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Perspective

The triglyceride-glucose index and obesity: A perspective review

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Obesity has become one of the major public health challenges worldwide, as its prevalence continues to rise[1]. The impact of obesity extends beyond a mere increase in body fat, as it is inherently linked to the development of various metabolic pathologies, such as arterial hypertension, type 2 diabetes, dyslipidemia, and metabolic syndrome. These diseases interact in a vicious cycle, where obesity serves as a factor that initiates and exacerbates metabolic alterations, while the latter perpetuate insulin resistance, chronic low-grade inflammation, and endothelial dysfunction. In this context, some indices enable an objective, standardized, and comparative assessment of excess body fat and its associated health risks, transforming simple measurements into diagnostic and predictive tools that guide prevention, early diagnosis, and intervention for obesity and its complications. Specifically, the triglyceride-glucose (TyG) index has emerged as a valuable clinical marker for assessing insulin resistance, a condition frequently linked to obesity and an elevated risk of metabolic diseases. Unlike traditional anthropometric indices, the TyG index incorporates biochemical parameters and is both easy and inexpensive to calculate, requiring only fasting triglyceride and glucose levels. It was first proposed and validated by Simental-Mendía *et al*[2] and has since demonstrated high diagnostic accuracy in studies on cardiometabolic diseases. The index is calculated using the following formula[2,3]: TyG index = ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL)] / 2.

Although the TyG index has been extensively studied in various metabolic conditions, its specific and exclusive relationship with obesity has not been sufficiently conceptualized or critically examined, leaving a notable gap in the current scientific literature. In this context, this perspective article aims to analyze the most recent evidence on the use of the TyG index in obesity and to discuss the prospects for its potential generalization across different levels of healthcare, focusing on its clinical applicability, limitations, and future directions for broader integration. This article was conceived as a perspective review rather than a systematic review, and the literature analyzed was identified through targeted searches in MEDLINE/PubMed, Scopus, and Web of Science, focusing on recent observational and cohort studies published between 2020 and 2025 that examined the association between the TyG index and obesity-related phenotypes. Priority was given to studies with large sample sizes, diverse populations, and clinically relevant outcomes to ensure the representativeness of current evidence. The selected studies are summarized in ***Table 1***.

The scientific rationale for applying the TyG index in obesity lies in the fact that triglyceride accumulation in the liver and skeletal muscle impairs insulin signaling, while elevated glucose levels reflect a state of metabolic compensation. Thus, the TyG index integrates two central pathophysiological pathways of obesity: dysregulated lipid metabolism and glycemic dysfunction[3–7]. Insulin resistance drives increased lipolysis, elevated free fatty acids, impaired glucose uptake, and compensatory hyperinsulinemia,

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Table 1 **Recent evidence on the triglyceride-glucose (TyG) index and obesity.**

Regions	Authors (year)	Population/Design	Main findings
China	Zhang et al. (2024)[4] Zhao et al. (2025)[5] Zuo et al. (2025)[6] Yang et al. (2025)[7] Wen et al. (2025)[8]	Cross-sectional and longitudinal studies	The TyG index is associated with diabesity and with visceral obesity, with reported odds ratios ranging from 2.5 to 3.1. It shows a positive correlation with BMI and WC. A high TyG index predicts sarcopenic obesity, being associated with a 2.4-fold higher risk of the condition and showing a strong correlation with fat mass and abdominal circumference. The TyG index is also correlated with visceral fat (r = 0.42), supporting its usefulness for identifying abdominal obesity, and with liver fat and other adiposity parameters (r = 0.38).
Republic of Korea	Kim S et al. (2023)[10] Kim B et al. (2024)[11]	Obese adults and seniors	The TyG index is correlated with LDL particle size, with an AUC of 0.897, and is associated with a higher risk in women. It is also associated with sarcopenic obesity and with metabolically unhealthy obesity.
Iran/Saudi Arabia	Gholami et al. (2023)[13] El-Sehrawy et al. (2025)[14]	Obese women and adults	Unhealthy dietary patterns and systemic inflammation are associated with elevated TyG levels, which are linked to higher visceral fat and a worse cardiometabolic profile. The TyG index also predicts risky eating behaviors and metabolic syndrome.
Italy	Pontiroli et al (2024)[15]	Cohort with obesity and type 2 diabetes	The TyG index predicts mortality and metabolic syndrome and serves as a prognostic marker of cardiometabolic risk.
USA	Huang et al. (2025)[16] Xiao et al. (2025)[17]	NHANES and cross-sectional studies	The TyG index is correlated with BMI, WC, and obesity, with an AUC greater than 0.80, demonstrating better discriminatory performance than BMI alone. In addition, TyG-derived indices, including TyG-BMI and TyG-WC, predict obesity and sarcopenia.

Abbreviations: TyG: triglyceride–glucose index; BMI: body mass index; WC: waist circumference; LDL: low-density lipoprotein; AUC: area under the curve.

creating a metabolic milieu that promotes visceral adiposity and cardiometabolic deterioration[6–8]. The TyG index has emerged as an accessible surrogate marker of insulin resistance, validated against the hyperinsulinemic-euglycemic clamp[3–8]. A key advancement is the development of composite indices, such as TyG-body mass index (BMI), TyG-waist circumference (WC), and TyG-waist-to-height ratio (WHtR), which demonstrate superior discriminatory power compared to traditional anthropometric measures in individuals with high-risk obesity[5,7–9]. Collectively, these findings support a growing consensus: the TyG index provides a more integrated assessment of metabolic health than isolated indicators and holds significant potential for guiding personalized interventions, including GLP-1 receptor agonists and digital health monitoring.

Recent international studies consistently demonstrate a strong association between the TyG index and various obesity phenotypes, particularly visceral and sarcopenic obesity, reinforcing its value as a marker of metabolic severity. Although most studies have been conducted in Asian populations, especially in China[6–9] and South Korea[10–12], emerging data from Europe and North America[16,17] confirm its broad applicability across diverse demographic contexts. Overall, elevated TyG levels are associated with a substantially increased risk of diabetes and obesity, an adverse body fat distribution, and metabolically unhealthy obesity profiles, suggesting that the TyG index reflects not only excess adiposity but also its pathological metabolic consequences. Regression models have reported significant associations, with a mean 2.6-fold higher odds of visceral obesity, sarcopenic obesity, or obesity-specific metabolic syndrome in individuals with elevated TyG (OR/HR range: 1.7–4.3; mean = 2.63 ± 0.79)[5-7,12,15].

Furthermore, a positive correlation has been consistently reported between the TyG index and obesity-related parameters, such as visceral/abdominal fat, body fat percentage, BMI, and waist circumference (r = 0.38; range: 0.30–0.45; SD = ±0.05)[7–9,11,13]. Therefore, the TyG index may serve as a robust predictor of visceral obesity, given its strong association with metabolic risk[7,8,11,14]. It has been positively correlated with visceral fat area (VFA) and the VFA-to-subcutaneous fat area ratio, a key diagnostic indicator of visceral-type abdominal obesity, which is associated with an elevated risk of metabolic diseases[6]. This suggests that the TyG index reflects not only overall adiposity but also the metabolically hazardous distribution of fat in the abdominal region[7,13,15] (***Table 1***).

Some studies have reported a higher risk associated with elevated TyG levels in women, older individuals, and populations with comorbidities[6,11]. Elevated TyG levels have also been associated with increased overall mortality in individuals with obesity, further underscoring its prognostic value[6,7]. Moreover, the TyG index may serve as a useful tool for distinguishing metabolically healthy obese individuals

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(those with normal TyG) from those at significant metabolic risk (those with elevated TyG)[12], thereby supporting more precise risk stratification and personalized clinical management (***Table 1***).

Beyond demonstrating strong associations, the available evidence also reveals considerable variability in optimal TyG index cutoff points across different populations, reflecting differences in lifestyle and metabolic profiles. This heterogeneity underscores the need for context-specific interpretation and highlights the importance of establishing population-specific thresholds to enhance the clinical accuracy of the TyG index as a tool for obesity screening and risk stratification.

In China, where visceral obesity and metabolic liver disease are predominant, higher TyG cutoff points (8.9–9.0) suggest that the index reflects a higher threshold for metabolic risk[4,7]. In contrast, in South Korea, slightly lower cut-offs (8.7–8.8) support its utility in the early detection of atherogenic dyslipidemia[11]. In Iran and Saudi Arabia, findings reinforce the association between the TyG index and behavioral and dietary factors, highlighting the contribution of unhealthy diets, systemic inflammation, and eating disorders to elevated cardiometabolic risk. In Europe, particularly in Italy, the progressive association between elevated TyG levels and mortality among obese patients with diabetes or impaired glycemia further underscores its prognostic value beyond the initial diagnosis of metabolic syndrome[13,14].

In the study by Wen et al.[8], the TyG index showed a positive correlation with BMI (β = 1.4; 95% confidence interval [CI]: 1.3–1.5). Huang et al.[16] reported significant associations between TyG-BMI, TyG-WHtR, and TyG-WC and hypertension, with odds ratios (ORs) and 95% CIs ranging from 1.01 to 1.02 for TyG-BMI, 2.17 to 2.83 for TyG-WHtR, and 1.00 to 1.01 for TyG-WC (all $P < 0.000\,1$), confirming the usefulness of TyG-derived indices as markers of obesity-related metabolic risk, with discriminative capacity that surpasses traditional indicators such as BMI. From this perspective, the available evidence positions the TyG index as a robust and practical biomarker with high potential to refine the diagnostic and prognostic approach to obesity. Beyond reflecting adiposity burden, TyG integrates metabolic risk and insulin resistance into a single composite indicator, enabling more accurate stratification of high-risk obesity phenotypes. Its application offers a strategic opportunity to guide individualized therapeutic and preventive interventions, particularly in settings where early identification of metabolic vulnerability is essential for optimizing clinical outcomes.

The TyG index, as a metabolic indicator derived from routine blood tests, is easily accessible and applicable, making it a practical and economical tool in both primary care and other levels of healthcare. Its strong association with obesity indicators, such as BMI, WC, ECI, and BAI, supports its use in individualized intervention strategies that include lifestyle modifications and comprehensive metabolic health management. In primary care settings, where early identification and targeted management of at-risk individuals can significantly improve public health outcomes, the TyG index is particularly valuable. By integrating information on glucose and triglyceride levels, it provides a comprehensive view of metabolic health and enables timely interventions, such as dietary modifications, increased physical activity, and control of metabolic risk factors, even before the onset of clinical symptoms. Thus, it could serve as an early marker for risk assessment, stratification, monitoring, and the prevention of complications in patients with obesity.

Its predictive accuracy could be influenced by factors such as age and sex, highlighting the need to define population-specific cutoff points. An elevated TyG index in a person with obesity may indicate a high-risk metabolic profile requiring more urgent interdisciplinary intervention. Furthermore, the TyG index may help differentiate metabolically healthy obese individuals (those with normal TyG values) from those with significant metabolic risk (those with elevated TyG). This distinction could facilitate personalized treatment approaches and more precise risk stratification, thereby consolidating the TyG index as a prognostic biomarker capable of identifying individuals who would benefit from targeted preventive or therapeutic strategies.

The integration of the TyG index into routine clinical practice for patients with obesity remains an ongoing proposal; however, its interpretation should be contextualized within a comprehensive clinical evaluation and approached with methodological caution until more longitudinal evidence becomes available to confirm its predictive utility and applicability across diverse clinical settings. Despite its advantages, the TyG index has methodological and clinical limitations that warrant consideration. Standardization is still lacking in both units and calculation formulas, hindering comparability across studies. Extrinsic factors, such as diet, physical activity level, and the use of lipid-lowering medications, can alter TyG values and affect result interpretation. Moreover, without adjustment for other

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biomarkers (*e.g.*, serum insulin or adiposity-derived indices), the TyG index may overestimate insulin resistance. Finally, ethnic and sex variability in reported cut-off points limits its universal applicability and underscores the need for population-specific criteria.

Although the TyG index and its derivatives, such as TyG-BMI, TyG-WC, and TyG-WHtR, have established roles as accessible biomarkers of insulin resistance in obese or at-risk individuals, challenges remain that hinder their full clinical integration. The lack of standardized cutoff points across heterogeneous populations, including children, older adults, and diverse ethnic groups stratified by sex and age, may limit generalizability. This limitation is compounded by the predominance of cross-sectional study designs, which introduce causal ambiguity. Future research should prioritize longitudinal, multicenter studies integrating genomics and metabolomics to validate the predictive value of the TyG index and to develop personalized cutoff thresholds that enhance its diagnostic accuracy and risk-stratification utility. Additionally, randomized controlled trials evaluating interventions such as glucagon-like peptide-1 (GLP-1) receptor agonists or lifestyle modifications, particularly in obese individuals with prehypertension, could clarify the potential causal relationships, the role of TyG in preventing cardiovascular complications, and its utility in therapeutic monitoring. Such research would address current knowledge gaps and enable more robust validation of the TyG index, ultimately positioning it as a key tool for early detection, risk stratification, comprehensive metabolic assessment, and personalized interventions in the context of the global obesity epidemic. Given that obesity is a multifactorial condition, the TyG index should complement, not replace, a holistic clinical evaluation that incorporates medical history, lifestyle factors, genetic predispositions, and other relevant diagnostic tests.

In conclusion, the TyG index has demonstrated a strong association with obesity, underscoring its value as both a diagnostic and prognostic tool that reflects not only total adiposity but also the deleterious distribution of fat in the abdominal region and its links to multiple obesity-related comorbidities. Its ease of application and cost-effectiveness position it as a promising biomarker for the early detection and stratification of metabolic risk in individuals with obesity across diverse levels of healthcare settings.

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