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Alcohol Intake Amplifies the Association Between Triglyceride–Cholesterol–Body Weight Index (TCBI) and Incident Hypertension: A Nationwide Cohort Study

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Article

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Abstract

Background Hypertension remains a major public health challenge, yet early identification of individuals at high risk is still inadequate. The triglyceride–cholesterol–body weight index (TCBI) is a simple metabolic and nutritional marker, but its role in incident hypertension is not well understood. It also remains unclear whether alcohol intake modifies this association. We therefore examined the relationship between TCBI and incident hypertension and explored the modifying effect of alcohol intake in a nationwide Chinese cohort.

Methods We analyzed data from the China Health and Retirement Longitudinal Study (CHARLS). The study included 4,887 adults aged 45 years or older who were free of hypertension at baseline. Alcohol intake was defined according to self-reported intake of any alcoholic beverage in the previous year. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for incident hypertension. We also added an interaction term for TCBI and alcohol intake. In addition, restricted cubic spline (RCS) models were used to examine dose–response patterns, receiver operating characteristic (ROC) analyses were conducted to assess predictive performance across drinking-status groups, and subgroup and sensitivity analyses were performed to evaluate the robustness of the findings.

Results During a median follow-up of 9 years, 1,743 participants developed hypertension, yielding an incidence density of 51.93 per 1,000 person-years. In the fully adjusted model, high TCBI was associated with a higher risk of incident hypertension (HR 1.35, 95% CI 1.23–1.49), whereas alcohol intake alone was not independently associated with risk (HR 1.10, 95% CI 0.99–1.23). Joint analyses showed that participants with both high TCBI and drinking had the greatest risk of incident hypertension (HR 1.54, 95% CI 1.33–1.78) compared with low-TCBI non-drinkers. The interaction between TCBI and alcohol intake was significant (P for interaction = 0.006). RCS analyses further showed a linear dose–response association, with a steeper increase in risk among drinkers. Adding TCBI to the base prediction model led to a modest improvement in model discrimination in the overall cohort when compared with the base model (AUC, 0.641 vs. 0.627; P < 0.001). A similar pattern was seen among drinkers, where the AUC increased from 0.631 to 0.649 (P = 0.026). Among non-drinkers, the improvement was relatively small and did not reach statistical significance (AUC, 0.645 vs. 0.639; P = 0.056). Sensitivity analyses yielded consistent results.

Conclusion Higher TCBI was independently associated with incident hypertension, and this association was stronger among drinkers. Individuals with both high TCBI and alcohol intake had the highest risk. These findings suggest that incorporating drinking status may improve risk stratification and support more targeted prevention of hypertension.

Introduction

As global population aging accelerates, hypertension has become a major public health challenge worldwide [1–3]. In China, it remains a leading risk factor for cardiovascular and cerebrovascular

diseases, with persistently high prevalence and a substantial contribution to the national disease burden [4, 5]. Although prevention efforts have partly slowed disease progression, control rates remain low, and many high-risk individuals in the preclinical stage are still missed during routine screening [6–8]. Because hypertension often develops gradually [9], a simple risk metric that captures early metabolic changes and can be applied in primary care is urgently needed. Such a metric may help identify high-risk individuals earlier and support more targeted preventive interventions.

The triglyceride–cholesterol–body weight index (TCBI) is an emerging nutritional indicator [10]. Unlike single lipid parameters or body mass index (BMI), TCBI integrates lipid metabolic dysregulation with body-weight burden and may therefore better reflect overall metabolic imbalance. Recent studies have linked TCBI to cardiometabolic and cardiovascular outcomes, including adverse outcomes after acute ischemic stroke, stroke burden among patients with hypertension, and long-term mortality after transcatheter aortic valve replacement [11–14], but evidence on its association with incident hypertension remains limited. Alcohol intake is also a recognized risk factor for multiple cardiovascular diseases [15, 16] and has complex interactions with lipid metabolism [17], which may jointly contribute to vascular injury [18]. Whether there is potential interaction between TCBI and alcohol intake in relation to hypertension risk therefore remains unclear.

To examine the association between TCBI and incident hypertension and to assess the potential modifying role of alcohol intake, we conducted a cohort analysis with up to 9 years of follow-up. These findings may help improve the early identification and risk stratification of individuals at high risk for hypertension.

Materials and methods

Study cohort and data source

Data were drawn from the China Health and Retirement Longitudinal Study (CHARLS), a nationwide program led by Peking University, which is designed to track health and aging patterns among Chinese adults aged 45 years and above. A multistage, stratified probability sampling strategy was adopted to recruit participants from 150 counties and 450 villages across 28 provinces. The present analysis covers five waves collected between 2011 and 2020. Data were collected through face-to-face interviews and physical examinations, and CHARLS gathered detailed information on demographics, lifestyle behaviors, medical history, and clinical biomarkers [19], in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Biomedical Ethics Review Committee of Peking University (IRB No. IRB00001052-11015), and written informed consent was obtained from all participants.

In the 2011 baseline wave, a total of 17,708 participants were initially enrolled. To identify eligible participants for the current analysis, the following exclusion criteria were applied: (1) missing demographic data (n = 140); (2) absence of lipid profile data or body weight measurements needed for calculating TCBI (n = 7,760); (3) loss to follow-up during 2013–2020 (n = 279); (4) age below 45 years (n = 249); (5) hypertension at baseline (n = 3,890); and (6) TCBI outliers or non-fasting blood samples (n =

503). After this screening process was carried out, the final analytic sample comprised 4,887 participants, as shown in Fig. 1.

Assessment of covariates

We collected baseline data on age, sex, smoking status, education, residence, marital status, and comorbidities. Education was stratified into four levels: illiterate or below primary school, primary school, middle school, and high school or above. Marital status was classified as married or other. Residence was divided into urban and rural. Comorbidities included diabetes, kidney disease, lung disease, digestive disease, heart disease, stroke, psychiatric conditions, and arthritis.

Calculation of the TCBI

Venous blood samples were collected, centrifuged, and then transported to the Chinese Center for Disease Control and Prevention, where they were stored at − 80°C, and biomarker assays were subsequently performed by the Clinical Laboratory Center of Capital Medical University. Using standardized enzymatic colorimetric methods, lipid measurements were obtained, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Based on these measurements, TCBI was calculated as follows: $\mathrm{TCBI} = \frac{\mathrm{TG(mg/dL)} \times \mathrm{TC(mg/dL)} \times \mathrm{Body\ Weight(kg)}}{1000}$ [10]. Given the skewed distribution, the TCBI values were natural log-transformed prior to analysis [20].

Ascertainment of incident hypertension

During the follow-up period, participants meeting any of the following criteria were identified as incident hypertension cases: (1) mean systolic blood pressure ≥ 140 mmHg or mean diastolic blood pressure ≥ 90 mmHg; (2) self-reported physician diagnosis of hypertension; or (3) current use of antihypertensive medication [21].

Identification of drinkers

Alcohol intake was assessed at baseline by self-report. Participants were asked the specific question: “Did you drink any alcoholic beverages, such as beer, wine, or liquor in the past year?” Those answering “Yes” were classified as drinkers and those answering “No” as non-drinkers.

Statistical analysis

Continuous variables were assessed for normality using the Shapiro–Wilk test, together with skewness, kurtosis, and visual inspection of histograms and Q–Q plots. Normally distributed variables were presented as the mean ± standard deviation (SD), whereas skewed variables were summarized as the

median with interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. The maximum missingness was 0.78% for any variable (Table S1) and missing data were imputed using multiple imputation [22]. Between-group differences were then examined using Student's t-test, Mann-Whitney U test, or Pearson's chi-square test, depending on the variable type and distribution. All analyses were carried out in the R software (version 4.3.0), and a two-sided P-value < 0.05 was considered statistically significant.

To visualize the cumulative incidence of hypertension across metabolic and behavioral profiles, Kaplan–Meier survival curves were plotted. First, TCBI was classified into low and high levels at the baseline median, and alcohol intake was coded as drinkers and non-drinkers. Participants were stratified into four mutually exclusive subgroups to evaluate the combined effect: (1) Low TCBI and Non-drinkers (reference); (2) High TCBI and Non-drinkers; (3) Low TCBI and Drinkers; and (4) High TCBI and Drinkers. Group differences were assessed using the log-rank test. Incidence density was calculated as the number of new cases divided by total person-years and expressed per 1,000 person-years.

To estimate the risk of incident hypertension in relation to TCBI, alcohol intake, as well as different TCBI–drinking combinations, Cox proportional hazards regression models were used. Results were expressed as a Hazard Ratio (HR) together with a 95% Confidence Interval (CI). In addition, the possible interaction between TCBI and alcohol intake was evaluated by incorporating a multiplicative interaction term into the model. Three stepwise models were constructed: Model 1 included no adjustments; Model 2 adjusted for age, sex, BMI, smoking status, education level, area of residence, and marital status; and Model 3, as the fully adjusted model, further adjusted for all comorbidities (diabetes, kidney disease, lung disease, digestive disease, heart disease, stroke, psychiatric disease, and arthritis). The proportional hazards (PH) assumption for fully adjusted Cox models was assessed using Schoenfeld residuals. Variance Inflation Factors (VIFs) were calculated to assess potential multicollinearity, and all VIFs were below 5 (Table S2).

To visualize the dose–response relationship between TCBI and hypertension risk across drinking strata, 4 knots (5%, 35%, 65% and 95%) restricted cubic spline (RCS) models were fitted on the fully adjusted model, and the Wald test was used to assess non-linearity. Prediction performance was evaluated using 9-year time-dependent receiver operating characteristic (ROC) curves. The fully adjusted model was taken as the reference model, and TCBI was then incorporated into this model to examine whether it could provide additional discriminatory value. ROC analyses were first carried out in the overall population and were then repeated separately in drinkers and non-drinkers. Model discrimination was reported as the AUC together with its 95% CI. Within each population, differences in AUC between the two models were assessed using DeLong's test. Finally, subgroup analyses were conducted based on the fully adjusted model to explore potential heterogeneity, with stratification by sex, age group (45–59 vs. ≥60 years), smoking status, diabetes status, and BMI category (≤ 24 vs. >24 kg/m²).

Sensitivity analysis

To assess the robustness of the primary findings, we performed three sensitivity analyses based on the fully adjusted model. First, we used logistic regression to examine whether the association remained stable under an alternative modeling approach. Second, we used a complete-case dataset to evaluate whether missing data materially influenced the results. Third, we excluded participants with abnormal TCBI values, defined as values beyond the mean ± 3 standard deviations, to determine whether extreme values materially influenced the results.

Results

Baseline Characteristics Stratified by Hypertension Status

Table 1 summarizes the baseline characteristics of the study cohort. Among the 4,887 participants, 52.9% were women, and 1,743 (35.7%) developed hypertension during follow-up. Compared with those who remained normotensive, participants who developed hypertension were older at baseline (59.5 ± 9.1 vs. 56.9 ± 8.4 years, $P < 0.001$) and had lower levels of education ($P < 0.001$). Residential distribution also differed slightly between the two groups ($P = 0.047$), with a somewhat higher proportion of urban residents in the hypertension group than in the non-hypertensive group (67.9% vs. 65.0%). Baseline TCBI was marginally higher in participants who later developed hypertension (7.0 ± 0.6 vs. 6.9 ± 0.6, $P < 0.001$). Participants in the hypertension group also showed a poorer metabolic and inflammatory status. They had higher levels of BMI, LDL-C, TC, TG, fasting blood glucose (FBG), uric acid, hemoglobin A1c (HbA1c), and C-reactive protein (CRP) (all $P < 0.05$). In addition, marital status ($P = 0.038$) and histories of digestive disease ($P = 0.031$), stroke ($P = 0.045$), and diabetes ($P = 0.013$) differed significantly between groups. By contrast, sex, smoking status, drinking status, HDL-C, and creatinine (Cr) were comparable between the two groups. No significant differences were observed for other comorbidities, including kidney disease, lung disease, heart disease, psychiatric disease, and arthritis (all $P > 0.05$).

Table 1

Baseline characteristics of participants according to incident hypertension status. HTN, hypertension; FBG, fasting blood glucose; HbA1c, hemoglobin A1c; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides; Cr, creatinine; CRP, C-reactive protein; BMI, body mass index; TCBI, triglyceride-cholesterol-body weight index.

Characteristics	Total (N = 4887)	Non-HTN (N = 3144)	HTN (N = 1743)	P-value
Age, years	57.8 ± 8.7	56.9 ± 8.4	59.5 ± 9.1	< 0.001
Sex, n(%)				0.149
Female	2584 (52.9%)	1687 (53.7%)	897 (51.5%)	
Male	2303 (47.1%)	1457 (46.3%)	846 (48.5%)	
Education, n(%)				< 0.001
No formal education	2383 (48.8%)	1482 (47.1%)	901 (51.7%)	
Primary school	1040 (21.3%)	649 (20.6%)	391 (22.4%)	
Middle school	963 (19.7%)	653 (20.8%)	310 (17.8%)	
High school or above	501 (10.3%)	360 (11.5%)	141 (8.1%)	
Marital status, n(%)				0.038
Other	688 (14.1%)	418 (13.3%)	270 (15.5%)	
Married	4199 (85.9%)	2726 (86.7%)	1473 (84.5%)	
Residence, n(%)				0.047
Urban	3227 (66.0%)	2044 (65.0%)	1183 (67.9%)	
Rural	1660 (34.0%)	1100 (35.0%)	560 (32.1%)	
BMI, kg/m²	22.49 (20.41–24.78)	22.27 (20.25–24.53)	22.94 (20.73–25.40)	< 0.001
Smoking, n(%)				0.470
No	3353 (68.8%)	2170 (69.2%)	1183 (68.1%)	
Yes	1522 (31.2%)	968 (30.8%)	554 (31.9%)	
Drinking, n(%)				0.140
No	3213 (65.7%)	2091 (66.5%)	1122 (64.4%)	
Yes	1674 (34.3%)	1053 (33.5%)	621 (35.6%)	
HDL-C, mg/dL	52.9 ± 15.2	53.0 ± 14.9	52.8 ± 15.7	0.638
LDL-C, mg/dL	116.5 ± 32.9	115.7 ± 32.1	117.9 ± 34.3	0.026

Characteristics	Total (N = 4887)	Non-HTN (N = 3144)	HTN (N = 1743)	P-value
TC, mg/dL	190.7 ± 36.6	189.4 ± 35.9	193.1 ± 37.7	< 0.001
TG, mg/dL	113.3 ± 63.6	110.9 ± 62.5	117.6 ± 65.4	< 0.001
FBG, mg/dL	100.62 (93.42-109.44)	99.72 (92.70-108.54)	102.06 (94.50-111.24)	< 0.001
HbA1c, %	5.10 (4.90–5.40)	5.10 (4.90–5.40)	5.20 (4.90–5.40)	< 0.001
CRP, mg/L	0.88 (0.50–1.84)	0.83 (0.48–1.73)	0.95 (0.53–2.09)	< 0.001
Uric acid, mg/dl	4.3 ± 1.2	4.2 ± 1.1	4.4 ± 1.2	< 0.001
Cr, mg/dl	0.8 ± 0.2	0.8 ± 0.2	0.8 ± 0.2	0.055
TCBI	7.0 ± 0.6	6.9 ± 0.6	7.0 ± 0.6	< 0.001
Diabetes, n(%)				0.013
No	4658 (96.1%)	3009 (96.6%)	1649 (95.1%)	
Yes	191 (3.9%)	106 (3.4%)	85 (4.9%)	
Heart disease, n(%)				0.163
No	4460 (91.6%)	2878 (92.1%)	1582 (90.9%)	
Yes	407 (8.4%)	248 (7.9%)	159 (9.1%)	
Stroke, n(%)				0.045
No	4812 (98.7%)	3102 (98.9%)	1710 (98.2%)	
Yes	64 (1.3%)	33 (1.1%)	31 (1.8%)	
Kidney disease, n(%)				0.914
No	4588 (94.4%)	2951 (94.3%)	1637 (94.5%)	
Yes	273 (5.6%)	177 (5.7%)	96 (5.5%)	
Lung disease, n(%)				0.266
No	4413 (90.6%)	2850 (91.0%)	1563 (90.0%)	
Yes	456 (9.4%)	282 (9.0%)	174 (10.0%)	
Digestive disease, n(%)				0.031
No	3678 (75.4%)	2333 (74.4%)	1345 (77.3%)	
Yes	1197 (24.6%)	801 (25.6%)	396 (22.7%)	
Psychiatric disease, n(%)				0.132

Characteristics	Total (N = 4887)	Non-HTN (N = 3144)	HTN (N = 1743)	P-value
No	4808 (98.7%)	3085 (98.5%)	1723 (99.1%)	
Yes	62 (1.3%)	46 (1.5%)	16 (0.9%)	
Arthritis, n(%)				0.099
No	3241 (66.4%)	2110 (67.3%)	1131 (64.9%)	
Yes	1639 (33.6%)	1027 (32.7%)	612 (35.1%)	

Cumulative Incidence of Hypertension Stratified by TCBI and Alcohol Intake

During a median follow-up of 9 years, 1,743 participants developed hypertension, with an overall incidence rate of 51.93 per 1,000 person-years. Participants with high TCBI had a higher incidence rate than those with low TCBI (58.42 vs. 45.67 per 1,000 person-years; $P < 0.001$), whereas alcohol intake alone showed no significant difference (54.85 vs. 50.45 per 1,000 person-years; $P = 0.102$). In the joint analysis, high-TCBI drinkers had the highest incidence rate (65.59 per 1,000 person-years) (Table 2), and Kaplan–Meier curves showed the steepest cumulative incidence in this group (Fig. 2).

Table 2
Incidence rates of hypertension according to baseline TCBI level, alcohol intake, and joint exposure groups. TCBI, triglyceride-cholesterol-body weight index.

Variable	Total (n)	Events (n)	Total person-years	Incidence rate per 1,000 person-years	Log-rank P value
TCBI Levels					< 0.001
Low TCBI	2443	780	17078	45.67	
High TCBI	2444	963	16485	58.42	
Alcohol Intake					0.102
Non-drinkers	3213	1122	22241	50.45	
Drinkers	1674	621	11322	54.85	
Joint Groups					< 0.001
Low TCBI and Non-drinkers	1593	514	11168	46.02	
High TCBI and Non-drinkers	1620	608	11073	54.91	
Low TCBI and Drinkers	850	266	5910	45.01	
High TCBI and Drinkers	824	355	5412	65.59	

Joint Effect of TCBI and Alcohol Intake on the Risk of Incident Hypertension

Elevated TCBI was associated with incident hypertension in multivariable models. After full adjustment in Model 3, the high TCBI group showed a 35% higher hazard of hypertension (HR = 1.35, 95% CI 1.23–1.49, P < 0.001). In the joint analysis, participants who had both high TCBI and alcohol intake exhibited the greatest hazard of incident hypertension (Model 3 HR = 1.54, 95% CI 1.33–1.78). High TCBI among non-drinkers remained associated with hypertension hazard (HR = 1.23, 95% CI 1.09–1.38, P < 0.001), but the effect size was lower than in the combined exposure group. While the low TCBI drinkers showed a hazard similar to the low TCBI non-drinker reference group (HR = 0.95, 95% CI 0.81–1.11, P = 0.524). A significant interaction was observed (P for interaction = 0.006), suggesting that the association between TCBI and incident hypertension differed by drinking status (Table 3). The PH assumption tests showed no clear evidence of violation for the main exposure terms, including TCBI, alcohol intake, joint exposure, and the TCBI–alcohol intake interaction term, with all P values > 0.05 (Table S3).

Table 3

Associations of TCBI and Alcohol Intake with the Risk of Incident Hypertension. Model 1 was unadjusted. Model 2 was adjusted for age, sex, BMI, smoking status, education level, residence, and marital status. Model 3 was further adjusted for diabetes, kidney disease, lung disease, digestive disease, heart disease, stroke, psychiatric disease, and arthritis. The P for interaction was calculated from the multiplicative interaction term between TCBI group and alcohol intake in Model 3. TCBI, triglyceride-cholesterol-body weight index; HR, hazard ratio; CI, confidence interval.

Variable	Total (n)	Events (n)	Model 1		Model 2		Model 3	
			HR (95% CI)	P	HR (95% CI)	P	HR (95% CI)	P
TCBI Levels								
Low TCBI	2443	780	1.00 (Reference)	-	1.00 (Reference)	-	1.00 (Reference)	-
High TCBI	2444	963	1.28 (1.17–1.41)	< 0.001	1.37 (1.24–1.50)	< 0.001	1.35 (1.23–1.49)	< 0.001
Alcohol Intake								
Non-drinkers	3213	1122	1.00 (Reference)	-	1.00 (Reference)	-	1.00 (Reference)	-
Drinkers	1674	621	1.09 (0.98–1.20)	0.102	1.10 (0.98–1.23)	0.095	1.10 (0.99–1.23)	0.085
Joint Groups								
Low TCBI and Non-drinkers	1593	514	1.00 (Reference)	-	1.00 (Reference)	-	1.00 (Reference)	-
High TCBI and Non-drinkers	1620	608	1.20 (1.06–1.35)	0.003	1.24 (1.10–1.40)	< 0.001	1.23 (1.09–1.38)	< 0.001
Low TCBI and Drinkers	850	266	0.98 (0.84–1.13)	0.747	0.95 (0.81–1.11)	0.530	0.95 (0.81–1.11)	0.524
High TCBI and Drinkers	824	355	1.43 (1.25–1.64)	< 0.001	1.55 (1.34–1.79)	< 0.001	1.54 (1.33–1.78)	< 0.001
P for interaction	0.006							

Linear Relationship Between TCBI and Hypertension Risk According to Drinking Status

RCS analysis demonstrated no evidence of non-linearity between TCBI and hypertension risk, with no significant non-linearity observed in either subgroup (both P for non-linearity > 0.05; Fig. 3). Overall, higher TCBI was linked to higher risk (P < 0.001), and testing showed no evidence of non-linearity (P for non-linearity = 0.168). Stratified analyses by drinking status revealed distinct patterns. The drinking group in Fig. 3A displayed a steeper linear rise, with risk increasing more markedly as TCBI increased (P < 0.001). The non-drinking group in Fig. 3B also remained linear (P = 0.003) but rose more gradually.

ROC Analysis of TCBI for Predicting Incident Hypertension According to Drinking Status

ROC curve analysis showed that the addition of TCBI increased the AUC from 0.627 (95% CI, 0.610–0.644) to 0.641 (95% CI, 0.624–0.658), and this difference was statistically significant (P < 0.001). Among drinkers, the AUC rose from 0.631 (95% CI, 0.602–0.660) to 0.649 (95% CI, 0.621–0.677), with the difference also reaching statistical significance (P = 0.0259). In non-drinkers, the AUC increased only slightly, from 0.639 (95% CI, 0.618–0.660) to 0.645 (95% CI, 0.624–0.666), without statistical significance (P = 0.056). These findings suggest that TCBI improved discrimination beyond conventional covariates, especially among drinkers (Fig. 4).

Subgroup analyses

Subgroup analyses showed that the elevated risk was broadly consistent across population subgroups. Significant associations were observed in both sexes, both age groups, both smoking strata, participants without diabetes, and those with BMI ≤ 24 kg/m², while all interaction tests were nonsignificant. In contrast, no significant association was found among participants with diabetes or those with BMI > 24 kg/m². The association was stronger in participants aged < 60 years (HR = 1.65) than in those aged ≥ 60 years (HR = 1.29), although the interaction did not reach statistical significance (Fig. 5).

Sensitivity analyses

Sensitivity analyses further supported the robustness of the primary findings. The results remained materially unchanged when logistic regression was used (odds ratio [OR] = 1.79, 95% CI: 1.48–2.16), in the complete-case dataset (HR = 1.55, 95% CI: 1.34–1.79), and after excluding participants with abnormal TCBI values (HR = 1.54, 95% CI: 1.33–1.78). Across all sensitivity analyses, participants with both high TCBI and alcohol intake consistently had the highest risk of incident hypertension compared with the low TCBI and non-drinking group (Table 4).

Table 4

Sensitivity analyses using alternative modeling strategies, a complete-case dataset, and excluding participants with abnormal TCBI values. Logistic regression results are reported as ORs, while complete-case analysis and the analysis excluding abnormal TCBI values are reported as HRs from Cox proportional hazards models. All estimates are presented with 95% CIs and were adjusted for age, sex, BMI, smoking status, education level, residence, marital status, diabetes, kidney disease, lung disease, digestive disease, heart disease, stroke, psychiatric disease, and arthritis (Model 3). OR, odds ratio; HR, hazard ratio; CI, confidence interval; TCBI, triglyceride–cholesterol–body weight index.

Logistic Regression	Total (n)	Events (n)	OR / HR (95% CI)	P
Low TCBI and Non-drinkers	1593	514	1.00 (Reference)	
High TCBI and Non-drinkers	1620	608	1.31 (1.13–1.52)	< 0.001
Low TCBI and Drinkers	851	266	0.94 (0.78–1.14)	0.535
High TCBI and Drinkers	823	355	1.79 (1.48–2.16)	< 0.001
P for interaction				0.004
Complete-case Dataset				
Low TCBI and Non-drinkers	1566	507	1.00 (Reference)	
High TCBI and Non-drinkers	1575	596	1.23 (1.09–1.39)	< 0.001
Low TCBI and Drinkers	829	260	0.95 (0.81–1.11)	0.518
High TCBI and Drinkers	807	351	1.55 (1.34–1.79)	< 0.001
P for interaction				0.006
Excluding Abnormal TCBI				
Low TCBI and Non-drinkers	1590	513	1.00 (Reference)	
High TCBI and Non-drinkers	1620	608	1.23 (1.09–1.38)	< 0.001
Low TCBI and Drinkers	848	264	0.94 (0.81–1.10)	0.472
High TCBI and Drinkers	823	355	1.54 (1.33–1.78)	< 0.001
P for interaction				0.005

Discussion

This study systematically evaluated the joint association of TCBI and alcohol intake with incident hypertension using the nationally representative CHARLS cohort. We found that higher TCBI was independently associated with an increased risk of incident hypertension. More importantly, alcohol intake appeared to strengthen this association. Participants with both high TCBI and alcohol intake had

the highest risk of developing hypertension, and the RCS analysis suggested that this relationship was approximately linear. Sensitivity analyses further supported the robustness of the findings. The joint exposure group consistently remained at the highest risk when we used logistic regression, analyzed the complete-case dataset, or excluded participants with abnormal TCBI values.

Several previous studies have linked lipid profiles and related composite metabolic indicators to incident hypertension. Sesso et al. reported that an unfavorable lipid profile predicted future hypertension in women [23], and Halperin et al. observed a similar pattern in men [24]. Later, other lipid-related composite markers, such as the triglyceride-glucose (TyG) index, were also shown to be positively associated with incident hypertension [25–28]. Mo et al. further demonstrated that the association between the atherogenic index of plasma (AIP) and incident hypertension varied across baseline blood pressure states [29]. TCBI is a novel lipid-related nutritional indicator originally proposed by Doi et al. for prognostic assessment in patients with coronary artery disease [10]. Subsequent studies also linked TCBI to outcomes in patients with acute decompensated heart failure, those receiving mechanical circulatory support, and those with dilated cardiomyopathy [30–32]. Chen et al. studied 5,068 participants and found that each 1-standard-deviation increase in cumulative TCBI was associated with an 18% higher risk of incident cardiometabolic multimorbidity. These findings suggest that TCBI may serve not only as a prognostic marker for different cardiovascular diseases, but also as an indicator of a broader burden of cardiometabolic damage [33]. Consistent with these findings, our study demonstrated a significant association between TCBI and the risk of incident hypertension. Notably, the magnitude of this association differed substantially according to drinking behavior, and the effect was markedly amplified among drinkers. By contrast, alcohol intake alone was not significantly associated with hypertension risk after full adjustment, which differs from some previous reports. This discrepancy may partly reflect the well-established dose-dependent relationship between alcohol intake and both blood pressure and hypertension risk [34–36], whereas our study did not capture detailed drinking patterns. Future studies should therefore examine whether the association between TCBI and incident hypertension differs according to drinking frequency and alcohol intake level.

The subgroup findings were largely consistent with the main analysis. However, we did not observe a significant association between high TCBI and incident hypertension among drinkers with diabetes, although the interaction across subgroups was not significant. This may partly reflect limited statistical power due to the relatively small sample size. In addition, patients with diabetes already experience substantial endothelial dysfunction, increased oxidative stress, and greater arterial stiffness [37–39], which may attenuate the incremental risk information provided by TCBI and alcohol intake. Larger studies are needed to clarify the association between TCBI and hypertension risk in specific high-risk populations.

Although the biological mechanisms underlying the modifying effect of alcohol on the association between TCBI and hypertension remain unclear, several pathways may be involved. Lipotoxicity can impair endothelial function through oxidative stress [40, 41]. Alcohol exposure may further aggravate vascular endothelial injury by reducing nitric oxide bioavailability, suppressing antioxidant systems,

increasing reactive oxygen species generation, and altering vascular tone regulation [42–44]. Taken together, our findings suggest that higher TCBI is associated with an increased risk of incident hypertension and that alcohol intake may further strengthen this association. These results may help refine risk stratification for incident hypertension and inform alcohol management and early intervention for metabolic abnormalities.

Several limitations should be acknowledged. First, alcohol exposure was self-reported, and detailed drinking patterns were unavailable. Recall bias may therefore have influenced both the direction and magnitude of the observed modifying effect of alcohol on the association between TCBI and hypertension risk. Second, although we adjusted for a broad range of covariates, residual confounding cannot be excluded because of the observational design. Third, despite using multiple approaches to define incident hypertension, some cases may still have been missed during follow-up. Fourth, among participants who developed hypertension during follow-up, we could not determine whether some cases were secondary in origin. Finally, because the study population consisted of middle-aged and older Chinese adults, the generalizability of our findings to younger individuals and other ethnic populations remains uncertain.

Conclusion

In this nationwide cohort of middle-aged and older Chinese adults, higher TCBI was independently associated with a greater risk of incident hypertension. This association was stronger among drinkers, and individuals with both high TCBI and alcohol intake had the highest risk. These findings suggest that alcohol intake may amplify the adverse association between TCBI and future hypertension. Assessment based on both TCBI and drinking status may therefore help identify individuals at elevated risk and support more targeted prevention strategies.

Abbreviations

TCBI Triglyceride–cholesterol–body weight index

CHARLS China Health and Retirement Longitudinal Study

BMI Body mass index

HR Hazard ratio

CI Confidence interval

RCS Restricted cubic spline

ROC Receiver operating characteristic

AUC Area under the curve

SD Standard deviation

IQR Interquartile range

TC Total cholesterol

TG Triglycerides

HDL-C High-density lipoprotein cholesterol

LDL-C Low-density lipoprotein cholesterol

FBG Fasting blood glucose

HbA1c Hemoglobin A1c

CRP C-reactive protein

Cr Creatinine

PH Proportional hazards

VIF Variance inflation factor

OR Odds ratio

HTN Hypertension

TyG Triglyceride-glucose index

AIP Atherogenic index of plasma

Declarations

Supplementary Information

Tables S1 - S3.

Declarations

Ethics approval and consent to participate

The CHARLS study was conducted in accordance with the Declaration of Helsinki, and the CHARLS protocol was approved by the Institutional Review Board of Peking University, with approval numbers

IRB00001052-11015 for the household survey and IRB00001052-11014 for blood sample collection; all participants provided written informed consent.

Conflicts of interest

The authors declare no conflicts of interest.

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Author Contribution

Shuya Zhang: Writing – original draft, Investigation, Methodology, Visualization. Jiaqing Dou: Data curation, Formal analysis, Methodology, Writing – original draft. Xiaoshen Yuan: Data curation. Fuzhong Xue: Writing – review & editing, Conceptualization, Funding acquisition, Project administration. All authors read and approved the final manuscript.

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Data Availability

The data used for this analysis were obtained from the publicly available CHARLS database (http://charls.pku.edu.cn/).

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Figures

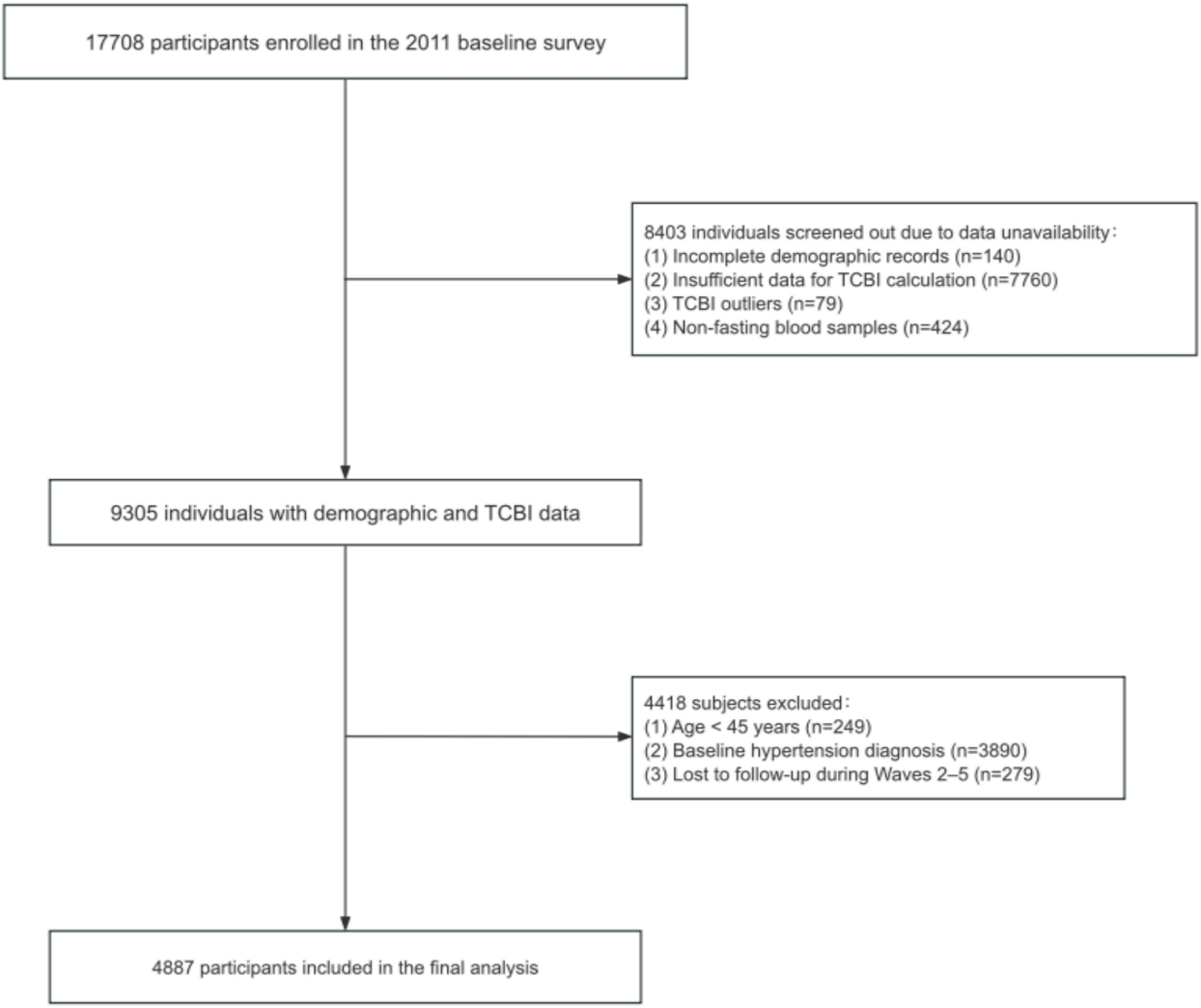

Figure 1

Screening of study participants.

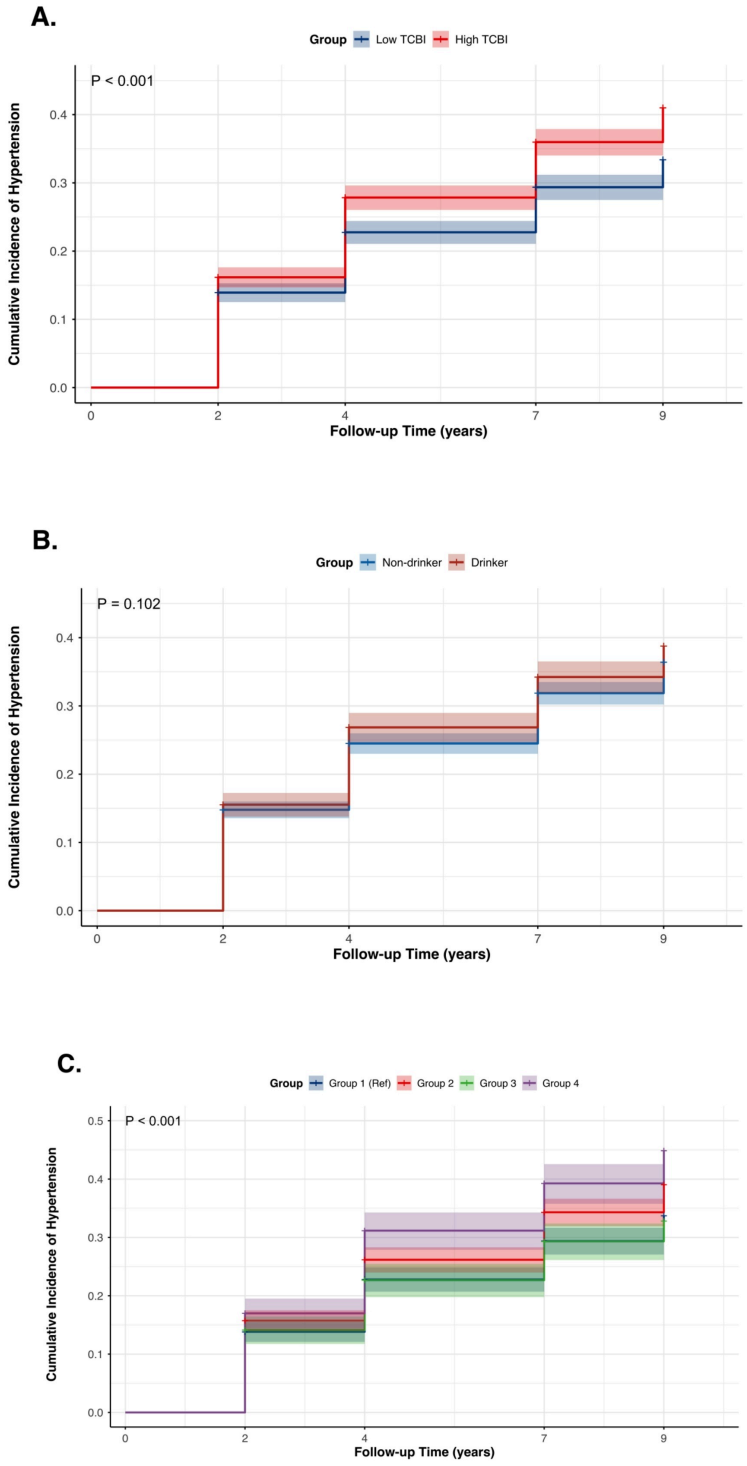

Figure 2

Kaplan–Meier cumulative incidence curves for incident hypertension according to baseline TCBI level, alcohol intake, and joint TCBI–alcohol exposure groups. (A) Participants stratified by low and high TCBI levels. (B) Participants stratified by alcohol intake status. (C) Participants stratified by joint TCBI–alcohol exposure groups: low TCBI and non-drinkers (Group 1), high TCBI and non-drinkers (Group 2), low TCBI

and drinkers (Group 3), and high TCBI and drinkers (Group 4). TCBI, triglyceride–cholesterol–body weight index; Ref, reference.

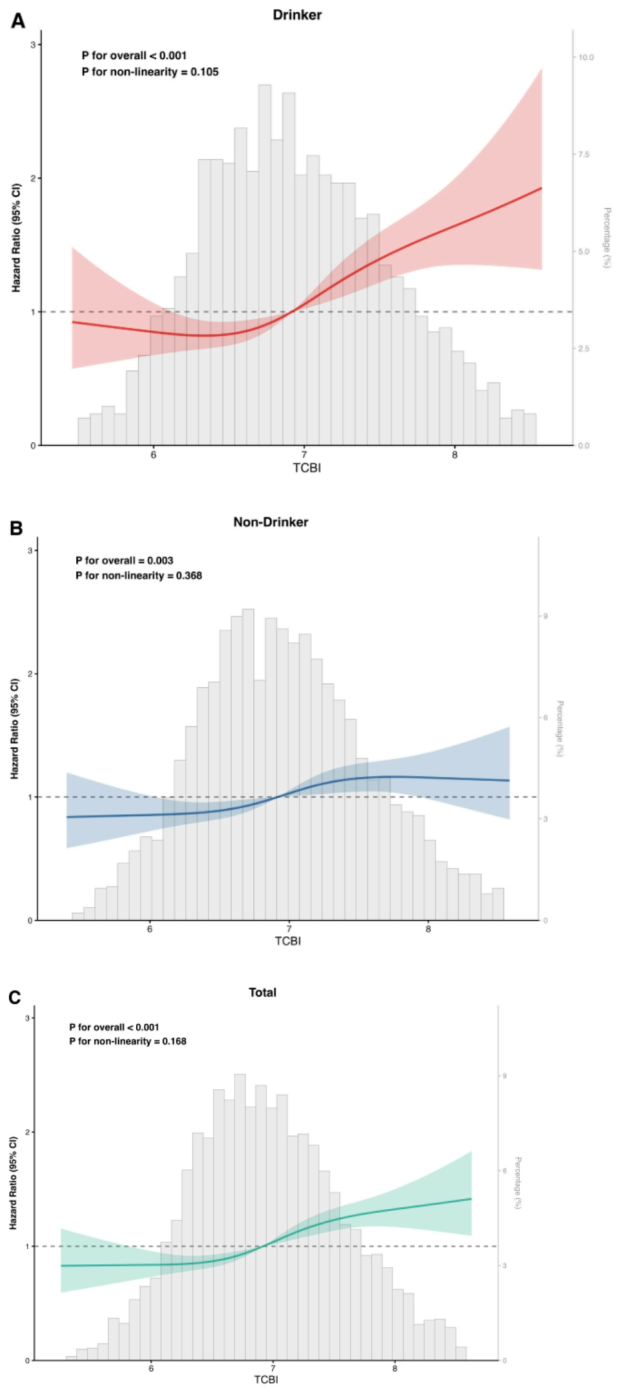

Figure 3

Restricted cubic spline analyses of the association between baseline TCBI and incident hypertension according to alcohol intake status. (A) Drinkers. (B) Non-drinkers. (C) Overall population. Solid lines

represent the estimated HRs for incident hypertension across TCBI levels, and shaded areas indicate the corresponding 95% CIs. Background histograms show the distribution of TCBI in the study population. TCBI, triglyceride–cholesterol–body weight index.

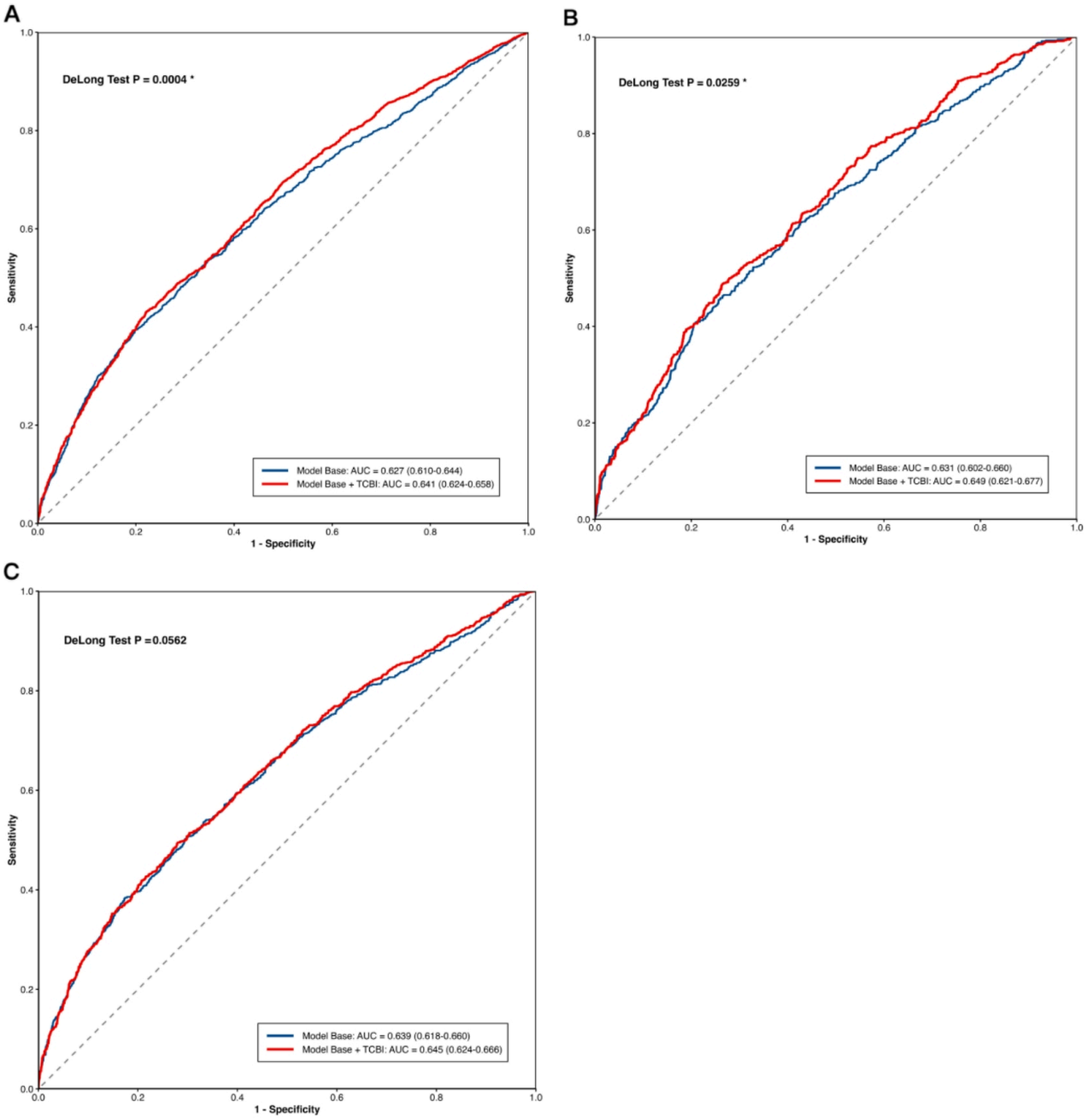

Figure 4

Time-dependent receiver operating characteristic curves for baseline TCBI predicting incident hypertension at different follow-up time points. (A) Overall population. (B) Drinkers. (C) Non-drinkers.The base model included age, sex, smoking status, education level, residence, marital status, BMI, diabetes,

kidney disease, lung disease, digestive disease, heart disease, stroke, psychiatric disease, and arthritis. TCBI, triglyceride–cholesterol–body weight index; ROC, receiver operating characteristic; AUC, area under the receiver operating characteristic curve; CI, confidence interval.

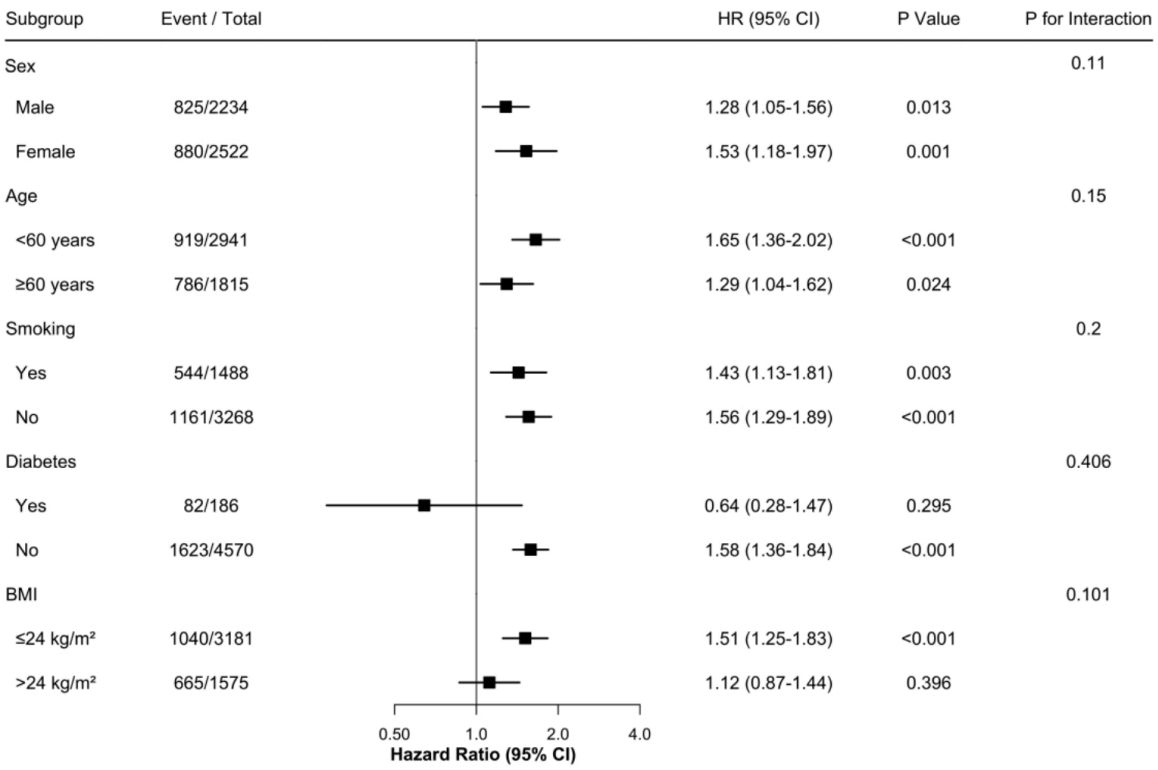

Figure 5

Subgroup analysis of the joint association between TCBI and alcohol intake with incident hypertension. Reference group: Low TCBI and Non-drinkers. All models are fully adjusted except for the stratification variable. TCBI, triglyceride–cholesterol–body weight index.

Supplementary Files

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

- SupplementaryMaterials.docx