Original Article

Triglyceride levels and risk of cardiovascular disease and all-cause mortality in Chinese adults younger than 40 years old: a prospective cohort study

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Background: Data on the associations of triglyceride (TG) levels with cardiovascular disease (CVD) and all-cause mortality mainly focused on the middle-aged or elderly population, with limited information available for younger adults. This study aimed to identify such associations among Chinese young adults.

Methods: This study included Chinese adults younger than 40 years free of CVD, cancer, and lipid-lowering agents at baseline in the Kailuan study who were enrolled during 2006 through 2016. All participants were biennially followed up till December 2020. The enzymatic colorimetric method was used to measure baseline fasting TG. Participants were categorized into four groups by quartiles of TG, with the lowest quartile (Q1) as the reference group. The primary outcomes were CVD [composite of myocardial infarction (MI) and ischemic stroke] and all-cause mortality. CVD and mortality risks were estimated with Cox regression models.

Results: A total of 43,882 participants were included. Their mean age was 30.6±5.56 years, and 80.2% were males. During a median follow-up of 11.2 years, 298 CVD events and 345 deaths occurred. The incidences of CVD and all-cause mortality were 0.67 and 0.76 per 1,000 person-years, respectively. Compared with individuals in the lowest quartile (Q1), participants in the highest quartile (Q4) showed a 126% higher risk of developing CVD [adjusted hazard ratio (HR) 2.26; 95% confidence interval (CI): 1.56 to 3.29; P=0.001] and a 61% higher risk of all-cause mortality (adjusted HR 1.61; 95% CI: 1.14 to 2.28; P=0.007). In addition, analyses of CVD subtypes showed that adjusted HRs (Q4 *vs.* Q1) were 3.25 (95% CI: 1.33 to 7.97; P=0.01) for MI, and 1.88 (95% CI: 1.16 to 3.04; P=0.01) for ischemic stroke.

Conclusions: Among Chinese young adults, elevated fasting TG levels were associated with increased CVD and all-cause mortality risks.

Keywords: Triglyceride (TG); myocardial infarction (MI); ischemic stroke; cardiovascular disease (CVD); all-cause mortality

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Introduction

Cardiovascular disease (CVD) is a leading cause of morbidity (1) and mortality (2). Globally, the incidence of CVD has risen rapidly in recent decades (1), especially among younger adults (3). Dyslipidemia is a key modifiable risk factor for CVD and can be modified through lifestyle changes and/or medications (4). As dyslipidemia has a cumulative effect on the risk of CVD, one of the most effective strategies to prevent CVD by slowing the development and progression of plaque is achieving a healthy lipid level as early as possible and maintaining this healthy lipid level throughout life (5).

Low-density lipoprotein cholesterol (LDL-C) is a well-established risk factor for CVD. In recent years, growing evidence suggests that elevated triglyceride (TG) levels may contribute to residual cardiovascular risk, even in patients with controlled LDL-C levels (6). The positive association between higher TG levels and CVD events has been reported in participants of different sexes (7,8) and ethnicities (9,10) as well as in certain high-risk individuals, such as those with abnormal glucose metabolism (11) and hypertension (12). Additionally, studies have reported a predictive role of TG levels for CVD mortality (13) and all-cause mortality (14).

However, prior studies regarding TG-associated risks of CVD, CVD subtypes, and mortality have mainly focused on the middle-aged or elderly population (7-14), with only three studies examining the younger population (15-17). In addition, all three studies focused on South Korean young adults (15-17), with no studies on Chinese young adults. Furthermore, the current guidelines mainly focus on the management of TG among adults aged 40 to 75 years (18). Therefore, it is imperative to carry out studies to determine whether more attention should be given to TG levels among adults younger than 40 years.

In the present study, Chinese adults younger than 40 years were recruited to examine the association of baseline fasting TG levels with CVD and all-cause mortality risks in later life. We present this article in accordance with the STROBE reporting checklist (available at https://cdt.amegroups.com/article/view/10.21037/cdt-23-412/rc).

Highlight box

Key findings

- Among Chinese young adults, elevated fasting triglyceride (TG) levels were associated with increased risk of cardiovascular disease (CVD) and all-cause mortality.

What is known and what is new?

- TG levels were positively associated with CVD and all-cause mortality in the middle-aged or elderly population.
- TG levels were positively associated with CVD and all-cause mortality in Chinese young adults.

What is the implication, and what should change now?

- TG levels have the potential to serve as a reference biomarker for long-term incidents of CVD and all-cause mortality among young adults.

Methods

Study population

The Kailuan study, a prospective dynamic cohort study, was started in 2006 and conducted in Tangshan, China. The study design and procedures have been previously described in detail (19). In brief, participants from the Kailuan Group, which is a coal miner group, received a unified standard physical health examination and questionnaire survey at 11 affiliated hospitals of the Kailuan Group and were followed up every 2 years. Every participant was then followed until their death or until December 31, 2020, whichever came first.

In the current study, participants who attended health checkups for the first time between 2006 and 2016 and were <40 years old were selected as the research subjects (n=44,715). Participants without baseline TG values were excluded (n=698). Individuals with a history of CVD or cancer were also excluded (n=77). Additionally, participants using lipid-lowering medications at baseline were excluded (n=58). Ultimately, 43,882 participants were enrolled as the analytical sample (Figure S1). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the Kailuan General Hospital (No. 200605) and informed consent was obtained from all individual participants. All participating hospitals were informed and agreed on the study.

Data collection and definitions

Baseline data on demographic, behavioral, and clinical information, including age, sex, smoking status, alcohol consumption, physical exercise, education level, medical history (hypertension and diabetes), medication use (antihypertensive agents, hypoglycemic agents, and lipid-

lowering agents), and family history of CVD, were collected via face-to-face questionnaires by trained interviewers. Smokers who smoked more than 1 cigarette per day on average over the past year were defined as current smokers. Drinkers who consumed more than 100 mL of alcohol per day on average with an alcohol concentration of ≥50% v/v in the past year were defined as current drinkers (20). Physical activity that exceeded 20 minutes per session and more than 3 times per week was classified as active (21). Education level was classified as high school or above and middle school or below. The height and weight of the participants were measured by trained nurses. Body mass index (BMI) was calculated by dividing the weight in kilograms by the height in meters squared. Obesity was defined as BMI ≥28 kg/m^2 according to the criteria of the Chinese Working Group on Obesity (20). A mercury sphygmomanometer was used to measure blood pressure in the seated position after at least five minutes of rest, and an average value of at least three measurements of both systolic blood pressure (SBP) and diastolic blood pressure (DBP) was obtained. Mean arterial pressure (MAP) was calculated using the following formula: MAP = DBP + 1/3 (SBP – DBP). Hypertension was defined as an SBP over 140 mmHg or a DBP over 90 mmHg, any use of antihypertensive agents, or a history of hypertension.

After overnight fasting (8–12 h), blood samples were obtained via the participants' elbow veins. At the Kailuan General Hospital, serum specimens were stored at –80 ℃ in the central laboratory. All serum samples were assessed using a Hitachi 747 autoanalyzer (Hitachi, Tokyo, Japan). The enzymatic colorimetric method was used to measure serum TG, LDL-C, high-density lipoprotein cholesterol (HDL-C), and total cholesterol (TC) (22). The hexokinase/glucose-6-phosphate dehydrogenase method was used for the measurement of fasting blood glucose (FBG). Diabetes was defined as self-reported physician-diagnosed type 2 diabetes, FBG levels ≥7.0 mmol/L, or any use of hypoglycemic agents. Lipid-lowering agents refer to self-reported lipid-lowering medication use, including statins, niacin, and fibrates.

Study outcomes and follow-up

The primary outcomes were incident CVD [a composite endpoint of myocardial infarction (MI) and ischemic stroke] and all-cause mortality. The secondary outcomes were individual CVD subtypes, including MI, and ischemic stroke. Assessment of CVD and all-cause mortality has been described previously (23,24). Briefly, during the follow-up period, the Municipal Social Insurance Institution of Tangshan and the Hospital Discharge Register of the Kailuan Group's 11 affiliated hospitals provided information on CVD, which was updated annually. MI was diagnosed based on the 2007 universal definition (25) determined by clinical symptoms, dynamic changes in myocardial enzymes, and electrocardiogram results. Ischemic stroke was defined following the World Health Organization criteria according to symptoms, signs, and neuroimaging data (computed tomography or magnetic resonance imaging) (26). Data on death were collected from provincial vital statistics offices and confirmed by physicians. The study population was followed up from the baseline date to the death date or December 31, 2020, whichever came first.

Statistical analysis

In the present study, the dose-response relationship was explored by restricted cubic splines, and non-linear positive associations were found between TG levels and CVD, CVD subtypes, and all-cause mortality (Figures S2,S3). To better examine these associations, the study participants were subdivided into four groups based on the baseline TG levels: quartile 1 (Q1) <67 mg/dL, Q2 67 to 97 mg/dL, Q3 98 to 150 mg/dL, and Q4 ≥151 mg/dL. Continuous variables are described as the mean ± standard deviation (SD) or the median (interquartile range) and were compared using analysis of variance (ANOVA) or Kruskal-Wallis tests based on distribution. Categorical variables are described as numbers (percentages) and were compared using the chi-square test. To handle the missing values of the covariates (as shown in Table S1, all covariates with a missing rate of less than 15%), we applied multiple imputation with 10 rounds using chained equations as previously described (27,28).

The person-years were determined from the baseline date to either the date of outcomes (CVD or death) or December 31, 2020, whichever came first. The Kaplan-Meier curve and log-rank test were adopted for univariable survival analysis. Two Cox proportional hazard regression models were constructed to estimate the hazard ratios (HRs) of CVD, all-cause mortality, and CVD subtypes. Model 1 was adjusted for age and sex; Model 2 was further adjusted for obesity, HDL-C, LDL-C, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of CVD. For each model, P values for the trend were determined using the quartiles of TG as an ordinal variable. All models were tested for proportional hazard assumption, and no

appreciable violation was observed. Based on the variance inflation factor, multicollinearity among covariables in Model 2 was found to be acceptable (Table S2). To gain insight into the linearity of associations between baseline TG levels and the risk of CVD, CVD subtypes, and all-cause mortality, restricted cubic splines were constructed. The receiver operating characteristic (ROC) curve was plotted to identify the optimal cutoff value (the best Youden Index: sensitivity + specificity –1) for each primary outcome.

To assess the robustness of our results, we also performed the following sensitivity analyses excluding participants with factors that impact TG levels, CVD events, or all-cause mortality (29,30): (I) exclusion of participants with LDL-C ≥190 mg/dL at baseline due to the elevated risk of CVD and mortality (31); (II) exclusion of participants on antihypertensive or hypoglycemic agents at baseline; (III) exclusion of participants with new-onset cancer during follow-up; (IV) exclusion of patients who experienced outcomes during the first 2-year follow-up period; (V) exclusion of participants with missing covariable values and complete-case analysis; (VI) exclusion of current drinkers at baseline; (VII) exclusion of participants with obesity at baseline; (VIII) exclusion of participants with hypertension at baseline; (IX) exclusion of participants with diabetes at baseline; (X) exclusion of participants with metabolic syndrome at baseline.

All analyses were carried out using Stata (version 15.1, StataCorp, College Station, TX, USA). A two-sided P<0.05 was considered statistically significant.

Results

The mean age of the 43,882 participants was 30.6±5.56 years, and 35,173 (80.2%) were males. Overall, participants with higher TG levels tended to be older, male, obese, current smokers, and current drinkers. They also had higher levels of MAP, FBG, and LDL-C and had a higher prevalence of hypertension and diabetes mellitus. In addition, participants with higher TG levels had a lower education level and were more likely to use antihypertensive and glucose-lowering medications (*Table 1*).

During a median follow-up of 11.2 (7.54 to 13.8) years, 298 incident CVD and 345 all-cause mortality cases were recorded. In terms of CVD subtypes, 74 cases of MI, 227 cases of ischemic stroke, and 3 cases of concurrent MI and stroke were documented.

The incidence of CVD and all-cause mortality were 0.67 and 0.76 per 1,000 person-years, respectively. From the lowest TG quartile to the second, third, and highest quartiles, the incidence of CVD increased from 0.25 to 0.47, 0.63, and 1.25 per 1,000 person-years, respectively, and the incidence of all-cause mortality increased from 0.45 to 0.55, 0.85, and 1.17 per 1,000 person-years, respectively (*Table 2*). Participants with higher baseline TG levels had a higher risk of CVD events and all-cause mortality (*Figure 1*; all log-rank P<0.001). After adjusting for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of CVD, compared with those for participants in the lowest quartile, the adjusted HR of incident CVD for participants in the highest quartile was 2.26 [95% confidence interval (CI): 1.56 to 3.29; P=0.001]. The corresponding HR of all-cause mortality for participants in the highest quartile was 1.61 (95% CI: 1.14 to 2.28; P=0.007) (*Table 2*). Additionally, the results of the prespecified sensitivity analyses showed consistent positive associations between TG levels and CVD and all-cause mortality as did the primary analysis (Tables S3,S4).

The area under the curve (AUC) of the TG levels for CVD and all-cause mortality was 0.664 (95% CI: 0.634 to 0.695) and 0.616 (95% CI: 0.586 to 0.645), respectively. The cutoff points of TG levels for incident CVD were 110 mg/dL with 66.4% sensitivity and 57.3% specificity; the corresponding cutoff points for all-cause mortality were 102 mg/dL with 66.7% sensitivity and 52.0% specificity, respectively (Table S5). In multivariable Cox-regression models, TG ≥110 mg/dL was associated with a higher risk for CVD (adjusted HR: 1.46; 95% CI: 1.13 to 1.88; P=0.003), and TG ≥102 mg/dL was associated with a higher risk for all-cause mortality (adjusted HR: 1.50; 95% CI: 1.18 to 1.91; P=0.001) (*Table 3*).

In terms of CVD subtypes, the incidence of MI, and ischemic stroke were 0.16, and 0.50 per 1,000 person-years, respectively. Participants with higher baseline TG levels tended to have higher risks of MI and ischemic stroke (*Figure 2*; all log-rank P<0.001). Compared with those for participants in the lowest quartile, the adjusted HRs (95% CIs) for MI for participants in the highest quartile was 3.25 (95% CI: 1.33 to 7.97; P=0.01), respectively; for ischemic stroke, that was 1.88 (95% CI: 1.16 to 3.04; P=0.01), respectively (*Table 4*).

Discussion

In the present study, we assessed the association between fasting TG and the risk of incident CVD and all-cause

Table 1 Baseline characteristics of participants according to the quartiles of baseline triglyceride levels[a]

Variables	TG (mg/dL)					P value
	Total	Q1 [<67]	Q2 [67–97]	Q3 [98–150]	Q4 [≥151]	
Participants, n	43,882	11,063	10,886	10,849	11,084	
Age, years	30.6±5.56	29.8±5.55	30.2±5.54	30.7±5.60	31.6±5.38	<0.001
Male	35,173 (80.2)	7,332 (66.3)	8,390 (77.1)	9,172 (84.5)	10,279 (92.7)	<0.001
Obesity	7,125 (16.2)	593 (5.4)	1,127 (10.4)	1,970 (18.2)	3,435 (31.0)	<0.001
MAP, mmHg	92.3 (83.8, 97.8)	88.3 (81.3, 94.0)	90.4 (83.3, 96.7)	93.3 (85.6, 98.7)	94.7 (90.0, 103)	<0.001
FBG, mg/dL	91.9±24.9	89.3±27.5	90.2±21.3	92.1±22.3	95.8±27.2	<0.001
HDL-C, mg/dL	53.4 (46.0, 61.9)	56.1 (48.0, 65.0)	53.8 (46.8, 61.5)	53.0 (46.0, 61.1)	50.7 (42.9, 59.9)	<0.001
LDL-C, mg/dL	92.8 (76.6, 112)	83.9 (66.9, 102)	92.8 (78.1, 109)	96.7 (82.4, 115)	98.2 (79.3, 118)	<0.001
Current smoker	15,863 (36.1)	3,144 (28.4)	3,583 (32.9)	4,000 (36.9)	5,136 (46.3)	<0.001
Current drinker	19,517 (44.5)	4,213 (38.1)	4,397 (40.4)	4,866 (44.9)	6,041 (54.5)	<0.001
Active physical activity	3,545 (8.1)	911 (8.2)	881 (8.1)	817 (7.5)	936 (8.4)	0.08
High school or above	24,352 (55.5)	6,697 (60.5)	6,223 (57.2)	5,859 (54.0)	5,573 (50.3)	<0.001
Hypertension	3,617 (8.2)	303 (2.7)	605 (5.6)	1,106 (10.2)	1,603 (14.5)	<0.001
Diabetes	532 (1.2)	54 (0.5)	74 (0.7)	132 (1.2)	272 (2.5)	<0.001
Antihypertensive agents	591 (1.3)	68 (0.6)	95 (0.9)	152 (1.4)	276 (2.5)	<0.001
Hypoglycemic agents	135 (0.3)	20 (0.2)	26 (0.2)	31 (0.3)	58 (0.5)	<0.001
Family history of CVD	1,127 (2.6)	244 (2.2)	263 (2.4)	277 (2.6)	343 (3.1)	<0.001

[a], continuous variables with normal distribution are described as mean ± standard deviation and were compared using ANOVA; continuous variables with skewed distribution are presented as median (interquartile range) and were compared using the Kruskal-Wallis test; categorical variables are described as number (percentage) and were compared using the chi-square test. TG, triglyceride; Q, quartile; MAP, mean arterial pressure, FBG, fasting blood glucose; HDL-C, high-density lipid cholesterol; LDL-C, low-density lipid cholesterol; CVD, cardiovascular disease; ANOVA, analysis of variance.

mortality in adults younger than 40 years from the Kailuan cohort. We reported two main findings: (I) elevated TG levels were associated with higher risks of CVD and all-cause mortality, which were independent of potential confounders such as age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of CVD; (II) this association still existed even in participants with "normal" TG levels (150 mg/dL) according to the current guidelines.

Until now, there has been no large prospective study reporting any association between baseline TG and long-term CVD among Chinese young adults. Although the association between TG levels and CVD risk has been extensively studied in observational studies (32,33), only three studies have focused on young adults and reported a positive association between baseline TG level and incident CVD (including MI and stroke) (15-17). However, all three studies were from the same database in South Korea (the claims database from the National Health Insurance Service), which limits the extrapolation of the results to other populations, so it makes sense to conduct studies among young people in other countries. Consistent with those studies, our study provided the first evidence that high levels of TG were independently related to increased future CVD risk in Chinese young adults. This finding highlights the necessity of early screening of TG levels.

The underlying mechanisms between TG and the risk of CVD are unclear. Evidence supports that TG may act as a biomarker of other risk factors rather than a direct driver for incident CVD. First, a high TG level has been regarded as an early marker of underlying metabolic dysfunction, leading to additional cardiovascular risk factor accumulation, such as elevated TC and LDL-C levels and obesity (34).

Table 2 Risk of incident cardiovascular disease and all-cause mortality by quartiles of baseline triglyceride levels

Triglyceride, mg/dL	Events/total	Incidence, per 1,000 person-years	Model 1			Model 2		
			HR (95% CI)	P value	P for trend	HR (95% CI)	P value	P for trend
CVD	298/43,882	0.67			<0.001			<0.001
Q1 [<67]	28/11,063	0.25	Reference	–		Reference	–	
Q2 [67–97]	52/10,886	0.47	1.57 (0.99, 2.48)	0.05		1.36 (0.87, 2.19)	0.17	
Q3 [98–150]	72/10,849	0.63	1.77 (1.14, 2.75)	0.01		1.38 (0.88, 2.14)	0.16	
Q4 [≥151]	146/11,084	1.25	2.96 (1.96, 4.46)	<0.001		2.26 (1.56, 3.29)	0.001	
Per SD increase			1.09 (1.05, 1.14)	<0.001		1.08 (1.02, 1.15)	0.01	
All-cause mortality	345/43,882	0.76			<0.001			0.001
Q1 [<67]	50/11,063	0.45	Reference	–		Reference	–	
Q2 [67–97]	61/10,886	0.55	1.08 (0.74, 1.57)	0.70		1.05 (0.72, 1.54)	0.78	
Q3 [98–150]	97/10,849	0.85	1.47 (1.04, 2.07)	0.02		1.40 (0.98, 1.99)	0.06	
Q4 [≥151]	137/11,084	1.17	1.75 (1.25, 2.45)	0.001		1.61 (1.14, 2.28)	0.007	
Per SD increase			1.07 (1.02, 1.12)	0.004		1.06 (1.01, 1.12)	0.02	

Model 1 was adjusted for age and sex; Model 2 was adjusted for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of CVD. HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; Q, quartile; SD, standard deviation.

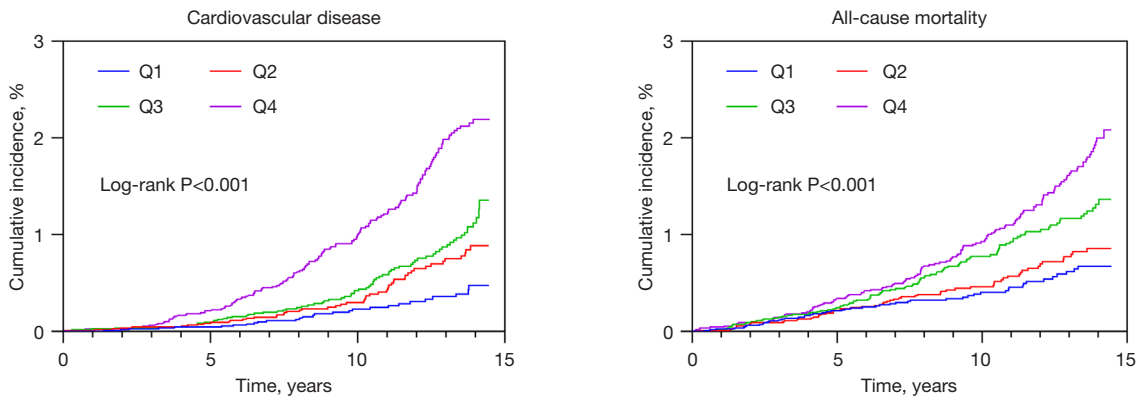

Figure 1 Cumulative incidence of cardiovascular disease and all-cause mortality by quartiles of baseline triglyceride levels. Q, quartile.

Second, high TG levels are more closely related to unhealthy lifestyles such as heavy alcohol consumption and lack of exercise, which reflect different pathophysiological effects in the development of CVD (35). Third, the majority of TG molecules are carried in two different lipoproteins: chylomicrons and very low-density lipoproteins. It is well-known that patients with familial chylomicronemia syndrome (FCS) have a low risk of CVD despite extremely high TG levels (>1,000 mg/dL) since birth (36). Therefore, the type of lipoproteins, rather than TG itself, is an important factor. Fourth, the lipolysis of those lipoproteins results in the formation of remnant lipoproteins that can penetrate arterial walls and can be taken up by macrophages, which will further transform into foam cells (37,38). Last, the lipolysis of TG-rich lipoproteins produces a high amount of oxidized free fatty acids, monoacylglycerols, and other molecules, which can induce endothelial cell inflammation and promote atherogenesis (38-40).

Table 3 Risk of incident cardiovascular diseases and mortality by dichotomous baseline triglyceride levels

Triglyceride, mg/dL	Events/total	Incidence, per 1,000 person-years	Model 1		Model 2	
			HR (95% CI)	P value	HR (95% CI)	P value
CVD	298/43,882	0.66				
<110	98/25,072	0.38	Reference	–	Reference	–
≥110	200/18,810	1.01	1.86 (1.45, 2.37)	<0.001	1.46 (1.13, 1.88)	0.003
All-cause mortality	345/43,882	0.76				
<102	115/22,747	0.50	Reference	–	Reference	–
≥102	230/21,135	1.03	1.59 (1.27, 2.00)	<0.001	1.50 (1.18, 1.91)	0.001

Model 1 was adjusted for age and sex; Model 2 was adjusted for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of CVD. HR, hazards ratio; CI, confidence interval; CVD, cardiovascular diseases.

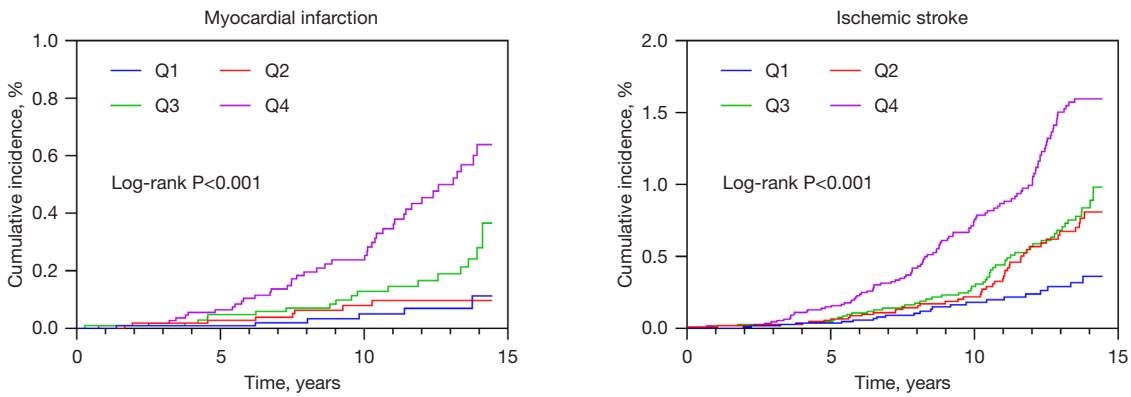

Figure 2 Cumulative incidence of myocardial infarction and ischemic stroke by quartiles of baseline triglyceride levels. Q, quartile.

Prior studies have reported conflicting findings regarding the association between TG levels and all-cause mortality risk among adults older than 40 years. Several studies have shown a significant rise in all-cause mortality risk among adults with a high level of TG (9,32). In contrast, a retrospective study of 373,389 American adults aged over 45 years receiving statin therapy found that elevated TG levels were associated with a reduced all-cause mortality risk (41). The possible reasons for this inconsistency may be population heterogeneity (i.e., age, ethnicity, and treatment), different follow-up durations, and various confounders. To date, limited studies have explored the association between TG levels and all-cause mortality risk among young adults. A nationwide cohort study included 5,688,055 Korean participants aged 20–39 years with a median follow-up of 7.1 years and found that elevated TG levels were independently associated with increased mortality (17). Our study with a much longer follow-up period (11.2 years) obtained a consistent result with this study. We provide the first epidemiological evidence in Chinese young adults that a higher TG level significantly contributes to an increased risk of long-term all-cause mortality.

According to current clinical practice guidelines, only individuals with TG >200 mg/dL were recommended to control hypertriglyceridemia to reduce CVD risk (42). However, several population-based studies consistently showed that elevated baseline TG levels even below 150 mg/dL, previously considered "optimal", were associated with increased risk of CVD (43-45) and all-cause mortality (14). In agreement with those studies, our data provided the first evidence that participants with a cutoff value of 110 mg/dL had a 46% higher risk of CVD and a cutoff value of 102 mg/dL had a 50% higher risk of all-cause mortality in young adults. The results suggested that TG-lowering intervention may prove beneficial for even moderately elevated TG levels (110 to 150 mg/dL) in young adults.

Table 4 Risk of incident cardiovascular disease subtypes by quartiles of baseline triglyceride levels

Triglyceride, mg/dL	Events/total	Incidence, per 1,000 person-years	Model 1			Model 2		
			HR (95% CI)	P value	P for trend	HR (95% CI)	P value	P for trend
Myocardial infarction	74/43,882	0.16			<0.001			0.001
Q1 [<67]	6/11,063	0.05	Reference	–		Reference	–	
Q2 [67–97]	8/10,886	0.07	1.26 (0.44, 3.63)	0.67		1.07 (0.37, 3.08)	0.90	
Q3 [98–150]	18/10,849	0.16	2.55 (1.01, 6.42)	0.04		1.83 (0.71, 4.70)	0.20	
Q4 [≥151]	42/11,084	0.36	5.35 (2.27, 12.6)	<0.001		3.25 (1.33, 7.97)	0.01	
Per SD increase			1.10 (1.04, 1.16)	0.001		1.09 (1.02, 1.19)	0.02	
Ischemic stroke	227/43,882	0.50			<0.001			0.01
Q1 [<67]	22/11,063	0.20	Reference	–		Reference	–	
Q2 [67–97]	45/10,886	0.40	1.73 (1.05, 2.85)	0.03		1.31 (0.79, 2.17)	0.29	
Q3 [98–150]	54/10,849	0.47	1.75 (1.06, 2.89)	0.03		1.53 (0.91, 2.56)	0.10	
Q4 [≥151]	106/11,084	0.91	2.80 (1.76, 4.48)	<0.001		1.88 (1.16, 3.04)	0.01	
Per SD increase			1.09 (1.04, 1.14)	0.001		1.08 (1.01, 1.15)	0.01	

Model 1 was adjusted for age and sex; Model 2 was adjusted for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of cardiovascular disease. HR, hazard ratio; CI, confidence interval; Q, quartile; SD, standard deviation.

Strengths and limitations

Several strengths are evident in our study. The associations between the fasting TG level and the risk of CVD, CVD subtypes, and all-cause mortality were examined for the first time among Chinese young adults comprising a prospective cohort. Our study involved only adults younger than 40 years, for whom few recommendations on TG management are provided in current guidelines (18). Accordingly, this study is poised to provide unprecedented insight into TG management among this age group.

However, there are some inherent limitations in this study. First, despite excluding participants with previous CVD at baseline and individuals who experienced outcomes during the first 2-year follow-up period to mitigate the potential concerns of reverse causality, we cannot prove causality in our study. Second, although we have made every effort to adjust for some important confounders, there is always a chance of unmeasured residual confounding (such as the level of lipoproteins (46) and hypertriglyceridemia-prone medications use). Third, since the study population was recruited from Tangshan City, which is located in northern China, the findings may not be generalizable to other parts of China, other countries, or ethnic populations. In addition, the majority of the participants are coal miners, which may limit the generalizability of the results as well. Last, due to the inherent limitations of our cohort, we were not able to provide detailed information on specific causes of mortality. As such, it may be worthwhile to investigate the effects of TG on specific causes of death in young adults in the future.

Conclusions

The present study shows that elevated fasting TG levels are significantly associated with increased long-term CVD and all-cause mortality risk among Chinese young adults. This study indicated that TG levels have the potential to serve as a reference biomarker for long-term prognosis among young adults.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://cdt.amegroups.com/article/view/10.21037/cdt-23-412/rc

Data Sharing Statement: Available at https://cdt.amegroups.com/article/view/10.21037/cdt-23-412/dss

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Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://cdt.amegroups.com/article/view/10.21037/cdt-23-412/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of the Kailuan General Hospital (No. 200605) and informed consent was obtained from all individual participants. All participating hospitals were informed and agreed on the study.

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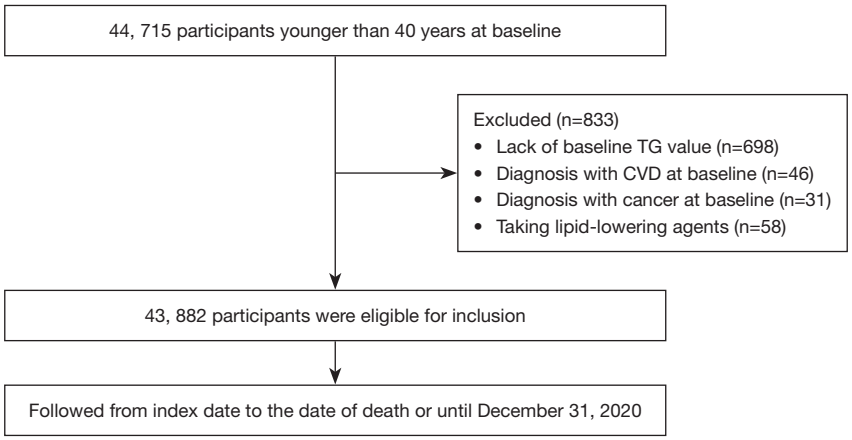

Figure S1 Study flow diagram. TG, triglyceride; CVD, cardiovascular disease.

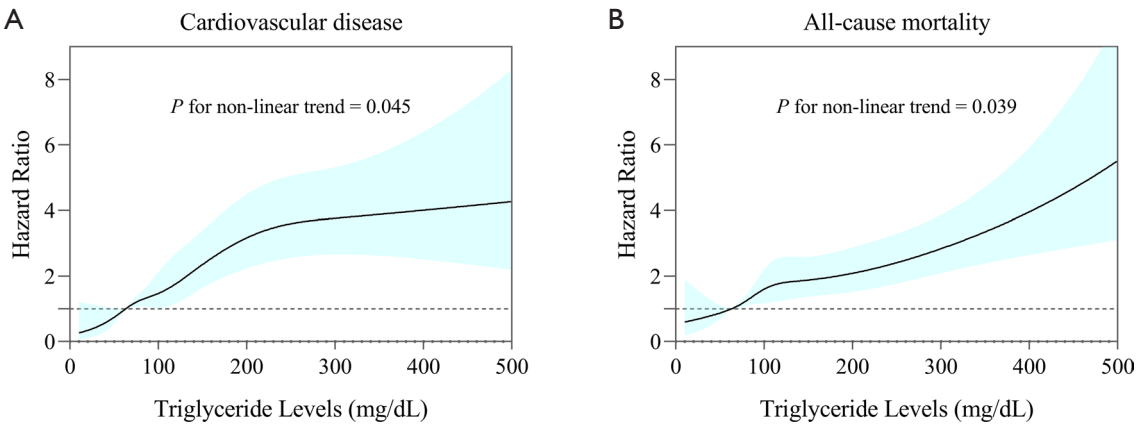

Figure S2 Full adjusted hazard ratios of incident cardiovascular disease (A) and all-cause mortality (B) by baseline triglyceride levels.

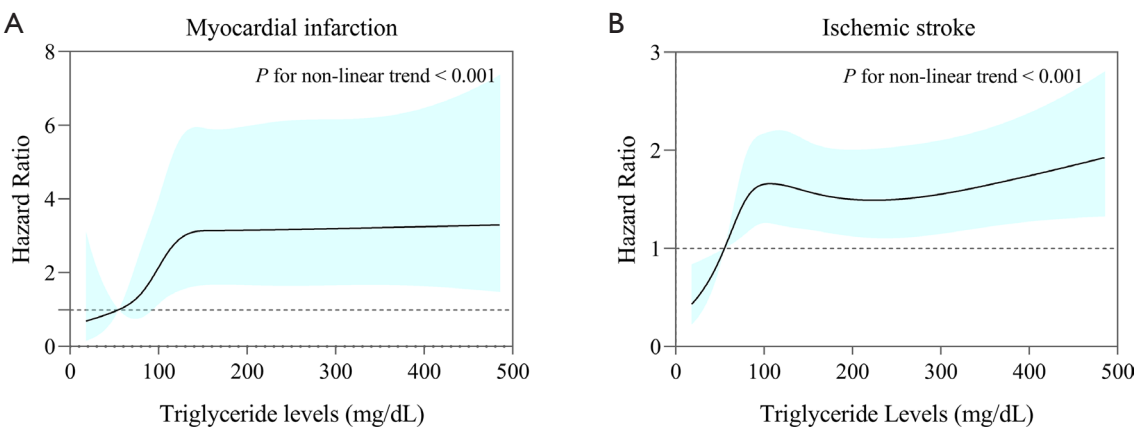

Figure S3 Full adjusted hazard ratios of incident myocardial infarction (A) and ischemic stroke (B) by baseline triglyceride levels.

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Table S1 Counts and proportions of missing data[a]

Variable	Count of missing values (%)
Triglyceride, mg/dL	0
Age, years	0
Sex	0
Obesity	1095 (2.5)
High-density lipoprotein cholesterol, mg/dL	23 (0.1)
Low-density lipoprotein cholesterol, mg/dL	60 (0.1)
Smoking status	4737 (10.8)
Alcohol consumption	4157 (9.5)
Physical activity	6229 (14.2)
Education level	6064 (13.8)
Hypertension	0
Diabetes	0
Family history of cardiovascular disease	2135 (4.9)

[a], Little's chi-square test for covariate-dependent missingness (CDM) showed that the missing values were missing at random (P>0.99).

Table S2 Variance inflation factors of the covariables in Model 2

Covariable	Variance inflation factor
Triglyceride, mg/dL	1.06
Age, years	1.24
Sex	1.31
Obesity	1.14
High-density lipoprotein cholesterol, mg/dL	1.04
Low-density lipoprotein cholesterol, mg/dL	1.03
Smoking status	1.36
Alcohol consumption	1.38
Physical activity	1.02
Education level	1.22
Hypertension	1.10
Diabetes	1.03
Family history of cardiovascular disease	1.02
Mean variance inflation factor	1.15

Table S3 Sensitivity analyses (A)

Triglyceride, mg/dL	HR (95% CI)[a]				
	Analysis 1[b]	Analysis 2[c]	Analysis 3[d]	Analysis 4[e]	Analysis 5[f]
CVD					
Events/total	295/43691	276/43218	294/43584	285/43834	288/33809
Q1 [<67]	Reference	Reference	Reference	Reference	Reference
Q2 [67–97]	1.42 (0.91, 2.24)	1.46 (0.89, 2.41)	1.40 (0.87, 2.18)	1.35 (0.84, 2.19)	1.39 (0.88, 2.20)
Q3 [98–150]	1.43 (0.89, 2.28)	1.53 (0.95, 2.46)	1.42 (0.90, 2.23)	1.37 (0.86, 2.17)	1.43 (0.89, 2.29)
Q4 [≥151]	2.11 (1.37, 3.24)	2.36 (1.50, 3.70)	2.07 (1.35, 3.18)	2.10 (1.36, 3.25)	2.08 (1.34, 3.21)
P for trend	<0.001	<0.001	<0.001	<0.001	0.01
Per SD increase	1.08 (1.02, 1.15)	1.08 (1.02, 1.15)	1.08 (1.01, 1.15)	1.09 (1.03, 1.15)	1.08 (1.02, 1.15)
All-cause mortality					
Events/total	344/43691	322/43218	282/43584	310/43847	323/33809
Q1 [<67]	Reference	Reference	Reference	Reference	Reference
Q2 [67–97]	1.01 (0.69, 1.48)	1.05 (0.72, 1.53)	1.05 (0.68, 1.60)	1.07 (0.71, 1.61)	1.05 (0.71, 1.55)
Q3 [98–150]	1.30 (0.92, 1.85)	1.39 (0.97, 1.98)	1.40 (0.94, 2.09)	1.47 (1.00, 2.14)	1.41 (0.98, 2.04)
Q4 [≥151]	1.46 (1.04, 2.06)	1.50 (1.05, 2.14)	1.70 (1.15, 2.51)	1.74 (1.20, 2.53)	1.60 (1.12, 2.31)
P for trend	0.008	0.005	0.001	0.001	0.002
Per SD increase	1.06 (1.01, 1.12)	1.06 (1.00, 1.12)	1.06 (1.01, 1.13)	1.06 (1.00, 1.12)	1.06 (1.00, 1.13)

[a], adjusted for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of cardiovascular disease; [b], excluded participants with LDL-C ≥190 mg/dL at baseline; [c], excluded participants on antihypertensive or hypoglycemic agents at baseline; [d], excluded participants with new-onset cancer during follow-up; [e], excluded participants who experienced outcomes during the first 2-year follow-up period; [f], excluded participants with missing covariable values and performed a complete-case analysis. HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; Q, quartile; SD, standard deviation.

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Table S4 Sensitivity analyses (B)

Triglyceride, mg/dL	HR (95% CI)[a]				
	Analysis 6[b]	Analysis 7[c]	Analysis 8[d]	Analysis 9[e]	Analysis 10[f]
CVD					
Events/total	141/24365	187/36757	191/40265	276/43350	249/41564
Q1 [<67]	Reference	Reference	Reference	Reference	Reference
Q2 [67–97]	2.20 (1.03, 4.72)	1.32 (0.81, 2.16)	1.27 (0.74, 2.17)	1.33 (0.83, 2.11)	1.35 (0.86, 2.11)
Q3 [98–150]	2.40 (1.14, 5.07)	1.39 (0.84, 2.28)	1.45 (0.86, 2.42)	1.47 (0.92, 2.36)	1.38 (0.87, 2.20)
Q4 [≥151]	3.86 (1.24, 8.00)	1.76 (1.10, 2.81)	2.24 (1.38, 3.64)	2.07 (1.34, 3.21)	1.87 (1.21, 2.90)
P for trend	<0.001	0.024	<0.001	<0.001	0.004
Per SD increase	1.08 (1.00, 1.16)	1.07 (1.00, 1.15)	1.08 (1.01, 1.15)	1.07 (1.00, 1.15)	1.07 (1.00, 1.15)
All-cause mortality					
Events/total	169/24365	263/36757	242/40265	325/43350	309/41564
Q1 [<67]	Reference	Reference	Reference	Reference	Reference
Q2 [67–97]	1.25 (0.74, 2.12)	1.00 (0.67, 1.50)	0.92 (0.61, 1.40)	1.06 (0.72, 1.55)	1.07 (0.74, 1.58)
Q3 [98–150]	1.74 (1.04, 2.92)	1.31 (0.90, 1.92)	1.42 (0.97, 2.09)	1.42 (1.00, 2.03)	1.41 (1.00, 2.02)
Q4 [≥151]	1.87 (1.14, 3.07)	1.66 (1.14, 2.40)	1.48 (1.01, 2.19)	1.54 (1.08, 2.20)	1.64 (1.14, 2.35)
P for trend	0.015	0.002	0.14	0.005	0.002
Per SD increase	1.02 (0.91, 1.15)	1.05 (0.98, 1.13)	1.02 (0.92, 1.14)	1.05 (1.00, 1.12)	1.06 (1.00, 1.13)

[a], adjusted for age, sex, obesity, high-density lipid cholesterol, low-density lipid cholesterol, smoking status, alcohol consumption, physical activity, education level, hypertension, diabetes, and family history of cardiovascular disease; [b], excluded current drinkers at baseline; [c], excluded participants with obesity at baseline; [d], excluded participants with hypertension at baseline; [e], excluded participants with diabetes at baseline; [f], excluded participants with metabolic syndrome at baseline. HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; Q, quartile; SD, standard deviation.

Table S5 The ROC curves and diagnostic characteristics of the triglyceride level as a marker to predict CVD and all-cause mortality

Primary outcomes	AUC (95% CI)	Cut-off value (mg/dL)	Sensitivity (%)	Specificity (%)
CVD	0.664 (0.634, 0.695)	110	66.4	57.3
All-cause mortality	0.616 (0.586, 0.645)	102	66.7	52.0

ROC, receiver operating characteristic; CVD, cardiovascular diseases; AUC, area under the curve; CI, confidence interval.

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