Review

The Triglyceride–Glucose (TyG) Index and Cardiovascular Diseases: A Narrative Review

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Review

The Triglyceride–Glucose (TyG) Index and Cardiovascular Diseases: A Narrative Review

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Abstract

Background: The triglyceride–glucose (TyG) index, calculated from fasting triglyceride and glucose levels, has emerged as a simple and cost-effective surrogate marker of insulin resistance (IR). Given that IR is a pivotal mechanism underlying cardiovascular disease (CVD), the TyG index has gained increasing attention in recent years. Objective: This review summarizes the current evidence on the association between the TyG index and cardiovascular risk, exploring its biochemical basis, role in metabolism, epidemiological findings, and clinical implications. Methods: A narrative review of published studies was conducted, focusing on the predictive value of the TyG index for insulin resistance, diabetes, metabolic syndrome, and CVD outcomes. Findings from cohort, cross-sectional, and meta-analysis studies were integrated to provide a comprehensive overview. Results: Multiple epidemiological and clinical studies demonstrate that higher TyG index values are associated with an increased risk of cardiovascular events, independent of traditional risk factors. Compared to the homeostasis model assessment of insulin resistance (HOMA-IR), the TyG index shows greater accessibility and predictive capacity. However, heterogeneity in cut-off thresholds and limited prospective and interventional trial data remain major challenges. Conclusions: The TyG index represents a promising biomarker for cardiovascular risk assessment and may serve as a supplementary tool for early screening, particularly in young and asymptomatic adults. Future large-scale prospective and interventional studies are warranted to standardize cut-off values and establish its role in clinical practice.

Keywords: triglyceride–glucose index; insulin resistance; cardiovascular disease; biomarker; metabolic syndrome; diabetes; cardiovascular risk prediction

1. Introduction

In recent years, several cost-effective markers of insulin resistance (IR) have been proposed. [1]. IR is widely recognized as a key contributor to the development of cardiovascular diseases (CVDs). Among these markers, the triglyceride–glucose (TyG) index—calculated as the product of fasting triglycerides and glucose—has gained attention as a simple and reliable surrogate for IR, with previous studies suggesting superior diagnostic accuracy compared with the homeostasis model assessment of insulin resistance (HOMA-IR) [2–5]. Notably, the TyG index has demonstrated greater sensitivity and specificity for detecting metabolic syndrome and CVD than HOMA-IR [3,6].

Despite the growing recognition of its clinical value, evidence regarding the predictive role of the TyG index in healthy young populations remains limited. Early identification of cardiovascular risk in this age group is particularly important, as preventive interventions may significantly reduce long-term morbidity and mortality. The present study therefore aimed to evaluate the TyG index as a predictor of incident CVD and all-cause mortality in a large, nationally representative cohort of young Korean adults, consistent with recent nationwide data [1,4]. Using data from the National Health Insurance Service–National Sample Cohort (NHIS-NSC), we analyzed 448,624 participants aged 20–39 years without prior CVD or diabetes who underwent baseline health examinations

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between 2009 and 2010. The TyG index was calculated as Ln (fasting triglycerides × fasting glucose / 2) and stratified into two categories based on the optimal cut-off value determined by time-dependent receiver operating characteristic (ROC) analysis. The primary outcomes were incident CVD events and all-cause mortality, assessed using Cox proportional hazards models to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) across TyG categories.

Understanding Triglycerides:

Since the mid-20th century, much of the dietary debate has focused on the role of carbohydrates, particularly refined sugars and high-fructose corn syrup (HFCS), in metabolic health. This emphasis contributed to the widespread promotion of "low-fat" and "fat-free" products, which often replaced dietary fats with simple carbohydrates. However, fats are essential macronutrients required for numerous physiological processes, and their indiscriminate elimination is neither biologically justified nor beneficial, especially in sedentary populations.

Excessive carbohydrate intake, particularly from refined sources, has been shown to elevate plasma triglyceride concentrations. Triglycerides, the most common form of dietary fat, serve as a primary energy source and are synthesized in the liver from surplus glucose and fructose. While triglycerides are vital for normal metabolism, persistently elevated levels (hypertriglyceridemia) are associated with an increased risk of atherosclerotic cardiovascular disease and stroke, even when cholesterol levels remain within normal ranges [1,2].

Lipoproteins play a central role in lipid transport: low-density lipoprotein (LDL) contributes to atherogenesis by promoting cholesterol deposition in arterial walls, whereas high-density lipoprotein (HDL) facilitates reverse cholesterol transport. Elevated triglycerides may exacerbate the atherogenic profile by altering LDL and HDL balance. Historical dietary studies have demonstrated that higher proportions of simple carbohydrates in total caloric intake lead to increased triglyceride levels, supporting the link between carbohydrate excess and lipid dysregulation [2].

Moreover, nutritional interventions such as increased intake of omega-3 fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]) and vitamin C–rich foods have been shown to lower plasma triglyceride concentrations. Hypertriglyceridemia is frequently observed in both type 1 and type 2 diabetes mellitus, reflecting impaired carbohydrate tolerance and underscoring the interrelationship between glucose metabolism and lipid regulation [3].

Biochemical Properties:

Atherogenic lipids, including cholesterol and triglycerides, are strongly associated with the risk of myocardial infarction and stroke, which remain urgent global health concerns. Circulating triglycerides, particularly when carried in lipoprotein-rich particles such as very-low-density lipoproteins (VLDL) and chylomicrons, contribute to fatty acid deposition within the arterial wall [4]. Systematic reviews and meta-analyses have confirmed that elevated triglyceride levels are independently associated with adverse cardiovascular outcomes [7–11].

Following a high-calorie meal, postprandial hypertriglyceridemia and hyperinsulinemia may occur, impairing the anti-inflammatory response and promoting endothelial stress. Excess free fatty acids released from adipose tissue stimulate endothelial dysfunction through several mechanisms: reduction in nitric oxide bioavailability, induction of oxidative stress, and activation of pro-inflammatory signaling pathways such as nuclear factor-κB (NF-κB). Endothelial dysfunction represents the initial stage of atherogenesis, characterized by increased vascular permeability, platelet activation, and leukocyte adhesion [10,11].

As these processes progress, oxidized lipoproteins are internalized by macrophages, forming foam cells and contributing to fatty streak development. Smooth muscle cell proliferation and extracellular matrix deposition ultimately lead to fibrous cap formation. However, inflammatory mediators and matrix metalloproteinases released by activated immune cells can weaken the fibrous cap, predisposing to plaque rupture. This cascade of events underlies the pathophysiology of acute coronary syndromes and ischemic stroke [5].

Given these risks, reliable markers of insulin resistance (IR) and lipid dysregulation are crucial for early detection. The triglyceride–glucose (TyG) index has emerged as a practical alternative to the

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homeostasis model assessment of insulin resistance (HOMA-IR). The TyG index, calculated from fasting triglyceride and glucose levels, has shown strong predictive value for cardiovascular events, including myocardial infarction and stroke [6–8]. Unlike HOMA-IR, which requires insulin assays that are costly, time-consuming, and less widely available, the TyG index is inexpensive, reproducible, and applicable in large-scale population screening. This makes it particularly valuable for identifying at-risk but asymptomatic individuals in the general population [11,12].

Role in Metabolism:

The fasting triglyceride–glucose (TyG) index, calculated as ln [fasting triglyceride (mg/dL) × fasting glucose (mg/dL) / 2], integrates two routinely measured biochemical parameters—fasting triglycerides (TG) and fasting glucose (FG). Accumulating evidence indicates that the TyG index is a reliable surrogate marker of insulin resistance (IR) and is now widely applied in large-scale clinical studies. A recent meta-analysis confirmed that TyG demonstrates high sensitivity and specificity compared with the euglycemic–hyperinsulinemic clamp, the current gold standard [6]. Beyond IR, the TyG index has been associated with an increased risk of type 2 diabetes, metabolic syndrome, and cardiovascular disease [11–15]. Recent findings also demonstrated a significant association between higher TyG index values and the severity of coronary atherosclerosis, as well as poor functional outcomes in stroke patients undergoing reperfusion therapy [7]. From a public health perspective, the TyG index represents a cost-effective, practical tool for risk stratification.

Glucose Metabolism:

Glucose metabolism is tightly regulated to maintain adequate energy supply to tissues and systemic homeostasis. Dysregulation contributes to the pathogenesis of both microvascular complications (retinopathy, nephropathy, neuropathy) and macrovascular disease (atherosclerosis, myocardial infarction, stroke). Large-scale analyses have confirmed that each 1% increase in glycated hemoglobin (HbA1c) confers an 18–30% higher risk of microvascular complications, while elevated fasting glucose is independently associated with CVD incidence [8]. Pharmacological glucose-lowering interventions reduce macrovascular risk, though the magnitude of benefit varies among drug classes [17,18].

Insulin Resistance:

IR is a reduced ability of insulin to stimulate glucose uptake in muscle or suppress hepatic glucose production, and it plays a central role in metabolic syndrome, diabetes, and CVD [9]. While fasting insulin has been proposed as a surrogate, its correlation with clamp-derived insulin sensitivity is limited. TyG-based measures have consistently shown stronger correlations with insulin action than fasting insulin or HOMA-IR [1,2,8]. Recent evidence suggests TyG may serve as a superior predictor of coronary artery disease severity and cardiovascular outcomes compared with other IR indices [14,15].

Glycemic Control:

Elevated TG and FG levels are consistently linked to IR. Epidemiological studies involving tens of thousands of participants have shown that TyG is a reliable and practical surrogate for IR [5]. Beyond IR detection, higher TyG quartiles have been linked to increased prevalence of ischemic heart disease, heart failure, and stroke [4,15,16]. A multinational cohort confirmed the association between elevated TyG and greater risk of myocardial infarction, stroke, and cardiovascular mortality [5]. Early detection of IR using TyG may therefore guide preventive interventions in populations with increasing rates of obesity and sedentary lifestyles.

The Triglyceride–Glucose Index:

The triglyceride–glucose (TyG) index, derived from fasting triglyceride and fasting glucose levels, has gained recognition as a novel marker of insulin resistance and cardiovascular risk. Recent cross-sectional studies have reported continuous positive associations between the TyG index and carotid atherosclerosis—measured via carotid intima–media thickness (cIMT)—independent of traditional risk factors [20,21]. Elevated TyG levels were also associated with increased carotid plaque and higher odds of subclinical arterial disease in healthy populations [10].

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Although diabetes mellitus is diagnosed primarily through plasma glucose criteria, vascular damage often develops early, raising the risk of macrovascular complications. This underscores the importance of early preventive measures. Owing to its simplicity and accessibility, the TyG index offers a practical tool for assessing metabolic dysfunction and stratifying cardiovascular risk.

Analyses have shown that higher TyG index values correlate with greater numbers of significantly stenosed carotid arteries and each component of carotid atherosclerosis. Indeed, in a cohort of ischemic stroke patients, those in the highest TyG quartile had significantly elevated odds of abnormal cIMT (adjusted ORs ~1.56) compared to the lowest quartile [11]. This reinforces TyG's role as a surrogate marker of subclinical atherosclerosis. Incorporating TyG into vascular assessment strategies may thus provide a cost-effective approach to improving vascular health and reducing cardiovascular morbidity.

The TyG index has also been recommended for population screening in Korean adults, with health checks every three years starting at age 20. In large cohort analyses, models adjusted for glucose, cholesterol, blood pressure, medications, smoking, alcohol, physical activity, education, BMI, and (in some cases) type 2 diabetes status support its robustness across confounders [1].

Calculated as ln [TG × FG / 2], the TyG index shows strong correlations with insulin resistance measured by hyperinsulinemic–euglycemic clamp tests, as well as hepatic and myocardial insulin sensitivity [6]. Other metabolic or lipoprotein-based indices often fail to outperform it in predictive strength.

In a Korean NHIS–based cohort of 679,498 young adults (19–39 years) free of CVD or diabetes, higher TyG values were significantly associated with incident chronic conditions including cardiovascular disease and kidney disorders. Analyses excluding implausible lab values and applying Cox and negative binomial regression models strengthened the evidence supporting TyG as a population-level risk marker [1].

Cardiovascular Diseases Overview:

Cardiovascular disease (CVD) includes coronary artery disease, stroke, and peripheral vascular disease—leading causes of global morbidity and mortality. In Korea, CVD is the top cause of death among men and women, with cerebrovascular disease ranking second overall [12]. The rising prevalence of CVD aligns closely with rapid socioeconomic changes, population aging, and unfavorable shifts in lifestyle.

Established risk factors include hypertension, dyslipidemia, impaired glucose metabolism, smoking, excessive alcohol consumption, physical inactivity, and obesity. The World Health Organization emphasizes these cardiometabolic factors as major contributors to global mortality, highlighting the need for early prevention and screening.

Triglycerides (TGs) and glucose represent key metabolic indices linked to cardiometabolic health. Under normal circumstances, TGs supply energy via β-oxidation, but sustained elevations lead to ectopic fat deposition, hepatic steatosis, lipotoxicity, and insulin resistance. These metabolic disruptions promote endothelial dysfunction and atherosclerosis, accelerating vascular injury and increasing risk of major cardiovascular events. Meta-analyses have shown that elevated TG levels independently elevate CVD risk and all-cause mortality in a dose–response fashion [25,26].

The TyG index, integrating fasting TG and glucose, has been validated in multiple populations as a surrogate IR marker and CVD risk predictor [20,23]. Its use enables early detection of at-risk individuals and may facilitate preventive lifestyle or pharmacological interventions.

Types of Cardiovascular Diseases:

Excluding congenital heart disease, there are five main categories of cardiovascular disease: coronary heart disease (CHD), cerebrovascular disease (stroke), hypertensive heart disease, rheumatic heart disease, and other forms of cardiac disease [6].CHD is the most frequent type. It evolves through atherosclerotic plaque formation in coronary vessels, with plaques composed of lipids, inflammatory cells, and necrotic debris. These plaques narrow vessel lumens and impair coronary perfusion, causing myocardial ischemia. Clinically, this manifests as angina. Whereas stable angina is predictable and relieved by rest, unstable angina can occur unpredictably and may precede

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myocardial infarction. Atrial fibrillation can occur via structural remodeling and fibrosis in CHD, disrupting normal electrical conduction.

Cerebrovascular disease includes ischemic and hemorrhagic stroke. Ischemic strokes (≈70% of cases) often result from thrombus formation triggered by plaque rupture or endothelial injury. A functional endothelium inhibits platelet aggregation and thrombosis, but injury leads to hypercoagulable states and vessel occlusion. Hemorrhagic strokes arise from vessel rupture under pressure. Clinically, stroke may cause deficits like aphasia along with emotional sequelae (depression, anxiety, irritability) that impair quality of life.

Risk Factors:

The triglyceride–glucose (TyG) index, calculated as ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL) / 2], has emerged as a promising surrogate marker of insulin resistance (IR). IR is a central feature of type 2 diabetes and metabolic syndrome and is strongly linked to the development of coronary heart disease and other cardiovascular conditions. Large meta-analyses have shown that higher TyG index values are associated with elevated incidence of coronary artery disease (CAD), myocardial infarction, and composite CVD [6].

The TyG index demonstrates a close linear correlation with the hyperinsulinemic–euglycemic clamp, the gold standard for assessing IR, and may outperform surrogate measures such as HOMA-IR and QUICKI in certain cohorts (2). Nevertheless, no universally accepted cut-off value exists, and further prospective studies are required to standardize thresholds for cardiovascular prediction.

In a large Korean cohort, individuals in the highest TyG quartile had significantly higher risks of myocardial infarction, stroke, and all-cause mortality compared to the lowest quartile, independent of traditional risk factors [6].

Association Between the TyG Index and Cardiovascular Diseases:

Because direct insulin sensitivity testing (e.g., clamp technique) is costly and impractical, the TyG index offers a simple, accessible alternative. A recent meta-analysis including >6 million participants found that individuals with the highest TyG values had significantly increased risks of CAD (HR ≈ 2.01), myocardial infarction (HR ≈ 1.36), and composite CVD (HR ≈ 1.46) [6]. These associations were also confirmed in large-scale Korean cohorts [1].

Epidemiological Studies:

Multiple epidemiological cohorts support the role of TyG in predicting metabolic and cardiovascular outcomes. In a Chinese population study, individuals in the top TyG quartile had a 1.48-fold higher risk of CVD and a 1.68-fold higher risk of CHD compared with those in the lowest quartile [13]. A Korean 16-year prospective study also reported baseline TyG as an independent predictor of incident ASCVD [14].

A meta-analysis of 13 cohort studies confirmed that subjects in the highest TyG group had nearly a twofold higher risk of atherosclerotic outcomes [13]. Moreover, in type 1 diabetes patients, TyG was associated with increased all-cause mortality and CVD risk [15].

Mechanisms of Action:

The TyG index captures the dual dysregulation of glucose and lipid metabolism. Elevated triglycerides increase free fatty acid (FFA) release, impair insulin signaling, and reduce clearance of glucose, while chronic hyperglycemia induces oxidative stress and endothelial dysfunction. Together, these synergistically promote vascular injury [16].

Additionally, TyG has been associated with arterial stiffness and coronary artery calcification (CAC). A meta-analysis of 26 studies found that each 1-unit increase in TyG was associated with ~1.5× higher odds of arterial stiffness and ~1.7× higher odds of CAC [17].

Clinical Implications:

Because TyG can be easily calculated from routine lab values, it may serve as a cost-effective tool for early risk stratification. In young Korean adults, higher TyG predicted incident stroke, myocardial infarction, and CVD-related mortality, independent of obesity and other risk factors [1].

Meta-analyses across diverse populations also reinforce its prognostic value [20,27].

Screening and Diagnosis:

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In a retrospective Chinese cohort (n=102,061), higher TyG quartiles were consistently associated with increased risk of new-onset CVD, with adjusted HR 1.17 for the highest vs lowest quartile [13]. These findings were robust across subgroups defined by fasting glucose, triglyceride levels, obesity, and hypertension.

Treatment Strategies:

Prospective cohorts demonstrate that higher baseline TyG predicts new-onset diabetes and CKD. ROC analyses show that TyG has higher predictive accuracy than HOMA-IR (AUC 0.77 vs 0.40) for diabetes development [18]. Lifestyle modification and pharmacological interventions targeting glucose and lipid metabolism can lower TyG, but randomized interventional data remain limited.

Comparative Analysis with Other Biomarkers:

Compared with HOMA-IR and QUICKI, TyG offers greater cost-effectiveness, reproducibility, and broader clinical applicability. Integrating TyG with traditional risk models enhances discrimination for CVD risk prediction [26,30].

Traditional Risk Factors:

TyG incorporates two classical risk factors—hyperglycemia and hypertriglyceridemia—and has outperformed isolated markers such as total cholesterol and HDL in predicting IR and CVD outcomes [23,26].

Novel Biomarkers:

Novel indices such as TyG-BMI and TyG-waist have been developed, combining TyG with anthropometric measures. A 2025 meta-analysis reported that higher TyG-BMI was associated with significantly greater CVD risk (HR ~1.62) [19]. Integrating TyG with inflammatory and genetic biomarkers is an emerging research direction.

Limitations of the TyG Index

Limitations include:

Heterogeneity of cut-off thresholds across populations [23,27].

Confounding by lifestyle and metabolic factors (diet, exercise, medications).

Observational nature of most evidence.

Lack of proof that TyG adds incremental predictive value over established CVD risk models [9].

Population Variability:

Recent studies have confirmed that the triglyceride–glucose (TyG) index is an independent predictor of cardiovascular disease (CVD) [6]. Although no absolute cut-off value has been universally established, evidence suggests that “lower is better” when considering target levels for cardiovascular prevention. Importantly, both the baseline TyG index and its variability over time appear to influence cardiovascular risk. While higher baseline TyG values are consistently associated with adverse outcomes, emerging data indicate that the magnitude and trajectory of change in TyG levels may be equally relevant. Longitudinal analyses suggest that individuals with persistently elevated TyG or greater variability exhibit higher incidence rates of CVD compared with those maintaining stable, low levels. For example, hazard ratios (HRs) of 1.82 and 1.64 were observed among individuals with optimal mean TyG but poor variability versus those with both optimal mean and stable variability over seven years [6]. These findings underscore the need to consider temporal patterns in addition to absolute TyG values in population risk assessment. Monitoring TyG variability may provide prognostic information beyond single-point measurements and improve stratification of high-risk individuals. At the population level, interventions targeting both glucose and lipid control may reduce TyG variability and improve long-term cardiovascular outcomes.

Measurement Challenges:

Although the TyG index is widely recognized as a surrogate marker of insulin resistance and is linked with cardiovascular outcomes, several challenges limit its routine clinical use. First, most studies focus on apparently healthy or CVD-free cohorts; data in populations with established heart disease are limited. Some studies report associations between TyG and rehospitalization or in-hospital mortality in heart failure patients, but results are inconsistent [1]. Second, TyG values may be significantly impacted by lifestyle, diet, and medications. Lipid-lowering agents can lower TG

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levels, whereas certain antihypertensive drugs (e.g., ACE inhibitors, ARBs, calcium channel blockers) may increase fasting glucose, confounding TyG-based risk estimation [13]. Third, methodological caveats include selection bias in recruitment, variability in laboratory assays, and lack of standardized cut-off values across populations. While long-term follow-up studies (≥7 years) show promising prognostic utility, TyG's predictive accuracy needs validation against gold-standard measures like the hyperinsulinemic–euglycemic clamp. Finally, the absence of universally accepted thresholds and the influence of comorbid conditions (ischemia, diabetes, metabolic syndrome) remain barriers to widespread adoption.

Future Research Directions:

Despite its limitations, the TyG index has seen broad uptake as a simple and reproducible IR surrogate, especially in high-risk populations. Evidence supports its association with early impaired glucose tolerance and development of type 2 diabetes [14]. In hypertensive patients, those in the highest TyG quartile exhibited more than twice the risk of incident diabetes compared to the lowest quartile [13]. These observations suggest that TyG may help identify individuals at elevated metabolic risk. Nonetheless, further research is needed to broaden its applicability: 1. Large, multiethnic prospective cohorts to define standardized cut-offs, 2. Studies assessing its incremental predictive value over conventional risk factors, 3. Focused evaluation in vulnerable populations (e.g., pregnancy, multi-morbidity) to reduce false-positive findings, and 4. Incorporation into multi-marker risk frameworks combining metabolic, inflammatory, and imaging biomarkers to enhance predictive performance.

Longitudinal Studies:

Multiple cohort studies support the TyG index as a stable and informative biomarker. In Korean young adults, elevated baseline TyG was predictive of cardiovascular events and mortality [1]. TyG correlates strongly with established IR measures including insulin suppression tests, hyperinsulinemic–euglycemic clamps, and HOMA-IR, suggesting that TyG is more than merely a surrogate [20]. Its utility as a screening tool in asymptomatic young populations has also been proposed, with some studies showing greater sensitivity than traditional indices [21]. However, TyG's predictive performance may differ based on the presence of existing cardiovascular disease, underscoring the need for disease-specific cut-off values. Longitudinal cohorts have reported optimal threshold values (e.g. ~5.34) for incident outcomes, but heterogeneity in study populations and designs limits generalizability. Prospective, well-powered studies are needed to validate these thresholds, explore TyG dynamics over time, and assess its added prognostic value beyond established risk factors.

Interventional Trials:

While observational data strongly associate TyG with metabolic and cardiovascular outcomes, intervention studies specifically targeting TyG are scarce. The existing evidence suffers from variability in proposed cut-offs (ranging >8.8 to ≥9.0), which undermines comparability and consistency across populations (10). Some cross-sectional analyses report high sensitivity and specificity values (e.g., ≥95% sensitivity, ~90% specificity) for detecting IR using TyG, but prevalence estimates vary widely across cohorts. Because prospective, randomized trials are lacking, causal inference regarding TyG modulation and outcome improvement remains unproven. Future interventional trials should (a) validate cut-off thresholds across ethnic groups, (b) test whether interventions that lower TyG translate into reduced cardiovascular events, and (c) evaluate how TyG-guided strategies compare with conventional risk-based interventions.

2. Conclusions

The TyG index, calculated as ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL) / 2], is emerging as a practical, low-cost, and effective surrogate marker of insulin resistance. Given the pivotal role of IR in cardiovascular disease pathogenesis, TyG offers significant promise, especially in resource-limited settings where gold-standard measures are impractical. While most evidence is observational and heterogeneity in thresholds and populations exists, the consistent associations

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between elevated TyG and adverse outcomes provide a compelling rationale for further validation. Ultimately, the TyG index may become an integral part of cardiovascular risk assessment and preventive cardiometabolic strategies—provided that future research establishes robust cut-offs, confirms incremental predictive value, and clarifies its role in interventional settings.

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Abbreviations

CVD	Cardiovascular disease
CHD	Coronary heart disease
CKD	Chronic kidney disease
DM	Diabetes mellitus
FG	Fasting glucose
HbA1c	Hemoglobin A1c
HDL-C	High-density lipoprotein cholesterol
HOMA-IR	Homeostasis model assessment of insulin resistance
HTN	Hypertension
IR	Insulin resistance
LDL	Low-density lipoprotein
MetS	Metabolic syndrome
NHIS	National Health Insurance Service
TG	Triglyceride
TyG	Triglyceride–glucose

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