stress align with the multifactorial pathogenesis of insulin resistance.[9,10] Interestingly, the weak negative correlations between lifestyle scores and prior

abnormal reports likely stem from reverse causality: participants who had previously been counseled by clinicians regarding borderline glucose or lipid values may have adopted healthier habits before enrolling in this study.

Limitations: This study is limited by its single-center, cross-sectional design and modest sample size (n = 100), which preclude causal inference and limit wider generalizability. Additionally, anthropometric indices such as BMI and waist circumference were not incorporated into composite TyG models, and lifestyle metrics relied on self-reported questionnaires subject to recall bias.

CONCLUSION

The TyG index correlates moderately with HOMA-IR and provides high sensitivity and negative predictive value for identifying insulin resistance in non-diabetic adults. Because it requires only routine fasting glucose and triglyceride measurements, the TyG index offers a practical, low-cost first-line screening tool for ruling out insulin resistance in resource-limited clinical settings. However, given its modest specificity (52.6%), positive screening results should be followed by confirmatory biochemical and clinical evaluation. Larger multi-center studies across Indian populations are needed to validate standardized diagnostic cutoffs.

REFERENCES

1. Gusev E, Sarapultsev A, Zhuravleva Y. Insulin resistance and inflammation. Int J Mol Sci. 2026;27:PMC12898699.
2. Ramalingam S, Kar AK, Senthil R. Comparison of triglyceride/glucose index and related parameters with Indian Diabetes Risk Score assessment in non-diabetic individuals visiting primary healthcare centre—A community-based cross-sectional study. J Family Med Prim Care. 2024;13(1):235–42.
3. Jog KS, Eagappan S, Santharam RK, Subbiah S. Comparison of novel biomarkers of insulin resistance with homeostasis model assessment of insulin resistance, its correlation to metabolic syndrome in South Indian population and proposition of population specific cutoffs for these indices. Cureus. 2023;15(1):e33653.
4. Kurniawan LB. Triglyceride-glucose index as a biomarker of insulin resistance, diabetes mellitus, metabolic syndrome, and cardiovascular disease: a review. EJIFCC. 2024;35(1):44–51.
5. Sun Y, Ji H, Sun W, An X, Lian F. Triglyceride glucose (TyG) index: a promising biomarker for diagnosis and treatment of different diseases. Eur J Intern Med. 2025;131:3–14.
6. Lelis DF, Baldo TOF, Andrade JM, Griep RH, Bensenor I, Lotufo PA, et al. High triglyceride-glucose index and HOMA-IR are associated with different cardiometabolic profiles in adults from the ELSA-Brasil study. Clin Biochem. 2024;131:110793.
7. Liu L, Luo Y, Liu M, Tang C, Liu H, Feng G, et al. Triglyceride glucose-related indexes and lipid accumulation products—reliable markers of insulin resistance in the Chinese population. Front Nutr. 2024;11:1373039.
8. Aliyu U, Toor SM, Abdalhakam I, Elrayess MA, Abou-Samra AB, Albagha OM. Evaluating indices of insulin resistance and estimating the prevalence of insulin resistance in a large biobank cohort. Front Endocrinol. 2025;16:1591677.
9. Chandrasekaran P, Weiskirchen R. Cellular and molecular mechanisms of insulin resistance. Curr Tissue Microenviron Rep. 2024;5(3):79–90.
10. Rogers EM, Banks NF, Jenkins ND. The effects of sleep disruption on metabolism, hunger, and satiety, and the influence of psychosocial stress and exercise: a narrative review. Diabetes Metab Res Rev. 2024;40(2):e3667.