Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Associations of baseline, cumulative exposure, and dynamic trajectories of the triglyceride– total cholesterol–body weight index with new-onset hypertension in middle-aged and older Chinese adults: a nationwide prospective cohort Study

Wenjing Huang
The First People's Hospital of Neijiang City
Li Chen
The First People's Hospital of Neijiang City
Yao Chen
The First People's Hospital of Neijiang City
Haibo Gong
2225968823@qq.com
The First People's Hospital of Neijiang City

Research Article

Keywords: triglycerides-total cholesterol-body weight index, hypertension, cumulative exposure, dynamic trajectories, CHARLS

Posted Date: November 16th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11316331/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Associations of baseline, cumulative exposure, and dynamic trajectories of the triglyceride–total cholesterol–body weight index with new-onset hypertension in middle-aged and older Chinese adults: a nationwide prospective cohort Study

Wenjing Huang[a] , Li Chen[a], Yao Chen[a], Haibo Gong[a*]

[a]The First People's Hospital of Neijiang City, Sichuan, China

Corresponding author：Haibo Gong 2225968823@qq.com

Wenjing Huang：541933708@qq.com

Li Chen: 625069853@qq.com

Yao Chen:1017780344@qq.com

Abstract

***Background*:** Hypertension is a major modifiable risk factor for cardiovascular disease (CVD) and mortality worldwide, and its development and progression is a dynamic process involving the accumulation of long-term metabolic burden. The Triglyceride–Total Cholesterol–Body Weight Index (TCBI) is a novel, simple, and readily available composite nutritional and metabolic indicator that has been associated with various adverse cardiovascular and metabolic outcomes. However, its prospective association with new-onset hypertension—particularly in terms of cumulative exposure and dynamic trajectories—has not yet been systematically studied. This study aimed to investigate the associations of baseline TCBI, time-weighted cumulative TCBI (cumTCBI), and TCBI dynamic trajectories with the risk of new-onset hypertension in middle-aged and older Chinese adults.

Methods: This study was based on data from the China Health and Retirement Longitudinal Study (CHARLS) cohort. The baseline analysis included 4,777 participants without hypertension at baseline, and the cumulative and trajectory analyses included 2,622 participants with complete TCBI data from Wave 1 (2011) and Wave 3 (2015). TCBI was calculated as triglyceride (TG, mg/dL) × total cholesterol (TC, mg/dL) × body weight (BW, kg) / 1000. cumTCBI was calculated using a time-weighted cumulative approach. K-means clustering was used to identify dynamic TCBI trajectories. Cox proportional hazards models were used to assess the association between TCBI and new-onset hypertension; restricted cubic spline (RCS) analysis was used to examine dose-response relationships; and subgroup and sensitivity analyses (logistic regression, complete-case analysis, and competing risk models) were conducted. Missing data were handled using multiple imputation by chained equations (MICE).

Conclusion: Baseline TCBI, cumulative TCBI, and TCBI dynamic trajectories were independently and positively associated with the risk of new-onset hypertension in middle-aged and older Chinese adults, and baseline TCBI exhibited a significant nonlinear threshold effect. As a simple and readily available composite nutritional and metabolic indicator, TCBI can effectively identify individuals at high risk of hypertension through multidimensional longitudinal assessment, providing a new tool for early risk stratification and primary prevention of hypertension.

Keywords: triglycerides-total cholesterol-body weight index; hypertension; cumulative exposure; dynamic trajectories; CHARLS

Introduction

Hypertension remains a major modifiable risk factor for cardiovascular disease and is one of the leading causes of death and disability worldwid(1, 2). According to data from the World Health Organization's Global Health Estimates, cardiovascular disease caused at least 19 million deaths globally in 2021, accounting for the largest share of deaths from noncommunicable diseases(3). The World Health Organization's *Global Hypertension Report* further notes that approximately one-third of adults worldwide are affected by hypertension, but less than one-quarter of them have their blood pressure adequately controlled (4). In China, the burden of hypertension is equally severe. The China Hypertension Survey (2012–2015) report shows that the age-standardized prevalence of hypertension among adults aged 18 and older in China was 23.2%, corresponding to approximately 244.5 million people; among those with hypertension, the awareness rate, treatment rate, and control rate were only 46.9%, 40.7%, and 15.3%, respectively (5, 6). Given the enormous disease burden and low control rates, the early identification of high-risk individuals through simple and readily available biomarkers is of significant public health importance (7, 8).

Triglyceride–Total Cholesterol–Body Weight Index (TCBI) is a recently proposed composite nutritional and metabolic indicator, calculated as: triglycerides (mg/dL) × total cholesterol (mg/dL) × body weight (kg) / 1000(9). TCBI integrates the two core components of lipid metabolism with body weight—which reflects

systemic metabolic burden—into a single indicator. It not only reflects the state of lipid metabolism disorder but also indirectly reflects nutritional reserves and energy balance. Its simplicity of calculation and high clinical feasibility give it unique advantages for application in large-scale epidemiological studies(10, 11) . Previous prospective studies have preliminarily confirmed that TCBI is associated with various adverse cardiometabolic outcomes. A study by Rezaee et al., based on the MASHAD cohort (n = 9,704) with a 10-year follow-up, demonstrated that TCBI was significantly positively associated with cardiovascular mortality (12). A study by Chen et al. based on the CHARLS cohort further demonstrated that cumulative TCBI and its dynamic trajectories were independently associated with new-onset cardiometabolic multimorbidity (13). However, the prospective association between TCBI and new-onset hypertension has not yet been systematically investigated.

The development and progression of hypertension is a dynamic process(14) , and a single baseline measurement is unlikely to fully capture long-term changes in metabolic burden(15) (16). Cumulative exposure analysis and trajectory analysis (such as K-means clustering) can better capture long-term metabolic burden and dynamic change patterns(17-20), and have been widely applied in cardiovascular and metabolic epidemiological studies(21, 22). Therefore, adopting a multidimensional analytical framework of “baseline level–cumulative burden–dynamic trajectory”(23) enables a more comprehensive assessment of the association between TCBI and new-onset hypertension.

Therefore, based on data from the nationally representative China Health and

Retirement Longitudinal Study (CHARLS) cohort, this study aimed to systematically evaluate the association between TCBI and the risk of new-onset hypertension in middle-aged and older adults across three dimensions: baseline TCBI, time-weighted cumulative TCBI, and TCBI dynamic trajectories identified via K-means clustering, aiming to provide new evidence for the early risk identification of hypertension based on a composite nutritional and metabolic indicator.

Study design and participants

The data for this study were drawn from the China Health and Retirement Longitudinal Study (CHARLS). CHARLS is a nationwide, prospective cohort study that targets community-dwelling Chinese adults aged 45 years and older(24). The baseline survey was conducted from 2011 to 2012, covering 28 provinces, 150 counties, and 450 villages or urban communities, and included a total of 17,708 participants. A standardized questionnaire was used to collect demographic, health, and lifestyle information. Follow-up surveys were conducted every two years, in 2013 (Wave 2), 2015 (Wave 3), 2018 (Wave 4), and 2020 (Wave 5). All participants provided written informed consent, and the study protocol was approved by the Biomedical Ethics Review Committee of Peking University (IRB approval number: IRB00001052-11015). The CHARLS dataset is publicly available at http://charls.pku.edu.cn.

Baseline TCBI analysis

This study used the 2011 wave (Wave 1) of CHARLS as the baseline. The exclusion criteria were as follows: (1) prevalent hypertension at Wave 1 (n = 6,868); (2) missing

hypertension data (n = 210); (3) missing triglyceride (TG) data (n = 3,796); (4) missing total cholesterol (TC) data (n = 2); (5) missing body weight data (n = 1,185); (6) age < 45 years or missing age data (n = 210); (7) missing follow-up data (n = 660). After applying these exclusion criteria, a total of 4,777 participants without hypertension at baseline were included. New-onset hypertension was ascertained during Waves 2–5 (2013–2020). The participant selection process is shown in Figure 1.

Cumulative TCBI and trajectory analyses

To assess dynamic changes in TCBI, we used data from Wave 1 (2011) and Wave 3 (2015). Of the 5,437 participants with complete TG, TC, and body weight data at Wave 1, we further required complete TG, TC, and body weight data at Wave 3, and excluded participants with prevalent hypertension at Wave 1 or Wave 3, as well as those who were lost to follow-up or had missing data during follow-up through Wave 4 or Wave 5. After these exclusions, a total of 2,622 participants were included in the cumulative and trajectory analyses. The participant selection process is shown in Figure 2.

Definitions of hypertension and new-onset hypertension

Hypertension was defined as meeting any of the following criteria at Wave 3: (1) self-reported physician diagnosis of hypertension; (2) current use of antihypertensive medication; or (3) systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg(25, 26).

For the baseline TCBI analysis, the follow-up period for survival analysis began

on the date of the Wave 1 survey and ended at the earliest of the Wave 5 (2020) survey, death, or loss to follow-up (27, 28). For cumulative TCBI and trajectory analyses, to ensure a clear temporal sequence between cumulative exposure and outcomes, the exposure period was defined as Wave 1 through Wave 3, and outcomes were assessed at Wave 4 (2018) or Wave 5 (2020) (29). For participants without hypertension at Wave 1 or Wave 3, follow-up began on the date of the Wave 3 survey and ended at the earliest of incident hypertension, death, loss to follow-up, or the last valid survey (Wave 5, 2020) (30, 31). The same definition of hypertension was applied at Wave 4 and Wave 5.

Because CHARLS surveys were conducted every two years, the exact date of hypertension diagnosis was unknown. Therefore, following standard methods for interval-censored data in longitudinal cohort studies (13, 32), the event time was assigned to the midpoint between the last hypertension-free survey and the first survey reporting a hypertension diagnosis.

TCBI assessment

TCBI was calculated as: TG (mg/dL) × TC (mg/dL) × BW (kg) / 1000.

Three exposure indicators were constructed in this study:

1. Baseline TCBI

Baseline TCBI was defined as the TCBI value measured at Wave 1 (2011).

2. Cumulative TCBI (cumTCBI)

To reflect long-term metabolic burden, the time-weighted cumulative TCBI from Wave 1 to Wave 3 was calculated as:

$$\text{cumTCBI} = [(\text{TCBI_Wave1} + \text{TCBI_Wave3}) / 2] \times (2015 - 2011)$$

The follow-up interval was 4 years. This method assumes linear changes between the two measurements and has been adopted in multiple cumulative exposure studies(13, 21, 22) and cross-cohort studies(26), and has been validated for estimating chronic metabolic burden.

3. TCBI dynamic trajectories

Using TCBI values from Wave 1 and Wave 3, K-means clustering was performed to identify long-term patterns of TCBI change. TCBI values were z-score standardized prior to clustering, and the optimal number of clusters (k = 2) was determined based on the elbow method and clinical interpretability. Clustering was performed using Euclidean distance with 1,000 random starts to ensure stability. Ultimately, cluster 1 (lower trajectory group) and cluster 2 (higher trajectory group) were identified.

Covariates

Covariates included the following categories:

1.Demographic characteristics: age (continuous and categorical: <60 years/≥60 years), sex, education (illiterate, primary school, middle school, high school or above), marital status (married/unmarried), residence (urban/rural), and region (east, central, west, and northeast China).

2.Lifestyle and psychosocial factors: smoking (never, former, current), drinking (never, former, current), sleep duration (hours, continuous variable), social activity (participation in at least one social activity: yes/no), work status (yes/no), self-rated health (very good, good, fair, poor, very poor), life satisfaction (completely satisfied,

very satisfied, somewhat satisfied, somewhat dissatisfied, completely dissatisfied), activities of daily living (ADL) disability (yes/no), history of falls (yes/no), physical pain (yes/no), disability (yes/no), and depressive symptoms (assessed using the CESD-10 scale, total score 0–30; a score ≥10 was defined as the presence of depressive symptoms and treated as a binary variable: yes/no)(33, 34).

3.Comorbidities: cardiovascular disease, diabetes, lung disease, arthritis, dyslipidemia, liver disease, kidney disease, and gastrointestinal disease. All comorbidities were based on self-reported physician diagnoses. Diabetes was defined as fasting blood glucose ≥126 mg/dL, glycated hemoglobin (HbA1c) ≥6.5%, self-reported physician-diagnosed diabetes, or current use of antidiabetic medication(35). Dyslipidemia was defined as total cholesterol ≥240 mg/dL, triglycerides ≥150 mg/dL, LDL-C ≥160 mg/dL, HDL-C <40 mg/dL, TC/HDL-C ratio >5.0, self-reported physician-diagnosed dyslipidemia, or current use of lipid-lowering medication (36).

4.Cognitive function: total cognitive function score (continuous variable), calculated from the CHARLS cognitive test, with higher scores indicating better cognitive function.

5.Laboratory indicators: white blood cell count, platelet count, blood urea nitrogen, creatinine, fasting blood glucose, HbA1c, hemoglobin, C-reactive protein(CRP), uric acid, LDL-C, HDL-C, total cholesterol, and triglycerides.

For cumulative TCBI and trajectory analyses, additional cumulative laboratory indicators were included as covariates, including cumulative body mass index (CumBMI), cumulative HbA1c (CumHbA1c), and cumulative C-reactive protein

(CumCRP) (see Table 3 footnote for the complete list).

Handling of missing data

The proportion of missing data was low for all covariates. The highest missing rate in the baseline dataset was 9.29% (life satisfaction), and the highest missing rate in the cumulative dataset was 9.00% (life satisfaction); the missing rates for all other variables were below 5% (Supplementary Figures S1 and S2). This study used multiple imputation by chained equations (MICE) to handle missing values. The imputation model included all covariates, exposure variables, and outcome variables, and generated 10 imputed datasets (37). For continuous variables, predictive mean matching was used, and for categorical variables, logistic regression was used. Results from the imputed datasets were combined using Rubin's rules (38).

Statistical analysis

All statistical analyses were performed using R software (version 4.4.1). Two-sided tests were conducted, and a P-value <0.05 was considered statistically significant.

Continuous variables were presented as the median (interquartile range), and categorical variables were presented as counts (percentages). Comparisons between groups were performed using the Kruskal-Wallis test (for continuous variables) and the chi-square test (for categorical variables). Multicollinearity was assessed using the variance inflation factor (VIF) (39, 40).

K-means clustering was used to identify long-term trajectories of TCBI(13). TCBI values from Wave 1 and Wave 3 were z-score standardized before clustering.

Cox proportional hazards models were used to estimate the hazard ratio (HR)

and 95% confidence interval (CI) for new-onset hypertension. Three progressively adjusted models were constructed: Model 1 was unadjusted; Model 2 was adjusted for sex, age, residence, marital status, education, and region; Model 3 was further adjusted based on Model 2 to include lifestyle factors (work status, drinking, smoking, sleep duration, social activity), health status and psychological factors (self-rated health, disability, history of falls, life satisfaction, ADL disability, total cognitive function score, depressive symptoms), comorbidities (lung disease, arthritis, dyslipidemia, liver disease, kidney disease, gastrointestinal disease, diabetes, cardiovascular disease), and laboratory indicators (pulse rate, white blood cell count, platelet count, blood urea nitrogen, fasting blood glucose, creatinine, HbA1c, hemoglobin, C-reactive protein, uric acid, LDL-C, HDL-C) (see Table 3 footnote for the complete list). For baseline TCBI and cumulative TCBI, exposure was analyzed as a continuous variable (per 1-SD increase) and by quartiles; for TCBI trajectory clusters, the HR for cluster 2 was estimated with cluster 1 as the reference. Quartile trend tests were performed by including the median of each quartile as a continuous variable in the model.

Restricted cubic spline (RCS) analysis was used to evaluate the dose-response relationships of baseline TCBI and cumulative TCBI with new-onset hypertension. In the fully adjusted model (Model 3), three knots (at the 10th, 50th, and 90th percentiles) were set, and the Wald test was used to assess the overall association (P for overall) and nonlinearity (P for nonlinearity)(13).

Kaplan–Meier survival curves were generated to compare hypertension-free survival

across quartiles of cumulative TCBI using the log-rank test.

Subgroup analyses were stratified by age, sex, smoking, drinking, region, education, ADL disability, diabetes, cardiovascular disease, work status, arthritis, lung disease, body pain, history of falls, and depression. Interaction P-values were calculated by including interaction terms between the exposure (per 1-SD increase) and each stratification variable in the Cox model.

Sensitivity analyses included: (1) logistic regression models to verify the robustness of the results; (2) complete-case analysis (excluding participants with missing values, i.e., unimputed baseline and cumulative analyses) to assess the impact of multiple imputation on the results; (3) competing risk analysis, treating all-cause death as the competing event, using the Fine-Gray subdistribution hazard model to evaluate and plot cumulative incidence function (CIF) curves (41).

Results

Baseline characteristics

The baseline analysis included 4,777 participants without hypertension at Wave 1 (2011), of whom 1,792 (37.5%) developed new-onset hypertension during follow-up; the cumulative and trajectory analyses included 2,622 participants, among whom 421 (16.1%) developed new-onset hypertension. Detailed baseline characteristics are presented in Tables 1 and 2. In the baseline analysis population, the new-onset hypertension group was older, had a higher proportion of men, lower education, and higher TCBI levels (all $P < 0.05$); in the cumulative analysis population, the differences in age, education, and TCBI remained significant (all $P < 0.05$), but the

difference in sex was no longer statistically significant (P = 0.19). Before statistical modeling, we assessed multicollinearity; all variance inflation factors (VIFs) were <5 (Supplementary Figure S3), indicating no significant multicollinearity.

TCBI trajectory cluster analysis

K-means clustering (k = 2) identified two distinct trajectory patterns based on TCBI values from Wave 1 and Wave 3 (Figure 3). In Cluster 1 (lower trajectory group), mean TCBI increased from 808.03 in 2011 to 1,614.83 in 2015, with a 4-year hypertension incidence of 14.5%. In Cluster 2 (higher trajectory group), mean TCBI increased from 884.96 to 1,818.84, with a hypertension incidence of 18.84%.

Association between different dimensions of TCBI and new-onset hypertension

Cox proportional hazards models showed that all three exposure dimensions were significantly and positively associated with the risk of new-onset hypertension (Table 3, all P < 0.01). In the fully adjusted model (Model 3), each 1-SD increase in baseline TCBI was associated with a 16% higher risk of hypertension (HR = 1.16, 95% CI: 1.10–1.22, P < 0.001); compared with the lowest quartile (Q1), the highest quartile (Q4) was associated with a 61% higher risk (HR = 1.61, 95% CI: 1.38–1.89, P < 0.001), with P for trend <0.001. For each 1-SD increase in cumulative TCBI, the risk increased by 17% (HR = 1.17, 95% CI: 1.05–1.30, P = 0.003); compared with the lowest quartile (Q1), the highest quartile (Q4) had an HR of 1.77 (95% CI: 1.26–2.48, P = 0.001), with P for trend = 0.003. For TCBI trajectories, the high-trajectory group (Cluster 2) had a 36% higher risk than the low-trajectory group (Cluster 1) (HR = 1.36, 95% CI: 1.09–1.70, P = 0.008).

Dose-response relationship: restricted cubic spline analysis and threshold effect analysis

Restricted cubic spline (RCS) analysis revealed a significant nonlinear dose-response relationship between baseline TCBI and new-onset hypertension (P for overall <0.001, P for nonlinearity <0.001; Figure 4A). Threshold effect analysis further identified an inflection point at 1539.29 (Table 4): when TCBI was below this threshold, each 1-unit increase was associated with a 73% higher risk of hypertension (HR = 1.73, 95% CI: 1.35–2.20, P < 0.001); once TCBI exceeded this threshold, the association tended to plateau and was no longer statistically significant (HR = 1.11, 95% CI: 0.99–1.23, P = 0.065). The log-likelihood ratio test confirmed that the piecewise linear model provided a significantly better fit than the standard linear model (P = 0.001).

In contrast, the RCS analysis of cumulative TCBI and new-onset hypertension showed a linear dose-response relationship (P for overall = 0.007, P for nonlinearity = 0.190; Figure 4B), suggesting that the risk increases at a constant rate as cumulative TCBI increases, with no apparent saturation effect.

Kaplan-Meier survival analysis

Kaplan-Meier survival curves were used to compare the cumulative incidence of new-onset hypertension across different TCBI dimensions (Figure 5A–C).

For the baseline TCBI quartile groups (Figure 5A), hypertension-free survival decreased as TCBI quartiles increased (Q1 to Q4), with statistically significant differences among groups (log-rank P < 0.001).

For the cumulative TCBI quartile groups (Figure 5B), the cumulative incidence

increased across TCBI quartiles; the cumulative incidence in the Q4 group was significantly higher than that in the Q1 group, while there was no significant difference between the Q2 and Q3 groups (log-rank $P < 0.001$). The trend test indicated a dose-response relationship between cumulative TCBI and the risk of new-onset hypertension (P for trend = 0.003).

For the two TCBI trajectory clusters (Figure 5C), hypertension-free survival was lower in the high-trajectory group (Cluster 2) than in the low-trajectory group (Cluster 1), and the difference between groups was statistically significant (log-rank $P < 0.05$).

Subgroup analysis

Subgroup analyses were stratified by age, sex, smoking, drinking, region, education, ADL disability, diabetes, cardiovascular disease, work status, arthritis, lung disease, body pain, history of falls, and depression.

Subgroup analyses of baseline TCBI (Figure 6A) showed that, overall, each 1-SD increase in TCBI was associated with a 16% higher risk of new-onset hypertension (HR = 1.16, 95% CI: 1.10–1.22, $P < 0.001$). Across all subgroups, the direction of the effect was consistent (all point estimates >1), and all interaction P-values were >0.05, suggesting that the association between baseline TCBI and hypertension was consistent across subgroups.

Subgroup analyses of cumulative TCBI (Figure 6B) showed that, in most subgroups, cumulative TCBI was positively associated with the risk of new-onset hypertension (HR > 1), and all interaction P-values were >0.05, suggesting that these factors did not modify the association with cumulative TCBI.

Subgroup analyses of TCBI trajectory clusters (Figure 6C) showed that the high-trajectory group was associated with a higher risk of hypertension in most subgroups (HR > 1), with all interaction P-values >0.05, suggesting that the association between trajectory clusters and hypertension was consistent across subgroups.

Sensitivity analysis

Logistic regression analysis yielded results consistent with the primary analysis (Supplementary Table S1): for each 1-SD increase in baseline TCBI, OR = 1.24 (95% CI: 1.16–1.33, P < 0.001); for each 1-SD increase in cumulative TCBI, OR = 1.19 (95% CI: 1.06–1.35, P = 0.004). The shape of the logistic RCS curve was consistent with that of the Cox RCS curve (Supplementary Figures S4 and S5).

Results from the complete-case analysis (excluding participants with missing values and without multiple imputation) were consistent with the main analysis (Supplementary Table S2): for each 1-SD increase in baseline TCBI, HR = 1.16 (95% CI: 1.10–1.22, P < 0.001); for each 1-SD increase in cumulative TCBI, HR = 1.19 (95% CI: 1.06–1.33, P = 0.004), suggesting that multiple imputation did not substantially affect the results.

Competing risk analysis was conducted using the Fine-Gray subdistribution hazard model, with death as the competing event, to further validate the findings regarding cumulative TCBI. The results showed that for each 1-SD increase in cumulative TCBI, the subdistribution hazard ratio (SHR) for new-onset hypertension was 1.14 (95% CI: 1.06–1.22, P < 0.001), consistent with the direction of the positive

association observed in the primary analysis; the SHR for the competing event of death was 0.80 (95% CI: 0.69–0.94, P = 0.005). These results suggest that the positive association between cumulative TCBI and new-onset hypertension remained robust after accounting for the competing event of death (Supplementary Figure S6 shows the cumulative incidence function curve; Supplementary Figures S7 and S8 show the Fine-Gray model forest plots for new-onset hypertension and death, respectively; Supplementary Table S3 summarizes the corresponding SHRs and P-values).

Discussion

Based on the nationally representative CHARLS cohort data, this study systematically evaluated the association between three dimensions—baseline TCBI, time-weighted cumulative TCBI, and TCBI dynamic trajectories identified by K-means clustering—and the risk of new-onset hypertension in middle-aged and older adults in China. The results showed that all three exposure measures were independently and positively associated with the risk of new-onset hypertension: for each 1-SD increase in baseline TCBI, the risk of hypertension increased by 16% (HR = 1.16, 95% CI: 1.10–1.22); each 1-SD increase in cumulative TCBI was associated with a 17% increase in risk (HR = 1.17, 95% CI: 1.05–1.30); and the high-TCBI trajectory group had a 36% higher risk than the low-TCBI trajectory group (HR = 1.36, 95% CI: 1.09–1.70). Notably, baseline TCBI exhibited a nonlinear dose-response relationship with new-onset hypertension (P for nonlinearity < 0.001), with a threshold at 1539.29; below this threshold, an increase in TCBI was associated with a sharp rise in risk, while above this threshold, the association tended to plateau; in contrast, cumulative

TCBI showed a linear association (P for nonlinearity = 0.190). Subgroup analyses revealed consistent effect directions across all subgroups, with all interaction P-values >0.05, suggesting that the association between TCBI and hypertension did not differ significantly among subgroups. Sensitivity analyses (logistic regression, complete-case analysis, and competing risk model) further confirmed the robustness of the results. These findings suggest that TCBI, as a simple and readily available composite nutritional and metabolic indicator, can effectively identify individuals at high risk for hypertension through multidimensional longitudinal assessment, providing a new tool for early risk stratification and primary prevention of hypertension.

Previous studies have suggested that TCBI is associated with cardiovascular outcomes. In a 10-year follow-up study of the MASHAD cohort (n = 9,704), Rezaee et al. found that TCBI was significantly positively associated with cardiovascular mortality (HR = 1.078, 95% CI: 1.012–1.15) (12). A study by Chen et al. based on the CHARLS cohort demonstrated that cumulative TCBI and its dynamic trajectories were independently associated with new-onset cardiometabolic multimorbidity (CMM) [13]. Building on these findings, the present study further confirmed that TCBI is also independently associated with the specific outcome of new-onset hypertension, and the effect sizes (HR = 1.16–1.36) were comparable to those in the aforementioned studies, indicating consistency in the predictive value of TCBI across the spectrum of cardiovascular and metabolic diseases. Regarding nonlinear associations, Chen et al. found a nonlinear association between cumulative TCBI and

CMM risk, with a threshold at 6553.04 (13); Luwen et al. found a nonlinear association between cumulative TCBI and stroke risk, with a threshold of 1263.9(42); the present study found a significant nonlinear association between baseline TCBI and new-onset hypertension, with a clear threshold effect; a piecewise linear model (43) identified a threshold at 1539.29: below this threshold, each 1-unit increase in TCBI was associated with a 73% higher risk of hypertension (HR = 1.73, 95% CI: 1.35–2.20, P < 0.001); above this threshold, the association was no longer significant (HR = 1.11, 95% CI: 0.99–1.23, P = 0.065). This "saturation effect" has important clinical implications: when TCBI was at low-to-moderate levels, an increase in metabolic burden was associated with a sharp rise in the risk of hypertension, suggesting that metabolic intervention at this stage may be more effective; however, once TCBI exceeded a certain threshold, its association with hypertension tended to plateau, at which point the marginal benefit of simply lowering TCBI may be limited, and comprehensive management of other risk factors must also be addressed. Although the location of the threshold varied across different outcomes in the aforementioned studies, these consistent findings suggest that a universal "saturation effect" may exist between metabolic markers and cardiovascular outcomes, providing a theoretical basis for setting metabolic intervention targets in clinical practice.

In terms of trajectory analysis, Yan et al., based on the CHARLS cohort, found a linear dose-response relationship between cumulative triglyceride-glucose index-body mass index (TyG-BMI) and the risk of new-onset hypertension, with an HR of 2.15 (95% CI: 1.43–3.23) in the highest quartile (44); this effect size was higher than that

for cumulative TCBI in the present study (HR = 1.17). This difference may reflect that TyG-BMI, as an indicator of insulin resistance, is more directly associated with hypertension, whereas TCBI, as a composite nutritional and metabolic indicator, encompasses a broader range of pathways, which may result in a more diluted effect. Nevertheless, given its simplicity (requiring only lipid profiles and body weight), TCBI may be more feasible for large-scale community-based screening.

The mechanisms through which TCBI influences the onset and progression of hypertension involve multiple interrelated biological pathways. First, triglycerides represent one of the core components of TCBI; long-term hypertriglyceridemia can promote the formation of triglyceride-rich lipoproteins and their remnant particles, which directly promote atherogenesis through endothelial dysfunction and activation of inflammatory pathways(45-47) . Second, total cholesterol—particularly low-density lipoprotein cholesterol (LDL-C)—plays a key role in the deposition, oxidative modification, and foam cell formation within the arterial intima, driving plaque formation and progression (48, 49) Dyslipidemia directly leads to elevated systolic blood pressure by increasing arterial stiffness and impairing vascular compliance. Third, body weight, as the third component of TCBI, reflects long-term energy imbalance and adipose tissue expansion, particularly the accumulation of visceral fat. As an active endocrine organ, adipose tissue secretes substantial amounts of free fatty acids, pro-inflammatory cytokines (such as tumor necrosis factor-α and interleukin-6), and chemokines, while simultaneously reducing the secretion of adiponectin, which has a vasoprotective effect(50, 51). This state of “adipose

inflammation" is a major source of systemic chronic low-grade inflammation, which can directly impair endothelial function, promote monocyte adhesion and foam cell formation, and accelerate the progression of atherosclerosis(52, 53). Furthermore, obesity is closely associated with insulin resistance, which directly leads to elevated blood pressure through multiple mechanisms (such as sympathetic nervous system activation and increased renal sodium reabsorption)(54-56).By integrating these three components into a single index, TCBI may more comprehensively capture the overall activation status of the "dyslipidemia–obesity–insulin resistance" metabolic axis, thereby predicting the risk of hypertension more effectively than individual indicators.

The introduction of cumulative exposure and trajectory analysis represents the core methodological innovation of this study. Hypertension develops gradually under cumulative metabolic stress spanning several years to several decades (21, 57, 58); a single baseline measurement may not adequately reflect changes in an individual's metabolic burden throughout the follow-up period, nor can it distinguish between the two distinct exposure patterns of "persistently high levels" and "intermittent fluctuations"(44). Cumulative TCBI more accurately reflects long-term metabolic burden by integrating both the intensity and duration of exposure(23). In this study, each 1-SD increase in cumulative TCBI was associated with a 17% higher risk of hypertension, suggesting that the cumulative effects of long-term metabolic burden should not be overlooked. More importantly, trajectory analysis (K-means clustering) extended the assessment of an individual's metabolic status from a "static point" based on a single measurement to a "dynamic line" tracked longitudinally, identifying

two distinct TCBI change patterns. Both groups exhibited an upward trend, but differed in their starting points and rates of increase: the low-trajectory group (TCBI rose from 808.03 to 1,614.83, with a 4-year hypertension incidence of 14.5%) and the high-trajectory group (rising from 884.96 to 1,821.70, with a 4-year incidence of 18.84%). The risk in the high-trajectory group was 36% higher than in the low-trajectory group (HR = 1.36), suggesting that a persistently high metabolic trajectory significantly increases the risk of hypertension(21). Although TCBI in the low-trajectory group was not high and the absolute risk was relatively low, its 4-year incidence of hypertension still reached 14.5%, indicating that even a metabolic trajectory with a low baseline TCBI should not be overlooked if it continues to rise. This highlights the clinical value of trajectory analysis: it identifies individuals whose baseline metabolic levels are not high but who exhibit a sustained upward trend over time. Through dynamic monitoring, clinicians can implement early interventions before their risk accumulates further and they shift into the high-trajectory group, thereby more effectively preventing hypertension(21, 59).

The findings of this study have multiple implications for clinical practice and public health. First, TCBI can be calculated using only three parameters—triglycerides, total cholesterol, and body weight—from routine physical examination, at no additional cost, making it particularly suitable for large-scale hypertension risk screening in primary care settings and resource-limited environments(59). Second, the multidimensional assessment framework of “baseline level–cumulative burden–dynamic trajectory” proposed in this study provides a more

refined tool for hypertension risk stratification: a single baseline TCBI can be used for initial screening, cumulative TCBI can be used to assess long-term risk, and trajectory analysis can be used for dynamic monitoring and early warning(21, 23). Third, the threshold of 1539.29 identified in this study provides a reference threshold for clinical practice: below this threshold, the focus should be on metabolic intervention to prevent progression beyond it; above this threshold, comprehensive management of other risk factors is warranted. Finally, trajectory analysis suggests that for individuals with a persistently increasing TCBI trajectory, lifestyle interventions (60)and regular monitoring(21, 44) should be strengthened, even if their current values remain within the normal range.

Strengths and Limitations

This study has the following strengths. First, to the best of our knowledge, this is the first prospective cohort study to systematically evaluate the association between the three dimensions of the TCBI (baseline, cumulative, and trajectory) and new-onset hypertension(13), providing new epidemiological evidence for the application of the TCBI in hypertension risk prediction. Second, the study is based on CHARLS, a nationally representative, large-sample prospective cohort with a long follow-up period and sufficient statistical power, ensuring good generalizability of the results. Third, methodologically, rigorous statistical approaches were employed, including multiple interpolation methods for handling missing values, VIF analysis for diagnosing multicollinearity, and various sensitivity analyses (logistic regression,

complete-case analysis, and competitive risk models), ensuring the robustness and reliability of the results. Fourth, through RCS analysis and piecewise linear models, a clinically actionable threshold (1,539.29) was identified, providing quantitative evidence for the application of TCBI in clinical practice.

This study also has several limitations. First, the diagnosis of hypertension relies in part on self-reported information regarding medical diagnoses and medication use, which may be subject to recall bias or reporting inaccuracies. Furthermore, blood pressure measurements were based on the average of multiple readings taken during a single visit, rather than multiple measurements taken on different days, which may have led to some misclassification bias due to white-coat hypertension or blood pressure variability. Although this study incorporated both blood pressure measurements and information on antihypertensive medication use to reduce the risk of misclassification, the influence of the aforementioned biases cannot be completely ruled out. Second, although the study adjusted for a wide range of covariates—including demographic factors, lifestyle, comorbidities, and laboratory indicators—residual confounding (such as detailed dietary habits, physical activity intensity, and genetic factors) may still exist. Third, the calculation of cumulative TCBI was based solely on two time points (2011 and 2015); assuming a linear change between the two measurements may not fully capture the complex fluctuations in metabolic trajectories. More frequent measurements would help to characterize metabolic dynamics in greater detail. Fourth, the study population was limited to middle-aged and older adults in China; the applicability of the findings to different

ethnic groups and age cohorts requires further validation. Fifth, as this study employed an observational design—despite utilizing a prospective cohort and various sensitivity analyses—it cannot establish causal relationships; future research could further validate these associations using causal inference methods such as Mendelian randomization.

Conclusion

Based on the nationally representative CHARLS cohort, to the best of our knowledge, this is the first study to systematically evaluate the association between TCBI and new-onset hypertension across three dimensions: baseline TCBI, time-weighted cumulative TCBI, and TCBI trajectories identified by K-means clustering. The results showed that all three exposure dimensions were independently and positively associated with the risk of new-onset hypertension. Baseline TCBI exhibited a significant nonlinear dose-response relationship with new-onset hypertension, with a threshold at 1539.29. Cumulative TCBI and trajectory analyses further suggested that the accumulation of long-term metabolic burden and a sustained upward trend also increased the risk of hypertension. These findings suggest that TCBI, as a simple and readily available composite nutritional and metabolic indicator, can effectively identify individuals at high risk for hypertension through multidimensional longitudinal assessment, providing a new tool for early risk stratification and primary prevention of hypertension. Future studies should validate the predictive value of TCBI in different populations and explore whether interventions targeting TCBI can reduce the incidence of hypertension.

Abbreviations

ADL: activities of daily living; BMI: body mass index; BW: body weight; CESD-10: 10-item Center for Epidemiologic Studies Depression Scale; CHARLS: China Health and Retirement Longitudinal Study; CI: confidence interval; CIF: cumulative incidence function; CMM: cardiometabolic multimorbidity; CRP: C-reactive protein; CumBMI: cumulative body mass index; CumCRP: cumulative C-reactive protein; CumHbA1c: cumulative glycated hemoglobin; cumTCBI: time-weighted cumulative triglyceride-total cholesterol-body weight index; CVD: cardiovascular disease; HbA1c: glycated hemoglobin; HDL-C: high-density lipoprotein cholesterol; HR: hazard ratio; IRB: Institutional Review Board; LDL-C: low-density lipoprotein cholesterol; MICE: multiple imputation by chained equations; OR: odds ratio; RCS: restricted cubic spline; SHR: subdistribution hazard ratio; TC: total cholesterol; TCBI: triglyceride-total cholesterol-body weight index; TG: triglyceride; TyG-BMI: triglyceride-glucose index-body mass index; VIF: variance inflation factor.

Acknowledgements

We sincerely thank the participants and staff of the CHARLS for their valuable contributions, and all members who contributed to this work.

Author contributions

WJH, LC, YC, and HBG contributed to the study conception and design. WJH, HBG ,

and LC organized the data and conducted the statistical analyses. WJH and HBG contributed to the interpretation of the results and revision of the manuscript. WJH and HBG wrote and edited the manuscript. All authors have reviewed and approved the final version of the manuscript.

Funding

This study was not funded

Availability of data and materials

The datasets generated and/or analysed during the current study are available in the China Health and Retirement Longitudinal Study (CHARLS) repository, http://charls.pku.edu.cn. The CHARLS data are publicly available to researchers who submit a data use application and obtain approval from the CHARLS team. The statistical code used in this study is available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The CHARLS study was approved by the Biomedical Ethics Review Committee of Peking University (IRB approval number: IRB00001052-11015). All participants provided written informed consent. This study used publicly available de-identified data from CHARLS, and therefore additional ethical approval was not required.

Consent for publication

Not applicable.

Clinical trial number

Not applicable.

Competing interests

The authors declare that they have no competing interests.

1. Wang S, Lv J, Cao X. Assessing the Global, Regional, and National Burden of Childhood Lower Respiratory Infections From Nonexclusive Breastfeeding, 1990-2021: Global Burden of Disease, Injuries, and Risk Factors Study 2021 Analysis and Implications for Public Health Strategies. J Hum Lact. 2026:8903344261431673.

2. Mensah GA, Fuster V, Murray CJL, Roth GA. Global Burden of Cardiovascular Diseases and Risks, 1990-2022. J Am Coll Cardiol. 2023;82(25):2350-473.

3. World Health Organization. Global health estimates 2021: deaths by cause, age, sex, by country and by region, 2000-2021. Geneva; 2024.

4. World Health Organization. Global report on hypertension: the race against a silent killer. Geneva:World Health Organization; 2023.

5. Wang Z, Chen Z, Zhang L, Wang X, Hao G, Zhang Z, et al. Status of Hypertension in China: Results From the China Hypertension Survey, 2012-2015. Circulation. 2018;137(22):2344-56.

6. Zhou T, Wang Y, Zhang H, Wu C, Tian N, Cui J, et al. Primary care institutional characteristics associated with hypertension awareness, treatment, and control in the China PEACE-Million Persons Project and primary health-care survey: a cross-sectional study. Lancet Glob Health. 2023;11(1):e83-e94.

7. Akyar S, Dağdeviren HN. Systemic immune-inflammation index: a novel tool for hypertension management in primary care. BMC Prim Care. 2026;27(1).

8. Ren X, Chen M, Lian L, Xia H, Chen W, Ge S, et al. The triglyceride-glucose index is associated with a higher risk of hypertension: evidence from a cross-sectional study of Chinese adults and meta-analysis of epidemiology studies. Front Endocrinol (Lausanne). 2025;16:1516328.

9. Doi S, Iwata H, Wada H, Funamizu T, Shitara J, Endo H, et al. A novel and simply calculated nutritional index serves as a useful prognostic indicator in patients with coronary artery disease. Int J Cardiol. 2018;262:92-8.

10. Li G, Li S. Exploring the prognostic value of the novel nutritional index for in-hospital mortality in acute coronary syndrome: a sex-specific analysis. Front Med (Lausanne). 2025;12:1498260.

11. Maruyama S, Ebisawa S, Miura T, Yui H, Kashiwagi D, Nagae A, et al. Impact of nutritional index on long-term outcomes of elderly patients with coronary artery disease: sub-analysis of the SHINANO 5 year registry. Heart Vessels. 2021;36(1):7-13.

12. Rezaee M, Kamrani F, Imannezhad M, Shahri HH, Saihood WK, Rezvani A, et al. Beyond traditional metrics: evaluating the triglyceride-total cholesterol-body weight index (TCBI) in cardiovascular risk assessment. BMC Cardiovasc Disord. 2025;25(1):39.

13. Chen X, Wang C, Huang Q, Luo Y, Wu Z, Chen J, et al. Associations of cumulative exposure and dynamic trajectories of the triglyceride-total cholesterol-body weight index with new-onset cardiometabolic multimorbidity in middle-aged and older Chinese adults: evidence from a nationwide prospective cohort study. Cardiovasc Diabetol. 2026;25(1).

14. Allen NB, Khan SS. Blood Pressure Trajectories Across the Life Course. Am J Hypertens. 2021;34(3):234-41.

15. Emberson JR, Whincup PH, Morris RW, Walker M, Lowe GD, Rumley A. Extent of regression dilution for established and novel coronary risk factors: results from the British Regional Heart Study. Eur J Cardiovasc Prev Rehabil. 2004;11(2):125-34.

16. Lewington S, Thomsen T, Davidsen M, Sherliker P, Clarke R. Regression dilution bias in blood total and high-density lipoprotein cholesterol and blood pressure in the Glostrup and Framingham prospective studies. J Cardiovasc Risk. 2003;10(2):143-8.

17. Avrahami D, Zahger D, Arbel R, Rokach L, Razi T, Sergienko R, et al. Effect of cumulative blood pressure exposure on long-term cardiovascular outcomes in the community, a nationwide cohort study. Am J Med. 2026;139(10):1330-6.e2.

18. Wang J, Zhang S, Jiao Y, Zheng L, Sun Y, Sun Z. Cumulative exposure to elevated blood pressure better predicts cardiovascular disease risk in rural Chinese adults. Front Public Health. 2022;10:1006220.

19. Xu J, Yang Y, Hu Y, Song Y, Ye X, Chen YM, et al. Associations between dynamic change of Chinese visceral adiposity index and hypertensive co-morbidities in the middle-aged and elderly population: a Chinese prospective cohort study. Front Nutr. 2025;12:1557868.

20. Amouzegar A, Honarvar M, Masoumi S, Khalili D, Azizi F, Mehran L. Trajectory patterns of metabolic syndrome severity score and risk of type 2 diabetes. J Transl Med. 2023;21(1):750.

21. Dou J, Zhang S, Ding C, Li H, Zhang P. Cumulative exposure and dynamic trajectories of the C-reactive protein-triglyceride-glucose index (CTI) versus the cholesterol, high-density lipoprotein, and glucose index (CHG) for incident hypertension prediction: a national cohort study. Cardiovasc Diabetol. 2026;25(1).

22. Wang H, Song G, Li G, Hu D, Zhang Y, Sun Z, et al. Associations of cumulative exposure and dynamic trajectories of the cholesterol-HDL-glucose index and its obesity-related derivatives with cardiovascular disease: a nationwide prospective cohort study. Cardiovasc Diabetol. 2026;25(1).

23. Zhao Y, Du D, Liu Z. Cumulative exposure to remnant cholesterol and new-onset hypertension in middle-aged and older adults: a nationwide prospective cohort study with mediation analysis. J Hum Hypertens. 2026;40(2):127-35.

24. Zhao Y, Hu Y, Smith JP, Strauss J, Yang G. Cohort profile: the China Health and Retirement Longitudinal Study (CHARLS). Int J Epidemiol. 2014;43(1):61-8.

25. 2018 Chinese Guidelines for Prevention and Treatment of Hypertension-A report of the Revision Committee of Chinese Guidelines for Prevention and Treatment of Hypertension. J Geriatr Cardiol. 2019;16(3):182-241.

26. Chen X, Liu S, Chu J, Hu W, Sun N, Shen Y. Joint effect of elevated-c-reactive protein level and hypertension on new-onset stroke: A nationwide prospective cohort study of CHARLS. Front Public Health. 2022;10:919506.

27. Clark TG, Bradburn MJ, Love SB, Altman DG. Survival analysis part I: basic concepts and first analyses. Br J Cancer. 2003;89(2):232-8.

28. Grimes DA, Schulz KF. Cohort studies: marching towards outcomes. Lancet. 2002;359(9303):341-5.

29. Zhang Y LH, Wu Z. Cumulative exposure of the combined triglyceride-glucose and frailty index for incident hypertension prediction in middle-aged and older adults: a national cohort study. BMC Cardiovasc Disord. 2026.

30. Suissa S. Immortal time bias in pharmaco-epidemiology. Am J Epidemiol. 2008;167(4):492-9.

31. Hernán MA, Robins JM. Using Big Data to Emulate a Target Trial When a Randomized Trial Is Not Available. Am J Epidemiol. 2016;183(8):758-64.

32. Lindsey JC, Ryan LM. Tutorial in biostatistics methods for interval-censored data. Stat Med. 1998;17(2):219-38.

33. Fu H, Si L, Guo R. What Is the Optimal Cut-Off Point of the 10-Item Center for Epidemiologic Studies Depression Scale for Screening Depression Among Chinese Individuals Aged 45 and Over? An Exploration Using Latent Profile Analysis. Front Psychiatry. 2022;13:820777.

34. Andresen EM, Malmgren JA, Carter WB, Patrick DL. Screening for depression in well older adults: evaluation of a short form of the CES-D (Center for Epidemiologic Studies Depression Scale). Am J Prev Med. 1994;10(2):77-84.

35. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2025. Diabetes Care. 2025;48(1 Suppl 1):S27-s49.

36. Zhao M, Cheng Y, Li M, Zhang W, Ji J, Lu F. Temporal changes in lipid concentrations and the prevalence of dyslipidemia among individuals with diabetes, prediabetes, and normal blood glucose from 2011 to 2015. Lipids Health Dis. 2024;23(1):394.

37. White IR, Royston P, Wood AM. Multiple imputation using chained equations: Issues and guidance for practice. Stat Med. 2011;30(4):377-99.

38. Marshall A, Altman DG, Holder RL, Royston P. Combining estimates of interest in prognostic modelling studies after multiple imputation: current practice and guidelines. BMC Med Res Methodol. 2009;9:57.

39. He ZY, Zhan ZW, Zhou LJ, Tang J, Chen C, Yang QJ, et al. Development and validation of a clinical factor-based nomogram for predicting imaging-defined cardiopulmonary abnormality risk in an asymptomatic screening population. World J Radiol. 2026;18(7):123894.

40. Xu Z, Zhang R, Chen Q, Peng G, Luo S, Liu C, et al. Machine learning algorithms for risk factor selection with application to 60-day sepsis morbidity risk for a geriatric hip fracture cohort. BMC Geriatr. 2025;25(1):591.

41. Austin PC, Fine JP. Practical recommendations for reporting Fine-Gray model analyses for competing risk data. Stat Med. 2017;36(27):4391-400.

42. Luwen H, Yunwei Z, Lei X, Linlin L, Ming Y. Unraveling the role of cumulative triglyceride-total cholesterol-body weight index in stroke development: evidence from the CHARLS cohort. Front Med (Lausanne). 2025;12:1616520.

43. Pastor-Barriuso R, Guallar E, Coresh J. Transition models for change-point estimation in logistic regression. Stat Med. 2003;22(7):1141-62.

44. Yan J, Zhang MZ, He QQ. Association of changes and cumulative measures of triglyceride-glucose index-body mass index with hypertension risk: a prospective cohort study. BMC Public Health. 2024;24(1):2652.

45. Cruz-Bautista I, Escamilla-Núñez C, Flores-Jurado Y, Rojas-Martínez R, Elías López D, Muñoz-Hernández L, et al. Distribution of triglyceride-rich lipoproteins and their remnants and their contribution to cardiovascular risk in the Mexican population. J Clin Lipidol. 2026;20(5):938-46.

46. Peng X, Wu H. Inflammatory Links Between Hypertriglyceridemia and Atherogenesis. Curr Atheroscler Rep. 2022;24(5):297-306.

47. Bai M, Liao J, Wang Y, Liang M, Wang C, Zhang J, et al. Remnant cholesterol and all-cause mortality risk: findings from the National Health and Nutrition Examination Survey, 2003-2015. Front Endocrinol (Lausanne). 2024;15:1417228.

48. Borén J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights: a consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J. 2020;41(24):2313-30.

49. Khatana C, Saini NK, Chakrabarti S, Saini V, Sharma A, Saini RV, et al. Mechanistic Insights into the Oxidized Low-Density Lipoprotein-Induced Atherosclerosis. Oxid Med Cell Longev. 2020;2020:5245308.

50. Fantuzzi G. Adipose tissue, adipokines, and inflammation. J Allergy Clin Immunol. 2005;115(5):911-9; quiz 20.

51. Ouchi N, Kihara S, Funahashi T, Matsuzawa Y, Walsh K. Obesity, adiponectin and vascular inflammatory disease. Curr Opin Lipidol. 2003;14(6):561-6.

52. Gustafson B. Adipose tissue, inflammation and atherosclerosis. J Atheroscler Thromb. 2010;17(4):332-41.

53. Niculescu R, Stoian A, Arbănași EM, Russu E, Babă DF, Manea A, et al. The Dual Role of Perivascular Adipose Tissue in Vascular Homeostasis and Atherogenesis: From Physiology to Pathological Implications. Int J Mol Sci. 2025;26(17).

54. Xiao J, Wen S, Huang Y, He X, Weng Y, Yu H. The Role of Insulin Resistance in Cardiorenal Metabolic Syndrome. Cardiol Rev. 2025.

55. Kosmas CE, Bousvarou MD, Kostara CE, Papakonstantinou EJ, Salamou E, Guzman E. Insulin resistance and cardiovascular disease. J Int Med Res. 2023;51(3):3000605231164548.

56. Ashraf FUN, Ghouri K, Someshwar F, Kumar S, Kumar N, Kumari K, et al. Insulin Resistance and Coronary Artery Disease: Untangling the Web of Endocrine-Cardiac Connections. Cureus. 2023;15(12):e51066.

57. Iiskala F, Meng Y, Juonala M, Magnussen CG, Pahkala K, Hutri N, et al. Life-Course Blood Pressure Levels, 38-Year Tracking, and Prediction of Hypertension. J Am Heart Assoc. 2026;15(1):e041805.

58. Ramezankhani A, Azizi F, Momenan AA, Hadaegh F. Sex Differences in Cumulative Exposure to Metabolic Risk Factors Before Hypertension Onset: The Cohort of the Tehran Lipid and Glucose Study. J Am Heart Assoc. 2021;10(23):e021922.

59. Luo F, Cao Y. A novel nutritional index for hypertension risk and prediction: Cross-sectional, prospective, and trajectory evidence from the English Longitudinal Study of Ageing. J Int Med Res. 2026;54(7):3000605261470980.

60. Mancia G, Kreutz R, Brunström M, Burnier M, Grassi G, Januszewicz A, et al. 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023;41(12):1874-2071.

Table 1. Baseline characteristics of the study population in the baseline analysis (N = 4,777)

Characteristic	Overall(N=4777)	Non-Hypertension (n=2985)	New-onset Hypertension (n=1792)	P-value
Categorical variable, n(%)				
Age				<0.001
≥60	1713(35.86)	875(29.31)	838(46.76)	
＜60	3064(64.14)	2110(70.69)	954(53.24)	
Sex				0.03
Male	2207(46.20)	1343(44.99)	864(48.21)	
Female	2570(53.80)	1642(55.01)	928(51.79)	
Education				<0.001
High school or above	484(10.13)	344(11.52)	140(7.81)	
Middle school	939(19.66)	615(20.60)	324(18.08)	
Primary school	1043(21.83)	636(21.31)	407(22.71)	
Illiterate	2311(48.38)	1390(46.57)	921(51.40)	
Marital				<0.001
Married	4151(86.90)	2631(88.14)	1520(84.82)	
Unmarried	626(13.10)	354(11.86)	272(15.18)	
Residence				0.31
Urban	3254(68.12)	2017(67.57)	1237(69.03)	
Rural	1523(31.88)	968(32.43	555(30.97)	
Region				0.27
East	2027(42.43)	1286(43.08)	741(41.35)	
Central	1591(33.31)	964(32.29)	627(34.99)	
West	857(17.94)	547(18.32)	310(17.30)	

Characteristic	Overall(N=4777)	Non-Hypertension (n=2985)	New-onset Hypertension (n=1792)	P-value
Categorical variable, n(%)				
Northeast	302(6.32)	188(6.30)	114(6.36)	
SRH				0.2
Very good	174(3.64)	105(3.52)	69(3.85)	
Good	981(20.54)	585(19.60)	396(22.10)	
Fair	2476(51.83)	1563(52.36)	913(50.95)	
Poor	854(17.88)	539(18.06)	315(17.58)	
Very poor	292(6.11)	193(6.47)	99(5.52)	
Smoking				0.02
Current	347(7.26)	193(6.47)	154(8.59)	
Former	1480(30.98)	922(30.89)	558(31.14)	
Never	2950(61.75)	1870(62.65)	1080(60.27)	
Drinking				0.02
Current	320(6.70)	188(6.30)	132(7.37)	
Former	1505(31.51)	908(30.42)	597(33.31)	
Never	2952(61.80)	1889(63.28)	1063(59.32)	
Life satisfaction				0.86
Completely satisfied	71(1.49)	44(1.47)	27(1.51)	
Very satisfied	899(18.82)	553(18.53)	346(19.31)	
Somewhat satisfied	3017(63.16)	1897(63.55)	1120(62.50)	
Somewhat dissatisfied	674(14.11)	423(14.17)	251(14.01)	
Completely dissatisfied	116(2.43)	68(2.28)	48(2.68)	
ADL				<0.001
No ADL disability	4112(86.08)	2618(87.71)	1494(83.37)	
Having ADL disability	665(13.92)	367(12.29)	298(16.63)	

Characteristic	Overall(N=4777)	Non-Hypertension (n=2985)	New-onset Hypertension (n=1792)	P-value
Categorical variable, n(%)				
Fall down				0.1
Yes	761(15.93)	455(15.24)	306(17.08)	
No	4016(84.07)	2530(84.76)	1486(82.92)	
Body pain				0.03
Yes	1634(34.21)	987(33.07)	647(36.10)	
No	3143(65.79)	1998(66.93)	1145(63.90)	
Social activity				0.99
Yes	2432(50.91)	1519(50.89)	913(50.95)	
No	2345(49.09)	1466(49.11)	879(49.05)	
Work status				<0.001
Yes	3498(73.23)	2251(75.41)	1247(69.59)	
No	1279(26.77)	734(24.59)	545(30.41)	
Disability				0.06
Yes	748(15.66)	444(14.87)	304(16.96)	
No	4029(84.34)	2541(85.13)	1488(83.04)	
Depression				0.13
Yes	1755(36.74)	1072(35.91)	683(38.11)	
No	3022(63.26)	1913(64.09)	1109(61.89)	
CVD				<0.001
Yes	468(9.80)	251(8.41)	217(12.11)	
No	4309(90.20)	2734(91.59)	1575(87.89)	
Diabetes				<0.001
Yes	182(3.81)	86(2.88)	96(5.36)	
No	4595(96.19)	2899(97.12)	1696(94.64)	

Characteristic	Overall(N=4777)	Non-Hypertension (n=2985)	New-onset Hypertension (n=1792)	P-value
Categorical variable, n(%)				
Lung disease				<0.001
Yes	562(11.76)	303(10.15)	259(14.45)	
No	4215(88.24)	2682(89.85)	1533(85.55)	
Arthritis				0.05
Yes	1630(34.12)	987(33.07)	643(35.88)	
No	3147(65.88)	1998(66.93)	1149(64.12)	
Dyslipidemia				0.18
Yes	300(6.28)	176(5.90)	124(6.92)	
No	4477(93.72)	2809(94.10)	1668(93.08)	
Liver disease				0.42
Yes	186(3.89)	122(4.09)	64(3.57)	
No	4591(96.11)	2863(95.91)	1728(96.43)	
Kidney disease				0.39
Yes	326(6.82)	196(6.57)	130(7.25)	
No	4451(93.18)	2789(93.43)	1662(92.75)	
Stomach disease				0.03
Yes	1366(28.60)	886(29.68)	480(26.79)	
No	3411(71.40)	2099(70.32)	1312(73.21)	
Continuous variable, Median(IQR)				
Age, years	56.00(50.00-62.00)	55.00(49.00-61.00)	59.00(53.00-66.00)	<0.001
Sleep duration, hours	7.00(5.00-8.00)	7.00(5.00-8.00)	6.00(5.00-8.00)	0.01
CESD_10	7.00(3.00-12.00)	7.00(3.00-12.00)	8.00(4.00-12.00)	0.01
SBP, mmHg	118.67(109.67-127.67)	115.33(107.67-123.67)	124.33(115.67-132.00)	<0.001

Characteristic	Overall(N=4777)	Non-Hypertension (n=2985)	New-onset Hypertension (n=1792)	P-value
Categorical variable, n(%)				
DBP, mmHg	70.67(64.67-76.67)	69.33(63.33-75.33)	73.33(66.67-79.00)	<0.001
Pulse, bpm	71.33(65.00-77.67)	70.67(64.67-77.00)	72.33(65.67-79.00)	<0.001
Total cognition	11.50(8.00-14.00)	12.00(8.50-14.50)	11.00(7.00-14.00)	<0.001
BMI, kg/m²	22.60(20.52-24.89)	22.38(20.37-24.63)	22.98(20.77-25.49)	<0.001
Weight, kg	56.40(50.20-63.60)	56.00(50.10-63.00)	57.30(50.60-64.80)	<0.001
HbA1c, %	5.10(4.90-5.40)	5.10(4.90-5.40)	5.20(4.90-5.50)	<0.001
CRP, mg/L	0.88(0.50-1.80)	0.82(0.48-1.69)	0.96(0.53-2.01)	<0.001
FPG, mg/dL	100.98(93.60-110.34)	99.90(92.88-109.08)	102.60(94.68-113.22)	<0.001
HDL_C, mg/dL	50.64(41.37-61.08)	51.03(42.14-61.08)	50.26(40.59-61.08)	0.27
LDL_C, mg/dL	113.66(92.78-134.92)	112.50(92.40-133.76)	114.82(93.17-138.02)	0.03
TC, mg/dL	188.27(165.85-213.02)	186.34(165.46-210.31)	190.98(166.62-216.59)	<0.001
TG, mg/dL	98.24(71.68-143.37)	95.58(69.92-136.29)	102.66(75.22-150.45)	<0.001
UA, mg/dL	4.17(3.49-4.97)	4.12(3.46-4.89)	4.27(3.52-5.16)	<0.001
WBC, ×10⁹/L	5.90(4.90-7.10)	5.90(4.90-7.10)	5.83(4.90-7.20)	0.72
PLT, ×10⁹/L	208.00(162.00-254.00)	208.00(163.00-252.00)	208.00(162.00-258.25)	0.63
BUN, mg/dL	15.15(12.55-18.12)	15.10(12.60-17.90)	15.21(12.46-18.47)	0.43
SCR, mg/dL	0.75(0.64-0.87)	0.75(0.63-0.86)	0.75(0.64-0.88)	0.03
HGB, g/dL	14.10(12.90-15.40)	14.10(12.90-15.40)	14.20(13.00-15.50)	0.01
TCBI	1034.59(697.75-1623.36)	994.51(678.18-1520.71)	1116.47(750.38-1778.74)	<0.001

Data are presented as n (%) for categorical variables and median (IQR) for continuous variables. Comparisons between groups were performed using the χ^2 test for categorical variables and the Kruskal-Wallis test for continuous variables. SRH, self-rated health; ADL, activities of daily living; CESD-10,

10-item Center for Epidemiologic Studies Depression Scale; SBP, systolic blood pressure; DBP, diastolic blood pressure; CVD, cardiovascular disease; CumBMI, cumulative body mass index; CumHbA1c, cumulative glycated hemoglobin A1c; CumCRP, cumulative C-reactive protein; CumUA, cumulative uric acid; CumLDL-C, cumulative low-density lipoprotein cholesterol; CumHDL-C, cumulative high-density lipoprotein cholesterol; CumTC, cumulative total cholesterol; CumTG, cumulative triglyceride; TCBI, triglyceride–total cholesterol–body weight index; CumTCBI, cumulative TCBI.

Table 2. Baseline characteristics of the study population in the cumulative and trajectory analyses (N = 2,622)

Characteristic	**Overall(N=2622)**	**Non-Hypertension(n=2201)**	**New-onset Hypertension(n=421)**	**P-value**
Categorical variable, n(%)				
Age group				<0.001
＜60	1734(66.13)	1481(67.29)	253(60.10)	
≥60	888(33.87)	720(32.71)	168(39.90)	
Sex				0.19
male	1157(44.13)	984(44.71)	173(41.09)	
Female	1465(55.87)	1217(55.29)	248(58.91)	
Education				<0.001
High school or above	263(10.03)	237(10.77)	26(6.18)	
Middle school	526(20.06)	457(20.76)	69(16.39)	
Primary school	571(21.78)	478(21.72)	93(22.09)	
Illiterate	1262(48.13)	1029(46.75)	233(55.34)	
Marital				0.44
Married	2330(88.86)	1961(89.10)	369(87.65)	
Unmarried	292(11.14)	240(10.90)	52(12.35)	
Residence				0.02
Urban	1805(68.84)	1494(67.88)	311(73.87)	
Rural	817(31.16)	707(32.12)	110(26.13)	
Region				0.56
East	1126(42.94)	958(43.53)	168(39.90)	
Central	858(32.72)	710(32.26)	148(35.15)	
West	476(18.15)	398(18.08)	78(18.53)	
Northeast	162(6.18)	135(6.13)	27(6.41)	
SRH				0.06
Very good	88(3.36)	72(3.27)	16(3.80)	
Good	559(21.32)	452(20.54)	107(25.42)	
Fair	1369(52.21)	1149(52.20)	220(52.26)	
Poor	455(17.35)	395(17.95)	60(14.25)	
Very poor	151(5.76)	133(6.04)	18(4.28)	
Smoking				0.81
Current	171(6.52)	146(6.63)	25(5.94)	

Former	781(29.79)	658(29.90)	123(29.22)	
Never	1670(63.69)	1397(63.47)	273(64.85)	
Drinking				0.85
Current	159(6.06)	136(6.18)	23(5.46)	
Former	800(30.51)	671(30.49)	129(30.64)	
Never	1663(63.42)	1394(63.33)	269(63.90)	
Life satisfaction				0.28
Completely satisfied	40(1.53)	32(1.45)	8(1.90)	
Very satisfied	496(18.92)	419(19.04)	77(18.29)	
Somewhat satisfied	1656(63.16)	1395(63.38)	261(62.00)	
Somewhat dissatisfied	368(14.04)	309(14.04)	59(14.01)	
Completely dissatisfied	62(2.36)	46(2.09)	16(3.80)	
ADL				<0.001
No ADL disability	2271(86.61)	1926(87.51)	345(81.95)	
Having ADL disability	351(13.39)	275(12.49)	76(18.05)	
Fall down				0.01
Yes	415(15.83)	331(15.04)	84(19.95)	
No	2207(84.17)	1870(84.96)	337(80.05)	
Body pain				0.01
Yes	897(34.21)	730(33.17)	167(39.67)	
No	1725(65.79)	1471(66.83)	254(60.33)	
Social activity				0.61
Yes	1297(49.47)	1094(49.70)	203(48.22)	
No	1325(50.53)	1107(50.30)	218(51.78)	
Work status				0.89
No	619(23.61)	518(23.53)	101(23.99)	
Yes	2003(76.39)	1683(76.47)	320(76.01)	
Disability				0.88
Yes	383(14.61)	320(14.54)	63(14.96)	
No	2239(85.39)	1881(85.46)	358(85.04)	
Depression				<0.001
Yes	983(37.49)	794(36.07)	189(44.89)	
No	1639(62.51)	1407(63.93)	232(55.11)	
CVD				0.01
Yes	256(9.76)	200(9.09)	56(13.30)	
No	2366(90.24)	2001(90.91)	365(86.70)	
Diabetes				0.02
Yes	95(3.62)	71(3.23)	24(5.70)	
No	2527(96.38)	2130(96.77)	397(94.30)	
Lung disease				<0.001
Yes	286(10.91)	217(9.86)	69(16.39)	
No	2336(89.09)	1984(90.14)	352(83.61)	
Arthritis				0.14

Yes	875(33.37)	721(32.76)	154(36.58)	
No	1747(66.63)	1480(67.24)	267(63.42)	
Dyslipidemia				0.18
Yes	176(6.71)	141(6.41)	35(8.31)	
No	2446(93.29)	2060(93.59)	386(91.69)	
Liver disease				0.05
Yes	97(3.70)	89(4.04)	8(1.90)	
No	2525(96.30)	2112(95.96)	413(98.10)	
Kidney disease				0.68
Yes	184(7.02)	152(6.91)	32(7.60)	
No	2438(92.98)	2049(93.09)	389(92.40)	
Stomach disease				0.90
Yes	750(28.60)	628(28.53)	122(28.98)	
No	1872(71.40)	1573(71.47)	299(71.02)	
Continuous variable, Median(IQR)				
Age, years	56.00(50.00-61.00)	56.00(50.00-61.00)	58.00(52.00-63.00)	<0.001
Sleep duration, hours	7.00(5.00-8.00)	7.00(5.00-8.00)	6.00(5.00-8.00)	0.07
CESD_10	7.00(3.00-12.00)	7.00(3.00-12.00)	9.00(5.00-15.00)	<0.001
SBP, mmHg	116.00(108.00-125.00)	115.33(107.33-123.67)	120.33(112.67-129.00)	<0.001
DBP, mmHg	69.67(63.67-75.67)	69.33(63.33-75.00)	71.67(65.67-78.00)	<0.001
Pulse, bpm	71.00(65.00-77.00)	71.00(65.00-77.00)	71.33(64.67-77.67)	0.54
Total cognition	12.00(8.50-14.00)	12.00(8.50-14.50)	11.00(6.50-13.50)	<0.001
CumBMI, kg/m²	90.42(82.59-99.87)	90.12(82.09-99.34)	93.03(84.55-102.12)	<0.001
CumWeight, kg	226.00(201.65-253.80)	225.00(200.80-252.80)	230.40(205.20-258.00)	0.01
CumHbA1c, %	21.80(20.80-22.80)	21.80(20.80-22.80)	22.00(21.20-23.00)	<0.001
CumCRP, mg/L	4.84(2.82-8.78)	4.72(2.78-8.60)	5.66(3.14-9.88)	<0.001
CumFPG, mg/dL	390.05(365.21-422.11)	388.26(364.14-420.69)	400.51(370.99-428.96)	<0.001
CumHDL_C, mg/dL	203.25(174.64-236.44)	203.99(176.18-236.44)	197.81(169.99-234.14)	0.09
CumLDL_C, mg/dL	426.56(358.09-497.65)	426.58(357.82-494.63)	426.52(360.83-506.94)	0.44
CumTC, mg/dL	734.89(656.26-823.71)	734.10(658.36-820.62)	745.69(653.79-844.57)	0.24
CumTG, mg/dL	424.79(318.59-591.16)	419.48(315.05-578.77)	460.19(332.75-658.43)	<0.001
CumUA,mg/dL	17.42(14.66-20.54)	17.32(14.62-20.49)	17.82(15.12-20.79)	0.06
CumWBC, ×10⁹/L	23.20(19.78-27.22)	23.20(19.80-27.22)	23.04(19.40-27.20)	0.35
CumPLT, ×10⁹/L	814.00(664.00-968.00)	812.00(664.00-968.00)	820.00(668.00-968.00)	0.97
CumBUN, mg/dL	60.28(51.50-70.81)	60.11(51.54-70.81)	61.06(51.43-70.92)	0.60
CumSCR, mg/dL	2.99(2.60-3.45)	2.99(2.59-3.44)	2.96(2.62-3.50)	0.46
CumHGB, g/dL	55.00(51.00-59.40)	55.00(51.00-59.40)	54.60(50.60-59.80)	0.47
TCBI2011	1011.98(684.69-1345.57)	1011.58(677.27-1327.45)	1011.98(739.58-1502.30)	<0.001
TCBI2015	1104.08(727.40-1555.93)	1103.72(719.37-1500.03)	1104.08(786.77-1695.53)	0.01
CumTCBI	4405.47(3032.05-6715.30)	4362.83(3002.39-6531.38)	4796.31(3204.86-7564.16)	<0.001

Data are presented as n (%) for categorical variables and median (IQR) for continuous variables.

Comparisons between groups were performed using the χ^2 test for categorical variables and the Kruskal-Wallis test for continuous variables. SRH, self-rated health; ADL, activities of daily living; CESD-10, 10-item Center for Epidemiologic Studies Depression Scale; SBP, systolic blood pressure; DBP, diastolic blood pressure; CVD, cardiovascular disease.

Table 3. Multivariate Cox regression analysis examining TCBI, cumulative TCBI, TCBI trajectory clusters, and the risk of new-onset hypertension

Characteristic	**Model 1**		**Model 2**		**Model 3**	
	HR (95% CI)	***P***	**HR (95% CI)**	***P***	**HR (95% CI)**	***P***
Hypertension by TCBI						
TCBI（Per 1 SD increase)	1.15 (1.10–1.20)	<0.001	1.20 (1.15–1.25)	<0.001	1.16 (1.10–1.22)	<0.001
Quartile TCBI						
Q1	Reference		Reference		Reference	
Q2	1.08 (0.94–1.24)	0.257	1.13 (0.99–1.30)	0.076	1.12 (0.97–1.29)	0.118
Q3	1.33 (1.16–1.53)	<0.001	1.43 (1.25–1.64)	<0.001	1.43 (1.24–1.65)	<0.001
Q4	1.47 (1.29–1.68)	<0.001	1.67 (1.46–1.91)	<0.001	1.61 (1.38–1.89)	<0.001
P for trend		<0.001		<0.001		<0.001
Hypertension by cumTCBI						
CumTCBI（Per 1 SD increase)	1.17 (1.08–1.28)	<0.001	1.24 (1.13–1.35)	<0.001	1.17 (1.05–1.30)	0.003
Quartile CumTCBI						
Q1	Reference		Reference		Reference	
Q2	1.22 (0.92–1.63)	0.167	1.26 (0.94–1.67)	0.118	1.18 (0.88–1.59)	0.278
Q3	1.09 (0.82–1.47)	0.551	1.19 (0.89–1.61)	0.242	1.11 (0.80–1.54)	0.518
Q4	1.70 (1.30–2.22)	<0.001	1.97 (1.50–2.59)	<0.001	1.77 (1.26–2.48)	0.001
P for trend		<0.001		<0.001		0.003
Hypertension by TCBI change						
TCBI change						
Cluster 1 (reference)	Reference		Reference		Reference	
Cluster 2	1.35 (1.11–1.63)	0.003	1.46 (1.20–1.78)	<0.001	1.36 (1.09–1.70)	0.008

Characteristic	Model 1		Model 2		Model 3	
	HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*
Hypertension by TCBI						
P for trend		0.003		<0.001		0.008

Model 1: unadjusted.

Model 2: adjusted for Sex, Age, Residence, Marital , Education,and Region.

Model 3:Sex, Age, Residence, Marital, Education, Region, Work status, Drinking, Smoking, SRH, Social activity, Disability, Fall down, Life satisfaction, ADL, Total cognition, Depression, Body pain, Lung disease, Arthritis, Dyslipidemia, Liver disease, Kidney disease, Stomach disease, Diabetes, CVD, Pulse, Sleep duration, WBC, PLT, BUN, FPG, SCR, HbA1c, HGB, CRP, UA, LDL-C, HDL-C.

Table 4. Threshold effect analysis of the association between baseline TCBI and the risk of new-onset hypertension

Model	Adjusted HR(95% CI)	P-value
TCBI		
Fitting by the standard linear model	1.24(1.16, 1.33)	<0.001
Fitting by the two-piecewise linear model		
Inflection point	1539.29	
TCBI≤1539.29	1.73(1.35, 2.20)	<0.001
TCBI>1539.29	1.11(0.99, 1.23)	0.065
P for Log-likelihood ratio	0.001	

Adjusted as Model 3: Sex, Age, Residence, Marital, Education, Region, Work status, Drinking, Smoking, SRH, Social activity, Disability, Fall down, Life satisfaction, ADL, Total cognition, Depression, Body pain, Lung disease, Arthritis, Dyslipidemia, Liver disease, Kidney disease, Stomach disease, Diabetes, CVD, Pulse, Sleep duration, WBC, PLT, BUN, FPG, SCR, HbA1c, HGB, CRP, UA, LDL-C, HDL-C.

Figure legends

Figure 1. Flowchart of participant selection for the baseline TCBI analysis.

Figure 2. Flowchart of participant selection for cumulative TCBI and trajectory

analyses.

Figure 3. TCBI trajectory patterns identified by K-means clustering (k = 2).

(A) Scatter plot of z-score-standardized TCBI values at Wave 1 (2011) and Wave 3 (2015) with K-means clustering (k = 2). (B) Elbow method for determining the optimal number of clusters, with the inflection point at k = 2. (C) Mean TCBI values over time for the two trajectory groups. (D) Four-year incidence of new-onset hypertension by cluster.

Figure 4. Restricted cubic spline analysis of the association between TCBI and new-onset hypertension.

(A)Baseline TCBI. (B) Cumulative TCBI.

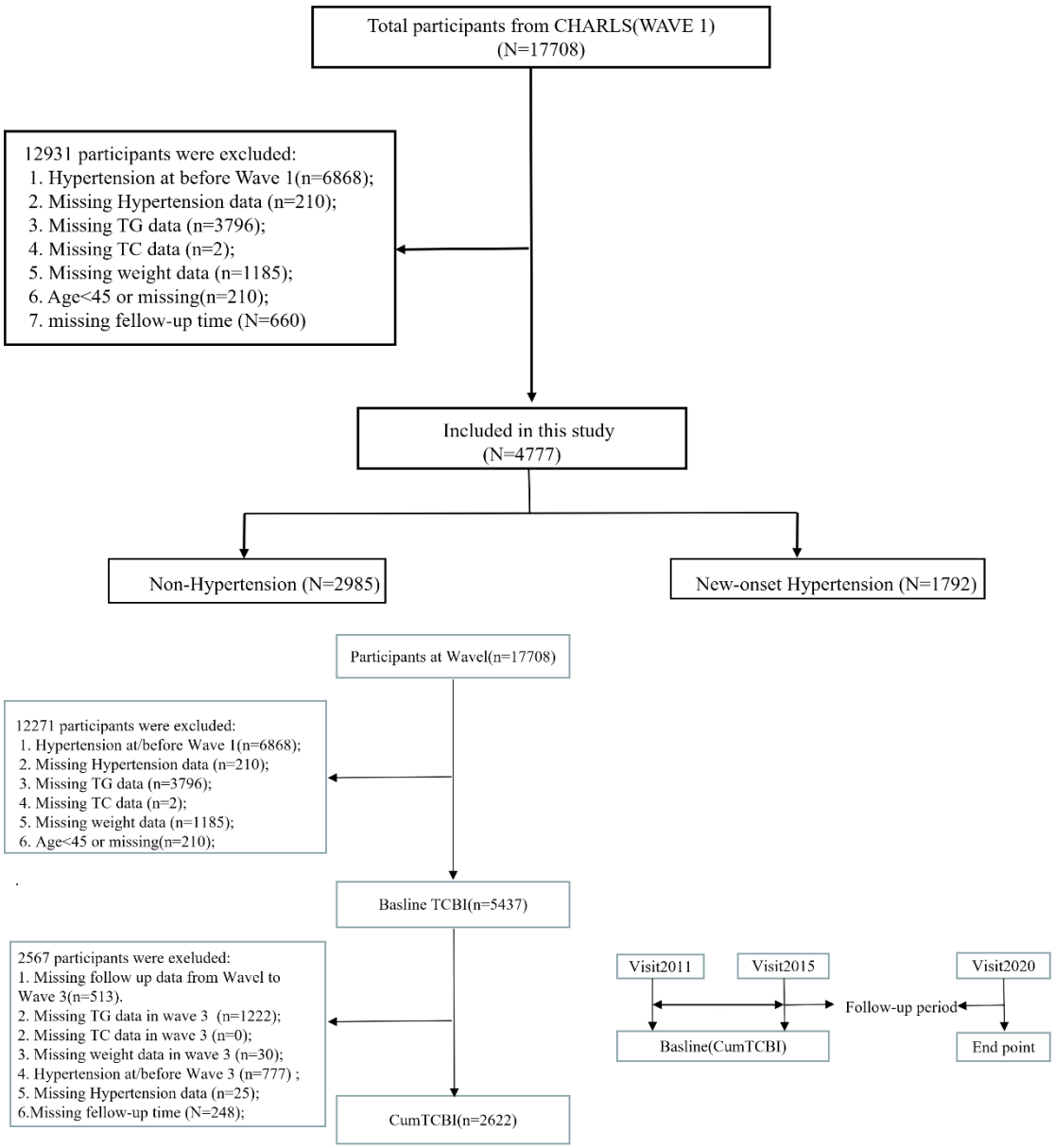
Figure 5. Kaplan-Meier survival curves for new-onset hypertension across different TCBI dimensions.

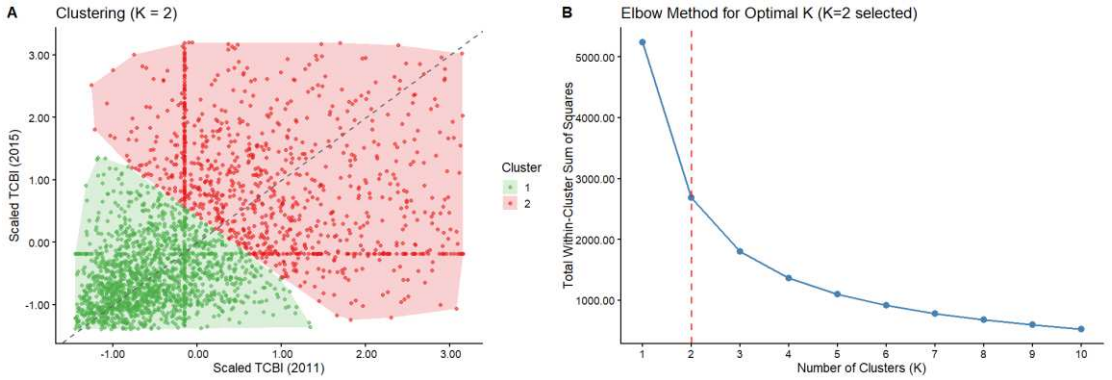
(A) Baseline TCBI quartiles (Q1–Q4). (B) Cumulative TCBI quartiles (Q1–Q4). (C) TCBI trajectory clusters (Cluster 1 vs. Cluster 2). The log-rank test was used to compare differences among groups.

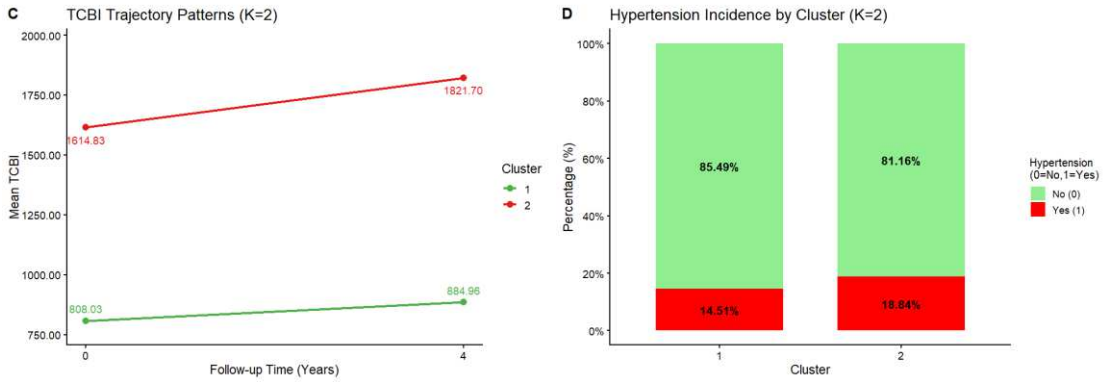
Figure 6. Subgroup analyses of the associations of baseline TCBI, cumulative TCBI, and TCBI trajectory clusters with new-onset hypertension.

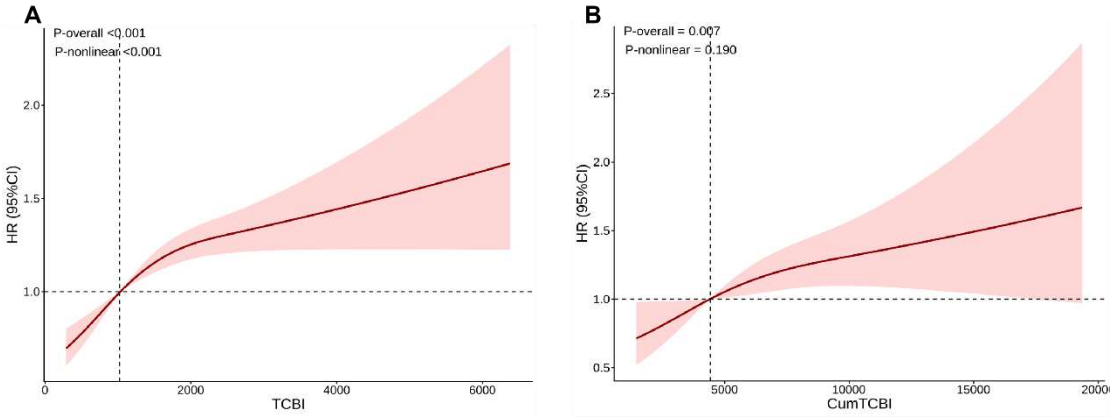
(A) Baseline TCBI (per 1-SD increase). (B) Cumulative TCBI (per 1-SD increase). (C) TCBI trajectory clusters (Cluster 2 vs. Cluster 1). Subgroup analyses were stratified by age, sex, smoking, drinking, region, education, ADL disability, diabetes, cardiovascular disease, work status, arthritis, lung disease, body pain, history of falls, and depression. P for interaction was used to assess the consistency of the associations

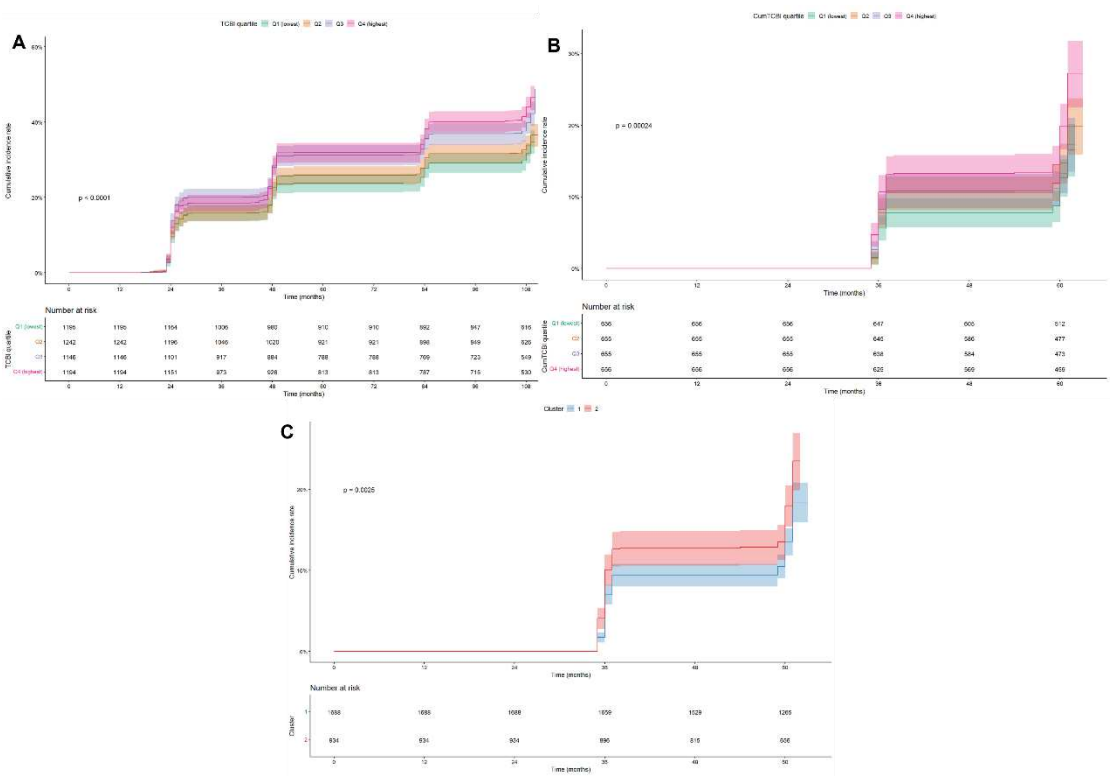
across subgroups.




A
Clustering (K = 2)
Scaled TCBI (2015)
Scaled TCBI (2011)
Cluster
1
2
B
Elbow Method for Optimal K (K=2 selected)
Total Within-Cluster Sum of Squares
Number of Clusters (K)

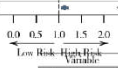

C
TCBI Trajectory Patterns (K=2)
Mean TCBI
Follow-up Time (Years)
1614.83
1821.70
808.03
884.96
Cluster
1
2
D
Hypertension Incidence by Cluster (K=2)
Percentage (%)
85.49%
81.16%
14.51%
18.84%
Cluster
Hypertension
(0=No,1=Yes)
No (0)
Yes (1)


A
P-overall <0.001
P-nonlinear <0.001
HR (95%CI)
TCBI
B
P-overall = 0.007
P-nonlinear = 0.190
HR (95%CI)
CumTCBI


A
TCB quartile Q1 (lowest) Q2 Q3 Q4 (highest)
Cumulative incidence rate
p < 0.0001
Time (months)
Number at risk
TCB quartile
Q1 (lowest) 1195 1195 1194 1186 982 910 910 892 847 515
Q2 1242 1242 1196 1166 1020 921 921 898 849 525
Q3 1146 1146 1101 917 884 788 788 769 723 549
Q4 (highest) 1194 1194 1151 873 828 813 813 787 715 530
0 12 24 36 48 60 72 84 96 108
B
CumTCB quartile Q1 (lowest) Q2 Q3 Q4 (highest)
Cumulative incidence rate
p = 0.00024
Time (months)
Number at risk
CumTCB quartile
Q1 (lowest) 606 606 606 547 500 312
Q2 655 655 655 546 386 277
Q3 655 655 655 538 384 273
Q4 (highest) 606 606 606 526 369 259
0 12 24 36 48 60
C
Cluster 1 2
p = 0.0025
Cumulative incidence rate
Time (months)
Number at risk
Cluster
1 1688 1688 1688 1659 1029 1265
2 934 934 934 896 815 606
0 12 24 36 48 60

A

Variable	N	HR (95%CI)	P-value	P for interaction
Overall	4777(100.00%)	1.160 (1.100, 1.220)	<0.001	
Age				0.157
< 60	3064(64.14%)	1.150 (1.080, 1.230)	<0.001	
≥ 60	1713(35.86%)	1.140 (1.050, 1.230)	0.002	
Sex				0.839
Female	2570(53.80%)	1.170 (1.110, 1.240)	<0.001	
Male	2207(46.20%)	1.150 (1.080, 1.220)	<0.001	
Smoking				0.486
Never	2950(61.75%)	1.150 (1.080, 1.220)	<0.001	
Former	1480(30.98%)	1.160 (1.050, 1.270)	0.003	
Current	347(7.26%)	1.000 (0.840, 1.180)	0.957	
Drinking				0.15
Never	2952(61.80%)	1.110 (1.040, 1.180)	0.003	
Former	1505(31.51%)	1.230 (1.130, 1.330)	<0.001	
Current	320(6.70%)	1.120 (0.910, 1.380)	0.289	
Region				0.387
East	2027(42.43%)	1.120 (1.040, 1.210)	0.003	
Central	1591(33.31%)	1.170 (1.070, 1.280)	<0.001	
West	857(17.94%)	1.120 (1.000, 1.250)	0.055	
Northeast	302(6.32%)	1.370 (1.090, 1.720)	0.008	
Education				0.499
Illiterate	2311(48.38%)	1.090 (1.010, 1.180)	0.019	
Primary_school	1043(21.83%)	1.170 (1.060, 1.300)	0.002	
Middle_school	939(19.66%)	1.250 (1.130, 1.390)	<0.001	
≥ High_school	484(10.13%)	1.290 (1.080, 1.530)	0.005	
ADL				0.846
Yes	665(13.92%)	1.130 (0.990, 1.290)	0.071	
No	4112(86.08%)	1.160 (1.100, 1.220)	<0.001	
Diabetes				0.253
Yes	182(3.81%)	1.130 (0.910, 1.420)	0.267	
No	4595(96.19%)	1.160 (1.100, 1.220)	<0.001	
CVD				0.332
Yes	468(9.80%)	1.070 (0.920, 1.250)	0.368	
No	4309(90.20%)	1.170 (1.110, 1.230)	<0.001	
Work				0.351
Yes	3498(73.23%)	1.150 (1.080, 1.210)	<0.001	
No	1279(26.77%)	1.190 (1.090, 1.300)	<0.001	
Arthritis				0.226
Yes	1630(34.12%)	1.090 (1.000, 1.180)	0.048	
No	3147(65.88%)	1.190 (1.120, 1.270)	<0.001	
Lung_disease				0.099
Yes	562(11.76%)	1.100 (0.970, 1.250)	0.148	
No	4215(88.24%)	1.160 (1.100, 1.220)	<0.001	
Body_pain				0.778
Yes	1634(34.21%)	1.130 (1.040, 1.230)	0.003	
No	3143(65.79%)	1.160 (1.090, 1.230)	<0.001	
Fall_down				0.664
Yes	761(15.93%)	1.160 (1.020, 1.320)	0.025	
No	4016(84.07%)	1.150 (1.090, 1.210)	<0.001	
Depression				0.366
Yes	1755(36.74%)	1.190 (1.090, 1.290)	<0.001	
No	3022(63.26%)	1.120 (1.060, 1.190)	<0.001	



B

Variable	N	HR (95%CI)	P-value	P for interaction
Overall	2622(100.00%)	1.170 (1.050, 1.300)	0.003	
Age				0.796
< 60	1734 (66.13%)	1.190 (1.040, 1.360)	0.009	
≥ 60	888 (33.87%)	1.170 (0.970, 1.420)	0.103	
Sex				0.305
Female	1465 (55.87%)	1.120 (0.970, 1.280)	0.129	
Male	1157 (44.13%)	1.270 (1.080, 1.500)	0.004	
Smoking				0.904
Never	1670 (63.69%)	1.150 (1.010, 1.310)	0.031	
Former	781 (29.79%)	1.170 (0.950, 1.440)	0.143	
Current	171 (6.52%)	24.570 (2.200, 273.770)	0.009	
Drinking				0.662
Never	1663 (63.42%)	1.140 (1.010, 1.300)	0.04	
Former	800 (30.51%)	1.130 (0.920, 1.380)	0.244	
Current	159 (6.06%)	2.890 (1.120, 7.420)	0.028	
Region				0.684
East	1126 (42.94%)	1.200 (1.020, 1.410)	0.027	
Central	858 (32.72%)	1.240 (1.040, 1.490)	0.02	
West	476 (18.15%)	1.160 (0.850, 1.580)	0.352	
Northeast	162 (6.18%)	0.840 (0.600, 1.180)	0.313	
Education				0.169
Illiterate	1262 (48.13%)	1.150 (0.980, 1.340)	0.08	
Primary_school	571 (21.78%)	1.070 (0.850, 1.350)	0.57	
Middle_school	526 (20.06%)	1.290 (1.000, 1.660)	0.054	
≥ High_school	263 (10.03%)	1.510 (0.870, 2.600)	0.139	
ADL				0.757
Yes	351 (13.39%)	1.160 (0.880, 1.530)	0.282	
No	2271 (86.61%)	1.170 (1.040, 1.310)	0.008	
Diabetes				0.068
Yes	95 (3.62%)	0	0	
No	2527 (96.38%)	1.190 (1.070, 1.330)	0.002	
CVD				0.573
Yes	256 (9.76%)	1.410 (0.960, 2.070)	0.076	
No	2366 (90.24%)	1.140 (1.020, 1.280)	0.022	
Work				0.83
Yes	2003 (76.39%)	1.160 (1.030, 1.320)	0.018	
No	619 (23.61%)	1.210 (0.970, 1.510)	0.099	
Arthritis				0.068
Yes	875 (33.37%)	1.010 (0.830, 1.230)	0.901	
No	1747 (66.63%)	1.210 (1.070, 1.380)	0.003	
Lung_disease				0.779
Yes	286 (10.91%)	1.330 (1.010, 1.750)	0.046	
No	2336 (89.09%)	1.140 (1.020, 1.280)	0.024	
Body_pain				0.918
Yes	897 (34.21%)	1.130 (0.950, 1.350)	0.16	
No	1725 (65.79%)	1.160 (1.020, 1.330)	0.029	
Fall_down				0.206
Yes	415 (15.83%)	0.800 (0.600, 1.080)	0.145	
No	2207 (84.17%)	1.260 (1.120, 1.400)	<0.001	
Depression				0.526
Yes	983 (37.49%)	1.090 (0.920, 1.300)	0.31	
No	1639 (62.51%)	1.180 (1.030, 1.350)	0.018	



C

Variable	N	HR (95%CI)	P-value	P for interaction
Overall	2622(100.00%)	1.360 (1.090, 1.700)	0.008	
Age				0.217
< 60	1734 (66.13%)	1.520 (1.140, 2.010)	0.004	
≥ 60	888 (33.87%)	1.080 (0.730, 1.580)	0.706	
Sex				0.18
Female	1465 (55.87%)	1.220 (0.910, 1.640)	0.177	
Male	1157 (44.13%)	1.710 (1.200, 2.430)	0.003	
Smoking				0.264
Never	1670 (63.69%)	1.210 (0.920, 1.590)	0.181	
Former	781 (29.79%)	1.940 (1.250, 3.030)	0.003	
Current	171 (6.52%)	0.990 (0.120, 8.200)	0.995	
Drinking				0.498
Never	1663 (63.42%)	1.270 (0.960, 1.690)	0.096	
Former	800 (30.51%)	1.520 (1.000, 2.310)	0.05	
Current	159 (6.06%)	2.560 (0.510, 12.970)	0.256	
Region				0.115
East	1126 (42.94%)	1.130 (0.790, 1.620)	0.501	
Central	858 (32.72%)	1.460 (0.970, 2.190)	0.067	
West	476 (18.15%)	1.770 (1.010, 3.120)	0.047	
Northeast	162 (6.18%)	0.290 (0.130, 0.620)	0.002	
Education				0.204
Illiterate	1262 (48.13%)	1.310 (0.960, 1.790)	0.09	
Primary_school	571 (21.78%)	1.210 (0.730, 2.020)	0.466	
Middle_school	526 (20.06%)	2.510 (1.390, 4.520)	0.002	
≥ High_school	263 (10.03%)	1.080 (0.350, 3.300)	0.891	
ADL				0.382
Yes	351 (13.39%)	1.090 (0.610, 1.960)	0.761	
No	2271 (86.61%)	1.470 (1.150, 1.890)	0.002	
Diabetes				0.552
Yes	95 (3.62%)	21.210 (8.150, 55.180)	0	
No	2527 (96.38%)	1.340 (1.070, 1.690)	0.012	
CVD				0.747
Yes	256 (9.76%)	1.230 (0.570, 2.650)	0.592	
No	2366 (90.24%)	1.370 (1.080, 1.750)	0.009	
Work				0.588
Yes	2003 (76.39%)	1.370 (1.060, 1.770)	0.015	
No	619 (23.61%)	1.390 (0.860, 2.240)	0.183	
Arthritis				0.037
Yes	875 (33.37%)	1.020 (0.700, 1.500)	0.901	
No	1747 (66.63%)	1.610 (1.210, 2.150)	0.001	
Lung_disease				0.961
Yes	286 (10.91%)	1.270 (0.660, 2.440)	0.478	
No	2336 (89.09%)	1.290 (1.010, 1.640)	0.039	
Body_pain				0.809
Yes	897 (34.21%)	1.280 (0.890, 1.850)	0.178	
No	1725 (65.79%)	1.370 (1.030, 1.840)	0.032	
Fall_down				0.932
Yes	415 (15.83%)	1.050 (0.600, 1.840)	0.851	
No	2207 (84.17%)	1.460 (1.130, 1.870)	0.003	
Depression				0.716
Yes	983 (37.49%)	1.340 (0.950, 1.880)	0.091	
No	1639 (62.51%)	1.240 (0.920, 1.680)	0.161	



Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile.pdf