

Association of Body Roundness Index with Cardiovascular Diseases in Middle-Aged and Older Chinese Adults with Cardiovascular-Kidney-Metabolic Syndrome Stage 0-3: Evidence from the CHARLS Study

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Article

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Abstract

Objective

This study aimed to evaluate the association between the body roundness index (BRI) and the risk of incident cardiovascular disease (CVD) among middle-aged and older Chinese adults in cardiovascular-kidney-metabolic (CKM) syndrome stages 0–3.

Methods

A total of 7,566 participants from the China Health and Retirement Longitudinal Study (CHARLS), free of baseline CVD and classified as CKM stage 0–3, were included. Multivariable Cox models, restricted cubic splines, mediation analyses, and receiver operating characteristic (ROC) curves were used to assess associations, dose-response relationships, potential mediators, and predictive performance.

Results

During a maximum 9.0-year follow-up, 1,836 incident CVD cases were documented. Each 1-unit increase in BRI was associated with a 15.4% higher risk of CVD (HR: 1.154, 95% CI: 1.112–1.199), 13.4% for heart disease, and 18.7% for stroke. Participants in the highest BRI quartile had significantly elevated risks compared to the lowest quartile. A linear dose-response relationship was observed. Hypertension and dyslipidemia mediated 10.8% and 11.6% of the association, respectively. BRI demonstrated superior predictive ability for CVD (AUC=0.71) compared to body mass index (BMI) and A Body Shape Index (ABSI).

Conclusion

Higher BRI levels are independently associated with increased CVD risk in CKM stages 0–3, with a dose-response relationship. BRI outperforms BMI and ABSI in risk prediction, partially mediated by hypertension and dyslipidemia, supporting its use for early CVD stratification in this population.

Highlights

- Higher body roundness index (BRI) increases cardiovascular disease risk in CKM stages 0–3.
- BRI shows a linear dose-response relationship with incident CVD, heart disease, and stroke.
- Hypertension and dyslipidemia partially mediate the association between BRI and CVD.
- BRI outperforms BMI and ABSI in predicting cardiovascular risk among Chinese adults.
- Supports using BRI for early risk stratification in cardiovascular-kidney-metabolic health.

1. Introduction

Cardiovascular disease (CVD), primarily including heart disease and stroke, is one of the leading causes of mortality and disability worldwide ^[1]. Between 1990 and 2022, the global prevalence of CVD increased markedly from 271 million to 523 million, constituting a major public health challenge in the 21st century^[2]. In China, CVD has become the leading cause of death, with CVD-related mortality increasing by 58.5% from 1990 to 2016, underscoring the importance of early identification and primary prevention at the population level ^[3].

Among the numerous modifiable risk factors for CVD, obesity is particularly prominent, with abdominal obesity showing a stronger association with CVD risk than general obesity as measured by body mass index (BMI) ^[4, 5]. BMI, widely used in clinical practice, cannot distinguish between fat and lean mass, nor can it effectively capture the heterogeneity of fat distribution, particularly visceral fat accumulation, which carries high metabolic risk ^[6]. To address this limitation, the body roundness index (BRI) was proposed^[7]. Derived from an elliptical geometric model of the human body, BRI integrates waist circumference and height, enabling a more precise estimation of body fat percentage and visceral fat area compared with traditional indices such as BMI, waist circumference (WC), and hip circumference. Multiple studies have demonstrated that BRI is significantly associated with metabolic syndrome, insulin resistance, diabetes, and CVD, suggesting its potential predictive value beyond conventional measures ^[8–10]. Furthermore, longitudinal studies have shown that higher BRI is strongly associated with increased risk of all-cause and CVD-specific mortality, displaying a U-shaped relationship ^[11–13]. Evidence from cohort studies has further indicated that higher BRI trajectories are linked to increased CVD risk ^[14]. Thus, BRI provides a more comprehensive reflection of abdominal obesity and may serve as a more sensitive and clinically meaningful predictor of CVD risk.

It is noteworthy that obesity often coexists and interacts with multiple disorders, including glucose metabolism abnormalities, dyslipidemia, hypertension, and impaired renal function. To integrate this complex comorbidity network, the American Heart Association (AHA) proposed the conceptual framework of cardiovascular-kidney-metabolic (CKM) syndrome ^[15]. This framework highlights the pathophysiological interactions among the heart, kidneys, and metabolic systems. Its early stages (0–3) encompass a broad population ranging from individuals with only risk factors to those with subclinical target organ damage. Epidemiological evidence indicates that the majority of adults worldwide fall within CKM stages 0–3, among whom the future risk of cardiovascular events and CVD-related mortality varies substantially ^[16–18]. The AHA recommends that CKM stages 0–3 should focus primarily on the prevention of CVD events ^[15]. Given that obesity lies at the core of CKM syndrome, refined risk stratification based on obesity assessment is essential to identify truly high-risk individuals. Although existing evidence suggests that BRI has potential utility, it remains unclear whether BRI can effectively elucidate the link between obesity and CVD, and whether its predictive value is independent of and superior to conventional indices within the CKM framework.

This study, based on data from the nationally representative prospective China Health and Retirement Longitudinal Study (CHARLS), aimed to examine the association between baseline BRI levels and

incident cardiovascular events—including overall CVD, coronary heart disease, and stroke—among middle-aged and older adults in CKM stages 0–3. We specifically investigated whether BRI was independently associated with CVD and exhibited a dose–response relationship, evaluated the potential mediating effects of key CKM intermediates (such as hypertension, dyslipidemia, diabetes, and metabolic syndrome), and compared the predictive value of BRI with other obesity-related indices for CVD risk.

2. Methods

2.1 Data Sources and Study Population

This study was a secondary analysis of the CHARLS (<http://charls.pku.edu.cn/>). CHARLS is a nationally representative cohort study that recruited participants using a multistage stratified probability sampling method from 150 counties/districts across 28 provinces in China. The baseline survey (2011–2012, wave 1) included 17,705 residents aged 45 years or older from 10,257 households ^[19, 20].

Sociodemographic characteristics, lifestyle factors, and health status were collected through standardized questionnaires ^[21]. To date, five waves of follow-up data have been released (2013–2014, 2015–2016, 2017–2018, and 2019–2020). For this analysis, we included the 17,708 baseline participants and excluded those with: (1) missing CKM data at baseline; (2) missing CVD data or a history of CVD at baseline; (3) missing waist circumference or height data, or extreme BRI values; and (4) age < 45 years. A total of 7,566 participants were ultimately included in the analysis. The detailed flowchart is shown in Fig. 1. The CHARLS study was approved by the Institutional Review Board of Peking University (IRB00001052-11015), and all participants provided written informed consent.

2.2 Definition of CKM

CKM syndrome staging was assessed according to the AHA Presidential Advisory Statement ^[15]. CKM stages were classified as follows: stage 0, no CKM risk factors; stage 1, overweight/obesity, abdominal obesity, or prediabetes without other metabolic abnormalities or CKD; stage 2, presence of hypertension, hypertriglyceridemia, diabetes, metabolic syndrome, or CKD; stage 3, subclinical cardiovascular disease, defined as high-risk CKD (KDIGO G4–G5, eGFR < 30 mL/min/1.73m²) or a 10-year CVD risk ≥ 20%, considered equivalent to subclinical CVD; and stage 4, clinically diagnosed CVD. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI equation ^[22], and CKD staging was classified according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines ^[23]. CVD risk was estimated using the Framingham risk score based on multiple risk factors ^[24]. Detailed definitions and Framingham scoring criteria are provided in Supplementary Tables S1–S4.

2.3 Assessment of BRI

BRI is a novel anthropometric index derived from height and waist circumference (WC) measurements [7]

It was calculated using the following formula: $364.2 - 365.5 \times \sqrt{1 - \frac{(WC/2\pi)^2}{0.5 \times height^2}}$

WC was measured with a non-elastic tape, and height was measured with a stadiometer, both to the nearest 0.1 cm.

2.4 Definition of Outcomes

CVD was defined as a composite endpoint of heart disease and stroke. The primary outcome was incident CVD during follow-up, and secondary outcomes were heart disease and stroke considered separately. CVD was identified using standardized questions: (1) “Have you been told by a doctor that you have been diagnosed with a heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems?” and (2) “Have you been told by a doctor that you have been diagnosed with a stroke?” Participants answering “yes” to either question were considered to have experienced a CVD event. Outcome ascertainment in this study was consistent with our prior research using CHARLS [25].

2.5 Covariates

Baseline covariates were obtained from questionnaires, physical examinations, and laboratory tests. Sociodemographic characteristics included age, sex (male/female), residence (urban/rural), marital status (currently married/other), and educational attainment (illiterate, primary school, middle school, high school or above). Lifestyle factors included smoking (yes/no), alcohol consumption (yes/no), and household fuel use (clean fuel/solid fuel). Clinical factors included body mass index (BMI), hypertension, diabetes, kidney disease, liver disease, dyslipidemia, and metabolic syndrome. Laboratory tests included fasting blood glucose, HbA1c, lipids (total cholesterol [TC], triglycerides [TG], high-density lipoprotein cholesterol [HDL-C], and low-density lipoprotein cholesterol [LDL-C]), uric acid, estimated glomerular filtration rate (eGFR), and high-sensitivity C-reactive protein (hs-CRP). Household fuel use was categorized as clean fuel (natural gas, biogas, liquefied petroleum gas, electricity) or solid fuel (coal, crop residues, wood, charcoal). Hypertension was defined as measured blood pressure $\geq 130/85$ mmHg or self-reported physician diagnosis/treatment [26]. Glucose metabolism status (GMS) was classified according to the American Diabetes Association (ADA) criteria as normal glucose regulation (NGR), prediabetes, or diabetes [27]. Dyslipidemia was defined as any of the following: self-reported diagnosis, current use of lipid-lowering medication, or abnormal laboratory lipid values [28]. Metabolic syndrome was defined according to the International Diabetes Federation (IDF) criteria [29]. Detailed definitions are provided in Supplementary Table S1.

2.6 Statistical analysis

Baseline characteristics were described across BRI quartiles. Normally distributed variables were presented as mean $\pm$ standard deviation, skewed variables as median (interquartile range), and categorical variables as frequency (percentage). Group comparisons were performed using analysis of variance (ANOVA), Kruskal–Wallis tests, or chi-square tests, as appropriate. Collinearity among BRI and

other covariates was assessed using variance inflation factors. Kaplan–Meier methods were applied to estimate cumulative incidence rates of CVD, HD, and stroke across groups. Participants were categorized into quartiles of BRI (Q1–Q4). Multivariable-adjusted Cox proportional hazards models were used to evaluate the association between BRI levels and CVD incidence, with hazard ratios (HRs) and 95% confidence intervals (CIs) calculated. Three models were progressively constructed: Model 1 adjusted for age and sex; Model 2 further adjusted for residence, marital status, education, smoking, alcohol consumption, and household fuel use; and Model 3 further adjusted for hypertension, diabetes, kidney disease, LDL-C, HbA1c, and hs-CRP. Trend tests were conducted by modeling the median value of each quartile as a continuous variable. Additionally, fully adjusted restricted cubic spline (RCS) analyses were conducted to examine dose–response relationships between BRI and risks of CVD, heart disease, and stroke. Mediation analysis using a bootstrap framework (1,000 resamples) was performed to estimate the mediating effects of hypertension, diabetes, dyslipidemia, and metabolic syndrome on the BRI–CVD association, with confidence intervals calculated for direct effects, average causal mediation effects, and the proportion mediated. Subgroup analyses were stratified by age, sex, education level, residence, BMI, hypertension, dyslipidemia, metabolic syndrome, and glucose metabolism status. Interaction terms were added to the models, and Wald tests were used to assess effect modification. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive performance of BRI and other anthropometric indices (e.g., BMI and A Body Shape Index [ABSI]) for CVD risk, and to assess BRI's predictive utility across CKM stages. Sensitivity analyses included: (1) repeating the primary analyses among participants with complete data (excluding physical activity; $n = 6,881$); (2) excluding individuals using lipid-lowering, hypoglycemic, or antihypertensive medications; and (3) excluding participants with missing physical activity data and additionally adjusting for physical activity in the model. All statistical analyses were conducted using R software (version 4.3.0), and two-sided $P < 0.05$ was considered statistically significant.

3. Results

3.1 Baseline Characteristics of Study Participants

A total of 7,566 participants from the CHARLS study were included in the final analysis. The mean age of participants was 59.3 (9.3) years, and 3,646 (48.2%) were men. Participants were categorized according to BRI quartiles, as shown in Table 1. Participants in higher BRI quartiles tended to be older, female, urban residents, have lower educational attainment, use clean fuels, and engage in lower levels of physical activity. They also had higher BMI, Total-C, TG, LDL-C, HbA1c, and hs-CRP, and were more likely to have comorbidities such as hypertension, diabetes, metabolic syndrome, and dyslipidemia. In contrast, they had significantly lower rates of smoking and alcohol consumption, as well as lower levels of HDL-c and eGFR. Marital status and history of kidney disease showed statistically significant differences across groups, whereas no significant differences were observed for liver disease or uric acid history. These findings suggest that higher BRI levels are associated with poorer cardiovascular risk profiles.

Table S5 summarizes baseline characteristics according to CVD status. No statistically significant differences were observed in residence, marital status, education level, smoking status, history of kidney disease, or uric acid levels ($P > 0.05$). Compared with participants without CVD, those with CVD were older, more likely to be female, and exhibited higher BMI, hs-CRP, TG, Total-C, LDL-C, and HbA1c, while showing lower levels of eGFR and HDL-C. They were also more likely to use solid fuels, have low physical activity, and present with comorbidities such as hypertension, diabetes, dyslipidemia, liver disease, and metabolic syndrome.

Table 1
Baseline characteristics of the study participants.

Characteristics	Total	Q1(< 3.20)	Q2(3.20-4.00)	Q3(4.00-5.04)	Q4(> 5.04)	P value
Participants, No.	7566	1892	1892	1891	1891	
Age, years	59.3 (9.3)	59.1 (9.2)	59 (9.2)	58.6 (9.2)	60.4 (9.5)	< 0.001
Sex, male	3646 (48.2%)	1294 (68.4%)	1059 (56%)	837 (44.3%)	456 (24.1%)	< 0.001
Residence, urban	2527 (33.4%)	480 (25.4%)	612 (32.3%)	706 (37.3%)	729 (38.6%)	< 0.001
Current married	6675 (88.2%)	1679 (88.7%)	1668 (88.2%)	1701 (90%)	1627 (86%)	0.002
Education						< 0.001
Illiterate	2191 (29%)	466 (24.6%)	501 (26.5%)	509 (26.9%)	715 (37.8%)	
Primary	3137 (41.5%)	843 (44.6%)	799 (42.3%)	782 (41.4%)	713 (37.7%)	
Middle school	1508 (19.9%)	400 (21.2%)	387 (20.5%)	383 (20.3%)	338 (17.9%)	
High school+	727 (9.6%)	182 (9.6%)	204 (10.8%)	217 (11.5%)	124 (6.6%)	
Smoking	3037 (40.1%)	1092 (57.7%)	873 (46.1%)	671 (35.5%)	401 (21.2%)	< 0.001
Alcohol consumption	2008 (26.5%)	650 (34.4%)	584 (30.9%)	478 (25.3%)	296 (15.7%)	< 0.001
Fuel for cooking						
Clean fuels	1379 (18.2%)	287 (15.2%)	335 (17.7%)	373 (19.7%)	384 (20.3%)	
Solid fuels	5657 (74.8%)	1514 (80.0%)	1436 (75.9%)	1367 (72.3%)	1340 (70.9%)	
Fuel for heating						< 0.001
Clean fuels	3138 (41.5%)	667 (35.3%)	802 (42.4%)	837 (44.3%)	832 (44.0%)	
Solid fuels	4367 (57.7%)	1208 (63.8%)	1077 (56.9%)	1037 (54.8%)	1045 (55.3%)	

Characteristics	Total	Q1(< 3.20)	Q2(3.20-4.00)	Q3(4.00-5.04)	Q4(> 5.04)	P value
BMI, kg/m2						
Continuous	23.4(3.7)	20.0(2.1)	22.1(2.1)	24.3(2.7)	27.2(3.5)	< 0.001
< 23.9	4491 (59.4%)	1843 (97.4%)	1569 (82.9%)	815 (43.1%)	264 (14.0%)	
24-27.9	2258 (29.8%)	42 (2.2%)	311 (16.4%)	995 (52.6%)	910 (48.1%)	
≥ 28	817 (10.8%)	7 (0.4%)	12 (0.6%)	81 (4.3%)	717 (37.9%)	
Hypertension	3533 (46.7%)	647 (34.2%)	769 (40.7%)	942 (49.8%)	1175 (62.1%)	
GMS						< 0.001
NGR	4875 (64.4%)	1402 (74.1%)	1298 (68.6%)	1158 (61.2%)	1017 (53.8%)	
Pre-diabetes	1419 (18.8%)	297 (15.7%)	334 (17.7%)	363 (19.2%)	425 (22.5%)	
Diabetes	1272 (16.8%)	193 (10.2%)	260 (13.7%)	370 (19.6%)	449 (23.7%)	
Dyslipidemia	549 (7.3%)	57 (3.0%)	95 (5.0%)	159 (8.4%)	238 (12.6%)	< 0.001
Liver disease	276 (3.6%)	61 (3.2%)	64 (3.4%)	82 (4.3%)	69 (3.6%)	0.273
Kidney disease	592 (7.8%)	173 (9.1%)	144 (7.6%)	149 (7.9%)	126 (6.7%)	0.041
Metabolic Syndrome	1183 (15.6%)	83 (4.4%)	219 (11.6%)	352 (18.6%)	529(28.0%)	< 0.001
Physical activity						< 0.001
Low	985 (13.0%)	188 (9.9%)	213 (11.3%)	248 (13.1%)	336 (17.8%)	
Moderate	941 (12.4%)	207 (10.9%)	231 (12.2%)	251 (13.3%)	252 (13.3%)	
High	1267 (16.7%)	390 (20.6%)	334 (17.7%)	306 (16.2%)	237 (12.5%)	
Total-C, mg/dl	194.7 (38.7)	187.0 (36.4)	191.8 (35.7)	197.8 (40.8)	202.0 (39.9)	< 0.001

Characteristics	Total	Q1(< 3.20)	Q2(3.20-4.00)	Q3(4.00-5.04)	Q4(> 5.04)	P value
LDL-C, mg/dl	116.6 (35.1)	110.9 (32.3)	114.7 (33.0)	119.1 (36.5)	121.8 (37.4)	< 0.001
HDL-C, mg/dl	52.0 (15.3)	57.7 (15.7)	53.8 (15.4)	49.7 (14.5)	46.8 (13.4)	< 0.001
TG, mg/dl	104.4(74.3-155.8)	82.3 (62.8-116.8)	96.5 (69.0-144.3)	112.4 (81.4-164.6)	132.8 (93.8-197.4)	< 0.001
HbA1c, %	5.1 (4.9–5.4)	5.1 (4.8–5.3)	5.1 (4.8–5.4)	5.2 (4.9–5.5)	5.2 (5.0-5.6)	< 0.001
hsCRP, mg/L	1.0 (0.5–2.1)	0.8 (0.4–1.7)	0.8 (0.5–1.8)	1.1 (0.6–2.1)	1.4 (0.8–2.8)	< 0.001
Uric acid, mg/dL	4.3 (3.6–5.2)	4.3 (3.5–5.1)	4.3 (3.6–5.2)	4.3 (3.6–5.2)	4.4 (3.7–5.2)	0.136
eGFR, ml/min/1.73 m ²	98.9 (88.7–105.6)	99.6(90.7–106.1)	99.6(89.5–106.0)	98.8 (88.8–105.8)	97.3 (85.9–104.4)	< 0.001
CKM stages						< 0.001
0	1051 (13.9%)	346 (18.3%)	618 (32.7%)	81 (4.3%)	6 (0.3%)	
1	1098 (14.5%)	143 (7.6%)	281 (14.9%)	368 (19.5%)	306 (16.2%)	
2	2832 (37.4%)	515 (27.2%)	633 (33.5%)	766 (40.5%)	918 (48.5%)	
3	2585 (34.2%)	616 (32.6%)	632 (33.4%)	676 (35.7%)	661 (35.0%)	
CVD, %	1836 (24.3%)	322 (17.0%)	413 (21.8%)	502 (26.5%)	599 (31.7%)	< 0.001
Heart disease, %	1407 (18.6%)	250 (13.2%)	314 (16.6%)	377 (19.9%)	466 (24.6%)	< 0.001
Stroke, %	642 (8.5%)	102 (5.4%)	143 (7.6%)	188 (9.9%)	209 (11.1%)	< 0.001

Data are presented as the mean ± SD, median (IQR), or number (%), as appropriate. Abbreviations: SD, standard deviation; IQR, interquartile range; hs-CRP, high sensitivity C-reactive Protein; CKM, Cardio-Kidney-Metabolic Syndrome; BMI, body mass index; GMS, glycemic metabolic status; NGR, normal glucose regulation; HbA1c, glycated Hemoglobin, Total-C, total cholesterol, TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; N of missing: Education(n =

3); Fuel for cooking (n = 530); Fuel for heating (n = 61); LDL-C(n = 15); HbA1c(n = 1); eGFR (n = 4); Physical activity (n = 4373).

3.2 Association Between BRI and CVD in Patients with CKM Syndrome

Collinearity analysis (**Supplementary Table S6**) indicated no significant multicollinearity among variables, with variance inflation factors (VIFs) < 5 for all covariates. During a maximum follow-up period of 9.0 years, 1,836 participants (24.3%) experienced new-onset CVD events, including 1,407 cases of heart disease (18.6%) and 642 cases of stroke (8.5%). We observed a significant association between BRI and CVD in patients with CKM syndrome (**Table 2**). In Model 1 (adjusted for age and sex only), per one-unit increase in baseline BRI was associated with an 18.3% higher risk of CVD (HR: 1.183, 95% CI: 1.144–1.224), a 23.9% higher risk of stroke (HR: 1.239, 95% CI: 1.171–1.310), and a 15.4% higher risk of heart disease (HR: 1.154, 95% CI: 1.111–1.200). When comparing Q4 with Q1, the HRs were 1.945 (95% CI: 1.686–2.244) for CVD, 2.448 (95% CI: 1.908–3.141) for stroke, and 1.773 (95% CI: 1.508–2.085) for heart disease. These findings indicate a significant association between BRI and CVD risk. After further adjustment for multiple covariates, the significant association between BRI and CVD risk remained robust. Moreover, CVD risk increased progressively across BRI quartiles (P for trend < 0.001), underscoring the importance of BRI in CVD risk assessment. Kaplan–Meier cumulative incidence curves demonstrated that participants in higher BRI quartiles had significantly higher incidence of CVD, heart disease, and stroke throughout the follow-up period (Fig. 2).

Table 2. Association between BRI levels and CVD in the population with CKM syndrome stages 0-3.

	Model 1		Model 2		Model 3	
	HR (95% CI)	<i>P</i> value	HR (95% CI)	<i>P</i> value	HR (95% CI)	<i>P</i> value
CVD						
BRI (per 1 unit)	1.183 (1.144-1.224)	<0.001	1.188 (1.146-1.232)	<0.001	1.154 (1.112-1.199)	<0.001
Q1	1(Ref.)		1(Ref.)		1(Ref.)	
Q2	1.319 (1.14-1.527)	<0.001	1.304 (1.119-1.519)	<0.001	1.272 (1.091-1.483)	0.002
Q3	1.624 (1.409-1.872)	<0.001	1.62 (1.395-1.881)	<0.001	1.515 (1.302-1.763)	<0.001
Q4	1.945 (1.686-2.244)	<0.001	1.984 (1.706-2.307)	<0.001	1.786 (1.529-2.087)	<0.001
<i>P</i> for trend	<0.001		<0.001		<0.001	
Heart disease						
BRI (per 1 unit)	1.154 (1.111-1.200)	<0.001	1.157 (1.109-1.206)	<0.001	1.134 (1.086-1.184)	<0.001
Q1	1(Ref.)		1(Ref.)		1(Ref.)	
Q2	1.251 (1.059-1.478)	0.008	1.212 (1.016-1.445)	0.032	1.193 (1.000-1.423)	0.050
Q3	1.477 (1.256-1.737)	<0.001	1.468 (1.236-1.742)	<0.001	1.400(1.177-1.666)	<0.001
Q4	1.773 (1.508-2.085)	<0.001	1.791 (1.509-2.127)	<0.001	1.673 (1.402-1.997)	<0.001
<i>P</i> for trend	<0.001		<0.001		<0.001	
Stroke						
BRI (per 1 unit)	1.239 (1.171-1.31)	<0.001	1.256 (1.184-1.333)	<0.001	1.187 (1.115-1.264)	<0.001
Q1	1(Ref.)		1(Ref.)		1(Ref.)	
Q2	1.486 (1.152-1.917)	0.002	1.491 (1.149-1.935)	0.003	1.41 (1.086-1.831)	0.01
Q3	2.089 (1.637-2.666)	<0.001	2.056 (1.596-2.648)	<0.001	1.806 (1.398-2.333)	<0.001
Q4	2.448 (1.908-3.141)	<0.001	2.543 (1.964-3.293)	<0.001	2.052 (1.573-2.677)	<0.001

<i>P</i> for trend	<0.001	<0.001	<0.001
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Abbreviations: HR, hazard ratio; CI, confidence interval; BRI, body roundness index; CVD, cardiovascular disease; Model 1: adjusted for age, sex; Model 2: model 1 plus residence, marriage, education, smoking, alcohol consumption and household fuel use; Model 3: model 2 plus hypertension, diabetes, Kidney disease, LDL-c, HbA1c and hs- CRP.

RCS analysis demonstrated a significant overall association between BRI and cardiovascular disease among individuals with CKM stages 0–3 (P overall < 0.001), with an evident dose–response relationship (P nonlinear > 0.05; see **Figure 3**). In the total population with CKM stages 0–3 (Panels A–C), higher BRI levels were associated with increased risks of CVD, heart disease, and stroke. The HR curves showed an overall upward trend, indicating a clear dose–response relationship. The reference point (red vertical line), corresponding to the median BRI, demonstrated that risk continued to increase above the median, whereas risk was relatively lower below the median. In sex-stratified analyses (Panels D–I), both men (Panels D–F) and women (Panels G–I) exhibited similar dose–response relationships, with risks increasing linearly throughout the BRI range. Overall, elevated BRI was positively associated with cardiovascular event risk in a dose–response manner, without evidence of a significant nonlinear pattern.

Panels A–C depict the associations between BRI and CVD (A), heart disease (B), and stroke (C) among individuals with CKM syndrome stages 0–3. Panels D–F present results for men, while panels G–I present results for women. The red vertical line indicates the median BRI. All models were adjusted for age, marital status, educational attainment, residence, smoking, alcohol consumption, household fuel use, LDL-c, HbA1c, CRP, hypertension, and diabetes.

3.3 Mediation Analysis

Figure 4 illustrates the mediating roles of metabolic disorders in the association between BRI and CVD. In unadjusted models, hypertension, diabetes, dyslipidemia, and metabolic syndrome significantly mediated the association, with mediation proportions of 12.4% (95% CI: 8.4%–17.8%), 4.4% (95% CI: 1.3%–7.6%), 9.6% (95% CI: 6.6%–13.1%), and 5.1% (95% CI: 0.6%–9.8%), respectively. After full adjustment, the mediating effects of hypertension and dyslipidemia remained robust and statistically significant, with proportions of 10.8% (95% CI: 5.9%–17.0%) and 11.6% (95% CI: 7.6%–17.4%), respectively. The mediating effects of diabetes and metabolic syndrome were substantially attenuated and no longer statistically significant. For heart disease, in fully adjusted models, hypertension and dyslipidemia significantly mediated the association between BRI and heart disease, with mediation proportions of 8.6% and 13.3%, respectively (**Figure S1**). For stroke, after full adjustment, hypertension and dyslipidemia significantly mediated the association between BRI and stroke, with mediation proportions of 16.1% and 8.7%, respectively (**Figure S2**).

The model was adjusted for age, marital status, educational level, residence, smoking, drinking, household fuel use, LDL-c, HbA1c, hs-CRP, hypertension, and diabetes.

3.4 Receiver Operating characteristic Analysis

This study employed ROC curves to evaluate the predictive performance of BRI, BMI, and ABSI for CVD, heart disease, and stroke (**Figure 5A–C**). For CVD prediction, BRI yielded the highest area under the curve (AUC) of 0.71, outperforming BMI (AUC = 0.573) and ABSI (AUC = 0.528). Similarly, BRI demonstrated superior predictive ability over BMI and ABSI in the prediction of both heart disease and stroke. We further assessed the predictive performance of BRI across different CKM stages (**Figure 5D–F**). The results indicated that the predictive ability of BRI progressively increased with advancing CKM stage. For CVD, the AUC of BRI increased from 0.526 in CKM stage 0 to 0.552 in stage 1, 0.577 in stage 2, and 0.582 in stage 3. Comparable trends were observed for heart disease and stroke, with the highest predictive performance of BRI evident in CKM stages 2 or 3.

Panels A–C: ROC curves illustrating the predictive performance of BRI, BMI, and ABSI for CVD (A), heart disease (B), and stroke (C). Panels D–F: ROC curves showing the discriminatory ability of BRI for predicting CVD (D), heart disease (E), and stroke (F) across CKM stages 0–3.

3.5 Subgroup Analysis

Stratified analyses revealed effect modification by sex, educational attainment, and place of residence (**Figure 6**). Men exhibited a greater increase in CVD risk (HR = 1.225, 95% CI: 1.146–1.308) compared with women (P for interaction = 0.026). With increasing educational level, risk rose progressively, with the highest risk observed among individuals with a high school education or above (HR = 1.307, 95% CI: 1.130–1.512; P for interaction = 0.010). Urban residents (HR = 1.204, 95% CI: 1.124–1.289) had a higher CVD risk compared with rural residents (P for interaction = 0.036). Age also demonstrated a potential modifying effect on the association between BRI and CVD risk: risk was more pronounced among individuals aged 45–59 years (HR = 1.201, 95% CI: 1.135–1.272) and 60–69 years (HR = 1.189, 95% CI: 1.120–1.262), while the association appeared attenuated in those aged ≥ 70 years (P for interaction = 0.083). No significant interactions were observed across subgroups defined by BMI, hypertension, dyslipidemia, metabolic syndrome, or glycemic status.

HR (95% CIs) of CVD risk per 1 unit increase in BRI. The model was adjusted for age, marital status, educational level, residence, smoking, drinking, household fuel use, LDL-c, HbA1c, hs-CRP, hypertension, and diabetes.

3.6 Sensitivity Analyses

Multiple sensitivity analyses confirmed the robustness of the results (**Table S8**). These included repeating the analyses in the full dataset ($n = 6973$) and excluding participants with a history of medication use ($n = 6052$). In addition, adjustment for physical activity was performed among 3,193 participants with available data, and the results remained consistent.

4. Discussion

This prospective, nationwide cohort study provides evidence that BRI are associated with the risk of CVD among adults with CKM syndrome stages 0–3. The main findings can be summarized as follows. First, after comprehensive adjustment for a wide range of potential confounders, higher baseline BRI was independently and robustly associated with increased risks of total CVD, heart disease, and stroke, showing a significant dose–response relationship. Second, mediation analysis indicated that a substantial proportion of this association was mediated by hypertension and dyslipidemia. Third, the strength of the association varied by sex, educational attainment, and place of residence, suggesting that the risk conveyed by BRI is not uniform across subpopulations.

Previous studies have mainly focused on the association between BRI and CVD incidence, identifying BRI as a predictor of CVD ^[14, 30]. Cross-sectional studies in China have demonstrated that BRI outperforms other anthropometric indices in predicting CVD risk factors ^[31]. In addition, Maessen et al. reported that individuals in the highest BRI quintile had a higher incidence of CVD compared with those in the lowest quintile, and the area under the ROC curve further confirmed that BRI accurately reflects cardiometabolic risk factors ^[32]. Coexistence of sarcopenia and elevated BRI has also been associated with increased CVD risk ^[33]. Our study extends this evidence by establishing a prospective longitudinal link within the framework of CKM syndrome, highlighting its potential application in this high-risk population. The biological plausibility of this association is supported by the well-established role of visceral adipose tissue (VAT) as an active endocrine organ. BRI, as a surrogate marker of VAT, reflects ectopic fat burden that drives cardiometabolic risk^[7]. Excess VAT serves as a major source of free fatty acids, proinflammatory cytokines (e.g., TNF- α , IL-6), and adipokines (e.g., reduced adiponectin and increased resistin), all of which contribute to chronic low-grade inflammation and insulin resistance ^[34–36]. These processes collectively lead to endothelial dysfunction, promote the formation and instability of atherosclerotic plaques, and directly impair cardiac and renal structure and function ^[37–39]. This multisystem pathophysiological mechanism reflects the core of CKM syndrome, in which dysregulated metabolic pathways damage both the cardiovascular and renal systems. Our mediation analysis provides direct empirical support for this mechanistic model. The findings indicated that hypertension and dyslipidemia exerted significant mediating effects, suggesting that the adverse impact of elevated BRI is partly transmitted through increased blood pressure and abnormal lipid metabolism. In contrast, the mediating roles of diabetes and metabolic syndrome were markedly attenuated after full adjustment, implying that their effects may be largely masked by the stronger mediation pathways of hypertension and dyslipidemia.

Our RCS analysis demonstrated a significant dose–response relationship between BRI levels and CVD risk. Specifically, within a reasonable BRI range (below the median), CVD risk remained relatively stable or slightly decreased. However, above this threshold (greater than the median), BRI was positively and strongly associated with increased CVD risk. This highlights that the risk is not limited to individuals with overt obesity; even modest elevations in BRI—commonly corresponding to CKM stages 1–2—are associated with substantially higher risk. Consistent with our findings, Kong et al. reported a significant linear dose–response relationship between BRI and incident CVD in non-diabetic populations, whereas

no such association was observed in individuals with diabetes, suggesting that hyperglycemia and insulin resistance may interfere with this relationship ^[40]. In our subgroup analyses stratified by CKM stage (**Figure S3**), we found that the linear association between BRI and incident CVD was significant in individuals with CKM stages 2–3 but not in those with CKM stages 0–1. This indicates that as CKM syndrome progresses, the predictive value of BRI for cardiovascular risk may become more pronounced. These findings align with our ROC curve analysis, further supporting the potential clinical utility of BRI in cardiovascular risk management for this population.

The results of the stratified analyses further highlight the complexity of the relationship between BRI levels and CVD risk, indicating that this association is substantially modified by demographic and socioeconomic factors. First, we observed a significant effect modification by sex (P for interaction = 0.026), with men exhibiting a markedly greater increase in risk compared with women. This finding may reflect sex-specific differences in body fat distribution and the distinct regulatory roles of sex hormones in cardiometabolic risk ^[41]. Men are more likely to present with central obesity and greater visceral fat accumulation, whereas premenopausal women typically demonstrate a more peripheral fat distribution pattern ^[41, 42]. The stronger association observed among individuals with higher educational attainment and those residing in urban areas may reflect the health-risk transition associated with socioeconomic development. In these groups, elevated BRI may be linked to more metabolically detrimental obesity phenotypes driven by sedentary occupations, dietary patterns, and reduced physical activity ^[43]. In contrast, obesity observed in rural populations or among individuals with lower educational levels may be less influenced by such behavioral factors, potentially attenuating the strength of the association. These findings carry important public health implications. They suggest that the clinical application of BRI and risk assessment strategies based on BRI may need to be tailored according to sex, educational background, and living environment. Future research should further explore the sociobehavioral and biological mechanisms underlying these effect modifications to provide a stronger theoretical basis for precision prevention of cardiovascular disease.

The strengths of this study lie in the use of a nationally representative cohort, with adjustment for a wide range of potential confounding variables, as well as the inclusion of mediation and subgroup analyses to elucidate the association between BRI levels and future CVD risk among individuals with CKM syndrome stages 0–3. These findings suggest that BRI may serve as a cost-effective biomarker for identifying individuals at increased risk of CVD within this population. Nevertheless, several limitations should be acknowledged. First, given the observational design of the study, we cannot establish a causal relationship between BRI levels and cardiovascular risk. Second, although extensive adjustment for major CVD risk factors was undertaken, residual or unmeasured confounding cannot be excluded, such as variations in dietary patterns and environmental exposures across regions. Third, due to the absence of medical records in the CHARLS dataset, information on chronic disease history (including heart disease, stroke, hypertension, and diabetes) was primarily based on self-reported data, which may introduce recall bias and disease misclassification, thereby affecting the accuracy of risk assessment.

Finally, as the study population was limited to middle-aged and older Chinese adults with CKM stages 0–3, the generalizability of the findings to other ethnic groups may be restricted.

5. Conclusion

In this nationally representative cohort of middle-aged and older Chinese adults, elevated BRI was significantly associated with an increased risk of incident cardiovascular disease across CKM syndrome stages 0–3. Mediation analyses suggested that this relationship was partially explained by metabolic abnormalities, including hypertension, diabetes, dyslipidemia, and metabolic syndrome. These findings indicate that BRI, as a simple anthropometric index, may be applied in both clinical and public health settings to facilitate early identification of high-risk individuals with CKM—particularly those at stages 2–3, thereby providing evidence to support strategies for primary prevention.

Abbreviations

CVD: Cardiovascular disease

BMI: Body mass index

WC: Waist circumference

ABSI: A Body Shape Index

BRI: Body roundness index

CKM: Cardiovascular-kidney-metabolic

CKD: Chronic kidney disease

CHARLS: China Health and Retirement Longitudinal Study

eGFR: Estimated glomerular filtration rate

TC: Total cholesterol

TG: Triglycerides

HDL-C: High-density lipoprotein cholesterol

LDL-C: Low-density lipoprotein cholesterol

HbA1c: Hemoglobin A1c

hs-CRP: High-sensitivity C-reactive protein

GMS: Glucose metabolism status

NGR: Normal glucose regulation

ROC: Receiver operating characteristic

Declarations

CRedit authorship contribution statement

P.-A.L. and X.W. designed the research framework, established the research methodology, organized raw data, and drafted the original manuscript. F.X. performed formal statistical analysis and sorted experimental data. S.-F.L. conducted experimental investigation and collected experimental resources. S.C. carried out result validation and data sorting. L.Y. and S.-W.Z. managed the research project, revised and edited the manuscript, and obtained financial support for this study. All authors read and approved the final version of the manuscript.

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Data availability

The datasets generated during and/or analysed during the current study are available in the CHARLS repository (<http://charls.pku.edu.cn/>).

Generative AI statement

The author(s) declare that no Generative AI was used in the creation of this manuscript.

Declaration of competing interest

The authors declare that they have no conflict financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethics declaration

The CHARLS protocol was approved by the Ethical Review Committee of Peking University (IRB: 00001052–11015). All participants provided written informed consent prior to enrollment.

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Figures

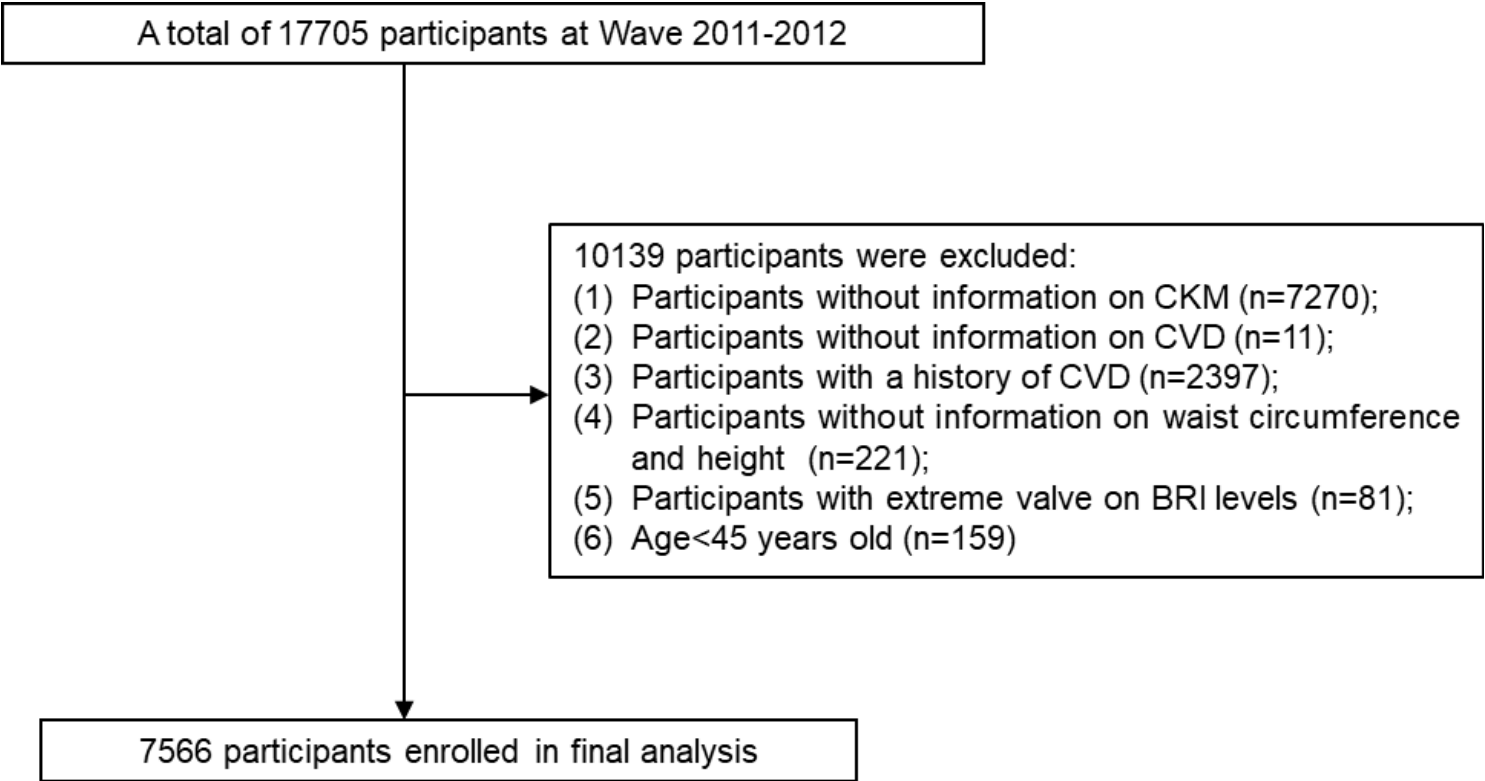


Figure 1

Flowchart of the study population.

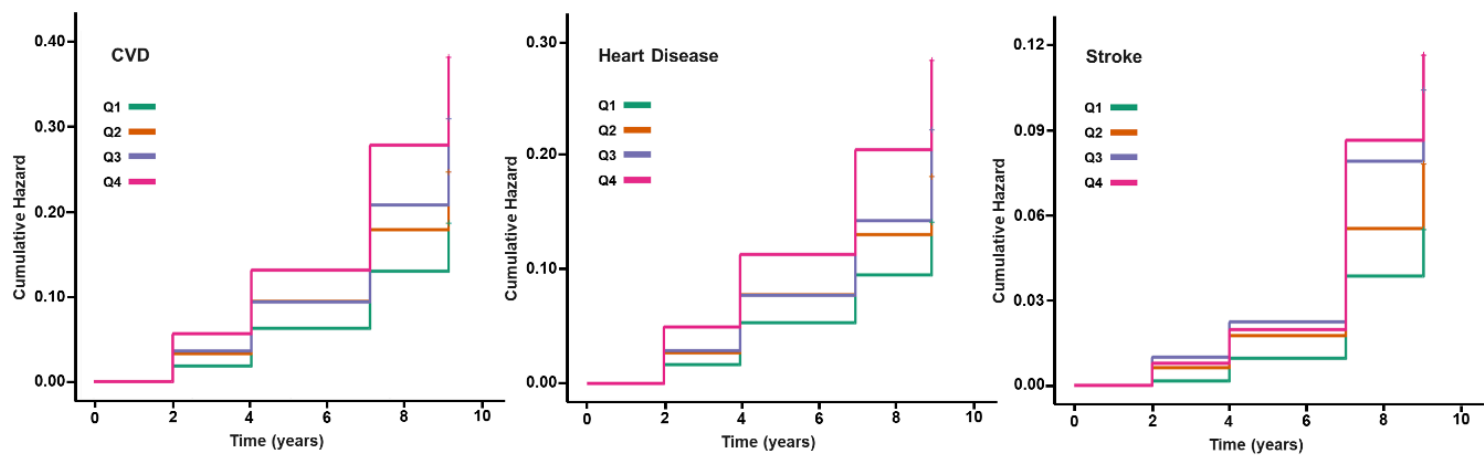


Figure 2

Kaplan–Meier cumulative incidence curves for CVD by BRI quartiles.

Abbreviations: CVD, cardiovascular disease; BRI, body roundness index.

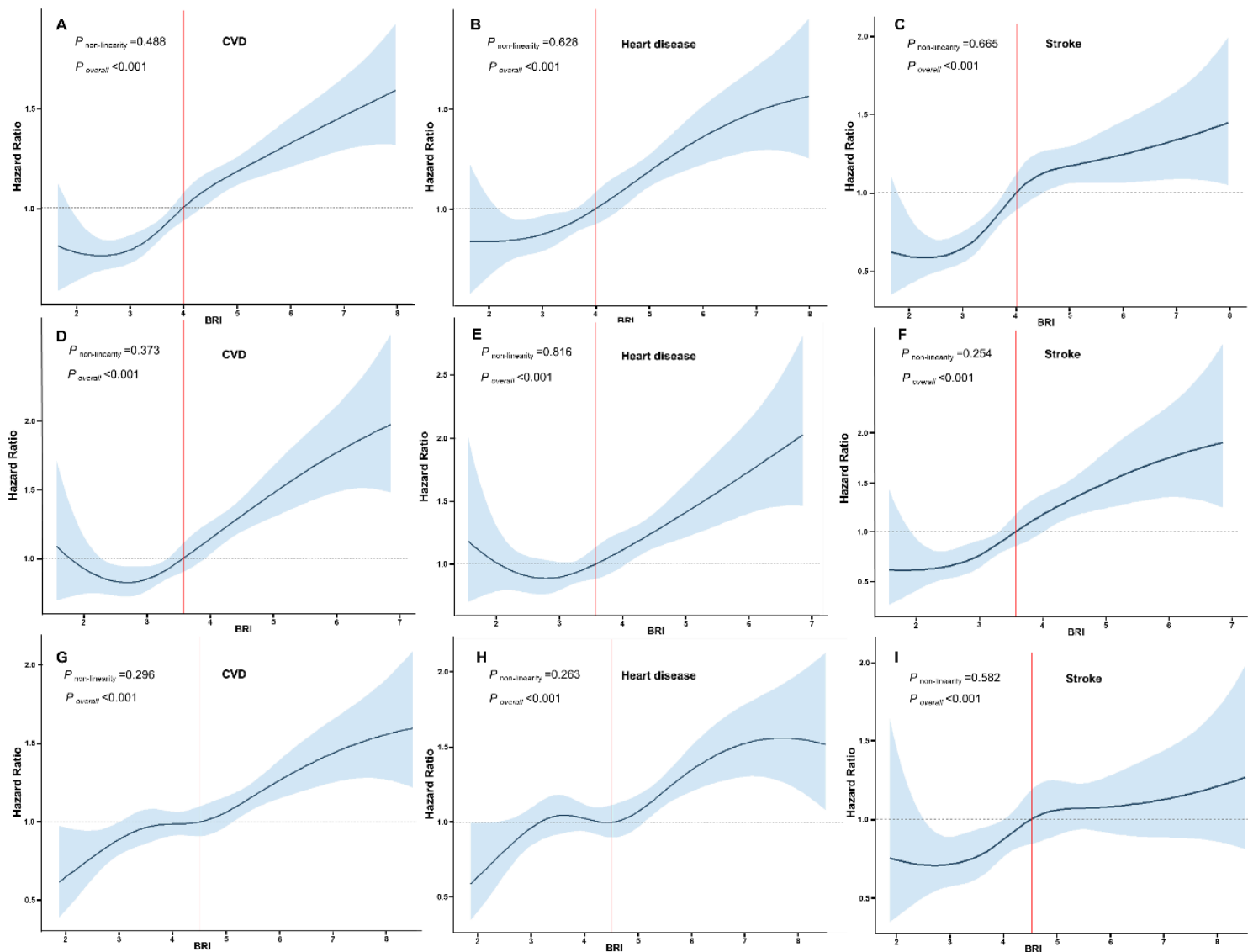


Figure 3

Restricted cubic spline analysis of the association between BRI and CVD.

Panels A–C depict the associations between BRI and CVD (A), heart disease (B), and stroke (C) among individuals with CKM syndrome stages 0–3. Panels D–F present results for men, while panels G–I present results for women. The red vertical line indicates the median BRI. All models were adjusted for age, marital status, educational attainment, residence, smoking, alcohol consumption, household fuel use, LDL-c, HbA1c, CRP, hypertension, and diabetes.

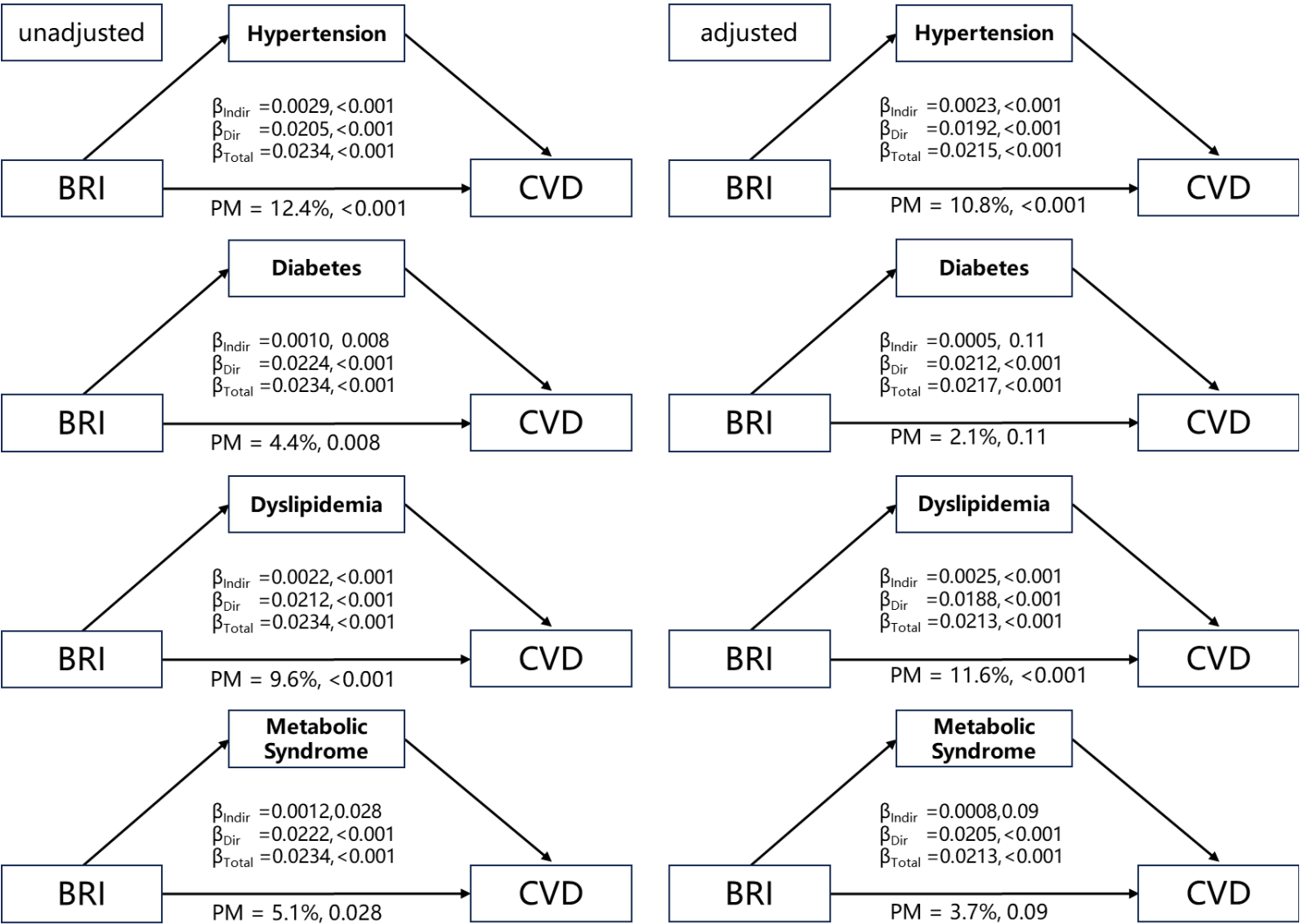


Figure 4

Mediation analysis of the association between Body Roundness Index and CVD.

The model was adjusted for age, marital status, educational level, residence, smoking, drinking, household fuel use, LDL-c, HbA1c, hs-CRP, hypertension, and diabetes.

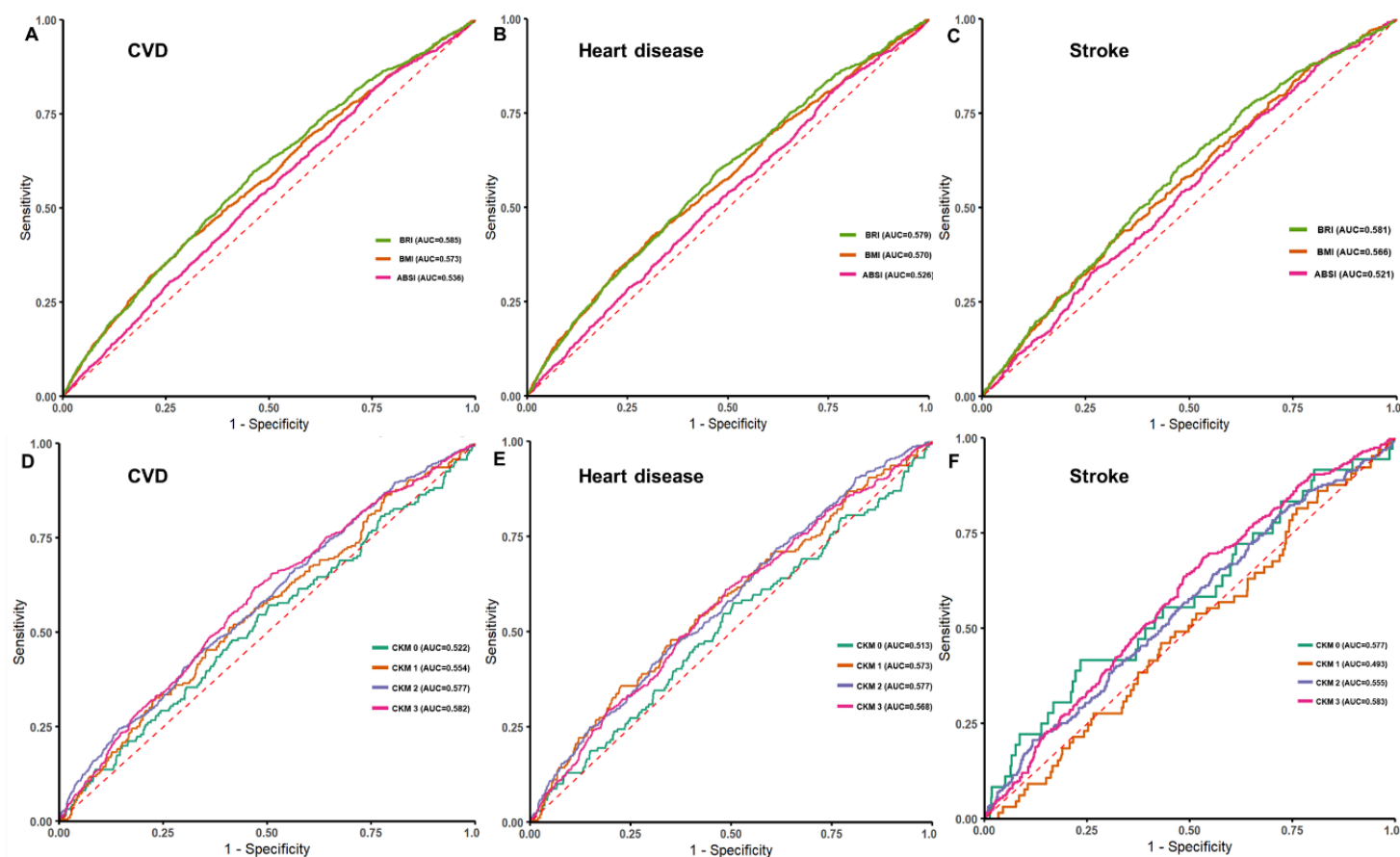


Figure 5

Predictive performance of BRI, BMI, and ABSI for CVD, heart disease, and stroke, and the discriminatory ability of BRI across different CKM stages.

Panels A–C: ROC curves illustrating the predictive performance of BRI, BMI, and ABSI for CVD (A), heart disease (B), and stroke (C). Panels D–F: ROC curves showing the discriminatory ability of BRI for predicting CVD (D), heart disease (E), and stroke (F) across CKM stages 0–3.

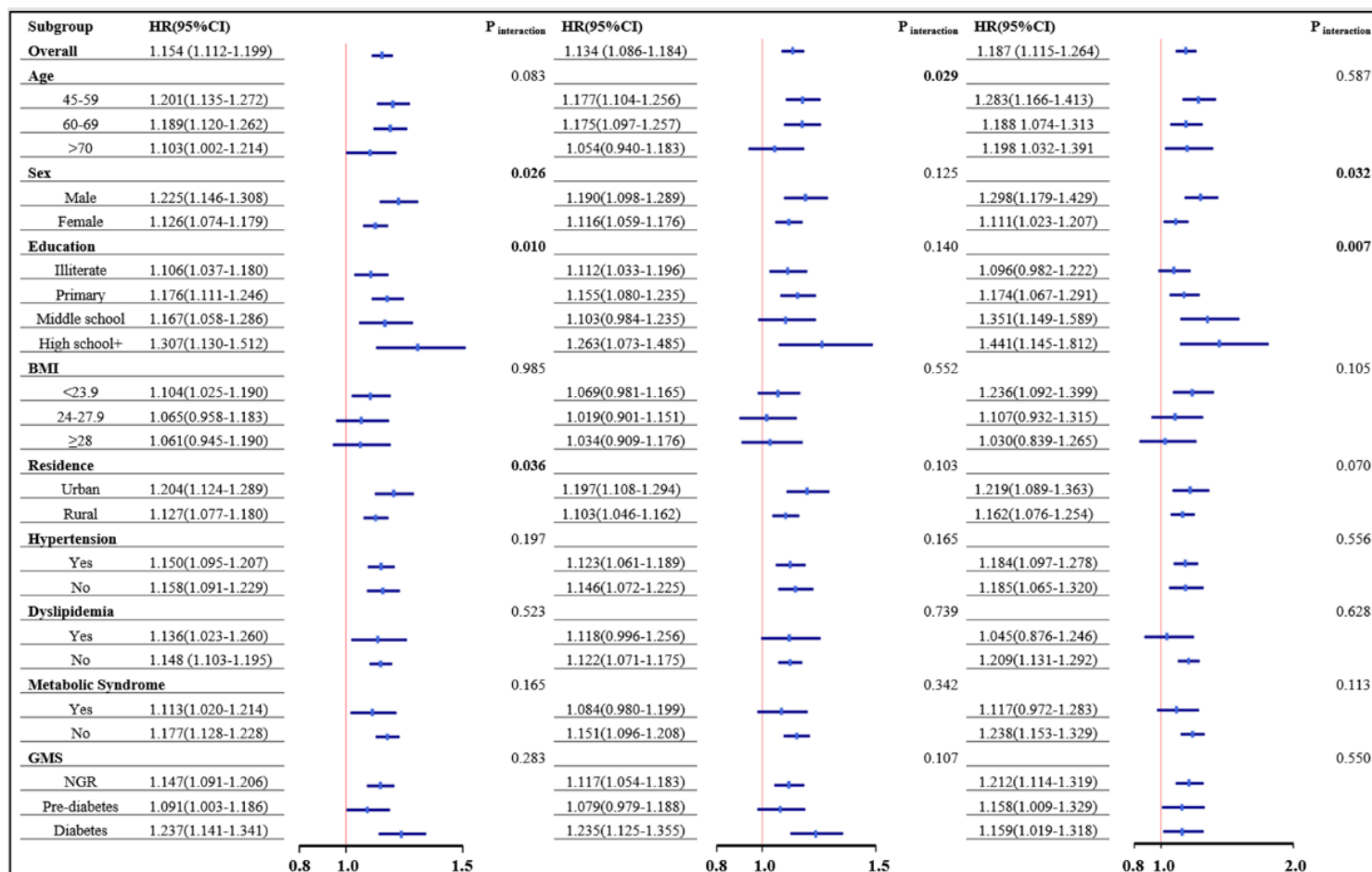


Figure 6

Subgroup analyses of the association between BRI and CVD risk across different demographic and clinical characteristics.

HR (95% CIs) of CVD risk per 1 unit increase in BRI. The model was adjusted for age, marital status, educational level, residence, smoking, drinking, household fuel use, LDL-c, HbA1c, hs-CRP, hypertension, and diabetes.

Supplementary Files

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