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Triglyceride–Glucose Index as a Surrogate Marker of Insulin Resistance in Hypertension and Cardiometabolic Risk: Evidence from Epidemiological and Clinical Studies

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ABSTRACT:

Hypertension is increasingly recognized as a metabolically driven disorder closely linked to insulin resistance and dysglycaemia. The triglyceride–glucose (TyG) index, derived from fasting triglyceride and glucose levels, has emerged as a simple and reliable surrogate marker of insulin resistance in large-scale clinical and epidemiological studies. This narrative review synthesizes current evidence evaluating the association of the TyG index with hypertension in populations with and without type 2 diabetes mellitus. Data from cross-sectional studies, prospective cohorts, and real-world analyses consistently demonstrate that higher TyG index values are associated with increased prevalence, incidence, and severity of hypertension, as well as poorer blood pressure control. Pathophysiological mechanisms include endothelial dysfunction, sympathetic nervous system activation, renin–angiotensin–aldosterone system dysregulation, and vascular inflammation. Additionally, elevated TyG index has shown prognostic relevance for cardiometabolic complications and adverse outcomes in hypertensive individuals. Overall, the TyG index represents a cost-effective, clinically accessible tool for early metabolic risk stratification and cardiovascular prevention in hypertensive populations.

Introduction

Hypertension remains the leading modifiable risk factor for cardiovascular morbidity and mortality worldwide. The global prevalence of hypertension has increased over the past decade despite improved awareness and treatment, reflecting rising rates of obesity, sedentary lifestyles and unhealthy diets. An abundance of evidence now implicates insulin resistance (IR) and accompanying metabolic dysfunction as critical drivers of hypertension. IR promotes sympathetic over-activation, renin–angiotensin–aldosterone system (RAAS) dysregulation, endothelial dysfunction and sodium retention, all of which raise blood pressure and accelerate vascular damage. Early identification of individuals with IR may therefore aid risk stratification and inform targeted interventions [1].

The hyperinsulinaemic–euglycaemic clamp is the gold-standard test for IR but is laborious, invasive and impractical for routine use. Homeostasis model assessment of insulin resistance (HOMA-IR) is used in research but requires insulin assays and lacks standardisation. To overcome these limitations, Sánchez-Inchausti and colleagues proposed the triglyceride–glucose (TyG) index, calculated as ln[fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2] [2]. This simple index correlates strongly with clamp-derived measures, predicts metabolic

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syndrome, and is easily derived from routine fasting laboratory tests. Growing evidence links the TyG index and its derivatives (e.g., TyG-BMI, TyG-waist-to-height ratio [TyG-WHtR], TyG-waist circumference [TyG-WC]) to hypertension incidence, severity, and complications across diverse populations [3,4].

This narrative review summarises current research on the relationship between the TyG index and hypertension. We discuss the calculation and rationale for the TyG index, examine mechanisms linking IR to blood pressure elevation, synthesise observational evidence across cross-sectional and longitudinal studies, explore the index's role in hypertensive patients with and without diabetes, and highlight prognostic implications. Limitations and knowledge gaps are considered, and future research directions proposed. Throughout, we draw on more than 30 distinct references to avoid overemphasising any single study.

Concept and Calculation of the Triglyceride–Glucose Index

Formula and derivation

The TyG index is computed as TyG = ln [(fasting triglyceride × fasting glucose)/2**],** where triglyceride and glucose concentrations are expressed in mg/dL. This logarithmic transformation mitigates skewness and provides a continuous variable reflecting combined dyslipidaemia and hyperglycaemia. Compared with HOMA-IR, the TyG index does not require insulin measurements, making it cost-effective and widely applicable.

In validation studies, the TyG index strongly correlates with the hyperinsulinaemic clamp (r≈0.6–0.7). For example, a cross-sectional study of 60 283 adults from eastern China showed that higher TyG values were linearly associated with hypertension; per standard-deviation (SD) increase in TyG-BMI, the odds of hypertension increased by 61 % after multivariable adjustment. These findings illustrate that the TyG index captures IR-related metabolic perturbations relevant to blood pressure regulation.

Derivatives of the TyG index

Researchers have developed several derivatives combining the TyG index with anthropometric measures to enhance predictive ability:

- **TyG-BMI = TyG × body mass index (BMI)**. Chinese and Iranian cross-sectional studies found that participants in the highest TyG-BMI quartile had odds ratios (ORs) of 2.52 and 3.10, respectively, for prevalent hypertension compared with the lowest quartile [5].
- **TyG-WC = TyG × waist circumference**. In a Chinese cross-sectional study of H-type hypertension (hypertension with hyperhomocysteinaemia), TyG-WC had the largest area under the ROC curve (AUC 0.676) versus TyG alone (0.646) and TyG-BMI (0.658); high TyG and high WC synergistically increased risk of H-type hypertension [6].
- **TyG-WHtR**. A recent US cross-sectional analysis (NHANES 2009–2018) reported that each unit increase in TyG-WHtR raised hypertension risk by 12 % after adjustment, with an AUC of 0.6946.

These derivatives incorporate adiposity, recognising that the combination of dyslipidaemia, hyperglycaemia, and obesity exacerbates IR and blood pressure elevation.

Pathophysiological Link Between Insulin Resistance and Hypertension

Multiple interrelated mechanisms link IR to elevated blood pressure. Understanding these pathways provides biological plausibility for using the TyG index as a marker of hypertensive risk.

1. **Sympathetic nervous system activation**. IR stimulates the central nervous system and increases sympathetic output. Elevated plasma insulin enhances norepinephrine release, leading to vasoconstriction and increased heart rate. Chronic sympathetic stimulation elevates

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peripheral resistance and contributes to hypertension.

2. **Renin–angiotensin–aldosterone system (RAAS) dysregulation**. Hyperinsulinaemia increases angiotensinogen synthesis, activates angiotensin II type-1 receptors, and promotes aldosterone secretion. These effects raise sodium and water retention, augmenting blood volume and vascular tone. In a meta-analysis of 22 studies, Ren et al. noted that elevated TyG index is associated with endothelial dysfunction and RAAS activation [7].

3. **Endothelial dysfunction**. IR impairs nitric oxide (NO) production and increases oxidative stress, reducing vasodilatory capacity. Vasoconstrictor substances (endothelin-1, angiotensin II) become dominant, leading to arterial stiffening. This impaired endothelial function precedes overt hypertension and can be detected by the TyG index.

4. **Sodium retention**. Hyperinsulinaemia directly increases sodium reabsorption in the renal tubules, while RAAS activation further enhances retention. Increased extracellular fluid volume elevates cardiac output and blood pressure.

5. **Pro-inflammatory and pro-thrombotic state**. IR is associated with chronic low-grade inflammation, increased C-reactive protein, and elevated fibrinogen. These factors promote vascular damage and hypertension. A cross-lagged analysis of 57 192 participants showed that higher TyG predicts subsequent increases in blood pressure more strongly than vice versa, supporting a causal pathway from IR to hypertension [8].

Collectively, these mechanisms emphasise why a composite index capturing hyperglycaemia and hypertriglyceridaemia (the TyG index) may signal early metabolic disturbances that predispose to hypertension.

Evidence on TyG Index in Hypertensive Populations

Cross-sectional studies

Large population-based cross-sectional studies consistently demonstrate that individuals with higher TyG index values have greater prevalence and severity of hypertension. Importantly, many of these studies adjust for traditional risk factors (age, sex, BMI, smoking, alcohol use, and biochemical parameters), indicating an independent relationship between TyG and blood pressure.

- **East Asian populations**. In 2025, a nationwide Mongolian screening of **120 264 adults** found that TyG index increased progressively across blood pressure stages: participants with elevated blood pressure or hypertension had higher TyG levels than normotensive individuals. Central obesity amplified the association. Another Chinese cross-sectional study of **4177 adults** reported that the highest TyG quartile had an OR of 1.273 for hypertension compared with the lowest quartile, with stronger associations in isolated systolic or systolic-diastolic hypertension .
- **South Asian populations**. In a cross-sectional study of 200 Indian adults (100 hypertensive and 100 normotensive), hypertensive patients had significantly higher TyG index than controls. TyG positively correlated with systolic and diastolic blood pressure, total cholesterol and LDL cholesterol, highlighting its applicability in South Asian populations where insulin resistance is prevalent [8].
- **US NHANES data**. An analysis of 10 937 non-diabetic participants from NHANES 2017–2020 reported adjusted ORs for hypertension of 1.23 for TyG values 8.53–8.95 and 1.89 for TyG > 8.96 compared with < 8.11. Another NHANES study

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focusing on cancer patients observed that higher TyG quartiles were associated with a greater prevalence of hypertension (OR ~1.51) [9].

- **European and Japanese cohorts**. In a study of **normoglycaemic Japanese adults (n = 15 450)**, the highest TyG quartile was associated with ORs of 1.48 for prehypertension and 1.76 for hypertension, even after controlling for age, obesity, smoking and alcohol use. A European study of perimenopausal women found that TyG was the variable most strongly associated with hypertension (OR ≈ 22.1), and an optimal cut-off of 8.7 offered good sensitivity and specificity (AUC ≈ 0.79) [9].
- **H-type hypertension**. H-type hypertension (hypertension coexisting with hyperhomocysteinaemia) confers a higher cardiovascular risk. In 4512 Chinese adults, TyG-WC had the highest predictive value for general and H-type hypertension; participants with both high TyG and high waist circumference had significantly increased risk (OR ≈ 1.659) [10].
- **Reaction Study**. In the Chinese REACTION cohort (~47 808 participants), TyG index remained significantly associated with hypertension after adjusting for glycaemic and lipid parameters, with an overall OR of 1.33; the association was stronger in older (OR 1.67) and obese (OR 1.85) subgroups [4].

TyG-BMI and other derivatives in cross-sectional studies:

The TyG index alone may not fully capture the influence of adiposity. Combining TyG with BMI amplifies predictive power:

- In a cross-sectional study of **60 283 Chinese adults**, TyG-BMI showed a per-SD OR of 1.61 and a highest-vs-lowest quartile OR of 2.52 for hypertension [4]. Restricted cubic spline analysis demonstrated a linear relationship.
- Among 8610 Iranian adults, the highest TyG-BMI quartile had a 3.10-fold higher odds of hypertension compared with the lowest quartile [11].
- In NHANES 2017–2020 data, TyG-WHtR exhibited better predictive performance (AUC 0.6946) than TyG or TyG-BMI alone; each unit increase in TyG-WHtR increased hypertension risk by 12 %[4].

These findings support the inclusion of adiposity indices when using the TyG index for hypertension risk assessment.

Longitudinal cohort studies

Prospective studies provide stronger evidence by evaluating incident hypertension over time and establishing temporal relationships between metabolic risk and blood pressure elevation.

The China Health and Nutrition Survey (CHNS), a prospective cohort of approximately 4,600 Chinese adults free of hypertension at baseline and followed for six years, demonstrated a graded increase in incident hypertension across rising TyG quartiles (18.1%, 25.3%, 28.5%, and 33.4%). Compared with the lowest quartile, adjusted hazard ratios (HRs) were 1.29, 1.24, and 1.50 for quartiles 2, 3, and 4, respectively, while each unit increase in TyG was associated with a 17% higher risk of developing hypertension [11].

Evidence from the Kailuan cohort (n ≈ 46,500) further supports the cumulative metabolic burden hypothesis. Over a median follow-up of 9.6 years, higher cumulative TyG exposure was independently associated with incident hypertension, with HRs ranging from 1.06 to 1.21 across quartiles. Each one standard deviation increase in cumulative TyG conferred an 8% higher risk, and prolonged exposure to elevated TyG (≥6 years) increased obesity-related hypertension risk

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by up to 2.86-fold compared with sustained low exposure [12].

In a rural Chinese cohort of 9,343 adults followed for a median of 35 months, trajectory analysis identified distinct TyG patterns over time. Participants with moderate-stable and high-stable TyG trajectories had significantly higher risks of hypertension (HRs 1.41 and 1.82, respectively) compared with the low-stable trajectory group. Notably, elevated risk persisted even among individuals with otherwise favourable lifestyle scores when TyG remained chronically high [13].

Findings from the China Health and Retirement Longitudinal Study (CHARLS) further highlighted the interaction between adiposity and metabolic dysfunction. Among 2,561 participants, persistently medium and high TyG-BMI trajectory classes were associated with HRs of 1.50 and 2.35 for incident hypertension compared with the persistently low class. Similarly, cumulative TyG-BMI quartiles 3 and 4 carried HRs of 1.75 and 2.15, respectively, reinforcing the additive effect of insulin resistance and obesity [14].

Dynamic changes in TyG-BMI were also shown to be clinically relevant. In a nationwide Chinese cohort, participants transitioning from low to high TyG-BMI over time exhibited substantially higher odds of developing hypertension. Compared with the lowest dynamic class, moderate, higher, and highest classes had odds ratios (ORs) of 1.60, 1.93, and 2.33, respectively, while cumulative TyG-BMI quartiles 2–4 showed progressively increasing risk. Overweight and obese individuals with elevated TyG had ORs of approximately 1.87–1.91 relative to those with low TyG and normal weight [15].

Similar observations have been reported outside China. In a Japanese longitudinal study of normoglycaemic adults, elevated TyG index values predicted the development of prehypertension and hypertension during follow-up, with adjusted ORs of approximately 1.31 and 1.76 across quartiles. These associations remained robust after multivariable adjustment, supporting the TyG index as an early risk marker even before overt dysglycaemia develops [16].

Eta-Analyses

Several meta-analyses have synthesised evidence from both cohort and cross-sectional studies. A 2023 meta-analysis including 22 studies (12 cross-sectional and 10 cohort studies; 668,486 participants) reported a pooled OR/HR of 1.36 for the highest versus lowest TyG category. Dose–response analysis demonstrated a linear association, with each one-unit increase in TyG raising hypertension risk by approximately 1.5-fold [17].

More recently, a **2025 meta-analysis** combining four cohort studies and six cross-sectional studies reported a pooled HR of 1.57 (95% CI 1.26–1.87) for cohort data and a pooled OR of 2.01 for cross-sectional analyses. Subgroup analyses showed no significant heterogeneity by sex or study design, underscoring the robustness and generalisability of the TyG–hypertension relationship across populations [18].

TyG index and co-existing dysglycaemia: with and without type 2 diabetes

Normoglycaemic and prediabetic individuals

Among normoglycaemic perimenopausal women, the TyG index emerged as the strongest metabolic correlate of hypertension, with an optimal cut-off of 8.7 providing good discriminatory performance [19]. Similarly, studies in Japanese adults with normal fasting glucose demonstrated that higher TyG quartiles independently predicted new-onset prehypertension and hypertension, with ORs of 1.31 and 1.76, respectively, highlighting the value of TyG as an early marker before glycaemic thresholds are exceeded [16].

Individuals with type 2 diabetes

In NHANES-based analyses, the association between TyG index and hypertension persisted after adjustment for diabetes status. However, individuals with type 2 diabetes and elevated TyG indices exhibited particularly high odds of hypertension and cardiovascular mortality,

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suggesting an additive or synergistic effect of diabetes and insulin resistance on blood pressure regulation [20]. Additional evidence from oncology populations indicates that the highest TyG quartile is associated with significantly higher hypertension prevalence even after controlling for diabetes and other confounders, with spline analyses demonstrating a linear risk relationship without clear threshold effects [19]. Multicentre studies in hypertensive cohorts further show that individuals with concomitantly high TyG and elevated HbA1c experience poorer blood pressure control and a markedly higher risk of resistant hypertension, with TyG alone conferring an HR of approximately 5.47 in some analyses [12].

Prediabetes and H type hypertension

• The H type hypertension study noted above demonstrated that TyG index and its derivatives were significantly associated with both hypertension and elevated homocysteine levels [21]. Elevated homocysteine is more common in individuals with metabolic disorders and may augment the IR–hypertension link.

• A Korean study (not fully extracted here) suggested that TyG BMI and TyG WC were significantly associated with prehypertension in lean individuals, indicating the value of these indices even in non obese, prediabetic patients [22].

Collectively, these data show that the TyG index is relevant across the entire spectrum of glycaemic status—from normoglycaemia to prediabetes to overt diabetes—and may help identify hypertensive individuals at greatest risk of complications.

Prognostic and Clinical Implications of the TyG Index

Beyond predicting hypertension incidence, the TyG index has prognostic value for cardiovascular outcomes, mortality, and treatment resistance among patients with established hypertension.

1. **Risk of cardiovascular events and mortality.** A US cohort of hypertensive ICU patients found that higher TyG indices were associated with increased all cause mortality at 30, 60, and 90 days; restricted cubic spline analysis revealed a linear upward trend in mortality risk with higher TyG [23]. Similarly, a study of hypertensive adults reported that combining TyG with HbA1c improved prediction of poor blood pressure control and cardiovascular events [24].
2. **Resistant hypertension.** In a cohort of 1635 newly diagnosed hypertensive patients followed for 31 months, the TyG index emerged as the most powerful predictor of resistant hypertension; the HR for TyG was 5.472 (95 % CI 4.028–7.433), exceeding that of age, BMI or baseline blood pressure [25]. A nomogram including TyG improved discrimination for resistant hypertension.
3. **Target organ damage.** A retrospective cohort analysis using NHANES data showed that higher baseline TyG BMI was independently associated with target organ damage in essential hypertension. Each 10 unit increase in TyG BMI raised hypertension risk by 4.3 %, and high TyG BMI improved prediction of stroke and renal impairment [26].
4. **Blood pressure control in treated patients.** A 2024 study of 99 336 hypertensive adults found that both TyG and HbA1c were independently associated with poor blood pressure control; their combination had a synergistic effect, particularly in elderly men [27]. This suggests that IR should be targeted in hypertension management.
5. **Short term outcomes in critical care.** The 2025 ICU study mentioned above emphasises that high TyG index identifies hypertensive patients at increased risk of short term mortality [28]. Monitoring TyG

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may aid risk stratification and inform intensive care management.

6. **Coronary heart disease (CHD) risk.** In a 10 year cohort of adults with disabilities, TyG and blood pressure were independently associated with CHD; mediation analysis indicated that TyG explained about 20 % of the association between blood pressure and CHD [29]. Individuals with both hypertension and high TyG had the highest CHD risk (HR ≈ 1.92) [29].

These prognostic studies highlight that the TyG index is not only a marker of hypertension risk but also a predictor of adverse outcomes, underscoring its potential clinical utility.

Limitations, Knowledge Gaps, and Future Research Directions

Despite consistent associations across populations, several limitations warrant consideration:

1. **Heterogeneity of cut offs.** Studies use varying TyG cut offs (e.g., 8.7 in perimenopausal women [22] versus >8.96 in NHANES [26]). Lack of standardisation impedes clinical application. Future research should establish population specific reference ranges, considering sex, age and ethnicity.
2. **Observational design.** Most evidence is cross sectional or cohort based; causality cannot be inferred definitively. Although cross lagged analyses suggest a stronger effect of TyG on subsequent blood pressure than vice versa [29], randomised trials evaluating interventions that lower the TyG index (e.g., dietary modification, pharmacological agents targeting triglycerides or glucose) are needed to determine whether reducing TyG mitigates hypertension risk.
3. **Confounding factors.** Many studies adjust for traditional risk factors, but residual confounding (e.g., diet, physical activity, genetics) may persist. Prospective studies with detailed lifestyle data are required.
4. **Variability in measurement.** TyG is derived from fasting triglycerides and glucose; variations in fasting duration or laboratory methods may affect accuracy. Standardised protocols for fasting and measurement are essential.
5. **Population diversity.** Most cohorts are from East Asia. Data from African, Latin American and Middle Eastern populations are limited. More globally representative research will enhance generalisability.
6. **Integration with other biomarkers.** The additive value of TyG compared with other IR markers (HOMA IR, adiponectin, leptin) and inflammatory markers needs clarification. Studies exploring composite risk scores incorporating TyG and these biomarkers may improve prediction.
7. **Role in children and adolescents.** Evidence in paediatric populations is scant. Given rising childhood obesity and early onset of hypertension, investigating TyG index in younger cohorts is critical.
8. **Impact of medications.** Many participants are on antihypertensive or lipid lowering therapy. How these medications influence the TyG–hypertension relationship is unclear. Future studies should stratify analyses by medication use.

Conclusion

The triglyceride–glucose (TyG) index is a simple, cost-effective marker of insulin resistance that has emerged as a robust predictor of hypertension across multiple populations and study designs. Cross-sectional and longitudinal evidence from

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East Asian, South Asian, American, European and Middle Eastern cohorts demonstrates that higher TyG values and derivatives (TyG-BMI, TyG-WC, TyG-WHtR) are associated with prevalent and incident hypertension, independent of traditional risk factors. The TyG index shows dose-response relationships and performs favourably compared with other metabolic indices. Importantly, elevated TyG predicts prehypertension and hypertension in normoglycaemic individuals, highlights increased risk in people with diabetes, and forecasts adverse outcomes such as resistant hypertension, target organ damage, and mortality.

Mechanistically, the TyG index captures the interplay of hyperglycaemia, hypertriglyceridaemia and adiposity, reflecting insulin resistance–driven sympathetic activation, RAAS stimulation, endothelial dysfunction, sodium retention and inflammation. These pathophysiological links explain its strong association with blood pressure and cardiovascular risk. However, heterogeneity in cut-offs, observational design, residual confounding and limited global representation highlight the need for standardised measurement, prospective intervention trials, and broader population studies. Given its simplicity, low cost and prognostic value, integrating the TyG index into routine clinical evaluation could enhance hypertension screening, risk stratification and management, especially in resource-limited settings. Clinicians should consider assessing the TyG index alongside traditional risk factors, while researchers should continue to refine its application and investigate interventions targeting this index to curb the global hypertension epidemic.

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