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Lipid–Glucose Metabolic Index (TyG) as an Early Indicator of Vascular Aging: Evidence of Body Mass Index Modification in a Chinese Community Cohort

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Article

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Abstract

Background and Aims: Cardiovascular disease remains a leading global health concern, with atherosclerosis being a major contributor to mortality. This study examines the association between the triglyceride–glucose (TyG) index—a novel surrogate marker of insulin resistance—and atherosclerosis in Chinese adults, and evaluates the modifying effect of body mass index (BMI) on this relationship.

Methods and Results: A total of 4729 healthy Chinese adults were included from a community-based health survey. The TyG index was calculated as ln[fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2]. Arterial stiffness was assessed via carotid-femoral (cfPWV), carotid-radial (crPWV), and carotid-dorsalis pedis (cdPWV) pulse wave velocities. After adjustment for conventional risk factors, the TyG index showed a significant association with peripheral arterial stiffness (cdPWV >10.57 cm/s or crPWV >11.12 cm/s), but not with cfPWV. A significant interaction between TyG index and BMI was observed for both crPWV and cdPWV. The risk of elevated crPWV and cdPWV increased across TyG index quartiles at varying BMI levels (P< 0.001). A dose-response relationship between TyG index and crPWV/cdPWV was evident in participants with normal weight and overweight, particularly among normal-weight adults, but was absent in participants with obesity.

Conclusion: The TyG index independently predicts peripheral arterial stiffness, particularly in normal-weight adults. These findings highlight the lipid–glucose metabolic imbalance as a driver of early vascular aging and suggest that TyG assessment may refine cardiometabolic risk stratification beyond BMI.

What is known about this research topic?

• The triglyceride–glucose (TyG) index is a well-validated proxy for insulin resistance and is consistently linked to cardiovascular events.

• Its interplay with body-mass index (BMI) in determining subclinical arterial stiffness—particularly in non-overweight individuals—remains insufficiently explored in large, healthy cohorts.

What this study adds and its future implications

• Among 4,729 Chinese adults, higher TyG index robustly predicted elevated peripheral pulse-wave velocity; this relationship was strongest in those with normal BMI and blunted in overweight or obese participants.

• A clear dose–response pattern established TyG as an early, BMI-independent marker of peripheral arterial stiffening.

These findings reposition risk stratification: normal-weight adults with elevated TyG emerge as a prime prevention cohort, warranting routine TyG assessment in population screening irrespective of BMI.

Introduction

Cardiovascular diseases (CVDs) continue to affect over half a billion individuals globally, accounting for 20.5 million deaths in 2021—nearly one-third of all global mortality and representing an increase from the estimated 121 million CVD deaths [1]. Atherosclerosis (AS), the leading cause of CVD morbidity and mortality, is primarily driven by abnormal blood lipid and glucose levels [2–3]. These abnormalities contribute to increased fibrotic collagen deposition, endothelial dysfunction, inflammation, and vascular smooth muscle hyperplasia, ultimately diminishing arterial elasticity and increasing stiffness [4–5]. Although clinically prevalent among middle-aged and older adults, AS begins insidiously early in life. Early detection and intervention in high-risk groups can decelerate its progression and postpone CVD onset.

Pulse wave velocity (PWV), a central marker of AS, is a widely utilised non-invasive method for assessing arterial stiffness and predicting cardiovascular events [6–7]. PWV measurements at various anatomical sites reflect stiffness in different arterial regions: carotid–femoral PWV (cfPWV) represents central arteries, while carotid–radial PWV (crPWV) and carotid–dorsalis pedis PWV (cdPWV) assess peripheral arteries. Insulin resistance (IR), closely associated with AS, is effectively evaluated using the triglyceride-glucose (TyG) index, which integrates fasting triglyceride and glucose concentrations [8–9].

With lifestyle changes, global rates of overweight and obesity have increased markedly [10–12]. Obesity is strongly linked to metabolic disorders, including type 2 diabetes, hypertension, dyslipidaemia, AS, and CVDs [13–14]. Body mass index (BMI) remains a common clinical tool for assessing obesity and stratifying risk [15–16]. Goossens et al. reported a positive association between BMI ≥18.5 kg/m² and cardiovascular and metabolic diseases [17]. However, emerging evidence suggests that the TyG index better identifies metabolic risk in individuals with normal BMI [18–20]. Notably, individuals without obesity may remain susceptible to AS in ways not captured by traditional obesity metrics. We hypothesise that the TyG index could provide additional insight into this phenomenon. Furthermore, the interactive effects of BMI and the TyG index on AS remains unclear.

This study aimed to investigate the relationship between the TyG index and arterial stiffness parameters (cfPWV, crPWV, cdPWV) in healthy Chinese adults, and to assess the modifying effect of BMI on this association. These findings are essential for identifying at-risk populations and mitigating the progression of AS.

Methods

1. Participants and study design

This cross-sectional study formed part of the Cardiometabolic Risk in Chinese (CRC) Study. Participants aged 19–90 years were randomly selected from the urban population in central China through a community health check-up survey. Collected data included PWVs, blood pressure (BP), BMI, neck circumference (NC), waist circumference (WC), age, and serum uric acid levels (SUA). Exclusion criteria comprised histories of vascular disease, coronary heart disease, diabetes, chronic inflammation, hyperlipidaemia, and acute or chronic hepatic or renal failure. Patients with anaemia, malignancies, pregnancy, or immune disorders were also excluded. A total of 4729 adults were ultimately enrolled. The study protocol received approval from the Ethics Committee of the Southeast University Affiliated Xuzhou Central Hospital, China. Written informed consent was obtained from all participants.

2. Assessment of PWVs

CfPWV, cdPWV, and crPWV were measured at rest using a device (Artech-Medical, Pantin, France) equipped with two sensors. For cfPWV, pulse waves at the carotid and femoral sites were analysed to calculate the relative ECG delay and determine PWV. Similar techniques were used for cdPWV and crPWV. Simultaneous recordings were obtained from the right carotid, radial, and dorsalis pedis arteries. From 16 ECG-gated waveforms, the three highest and three lowest values were excluded; the remaining 10 were averaged. PWV was calculated as the path length divided by transmission time (distance/time ratio, expressed in meters per second) [21]. Participants were classified into four groups based on PWV quartiles, with the highest quartile indicating elevated arterial stiffness risk [22–23].

3. Biomarker assessment

After a 10-hour overnight fast, fasting elbow venous blood samples were collected in the morning. Measurements included fasting plasma glucose (FPG), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), and SUA levels. Participants subsequently underwent a 75 g oral glucose tolerance test (OGTT), with blood samples drawn at 120 minutes post-glucose ingestion. Haemoglobin A1c

(HbA1c) was measured using high-performance liquid chromatography. The TyG index was calculated using the following formula: ln [fasting TG (mg/dL) × fasting glucose (mg/dL) / 2] [24].

4. Anthropometric measurements

Body weight was measured to the nearest 0.1 kg with participants wearing light indoor clothing and no footwear. Height was recorded to the nearest 0.5 cm using a wall-mounted stadiometer. BMI was calculated as weight (kg) divided by height squared (m^2). Based on Chinese guidelines [25], BMI classifications were: normal weight (<24.0 kg/m^2), overweight (24.0–28.0 kg/m^2), and obese (≥28.0 kg/m^2). NC was measured horizontally at the upper margin of the larynx using a flexible tape [26]. WC was measured midway between the lower rib margin and iliac crest. BP was assessed using a mercury sphygmomanometer by trained physicians after a minimum rest of 5 minutes. Three measurements were recorded at 60-second intervals, and the mean was used for analysis.

5. Statistical analysis

Data were analysed using SPSS version 22.0 (SPSS Inc., Chicago, USA) and Stata version 14.0 (StataCorp, College Station, TX, USA). Categorical variables are presented as frequencies (percentages), and continuous variables as means ± standard deviations (SD). Analysis of variance (ANOVA) was employed to compare metabolic markers across TyG index quartiles, while chi-square tests were used for categorical variable comparisons. Multivariate logistic regression assessed the independent association between TyG index and elevated PWVs, adjusting for covariates including age, sex, BMI, BP, LDL-C, HbA1c, SUA, and heart rate (HR). Generalised linear models were applied to explore dose–response relationships between TyG index and elevated crPWV and cdPWV at varying BMI levels using Stata version 14.0, with interaction terms used to evaluate TyG index × BMI effects. A p-value < 0.05 was considered statistically significant.

Results

1. Characteristics of the study participants based on TyG index levels

Table 1 displays the baseline clinical and biochemical characteristics of the 4729 participants, stratified by quartiles of the TyG index. Significant positive correlations were observed between the TyG index and crPWV, cfPWV, cdPWV, BMI, NC, WC, age, SBP, DBP, 2-hour OGTT glucose, HbA1c, TC, LDL-C, SUA, and HR (all $P < 0.05$). A significant negative correlation was found with HDL-C ($P < 0.05$).

2. Association between TyG index and arterial stiffness markers

Participants were classified into two groups based on PWV thresholds: cdPWV > 10.57 cm/s, cfPWV > 11.51 cm/s, and crPWV > 11.12 cm/s were defined as elevated. Table 2 summarises the associations between TyG index quartiles and high PWV. After adjustment for sex, age, and BMI, the TyG index remained significantly associated with elevated cdPWV, cfPWV, and crPWV (all $P < 0.001$). Following further adjustment for SBP, DBP, LDL-C, HbA1c, SUA, and HR (Model 2), the association with cfPWV was attenuated ($P = 0.112$), while associations with cdPWV and crPWV remained robust ($P < 0.001$), indicating a stronger link with peripheral arterial stiffness.

3. Relationship between TyG index and crPWV and cdPWV under different BMI levels

The interaction between TyG index and BMI in relation to crPWV and cdPWV was further assessed (Table 3). Participants were categorised into three BMI groups, revealing significant interactions for both PWV parameters ($P < 0.001$). The associations were strongest in individuals with BMI < 24.0 kg/m^2 ($P < 0.001$). Across all BMI categories, the risk of elevated crPWV and cdPWV increased progressively with higher TyG index quartiles ($P < 0.001$).In the normal BMI group, using the lowest TyG quartile as reference, the risk of elevated crPWV and cdPWV increased markedly with each

successive quartile (odds ratios: 1.950 and 2.951, respectively). Notably, the obese group exhibited significantly lower risks compared to the normal and overweight groups.

Dose-response analysis of TyG index and elevated crPWV and cdPWV at different BMI levels is illustrated in Figures_1 and Figures_2. After adjustment for age, sex, SBP, DBP, FPG, and TC, a clear dose–response relationship was observed in the normal and overweight groups, most prominently among individuals with normal BMI. However, no significant dose-response association was found in the obese group, where increased TyG index did not correspond to higher risk of crPWV or cdPWV.

Discussion

This study demonstrated a positive association between the TyG index and peripheral arterial stiffness, as indicated by cdPWV > 10.57 cm/s or crPWV > 11.12 cm/s. However, the TyG index was not associated with cfPWV, a measure of central arterial stiffness. Furthermore, a significant interaction was identified between the TyG index and BMI in relation to both crPWV and cdPWV.

Previous research has linked the TyG index to CVDs [27–29], with evidence suggesting that its detrimental effects may emerge in the early stages of AS [30–31]. A recent cross-sectional study involving Korean participants reported an independent association between the TyG index and brachial-ankle PWV (baPWV) [32]. Nevertheless, as baPWV is influenced by both central and peripheral factors [33], it remains uncertain whether the TyG index preferentially affects central or peripheral atherosclerosis. Our findings support the conclusion that an elevated TyG index is more strongly related to peripheral arteriosclerosis, with a comparatively weaker impact on central arteriosclerosis Poon et al. [34] conducted a cross-sectional and cohort follow-up study of 2,571 middle-aged and older American adults, identifying an association between the TyG index and cfPWV in cross-sectional analysis. However, during follow-up, high aortic stiffness was not associated with HOMA-IR or the annualised rate of log-TyG index increase. Differences in study population, ethnicity, and sample size may explain the discrepancies between their findings and ours. Nonetheless, our larger sample and their longitudinal observations collectively reinforce our conclusions. Further research is warranted to clarify the role of the TyG index in aortic wall remodelling.

Several mechanisms may underlie the association between an elevated TyG index and AS. IR is a potential factor that impairs insulin-mediated glucose regulation and lipid oxidation, resulting in functional insulin deficiency. This promotes hyperglycaemia and hyperlipidaemia [35]. Elevated free fatty acids infiltrate non-adipose tissues, deposit triglycerides, and exacerbate metabolic disturbances, ultimately contributing to AS [36–37]. As a surrogate marker for IR [38–40], the TyG index is closely related to PWV [8], suggesting that IR may mediate arterial stiffness through the TyG index. Although AS has long been attributed to lipid accumulation [36–37], emerging evidence indicates a multifactorial, inflammatory aetiology [41–42]. Risk factors including hyperglycaemia, dyslipidaemia, and hypertension foster a hypoxic, oxidative stress environment [43], promoting the generation of reactive oxygen species and activation of inflammatory pathways. These processes accelerate the progression of AS [44]. Thus, the TyG index may influence AS through mechanisms involving both metabolic and inflammatory dysfunction.

The relationship between the TyG index and AS appears to vary based on BMI. Although BMI is recognised as an independent risk factor for AS [45], this study identified a significant interaction between the TyG index and BMI. As BMI increases, the contribution of the TyG index to crPWV and cdPWV is diminished. Among individuals with normal weight, the adverse association between the TyG index and arterial stiffness is more pronounced than among those with obesity, suggesting that the TyG index may serve as a more sensitive predictor of AS risk in normal-weight populations. Subgroup analyses revealed a clear dose-response relationship between the TyG index and both crPWV and cdPWV in normal-weight and overweight groups, particularly among the former.

The superior predictive value of the TyG index in lean individuals may be attributed to its stronger reflection of hepatic IR, in contrast to HOMA-IR, which predominantly reflects muscular IR [46]. TyG index may differ functionally from HOMA-IR among non-diabetic older adults [44]. Lamb et al. [30], in a cross-sectional study of 473 postmenopausal Greek women and, found that the TyG index was a better predictor of subclinical AS than metabolic syndrome in lean postmenopausal women. Specifically, it correlated significantly with carotid intima–media thickness, atherosclerotic plaque formation, and aortic stiffness assessed by PWV—findings consistent with our results.

The principal strength of this study lies in identifying the interaction between BMI and the TyG index in the context of AS. As an alternative IR marker, the TyG index may aid in detecting high-risk individuals, particularly those with normal weight who may otherwise be misclassified as low-risk. Nonetheless, several limitations must be acknowledged. First, the cross-sectional design precludes causal inference between the TyG index and AS. Second, the study population comprised Chinese adults, limiting generalisability to other ethnic groups. Third, the sample size was relatively modest. Additionally, reliance on data from community health check surveys may restrict broader applicability. Future longitudinal studies with larger cohorts are needed to elucidate the causal relationship between the TyG index and AS.

Conclusion

In healthy Chinese adults, the triglyceride-glucose (TyG) index is significantly associated with arterial stiffness (AS) risk and independently predicts peripheral AS, particularly in normal-weight individuals—higher BMI attenuates the TyG index's impact on AS, making it a more reliable indicator in this subgroup. These findings identify lipid-glucose metabolic imbalance as a driver of early vascular aging and suggest that TyG index assessment can refine cardiometabolic risk stratification beyond BMI, thereby enhancing cardiovascular disease prevention when targeted alongside conventional high-risk groups.

Abbreviations

AS: atherosclerosis; ANOVA: analysis of variance; BMI: body mass index; BP: blood pressure; cdPWV: carotid–dorsalis pedis pulse wave velocity; cfPWV: carotid–femoral pulse wave velocity; CRC: cardiometabolic risk in Chinese; crPWV: carotid–radial pulse wave velocity; CVD: cardiovascular disease; DBP: diastolic blood pressure; ECG: electrocardiogram; FPG: fasting plasma glucose; HbA1c: haemoglobin A1c; HDL-C: high-density lipoprotein cholesterol; HOMA-IR: homeostasis model assessment of insulin resistance; HR: heart rate; IR: insulin resistance; LDL-C: low-density lipoprotein cholesterol; NC: neck circumference; OGTT: oral glucose tolerance test; PWV: pulse wave velocity; ROS: reactive oxygen species; SBP: systolic blood pressure; SD: standard deviation; SUA: serum uric acid; TC: total cholesterol; TG: triglyceride; TyG: triglyceride–glucose

Declarations

Authors' contributions

Xiu Zang and Yun Wang drafted this manuscript. Jia Zhang and **Yanhong Lin** collated and organized the data. Xuekui Liu analysed the data. Houfa Geng and Jun Liang are responsible for the integrity of the work as a whole. All authors have read and approved the final manuscript.

Availability of data and materials

Not applicable.

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Ethics approval and consent to participate

This study involves human participants. The study protocol was reviewed and approved by the Ethics Committee of Xuzhou Central Hospital, Southeast University. All research was conducted strictly in accordance with relevant ethical guidelines and regulatory requirements. Written informed consent was obtained from all eligible participants; for participants unable to provide independent consent (e.g., minors or individuals with impaired decision-making capacity), written informed consent was obtained from their legal guardians.

Data Availability Statement

The data of this study are not publicly available at this time due to the ongoing related follow-up research, but can be obtained from the corresponding author [Dr. Jun Liang, MD, PhD] upon reasonable request for scientific research purposes (email address: liangjun@xzhmu.edu.cn). Use of the data must comply with relevant ethical review regulations.

Consent for publication

All authors approved the current version of the manuscript.

Conflict of interest

The authors declare that they have no conflicts of interest.

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References

1. Xue, Xia,Xue, Tian,Qin, Xu et al. Global trends and regional differences in mortality of cardiovascular disease and its impact on longevity, 1980-2021: Age-period-cohort analyses and life expectancy decomposition based on the Global Burden of Disease study 2021.[J] .Ageing Res Rev, 2024, 103: 0.
2. Pauline C E, Schuitema,Frank L J, Visseren,Børge G, Nordestgaard et al. Elevated triglycerides are related to higher residual cardiovascular disease and mortality risk independent of lipid targets and intensity of lipid-lowering therapy in patients with established cardiovascular disease.[J] .Atherosclerosis, 2025, undefined: 0.
3. Sidhi, Laksono,Hillary, Kusharsamita,Unravelling the role of carotid atherosclerosis in predicting cardiovascular disease risk: A review.[J] .ARYA Atheroscler, 2024, 20: 0.
4. Aukie, Hooglugt,Olivia, Klatt,Stephan, Huveneers,Vascular stiffening and endothelial dysfunction in atherosclerosis.[J] .Curr Opin Lipidol, 2022, 33: 0.
5. Davi Vieira Teixeira, da Silva,Diego Dos Santos, Baião,Cristine Couto, Almeida et al. A Critical Review on Vasoactive Nutrients for the Management of Endothelial Dysfunction and Arterial Stiffness in Individuals under Cardiovascular Risk.[J] .Nutrients, 2023, 15: 0.
6. Kim HL. Pulse Wave Velocity in Atherosclerosis. Front Cardiovasc Med 2019; 6: 41.
7. Jin, Zhi,Zhao, Li,Study on the correlation between the level of insulin resistance and changes in the degree of cerebral atherosclerosis in non-diabetic patients.[J] .Technol Health Care, 2024, 32: 0.

8. Wu Jingjing, Li Fei, Wang Jie, et al. Clinical research progress on pulse wave conduction velocity evaluation of vascular aging[J].Journal of Central South University(Health Sciences), 2024, 49(12).
9. Su WY, Chen SC, Huang YT, et al. Comparison of the Effects of Fasting Glucose, Hemoglobin A, and Triglyceride-Glucose Index on Cardiovascular Events in Type 2 Diabetes Mellitus. Nutrients 2019; 11:.
10. Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults.[J] .Lancet, 2024, 403: 0.
11. Kim CS, Ko SH, Kwon HS, et al. Prevalence, awareness, and management of obesity in Korea: data from the Korea national health and nutrition examination survey (1998-2011). Diabetes Metab J 2014; 38: 35 - 43.
12. Barazzoni R, Bischoff S, Boirie Y, et al. Sarcopenic Obesity: Time to Meet the Challenge. Obes Facts 2018; 11: 294 - 305.
13. Yingxin, Liu,Ai, Zhao,Guiju, Sun et al. Relationship between metabolic status, physical activity and cardiovascular disease in participants with obesity.[J] .Int J Obes (Lond), 2024, 48: 0.
14. Neeland IJ, Poirier P. Cardiovascular and Metabolic Heterogeneity of Obesity: Clinical Challenges and Implications for Management. Circulation 2018; 137: 1391 - 1406.
15. Ji-Hee, Haam,Bom Taeck, Kim,Eun Mi, Kim et al. Diagnosis of Obesity: 2022 Update of Clinical Practice Guidelines for Obesity by the Korean Society for the Study of Obesity.[J] .J Obes Metab Syndr, 2023, undefined: 0.
16. Kommuri NV, Zalawadiya SK, Veeranna V, et al. Association between various anthropometric measures of obesity and markers of subclinical atherosclerosis. Expert Rev Cardiovasc Ther 2016; 14: 127 - 135.
17. The Metabolic Phenotype in Obesity: Fat Mass, Body Fat Distribution, and Adipose Tissue Function. Obes Facts 2017; 10: 207 - 215.
18. Thurka, Paramanathan,Balakumar, Sandrasegarampillai,Vasanthy, Arasaratnam et al. The discriminative ability of the triglyceride-glucose index to identify metabolic syndrome among adults of the northern Sri Lankan population. [J] .BMC Endocr Disord, 2024, 24: 0.
19. Jong Seo, Yoon,Young Suk, Shim,Hae Sang, Lee et al. A population-based study of TyG index distribution and its relationship to cardiometabolic risk factors in children and adolescents.[J] .Sci Rep, 2021, 11: 0.
20. Man, Wu,Chaoyang, Li,Yiqing, Yu et al. Association between the triglyceride-glucose (TyG) index and stroke risk in Chinese normal-weight adults: a population-based study.[J] .Diabetol Metab Syndr, 2024, 16: 0.
21. Liang J, Wang Y, Li H, et al. Neck circumference and early stage atherosclerosis: the cardiometabolic risk in Chinese (CRC) study. Cardiovasc Diabetol 2014; 13: 107.
22. Ohishi M, Tatara Y, Ito N, et al. The combination of chronic kidney disease and increased arterial stiffness is a predictor for stroke and cardiovascular disease in hypertensive patients. Hypertens Res 2011; 34: 1209 - 1215.
23. Yen CH, Hung CL, Lee PY, et al. Segmental arterial stiffness in relation to B-type natriuretic peptide with preserved systolic heart function. PLoS One 2017; 12: e0183747.
24. Lanfranco, D'Elia,Is the triglyceride-glucose index ready for cardiovascular risk assessment?[J] .Nutr Metab Cardiovasc Dis, 2025, 35: 0.
25. Sun H, Ren X, Chen Z, et al. Association between body mass index and mortality in a prospective cohort of Chinese adults. Medicine (Baltimore) 2016; 95: e4327.
26. Liang J, Teng F, Liu X, et al. Synergistic effects of neck circumference and metabolic risk factors on insulin resistance: the Cardiometabolic Risk in Chinese (CRC) study. Diabetol Metab Syndr 2014; 6: 116.
27. Abdulhadi, Alotaibi,Abinash, Mahapatro,Mohit, Mirchandani et al. Triglyceride-glucose index as a marker in cardiovascular diseases; a bibliometric study and visual analysis.[J] .Ann Med Surg (Lond), 2025, 87: 0.
28. Yusong, Ye,Shu, Huang,Ruiyu, Wang et al. Global trends and emerging topics related to triglyceride-glucose index: A bibliometric analysis and visualization from 2000 to 2024.[J] .Medicine (Baltimore), 2024, 103: 0.

29. Yahui, Liu,Binbin, Zhu,Weicen, Zhou et al. Triglyceride-glucose index as a marker of adverse cardiovascular prognosis in patients with coronary heart disease and hypertension.[J] .Cardiovasc Diabetol, 2023, 22: 0.
30. Lambrinoudaki I, Kazani MV, Armeni E, et al. The TyG Index as a Marker of Subclinical Atherosclerosis and Arterial Stiffness in Lean and Overweight Postmenopausal Women. Heart Lung Circ 2018; 27: 716 - 724.
31. Alizargar J. Comparison of Carotid Ultrasound Indices and the Triglyceride Glucose Index in Hypertensive and Normotensive Community - Dwelling Individuals: A Case Control Study for Evaluating Atherosclerosis. Medicina (Kaunas) 2018; 54:.
32. Won KB, Park GM, Lee SE, et al. Relationship of insulin resistance estimated by triglyceride glucose index to arterial stiffness. Lipids Health Dis 2018; 17: 268.
33. Liang J, Li Y, Zhou N, et al. Synergistic effects of serum uric acid and cardiometabolic risk factors on early stage atherosclerosis: the cardiometabolic risk in Chinese study. PLoS One 2012; 7: e51101.
34. Poon AK, Meyer ML, Tanaka H, et al. Association of insulin resistance, from mid-life to late-life, with aortic stiffness in late-life: the Atherosclerosis Risk in Communities Study. Cardiovasc Diabetol 2020; 19: 11.
35. Wainwright P. Bidirectional Relationships and Disconnects between NAFLD and Features of the Metabolic Syndrome. Int J Mol Sci 2016; 17: 367.
36. Khojasteh, Malekmohammad,Evgeny E, Bezsonov,Mahmoud, Rafieian- Kopaei, Role of Lipid Accumulation and Inflammation in Atherosclerosis: Focus on Molecular and Cellular Mechanisms.[J] .Front Cardiovasc Med, 2021, 8: 0.
37. Melania, Gaggini,Francesca, Gorini,Cristina, Vassalle,Lipids in Atherosclerosis: Pathophysiology and the Role of Calculated Lipid Indices in Assessing Cardiovascular Risk in Patients with Hyperlipidemia.[J] .Int J Mol Sci, 2023, 24: 0.
38. Kang B, Yang Y, Lee EY, et al. Triglycerides / glucose index is a useful surrogate marker of insulin resistance among adolescents. Int J Obes (Lond) 2017; 41: 789 - 792.
39. Unger G, Benozzi SF, Perruzza F. Triglycerides and glucose index: a useful indicator of insulin resistance. Endocrinol Nutr 2014; 61: 533 - 540.
40. Guerrero-Romero F, Villalobos-Molina R, Jiménez-Flores JR, et al. Fasting Triglycerides and Glucose Index as a Diagnostic Test for Insulin Resistance in Young Adults. Arch Med Res 2016; 47: 382 - 387.
41. Amir, Ajoolabady,Domenico, Pratico,Ling, Lin et al. Inflammation in atherosclerosis: pathophysiology and mechanisms.[J] .Cell Death Dis, 2024, 15: 0.
42. Wolf D. Immunity and Inflammation in Atherosclerosis. Circ Res 2019; 124: 315 - 327.
43. Varghese JF, Patel R. Novel Insights in the Metabolic Syndrome-induced Oxidative Stress and Inflammation-mediated Atherosclerosis. Curr Cardiol Rev 2018; 14: 4 - 14.
44. Marcin, Myszko,Jerzy, Bychowski,Elżbieta, Skrzydlewska et al. The Dual Role of Oxidative Stress in Atherosclerosis and Coronary Artery Disease: Pathological Mechanisms and Diagnostic Potential.[J] .Antioxidants (Basel), 2025, 14: 0.
45. van den Munckhof ICL, Holewijn S, de Graaf J. Sex differences in fat distribution influence the association between BMI and arterial stiffness. J Hypertens 2017; 35: 1219 - 1225.
46. Katsuki A, Sumida Y, Urakawa H, et al. Increased visceral fat and serum levels of triglyceride are associated with insulin resistance in Japanese metabolically obese, normal weight subjects with normal glucose tolerance. Diabetes Care 2003; 26: 2341 - 2344.

Tables

Table 1. The clinical characteristics of participants under different levels of TyG index

	TyG in quartiles				*p* for trend
Variables	Q1	Q2	Q3	Q4	
	<3.97	3.97-5.92	5.93-9.3	≥9.3	
n	1181	1184	1180	1184	
crPWV（cm/s）	10.03±1.56	10.09±1.47	10.31±1.57	10.41±1.59	<0.001
cfPWV（cm/s）	10.37±1.83	10.57±1.95	10.81±1.92	10.93±1.80	<0.001
cdPWV（cm/s）	9.35±1.41	9.51±1.49	9.77±1.54	9.93±1.46	<0.001
age(years)	48.08±13.14	47.85±12.57	49.69±13.01	48.86±12.66	0.002
BMI(kg/m^2)	23.27±2.89	24.12±3.10	25.24±3.06	26.08±3.01	<0.001
NC (cm)	34.55±3.72	35.18±3.45	36.00±3.61	36.66±4.14	<0.001
WC (cm)	81.51±9.47	84.38±9.77	87.86±9.58	90.26±8.73	<0.001
SBP(mmHg)	121.17±16.02	124.27±17.01	127.89±17.04	130.51±16.89	<0.001
DBP(mmHg)	76.56±10.94	78.43±11.25	80.63±11.50	82.70±11.71	<0.001
2h OGTT(mmol/l)	5.90±1.46	6.02±1.46	6.24±1.52	6.52±1.49	<0.001
HbA1c (%)	5.21±0.39	5.26±0.40	5.31±0.40	5.36±0.40	<0.001
TC (mmol/l)	4.64±0.79	4.92±0.80	5.17±0.86	5.40±0.98	<0.001
HDL (mmol/l)	1.42±0.30	1.32±0.28	1.22±0.26	1.05±0.24	<0.001
LDL (mmol/l)	2.69±0.66	2.99±0.64	3.22±0.71	3.03±0.88	<0.001
SUA(μmol/l)	265.02±69.98	285.36±73.28	307.99±76.87	330.76±83.82	<0.001
HR(bpm)	69.76±10.37	70.05±9.99	70.68±10.29	71.22±10.02	0.002

Quantitative variables are shown as mean ± SD.

Abbreviations: crPWV, carotid-radial pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cdPWV, carotid-dorsalis pedis pulse wave velocity; BMI, body mass index; NC, neck circumference; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; 2h OGTT, 2 hours blood glucose of oral glucose tolerance test; HbA1c, hemoglobin A1c; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SUA, serum uric acid; HR, heart rate; bpm, beats per minute.

Table 2. Associations of TyG index with central and peripheral arterial stiffness.

				Model1		Model2	
	TyG in quartiles	Normal level	High level	OR	95%CI	OR	95%CI
crPWV（cm/s）		≤11.12	>11.12				
	Q1	971(82.2%)	210(17.8%)	1	1	1	1
	Q2	927(78.3%)	257(21.7%)	1.36	1.096~1.689	1.22	0.881~1.690
	Q3	835(70.8%)	345(29.2%)	1.889	1.528~2.334	1.937	1.401~2.678
	Q4	814(69.8%)	370(31.3%)	2.148	1.732~2.664	1.767	1.273~2.453
	p for trend	<0.001		<0.001		<0.001	
cfPWV（cm/s）		≤11.51	>11.51				
	Q1	954(80.8%)	227(19.2%)	1	1	1	1
	Q2	923(78.0%)	261(22.0%)	1.346	1.079~1.680	1.054	0.755~1.472
	Q3	880(74.6%)	300(25.4%)	1.447	1.161~1.804	1.213	0.865~1.702
	Q4	844(71.3%)	340(28.7%)	1.944	1.556~2.429	1.276	0.908~1.795
	p for trend	<0.001		<0.001		0.112	
cdPWV（cm/s）		≤10.57	>10.57				
	Q1	942(79.8%)	239(20.2%)	1	1	1	1
	Q2	923(78.0%)	261(22.0%)	1.143	0.929~1.405	1.075	0.814~1.420
	Q3	861(73.0%)	319(27.0%)	1.606	1.307~1.973	1.489	1.121~1.976
	Q4	820(69.3%)	364(30.7%)	2.015	1.635~2.484	1.586	1.190~2.112
	p for trend	<0.001		<0.001		<0.001	

Model1:adjusted sex age BMI

Model2:adjusted sex age BMI ,SBP, DBP ,LDL, HbA1c, SUA and HR

Abbreviations: crPWV, carotid-radial pulse wave velocity; cfPWV, carotid-femoral pulse wave velocity; cdPWV, carotid-dorsalis pedis pulse wave velocity; TyG: triglyceride-glucose; OR, odds ratio; CI, confidence interval.

Table 3. The relationship of TyG index with crPWV and cdPWV under different BMI levels

		TyG in quartiles					
		OR（95%C.I）					
	BMI (kg/m^2)	Q1	Q2	Q3	Q4	*p* for trend	*p* for interaction
crPWV（m/s）	<24	1	1.081 (0.809~1.446)	1.457 (1.056~2.009)	1.950 (1.338~2.843)	<0.001	<0.001
	[24~28)	1	1.167 (0.794~1.716)	1.042 (0.717~1.515)	1.702 (1.171~2.474)	0.003	
	≥28	1	0.568 (0.202~1.598)	1.291 (0.503~3.310)	1.571 (0.617~4.000)	0.006	
cdPWV（m/s）	<24	1	1.267 (0.898~1.787)	2.118 (1.485~3.021)	2.051 (1.373~3.063)	<0.001	<0.001
	[24~28)	1	1.368 (0.903~2.073)	1.696 (1.149~2.503)	2.347 (1.602~3.438)	<0.001	
	≥28	1	1.267 (0.513~3.125)	1.174 (0.494~2.792)	1.189 (0.508~2.785)	0.944	

Abbreviations: crPWV, carotid-radial pulse wave velocity; cdPWV, carotid-dorsalis pedis pulse wave velocity; TyG: triglyceride-glucose; BMI, body mass index; OR, odds ratio; CI, confidence interval.

Analysis was adjusted for age, sex, SBP, DBP,FPG and TC.

Figures

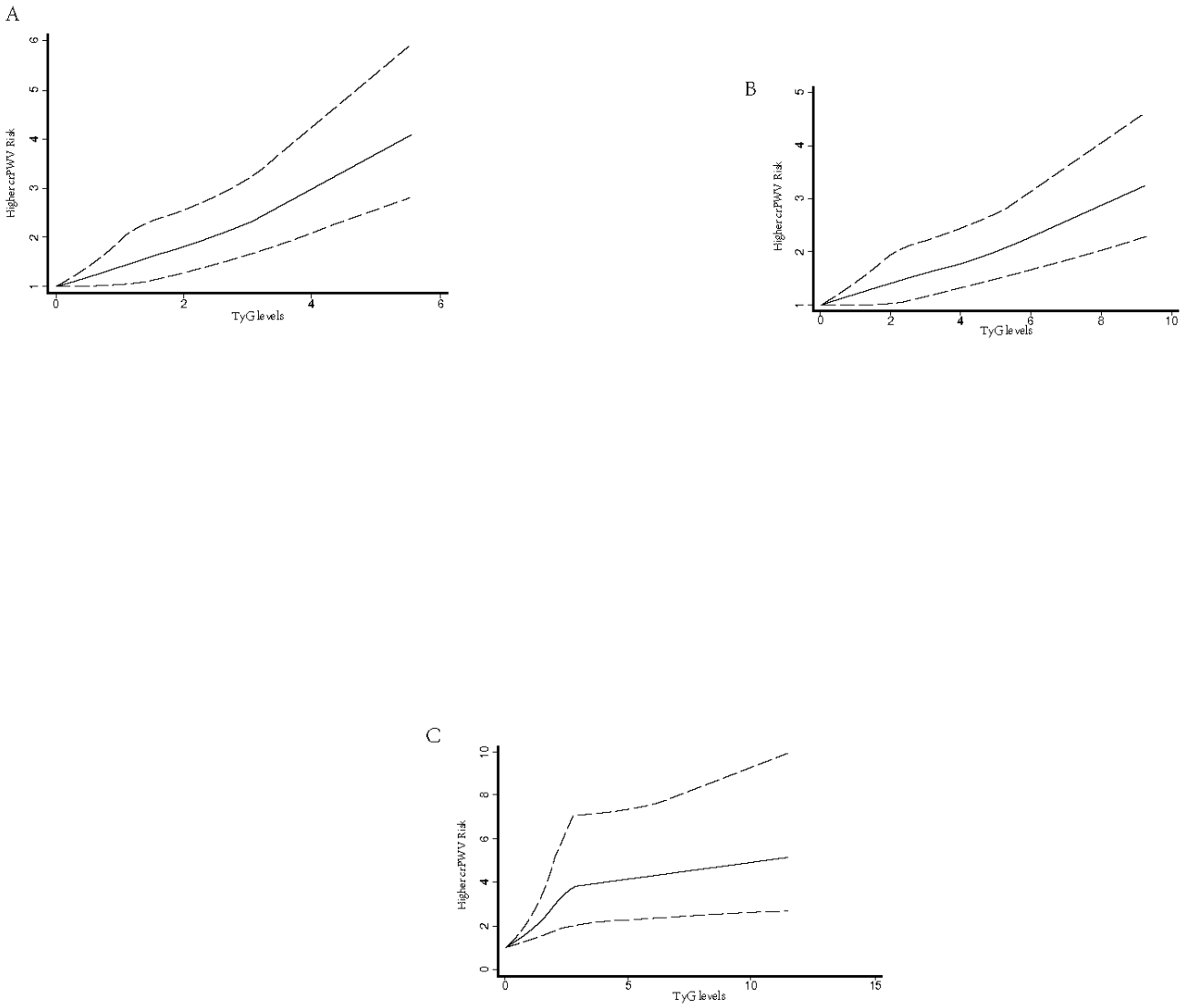


Figure 1

Dose-response relationship between TyG index and high crPWV risk under different BMI levels (A: BMI<24kg/m^2, B: 24kg/m^2≤BMI<28 kg/m^2, C: BMI≥28 kg/m^2). Analysis was adjusted for age, sex, SBP, DBP,FPG and TC .

Abbreviations:crPWV,carotid-radial pulse wave velocity; TyG:triglyceride-glucose;BMI, body mass index.

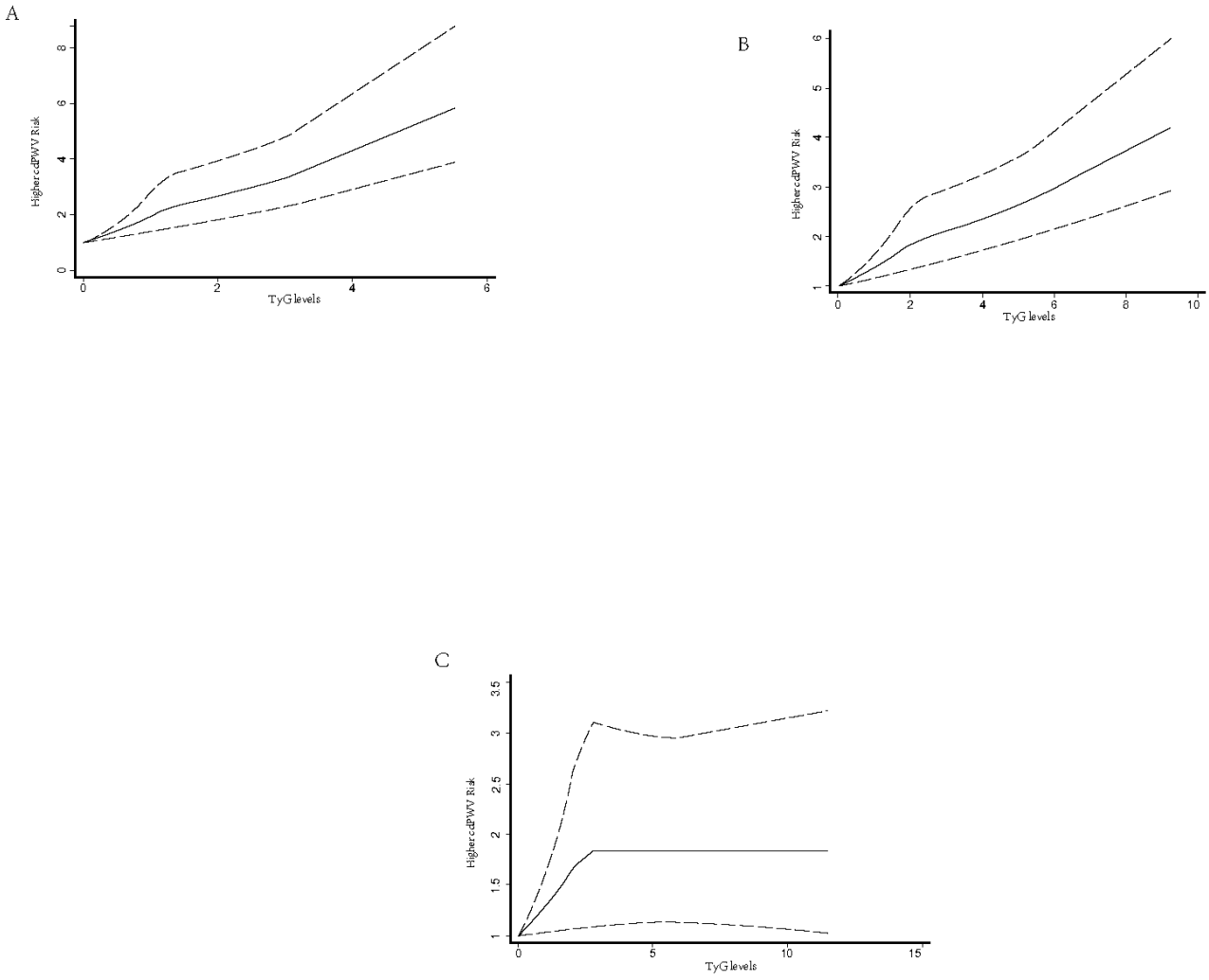


Figure 2

Dose-response relationship between TyG index and high cdPWV risk under different BMI levels (A: BMI<24kg/m^2, B: 24kg/m^2≤BMI<28 kg/m^2,

C: BMI≥28 kg/m^2). Analysis was adjusted for age, sex, SBP, DBP,FPG and TC. Abbreviations: cdPWV, carotid-dorsalis pedis pulse wave velocity; TyG: triglyceride-glucose; BMI, body mass index.