Triglyceride glucose-body mass index as a practical screening tool for metabolic dysfunction-associated steatotic liver disease in older adults: A community-based cohort study

Running title: TWI of young male athletes increases with PAEE

Ying Jiang BSc[1†], Weiqi Hong BSc[2†], Tianwei Xu PhD[3], Jialu Wang MPH[4], Molian Tang BSc[4], Qin Cao BSc[5], Yun Li PhD[1], Renying Xu MD, PhD[1,4]

[1]School of Public Health, North China University of Science and Technology, Tangshan, China
[2]Shanghai Pudong New Area Caolu Community Health Center, Shanghai, China
[3]Stress Research Institute, Department of Psychology, Stockholm University, Stockholm, Sweden
[4]Department of Clinical Nutrition, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China
[5]Health Management Center, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine, Shanghai, China
[†]Both authors contributed equally to this manuscript

Authors' email addresses and contributions:
Ying Jiang (School of Public Health, North China University of Science and Technology), Email: jiangying2558@renji.com. Contribution: conceptualization, data analysis, methodology, visualization, formal analysis, and writing—original draft.

Weiqi Hong (Associate Chief Physician, Chief of Preventive Medicine Section, Shanghai Pudong New Area Caolu Community Health Center), Email: narcessis@alumni.sjtu.edu.cn. Contribution: conceptualization, data analysis, investigation, visualization, and writing—original draft.

Tianwei Xu (Department of Psychology, Stockholm University), Email: tianwei.xu@psychology.su.se. Contribution: conceptualization, formal analysis, and supervision.

Jialu Wang (Department of Clinical Nutrition, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine), Email: 14775@renji.com. Contribution: data collection and data analysis.

Molian Tang (Department of Clinical Nutrition, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine), Email: 17731@renji.com. Contribution: conceptualization, formal analysis, and visualization.

Qin Cao (Health Management Center, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine), Email: caoqin@renji.com. Contribution: conceptualization, supervision, and writing—review & editing.

Yun Li (School of Public Health, North China University of Science and Technology), Email: liyun@ncst.edu.cn. Contribution: supervision and writing—review & editing.

Renying Xu (Department of Clinical Nutrition, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine), Email: 721001735@shsmu.edu.cn. Contribution: data analysis, methodology, supervision, and writing—review & editing.

Corresponding Author: Dr Renying Xu, Department of Clinical Nutrition, Ren Ji Hospital, Shanghai Jiao Tong University School of Medicine, No. 160, Pujian Road, Shanghai, China. Tel: +86-021-68383335. Email: 721001735@shsmu.edu.cn

ABSTRACT

Background and Objectives: As the prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD) rises, identifying reliable biomarkers for risk prediction is essential. This study assessed baseline triglyceride glucose (TyG) index and TyG-derived parameters as predictors of future MASLD risk in older adults. **Methods and Study Design:** We analyzed data from 2,757 Chinese adults (60-91 years) undergoing annual health checks (2017-2023). Incident MASLD was detected using ultrasound after excluding baseline cases. Cox proportional hazards models (with parameters in quartiles) and time-dependent ROC curves quantified associations and time-varying predictive performance among TyG-related parameters. The predictive performance of TyG-BMI was also compared with the hepatic steatosis index (HSI) in ROC analysis. **Results:** Over a median six-year follow-up, 584 incident MASLD cases occurred. TyG-derived parameters showed stronger associations than TyG index. Specifically, triglyceride glucose-body mass index (TyG-BMI) demonstrated the highest risk, with an adjusted hazard ratio of 6.42 (95% CI: 5.33-7.73) for the highest versus lowest quartile. This association remained robust in sensitivity analysis. TyG-BMI demonstrated numerically higher AUC values than the HSI (Men: TyG-BMI 0.758 vs. HSI 0.747; Women: TyG-BMI 0.759 vs. HSI 0.756), with overlapping confidence intervals. Time-dependent ROC analysis confirmed TyG-BMI's superior predictive accuracy over time. The optimal TyG-BMI cut-offs were 187.97 for men and 191.33 for women. **Conclusions:** TyG-BMI demonstrated superior predictive performance compared to TyG index itself, other TyG-derived parameters, and the HSI, supporting its utility as a practical screening tool in primary care.

Key Words: TyG index, obesity, metabolic dysfunction-associated steatotic liver disease, aged population, time-dependent ROC analysis

INTRODUCTION

Evidence from the Global Burden of Disease study indicates that metabolic dysfunction-associated steatotic liver disease (MASLD) has become the most rapidly escalating global health burden, including cirrhosis, liver cancer, and even hepatic failure.[1] A 2023 systematic review synthesized epidemiological data confirming MASLD affects 30% of the global population, with persistent upward trajectory over recent decades.[2] Early detection of high-risk MASLD individuals is clinically imperative.

The development and progression of MASLD are fundamentally driven by insulin resistance (IR).[3] Triglyceride glucose (TyG) index was reported as an effective tool to determine IR by integrating fasting triglycerides and fasting blood glucose level together,[4] showing potential value in early screening of MASLD.[5] Conventional MASLD biomarkers—notably fatty liver index (FLI) and MASLD Liver Fat Score—faced limitations in clinical applicability due to their complex calculations and high detection costs.[6] For expansive epidemiological investigations, the TyG index proves exceptionally viable owing to simplicity, cost-effectiveness, and reliability.[4] TyG index might have broad applications in clinical practices. Multiple cross-sectional analyses had proved that TyG index was a good indicator for detecting MASLD.[5] Further, TyG-derived parameters integrate TyG index with anthropometrical measurements, such as body mass index (BMI), waist circumference (WC), waist-to-height ratio (WHtR), and waist-to-hip ratio (WHR). These measurements capture different dimensions of adiposity, namely overall obesity (BMI), central obesity (WC, WHtR), and fat distribution (WHR); everyone was previously reported to be independently linked to MASLD progression.[7] Combining TyG index with these anthropometrical indicators may therefore provide a more accurate assessment of metabolic risk. Several cross-sectional studies have reported that TyG-derived parameters possessed the potential to enhance the identification of MASLD.[8, 9] However, existing evidences were predominantly cross-sectional and inconsistent regarding which TyG-derived parameter most robustly predicts MASLD: some studies favored TyG-BMI while others suggested TyG-WC, and still others reported gender-specific differences.[9-12] Moreover, studies specifically focusing on older adults, who have distinct metabolic and body composition characteristics, remain scarce.[13, 14]

We hypothesized that one of TyG-derived parameters would surpass TyG index itself in predicting incident MASLD among community-dwelling older adults. This study compared predictive capacity of baseline TyG index versus TyG-derived parameters regarding incident MASLD, establishing optimal cutoff values for older adults.

MATERIALS AND METHODS

Study cohort

This study was conducted in accordance with the ethical standards of the Declaration of Helsinki. Conducted as an observational retrospective cohort at a single center, this investigation enrolled 8,909 older Chinese adults undergoing annual health assessments at Shanghai Pudong New Area Caolu Community Health Service Center (2017-2023). The physical examination included general check-up, blood samples [blood routine test, fasting

plasma glucose (FPG), liver and renal function, lipid profiles], and abdominal ultrasound examination. Trained medical staff performed a face-to-face interview to document demographic characteristics, medical history, and information about exercise, drinking, and smoking. Alcohol consumption was assessed by asking participants about their drinking frequency over the past 12 months. Responses were categorized into four groups: never or almost never, occasional (<1 time/week), regular (1-6 times/week), and daily. For participants in regular and daily categories, further details on beverage type and typical quantity consumed were collected to estimate daily alcohol intake (g/day). A total number of 6,152 participants were excluded: (1) presence of hepatic steatosis detected by ultrasound at baseline (n = 5,203); (2) missing data on height, body weight, WC, hip circumference (HC), blood pressure, drinking, smoking, and physical exercise (n = 783); (3) alcohol intake exceeding sex-specific limits (men: ≥30 g/d; women: ≥20 g/d) (n = 98);15 (4) refused liver ultrasound at baseline (n = 36); (5) Follow-up <1 year (n = 32). Finally, 2,757 Chinese community-dwelling older participants (980 men and 1,777 women, aged 60-91 years) were included (Figure 1). Study protocol was approved by the Ethical Committee of Ren Ji Hospital, Shanghai Jiao Tong University (approval number: KY-2019-112). Written informed consent was obtained from all participants prior to their inclusion in the study.

Anthropometrical measurement and clinical information

All participants wore light clothing and bare feet when performing anthropometric measurements. Briefly, WC assessment adhered to the midpoint protocol between the lowermost rib and iliac crest. HC was recorded at the maximal gluteal protrusion level, corresponding to the superior iliac crest plane. Height, weight, WC, and HC were measured. BMI, waist-to-height ratio (WHtR), and waist-to-hip ratio (WHR), were calculated. Following a 10-min rest period, two sequential blood pressure readings were obtained using an automated oscillometric device (Omron HBP-9020, China), with the mean value retained for analysis.

Morning venous blood was collected after ≥8-hour fasting. FPG and total triglycerides (TG) were quantified via automated biochemistry analyzer (Roche Cobas C701module, Germany). Diagnostic thresholds followed established criteria: hyperglycemia: FPG ≥7.0 mmol/L or prior diabetes;[16] hypertension: systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg or confirmed with hypertension;[17] dyslipidemia: total cholesterol (TC) ≥6.2 mmol/L and/or triglyceride (TG) ≥2.3 mmol/L or prior dyslipidemia;[18] hyperuricemia: serum uric acid (SUA) ≥420 μmol/L or documented hyperuricemia or gout.[19]

Determination of MASLD (Outcome)

The diagnosis of incident MASLD was based on contemporary international consensus criteria, which mandate the concurrent presence of hepatic steatosis and at least one cardiometabolic risk factor.[20] In this cohort of older Chinese adults, all participants met at least one of the specified cardiometabolic criteria at baseline. These included: BMI ≥23 kg/m²; FPG ≥5.6 mmol/L or known type 2 diabetes; systolic/diastolic blood pressure ≥130/85 mmHg or use of specific antihypertensive treatment; or TG ≥1.70 mmol/L (data on high-density lipoprotein cholesterol were unavailable). Accordingly, for this study, incident MASLD was operationally defined as the new detection of hepatic steatosis via abdominal ultrasound during follow-up in participants free of steatosis at baseline. Sonographic steatosis was defined by the presence of at least two of the following features: (1) diffusely heightened hepatic near-field echogenicity with distal attenuation, (2) liver echogenicity greater than that of the renal cortex, and (3) poor visualization of the intrahepatic vessel borders.

Calculation of TyG index and derived parameters (Exposures)

The parameters were calculated as follows: TyG index = ln [fasting TG (mg/dL) × FPG (mg/dL)/²]; TyG-BMI=TyG × BMI; TyG-WHtR = TyG × WHtR; TyG – WHR = TyG × WHR; TyG - WC = TyG × WC.[21, 22]

Statistical analyses

Statistical analyses employed SAS version 9.4 (SAS Institute Inc., Cary, North Carolina). A two-tailed *p*-value of less than 0.05 was considered statistically significant for all tests. Participants were classified into two distinct groups based on the presence or absence of MASLD during the follow-up period. Baseline characteristics for the entire cohort were then summarized, with continuous variables expressed as either mean [standard deviation (SD)] or median (interquartile range), and categorical variables presented as numbers (percentage). For descriptive analyses, group comparisons between participants developing incident MASLD and those without incident MASLD were conducted using the Wilcoxon test for continuous variables and the chi-square test for categorical variables.

To examine the shape of the association between TyG index, TyG-derived parameters, and MASLD risk, we first used restricted cubic splines with five knots in Cox proportional hazards models, adjusted for the following covariates: sex (binary), age (continuous), exercise routines (binary: ≥1 time/week vs. <1 time/week), alcohol consumption frequency (categorical: never/almost never, occasional, regular, or daily), and smoking habits (binary:

current smoker vs. non-smoker). The results indicated significant nonlinear relationships for TyG-BMI, TyG-WHtR, TyG-WHR, and TyG-WC (all p for nonlinearity <0.05), while the TyG index did not show significant deviation from linearity ($p = 0.20$) (Supplementary Figure 1). Therefore, we categorized TyG index and each TyG-derived parameter into quartiles for subsequent risk analysis. Although the TyG index demonstrated a linear relationship, it was also categorized into quartiles to maintain consistency in presentation and facilitate direct comparison with the non-linear TyG-derived parameters.

To quantify the association between TyG index, TyG-derived parameters, and MASLD risk, we employed multivariate Cox proportional hazard regression models, reporting hazard ratios (HRs) with corresponding 95% confidence intervals (CIs). Model construction followed a sequential approach: an initial unadjusted Model was created; Model I adjusted for demographic factors (sex, age); and Model II additionally incorporated lifestyle covariates (exercise routines, alcohol consumption, and smoking habits). We verified compliance with the proportional hazards assumption using Kaplan-Meier curves and log-rank tests (Shown in Supplementary Figure 2). Furthermore, variance inflation factors (VIFs) for all model variables were examined to assess potential collinearity, with results indicating no significant multicollinearity concerns (determined by VIF <3). To evaluate the robustness of the primary findings across diverse populations, we employed Model II and systematically excluded participants diagnosed with dyslipidemia, hyperglycemia, hypertension, hyperuricemia, overweight and obesity at baseline in sensitivity analyses. Additionally, to address potential selection bias, participants with under 6 year-follow-up were excluded in another sensitivity analysis. Furthermore, considering the potential for MASLD regression over time, we performed an additional sensitivity analysis (Sensitivity-7) in which we redefined the outcome as "persistent MASLD." Cases considered likely to represent transient MASLD (i.e., a single positive diagnosis before the final visit, with MASLD free status at the final follow up) were excluded from this analysis.

Given the recognized association between alanine aminotransferase (ALT) levels and MASLD development, we also conducted a stratified analysis by baseline ALT levels (using the median value of 15.6 U/L as the cutoff) to examine whether the associations between TyG-related parameters and MASLD risk remained consistent across subgroups.[23]

We employed ROC curve analysis to determine the optimal discriminative thresholds of baseline TyG index and its derived parameters for predicting MASLD onset within this 6-year follow-up. To evaluate their predictive performance against an existing MASLD prediction tool, we also calculated the hepatic steatosis index (HSI) using the formula: HSI = 8 ×

(ALT/AST ratio) + BMI (+2, if female; +2, if diabetes mellitus), and compared its predictive performance with that of the TyG-related parameters in the same ROC analysis.[24] Subsequently, the time-dependent predictive performance of the baseline TyG index and TyG-derived parameters for incident MASLD at multiple follow-up timepoints was evaluated and compared using time-dependent ROC curve analysis.

RESULTS

Baseline characteristics

A total of 2,757 community-dwelling older adults were included (980 men and 1,777 women, ages ranging from 60 to 91 years). The proportion of participants who were followed up for 1-3 years, 4-5 years, and six-year, was 11.2%, 13.0%, and 75.8% respectively. Table 1 displayed baseline characteristics stratified by incident MASLD development during a median follow-up of 6 years, and 584 participants (197 men and 387 women) developed MASLD. The incidence of MASLD was 394/10,000 person-years. Compared to participants without MASLD, those developed MASLD exhibited significantly younger age and larger anthropometric measures (height, weight, WC, BMI, WHtR, WHR), TyG index, TyG BMI, TyG WHtR, TyG WHR, TyG WC, TG, FPG, alanine aminotransferase (ALT), uric acid level, and lower creatinine level. Furthermore, those developed MASLD demonstrated significantly higher prevalence of hypertension and dyslipidemia.

Association of TyG index and TyG-derived parameters with MASLD risk

Prior to multivariable-adjusted Cox regression, the proportional hazards assumption was confirmed as appropriate (Supplementary Figure 2). The unadjusted Cox regression model demonstrated statistically significant positive associations between quartiles of TyG index, TyG-derived parameters, and incident MASLD risk, with HRs generally increasing across quartiles (Table 2). Further adjustment of sex, age, and lifestyle behaviors (exercise routines, alcohol consumption, and smoking habits), these graded associations remained significant. Among the evaluated parameters, TyG-BMI demonstrated the strongest association with incident MASLD risk, with participants in the highest quartile (Q4) exhibiting an HR of 6.42 (95% CI 5.33-7.73) compared to the lowest quartile (Q1). Subsequently, progressively less pronounced associations were observed for TyG-WC (Q4 HR = 4.39, 95% CI 3.70-5.20), TyG-WHtR (Q4 HR = 3.77, 95% CI 3.19-4.45), TyG-WHR (Q4 HR = 2.40, 95% CI 2.06-2.82), and TyG index (Q4 HR = 2.30, 95% CI 1.98-2.68).

Sensitivity analysis

Sensitivity analysis confirmed stable associations between TyG index, TyG-derived parameters and incident MASLD risk (Table 3). Across all sensitivity analyses, the associations remained significant, with TyG-BMI consistently exhibiting the strongest effect. Specifically, after excluding participants with dyslipidemia at baseline (Sensitivity-1), the adjusted HR for TyG-BMI (Q4 vs. Q1) was 6.48 (95% CI: 5.26-7.97). Similar robust associations were observed after excluding those with hyperglycemia (HR = 6.64, 95% CI: 5.46-8.08), hypertension (HR = 6.25, 95% CI: 4.83-8.08), hyperuricemia (HR = 6.23, 95% CI: 5.13-7.56), or overweight/obesity (HR = 4.74, 95% CI: 3.68-6.12). In sensitivity analysis 7, which redefined the outcome as "persistent MASLD" by excluding likely transient cases, TyG-BMI continued to demonstrate the strongest association, with a markedly elevated HR of 10.45 (95% CI: 7.97-13.69) for the highest versus lowest quartile.

Stratified analysis by baseline ALT level

Stratified analysis by median baseline ALT level (≤15.6 U/L vs. >15.6 U/L) revealed that the positive associations between TyG index, TyG-derived parameters, and MASLD risk were consistent in direction across both subgroups (Supplementary Table 1). The magnitude of association was generally stronger in the low ALT group for most parameters, particularly for TyG-BMI, where the HR for Q4 versus Q1 was 7.36 (95% CI 5.59-9.70) in the low ALT group and 4.61 (95% CI 3.65-5.83) in the high ALT group.

Optimal thresholds of baseline TyG index, TyG-derived parameters and HSI for MASLD risk prediction

As shown in Supplementary Table 2, TyG-BMI demonstrated the highest AUC of 0.758 (95% CI: 0.728, 0.788) among men and 0.759 (95% CI: 0.737, 0.781) among women, while TyG index presented the lowest AUC. In a direct comparison, The HSI also showed good predictive performance, with AUCs of 0.747 (95% CI: 0.716, 0.778) among men and 0.756 (95% CI: 0.734, 0.779) among women. The AUC point estimates for TyG-BMI were numerically higher than those for the HSI, although their confidence intervals overlapped in both sexes. The optimal TyG-BMI cut-off value was 187.97 (sensitivity: 75.7%, specificity: 66.1%) for men and 191.33 (sensitivity: 74.7%, specificity: 63.2%) for women.

Time dependent ROC analysis for MASLD prediction using the TyG index and its derived parameters

Supplementary Figure 3 displayed temporal trajectories of AUC values via fluctuation curves, illustrating how the predictive accuracy of these indicators evolved across the follow-up period. All baseline TyG index and its derived parameters maintained robust predictive capacity for incident MASLD across multiple timepoints, with TyG-derived parameters consistently outperforming the standalone TyG index in AUC values. Notably, TyG-BMI achieved the highest predictive accuracy for MASLD onset in both genders (Table 4 and Supplementary Figure 3).

DISCUSSION

This large longitudinal cohort study of Chinese adults demonstrated that higher quartiles of the baseline TyG index and its derived parameters were associated with progressively increased risks of incident MASLD. Moreover, combining TyG index with anthropometrical measurements (BMI, WHtR, WHR, WC) enhanced both risk assessment and predictive accuracy for MASLD. Importantly, TyG-BMI showed superior performance, as reflected by the highest quartile hazard ratio and the greatest time-dependent AUC, in forecasting MASLD risk compared to the TyG index and other derived parameters. TyG-BMI emerged as the optimal biomarker for identifying high-risk older adults, underscoring its clinical utility in MASLD screening strategies.

As a cost-effective surrogate marker for IR,[3, 5] TyG index reflected IR's central role in MASLD pathogenesis. The core pathophysiological impact of IR manifested as impaired glucose uptake in adipose and muscular tissues. This defect suppressed triglyceride hydrolysis while simultaneously elevating hepatic free fatty acid uptake, which exacerbated triglyceride synthesis in the liver.[25-27] Consequently, IR, along with subsequent hyperinsulinemia and elevated systemic glucose and free fatty acid levels, promoted substantial hepatic lipid deposition, thereby initiating steatosis and advancing MASLD. Moreover, impaired insulin sensitivity in IR patients induced persistent hyperglycemia, triggering compensatory hyperinsulinemia and appetite stimulation.[28] This metabolic dysfunction promoted dietary preferences favoring high-carbohydrate intake and reinforced a self-perpetuating cycle. Simultaneously, IR correlated with low-grade chronic inflammation mediated by pro-inflammatory cytokines that both aggravated insulin resistance and accelerated MASLD pathogenesis. Key mechanisms comprised dysregulated adipose tissue lipolysis, enhanced de novo lipogenesis, compromised mitochondrial β-oxidation of fatty acids, abnormal fat

distribution, gut microbiome compositional shifts, and adipokine/cytokine level alterations.3 Recent Japanese and Chinese investigations identified significant TyG index-MASLD association, supporting regular TyG monitoring for risk mitigation.[29, 30] Furthermore, another large-scale cohort study established that elevated baseline TyG index independently correlated with increased risk of MASLD.[31] Building upon this framework, TyG-BMI extended predictive capacity of TyG index by incorporating BMI, which captured additional burden of adiposity. Excess adiposity, reflected by higher BMI, exacerbates IR through the release of free fatty acids and pro-inflammatory adipokines from dysfunctional adipose tissue, thereby amplifying the pathways described above.[32, 33] By multiplying TyG index (a marker of IR) with BMI (a measure of overall obesity), TyG-BMI effectively quantifies the synergistic crosstalk between systemic IR and adiposity, where a high degree of IR in the setting of significant obesity leads to an amplified risk of hepatic steatosis.[34] This integration of metabolic dysfunction and obesity-related pathways likely explained its superior predictive performance for MASLD.

This retrospective cohort research documented a MASLD incidence density of 394/10,000 person-years, closely aligning with findings from another Chinese investigation.[30] The similarity in incidence between the two studies resulted from the shared demographic characteristics of community residents in China aged over 60 years who underwent health examinations. Recent studies underscored the critical role of body fat percentage and adipose tissue distribution in driving hepatic steatosis progression. Our findings extend this understanding by demonstrating that integrating the TyG index with BMI (TyG-BMI) provides a stronger predictive signal than either component alone or its combination with other adiposity measures (WC, WHtR, WHR). TyG-derived parameters, which incorporated fasting plasma glucose, triglycerides, and simple anthropometrical measurements, were easily obtained and applied in clinical practice.[8, 35] Furthermore, investigations have sought to identify which TyG-derived parameters was the most suitable one to screen MASLD. A large-scale study found that TyG-BMI was a more effective predictor of future MASLD risk than traditional indicators, particularly among young and middle-aged participants who were not fat.[35] However, another research performed in overweight and obese populations demonstrated that TyG-WC was better than either TyG-BMI or TyG index in detecting MASLD.[8] The research on the American population established TyG-WC as the strongest predictor of MASLD, followed by TyG-WHtR and TyG-BMI.[9] Furthermore, another investigation revealed distinct gender-specific patterns, identifying TyG-WHtR as exhibiting enhanced efficacy for detecting MASLD in women, while TyG-WC was more effective in

men.[12] The discrepancies among previous studies could be explained by differences in study populations: many studies focused on adults with a small number of elderly participants whereas our research exclusively included the older population. Our longitudinal analysis in an exclusively older Chinese cohort confirmed TyG-BMI as the most predictive parameter in this demographic. Importantly, our results remained robust in multiple sensitivity analyses, including addressing potential outcome misclassification by excluding likely transient MASLD cases. Additionally, positive association was consistent across subgroups stratified by baseline ALT level, suggesting that its applicability extends beyond individuals with elevated liver enzymes. Notably, in a head-to-head comparison within our cohort, TyG-BMI demonstrated a comparable discriminative ability to the established HSI. The superiority of TyG-BMI lied not merely in a marginal statistical advantage but, more importantly, in its extreme simplicity of composition. While the HSI incorporates the ALT/AST ratio and diabetes status, TyG-BMI requires only TG, FPG, and BMI. This elimination of the need for liver enzymes or diabetes history constituted a major practical advantage for risk stratification in primary care settings and large-scale epidemiological screening. Furthermore, WC was subject to considerable error in older adults, due to age-related changes such as abdominal muscle laxity and postural variations.[13] This might be partly attributed to age-related body composition alterations, such as visceral fat redistribution, which could distort waist circumference as a reliable marker of adiposity and metabolic risk in older adults, whereas BMI might better reflect the overall burden of adiposity and muscle loss relevant to MASLD pathogenesis.[14] Consistently, a recent large-scale comparative study reported that TyG-BMI achieved the highest AUC for MASLD prediction among TyG-derived indices, outperforming both TyG-WC and TyG-WHtR in both sexes.[36]

The sex specific TyG BMI cut off values identified in this study were derived from our community based cohort of Chinese older adults and should therefore be regarded as preliminary. Their definitive calibration and validation represent necessary next steps. We acknowledge that the stability of these cut offs requires internal validation, and more importantly, their applicability to other populations, such as younger individuals, different ethnic groups, or patients with various comorbidities, is unknown and must be tested through external validation in independent and demographically diverse cohorts. Consequently, these thresholds should not be generalized to clinical settings beyond populations with similar characteristics without further confirmation.

Key strengths of the present investigation included its cohort design and the application of time-dependent ROC curves, which directly compared the predictive performance of baseline

TyG index and its derived parameters for MASLD risk at various follow-up time points. Additionally, the incorporation of multiple sensitivity analyses and adjustments for confounders further strengthened the reliability of the findings. Several limitations should be noted. The primary limitation was that MASLD diagnosis relied on abdominal ultrasound, which, despite its convenience and practicality, had limited sensitivity for mild hepatic steatosis and could not assess liver fibrosis or inflammation.[37] Future studies is necessary to consider incorporating complementary non-invasive tests, such as transient elastography (FibroScan) for fibrosis assessment, and the Fibrosis-4 (FIB-4) index to enhance diagnostic accuracy and risk stratification. Another limitation was the lack of data on important confounding factors. Specifically, we had no information on medication use, for example lipid-lowering, antidiabetic, and antihypertensive drugs, or on detailed dietary patterns, including intake of total fat, saturated and polyunsaturated fatty acids.[38] These factors directly influence triglyceride, glucose, and uric acid levels, which were core components of TyG-BMI or closely related metabolic pathways, and were also associated with MASLD risk.[39, 40] Their absence might limit our ability to fully adjust for potential confounding. Future prospective studies should therefore prioritize collecting such data to clarify the independent association of TyG-BMI with MASLD. Additionally, data on other potential confounders such as socioeconomic status were deficient.[41] Another constraint was the lack of systematic follow-up data on mortality and other severe comorbidities, including malignancies, which prevented us from accounting for competing risks that may influence absolute risk estimates in this older cohort. Moreover, all the participants were from one single-center, thus limiting its generalizability. Also, as the cohort was derived from individuals voluntarily undergoing health check-ups, it is susceptible to the "healthy volunteer" selection bias[42]. Participants in such settings were often more health-conscious, with better access to healthcare and potentially a lower baseline risk profile than the general elderly population. This might lead to an underestimation of the true MASLD incidence and could affect the absolute risk estimates, although the internal validity of the observed relative associations and the comparative performance among different TyG-derived parameters was likely preserved. Therefore, future multi-center, community-based studies encompassing a broader spectrum of older adults, including those not engaged in regular health screenings, are warranted to validate the generalizability and clinical utility of TyG-BMI as a screening tool.

Conclusions

TyG-BMI demonstrated superior performance in forecasting MASLD risk, compared to TyG index itself, other TyG-derived parameters, and the HSI. The robust association observed across sensitivity analysis and ALT subgroups supported its reliability. This practical tool enabled primary care providers to stratify older adults' risk during wellness visits, supporting timely interventions without specialized resources.

SUPPLEMENTARY MATERIALS

All supplementary tables and figures are available upon request from the editorial office, and are also accessible on the journal's webpage (apjcn.qdu.edu.cn).

DATA AVAILABILITY STATEMENT

The analytic code and data coding schemes for this study can be shared with other researchers for replication purposes. If necessary, please contact the corresponding author (Renying Xu, email address: 721001735@shsmu.edu.cn).

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

The authors declare no conflict of interest.

REFERENCES

1. Paik JM, Golabi P, Younossi Y, Srishord M, Mishra A, Younossi ZM. The Growing Burden of Disability Related to Nonalcoholic Fatty Liver Disease: Data From the Global Burden of Disease 2007-2017. Hepatol Commun. 2020;4(12):1769-80. doi:10.1002/hep4.1599.
2. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH): a systematic review. Hepatology. 2023;77(4):1335-47. doi:10.1097/hep.0000000000000004.
3. Khan RS, Bril F, Cusi K, Newsome PN. Modulation of Insulin Resistance in Nonalcoholic Fatty Liver Disease. Hepatology. 2019;70(2):711-24. doi:10.1002/hep.30429.
4. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord. 2008;6(4):299-304. doi:10.1089/met.2008.0034.
5. Wang J, Yan S, Cui Y, Chen F, Piao M, Cui W. The diagnostic and prognostic value of the triglyceride-glucose index in metabolic dysfunction-associated fatty liver disease (MAFLD): a systematic review and meta-analysis. Nutrients. 2022;14(23). doi:10.3390/nu14234969.

6. Simental-Mendía LE, Simental-Mendía E, Rodríguez-Hernández H, Rodríguez-Morán M, Guerrero-Romero F. The product of triglycerides and glucose as biomarker for screening simple steatosis and NASH in asymptomatic women. Ann Hepatol. 2016;15(5):715-20. doi:10.5604/16652681.1212431.

7. Er LK, Wu S, Chou HH, Hsu LA, Teng MS, Sun YC, et al. Triglyceride glucose-body mass index is a simple and clinically useful surrogate marker for insulin resistance in nondiabetic individuals. PLoS One. 2016;11(3):e0149731. doi:10.1371/journal.pone.0149731.

8. Khamseh ME, Malek M, Abbasi R, Taheri H, Lahouti M, Alaei-Shahmiri F. Triglyceride glucose index and related parameters (triglyceride glucose-body mass index and triglyceride glucose-waist circumference) identify nonalcoholic fatty liver and liver fibrosis in individuals with overweight/obesity. Metab Syndr Relat Disord. 2021;19(3):167-73. doi:10.1089/met.2020.0109.

9. Xue Y, Xu J, Li M, Gao Y. Potential screening indicators for early diagnosis of NAFLD/MAFLD and liver fibrosis: triglyceride glucose index-related parameters. Front Endocrinol (Lausanne). 2022;13:951689. doi:10.3389/fendo.2022.951689.

10. Wang M, Chang M, Shen P, Wei W, Li H, Shen G. Application value of triglyceride-glucose index and triglyceride-glucose body mass index in evaluating the degree of hepatic steatosis in non-alcoholic fatty liver disease. Lipids Health Dis. 2023;22(1):186. doi:10.1186/s12944-023-01954-5.

11. Zhang S, Du T, Zhang J, Lu H, Lin X, Xie J, et al. The triglyceride and glucose index (TyG) is an effective biomarker to identify nonalcoholic fatty liver disease. Lipids Health Dis. 2017;16(1):15. doi:10.1186/s12944-017-0409-6.

12. Peng H, Pan L, Ran S, Wang M, Huang S, Zhao M, et al. Prediction of MAFLD and NAFLD using different screening indexes: A cross-sectional study in U.S. adults. Front Endocrinol (Lausanne). 2023;14:1083032. doi:10.3389/fendo.2023.1083032.

13. Verweij LM, Terwee CB, Proper KI, Hulshof CT, van Mechelen W. Measurement error of waist circumference: gaps in knowledge. Public Health Nutr. 2013;16(2):281-8. doi:10.1017/s1368980012002741.

14. Polyzos SA, Vachliotis ID, Mantzoros CS. Sarcopenia, sarcopenic obesity and nonalcoholic fatty liver disease. Metabolism. 2023;147:155676. doi:10.1016/j.metabol.2023.155676.

15. National Workshop on Fatty Liver and Alcoholic Liver Disease CSoH, Chinese Medical Association, Fatty Liver Expert Committee, Chinese Medical Doctor Association. Guidelines of prevention and treatment for nonalcoholic fatty liver disease: a 2018 update. Chin J Hepatol. 2018;26(3):195-203. doi:10.3760/cma.j.issn.1007-3418.2018.03.008.

16. Committee ADAPP. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. Diabetes Care. 2022;45(Suppl 1):S17-S38. doi:10.2337/dc22-S002.

17. Revision JCfG. 2018 Chinese Guidelines for Prevention and Treatment of Hypertension-A report of the Revision Committee of Chinese Guidelines for Prevention and Treatment of Hypertension. J Geriatr Cardiol. 2019;16(3):182-241. doi:10.11909/j.issn.1671-5411.2019.03.014.

18. Adults JCfDCgoPaToDi. Chinese guidelines on prevention and treatment of dyslipidemia in adults. Chin J Cardiol. 2007;35(5):390-419. doi:10.3760/j.issn.0253-3758.2007.05.003.

19. The Chinese Society of Endocrinology CMA. Guideline for the diagnosis and management of hyperuricemia and gout in China (2019). Chin J Endocrinol Metab. 2020;36(1):1-13. doi:10.3760/cma.j.issn.1000-6699.2020.01.001.

20. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. J Hepatol. 2023;79(6):1542-56. doi:10.1016/j.jhep.2023.06.003.

21. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, Martínez-Abundis E, Ramos-Zavala MG, Hernández-González SO, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab. 2010;95(7):3347-51. doi:10.1210/jc.2010-0288.

22. Kuang M, Yang R, Huang X, Wang C, Sheng G, Xie G, et al. Assessing temporal differences in the predictive power of baseline TyG-related parameters for future diabetes: an analysis using time-dependent receiver operating characteristics. J Transl Med. 2023;21(1):299. doi:10.1186/s12967-023-04159-7.

23. Zhang X, Mu Y, Yan W, Ba J, Li H. Alanine aminotransferase within reference range is associated with metabolic syndrome in middle-aged and elderly Chinese men and women. Int J Environ Res Public Health. 2014;11(12):12767-76. doi:10.3390/ijerph111212767.

24. Lee JH, Kim D, Kim HJ, Lee CH, Yang JI, Kim W, et al. Hepatic steatosis index: a simple screening tool reflecting nonalcoholic fatty liver disease. Dig Liver Dis. 2010;42(7):503-8. doi:10.1016/j.dld.2009.08.002.

25. Angulo P, Lindor KD. Insulin resistance and mitochondrial abnormalities in NASH: a cool look into a burning issue. Gastroenterology. 2001;120(5):1281-5. doi:10.1053/gast.2001.23591.

26. Wong VW, Chitturi S, Wong GL, Yu J, Chan HL, Farrell GC. Pathogenesis and novel treatment options for non-alcoholic steatohepatitis. Lancet Gastroenterol Hepatol. 2016;1(1):56-67. doi:10.1016/s2468-1253(16)30011-5.

27. Timlin MT, Parks EJ. Temporal pattern of de novo lipogenesis in the postprandial state in healthy men. Am J Clin Nutr. 2005;81(1):35-42. doi:10.1093/ajcn/81.1.35.

28. Petersen MC, Shulman GI. Mechanisms of insulin action and insulin resistance. Physiol Rev. 2018;98(4):2133-223. doi:10.1152/physrev.00063.2017.

29. Kitae A, Hashimoto Y, Hamaguchi M, Obora A, Kojima T, Fukui M. The triglyceride and glucose index is a predictor of incident nonalcoholic fatty liver disease: a population-based cohort study. Can J Gastroenterol Hepatol. 2019;2019:5121574. doi:10.1155/2019/5121574.

30. Huanan C, Sangsang L, Amoah AN, Yacong B, Xuejiao C, Zhan S, et al. Relationship between triglyceride glucose index and the incidence of non-alcoholic fatty liver disease in the elderly: a retrospective cohort study in China. BMJ Open. 2020;10(11):e039804. doi:10.1136/bmjopen-2020-039804.

31. Wang Y, Wang J, Liu L, Yang P, Deng S, Liu X, et al. Baseline level and change trajectory of the triglyceride-glucose index in relation to the development of NAFLD: a large population-based cohort study. Front Endocrinol (Lausanne). 2023;14:1137098. doi:10.3389/fendo.2023.1137098.

32. Zou H, Xie J, Ma X, Xie Y. The value of TyG-related indices in evaluating MASLD and significant liver fibrosis in MASLD. Can J Gastroenterol Hepatol. 2025;2025:5871321. doi:10.1155/cjgh/5871321.

33. Boushehri YG, Meymanatabadi Z, Tanha AE, Azami P, Alaei M, Alamdari AA, et al. Association of triglyceride glucose-body mass index (TyG-BMI) with metabolic dysfunction-associated steatotic liver disease: a systematic review and meta-analysis. PLoS One. 2025;20(8):e0324483. doi:10.1371/journal.pone.0324483.

34. Chen Y, Lin X, Chen Y, Wang B, Xu C, Wang Y. Triglyceride-glucose index and related parameters in the diagnosis and predictive efficacy for older adults with NAFLD/MASLD: a prospective cohort study. Front Med (Lausanne). 2025;12:1626672. doi:10.3389/fmed.2025.1626672.

35. Wang R, Dai L, Zhong Y, Xie G. Usefulness of the triglyceride glucose-body mass index in evaluating nonalcoholic fatty liver disease: insights from a general population. Lipids Health Dis. 2021;20(1):77. doi:10.1186/s12944-021-01506-9.

36. Sheng G, Lu S, Xie Q, Peng N, Kuang M, Zou Y. The usefulness of obesity and lipid-related indices to predict the presence of non-alcoholic fatty liver disease. Lipids Health Dis. 2021;20(1):134. doi:10.1186/s12944-021-01561-2.

37. Khov N, Sharma A, Riley TR. Bedside ultrasound in the diagnosis of nonalcoholic fatty liver disease. World J Gastroenterol. 2014;20(22):6821-5. doi:10.3748/wjg.v20.i22.6821.

38. Semmler G, Datz C, Reiberger T, Trauner M. Diet and exercise in NAFLD/NASH: Beyond the obvious. Liver Int. 2021;41(10):2249-68. doi:10.1111/liv.15024.

39. Del Bo C, Perna S, Allehdan S, Rafique A, Saad S, AlGhareeb F, et al. Does the Mediterranean diet have any effect on lipid profile, central obesity and liver enzymes in non-alcoholic fatty liver disease (NAFLD) subjects? a systematic review and meta-analysis of randomized control trials. Nutrients. 2023;15(10). doi:10.3390/nu15102250.

40. Chooi YC, Zhang QA, Magkos F, Ng M, Michael N, Wu X, et al. Effect of an Asian-adapted Mediterranean diet and pentadecanoic acid on fatty liver disease: the TANGO randomized controlled trial. Am J Clin Nutr. 2024;119(3):788-99. doi:10.1016/j.ajcnut.2023.11.013.

41. Kardashian A, Serper M, Terrault N, Nephew LD. Health disparities in chronic liver disease. Hepatology. 2023;77(4):1382-403. doi:10.1002/hep.32743.

42. Lindsted KD, Fraser GE, Steinkohl M, Beeson WL. Healthy volunteer effect in a cohort study: temporal resolution in the Adventist Health Study. J Clin Epidemiol. 1996;49(7):783-90. doi:10.1016/0895-4356(96)00009-1.

Chinese participants aged ≥ 60 years in 2017 (n= 8,909)

Baseline: 2017

6,152 participants were excluded:

(1) presence of hepatic steatosis detected by ultrasound at baseline (n = 5,203; 58.4%)

(2) missing data on height, body weight, waist circumference, hip circumference, blood pressure, drinking, smoking, and physical exercise (n = 783; 8.8%)

(3) history of excessive alcohol consumption, equivalent to ≥ 30 g/d for men and ≥ 20 g/d for women (n=98; 1.1%)

(4) refused liver ultrasound at baseline (n = 36; 0.4%)

(5) follow-up < 1 year (n = 32 ; 0.4%)

Endpoint: 2023

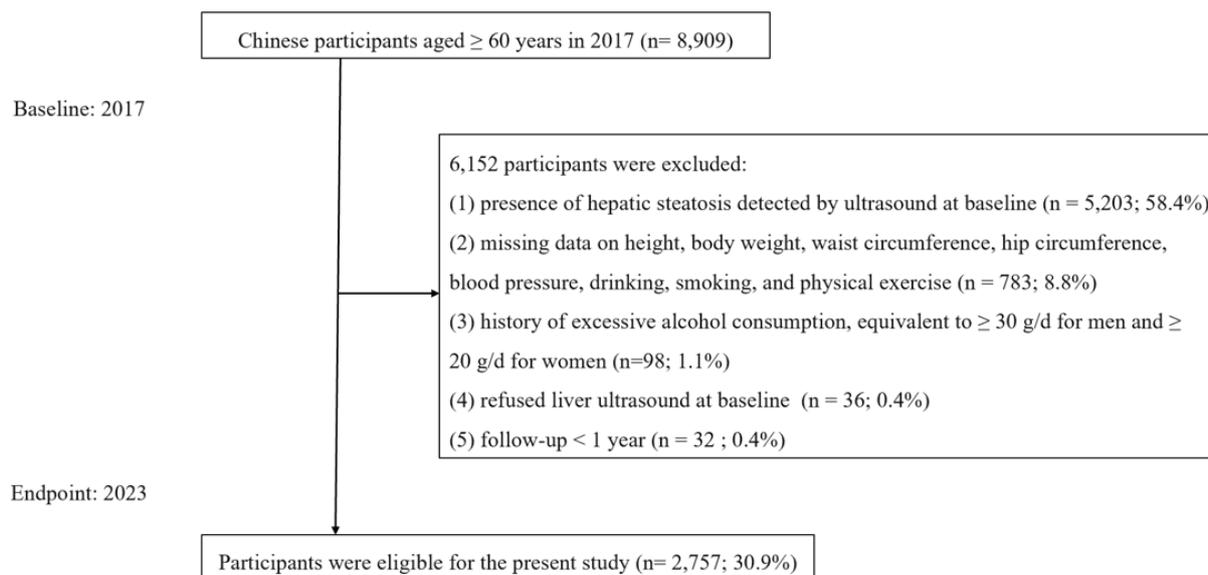Participants were eligible for the present study (n= 2,757; 30.9%)

Figure 1. Flow chart for participants selection

Table 1. Baseline characteristics of 2757 Chinese older adults MASLD-free at enrollment grouped by 6-year MASLD incidence[†‡]

Variables	Total (n= 2,757)	Incident MASLD (n= 584)	Without incident MASLD (n= 2,173)	p value
Age (years)	67.0 (63.0, 72.0)	66.0 (63.0, 69.0)	68.0 (63.0, 73.0)	< 0.001[**]
Height (cm)	158.0 (152.5, 164.0)	158.0 (153.0, 164.0)	157.5 (152.0, 164.0)	0.04[*]
Body weight (kg)	57.1 (51.6, 63.6)	61.7 (55.4, 68.6)	56.0 (50.8, 62.4)	< 0.001[**]
WC (cm)	80.0 (76.0, 85.0)	83.0 (78.0, 88.0)	80.0 (75.0, 85.0)	< 0.001[**]
BMI (kg/m^2)	22.97 (21.15, 24.90)	24.38 (22.60, 26.51)	22.60 (20.85, 24.40)	< 0.001[**]
WHtR	0.51 (0.48, 0.54)	0.52 (0.49, 0.55)	0.50 (0.47, 0.54)	< 0.001[**]
WHR	0.89 (0.85, 0.92)	0.89 (0.86, 0.92)	0.88 (0.85, 0.92)	< 0.001[**]
TyG	8.46 (8.14, 8.81)	8.59 (8.30, 8.94)	8.41 (8.11, 8.76)	< 0.001[**]
TyG-BMI	194.42 (175.72, 214.55)	210.50 (193.02, 231.88)	190.55 (172.64, 208.71)	< 0.001[**]
TyG-WHtR	4.30 (3.96, 4.66)	4.50 (4.17, 4.84)	4.25 (3.90, 4.62)	< 0.001[**]
TyG-WHR	7.48 (7.05, 7.92)	7.65 (7.30, 8.03)	7.42 (6.99, 7.88)	< 0.001[**]
TyG-WC	681.81 (627.41, 738.21)	713.91 (662.18, 769.08)	671.57 (618.84, 726.78)	< 0.001[**]
TC (mmol/L)	5.02 (4.43, 5.66)	5.00 (4.46, 5.63)	5.03 (4.41, 5.66)	0.87
TG (mmol/L)	1.19 (0.89, 1.60)	1.35 (1.01, 1.82)	1.14 (0.87, 1.53)	< 0.001[**]
FPG (mmol/L)	4.80 (4.50, 5.40)	4.90 (4.50, 5.50)	4.80 (4.40, 5.30)	0.02[*]
ALT (U/L)	15.60 (12.70, 19.50)	17.10 (13.80, 21.20)	15.20 (12.40, 18.90)	< 0.001[**]
AST (U/L)	22.40 (19.70, 25.90)	22.10 (19.70, 25.75)	22.50 (19.70, 26.00)	0.78
Creatinine (μmol/L)	66.00 (57.00, 76.00)	64.50 (56.00, 75.00)	66.00 (58.00, 76.00)	0.01[*]
Uric acid (mmol/L)	295.40 (246.40, 347.10)	309.85 (256.60, 369.25)	291.50 (243.60, 341.60)	< 0.001[**]
Hyperglycemia, N (%)	215 (7.80%)	50 (8.56%)	165 (7.59%)	0.44
Hypertension, N (%)	1,301 (47.19%)	298 (51.03%)	1,003 (46.16%)	0.04[*]
Dyslipidemia, N (%)	456 (16.54%)	127 (21.75%)	329 (15.14%)	< 0.001[**]
Hyperuricemia, N (%)	235 (8.52%)	57 (9.76%)	178 (8.19%)	0.23
Alcohol consumption frequency, N (%)				0.90
Never/almost never	2612 (94.74%)	554 (94.86%)	2058 (94.71%)	
Occasional (<1 time/week)	70 (2.54%)	15 (2.57%)	55 (2.53%)	
Regular (1–6 times/week)	15 (0.54%)	4 (0.68%)	11 (0.51%)	
Daily	60 (2.18%)	11 (1.88%)	49 (2.25%)	
Current smoking, N (%)	423 (15.34%)	84 (14.38%)	339 (15.60%)	0.47
Exercise (≥ 1 times/week), N (%)	1,625 (58.94%)	358 (61.30%)	1267 (58.31%)	0.19

MASLD: metabolic dysfunction-associated steatotic liver disease; WC: waist circumference; BMI: body mass index; WHtR: waist-to-height ratio; WHR: waist-to-hip ratio; TyG: triglyceride-glucose; TyG-BMI: triglyceride glucose-body mass index; TyG-WHtR: triglyceride glucose-waist-to-height ratio; TyG-WHR: triglyceride glucose-waist-to-hip ratio; TyG-WC: triglyceride glucose-waist circumference; TC: total cholesterol; TG: triglyceride; FBG: fasting blood glucose; ALT: alanine aminotransferase; AST: aspartate aminotransferase.

[†]Continuous variables are presented as medians (P25, P75) and categorical variables are presented as N (%)

[‡]Continuous variables were tested for differences between men and women using the Wilcoxon test, while categorical variables were tested using the chi-square test.

[*]p <0.05

[**]p <0.001

Table 2. Hazard ratios and 95% confidence intervals for future risk of MASLD by quartiles of baseline TyG index and TyG-derived parameters among 2,757 Chinese older adults

Parameters/ Quartile	Unadjusted model	Model I[†]	Model II[‡]
TyG			
Q1 (7.00-8.14)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2 (8.14-8.46)	1.21 (1.03, 1.43)	1.23 (1.04, 1.44)	1.23 (1.04, 1.45)
Q3 (8.46-8.81)	1.60 (1.37, 1.87)	1.64 (1.40, 1.91)	1.63 (1.40, 1.91)
Q4 (8.81-11.14)	2.23 (1.92, 2.60)	2.30 (1.97, 2.67)	2.30 (1.98, 2.68)
TyG-BMI			
Q1 (117.54-175.65)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2 (175.72-194.41)	2.42 (1.99, 2.95)	2.42 (1.98, 2.95)	2.42 (1.98, 2.95)
Q3 (194.42-214.55)	3.91 (3.24, 4.73)	3.95 (3.26, 4.77)	3.95 (3.26, 4.77)
Q4 (214.66-426.35)	6.35 (5.27, 7.64)	6.40 (5.32, 7.71)	6.42 (5.33, 7.73)
TyG-WHtR			
Q1 (2.71, 3.96)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2 (3.96, 4.30)	1.71 (1.44, 2.04)	1.78 (1.49, 2.12)	1.77 (1.49, 2.11)
Q3 (4.30, 4.66)	2.49 (2.11, 2.95)	2.66 (2.25, 3.15)	2.67 (2.25, 3.15)
Q4 (4.66, 7.14)	3.39 (2.88, 3.99)	3.77 (3.19, 4.45)	3.77 (3.19, 4.45)
TyG-WHR			
Q1 (5.13, 7.04)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2 (7.05, 7.48)	1.49 (1.27, 1.76)	1.51 (1.28, 1.78)	1.51 (1.28, 1.78)
Q3 (7.48, 7.92)	2.04 (1.74, 2.39)	2.06 (1.76, 2.42)	2.06 (1.76, 2.42)
Q4 (7.92, 10.65)	2.36 (2.02, 2.76)	2.40 (2.05, 2.81)	2.40 (2.06, 2.82)
TyG-WC			
Q1 (420.48, 627.39)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2 (627.41, 681.81)	1.98 (1.66, 2.38)	2.00 (1.67, 2.40)	2.00 (1.67, 2.40)
Q3 (682.05, 738.20)	2.80 (2.35, 3.33)	2.85 (2.40, 3.40)	2.85 (2.40, 3.40)
Q4 (738.30, 1227.24)	4.31 (3.64, 5.10)	4.38 (3.69, 5.20)	4.39 (3.70, 5.20)

MASLD: metabolic dysfunction-associated steatotic liver disease; TyG: triglyceride-glucose; TyG-BMI: triglyceride glucose-body mass index; TyG-WHtR: triglyceride glucose-waist-to-height ratio; TyG-WHR: triglyceride glucose-waist-to-hip ratio; TyG-WC: triglyceride glucose-waist circumference.

[†]Model I was adjusted for sex, age

[‡]Model II was adjusted for sex, age, exercise routines (binary: ≥1 time/week vs. <1 time/week), alcohol consumption frequency (categorical: never/almost never, occasional, regular, or daily), and smoking habits (binary: current smoker vs. non-smoker).

Table 3. Sensitivity analysis: adjusted hazard ratios and 95% confidence intervals for future risk of MASLD associated with baseline TyG index and TyG- derived parameters among Chinese older adults with different baseline health status

Parameters	Sensitivity-1[†] (n= 2,301)	Sensitivity-2[‡] (n= 2,542)	Sensitivity-3[§] (n= 1,456)	Sensitivity-4[¶] (n= 2,522)	Sensitivity-5[††] (n= 1,778)	Sensitivity-6[‡‡] (n= 2,091)	Sensitivity-7[¶¶] (n=2,393)
TyG							
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	1.25 (1.05, 1.50)	1.24 (1.05, 1.48)	1.14 (0.90, 1.44)	1.22 (1.03, 1.45)	1.31 (1.03, 1.66)	1.35 (1.13, 1.62)	1.45 (1.17, 1.81)
Q3	1.63 (1.37, 1.93)	1.61 (1.37, 1.90)	1.61 (1.29, 2.02)	1.49 (1.26, 1.76)	1.69 (1.34, 2.12)	1.69 (1.42, 2.01)	2.00 (1.63, 2.46)
Q4	2.11 (1.78, 2.50)	2.26 (1.92, 2.65)	2.49 (2.01, 3.09)	2.20 (1.87, 2.57)	2.41 (1.92, 3.01)	2.44 (2.06, 2.89)	3.00 (2.46, 3.66)
TyG-BMI							
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	2.49 (1.99, 3.10)	2.42 (1.96, 2.98)	1.95 (1.47, 2.59)	2.28 (1.85, 2.80)	1.98 (1.50, 2.62)	2.59 (2.09, 3.22)	2.92 (2.18, 3.91)
Q3	3.87 (3.13, 4.78)	3.82 (3.12, 4.66)	3.55 (2.73, 4.62)	3.79 (3.11, 4.62)	3.04 (2.33, 3.96)	4.01 (3.25, 4.94)	5.94 (4.51, 7.82)
Q4	6.48 (5.26, 7.97)	6.64 (5.46, 8.08)	6.25 (4.83, 8.08)	6.23 (5.13, 7.56)	4.74 (3.68, 6.12)	6.62 (5.39, 8.12)	10.45 (7.97, 13.69)
TyG-WHtR							
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	1.90 (1.57, 2.30)	1.75 (1.45, 2.10)	1.92 (1.49, 2.47)	1.73 (1.44, 2.08)	2.00 (1.57, 2.56)	1.84 (1.51, 2.23)	2.17 (1.71, 2.75)
Q3	2.53 (2.10, 3.06)	2.58 (2.16, 3.07)	2.73 (2.14, 3.49)	2.62 (2.19, 3.12)	2.44 (1.91, 3.10)	2.69 (2.23, 3.24)	3.56 (2.84, 4.46)
Q4	3.74 (3.11, 4.49)	3.77 (3.17, 4.49)	4.24 (3.33, 5.39)	3.67 (3.09, 4.37)	2.98 (2.34, 3.80)	3.81 (3.17, 4.58)	5.15 (4.12, 6.44)
TyG-WHR							
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	1.46 (1.21, 1.75)	1.49 (1.25, 1.77)	1.65 (1.30, 2.09)	1.51 (1.27, 1.79)	1.46 (1.15, 1.85)	1.51 (1.26, 1.82)	1.81 (1.45, 2.27)
Q3	2.11 (1.77, 2.51)	2.17 (1.84, 2.56)	2.25 (1.79, 2.83)	2.07 (1.75, 2.45)	1.81 (1.44, 2.28)	2.19 (1.83, 2.60)	2.74 (2.22, 3.38)
Q4	2.28 (1.91, 2.72)	2.38 (2.01, 2.81)	2.70 (2.15, 3.40)	2.32 (1.97, 2.74)	2.17 (1.73, 2.72)	2.42 (2.03, 2.88)	3.21 (2.60, 3.96)
TyG-WC							
Q1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Q2	2.05 (1.67, 2.51)	2.01 (1.66, 2.43)	2.12 (1.63, 2.76)	2.01 (1.66, 2.43)	1.84 (1.42, 2.38)	2.01 (1.65, 2.46)	2.55 (1.98, 3.30)
Q3	2.84 (2.34, 3.45)	2.88 (2.40, 3.46)	3.08 (2.39, 3.97)	2.80 (2.33, 3.36)	2.50 (1.95, 3.20)	2.90 (2.40, 3.52)	4.22 (3.31, 5.37)
Q4	4.27 (3.53, 5.16)	4.49 (3.75, 5.37)	4.86 (3.80, 6.23)	4.23 (3.54, 5.06)	3.37 (2.65, 4.29)	4.32 (3.58, 5.21)	6.38 (5.03, 8.10)

MASLD: metabolic dysfunction-associated steatotic liver disease; TyG: triglyceride-glucose; TyG-BMI: triglyceride glucose-body mass index; TyG-WHtR: triglyceride glucose-waist-to-height ratio; TyG-WHR: triglyceride glucose-waist-to-hip ratio; TyG-WC: triglyceride glucose-waist circumference.

[†]Sensitivity-1: Participants diagnosed with dyslipidemia at baseline were excluded.
[‡]Sensitivity-2: Participants diagnosed with hyperglycemia at baseline were excluded.
[§]Sensitivity-3: Participants diagnosed with hypertension at baseline were excluded.
[¶]Sensitivity-4: Participants diagnosed with hyperuricemia at baseline were excluded.
[††]Sensitivity-5: Participants with BMI $\geqslant$ 24 kg/m2 at baseline were excluded.
[‡‡]Sensitivity-6: Participants with a follow-up of less than 6 years were excluded.
[¶¶]Sensitivity-7: Participants with non-persistent MASLD (single diagnosis not at final follow-up) were excluded, redefining the outcome as persistent MASLD.
Every sensitivity analysis adjusted for sex, age, exercise routines (binary: $\geqslant$1 time/week vs. <1 time/week), alcohol consumption frequency (categorical: never/almost never, occasional, regular, or daily), and smoking habits (binary: current smoker vs. non-smoker).

Table 4. Areas under the time-dependent receiver operating characteristic curves for baseline TyG index and TyG- derived parameters predicting incident MASLD among Chinese older adults[†]

Predict time	AUC (95% CI)				
	TyG	TyG-BMI	TyG-WHtR	TyG-WHR	TyG-WC
Men (n= 980)					
1 year	0.643 (0.605, 0.682)	0.731 (0.698, 0.764)	0.664 (0.628, 0.700)	0.642 (0.606, 0.679)	0.688 (0.653, 0.723)
3 years	0.653 (0.614, 0.693)	0.770 (0.737, 0.803)	0.698 (0.662, 0.735)	0.661 (0.623, 0.700)	0.719 (0.683, 0.754)
4 years	0.678 (0.638, 0.718)	0.789 (0.756, 0.822)	0.714 (0.676, 0.751)	0.678 (0.639, 0.718)	0.735 (0.698, 0.771)
5 years	0.650 (0.611, 0.688)	0.782 (0.750, 0.815)	0.706 (0.669, 0.743)	0.657 (0.618, 0.696)	0.734 (0.699, 0.769)
6 years	0.649 (0.610, 0.688)	0.796 (0.764, 0.828)	0.710 (0.673, 0.747)	0.656 (0.617, 0.696)	0.735 (0.699, 0.771)
Women (n= 1,777)					
1 year	0.633 (0.603, 0.663)	0.776 (0.752, 0.800)	0.730 (0.704, 0.756)	0.659 (0.631, 0.687)	0.741 (0.716, 0.766)
3 years	0.646 (0.617, 0.675)	0.764 (0.739, 0.789)	0.721 (0.695, 0.748)	0.659 (0.630, 0.687)	0.728 (0.702, 0.755)
4 years	0.647 (0.617, 0.677)	0.766 (0.741, 0.792)	0.717 (0.690, 0.745)	0.655 (0.625, 0.684)	0.726 (0.699, 0.753)
5 years	0.645 (0.617, 0.674)	0.780 (0.757, 0.804)	0.719 (0.693, 0.746)	0.654 (0.626, 0.683)	0.731 (0.705, 0.757)
6 years	0.642 (0.613, 0.672)	0.775 (0.750, 0.800)	0.711 (0.684, 0.739)	0.648 (0.618, 0.678)	0.724 (0.697, 0.751)

AUC: area under the curves; MASLD: metabolic dysfunction-associated steatotic liver disease; TyG: triglyceride-glucose; TyG-BMI: triglyceride glucose-body mass index; TyG-WHtR: triglyceride glucose-waist-to-height ratio; TyG-WHR: triglyceride glucose-waist-to-hip ratio; TyG-WC: triglyceride glucose-waist circumference.
[†]Liver ultrasound data were unavailable in the second year of follow-up