Estimated pulse wave velocity, waist-to-height ratio, and risk of cardiometabolic multimorbidity: A secondary dataset analysis of the China Health and Retirement Longitudinal Study (CHARLS)

ZhiYing Fei[1], LingLing Bian[1], ShuLin Lu[1], ChunQiao Wu[1]

ABSTRACT

INTRODUCTION Obesity, smoking, and atherosclerosis increase the risk of developing various cardiometabolic diseases. The estimated pulse wave velocity (ePWV) is a new indicator of arterial stiffness. However, the relationship between ePWV, waist-to-height ratio (WHtR), smoking, and cardiometabolic multimorbidity (CMM) remains unclear.

METHODS The study is a secondary dataset analysis of CHARLS, which included 8414 participants from the China Health and Retirement Longitudinal Study conducted between 2011 and 2018. The ePWV was calculated using the mean blood pressure and age. Cox proportional hazards models were used to explore the relationship between ePWV, WHtR, and CMM in both smoking and non-smoking populations. Additionally, we also employed restricted cubic spline (RCS) analysis and mediation analysis to investigate the relationship between ePWV, WHtR, and CMM. A $p<0.05$ was considered statistically significant.

RESULTS During the 7-year follow-up period, 1545 participants (18.36%) developed new-onset CMM. The RCS model exhibited a U-shaped relationship between WHtR and CMM incidence, with a positive correlation when WHtR exceeded 0.5. Cox regression analysis revealed that ePWV and WHtR were independent predictors of CMM in both smoking and non-smoking populations. Additionally, ePWV significantly mediated the association between WHtR and CMM risk.

CONCLUSIONS Our findings indicate that ePWV and WHtR are associated with increased CMM risk. Early detection of ePWV and WHtR, combined with smoking cessation, may help identify high-risk individuals and provide a basis for future preventive research.

AFFILIATION
1 Department of Nursing, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, People's Republic of China

CORRESPONDENCE TO
ChunQiao Wu. Nursing Department, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, 3 Qingchundong Road, Hangzhou, Zhejiang Province, 310016, People's Republic of China
E-mail: 13747256@qq.com

KEYWORDS
estimated pulse wave velocity, waist-to-height ratio, cardiometabolic multimorbidity

Received: 1 December 2025
Revised: 4 January 2026
Accepted: 11 January 2026

Tob. Induc. Dis. 2026;24(March):40 https://doi.org/10.18332/tid/216379

INTRODUCTION

Cardiometabolic diseases (CMDs) such as stroke, diabetes, and hypertension are becoming increasingly prevalent, posing a significant burden on public health[1]. Compared to individuals with a single CMD, those with multiple face significantly higher mortality rates and shorter life expectancies[2]. Cardiometabolic multimorbidity (CMM) is defined as the coexistence of more than one of the following conditions: Type 2 diabetes, coronary heart disease, and stroke[3]. Studies have demonstrated that lifestyle factors, particularly smoking, are among the primary risk factors for the development and progression of cardiovascular

diseases. Passive smokers (1/100) who are excessively exposed to tobacco smoke have approximately 30% higher risk factors for coronary artery disease (CAD) compared to active smokers (80%)[4]. However, evidence regarding emerging biomarkers (e.g. ePWV and WHtR) and CMM progression remains limited.

Obesity is a potential risk factor for CMM[5]. Generally, the relationship between obesity and CMM is assessed using body mass index (BMI) as an indicator, with some studies reporting its impact on CMM[5,6]. Fat distribution is gaining attention amid growing focus on body composition research. However, BMI cannot distinguish between fat and muscle or assess fat distribution[7]. Studies have illustrated that the waist-to-height ratio (WHtR) has superior predictive ability for cardiovascular disease than BMI[8,9]. A large-scale cross-sectional study in China demonstrated that WHtR performs significantly better in detecting CMM[10]. The effect of obesity on cardiovascular metabolic diseases (CVDs) is related to atherosclerosis[11]. The estimated pulse wave velocity (ePWV) is a non-invasive marker of arterial stiffness and has been extensively studied in cardiovascular research[12]. ePWV is calculated based on age and mean blood pressure (MBP) and can predict the CVD risk and adverse outcomes in patients. However, research on the interaction between WHtR and ePWV in CMM development is limited.

We utilized the large-scale data and prospective cohort design of the China Health and Retirement Longitudinal Study (CHARLS) from 2011 to 2018, to investigate the effects of waist-to-hip ratio (WHtR) and ankle-arm pulse wave velocity (ePWV) on CMM in both smoking and non-smoking populations, and to verify whether ePWV mediates the association between WHtR and CMM risk.

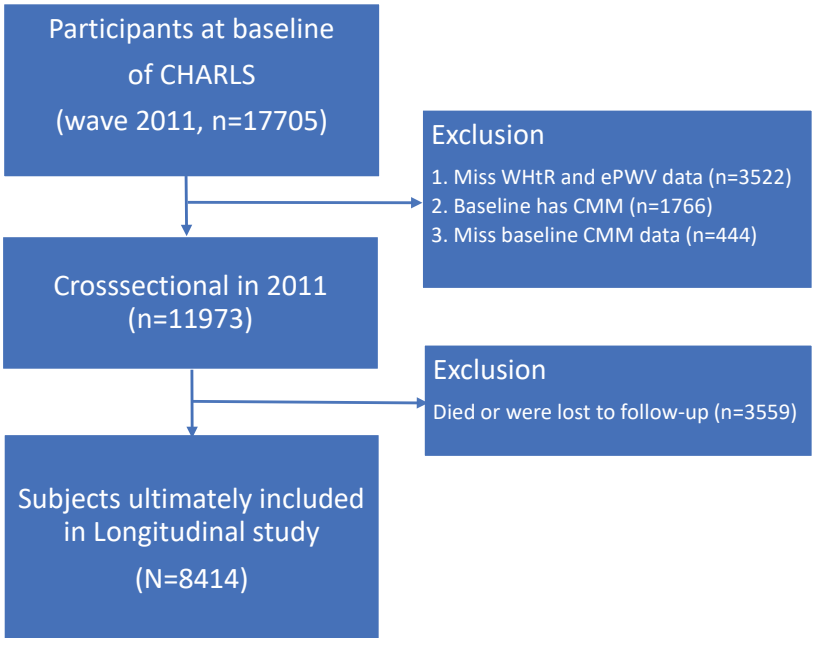
METHODS

Population and data sources

CHARLS was established in 2011 through multi-stage, stratified, and cluster sampling of Chinese adults aged ≥45 years, with follow-up surveys conducted every two years thereafter. This study was approved by the Beijing University Biomedical Ethics Review Committee (IRB00001052-11015), and written informed consent was obtained from all participants.

This study used a unique ID to track participants. Starting with 17705 participants at the 2011 baseline, we excluded 9291 participants for the following reasons: CMM presence at baseline (n=1766), missing CMM data (n=444), missing WHtR and ePWV data (n=3522), and missing follow-up data (n=3559). Ultimately, 8414 participants were included in this study (Figure 1).

Figure 1. The flow chart of participants selection process



ePWV assessment

ePWV, a marker of arterial stiffness, is calculated using a validated formula developed by Greve et al.[13] enabling non-invasive assessment based on age and blood pressure parameters. The calculation formula is as follows:

$ePWV = 9.587 - 0.402 \times age + 4.560\times10^{-3} \times age^2 - 2.621\times10^{-5} \times age^2 \times MBP + 3.176 \times10^{-3} \times age \times MBP - 1.832\times10^{-2} \times MBP$

where MBP is calculated as follows:

$MBP = DBP + 0.4 \times (SBP - DBP)$

Blood pressure was measured using the Omron HEM-7200 device. The participants were seated with their left arm resting on a flat surface at heart level. The cuff was placed 1–2 cm above the elbow. Three consecutive readings were taken, and the average was calculated to ensure measurement reliability.

WHtR assessment

WHtR was calculated by dividing WC by height. Height was measured using standard methods, with participants not wearing shoes. WC was measured to the nearest ±0.5 cm at the smallest circumference between the lowest ribs. The calculation formula is as follows:

WHtR = WC (cm)/height (cm)

CMM definition

In this study, CMM was defined as having at least two CVD conditions, including diabetes, heart disease, and stroke[14]. For the CHARLS database, the diagnosis of heart disease was determined based on questionnaire responses indicating that participants had been diagnosed by a doctor with conditions such as heart failure, coronary heart disease, myocardial infarction, or other heart diseases, or were taking heart disease-related medications. Diabetes was defined as a self-reported physician's diagnosis or the presence of any one of the following biochemical criteria: fasting blood glucose ≥7.8 mmol/L, HbA1c ≥6.0%, or random blood glucose ≥11.1 mmol/L. Additionally, individuals taking diabetes-related medications or receiving insulin injections were also classified as having diabetes. Stroke was identified based on either a participant's report of a physician-diagnosed cerebrovascular event (such as cerebral infarction or cerebral hemorrhage) or the use of antithrombotic or other stroke-specific medications. In the clinical data, heart disease was primarily defined by discharge diagnoses including coronary heart disease, heart failure, myocardial infarction, or other heart diseases. Diabetes was determined based on discharge diagnoses of type 1 or type 2 diabetes. Stroke was identified by discharge diagnoses of cerebrovascular accidents such as cerebral infarction or cerebral hemorrhage.

Accompanying factors

Covariates included age, sex, marital status (married and cohabiting, married but separated, and single), residence (rural, urban), education level (no junior high school education, junior high school or above), annual household expenditure, smoking habits (smoker, non-smoker), drinking habits (drinker, non-drinker), nighttime sleep duration, and BMI.

Statistical methods

The Kolmogorov-Smirnov and Levene tests were used to assess the normality of the distribution and the equality of variances for continuous datasets. All continuous datasets were normally distributed, and continuous variables were compared using t-tests with mean ± standard deviation. Categorical variables were expressed as frequencies and percentages, and compared using the chi-squared test. We recorded the number of follow-up visits from baseline to diagnosis or 31 December 2018 (whichever occurred first). The Cox proportional hazards model was used to assess the association between ePWV, WHtR, and CMM progression in both smoking and non-smoking populations. To investigate the linear and nonlinear relationships between ePWV, WHtR, and CMM risk, both ePWV and WHtR were analyzed as continuous and categorical variables (quartiles). In the preliminary analysis, we separately established Model 1 (crude model) and Model 2 in smoking and non-smoking populations, with the latter adjusted for age, sex, residence, marital status, education level, household expenditure, alcohol consumption, and nighttime sleep duration. We also conducted a mediation analysis to quantify the influence of ePWV on WHtR and CMM. Specifically, WHtR was the

predictor variable (X), ePWV served as the mediator (M), and CMM onset was the outcome variable (Y). This method has been widely used to quantify mediation effects[15].

Additionally, we employed a four-node restricted cubic spline (RCS) function to explore potential non-linear relationships between ePWV and CMM progression, as well as between WHtR and CMM progression. We conducted several sensitivity analyses for further validation of the interaction robustness between ePWV, WHtR, and CMM onset. In sensitivity analysis 1, we excluded participants with strokes at baseline. In sensitivity analysis 2, participants with diabetes or those who had received diabetes treatment at baseline were excluded. In sensitivity analysis 3, participants with heart disease at baseline were excluded. In sensitivity analysis 4, we excluded patients who developed CMM within two years of the baseline survey to minimize latency bias.

All statistical analyses were performed using R statistical software (Version 4.3.0). Statistical significance was set at $p<0.05$ in each analysis.

RESULTS

Descriptive statistics

This study was based on a total of 8414 participants from the CHARLS survey, which was conducted from 2011 to 2018 (Table 1), with an average age of 57.6

Table 1. Baseline characteristics of the longitudinal study population by CMM, CHARLS, 2011–2018 (N=8414)

Characteristics	*Overall (N=8414) n (%)*	*Non-CMM (N=6869) n (%)*	*CMM (N=1545) n (%)*	*p*
Age (years), mean (SD)	57.6 (9.2)	57.0 (9.1)	60.2 (9.1)	<0.001
Gender				
Female	4528 (53.8)	3635 (52.9)	893 (57.8)	0.001
Male	3886 (46.2)	3234 (47.1)	652 (42.2)	
Residence				
Rural	7126 (84.7)	5865 (85.4)	1261 (81.6)	<0.001
Urban	1288 (15.3)	1004 (14.6)	284 (18.4)	
Marital status				
Married and living with a spouse	7211 (85.7)	5922 (86.2)	1289 (83.4)	<0.001
Married but living without a spouse	350 (4.2)	299 (4.4)	51 (3.3)	
Single/divorced/windowed	853 (10.1)	648 (9.4)	205 (13.3)	
Education level				
Less than lower secondary education	7577 (90.1)	6185 (90.0)	1392 (90.1)	0.986
Secondary or higher	837 (9.9)	684 (10.0)	153 (9.9)	
Smoking				
Non-smoker	3242 (38.5)	2686 (39.1)	556 (36.0)	0.025
Smoker	5172 (61.5)	4183 (60.9)	989 (64.0)	
Drinking				
Non-drinker	6054 (72.0)	4879 (71.0)	1175 (76.1)	<0.001
Drinker	2360 (28.0)	1990 (29.0)	370 (23.9)	
Sleep (hours), mean (SD)	6.4 (1.9)	6.4 (1.9)	6.3 (1.9)	<0.001
Household expenditure (RMB), mean (SD)	6764.7 (8612.0)	6813.6 (8802.1)	6547.1 (7709.5)	0.272
BMI (kg/m^2), mean (SD)	23.9 (32.6)	23.1 (6.6)	27.3 (74.7)	<0.001
WHtR, mean (SD)	0.5 (0.1)	0.5 (0.1)	0.6 (0.2)	<0.001
ePWV (m/s), mean (SD)	9.2 (1.8)	9.0 (1.8)	10.1 (1.8)	<0.001

CMM: cardiometabolic multimorbidity. BMI: body mass index. WHtR: waist-to-height ratio. ePWV estimated pulse wave velocity.

years and 46.2% being male. During the maximum 7-year follow-up period, 1545 participants who did not have CMM at baseline (18.36% of the total) subsequently developed CMM. Table 1 presents the baseline characteristics of the study population, categorized by CMM diagnosis. Participants in the CMM group were more likely to be older, female, smokers, single, have lower household expenditure, be non-alcoholic, and have shorter nighttime sleep duration.

Association between ePWV and CMM

Non-smokers

In univariate analyses, each 1-unit increase in ePWV was associated with a 28.9% increase in CMM risk (HR=1.289; 95% CI: 1.253–1.326) (Table 2). After confounding factors adjustment, the HR for CMM was 1.496 (95% CI: 1.421–1.576). EPWV was divided into quartiles, and participants in the first quartile (Q1) were used as the reference group, with an HR of 1.00. Compared with Q1, the HRs for CMM risk in the Q2, Q3, and Q4 groups were 1.994 (95% CI: 1.376–2.890), 3.193 (95% CI: 2.248–4.534), and 5.723 (95% CI: 4.088–8.013) (all p<0.001). After adjusting for confounding factors, the HR for CMM were 2.339 (95% CI: 1.604–3.412), 4.162 (95% CI: 2.871–6.033), and 9.353 (95% CI: 6.271–13.951) (all p<0.001) in subjects with Q2, Q3, and Q4 compared with those with Q1 values.

Smokers

In univariate analyses, each 1-unit increase in ePWV was associated with a 33.5% increase in CMM risk (HR=1.335; 95% CI: 1.281–1.391) (Table 2). After confounding factors adjustment, the HR for CMM was 1.594 (95% CI: 1.487–1.709). ePWV was divided into quartiles, and participants in the first quartile (Q1) were used as the reference group, with an HR of 1.00. Compared with Q1, the HRs for CMM risk in the Q2, Q3, and Q4 groups were 2.549 (95% CI: 2.001–3.245), 4.112 (95% CI: 3.274–5.166), and 5.735 (95% CI: 4.596–7.158) (all p<0.001). After adjusting for confounding factors, the HR for CMM were 2.797 (95% CI: 2.183– 3.584), 4.953 (95% CI: 3.867–6.344), and 8.131 (95% CI: 6.129–10.787) (all p<0.001) in subjects with Q2, Q3, and Q4 compared with those with Q1 values.

Additionally, the RCS model exhibited a non-linear relationship between ePWV and CMM incidence among all participants (non-linear p<0.001) (Figure 2). As ePWV increased, the HR for CMM gradually increased. The dose–response relationship between

Table 2. Hazard ratio and 95% CI of CMM status for ePWV and WHtR, by smoking status, CHARLS, 2011–2018 (N=8414)

Variables	Non-smoker (N=3242)				Smoker (N=5172)			
	Model 1		Model 2		Model 1		Model 2	
	OR (95 % CI)	p	AOR (95 % CI)	p	OR (95 % CI)	p	AOR (95 % CI)	p
ePWV (quartiles)	1.289 (1.253–1.326)	<0.001	1.496 (1.421–1.576)	<0.001	1.335 (1.281–1.391)	<0.001	1.594 (1.487–1.709)	<0.001
Q1 (ref.)								
Q2	1.994 (1.376–2.890)	<0.001	2.339 (1.604–3.412)	<0.001	2.549 (2.001–3.245)	<0.001	2.797 (2.183–3.584)	<0.001
Q3	3.193 (2.248–4.534)	<0.001	4.162 (2.871–6.033)	<0.001	4.112 (3.274–5.166)	<0.001	4.953 (3.867–6.344)	<0.001
Q4	5.723 (4.088–8.013)	<0.001	9.353 (6.271.13.951)	<0.001	5.735 (4.596–7.158)	<0.001	8.131 (6.129–10.787)	<0.001
WHtR (quartiles)	2.082 (1.809–2.396)	<0.001	163.107 (61.217–434.583)	<0.001	5.860 (3.690–9.324)	<0.001	236.281 (44.672–1249.744)	<0.001
Q1 (ref.)								
Q2	1.286 (1.011–1.635)	<0.05	1.276 (1.003–1.624)	<0.05	1.434 (1.115–1.845)	<0.01	1.461 (1.135–1.880)	<0.01
Q3	1.859 (1.466–2.358)	<0.001	1.844 (1.449–2.334)	<0.001	1.844 (1.458–2.332)	<0.001	1.855 (1.465–2.349)	<0.001
Q4	2.914 (2.301–3.689)	<0.001	2.819 (2.198–3.617)	<0.001	3.173 (2.548–3.951)	<0.001	2.966 (2.371–3.712)	<0.001

Model 1: non-adjusted. Model 2: adjusted for age, sex, residence, marital status, education level, household expenditure, alcohol consumption. and nighttime sleep duration.
AOR: adjusted odds ratio.

ePWV and CMM was consistent with the logistic model.

Association between WHtR and CMM

Non-smokers

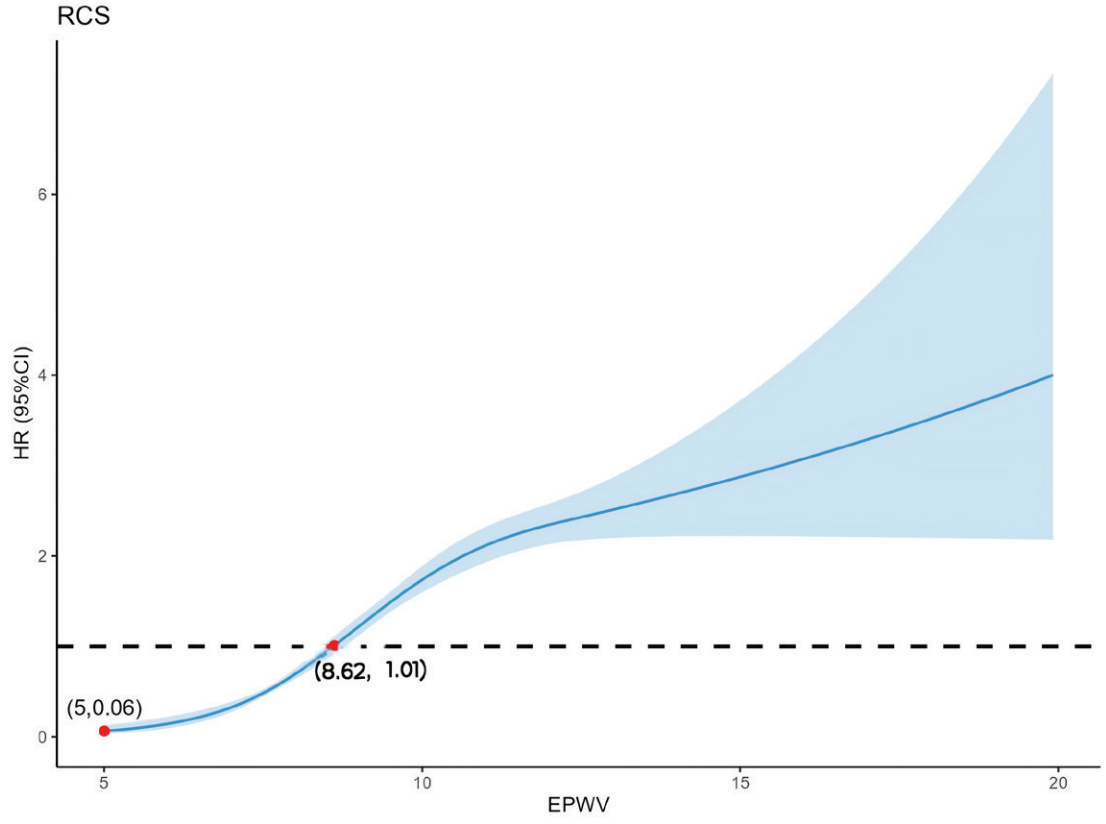
In univariate analyses, each 1-unit increase in WHtR was associated with a 108.2% increase in CMM risk (HR=2.082; 95% CI: 1.809–2.396) (Table 2). After confounding factors adjustment, the HR for CMM was 163.107 (95% CI: 61.217–434.583). WHtR was divided into quartiles, and Q1 participants were used as the reference group, with an OR of 1.00. Compared with Q1, the HRs for CMM risk in the Q2, Q3, and Q4 groups were 1.286 (95% CI: 1.011–1.635), 1.859 (95% CI: 1.466–2.358), and 2.914 (95% CI: 2.301–3.689) (all p<0.05). After adjusting for confounding factors, the HR for CMM were 1.27 (95% CI: 1.003–1.624), 1.844 (95% CI: 1.449–2.3334), and 2.819 (95% CI: 2.198–3.617) (all p<0.05) in subjects with Q2, Q3, and Q4 compared with those with Q1 values.

Smokers

In univariate analyses, each 1-unit increase in ePWV was associated with a 486.00% increase in CMM risk (HR=5.860; 95% CI: 3.690–9.324) (Table 2). After confounding factors adjustment, the HR for CMM was 236.281 (95% CI: 44.672–1249.744). WHtR was divided into quartiles, and participants in the first quartile (Q1) were used as the reference group, with an HR of 1.00. Compared with Q1, the HRs for CMM risk in the Q2, Q3, and Q4 groups were 1.434 (95% CI: 1.115– 1.845), 1.844 (95% CI: 1.458–2.332), and 3.173 (95% CI: 2.548–3.951) (all p<0.001). After adjusting for confounding factors, the HR for CMM were 1.461 (95% CI: 1.135–1.880), 1.855 (95% CI: 1.465–2.349), and 2.966 (95% CI: 2.371–3.712) (all p<0.001) in subjects with Q2, Q3, and Q4 compared with those with Q1 values.

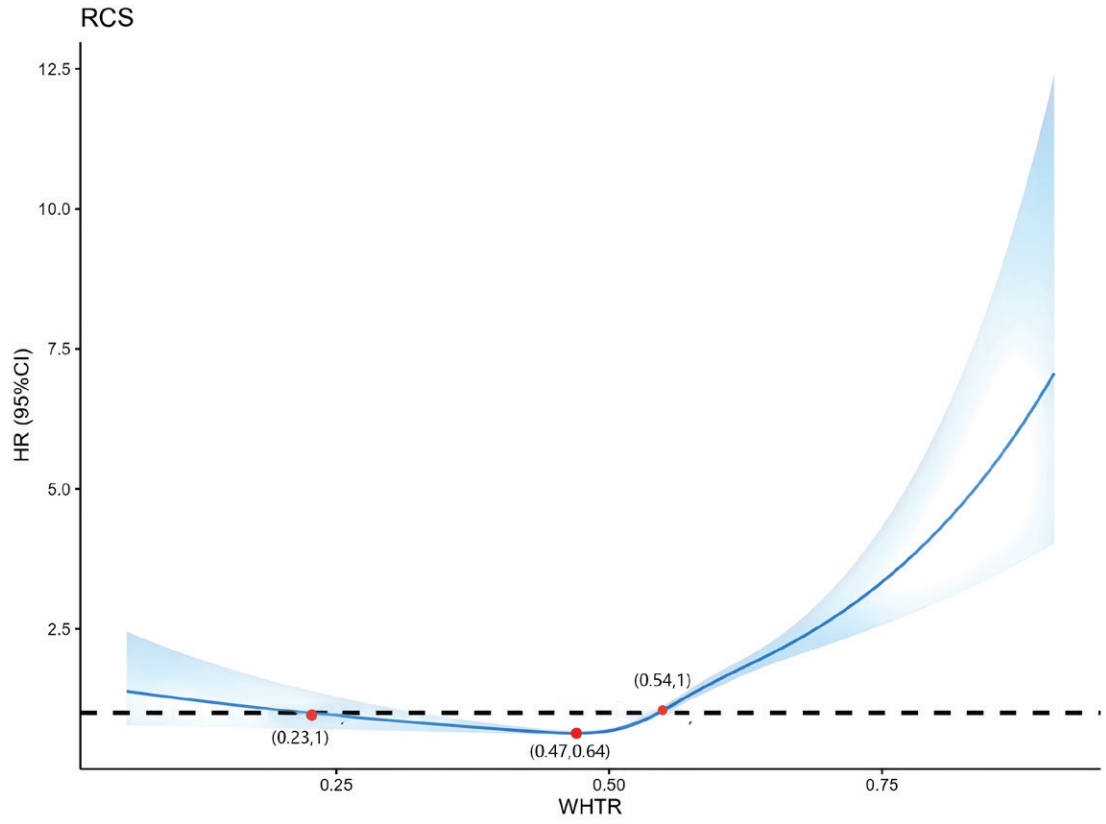
The RCS model exhibited a non-linear relationship between WHtR and CMM incidence among all participants (non-linear p<0.001). WHtR and CMM

Figure 2. Adjusted restricted cubic spline showing the association between ePWV and incident CMM among CHARLS participants, 2011–2018 (N=8414)



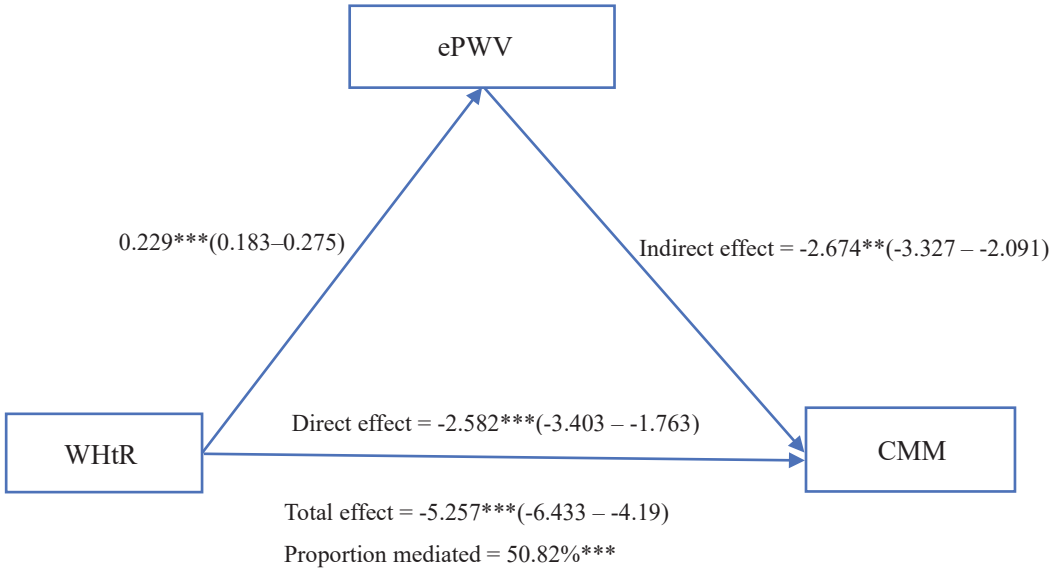
RCS: restricted cubic spline. EPWV: estimated pulse wave velocity.

Figure 3. Adjusted cubic spline models showing the association between WHTR and the prevalence of CMM among CHARLS participants, 2011–2018 (N=8414)



RCS: restricted cubic spline. WHTR: waist-to-height ratio.

Figure 4. Path diagram of the association between WHtR and CMM, with ePWV as a mediator



Mediation analysis of the association between WHtR and risk of CMM by ePWV. Adjusted for age, sex, residence, marital status, education level, household expenditure, alcohol consumption, smoking, and nighttime sleep duration. **p<0.01. ***p<0.001.

incidence exhibited a U-shaped relationship. When WHtR was >0.47, WHtR positively correlated with CMM incidence, and when WHtR was >0.54, HR was >1 (Figure 3).

Sensitivity analyses

We conducted several sensitivity analyses to assess the robustness of the interaction between ePWV, WHtR, and CMM episodes. In sensitivity analysis 1, we excluded participants with a history of stroke at baseline (Supplementary file 1). In sensitivity analysis 2, participants with diabetes or who had received diabetes treatment at baseline were excluded (Supplementary file 2). In sensitivity analysis 3, participants with baseline heart disease were excluded (Supplementary file 3). In sensitivity analysis 4, we excluded participants who developed CMM within two years of the baseline survey to minimize latency bias in the results (Supplementary file 4). The results of the sensitivity analyses were consistent with those of this study.

Mediation analysis

Figure 4 illustrates the potential mediating effect of ePWV on the association between WHtR and CMM risk. In the fully adjusted analysis, the ePWV mediation proportion was 50.82% ($p<0.001$). These results suggest that WHtR may promote CMM development by partially affecting arterial stiffness.

DISCUSSION

To our knowledge, this is the first large-scale longitudinal study to explore the independent and combined effects of ePWV and WHtR on CMM development in middle-aged and older Chinese adults. Our results indicate that higher ePWV and WHtR are associated with greater CMM. Sensitivity analysis further confirmed these findings. Our results also suggest that WHtR promotes CMM development by partially mediating arterial stiffness effects.

Aortic stiffness is an indicator of subclinical disease and is associated with increased risk of various conditions, including hypertension, CKD, and stroke[16]. ePWV is a new, simple parameter proposed by Greve et al.[17] to predict aortic stiffness. Studies have demonstrated that ePWV has predictive value for future CVD events, particularly in healthy individuals and untreated hypertensive patients[18,19]. A small-scale study conducted by Ji et al.[20] in China also revealed that ePWV can predict CVD risk. A longitudinal study conducted by Kim et al.[21] in South Korea further validated this finding. Our study also found that ePWV was associated with the onset of CMM.

Obesity, particularly abdominal fat accumulation, is closely linked to cardiovascular and metabolic diseases[22]. Previous studies have revealed that obesity is associated with individual CMDs[23]. A study in Iran demonstrated that WHtR can effectively predict CVD occurrence[24]. A study conducted in Medellín, Colombia, similarly found that WHtR is a strong predictor of full cardiometabolic risk (CMR)[25]. A Chinese study demonstrated that WHtR is a potential obesity indicator for distinguishing high CMR, with excellent predictive ability[8]. Another Chinese study exhibited that WHtR can independently predict diabetes risk[26], consistent with our results.

CMM is defined as the coexistence of two or three CMDs. Given its higher mortality rate and shorter life expectancy, CMM was considered as an outcome measure. The CHARLS database was used to investigate the relationship between WHtR and CMM in middle-aged and older Chinese adults, providing a nationally representative sample. Among the 5996 participants from 2011 to 2018, 1159 (19.33%) had new-onset CMD. Overall, individuals with CMD had lower WHtR and ePWV than those without CMD. Logistic regression analysis illustrated that ePWV and WHtR were independent predictors of CMM. Moreover, RCS analysis adjusted for confounding factors indicated that ePWV was positively correlated with CMM in all participants (non-linear association), whereas WHtR exhibited a U-shaped relationship with CMM incidence. When WHtR exceeded 0.47, it exhibited a positive correlation with CMM incidence.

The exact mechanisms linking obesity, ePWV, and CMM are still unclear. Studies have revealed that as obesity progresses, macrophages infiltrate ectopic fat, and certain adipokines are up-regulated[26]. Adipokines play a crucial role in inflammation pathogenesis and insulin resistance (IR)[26]. As fat tissue accumulates in organs, it disrupts normal metabolic function and ultimately develops systemic metabolic disorders, such as type 2 diabetes and cardiovascular disease[27].

Our findings indicate a significant association between ePWV, WHtR, and CMM. The mediating role of ePWV between obesity and CMM may result from visceral fat actively secreting excessive inflammatory mediators, which intensify IR and oxidative stress, and worsen endothelial dysfunction[28], thereby increasing CMM risk. Adipocyte proliferation and hypertrophy may induce adipokine dysregulation[29], where adipokines are bioactive peptides secreted by visceral adipose tissue with endocrine functions. Adipokine dysregulation can lead to vascular inflammation and remodeling, as well as endothelial dysfunction[30].

Our study results demonstrated a significant association between smoking and the prevalence of CMM in baseline characteristics, consistent with previous research[4,31]. In the Cox regression analysis, although ePWV and body mass index (WHtR) were independent predictors of CMM in both smoking and non-smoking populations, the hazard ratio (HR) in the smoking group was significantly higher than that in the non-smoking group. This further underscores the importance of smoking cessation as a critical measure for CMM prevention.

This study fills the previous research gap by exploring WHtR and ePWV potential in predicting CMM risk. Our findings remained significant even after adjusting for potential confounding factors. The results remained consistent in sensitivity analyses. Using a large sample from a nationally representative population, we estimated the relationship between WHtR, ePWV, and CMM using Cox regression and RCS models. We also explored the potential mediating role of ePWV in the association between WHtR and CMM risk.

Limitations

This study has several limitations. First, CMM was determined using self-reported diagnostic information from physicians, without medical record verification, which may have led to classification bias. Second, participants with missing data were excluded during the sample selection process, which may introduce selection bias. Third, the CMM definition used in this study only covers diabetes, heart disease, and stroke. This narrower definition may lead to a potential underestimation of the true prevalence of CMM, thereby affecting the results.

CONCLUSIONS

Our findings indicate that ePWV significantly mediates the association between WHtR and new-onset CMM, and that both increased ePWV and WHtR are significantly linked with an increased risk of developing CMM. Early identification and intervention of ePWV and WHtR may be useful for CMM prevention and treatment.

REFERENCES

1. Sawaf B, Swed S, Alibrahim H, et al. Triglyceride-Glucose Index as predictor for hypertension, CHD and stroke risk among non-diabetic patients: A NHANES cross-sectional study 2001-2020. J Epidemiol Glob Health. 2024;14(3):1152-1166. doi:10.1007/s44197-024-00269-7
2. Emerging Risk Factors Collaboration, Di Angelantonio E, Kaptoge S, et al. Association of cardiometabolic multimorbidity with mortality. JAMA. 2015;314(1):52-60. doi:10.1001/jama.2015.7008
3. Busija L, Lim K, Szoeke C, Sanders KM, McCabe MP. Do replicable profiles of multimorbidity exist? Systematic review and synthesis. Eur J Epidemiol. 2019;34(11):1025-1053. doi:10.1007/s10654-019-00568-5
4. Salahuddin S, Prabhakaran D, Roy A. Pathophysiological mechanisms of tobacco-related CVD. Glob Heart. 2012;7(2):113-120. doi:10.1016/j.gheart.2012.05.003
5. Kivimäki M, Kuosma E, Ferrie JE, et al. Overweight, obesity, and risk of cardiometabolic multimorbidity: Pooled analysis of individual-level data for 120 813 adults from 16 cohort studies from the USA and Europe. Lancet Public Health. 2017;2(6):e277-e285. doi:10.1016/S2468-2667(17)30074-9
6. Wall-Medrano A, Ramos-Jiménez A, Hernandez-Torres RP, et al. Cardiometabolic risk in young adults from northern Mexico: Revisiting body mass index and waist-circumference as predictors. BMC Public Health. 2016;16:236. doi:10.1186/s12889-016-2896-1
7. Luo X, Cai B. Association between cardiometabolic index and congestive heart failure among US adults: A cross-sectional study. Front Cardiovasc Med. 2024;11:1433950. doi:10.3389/fcvm.2024.1433950
8. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0·5 could be a suitable global boundary value. Nutr Res Rev. 2010;23(2):247-269. doi:10.1017/S0954422410000144
9. Xu J, Zhang L, Wu Q, et al. Body roundness index is a superior indicator to associate with the cardio-metabolic risk: Evidence from a cross-sectional study with 17,000 Eastern-China adults. BMC Cardiovasc Disord. 2021;21(1):97. doi:10.1186/s12872-021-01905-x
10. Han Y, Hu Y, Yu C, et al. Lifestyle, cardiometabolic disease, and multimorbidity in a prospective Chinese study. Eur Heart J. 2021;42(34):3374-3384. doi:10.1093/eurheartj/ehab413

11. Koenen M, Hill MA, Cohen P, Sowers JR. Obesity, Adipose Tissue and Vascular Dysfunction. Circ Res. 2021;128(7):951-968. doi:10.1161/CIRCRESAHA.121.318093
12. Townsend RR, Wilkinson IB, Schiffrin EL, et al. Recommendations for improving and standardizing vascular research on arterial stiffness: A scientific statement from the American Heart Association. Hypertension. 2015;66(3):698-722. doi:10.1161/HYP.0000000000000033
13. Ke WK, Cheng JP, Xu LL. Association between estimated pulse wave velocity and hip fracture in middle-aged and older adults: A prospective cohort study in China. Bone. 2025;197:117499. doi:10.1016/j.bone.2025.117499
14. Zhang R, Ma J, Wang L. Cardiometabolic multimorbidity in relation to the metabolic score for insulin resistance and creatinine-to-cystatin C ratio in a middle-aged and aged population. Front Endocrinol (Lausanne). 2025;16:1694959. doi:10.3389/fendo.2025.1694959
15. Yang L, Zhang Z, Zhang J, et al. Stage-specific risk factors of cardiometabolic multimorbidity: A systematic review and meta-analysis from incidence to mortality. Ageing Res Rev. 2026;114:102991. doi:10.1016/j.arr.2025.102991
16. Oh YS. Arterial stiffness and hypertension. Clin Hypertens. 2018;24:17. doi:10.1186/s40885-018-0102-8
17. Reference Values for Arterial Stiffness' Collaboration. Determinants of pulse wave velocity in healthy people and in the presence of cardiovascular risk factors: 'establishing normal and reference values'. Eur Heart J. 2010;31(19):2338-2350. doi:10.1093/eurheartj/ehq165
18. Greve SV, Blicher MK, Kruger R, et al. Elevated estimated arterial age is associated with metabolic syndrome and low-grade inflammation. J Hypertens. 2016;34(12):2410-2417. doi:10.1097/HJH.0000000000001083
19. Vlachopoulos C, Terentes-Printzios D, Laurent S, et al. Association of estimated pulse wave velocity with survival: A secondary analysis of SPRINT. JAMA Netw Open. 2019;2(10):e1912831. doi:10.1001/jamanetworkopen.2019.12831
20. Ji C, Gao J, Huang Z, et al. Estimated pulse wave velocity and cardiovascular events in Chinese. Int J Cardiol Hypertens. 2020;7:100063. doi:10.1016/j.ijchy.2020.100063
21. Kim BS, Lee Y, Park JK, Lim YH, Shin JH. Association of the estimated pulse wave velocity with cardio-vascular disease outcomes among men and women aged 40-69 years in the Korean Population: An 18-Year follow-up report on the Ansung-Ansan Cohort in the Korean Genome Environment Study. J Pers Med. 2022;12(10):1611. doi:10.3390/jpm12101611
22. Su Z, Cao L, Chen H, et al. Obesity indicators mediate the association between the aggregate index of systemic inflammation (AISI) and type 2 diabetes mellitus (T2DM). Lipids Health Dis. 2025;24(1):176. doi:10.1186/s12944-025-02589-4
23. Khademi Kolah Loui MH, Jambarsang S, Namayandeh SM, Tabatabaei SM, Hozhabrnia A, Sefidkar R. Comparing the power of obesity indices to predict cardiovascular diseases at different ages: An application of conditional time-dependent ROC curve in Healthy Heart Cohort of Yazd, Iran. ARYA Atheroscler. 2025;21(1):36-43. doi:10.48305/arya.2025.42469.2938
24. Montoya Castillo M, Martínez Quiroz WJ, Suarez-Ortegón MF, Higuita-Gutiérrez LF. Waist-to-Height Ratio, Waist circumference, and Body Mass Index in relation to full cardiometabolic risk in an adult population from Medellin, Colombia. J Clin Med. 2025;14(7):2411. doi:10.3390/jcm14072411
25. Zhang Y, Gao W, Li B, et al. The association between the visceral obesity indices and the future diabetes mellitus risk: A prospective cohort study. Diabetes Obes Metab. 2025;27(8):4490-4498. doi:10.1111/dom.16492
26. Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. Nat Rev Immunol. 2011;11(2):85-97. doi:10.1038/nri2921
27. Britton KA, Fox CS. Ectopic fat depots and cardiovascular disease. Circulation. 2011;124(24):e837-e841. doi:10.1161/CIRCULATIONAHA.111.077602
28. Caballero AE. Endothelial dysfunction in obesity and insulin resistance: A road to diabetes and heart disease. Obes Res. 2003;11(11):1278-1289. doi:10.1038/oby.2003.174
29. Fantin F, Di Francesco V, Rossi A, et al. Abdominal obesity and subclinical vascular damage in the elderly. J Hypertens. 2010;28(2):333-339. doi:10.1097/HJH.0b013e328333d23c
30. Tsioufis C, Tsiachris D, Stefanadis C. Abdominal obesity and arterial stiffness: The differential role of adipokines. Am J Hypertens. 2010;23(5):457. doi:10.1038/ajh.2010.21
31. Sun P, Gao J, Liang X, et al. Gender-specific effects of smoking and alcohol consumption on cardiometabolic diseases and multimorbidity: A cross-sectional study. Tob Induc Dis. 2025;23(September):135. doi:10.18332/tid/208109

CONFLICTS OF INTEREST
The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none was reported.

FUNDING
This research had no source of funding.

ETHICAL APPROVAL AND INFORMED CONSENT
The data comes from a public database, and the project was approved by the Biomedical Ethics Committee of Peking University, Beijing, China (Approval number: IRB00001052-11015; Date: 20 January 2011). All of the participants signed a written informed consent form.

DATA AVAILABILITY
The data supporting this research are available from the following source: The CHARLS dataset is publicly available online, accessible at: http://charls.pku.edu.cn/en

AUTHORS' CONTRIBUTIONS
ZF: research concept and design. LB and SL: data analysis and interpretation. ZF and SL: writing of the manuscript. CW: final approval of the manuscript. All authors read and approved the final version of the manuscript.

PROVENANCE AND PEER REVIEW
Not commissioned; externally peer reviewed.