Cardiovascular Diabetology

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Association between the triglyceride glucose index:Chinese visceral adiposity index (TyG-CVAI) and new-onset cardiovascular disease in middle-aged and older adults—insights from the China Health and Retirement Longitudinal Study (CHARLS)

Wenling Zheng[1*], Ziyue Man[2], Yu Li[1] and Xiaohong Zhu[1]

Abstract

Background Insulin resistance and visceral adiposity are established pathophysiological drivers of cardiovascular disease (CVD). The triglyceride–glucose (TyG) index and the Chinese visceral adiposity index (CVAI) are respective indicators of these conditions. However, the association of their combination, the TyG-CVAI, with the risk of new-onset CVD in the general population remains unclear. This study aimed to investigate this relationship using data from a large national cohort.

Methods This prospective study included participants in the China Health and Retirement Longitudinal Study (CHARLS) who lacked CVD at baseline. The TyG-CVAI was calculated and used to categorize participants into quartiles. The primary endpoint was the incidence of new-onset CVD. Kaplan–Meier analysis was performed to assess the cumulative incidence, while multivariable Cox proportional hazards models were used to estimate risk ratios (HRs) and 95% confidence intervals (CIs). The nonlinearity of the relationship was examined using restricted cubic splines (RCSs), and the predictive performance of the TyG-CVAI was evaluated and compared to that of its components using receiver operating characteristic (ROC) curve analysis.

Results Among the 7977 participants, 1221 ultimately developed a CVD. Kaplan–Meier analysis showed a significantly higher cumulative incidence of CVD with increasing TyG-CVAI quartiles (log-rank $P < 0.001$). After full adjustment for demographic, lifestyle, and cardiometabolic risk factors, participants in the highest quartile (Q4) had a significantly higher CVD risk (HR = 1.64, 95% CI 1.35–2.00) than those in the lowest quartile (Q1). RCS analysis revealed a significant nonlinear association between the TyG-CVAI and CVD risk, with a threshold at 778.62; below this point, no significant association was observed, whereas above it, each unit increase in the index conferred a 47% higher

*Correspondence:
Wenling Zheng
zwl08201032@126.com

Full list of author information is available at the end of the article

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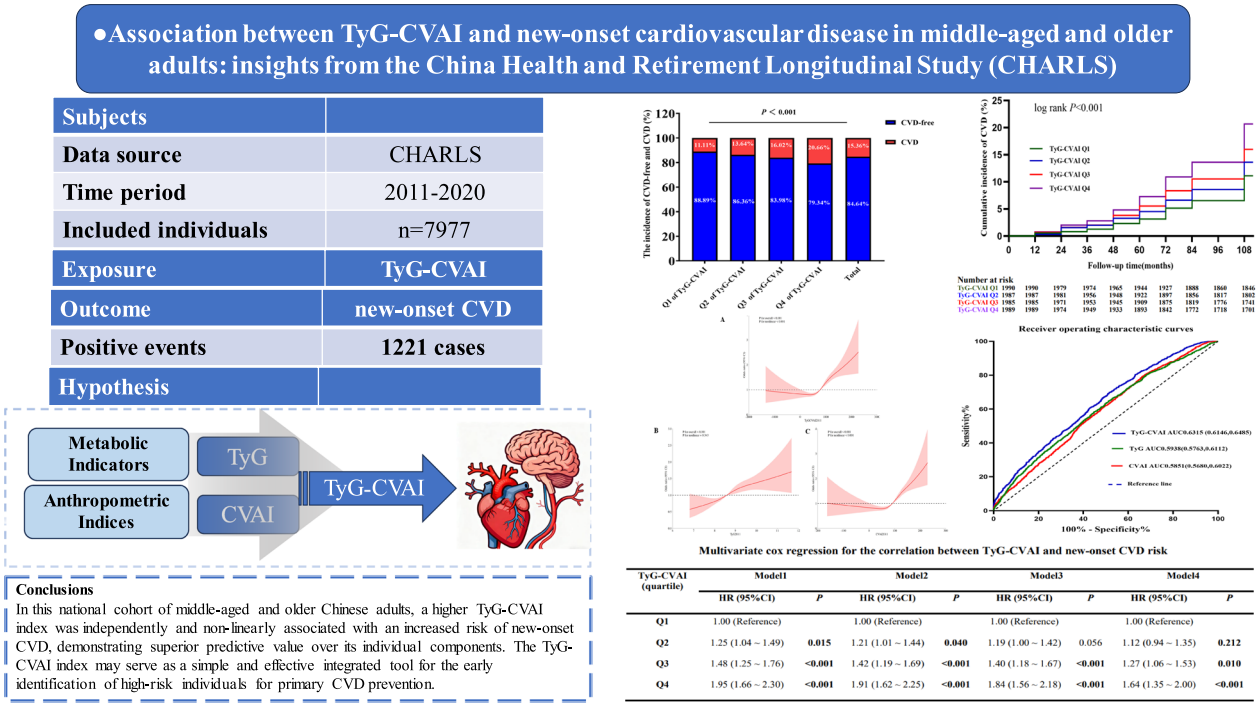
CVD risk (HR 1.47, 95% CI 1.31–1.64; $P < 0.001$). Furthermore, the TyG-CVAI (AUC = 0.6315) outperformed the TyG index (AUC = 0.5938) and the CVAI (AUC = 0.5851) alone in predicting the risk of CVD.

Conclusions In this national cohort of middle-aged and older Chinese adults, a higher TyG-CVAI was independently and nonlinearly associated with an increased risk of new-onset CVD, demonstrating superior predictive value over its individual components. The TyG-CVAI may serve as a simple and effective integrated tool for the early identification of high-risk individuals for primary CVD prevention.

Keywords TyG-CVAI, TyG, CVD, Heart disease, Stroke

Graphical abstract



Research insights

What is currently known about this topic?

The TyG-CVAI integrates insulin resistance and visceral adiposity, two established CVD risk drivers. Its association with stroke is recognized.

What is the key research question?

Does the TyG-CVAI index independently predict new-onset CVD risk, and is this relationship linear or threshold-dependent?

What is new?

This study confirms that TyG-CVAI is an independent predictive predictor for new-onset CVD and, for the first time identifies a significant non-linear threshold effect, defining a specific risk inflection point.

How might this study influence clinical practice?

It offers a simple, cost-effective tool for improved early risk stratification, potentially aiding targeted prevention in resource-limited settings by identifying individuals surpassing a critical risk threshold.

Introduction

Cardiovascular disease (CVD) remains the leading cause of global mortality and disability [1, 2]. In 2021, CVD accounted for approximately 44% of all deaths in China [3], while as of 2022, an estimated 330 million prevalent cases have been identified [4, 5]. Despite advances in the management of traditional risk factors such as hypertension [6], dyslipidaemia [7], and diabetes [8], a significant residual risk persists, underscoring the need for better early risk stratification tools.

Two central pathophysiological drivers of CVD are insulin resistance (IR) [9–11] and visceral adiposity [12–14]. The triglyceride–glucose (TyG) index, which is derived from routine fasting measurements, is a well-validated and low-cost surrogate marker for the former [15] and is strongly associated with incident CVD [16, 17], stroke [17, 18], and other metabolic disorders [19–21]. Similarly, a precise assessment of visceral fat—a metabolically active depot associated with inflammation [22], lipotoxicity [23], and cardiometabolic risk [24]—is

crucial. The Chinese visceral adiposity index (CVAI), a sex- and age-specific indicator, has been suggested to be superior to conventional measures such as body mass index for predicting cardiovascular events in Chinese populations [25–27].

Given the synergistic interaction between IR and visceral fat, composite indices that integrate both components may offer superior predictive value than those reflecting either factor alone [28–31]. To this end, the TyG-CVAI [32], which combines these two core pathways, represents a logical and promising tool. Recent research has shown that the TyG-CVAI has good predictive value for incident stroke [31, 32]. In addition, the longitudinal trajectories of the TyG-CVAI have been associated with CVD [33]. However, the predictive value of the baseline TyG-CVAI for new-onset CVD and its potential nonlinear relationship and threshold effect on the overall CVD risk, remains underexplored.

Therefore, using data from the China Health and Retirement Longitudinal Study (CHARLS), this prospective cohort study aimed to investigate the relationship between the TyG-CVAI at baseline and the risk of new-onset CVD. In addition, we sought to evaluate its predictive utility and threshold effect. Our findings may contribute to the development of a practical, accessible tool for the early identification of high-risk individuals, facilitating the development of targeted prevention strategies.

Methods

Study design

This study used data from the CHARLS, a nationally representative prospective cohort survey designed to investigate health and ageing related issues among Chinese adults aged 45 years and older. The baseline survey (Wave 1, n = 17,705) was conducted between 2011 and 2012, with follow-up surveys conducted in 2013 (Wave 2, n = 15,960), 2015 (Wave 3, n = 14,286), 2018 (Wave 4, n = 12,889), and 2020 (Wave 5, n = 11,572) [34]. The study design and sampling methods have previously been described in detail [32, 35]. The present analysis used data from Waves 1 to 5 to examine the association between the TyG-CVAI and new-onset CVD.

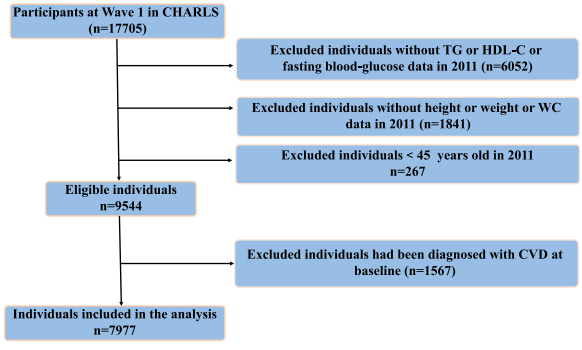


Fig. 1 Flowchart of the study population selection process in the analysis

Study population

A total of 17,705 participants were initially enrolled in CHARLS at baseline. We applied the following exclusion criteria to construct the final analytical sample: (1) missing data required to calculate the TyG-CVAI (including triglyceride (TG) levels, fasting blood glucose (FBG) levels, high-density lipoprotein cholesterol (HDL-C) levels, waist circumference (WC), body mass index (BMI), age and sex); (2) age < 45 years at baseline; and (3) a diagnosis of with CVD (including heart disease or stroke) at baseline or a missing CVD status during the follow-up period.

After application of the exclusion criteria, a total of 7977 participants were included in the final analysis. A flow chart detailing participant selection is provided in Fig. 1.

Physical measurement

Information, including sex, age, nationality, educational level, marital status, smoking status, drinking status and medically diagnosed diseases such as hypertension, dyslipidaemia, and diabetes, was obtained through questionnaires. Trained interviewers collected anthropometric measurements using standardised protocols. Height and weight were measured with participants wearing light clothing without shoes. BMI was calculated as weight in kilograms divided by height in metres squared (kg/m^2). WC was measured at the navel with the participants in a standing posture using a tape measure [36]. Blood pressure (BP) and heart rate (HR) were measured three times using an electronic sphygmomanometer after the participants had rested for 5 min, after which the average of the three readings was recorded.

Laboratory analyses

Fasting blood samples were collected from all participants after at least an 8 h fast overnight. Serum levels of TG, FBG, and HDL-C and other lipid profiles were measured using standard enzymatic methods. All laboratory procedures were performed using strict quality control protocols, as previously described in CHARLS [35].

Calculation of the TyG-CVAI

The TyG index was calculated as follows: $TyG = \ln[TG\ (mg/dL) \times FBG\ (mg/dL)/2]$. The CVAI was calculated using sex-specific formulas [26, 32]: $CVAI = 267.93 + 0.68 \times age\ (years) + 0.03 \times BMI\ (kg/m^2) + 4.00 \times WC\ (cm) + 22.00 \times \log_{10}(TG)\ (mmol/L) - 16.32 \times HDL\text{-}C\ (mmol/L)$ for men, and $CVAI = -87.32 + 1.71 \times age\ (years) + 4.23 \times BMI\ (kg/m^2) + 1.12 \times WC\ (cm) + 39.76 \times \log_{10}(TG)\ (mmol/L) - 11.66 \times HDL\text{-}C\ (mmol/L)$ for women. The

TyG-CVAI was then derived as the product of the TyG index and the CVAI: TyG-CVAI = TyG × CVAI [31, 32].

Diagnosis of CVD

CVD was evaluated on the basis of self-reported physician diagnoses of heart disease or stroke. Participants were asked "*Have you been diagnosed with heart disease (including heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems) by a doctor?*" or "*Have you been diagnosed with stroke by a doctor?*" at baseline and at each follow-up activity. Subjects were considered to have CVD when they reported being diagnosed with heart disease or stroke. At the next follow-up, individuals were required to confirm the presence of heart disease or stroke. The definition of CVD is consistent with that of previous studies based on CHARLS [35, 37] and with the diagnosis of CVD in mainstream international cohort studies [38].

The primary outcome was new-onset CVD, which was defined as the first CVD event recorded between follow-up Waves 2 and 5; individuals with a baseline CVD diagnosis were excluded. The time to event was calculated as the interval between the last survey without a CVD diagnosis and the survey at which the new event was first reported.

Statistical analysis

Continuous variables are summarised as the means ± standard deviations or medians with interquartile ranges, and categorical variables are summarised as frequencies and percentages. Baseline characteristics were compared between groups using ANOVA, the Kruskal–Wallis test, or the chi-square test, as appropriate.

Kaplan–Meier analysis was performed to assess the cumulative incidence of new-onset CVD within TyG-CVAI quartiles, and differences were evaluated using the log-rank test. Multivariable Cox regression analysis was performed to visualise the association between the TyG-CVAI and new-onset CVD. Four models were constructed: Model 1 was unadjusted; Model 2 was adjusted for sex, nationality, marital status, and educational level; Model 3 was adjusted for sex, age, nationality, marital status, educational level, smoking status, and drinking status; and Model 4 was adjusted for sex, age, nationality, marital status, educational level, smoking status, drinking status, and continuous variables, including FBG levels, TG levels, LDL-C levels, HbA1c levels, SBP, DBP and HR. The TyG-CVAI was analysed both as a continuous variable and categorical variable according to quartiles. The proportional hazards assumption was tested using Schoenfeld residuals.

Restricted cubic splines (RCSs) with four knots were used to examine potential nonlinear relationships between the TyG-CVAI and the risk of new-onset CVD. Two-piecewise linear regression analysis was applied to quantify the threshold effect. Receiver operating characteristic (ROC) curve analysis was performed to evaluate and compare the performance of the TyG-CVAI, TyG, and CVAI for predicting new-onset CVD. The area under the curve (AUC) was calculated for each index to quantify the predictive value of the TyG-CVAI, TyG and CVAI.

Sensitivity analyses were conducted to test the robustness of the results. First, we applied interpolation to handle missing data due to the presence of missing values and the exclusion of too many individuals with missing values in the aforementioned study. Approximately 20% of the total data were missing. The number and proportion of key missing data closely related to this study are shown in Supplementary Table 1. An analysis of the missingness pattern revealed strong correlations with missing indicators such as FBG, TG and HDL-C levels (Supplementary Table 2), suggesting a systematic mechanism consistent with the Missing at Random concept. For these variables, missing values were imputed using the Multiple Imputation by Chained Equations method. In contrast, missing data for sex and age were minimally correlated with the other variables (Supplementary Table 2), indicating a Completely at Random pattern. Given the low proportion and independence of these missing values, the corresponding observations were excluded. Statistical inferences were derived from five imputed datasets and pooled using Rubin's rules. Furthermore, given that underlying conditions such as hypertension, hyperlipidaemia, and diabetes are established risk factors for CVD, we excluded patients with these conditions at baseline from the respective analyses to minimise potential confounding.

All analyses were performed using R software (version 4.3.3). A two-tailed P value < 0.05 was considered to indicate statistical significance.

Results

Demographic characteristics of individuals based on TyG-CVAI quartiles

A total of 7977 individuals were included in this study, including 3788 males (47.49%) and 4189 females (52.51%), with a median age of 58 (51, 65) years. CVD risk factors were investigated according to the baseline TyG-CVAI quartiles. Significant increases in the TyG-CVAI quartiles for age, FBG level, total cholesterol (TC) level, TG level, C-reactive protein (CRP) level, glycosylated haemoglobin (HbA1c) levels, TyG, CVAI, systolic blood pressure (SBP), diastolic blood pressure (DBP), HR, weight, BMI, and WC, along with increased proportions of hypertension, dyslipidaemia, and diabetes, were detected. In contrast, HDL-C levels decreased with increasing TyG-CVAI quartile (Table 1).

Incidence of CVD

During the follow-up period, a total of 1221 new-onset CVD cases were reported, for an overall incidence of 15.36%. A progressive increase in CVD incidence was recorded for higher quartiles. The corresponding CVD incidence values were 11.11%, 13.64%, 16.02%, and 20.66% in TyG-CVAI quartiles Q1, Q2, Q3, and Q4, respectively. The CVD incidence rate differed significantly between the groups (Supplementary Fig. 1).

Association between the TyG-CVAI and CVD

During the 9-year follow-up period (2011–2020), a total of 1221 new-onset CVD events were recorded among the analysed population. Kaplan–Meier curves were constructed to evaluate the cumulative incidence of new-onset CVD based on the quartiles of the TyG-CVAI. Analysis of the curves showed a clear and graded association between the TyG-CVAI and the risk of developing CVD. As illustrated in Fig. 2, participants in the highest TyG-CVAI quartile (Q4) exhibited the highest cumulative incidence of CVD, followed by those in Q3, Q2 and Q1. The logarithmic rank test for the trend across the quartiles was statistically significant ($P < 0.001$), indicating a progressive increase in CVD risk with increasing TyG-CVAI value.

We performed multivariable Cox proportional hazards regression analyses to further quantify this association (Table 2). In the unadjusted model (Model 1), a clear dose–response relationship was observed. With respect to the lowest quartile (Q1, reference), the hazard ratios (HRs) and 95% confidence intervals (CIs) for the CVD risk increased progressively across TyG-CVAI quartiles: Q2 (HR = 1.25, 95% CI 1.04–1.49, $P = 0.015$), Q3 (HR = 1.48, 95% CI 1.25–1.76, $P < 0.001$), and Q4 (HR = 1.95, 95% CI 1.66–2.30, $P < 0.001$). This significant trend persisted after sequential adjustments for potential confounders. Adjusting for demographic factors (sex, marital status, educational level, and nationality) did not markedly attenuate this association. After further adjustment for additional variables (age, smoking status, and drinking status) in Model 3, the HRs for Q3 and Q4 remained statistically significant. Most importantly, after additional adjustment for traditional cardiometabolic risk factors (continuous variables that include FBG level, TG level, LDL-C level, HbA1c level, SBP, DBP, and HR) (Model 4), participants in the highest TyG-CVAI quartile (Q4) still exhibited a 64% increased risk of developing CVD relative to those in the lowest quartile (HR = 1.64, 95% CI 1.35–2.00; $P < 0.001$). A significant, albeit attenuated, increase in risk was also observed for Q3 (HR = 1.27; 95% CI 1.06–1.53; $P = 0.010$).

The results of both the Kaplan–Meier analysis and the multivariable Cox regression models consistently indicated that a higher TyG-CVAI was independently and positively associated with an increased risk of new-onset CVD in a nationally representative cohort of middle-aged and older Chinese adults.

Nonlinear association between the TyG-CVAI and CVD risk

RCS analysis revealed a significant nonlinear relationship between the TyG-CVAI at baseline and the risk of new-onset CVD (P for nonlinearity < 0.05) (Fig. 3A). A two-piecewise linear regression model was fitted to quantify this threshold effect. The analysis revealed a critical inflection point at a TyG-CVAI value of 778.62. In the fully adjusted model (Supplementary Table 3, Model 3), the association between the TyG-CVAI and CVD risk differed markedly above this threshold. Specifically, below the inflection point (< 778.62), the TyG-CVAI was not significantly associated with the CVD risk (HR 0.95, 95% CI 0.83–1.08; $P = 0.821$). Conversely, above the threshold (≥ 778.62), each unit increase in the TyG-CVAI was associated with a significant 47% increase in the risk of CVD (HR 1.47, 95% CI 1.31–1.64; $P < 0.001$) (Supplementary Table 3). A similar nonlinear pattern with a distinct threshold effect was observed for the CVAI component alone, with its inflection point at 90.74 (Supplementary Table 3). The logarithmic likelihood ratio test confirmed the statistically significant superiority of this segmented, nonlinear model over a standard linear assumption ($P < 0.001$), solidifying the presence of a threshold-driven, nonlinear dose–response relationship between the TyG-CVAI and CVD risk in this population.

Utility of TyG-CVAI in predicting new-onset CVD

We evaluated and compared the predictive performance of the TyG-CVAI and its components for new-onset CVD by performing ROC curve analyses. As shown in Fig. 4, the TyG-CVAI showed good predictive performance, with an AUC of 0.6315 (95% CI 0.6146–0.6485). This value was significantly higher than the AUC values for the TyG index alone (AUC = 0.5938, 95% CI 0.5763–0.6112) and the CVAI alone (AUC = 0.5851, 95% CI 0.5680–0.6022). The cut-off value for the TyG-CVAI was 737.48, with a sensitivity of 68.9% and a specificity of 65.3%.

Subgroup analyses

Subgroup analyses were performed to assess the consistency of the association between higher TyG-CVAIs and increased CVD risks across groups stratified by various demographic and clinical factors (Fig. 5). A significantly increased risk (HR > 1) was consistently observed in almost all the subgroups, including those stratified by sex, age (except in the 75 year age group), ethnicity, and cardiometabolic status (hypertension, dyslipidaemia, and diabetes). Notably, the association was not significant among participants aged ≥ 75 years (HR 1.04, 95%

Table 1 Characteristics of the study subjects at baseline in CVD analysis by TyG-CVAI quartiles

Variables	Total (n = 7977)	Q1 (n = 1994)	Q2 (n = 1995)	Q3 (n = 1994)	Q4 (n = 1994)	*P*
Age, year	58 (51, 65)	56 (49,62)	57 (51,64)	58 (51,65)	60 (54,67)	**< 0.001**
Sex, n(%)						**< 0.001**
Male	3788 (47.49)	1187 (59.53)	885 (44.36)	801 (40.17)	915 (45.89)	
Female	4189 (52.51)	807 (40.47)	1110 (55.64)	1193 (59.83)	1079 (54.11)	
Nationality, n(%)						0.157
Han nationality	6799 (85.23)	1671 (83.80)	1706 (85.51)	1701 (85.31)	1721 (86.31)	
National minority	1178 (14.77)	323 (16.20)	289 (14.49)	293 (14.69)	273 (13.69)	
Marital status, n(%)						0.299
Married	6672 (83.64)	1672 (83.85)	1686 (84.51)	1642 (82.35)	1672 (83.85)	
Others	1305 (16.36)	322 (16.15)	309 (15.49)	352 (17.65)	322 (16.15)	
Educational level, n(%)						**< 0.001**
Illiterate	2352 (29.49)	489 (24.52)	624 (31.29)	632 (31.70)	607 (30.44)	
Primary school	3278 (41.10)	896 (44.93)	821 (41.17)	791 (39.67)	770 (38.62)	
Middle or high school	2245 (28.15)	583 (29.24)	533 (26.73)	541 (27.13)	588 (29.49)	
College or above	101 (1.27)	26 (1.30)	16 (0.80)	30 (1.50)	29 (1.45)	
BUN, mg/dL	15.77 ± 4.60	16.17 ± 4.71	15.61 ± 4.80	15.57 ± 4.49	15.73 ± 4.36	**< 0.001**
FBG, mg/dL	110.08 ± 37.15	101.75 ± 23.48	104.04 ± 28.15	110.26 ± 35.50	124.27 ± 51.11	**< 0.001**
Serum creatinine, mg/dL	0.78 ± 0.24	0.78 ± 0.18	0.77 ± 0.23	0.78 ± 0.32	0.81 ± 0.20	**< 0.001**
TC, mg/dL	193.72 ± 38.82	185.38 ± 35.49	190.24 ± 36.88	195.78 ± 37.76	203.47 ± 42.48	**< 0.001**
TG, mg/dL	131.70 ± 108.38	81.33 ± 41.43	101.46 ± 47.52	132.39 ± 67.56	211.62 ± 169.20	**< 0.001**
HDL-C, mg/dL	51.59 ± 15.34	61.48 ± 16.29	54.84 ± 13.38	48.80 ± 12.15	41.24 ± 11.25	**< 0.001**
LDL-C, mg/dL	116.43 ± 35.04	109.64 ± 31.12	116.68 ± 32.50	121.24 ± 34.10	118.18 ± 40.67	**< 0.001**
CRP, mg/dL	2.73 ± 7.68	2.46 ± 7.70	2.59 ± 8.40	2.69 ± 7.72	3.16 ± 6.82	**0.025**
HbA1c, %	5.26 ± 0.81	5.10 ± 0.58	5.16 ± 0.67	5.26 ± 0.83	5.51 ± 1.03	**< 0.001**
Serum UA, mg/dL	4.46 ± 1.25	4.29 ± 1.17	4.24 ± 1.17	4.41 ± 1.23	4.89 ± 1.33	**< 0.001**
Cystatin C, mg/dL	1.02 ± 0.29	1.02 ± 0.23	1.01 ± 0.29	1.02 ± 0.34	1.03 ± 0.28	0.392
TyG	8.67 ± 0.67	8.22 ± 0.47	8.46 ± 0.46	8.76 ± 0.51	9.25 ± 0.71	**< 0.001**
CVAI	91.65 ± 44.51	39.22 ± 36.72	78.24 ± 8.73	104.47 ± 10.29	144.69 ± 22.14	**< 0.001**
TyG-CVAI	808.14 ± 423.05	320.12 ± 308.75	660.48 ± 68.31	913.24 ± 81.35	1338.79 ± 238.24	**< 0.001**
SBP, mmHg	134 ± 57	125 ± 45	130 ± 51	136 ± 66	144 ± 61	**< 0.001**
DBP, mmHg	76 ± 12	72 ± 12	74 ± 12	76 ± 12	80 ± 12	**< 0.001**
HR, beats/min	72 (65, 79)	70 (64,78)	71 (64,77)	72 (66,79)	73 (67,80)	**< 0.001**
Height, cm	157.9 ± 8.6	158.4 ± 7.9	157.4 ± 8.5	157.0 ± 8.7	158.8 ± 9.1	**< 0.001**
Weight, kg	57.4 (50.7, 65.1)	51.0 (45.8,55.9)	55.1 (49.4,60.6)	59.6(53.5,65.2	67.8 (60.2,75.1)	**< 0.001**
BMI, kg/m^2	22.99 (20.75, 25.53)	20.17 (18.80,21.67)	22.17 (20.57,23.64)	23.91 (22.22,25.60)	26.57 (24.66,28.74)	**< 0.001**
WC, cm	84.0 (77.2, 91.2)	74.2 (70.1,77.8)	81.2 (78.0,84.4)	87.2 (84.0,90.0)	96.0 (91.4,100.0)	**< 0.001**
Smoke, n(%)						**< 0.001**
No	4815 (60.36)	983 (49.30)	1245 (62.41)	1325 (66.45)	1262 (63.29)	
Yes	3162 (39.64)	1011 (50.70)	750 (37.59)	669 (33.55)	732 (36.71)	
Drink, n(%)						**< 0.001**
No	5882 (73.74)	1325 (66.45)	1499 (75.14)	1553 (77.88)	1505 (75.48)	
Yes	2095 (26.26)	669 (33.55)	496 (24.86)	441 (22.12)	489 (24.52)	
Hypertension, n(%)						**< 0.001**
No	4977 (62.83)	1551 (78.25)	1336 (67.58)	1203 (60.73)	887 (44.75)	
Yes	2945 (37.17)	431 (21.75)	641 (32.42)	778 (39.27)	1095 (55.25)	
Dyslipidemia, n(%)						**< 0.001**
No	6113 (78.39)	1709 (87.37)	1634 (83.62)	1475 (76.07)	1295 (66.44)	
Yes	1685 (21.61)	247 (12.63)	320 (16.38)	464 (23.93)	654 (33.56)	
Diabetes, n(%)						**< 0.001**

Table 1 (continued)

Variables	Total (n = 7977)	Q1 (n = 1994)	Q2 (n = 1995)	Q3 (n = 1994)	Q4 (n = 1994)	*P*
No	6869 (87.16)	1846 (93.80)	1814 (91.89)	1688 (85.82)	1521 (77.13)	
Yes	1012 (12.84)	122 (6.20)	160 (8.11)	279 (14.18)	451 (22.87)	

BUN, blood urea nitrogen; FBG, fasting blood glucose; TC, Total cholesterol; TG, Triglycerides; LDL-C, low-density lipoproteins cholesterol; HDL-C, high-density lipoproteins cholesterol; CRP, C-reactive protein; HbA1c, glycosylated hemoglobin; UA, uric acid; TyG, triglyceride-glucose index; CVAI, Chinese visceral adiposity index; TyG-CVAI, triglyceride glucose Chinese visceral adiposity index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; BMI, body mass index; WC, waist circumference

The statistical significance is set to a two-sided *P* value less than 0.05 and is marked in bold

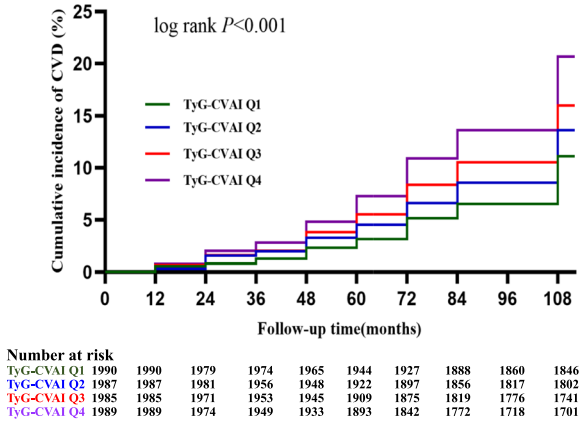


Fig. 2 Kaplan–Meier curves for new-onset CVD across quartiles of TyG-CVAI

CI 0.60–1.81) or those with a college education or above (HR 0.79, 95% CI 0.29–2.17). The formal tests for interactions were not significant for any variable (all *P* values for interactions > 0.05), indicating that the positive association between the TyG-CVAI and CVD risk was generally homogeneous across the studied population.

Sensitivity analysis

We performed a sensitivity analysis using Cox regression models with multiple imputation of the missing data to assess the robustness of our findings. The results remained consistent, showing a graded positive association between TyG-CVAI quartiles and the CVD risk after sequential adjustments. In the fully adjusted model, which accounted for demographics, lifestyle, and interpolated cardiometabolic parameters, participants in the highest quartile (Q4) retained a 60% higher CVD risk (HR 1.60, 95% CI 1.34–1.92, $P < 0.001$) than those in the lowest quartile (Q1) (Supplementary Table 4).

Furthermore, we performed sensitivity analyses after excluding participants with baseline hypertension (Supplementary Table 5), dyslipidaemia (Supplementary Table 6), or diabetes (Supplementary Table 7) to evaluate the robustness of the primary association. The graded association between higher TyG-CVAI quartiles and an increased CVD risk consistently remained significant. Specifically, the risk for participants in the highest quartile (Q4) was substantially increased in all three exclusion analyses (adjusted HR: 1.64 for the nonhypertensive cohort, 1.65 for the nondyslipidaemic cohort, and 1.67 for the nondiabetic cohort), confirming that the predictive value of the TyG-CVAI is independent of these common cardiometabolic comorbidities.

Discussion

In this large, nationally representative cohort of middle-aged and older Chinese adults, we observed that an elevated TyG-CVAI at baseline was independently associated with an increased risk of new-onset CVD after comprehensive adjustment for demographic, lifestyle and clinical confounders. Specifically, compared with participants in the lowest quartile, participants in the highest quartile of the TyG-CVAI had a significantly higher risk of developing CVD (64%).

The robustness of our findings is supported by the consistent results obtained using different analytical methods. The persistence of a significant association in the

Table 2 Multivariate cox regression for the correlation between TyG-CVAI and new-onset CVD risk

TyG-CVAI (quartile)	Model1		Model2		Model3		Model4	
	HR (95%CI)	*P*	HR (95%CI)	*P*	HR (95%CI)	*P*	HR (95%CI)	*P*
Q1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2	1.25 (1.04–1.49)	**0.015**	1.21 (1.01–1.44)	**0.040**	1.19 (1.00–1.42)	0.056	1.12 (0.94–1.35)	**0.212**
Q3	1.48 (1.25–1.76)	**< 0.001**	1.42 (1.19–1.69)	**< 0.001**	1.40 (1.18–1.67)	**< 0.001**	1.27 (1.06–1.53)	**0.010**
Q4	1.95 (1.66–2.30)	**< 0.001**	1.91 (1.62–2.25)	**< 0.001**	1.84 (1.56–2.18)	**< 0.001**	1.64 (1.35–2.00)	**< 0.001**

Model1: Unadjusted

Model2: Adjust: Sex, marital status, educational level and nationality

Model3: Adjust: Sex, marital status, educational level, nationality, age, smoking status and drinking status

Model4: Adjust: Sex, marital status, educational level, nationality, age, smoking status, drinking status, FBG, TG, LDL-C, HbA1c, SBP, DBP and HR

The statistical significance is set to a two-sided *P* value less than 0.05 and is marked in bold

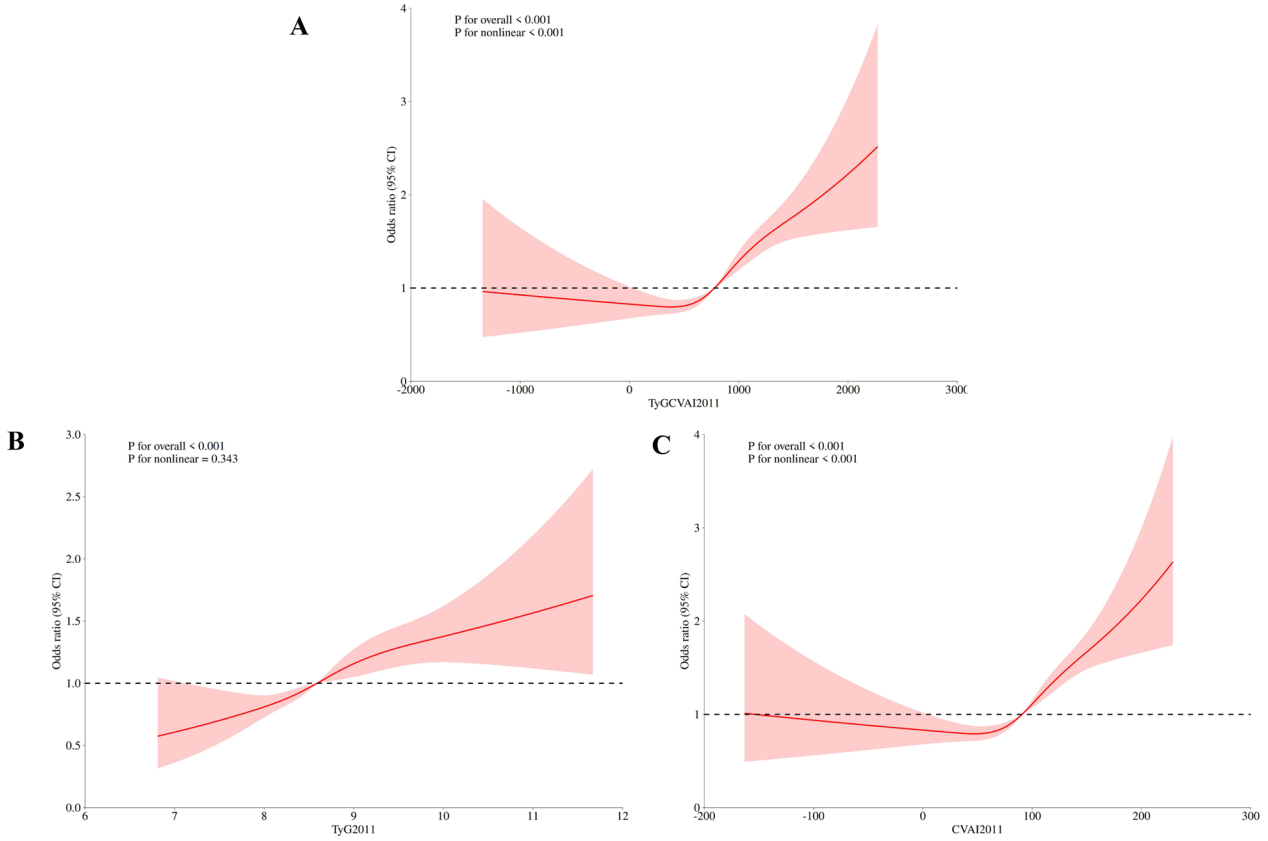


Fig. 3 Restricted cubic splines (RCS) analysis exploring the relationship between TyG-CVAI (**A**), TyG(**B**), CVAI (**C**) and CVD

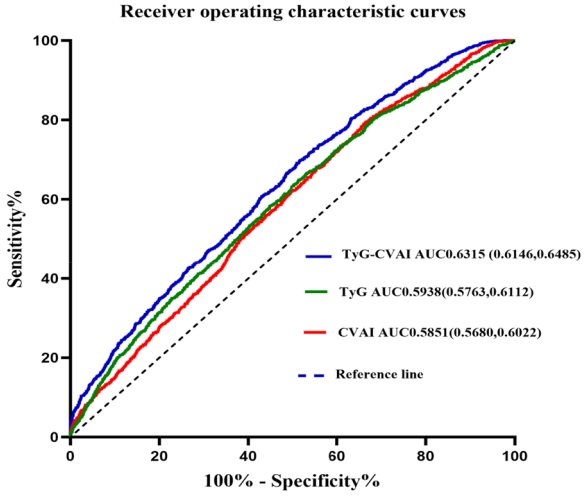


Fig. 4 The ROC curve of TyG-CVAI, TyG and CVAI for predicting new-one-set CVD

fully adjusted Cox model suggests that the pathophysiological pathways captured by the TyG-CVAI extend beyond those of traditional risk factors. The TyG-CVAI index synergistically captures two fundamental pathophysiological processes: insulin resistance (IR) and visceral adiposity. The TyG index, which is derived from fasting triglyceride and glucose levels, is a well-validated surrogate marker of IR [39]. IR promotes endothelial dysfunction [40], oxidative stress [41], and chronic low-grade inflammation [42] through mechanisms involving advanced glycation end products [43] and activation of the protein kinase C [44] and NADPH oxidase pathways [45], which together accelerate atherosclerosis [46]. Gleichzeitig, CVAI, a sex- and age-specific indicator customised for the Chinese population [47], was confirmed to reflect visceral fat accumulation [48]. Visceral adipose tissue acts as an active endocrine organ [49], secreting proinflammatory cytokines and free fatty acids [50] to further exacerbate IR [51], promote macrophage infiltration into vascular walls, and contribute to plaque instability [52]. The integration of the TyG index and CVAI into the TyG-CVAI composite indicator is based on the synergistic pathophysiological roles of IR and visceral adiposity, capturing the interplay between metabolic dysfunction and adverse body fat distribution in CVD development. Therefore, the integration of the TyG index and CVAI into a composite index has been reported to provide a more holistic reflection of the intertwined metabolic and inflammatory pathways that drive stroke pathogenesis [32]. Our results suggest that by synergistically integrating the pathways of insulin resistance and visceral obesity, the TyG-CVAI provides a more holistic

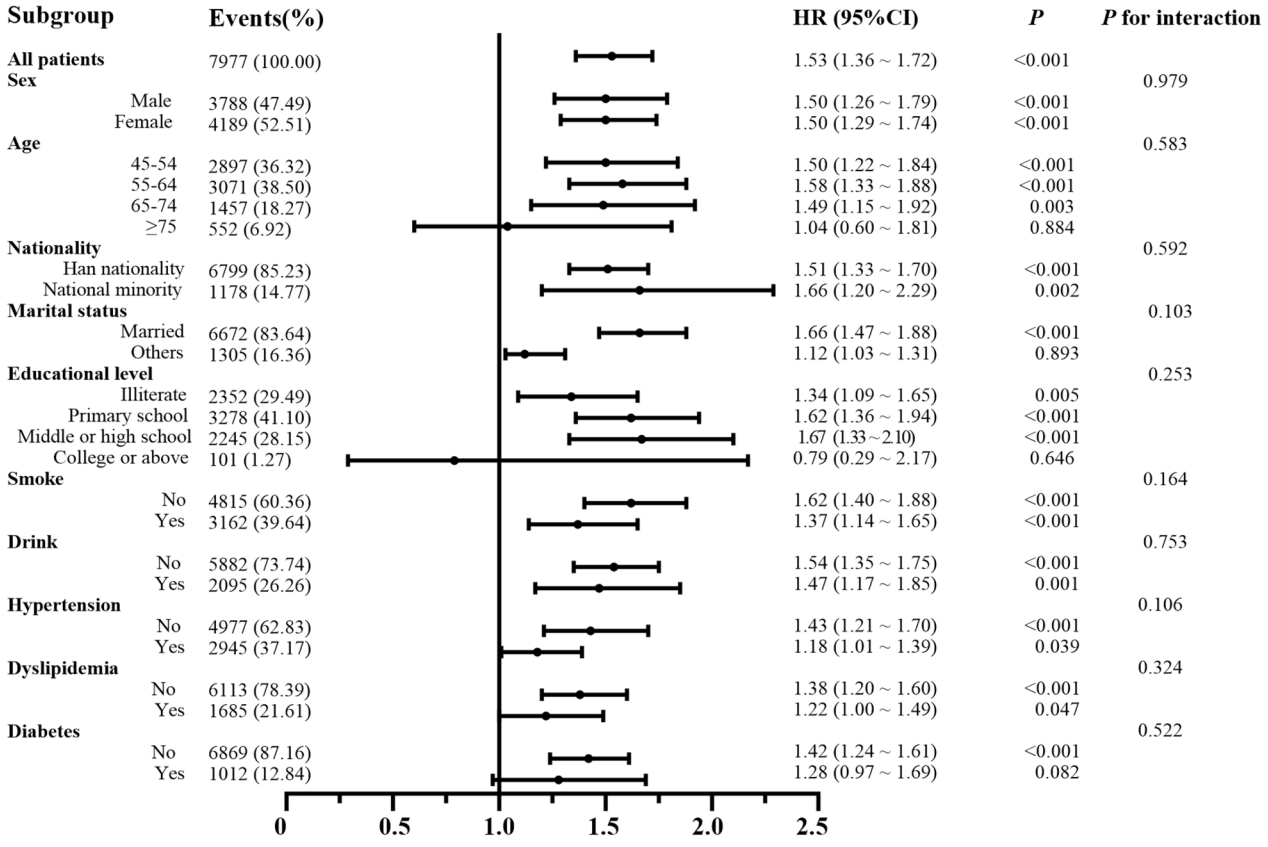


Fig. 5 Subgroup analysis for the TyG-CVAI on CVD risk

reflection of the underlying metabolic dysfunction driving CVD development.

Our findings are consistent with and extend the growing body of evidence supporting the utility of composite indices in the prediction of the cardiovascular risk. For example, a recent analysis of the CHARLS cohort showed that TyG-related indices, such as TyG-BRI (TyG-related body roundness index) and the TyG-weighted-adjusted waist index (TyG-WWI), were significantly associated with incident stroke, with the TyG-CVAI showing the strongest predictive performance (AUC = 0.62) [31]. Similarly, another CHARLS-based study reported a U-shaped relationship between the cumulative TyG-CVAI and stroke risk [32], which is consistent with our observation of nonlinear associations. These consistent findings obtained from independent analyses of the same cohort highlight the robustness of the TyG-CVAI as a predictor of cerebrovascular and cardiovascular outcomes.

However, comparisons with other integrated indices reveal both similarities and differences. CHARLS data showed that the TyG index alone exhibited the highest C index (0.742) for predicting CVD, while the CVAI showed the highest hazard ratio (1.13 (1.08–1.19)) among the nine IR surrogates [53]. Another analysis based on data from 12 communities in eastern China confirmed that CVAI was independently associated with a higher incidence of coronary heart disease (CHD) and was the most powerful predictor of incidence in Chinese populations, indicating its superiority to TyG [54]. A recent study attempted to combine the TyG index with the CVAI to predict the CVD risk. In this study, the median values of the TyG index and CVAI were used as cut-off points, and the patients were classified into 'low' and 'high' groups. The participants were subsequently divided into four groups: low TyG and low CVAI, low TyG and high CVAI, high TyG and low CVAI, and high TyG and high CVAI. This analysis confirmed that the interaction between the TyG index and CVAI played a complex role in the CVD risk, with a potential antagonistic effect between a high TyG and high CVAI on the CVD risk [26]. These findings suggest that while the TyG index or CVAI is a powerful standalone indicator of CVD, the combination of the above two indices may offer complementary risk stratification capabilities, particularly in populations with a high incidence of abdominal obesity, such as middle-aged and elderly individuals. Importantly, our study innovatively advances the field by evaluating TyG-CVAI exposure, which accounts for the metabolic–visceral adiposity burden and shows a stronger predictive

ability than the individual components (TyG or CVAI). Furthermore, we uniquely identified a significant nonlinear threshold effect, quantifying a critical inflection point (TyG-CVAI = 778.62) beyond which the CVD risk increases sharply. This study provides a potential quantitative anchor for risk stratification that complements the findings of longitudinal trajectories of related indices [33].

The potential mechanism by which the TyG-CVAI can predict the CVD risk likely involves the vicious cycle between IR and the accumulation of visceral fat. IR contributes to metabolic abnormalities such as dyslipidaemia and hyperglycaemia, which promote endothelial dysfunction and atherosclerosis. Visceral adipose tissue acts as an active endocrine organ, releasing proinflammatory cytokines and free fatty acids that further exacerbate IR and systemic inflammation, thereby accelerating atherosclerotic progression.

From a practical perspective, the TyG-CVAI represents a cost-effective and accessible tool for CVD risk stratification, particularly in resource-limited primary care settings. The index can be derived from routine biochemical (triglyceride, glucose, and HDL-C levels) and anthropometric (waist circumference, BMI, age, and sex) measurements, which cost approximately USD 20 per assessment. Its superior predictive performance supports its use to identify high-risk individuals who might be missed with the use of conventional risk scores.

Several limitations should be considered when interpreting our results. First, the definition of CVD events relies primarily on self-reported physician diagnoses, which can introduce misclassification bias because of underreporting of asymptomatic cases or recall errors. However, this method has been widely used in previous studies and is considered acceptable for large cohort surveillance. Second, as an observational study, residual confounding from unmeasured factors (e.g., dietary patterns, genetic predispositions, and environmental influences) cannot be entirely excluded, despite our extensive adjustment for known confounders. Third, the generalizability of our findings may be limited to middle-aged and older Chinese adults, and external validation in younger or other ethnic populations is needed, especially given that the CVAI formula was specifically developed for Chinese adults. Finally, while we demonstrated the predictive value of the TyG-CVAI, we were unable to compare it directly with established CVD risk scores such as the Framingham risk score because of the lack of key variables in the CHARLS dataset.

Conclusions

In this large, national cohort study, we showed that a higher TyG-CVAI was independently and nonlinearly associated with an increased risk of new-onset CVD, even after comprehensive adjustment for traditional cardiometabolic risk factors. The TyG-CVAI exhibited greater predictive performance for CVD than its individual components: TyG and CVAI. These findings suggest that the TyG-CVAI, a simple and integrated biomarker that combines insulin resistance and visceral adiposity, could serve as a valuable and practical tool for the early identification of high-risk individuals, potentially helping to prevent CVD in the general population.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12933-025-03063-2.

Supplementary material 1

Supplementary material 2

Supplementary material 3

Supplementary material 4

Supplementary material 5

Supplementary material 6

Supplementary material 7

Supplementary material 8

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Author contributions

WLZ and ZYM were responsible for the conception and design of the study. WLZ, YL and XHZ were responsible for software support, statistical support, analysis, and interpretation of the study. WLZ and ZYM drafted and revised the manuscript. WLZ provided fund support. All authors have read and approved the final manuscript.

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Data availability

The CHARLS dataset utilized in this study is publicly available from the official CHARLS website https://charls.pku.edu.cn/.

Declarations

Ethics approval and consent to participate

The Peking University Biomedical Ethics Review Board granted this program's ethical approval, with the approval numbers IRB00001052-11015 and IRB00001052-11014.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Geriatrics Medicine, First Affiliated Hospital of Xi'an Jiaotong University, No.277, Yanta West Road, Xi'an 710061, People's Republic of China

[2]Department of Cardiovascular Medicine, First Affiliated Hospital of Xi'an Jiaotong University and Key Laboratory of Environment and Genes Related to Diseases, Ministry of Education, Xi'an, People's Republic of China

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