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Association between TG/HDL-C and hypertension in Chinese middle-aged and older adults: findings from CHARLS

Lerui Wang[1,2†], Xiao Chen[1,2†], Weicheng Lai[3†], Junxi Liu[4] and Boda Zhou[1*]

Abstract

Background The triacylglycerol to high-density lipoprotein cholesterol ratio (TG/HDL-C) has been recognized as one of the risk factors for cardiovascular diseases, insulin resistance and metabolic syndrome. We aimed to investigate the relationship between TG/HDL-C and hypertension in a Chinese middle-aged and elderly population.

Methods We used data from the CHARLS database 2011–2018 to explore the relationship between TG/HDL-C and hypertension through cross-sectional and longitudinal studies. Hypertension was identified by self-report or taking anti-hypertensive medications. Participants aged below 45, or with missing data on TG/HDL-C or hypertension record, or taking lipid-lowering medication were excluded. Participants were divided into three or two groups based on triplets TG/HDL-C and median TG/HDL-C in cross-sectional and longitudinal analysis. Multivariate logistic regression analysis, subgroup analysis, and restricted cubic splines were used in statistics.

Results In the cross-sectional analysis, a total of 12,824 participants were included, after adjusting for potential confounders, there was a significant association between higher TG/HDL-C and increased prevalence of hypertension (OR = 1.86, 95% CI: 1.65–2.09, $p < 0.001$), systolic blood pressure (SBP) above 140 mmHg (OR = 1.37, 95% CI: 1.25–1.50, $p < 0.0001$), diastolic blood pressure (DBP) above 90 mmHg (OR = 1.47, 95% CI: 1.29–1.67, $p < 0.0001$), and pulse pressure (PP) above 60 mmHg (OR = 1.17, 95% CI: 1.07–1.29, $p < 0.0011$). The longitudinal analysis included 7909 participants, there was a significant association between higher TG/HDL-C and increased incidence of hypertension (OR = 1.51, 95% CI: 1.32–1.73,$p < 0.001$). Restricted cubic splines show nonlinear relationship between TG/HDL-C and hypertension.

Conclusion These results demonstrated significant positive association between TG/HDL-C and the prevalence & incidence of hypertension, in a nationwide representative middle-aged and elderly population in China.

Keywords CHARLS, Hypertension, TG/HDL-C, Nonlinear, Elderly

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Introduction

Hypertension is the leading cause of cardiovascular disease and premature death worldwide [1]. Unfortunately, only half of people with hypertension know they have the disease, and only about one in five have hypertension under effective control [2, 3]. Hypertension and its complications affect more than 1 billion people worldwide, which is a major public health problem [4]. Known risk factors for hypertension include high density lipoprotein cholesterol (HDL), triglycerides (TG), body mass index (BMI), alcohol dependence, insomnia, and education level [5]. Of them, dyslipidemia may promote hypertension progression [6], while elevated plasma triglyceride (TG) and reduced high HDL-C was associated with Metabolic Syndrome (MetS) and cardiovascular disease (CVD). The TG/HDL-C ratio (TG/HDL-C) was associated with insulin resistance (IR) [7], as well as significantly increased five-year risk of death from chronic heart failure [8], thus TG/HDL-C has been proposed as novel biomarker for predicting the risk of several CVDs [9], as well as cardiometabolic diseases [10].

Previous studies have mostly focused on TG/HDL-C to predict in-hospital mortality, while only one study in China has examined the association between TG/HDL-C and the prevalence of hypertension in adults (18 years and older) using a cross-sectional study approach. The results showed that TG/HDL-C was positively associated with an increased risk of hypertension in Chinese adults, especially in women and those with normal BMI [11]. Another study showed TG/HDL-C significantly correlated with progression of arterial stiffness in hypertensive population [12]. However, the relationship between TG/HDL-C and hypertension has not been examined through cross-sectional and longitudinal analysis in a middle-aged and elderly population, who were at high risk for CVDs and cardiometabolic diseases. Thus the aim of our study was to investigate the relationship between TG/HDL-C and hypertension through cross-sectional and longitudinal analysis of CHARLS.

Methods

Study design and population

This study used data from the China Health and Retirement Longitudinal Study (CHARLS), a nationwide representative population-based longitudinal study. The CHARLS baseline survey (Wave 1) was conducted from June 2011 to March 2012, which included a total of 17,708 participants. A multi-stage probability sampling method was used to draw study participants from 150 counties (districts) and 450 villages (communities) in China. Up to now, the baseline participants has been followed up every two years (Wave 2 in 2013, Wave 3 in 2015, Wave 4 in 2018 and Wave 5 in 2020).

In the cross-sectional analysis, we included participants in the 2011 or 2015 wave with non-duplicated IDs. Inclusion criteria include (1) complete TG/HDL-C value, and (2) complete hypertension diagnostic data. Reasons for exclusion included (1) missing age information or age below 45 years, or (2) missing TG/HDL-C value or hypertension diagnostic data in both 2011 and 2015, or (3) missing major covariables (diabetes mellitus, heart disease, stroke, drinking status), or (4) taking lipid-lowering medication. Detailed inclusion and exclusion criteria are shown in Fig. 1.

In the longitudinal analysis, we selected participants without hypertension in 2011, focusing on the incidence of hypertension during followup. Inclusion criteria include (1) complete TG/HDL-C value in 2011, and (2) complete hypertension diagnostic data during followup. Exclusion criteria included (1) missing age information or age under 45 years, or (2) missing TG/HDL-C value or hypertension diagnostic data, or (3) those who already had hypertension in 2011, or (4) taking lipid-lowering medication. Detailed inclusion and exclusion criteria are shown in Fig. 2.

Ethical approval

CHARLS project and the protocol for biomarker sample collection were approved by the Biomedical Ethics Review Committee of Peking University (IRB00001052-11014) and the Institutional Review Board of the National School of Development at Peking University (IRB00001052-11015), with informed consent obtained from all participants.

Data collection and potential covariates

Data collection was performed by professionally trained personnel via structured questionnaires [13]. When choosing covariates, we first identified indicators that were correlated with the outcome variable through univariate analysis and then corrected them as covariates in multivariate logistic regression. And based on previous studies [14], our covariates included demographic characteristics, health behaviors and health-related factors, chronic diseases, physical examination data, and hematological screening indicators. Demographic covariates included sex, age, education level, area of residence (rural or urban), health behaviors and health-related factors included smoking status, drinking status, chronic diseases (hypertension, diabetes mellitus, heart disease, stroke), physical examination data (SBP-systolic blood pressure, DBP-diastolic blood pressure), BMI, blood test results (BUN-blood urea nitrogen, SUA-serum uric acid, Scr-serum creatinine, TC-total cholesterol, FBG-fasting blood glucose, Cysc-Cystatin C, HbA1c-glycosylated hemoglobin).

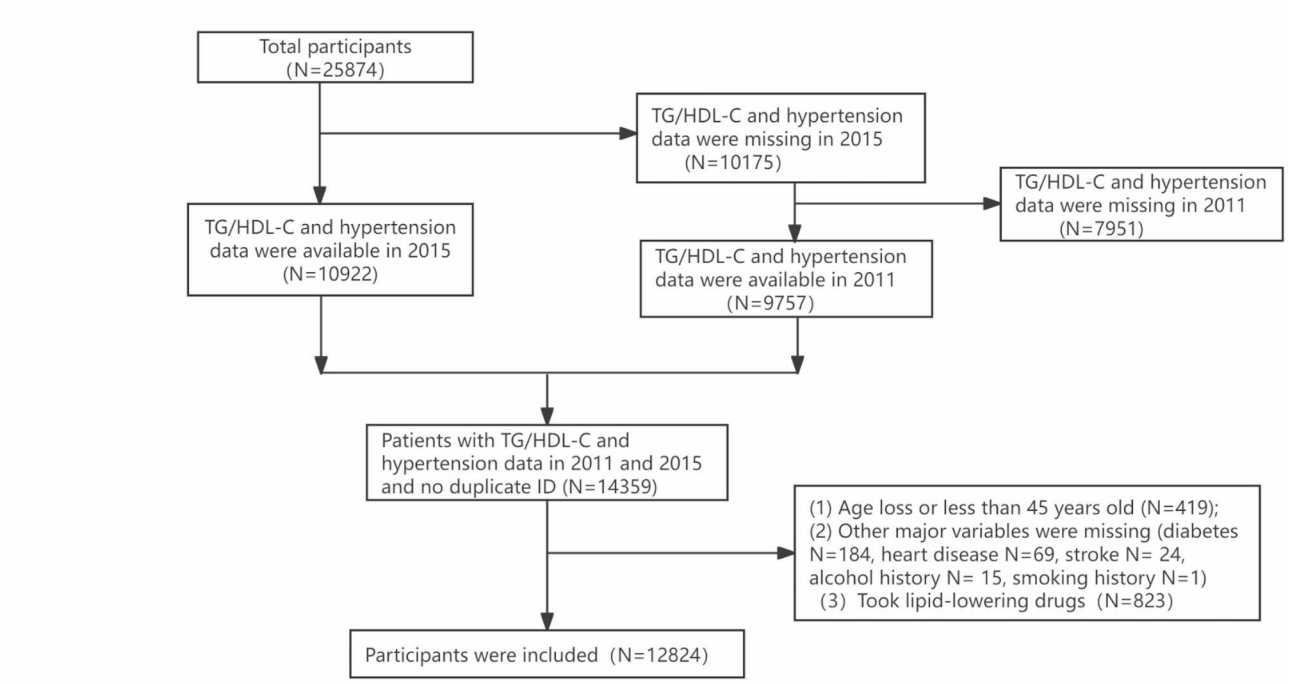

Fig. 1 Research flowchart for the cross-sectional analysis

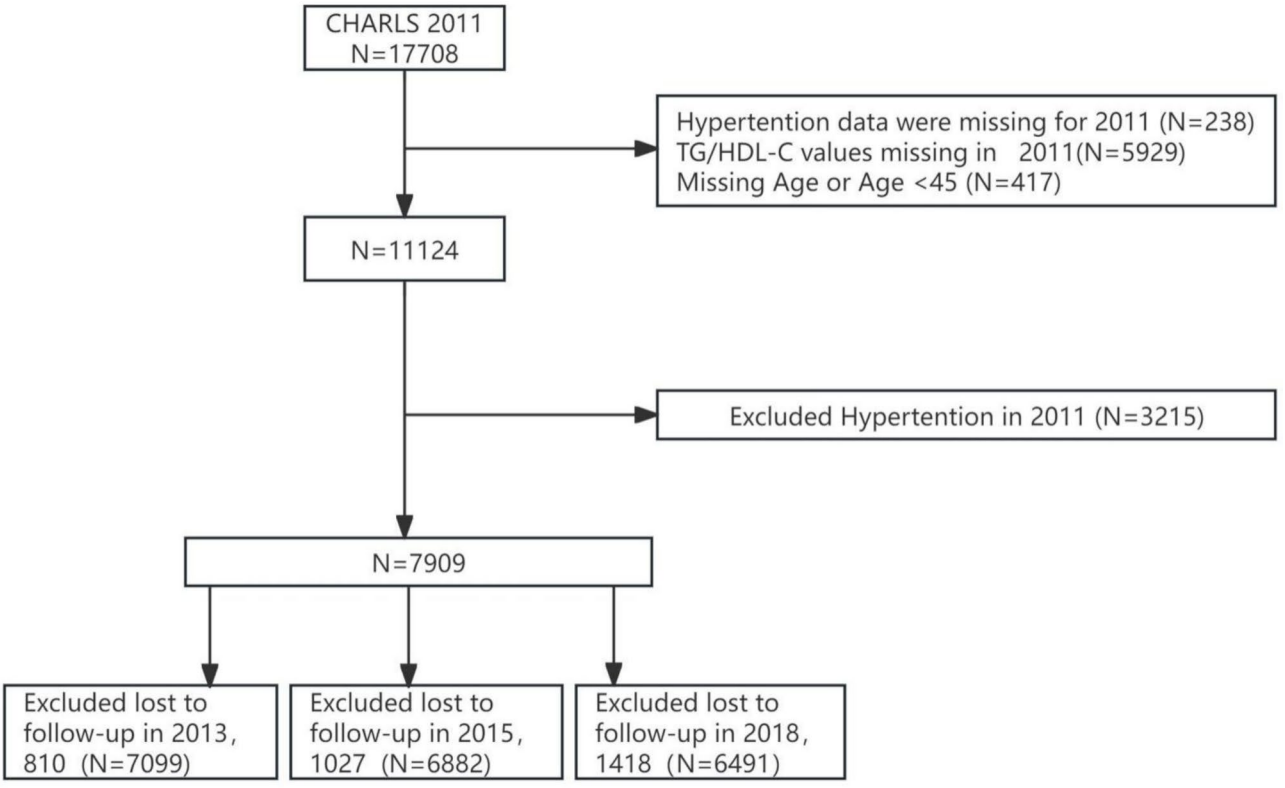

Fig. 2 Research flowchart for the longitudinal analysis

Measurement and grouping of TG/HDL-C

Blood samples were collected by medical staff at the Chinese Center for Disease Control and Prevention (CDC) after an overnight fast. These samples were analyzed in the central laboratory to measure various biomarkers of TG, HDL-C, and low-density lipoprotein cholesterol (LDL-C) using enzymatic colorimetric assays. In this study, the TG/HDL-C ratio was calculated using TG (mg/dL) divided by HDL-C (mg/dL). In different studies, TG/HDL-C was divided into three and two groups based on triplicates and medians, respectively. In the cross-sectional analysis, participants were divided into three groups based on triplets TG/HDL-C in cross-sectional analysis, Q1 group: (TG/HDL-C ≤ 1.58);Q2 group: (1.58 < TG/HDL-C < 2.93);Q3 group: (TG/HDL-C ≥ 2.93). In the longitudinal analysis, participants were divided into two groups based on median TG/HDL-C in longitudinal analysis (1.97), low level group: (TG/HDL-C < 1.97); high level group: (TG/HDL-C ≥ 1.97).

Definition of hypertension

Hypertension was defined by one of the following criteria: ① positive answer to question "Have you ever been diagnosed with hypertension by a doctor?" or ② positive answer to question "Are you currently taking any treatments to manage or control your hypertension, such as Traditional Chinese Medicine or Western modern medicine?". We also tried to add systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg in the diagnosis of hypertension with the results presented in Table S1, S2.

Statistical analysis

Quantitative data were expressed as medians and 95% confidence intervals, and differences between TG/HDL-C groups were assessed using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages, and statistical significance was tested using the Pearson chi-square test. To address the missing clinical variables, multiple interpolation via chained equations was used for modeling purposes (Figure S3, Table S4-S7). Multivariate binary logistic regression was used to analyze the association between different TG/HDL-C groups and hypertension. Results were expressed as odds ratios (OR) and their 95% confidence intervals (CI). Three models were used: model 1 was a crude model, uncorrected for covariates; model 2 was corrected for age and sex; and model 3 was further corrected for education level, marital status, region of residence, health behaviors, and health-related factors, including smoking status, drinking status, chronic diseases (diabetes mellitus, heart disease, stroke), and blood test results (Scr, FBG, Cysc, and HbA1c). We also tested for multicollinearity by calculating the variance inflation factor (VIF) for each covariate, and found that there is no covariance between covariates (Figure S2). To explore the nonlinear relationship between TG/HDL-C and hypertension, we used the restricted cubic spline (RCS) model, setting 4 nodes (knots) located in the 5th, 35th, 65th, and 95th percentiles of the TG/HDL-C distribution and using a threshold effect model to analyze the folds and compare the likelihood ratio tests of the linear and spline models (Table S3); further stratified analyses were performed to investigate the effects of potential changes in the following factors: sex, age, place of residence, drinking status, and smoking status. The impact of this modification may have been assessed by modeling the interaction of stratified covariates using TG/HDL-C. In this study, a difference with a two-sided P less than 0.05 was considered statistically significant. Statistical analyses were performed using R software (version 4.3.1) and Empower 6.0.

Results

Baseline characteristics

The cross-sectional analysis enrolled a total of 12,824 participants. As shown in Table 1, the median age of participants was 59.00 years, with 47.31% males ($N = 6,067$) and 52.69% females ($N = 6757$). Hypertension was present in 27.34% ($N = 3506$) participants. Statistical analysis revealed significant differences in age, sex, marital status, education level, area of residence, drinking status, smoking status between dichotomized TG/HDL-C groups (all p values < 0.05). Notably, all selected serum biochemical indicators showed significant differences between the dichotomized TG/HDL-C groups (all p values < 0.05). Specifically, compared to low TG/HDL-C group, participants in high TG/HDL-C group exhibited higher SBP, DBP, pulse pressure (PP), TC, Scr, SUA, FBG, and HbA1c, highlighting the close link between TG/HDL-C levels and various CVD risk factors.

Association between TG/HDL-C and prevalence of hypertension

After adjusting for potential covariates, when TG/HDL-C was used as a continuous or categorical variable, the prevalence of hypertension tended to increase with increased TG/HDL-C level/ tertile (Table 2, Table S1, S2). Compared to TG/HDL-C Q1 group, TG/HDL-C Q3 group showed significantly higher hypertension prevalence (OR = 1.86, 95% CI: 1.65–2.09, $p < 0.001$). These results collectively confirmed significant positive association between TG/HDL-Cand prevalence of hypertension.

Association between TG/HDL-C and blood pressure measurement

Multivariate logistics regression analysis between TG/HDL-C and blood pressure measurement showed that

Table 1 Baseline characteristics of study participants according to TG/HDL-C in the cross-sectional analysis

Characteristics	Total ($n=12824$)	Quartiles of TG/HDL-C		*P*-value
		Low TG/HDL-C group ($n=6412$)	High TG/HDL-C group ($n=6412$)	
Age, M (Q_1, Q_3), years	59.00 (52.00, 66.00)	59.00 (52.00, 67.00)	58.00 (51.00, 65.00)	**< 0.001**
sex, N and percentage (%)				**< 0.001**
Male	6067 (47.31)	3177 (49.55)	2890 (45.07)	
Female	6757 (52.69)	3235 (50.45)	3522 (54.93)	
Education, N and percentage (%)				**< 0.001**
Primary school or lower	11,538 (89.97)	5842 (91.11)	5696 (88.83)	
Secondary school	1122 (8.75)	511 (7.97)	611 (9.53)	
Higher	164 (1.28)	59 (0.92)	105 (1.64)	
Area of residence, N and percentage (%)				**< 0.001**
Rural areas	4658 (36.32)	2034 (31.72)	2624 (40.92)	
Urban areas	8166 (63.68)	4378 (68.28)	3788 (59.08)	
Drinkng status, N and percentage (%)				**< 0.001**
No	7487 (58.38)	3602 (56.18)	3885 (60.59)	
Yes	5337 (41.62)	2810 (43.82)	2527 (39.41)	
Smoking status, N and percentage (%)				**< 0.001**
No	7539 (58.79)	3665 (57.16)	3874 (60.42)	
Yes	5285 (41.21)	2747 (42.84)	2538 (39.58)	
Hypertention, N and percentage (%)				**< 0.001**
No	9318 (72.66)	5022 (78.32)	4296 (67.00)	
Yes	3506 (27.34)	1390 (21.68)	2116 (33.00)	
Diebetes or hyperglycemia, N and percentage (%)				**< 0.001**
No	12,051 (93.97)	6162 (96.10)	5889 (91.84)	
Yes	773 (6.03)	250 (3.90)	523 (8.16)	
Heart disease, N and percentage (%)				**< 0.001**
No	11,249 (87.72)	5754 (89.74)	5495 (85.70)	
Yes	1575 (12.28)	658 (10.26)	917 (14.30)	
Stroke, N and percentage (%)				**0.001**
No	12,504 (97.50)	6281 (97.96)	6223 (97.05)	
Yes	320 (2.50)	131 (2.04)	189 (2.95)	
BMI group, N and percentage (%)				**< 0.001**
< 18.5	854 (6.75)	647 (10.21)	207 (3.27)	
18.5 ~ 23	5259 (41.55)	3228 (50.96)	2031 (32.13)	
23 ~ 25	2589 (20.46)	1212 (19.13)	1377 (21.78)	
≥ 25	3954 (31.24)	1248 (19.70)	2706 (42.81)	
SBP, M (Q_1, Q_3) mmHg	126.00 (113.00, 140.50)	124.00 (111.50, 138.00)	127.50 (115.00, 142.50)	**< 0.001**
DBP, M (Q_1, Q_3) mmHg	74.00 (66.50, 82.50)	72.50 (65.50, 81.00)	75.50 (68.00, 84.00)	**< 0.001**
PP, M (Q_1, Q_3) mmHg	50.50 (43.00, 61.00)	50.00 (43.00, 60.50)	51.00 (43.50, 61.00)	**< 0.001**
BMI, M (Q_1, Q_3) kg/m^2	23.15 (20.85, 25.76)	22.06 (20.06, 24.35)	24.38 (22.01, 26.86)	**< 0.001**
BUN, M (Q_1, Q_3) mg/dl	15.10 (12.55, 18.15)	15.52 (12.89, 18.71)	14.59 (12.32, 17.53)	**< 0.001**
FPG, M (Q_1, Q_3) mg/dl	100.26 (91.89, 111.19)	97.38 (90.09, 106.38)	102.78 (93.96, 116.28)	**< 0.001**
Scr, M (Q_1, Q_3) mg/dl	0.76 (0.66, 0.89)	0.76 (0.65, 0.88)	0.77 (0.66, 0.90)	**< 0.001**
TC, M (Q_1, Q_3) mg/dl	187.11 (164.69, 211.86)	183.25 (161.99, 206.83)	190.98 (167.57, 216.50)	**< 0.001**
CRP, M (Q_1, Q_3) mg/dl	1.11 (0.60, 2.30)	0.90 (0.50, 1.90)	1.39 (0.73, 2.69)	**< 0.001**
HbA1c, M (Q_1, Q_3) mg/dl	5.30 (5.00, 5.70)	5.30 (5.00, 5.60)	5.40 (5.00, 5.80)	**< 0.001**
SUA, M (Q_1, Q_3) mg/dl	4.43 (3.69, 5.37)	4.28 (3.55, 5.10)	4.63 (3.82, 5.60)	**< 0.001**
CystatinC, M (Q_1, Q_3) mg/dl	0.93 (0.79, 1.07)	0.93 (0.80, 1.08)	0.92 (0.79, 1.06)	**< 0.001**
SBPgroup, N and percentage (%)				**< 0.001**
< 140mmHg	10,041 (73.58)	5212 (76.39)	4829 (70.76)	
≥ 140mmHg	3606 (26.42)	1611 (23.61)	1995 (29.24)	
DBPgroup, N and percentage (%)				**< 0.001**
< 90mmHg	12,048 (88.28)	6177 (90.53)	5871 (86.03)	

Table 1 (continued)

Characteristics	Total ($n=12824$)	Quartiles of TG/HDL-C		*P*-value
		Low TG/HDL-C group ($n=6412$)	High TG/HDL-C group ($n=6412$)	
≥ 90mmHg	1599 (11.72)	646 (9.47)	953 (13.97)	
PPgroup, N and percentage (%)				**0.028**
< 60mmHg	9932 (72.78)	5038 (73.84)	4894 (71.72)	
≥ 60mmHg	3715 (27.22)	1785 (26.16)	1930 (28.28)	

Table 2 Multivariate regression analysis of the association between TG/HDL-C and hypertention prevalence

Model	TG/HDL-C Group [OR(95%CI)]					*P*-value
	TG/HDL-C	*P*-value	TG/HDL-C Q1 ($n=4232$)	TG/HDL-C Q2 ($n=4360$)	TG/HDL-C Q3 ($n=4232$)	
Model 1	1.04 (1.03,1.05)	**< 0.001**	1.000(Reference)	1.40 (1.26 ~ 1.54)	2.08 (1.88 ~ 2.29)	**< 0.001**
Model 2	1.04 (1.03, 1.05)	**< 0.001**	1.000(Reference)	1.41 (1.28 ~ 1.57)	2.23 (2.02 ~ 2.46)	**< 0.001**
Model 3	1.04 (1.03, 1.06)	**< 0.001**	1.000(Reference)	1.26 (1.13 ~ 1.42)	1.86 (1.65 ~ 2.09)	**< 0.001**

OR: Odds Ratio, CI: Confidence Interval

Model1: Crude

Model2: Adjust: age, sex

Model3: Adjust: age, sex, education, residence, diabetes, heart disease, stroke, drinkng status, smoking status, marital status, BMI, BUN, FBG, Scr, HbA1c, CystatinC

Table 3 Multivariate regression analysis of TG/HDL-C status and blood pressure measurement

Model	TG/HDL-C Group [OR(95%CI)]				
	TG/HDL-C	*P*-value	Low TG/HDL-C group ($N=6412$)	The high TG/HDL-C group ($N=6412$)	*P*-value
SBP ≥ 140 mmHg					
Model 1	1.02 (1.01,1.03)	**< 0.0001**	1.000(Reference)	1.33 (1.23, 1.44)	**< 0.0001**
Model 2	1.03(1.02,1.04)	**< 0.0001**	1.000(Reference)	1.43 (1.32, 1.55)	**< 0.0001**
Model 3	1.03(1.02,1.05)	**< 0.0001**	1.000(Reference)	1.37 (1.25, 1.50)	**< 0.0001**
DBP ≥ 90 mmHg					
Model 1	1.03 (1.02,1.04)	**< 0.0001**	1.000(Reference)	1.53 (1.36, 1.70)	**< 0.0001**
Model 2	1.03(1.02,1.04)	**< 0.0001**	1.000(Reference)	1.53 (1.36, 1.71)	**< 0.0001**
Model 3	1.04 (1.02,1.05)	**< 0.0001**	1.000(Reference)	1.47 (1.29, 1.67)	**< 0.0001**
PP ≥ 60mmHg					
Model 1	1.01(1.0,1.02)	**0.004**	1.000(Reference)	1.09 (1.01, 1.18)	**0.0283**
Model 2	1.02(1.01,1.03)	**< 0.0001**	1.000(Reference)	1.21 (1.11, 1.31)	**< 0.0001**
Model 3	1.02 (1.01,1.04)	**< 0.0001**	1.000(Reference)	1.17 (1.07, 1.29)	**0.0011**

OR: Odds Ratio, CI: Confidence Interval

Model1: Crude

Model2: Adjust: age, sex

Model3: Adjust: age, sex, education, residence, diabetes, heart disease, stroke, drinkng status, smoking status, marital status, BMI, BUN, FBG, Scr, HbA1c, CystatinC

each unit increase in TG/HDL-C was significantly associated with SBP ≥ 140 mmHg (OR = 1.03, 95% CI: 1.02–1.05, $p < 0.0001$), DBP ≥ 90 mmHg (OR = 1.04, 95% CI: 1.02–1.05, $p < 0.0001$), PP ≥ 60 mmHg (OR = 1.02, 95% CI: 1.01–1.04,$p < 0.0001$), after adjusting potential covariates. When TG/HDL-C was analyzed as a categorical variable, after adjusting potential covariates, high TG/HDL-C group was significantly associated with increased risk of SBP ≥ 140 mmHg (OR = 1.37, 95% CI: 1.25–1.50, $p < 0.0001$), DBP ≥ 90 mmHg (OR = 1.47, 95% CI: 1.29–1.67, $p < 0.0001$), PP ≥ 60 mmHg (OR = 1.17, 95% CI: 1.07–1.29, $p = 0.0011$), compared to low TG/HDL-C group. (Table 3)

Nonlinear relationship between TG/HDL-Cand hypertension prevalence

We performed a restricted cubic spline (RCS) to flexibly model and visualize the association between TG/HDL-C and the prevalence of hypertension. After adjusting for potential covariates, a nonlinear trend was observed in the association between the TG/HDL-C and the prevalence of hypertension (P for nonlinear < 0.001) (Fig. 3). We found a threshold effect for the association between TG/HDL-C and hypertention by threshold effect analysis with a fold point of 4.098 (P for likelihood test < 0.001). When TG/HDL-C was lower than 4.098, TG/HDL-C were positively associated [OR (95%CI): 1.25 (1.18–1.33)]

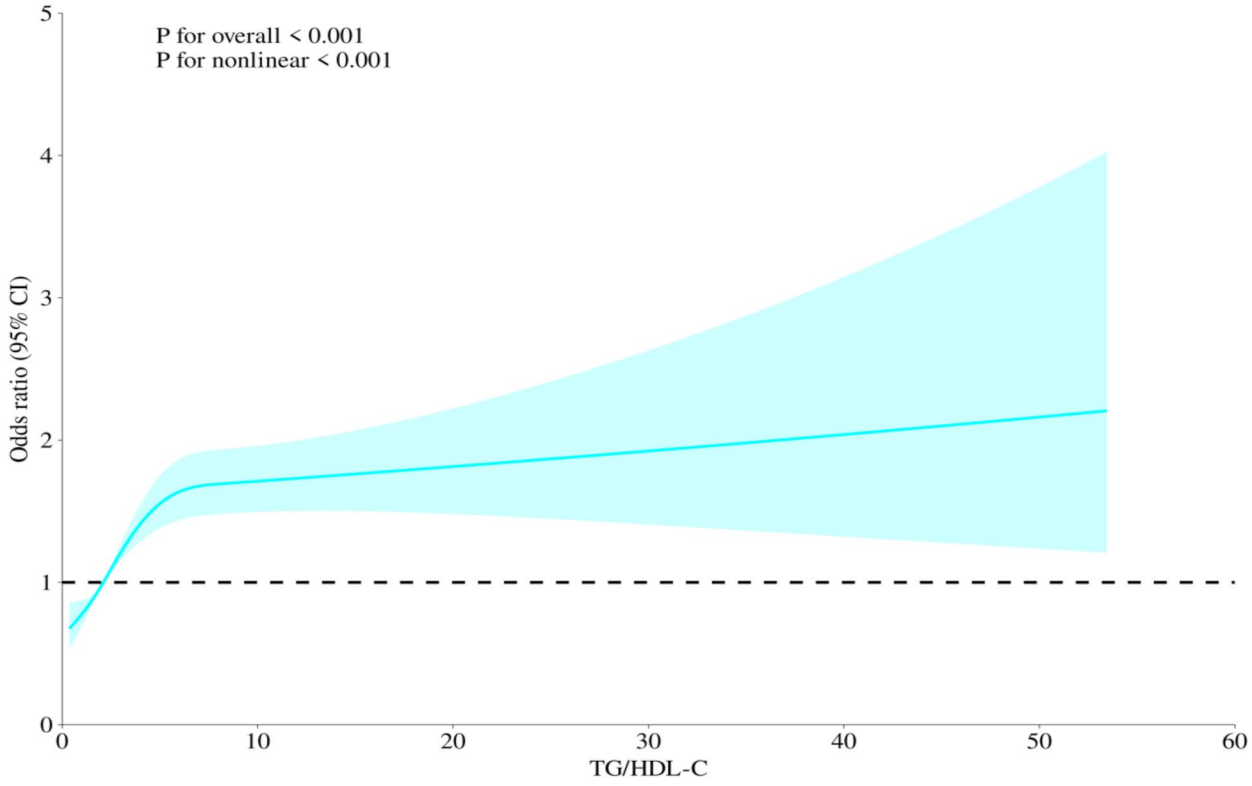

Fig. 3 Restricted cubic spline curve analysis between TG/HDL-C and hypertension prevalence. Results are presented as OR (95% CI).The solid line in the figure represents the OR, and the shaded area indicates the 95% confidence interval. BMI, body mass index; BUN, blood urea nitrogen; FPG, fasting plasma glucose; Scr, serum creatinine; HbA1c, glycated hemoglobin; CysC, Cystatin C; OR, odds ratio; CI, confidence interval

with hypertention, and when TG/HDL-C was higher than 4.098, no association was found between TG/HDL-C and hypertention.(Table S3).

Subgroup analysis

After adjusting for potential covariables, we performed stratified analysis to validate the associations betweenTG/HDL-C and hypertension prevalence in certain subgroups. The subgroups were categorized according to age, sex, area of residence, drinking, smoking to explore the association between TG/HDL-C and hypertension. The results of the subgroup analyses showed an overall consistent association between TG/HDL-C and hypertension prevalence. The interaction of TG/HDL-C with sex and smoking was significant ($p < 0.05$). Specificly, we found the association of TG/HDL-C with hypertension prevalence was more prominent in female participants ($p = 0.032$); and in non-smoking participants ($p = 0.029$) (Fig. 4).

Analysis of the association between TG/HDL-C and the incidence of hypertension

In the longitudinal study, participants were divided into two groups based on median TG/HDL-C: low TG/HDL-C group: (TG/HDL-C < 1.97) and high TG/HDL-C group: (TG/HDL-C ≥ 1.97). The participants who did not suffer from hypertension in 2011 (7909, Fig. 2) were included and followed up for 7 years, a total of 1902 participants had new-onset hypertension during 7 years followup. A multivariate logistic regression was performed to explore the association between TG/HDL-C and hypertension incidence in the longitudinal study. The results showed that higher levels of TG/HDL-C were significantly associated with increased hypertension incidence [OR = 1.51 (1.32, 1.73), $p < 0.001$] (Table 4). Further stratified analyses were performed in different subgroups (Figure S1).

Discussion

Our results show that in this large, representative population of middle-aged and older Chinese adults, significant association between higher TG/HDL-C ratio and prevalence of hypertension, significant association between TG/HDL-C ratio and blood pressure measurements including SBP, DBP, and PP. Nonlinear relationship was found between TG/HDL-C and the prevalence of hypertension. Subgroup analyses showed an overall consistent association between TG/HDL-C and hypertension prevalence. In the longitudinal study, we used the 2011 non-hypertensive participants as baseline, and analyzed the

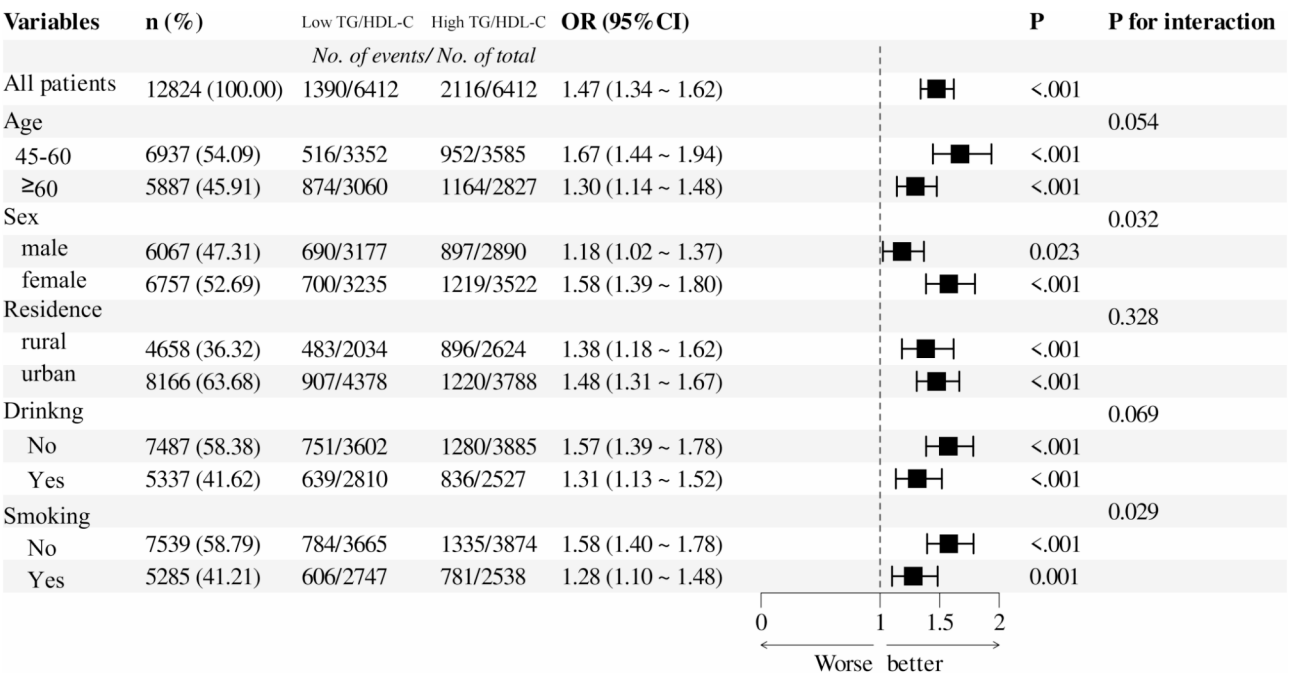

Fig. 4 Association of the TG/HDL-C and hypertension prevalence in different subgroups

Table 4 Multivariate regression annlysis of the association between TG/HDL-C and hypertension incidence

Model	TG/HDL-C Group [OR(95%CI)]		
	LowTG/HDL-C group (TG/HDL-C < 1.97)	High TG/HDL-C group (TG/HDL-C ≥ 1.97)	*P* value
Model 1	1.000(Reference)	1.45 (1.30, 1.61)	**< 0.001**
Model 2	1.000(Reference)	1.46 (1.31,1.62)	**< 0.001**
Model 3	1.000(Reference)	1.51(1.32, 1.73)	**< 0.001**

OR: Odds Ratio, CI: Confidence Interval

Model1: Crude

Model2: Adjust: age, sex

Model3: Adjust: age, sex, education, residence, diabetes, heart disease, stroke, drinkng status, smoking status, marital status, BMI, BUN, FBG, Scr, HbA1c, CystatinC

association between TG/HDL-C levels and new-onset hypertension after 7 years of follow-up, and the findings showed a significant association between high TG/HDL-C level and the increased incidence of hypertension. TG/HDL-C, a new indicator of insulin resistance, is expected to be used as a new serological indicator to assess the risk and extent of hypertension.

TG/HDL-C is not only a marker of lipid metabolism disorders, but also a core indicator of insulin resistance. IR can lead to a variety of diseases, such as hyperglycemia, hypertension and dyslipidemia, all of which are closely related to atherosclerosis by their very nature, as well as causing vasoconstriction, inflammation and thrombosis, which accelerate atherosclerosis [15]. High TG/HDL-C contributes to elevated blood pressure through a combination of increased free fatty acid release, endothelial dysfunction, and activation of the renin-angiotensin system. Pathophysiologic processes in vascular endothelial cells, smooth muscle cells, and macrophages may be enhanced through inflammation, promoting atherosclerosis-associated foam cell and vulnerable plaque formation. In addition, IR may have atherogenic effects through impaired fibrinolysis and dyslipidemia [16]. Free fatty acids increase with lipolysis as TG levels increase and HDL-C levels decrease. Deterioration of insulin sensitivity can lead to increased levels of free fatty acids; induction of tissue oxidative stress can lead to the development of tissue insulin resistance [17]. Previous studies have shown TG/HDL-C as a marker of metabolic syndrome and cardiovascular disease [9], and that disorders of lipid metabolism and consequent dyslipidemia are important causes of plaque formation, with low-density lipoprotein cholesterol (LDL-C) being the main causative factor. However, there is still a risk of CVD by controlling low-density lipoprotein (LDL-C) levels alone, which is attributed to disturbances in other lipid components, changes in the levels of triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C). An analysis of data from the UK Biobank [18] found that elevated baseline TyG indices and TG/HDL-C were associated with an increased risk of CVD in a European population. These associations were mainly mediated by an increased prevalence of dyslipidemia and hypertension. Nie [19] et al. found a significant association between TG/HDL-C and metabolic syndrome. High levels of TG/HDL-C indicate a higher risk of metabolic syndrome in the Chinese elderly population. This further suggests an association between TG/HDL-C and hypertension; A

study [20] on an elderly population also found that higher TG/HDL-C was associated with higher risk of metabolic diseases including hypertension. These studies, which have focused on the association between TG/HDL-C and cardiovascular disease risk or the metabolic syndrome, indirectly reflect the possible association between TG/HDL-C and hypertension.

Compared with previous studies, we utilized the CHARLS database, which is more representative of the middle-aged and elderly population and includes a wider range of regions and populations. Meanwhile, in terms of methodology, we explored the association between TG/HDL-C and the prevalence of hypertension through cross-sectional study, also TG/HDL-C and the incidence of hypertension through longitudinal study. And for the first time, a significant association was also found between TG/HDL-C and blood pressure measurements, indicating TG/HDL-C was also associated with the extent of hypertension.

However, there are some limitations in our study, for example, the study population mainly focuses on Chinese people, and whether it is applicable to other ethnic groups needs to be further verified; in addition, when correcting covariates in the multivariate logistic regression analysis, due to limited information in the database, other factors that may be related to hypertension, such as sodium intake, lifestyle, and stress status, were not included, which may have a certain impact on the results. The results may be affected to a certain extent. Although we explored the association of TG/HDL-C and hypertension prevalence and incidence, the relationship between TG/HDL-C with hypertension-related serious clinical outcomes such as cardiovascular disease, myocardial infarction, or stroke was not further evaluated. The CHARLS database currently lacks detailed documentation of endpoints such as time to event, imaging evidence, and the duration of follow-up may be insufficient to capture a sufficient number of serious events. However, previous studies have shown that elevated TG/HDL-C is significantly associated with an increased risk of atherosclerotic cardiovascular disease [21, 22] (ASCVD), and hypertension is a key driver of ASCVD. Future studies are needed to validate the predictive value of TG/HDL-C for stratified prognosis in hypertensive patients in cohorts with long-term follow-up and precise endpoint assessment, and to explore the underlying mechanisms in conjunction with multi-omics data; integrating such biomarkers with clinical outcomes may provide a more comprehensive risk assessment tool for personalized intervention.

In clinical practice, TG/HDL-C could be used as an easy-to-use, cost-effective adjunct to risk screening: in normotensive populations, elevated TG/HDL-C may suggest prehypertensive risk and the need for enhanced lifestyle interventions such as limiting refined carbohydrate intake and increasing aerobic exercise; in populations with established hypertension, high TG/HDL-C may reflect a subtype of underlying metabolic hypertension, suggesting the need to prioritize drugs that improve insulin sensitivity; also by dynamically monitoring TG/HDL-C changes, the synergistic effect of lipid-lowering or metabolic interventions as well as blood pressure control could be assessed. The present results call for the integration of TG/HDL-C testing in evaluation, especially for hypertensive high-risk groups such as those with obesity and a family history of diabetes, and for the enhancement of the public's awareness of the lipid metabolism-blood pressure axis associations through science education. Prospective intervention studies are needed to validate the effectiveness of TG/HDL-C lowering in preventing hypertension and to explore the value of combining it with novel biomarkers such as lipoprotein subfractions.

Conclusions

These results demonstrated significant positive association between TG/HDL-C and the prevalence & incidence of hypertension, in a nationwide representative middle-aged and elderly population in China. Our study highlights the value of TG/HDL-C, as new serological indicator to assess the risk and extent of hypertension. Monitoring the TG/HDL-C ratio may help identify people at high risk of hypertension and provide early intervention.

Abbreviations

CHARLS	China Health and Retirement Longitudinal Study
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
PP	Pulse pressue
LDL-C	Low-density lipoprotein cholesterol
TC	Total cholesterol
TG	Triglycerides
HDL-C	High-density lipoprotein cholesterol
BMI	Body Mass Index
HbA1c	Glycated hemoglobin
FPG	Fasting plasma glucose
CRP	C-reactive protein
Scr	Serum ceatinine
CysC	Cystatin C
HbA1c	Glycosylated hemoglobin
SUA	Serum Uric Acid
OR	Odds Ratios
CI	Confidence Intervals

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

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Author contributions
LW and BZ designed the study. LW, XC, JL and WL performed the research procedures, including data collection, data analysis, and data interpretation. LW, XC, JL and WL were involved in the preparation of figures, drafting and revising the manuscript. BZ provided academic guidance. All authors read and approved the final version of the manuscript.

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Data availability
The CHARLS data that support the findings of this study are openly available at https://charls.pku.edu.cn/.

Declarations

Ethics approval and consent to participate
The CHARLS protocol was approved by the Biomedical Ethics Review Committee of Peking University (IRB00001052-11014) and the Institutional Review Board of the National School of Development at Peking University (IRB00001052-11015), with informed consent obtained from all participants.

Consent for publication
Not applicable.

Clinical trial number
Not applicable.

Competing interests
The authors declare no competing interests.

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