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# Body roundness index and the risk of hypertension: a prospective cohort study in Southwest China



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# **Abstract**

**Background** Body roundness index (BRI) is an anthropometric measure related to obesity, combining waist circumference (WC) and height to more accurately reflect body fat. This study aims to investigate the relationship between BRI and the risk of hypertension using data from a prospective cohort study in Southwest China.

**Methods** Data for the study were derived from Guizhou Population Health Cohort Study (GPHCS), established in 2010. A total of 9,280 participants (aged 18 to 95 years, mean 41.53±14.15 years) from 48 townships across 12 districts/counties were surveyed at baseline through multistage stratified random cluster sampling. Cox proportional risk models were employed to analyze the association between BRI and the risk of hypertension, estimating hazard ratios (HRs) and 95% confidence intervals (CIs) after adjusting for confounding factors. The relationship between BRI and the onset time of hypertension was analyzed using the time failure acceleration model.

**Results** Over a median follow-up period of 6.64 years, 1,157 participants were diagnosed with hypertension. After adjusting for confounding variables, each unit increase in BRI was associated with a 17% increase in hypertension risk (HR=1.17, 95% CI: 1.11, 1.24, *P* for trend<0.001). Compared to participants in the first quartile (Q1) of BRI, the risk of hypertension for those in the third quartile (Q3) and fourth quartile (Q4) was 1.31 (95% CI: 1.10, 1.56) and 1.53 (95% CI: 1.28, 1.84), respectively. Each unit increase in BRI advanced the onset of hypertension by 0.26 years (95% CI: 0.16, 0.35).

**Conclusion** This study indicates that BRI has a positive association with hypertension and can accelerate the onset of hypertension in the Chinese population. It is suggested that reducing BRI by controlling abdominal fat may be one of the effective measure to prevent hypertension.

**Keywords** Hypertension, Body roundness index (BRI), Cohort study, Hazard ratio

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# **Introduction**

Hypertension is one of the most significant modifiable risk factors for cardiovascular disease and premature mortality globally [[1\]](#page-8-0). It is projected that by 2025, hypertension will affect more than 1.5 billion individuals globally, with an anticipated rise in blood pressurerelated deaths to 10.4 million annually, a 49% increase [\[2](#page-8-1)]. Therefore, it is crucial to identify and reduce risk factors for hypertension. Previous studies have demonstrated a strong association between overweight, obesity, and hypertension [\[3](#page-8-2)], particularly visceral obesity [[4\]](#page-8-3). Studies have shown that a large proportion of variation in blood pressure was explained by the relationship between lean body mass (muscle) and adipose tissue [\[5\]](#page-8-4). Body round index (BRI), proposed by Thomas et al. in 2013, is a novel anthropometric index related to obesity [\[6](#page-8-5)]. It combines waist circumference (WC) and height to describe a person's body shape and is more reflective of the proportion of body fat and visceral fat compared to traditional indicators like Body Mass Index (BMI), WC, and hip circumference (HC) [[7](#page-8-6)[–10](#page-8-7)]. BRI has demonstrated strong predictive ability for fat distribution ( $\mathbb{R}^2$  for predicting male body fat percentage is 0.78;  $\mathbb{R}^2$  for predicting male visceral fat percentage is 0.56) [[11](#page-8-8)], and has also been used to predict diabetes, hypertension, metabolic syndrome, and cardiovascular diseases [[12](#page-8-9)[–14](#page-8-10)]. Previous studies have indicated that visceral obesity is closely associated with the risk of hypertension, and BRI is an effective indicator of visceral obesity [\[6](#page-8-5), [15](#page-9-0)]. Nevertheless, the related research on BRI and hypertension is mostly limited to cross-sectional research, which can not provide evidence for causal inference [[16](#page-9-1)[–18](#page-9-2)]. To date, only one longitudinal study in China has evaluated the relationship between BRI and the risk of hypertension [\[19](#page-9-3)]. Therefore, this study used a population cohort in Southwest China to analyze the differences in hypertension at various BRI levels aiming to clarify the relationship between BRI, a new obesity index, and hypertension risk. This study aimed to explore the relationship between BRI and hypertension, providing a scientific basis for hypertension prevention and control.

# **Methods**

# **Study population**

We used data from the Guizhou Population Health Cohort Study (GPHCS), a large population database designed to investigate the incidence of risk factors and chronic diseases. A multi-stage cluster random sample approach was adopted. The baseline survey between 2010 and 2012 included 9,280 adults who were permanent inhabitants in 12 districts (counties) of Guizhou Province. Participants were followed-up once between from 2016 to 2020 to monitor their main chronic diseases and life status, with the response rate of 88% (loss in follow-up *N*=1,117). The follow-up methods were consistent with the baseline survey methods. Details of the cohort and sampling process have been previously published [\[20](#page-9-4)]. The inclusion criteria for this study were: (1) participants aged 18 years or older; (2) participants who were residents of the research area and had no intention of leaving; (3) participants without a hypertension diagnosis at baseline; (4) participants who completed the questionnaire and blood sample collection; (5) participants who provided written consent before data collection. Then, we excluded participants who met the following criteria: (1) died or lost to follow-up; (2) had a hypertension diagnosis at baseline; (3) unclear blood pressure diagnosis information; (4) had missing baseline BRI variables. A total of 5,230 participants were included in the analysis ( Fig. [1](#page-2-0)).

#### **Data collection and definitions**

To gather information on each participant's basic demographics (age, residence, gender, ethnicity, marital status, education level, and occupation), lifestyle (smoking, drinking, and physical activity), history of chronic diseases (hypertension, dyslipidemia, T2DM, and cardiovascular diseases), and dietary factors, trained investigators used a structured one-on-one face-to-face questionnaire. The questionnaires were designed by the Chinese Center for Disease Control and Prevention [[21\]](#page-9-5) and applied in China's chronic disease surveillance (2010). Measurements of blood pressure, waist circumference (WC), height, weight, and other anthropometric data were also collected. Venous blood samples were collected after overnight fasting for at least 8 h, and a fully automated biochemical analyser (Olympus 400 analyzer, Beckman Coulter, CA, USA) was used to measure total cholesterol (TC), triglycerides (TG), high-density lipoprotein-Cholesterol (HDL-C), low-density lipoprotein-Cholesterol (LDL-C), fasting blood glucose (FBG), 2-hour postprandial blood glucose (PBG 2 h). The aforementioned data collection methods were identical for both baseline and follow-up.

Smoking was defined as adults who smoked at the time of the survey, including daily smokers and occasional smokers. Harmful drinking was defined as consuming≥61 ml/d for men or ≥41 ml/d for women [\[22](#page-9-6)]. Diabetes was defined if any of the following conditions were met: (1) diagnosed as diabetic or on hypoglycemic therapy by a physician at the township level or above; (2) FBG level≥7.0 mmol/L or PBG 2 h level≥11.1 mmol/L or glycated hemoglobin level≥6.5%, according to the diagnostic criteria for type 2 diabetes mellitus in the Chinese Guidelines for the Prevention and Control of Type 2 Diabetes Mellitus (2020 Edition); meeting one of the above criteria was sufficient [\[23](#page-9-7)]. Dyslipidemia was determined if participants met any of the following criteria  $[24]$  $[24]$ : 1) diagnosed as dyslipidemia by doctors at the township

<span id="page-2-0"></span>

**Fig. 1** The flow chart of the study. GPHCS, Guizhou population health cohort study

level or above, or took lipid-lowering drugs; 2) high TC: TC≥6.22mmol/L; (3) high TG: TG≥2.26mmol/L; (4) low HDL-C: HDL-C<1.04mmol/L; (5) high LDL-C:  $\geq 4.14$ mmol/L.

#### **Blood pressure measurements**

The trained staff performed blood pressure measurements following the standard protocol, using appropriately sized cuffs for each survey. Before the measurement, all participants need to take a 10-minute seated rest and were advised not to drink tea, coffee, etc. Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) were measured using an Omron electronic sphygmomanometer (HBP-1300, OMRON, Liaoning, China), with at least 1 min between recordings. The following methods were used to evaluate blood pressure: (1) the average value of three measurements should be used as the final reading if the difference between the three measurements is less than or equal to 10 mmHg; (2) if the difference between the three measurements is greater than or equal to 10 mmHg, the average value of two similar measurements should be used as the final reading; and (3) if only one measurement was obtained, it should be used as the final reading.

#### **Study outcome**

Hypertension was defined as meeting one of the following criteria:1) self-reported diagnosis of high blood pressure by a physician or took hypertension medication;2) systolic blood pressure≥140mmHg (18.6 kPa) and/or

diastolic blood pressure≥90mmHg (12.0 kPa) [\[25\]](#page-9-9). The time of hypertension diagnosis was considered the final event. The final survey date was used to determine the follow-up time for those who did not develop hypertension during the follow-up.

#### **Anthropometric measurements**

Before the anthropometric measurements, all participants were required to remove heavy clothes and shoes, and the measurements were taken by trained professionals in a private and comfortable room. Height and WC were measured using the calibrated equipment according to standard procedure. A unified height meter (accuracy=0.1 cm) was used to measure standing height without shoes. An electronic scale with a 0.1 kg precision was used to measure weight. WC was measured at the midpoint between the iliac crest and the lowest rib using a waist ruler with an accuracy of 0.1 cm. The BRI was calculated based on WC and height [\[19](#page-9-3)].

$$
BRI = 364.2 - 365.5 * \sqrt{1 - \left[\frac{WC/2\pi}{0.5 * Height}\right]^2}
$$

# **Statistical analysis**

Categorical variables were summarised using frequencies and percentages. The continuous variables that were not normally distributed were represented as M (P25, P75). To compare baseline characteristics, the Wilcoxon

rank-sum or Chi-square test was employed. The incidence density of the population in different BRI groups was computed, as well as the person-years from the baseline survey to the onset of hypertension or the end of follow-up.

We divided BRI into four groups according to quartiles: Q1<2.38; 2.39≤Q2<3.01; 3.02≤Q3<3.83; 3.84≤Q4. with Q1 as the reference level. The association between BRI (continuous variable, or grouped based on quartiles) and the risk of hypertension was evaluated using the Cox proportional hazards model. The hazard ratio (HR), adjusted HR (aHR), and 95% confidence interval (CI) were estimated by using three Cox proportional hazard regression models. We progressively adjusted the covariates and applied three analysis models. Model 1 was without any adjustment for covariates. In Model 2, adjusted for age  $($ <45years,  $\geq$  45years), gender (male, female), ethnicity (Han Chinese, minorities), residence (urban, rural), sleep duration  $(7-9 h/d, > 9 or < 7 h/d)$ , oil intake (25–30 g/d, < 25 g/d or >30 g/d), salt intake (<5 g/d, ≥ 5 g/d), vegetable intake (<300 g/d,  $\geq$ 300 g/d), fruit intake  $\left($  < 200 g/d,  $\geq$  200 g/d), smoking (No, Yes), harmful drinking (No, Yes), sedentary time (≥4 h/d, < 4 h/d), educational level (≥9 years, < 9 years). Model 3 included all variables in Model 2 plus family history of hypertension (yes, no, unclear), diabetes (yes, no), triglycerides(TG), total cholesterol (TC), high-density lipoprotein(HDL-C), low-density lipoprotein(LDL-C). Model 3 was considered the main model. The dose-response relationship between baseline BRI levels and hypertension risk was illustrated using a restricted cubic-like spline curve. The Akek Information Standard (AIC) was used to choose among various survival distributions (such as Weibull, exponential, logistic and Gaussian), and the logistic distribution was selected for the accelerated failure time (AFT) model to evaluate the influence of BRI on the onset time of hypertension. We conducted stratified analyses by individuallevel characteristics and residence to examine susceptible subgroups. We also adjusted other variables and interactions to identify differences between subgroups. A series of sensitivity analysis were conducted to ensure the robustness of the relationship between BRI and incident hypertension. Multivariate Cox proportional risk models were repeated to exclude participants who had hypertension in the 2 years before follow-up and those diagnosed with baseline diabetes. The Statistical Package for the Social Sciences (Version 26.0; IBM Corporation, Armonk, NY, USA) and R software (Version 4.2.2; R Foundation for Statistical Computing, Vienna, Austria) were used to perform statistical analyses. All tests were conducted on two-sided, and a *P*value less than or equal to 0.05 was considered statistically significant.

# **Results**

# **Baseline characteristics**

The study included 5,230 participants, comprising 2,394 (45.8%) male participants and 2,836 (54.2%) female participants. The age ranged of the participants was 18 to 95 years (mean age:  $41.53 \pm 14.15$  years). The total personyears of follow-up were 36,950.24 years, with a mean follow-up time of 6.64 years and the incidence density of 1,157 newly diagnosed hypertension cases at 31.31 per 1000 person-years (PYs). Differences in residence, gender, age, educational level, baseline diabetes, triglyceride (TG), total cholesterol (TC), high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), oil intake, smoking, vegetable intake, fruit intake, sleep duration, and sedentary time were observed among BRI quartiles (*P*<0.05; Table [1\)](#page-4-0). There were differences in residence, gender, age, educational level, family history of hypertension, TG, smoking, harmful drinking, fruit intake, and BRI between participants with hypertension and those without hypertension ( $P < 0.001$ ; Table S1).

#### **Associations of BRI with risk of hypertension**

The Cox proportional hazards regression model showed the association between BRI and incident hypertension. BRI had a positive association with hypertension incidents. The hazard of hypertension incidents increased by 22% for every unit increase in BRI (Table [2\)](#page-5-0): unadjusted HR=1.22 (95%CI: 1.16, 1.28). The association weakened slightly when adjusting for demographic, lifestyle and socio-economic factors, and persisted (unchanged) when further adjusting for family history of hypertension, diabetes, and lipids: adjusted HR=1.17 (95%CI: 1.11, 1.24). Additionally, in the fully adjusted Model 3, the risk of hypertension in Q3 and Q4 participants was 1.31 times (HR=1.31, 95%CI: 1.10, 1.56) and 1.53 times (HR=1.53, 95%CI: 1.28, 1.84) higher than that of the Q1 participants, respectively. The risk of hypertension increased with higher BRI levels (*Pfor trend* < 0.001, Table [2](#page-5-0)). Between BRI and the risk of hypertension, Fig. [2](#page-5-1) consistently illustrated a positive dose-response association (*P*-overall<0.001, *P*for non-linearity=0.634).

## **Subgroup analysis**

To explore the effect of other variables on the relationship between BRI and the incidence of hypertension, a stratified analysis was conducted. The results of the subgroup analysis (Fig. [3](#page-6-0)) showed an interaction between ethnicity and BRI (*P*for interaction<0.05). Han Chinese participants had a higher risk of new hypertension (HR=1.17,95%CI: 1.10, 1.26) for each unit increase in BRI, while minority participants had an HR of 1.15 (95%CI: 1.05, 1.27). Residence, age, and gender did not alter the relationship between BRI and hypertension (*P*for interaction>0.05).

<span id="page-4-0"></span>Table 1 Base line characteristics of the participants according to the quartiles of BRI

<b>BRI</b>	$Q1 (\leq 2.38)$	$Q2(2.39 - 3.01)$	$Q3(3.02 - 3.83)$	Q4(≥3.84)	Pvalue
Participants	1,422	1,425	1,340	1,043	
Residence[N(%)]					$0.011*$
Urban	493(34.7)	461(32.4)	471(35.2)	405(38.8)	
Rural	929(65.3)	964(67.7)	869(64.9)	638(61.2)	
Gender[N(%)]					$< 0.001*$
Male	750(52.7)	662(46.5)	572(42.7)	410(39.3)	
Female	672(47.3)	763(53.5)	768(57.3)	633(60.7)	
Ethnicity[N(%)]					0.149
Minorities	622(43.7)	617(43.3)	546(40.8)	417(40.0)	
Han Chinese	800(56.3)	808(56.7)	794(59.3)	626(60.0)	
Age, years[N(%)]					$< 0.001*$
$<$ 45	1,009(71.0)	881(61.8)	801(59.8)	524(50.2)	
$\geq 45$	413(29.0)	544(38.2)	539(40.2)	519(49.8)	
Educational level, years[N(%)]					$< 0.001*$
< 9	635(44.7)	820(57.5)	720(53.7)	605(58.0)	
$\geq 9$	787(55.3)	605(42.5)	620(46.3)	438(42.0)	
Oil intake(g/d) <sup>a</sup> [N(%)]					$0.040*$
$25 - 30$	210(14.9)	183(13.0)	190(14.3)	115(11.1)	
$< 25$ or $> 30$	1,199(85.1)	1,228(87.0)	1,141(85.7)	917(88.9)	
Salt intake(g/d) <sup>a</sup> [N(%)]					0.470
< 5	240(16.9)	255(17.9)	256(19.1)	181(17.4)	
$\geq 5$	1,182(83.1)	1,170(82.1)	1,084(80.9)	862(82.7)	
Smoking[N(%)]					$< 0.001*$
No	954(67.1)	1,047(73.5)	1,023(76.3)	807(77.4)	
Yes	468(32.9)	378(26.5)	317(23.7)	236(22.6)	
Harmful Drinking[N(%)]					0.788
No	1,391(97.8)	1,395(97.9)	1,308(97.6)	1,015(97.3)	
Yes	31(2.2)	30(2.1)	32(2.4)	28(2.7)	
Vegetable intake(g/d) <sup>a</sup> [N(%)]					$0.004*$
< 300	394(28.1)	424(30.4)	429(32.9)	351(34.5)	
$\geq 300$	1,006(71.9)	971(69.6)	874(67.1)	666(65.5)	
Fruit intake $(q/d)^{a}$ [N $(\%)$ ]					0.148
< 200	1,319(93.8)	1,325(94.0)	1,230(93.1)	947(91.9)	
$\geq 200$	87(6.2)	84(6.0)	91(6.9)	84(8.2)	
Sleep time(h/d)[N(%)]					0.050
$< 7$ or $> 9$	283(19.9)	286(20.1)	301(22.5)	248(23.8)	
$7 - 9$	1,139(80.1)	1,139(79.9)	1,039(77.5)	795(76.2)	
Sedentary time (h/d)[N(%)]					$\leq 0.001*$
$<$ 4	577(40.6)	674(47.3)	639(47.7)	499(47.8)	
$\geq 4$	845(59.4)	751(52.7)	701(52.3)	544(52.2)	
Family history of hypertension[N(%)]					0.455
No	912(64.1)	890(62.5)	851(63.5)	678(65.0)	
Yes	131(9.2)	144(10.1)	143(10.7)	114(10.9)	
Unclear	379(26.7)	391(27.4)	346(25.8)	251(24.1)	
History of diabetes <sup>a</sup> [N(%)]					$< 0.001*$
No	1,355(95.8)	1,345(94.9)	1,262(94.6)	928(89.2)	
Yes	60(4.2)	73(5.2)	72(5.4)	112(10.8)	
TC(mmol/L) <sup>a</sup>	$4.29(3.66 - 5.18)$	4.62(4.00-5.38)	4.87(4.00-5.64)	$5(4.00 - 5.83)$	$< 0.001*$
TG(mmol/L) <sup>a</sup>	$1.00(0.92 - 1.51)$	$1.04(0.95 - 1.77)$	$1.31(1.00 - 2.00)$	$1.88(1.00 - 2.67)$	$< 0.001*$
HDL-C(mmol/L) <sup>a</sup>	$1.42(1.00-1.84)$	1.43(1.00-1.82)	$1.4(1.00-1.75)$	$1.32(1.00-1.67)$	$< 0.001*$
$LDL-C(mmol/L)a$	$2.13(1.78 - 3.01)$	2.36(2.00-3.11)	2.66(2.00-3.41)	3.00(2.00-3.72)	$< 0.001*$

N: number; BRI: body roundness index; TC: total cholesterol; TG: triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol Note: Continuous variables are presented as mean±standard deviation (normally distributed) or median with 25–75th percentile (not normally distributed); categorical variables are expressed as percentage; <sup>a</sup> missing values; \* P value<0.05

<span id="page-5-0"></span>



BRI: body roundness index; n: number; HR: hazard ratio; CI: confidence interval;

Model 1: unadjusted;

Model 2: adjusted age (<45, ≥45), gender, ethnicity, residence, sleep duration, oil intake, salt intake, vegetable intake, fruit intake, smoking, harmful drinking, sedentary time and educational level;

<span id="page-5-1"></span>Model 3: Model 2 added family history of hypertension, diabetes, total cholesterol(TC), triglycerides(TG), high-density lipoprotein(HDL-C), low-density lipoprotein(LDL-C)



**Fig. 2** The dose-response relationship between BRI and the risk of hypertension BRI: body roundness index; HR: hazard ratio; CI: confidence interval;

# **Association between BRI and the onset time of hypertension**

The association between BRI and the onset time of hypertension was analyzed using an AFT model with a logistic distribution, chosen according to the Akaike information criterion (Table S2). Each unit increase in BRI advanced the onset of hypertension by 0.26 years (95% CI: 0.16, 0.35; Table [3\)](#page-7-0).

#### **Sensitivity analysis**

A series of sensitivity analyses were conducted by excluding participants who were followed up for less than 2 years and patients with diabetes at baseline. The results did not differ substantially from those of the primary analyses (Figure S1).

# **Disscussion**

We used data from the GPHCS to demonstrate a positive association between BRI and the risk of hypertension, as indicated by the dose-response curve. Additionally, increased BRI accelerated the onset of hypertension. Furthermore, among Han Chinese, this association was more pronounced.

#### **Comparison with other studies**

Traditional anthropometricindices (AHIs) such as body mass index (BMI), waist circumference (WC), and

<span id="page-6-0"></span>

HR (95% CI)

**Fig. 3** Stratified analysis of BRI and risk of hypertension

BRI: body roundness index; HR: hazard ratio; CI: confidence interval; Note: \* *P value*<0.05

waist-to-hip ratio (WHR) are commonly used to evaluate overweight and obesity. Despite the fact that these indicators have a significant association with hypertension, each of them has some disadvantages [[26,](#page-9-10) [27\]](#page-9-11). BMI cannot distinguish between excess fat and high muscle mass [[28\]](#page-9-12), and WHR is a measure of visceral fat, calculated by dividing WC by hip circumference (HC). However when BMI exceeds 35  $\text{kg/m}^2$ , WC is difficult to measure, and WHR is strongly influenced by gender, which may make

WHR inaccurate. Although the new anthropometric index BRI computation is more complicated than that of the conventional AHIs due to the inclusion of WC and Height, it is significant to note that it more accurately represents visceral adipose tissue and body fat [\[19\]](#page-9-3).

Several cross-sectional studies have shown that BRI levels is strongly associated with pre-hypertension in Mexican population (OR=2.08, 95% CI: 1.49, 2.91) [\[29](#page-9-13)]. Another study based on non-obese people showed that

<span id="page-7-0"></span>**Table 3** Association between body roundness index and the onset time of hypertension

	$\sim$			
<b>Covariates</b>	<b>Estimates</b>	<b>SE</b>	95%CI	Pvalue
<b>BRI</b>	$-0.26$	0.05	$(-0.35, -0.16)$	< 0.001
BRI (quartiles)				
Q1	1.00			
Q <sub>2</sub>	$-0.14$	0.14	$(-0.42, -0.14)$	0.312
Q <sub>3</sub>	$-0.41$	0.14	$(-0.69, -0.13)$	0.004
Q4	$-0.67$	0.15	$(-0.97, -0.37)$	< 0.001
----	$\sim$ $\sim$ $\sim$	$\sim$ $\sim$ $\sim$ $\sim$	.	

BRI: body roundness index; CI: confidence interval; SE: standard error;

the OR of hypertension in the fourth quartile was 2.76 (95% CI: 2.17 2.17,3.49) compared with the first quartile of BRI [[17\]](#page-9-14). Xiao et al.'s latest study demonstrates a positive association between BRI and pre-hypertension after adjusting for confounding factors [\[30](#page-9-15)]. Currently, only one longitudinal study has shown a positive association between BRI and the risk of hypertension (Q4 vs. Q1: HR=1.99, 95% CI: 1.82, 2.19) in China, with a greater risk of new-onset hypertension in participants under 40 years old (*P*for interaction<0.05). However, the above research may have some limitations (such as self-reported covariates, cross-sectional studies unable to provide evidence for causal inference, unadjusted family history data of hypertension, and unmeasured blood biochemical examination parameters, etc.) [[19\]](#page-9-3). In this longitudinal study, we found that BRI has a positive association with the risk of hypertension, which is consistent with previous similar studies, and suggests that excessive visceral fat tissue may be a risk factor for hypertension. Thus, controlling weight, particularly abdominal fat may comtribute to prevent hypertension. Nevertheless, additional research, such as randomized controlled trials and Mendel randomized analysis, is required to verify these results due to the limitations and characteristics of the observational study.

#### **The explanation for the stratified analyses results**

In the subgroup analysis, we further found the interaction between BRI and ethnicity, which is more pronounced in the Han Chinese. Other factors did not alter the relationship between BRI and hypertension. Similarly, a cross-sectional study of 45,853 American adults investigated the relationship between BRI and the incidence of hypertension, and found that non-Hispanic white people were more sensitive to BRI than non-Hispanic black people and other people  $[31]$  $[31]$ . To date, the ethnic differences between BRI and hypertension are uncertain. Some studies have shown that there are obvious differences between obesity and race or ethnicity, which may be due to differences in physical activity and eating behavior, as well as various potential social and cultural determinants. These factors may lead to changes in the role of the sympathetic nervous system, renal function, and microvascular level, thereby affecting the risk of hypertension [[32](#page-9-17)[–34](#page-9-18)]. Therefore, it is necessary to further study the mechanism involved in ethnic differences.

# **Potential biological mechanism**

There may not be a single mechanism linking BRI and the development of hypertension: (1) physical compression of the kidneys due to increased visceral, retroperitoneal, and renal sinus fat; (2) activation of the renin-angiotensin-aldosterone system (RAAS), including activation of mineralocorticoid receptor (MR), which is not aldosterone-dependent; (3) activation of the sympathetic nervous system (SNS), especially renal sympathetic nerve activity ( RSNA) increases [[35](#page-9-19)].

Since BRI reflects the accumulation of visceral fat, excessive accumulation of fat in adipose tissue and ectopic sites leads to impaired adipogenesis, dysregulation of adipokines, elevation of pro-atherosclerotic inflammatory factors, circulating free fatty acids, pro-inflammatory cytokines, increased oxidative stress and lipotoxicity, and hemodynamic overload, which leads to atherosclerosis and endothelial cell dysfunction, thereby contributing to hypertension and other disease development [[36](#page-9-20), [37\]](#page-9-21). Recent studies have demonstrated that abdominal obesity independently affects hypertension, serving as a surrogate indicator for the impact of abdominal subcutaneous and visceral fat on hypertension. Excess visceral fat leads to increased insulin resistance, affects endothelial cell metabolism, stimulates nitric oxide production, and further affects angiotensin production. This sequence of events reduces the ability of insulin-mediated diastole of vascular tissues, ultimately leading to increased blood pressure [\[38](#page-9-22)]. Additionally, increased systemic inflammation and activation of the sympathetic nervous system triggered by insulin resistance are important factors in the development of hypertension [\[39](#page-9-23)].

#### **Strengths and limitations**

Our research has several strengths. Firstly, we analyzed the association between BRI levels and hypertension though a long-term and population based prospective cohort study. Secondly, Sensitivity analyses made the results more robust and reliable. Our findings provided a reference basis for the early detection of hypertension and intervention in the population. However, there are some limitations to our study. Firstly, our results may not be applicable to other race or ethnic populations. Secondly, dietary information is self-reported by the population, which inevitably leads to measurement error and information bias. Thirdly, despite adjusting for a variety of confounders, there is always a risk of unmeasured or residual confounders in observational studies. This means that there may be unconsidered variables that could bias our findings.

# **Conclusion**

This study indicates that BRI has a positive association with hypertension and can accelerate the onset of hypertension in the Chinese population. It is suggested that reducing BRI by controlling abdominal fat may be one of the effective measure to prevent hypertension. However, further research is needed to confirm these findings and to explore the mechanisms underlying the association between BRI and hypertension.

#### **Abbreviations**



#### **Supplementary Information**

The online version contains supplementary material available at [https://doi.](https://doi.org/10.1186/s12889-024-20049-z) [org/10.1186/s12889-024-20049-z.](https://doi.org/10.1186/s12889-024-20049-z)

Supplementary Material 1

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#### **Author contributions**

QQZ completed data cleaning and analysis and wrote this initial manuscript. QYA, FYZ and TLZ participated in the data analysis, and collection of the literature. YYW undertook the statistical analysis and participated in manuscript preparation. QQZ and YYW interpreted the results and manufactured the tables and figures. TL revised the manuscript for important intellectual contents and languages. All authors read and approved the final manuscript.

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#### **Data availability**

Application for data sets generated during and/or analyzed during the current study may be considered by the corresponding author on reasonable request.

#### **Declarations**

#### **Ethics approval and consent to participate**

This study was approved by the Institutional Review Board of Guizhou Province Centre for Disease Control and Prevention (No. s2017-02), and written informed consent was signed by all subjects.

## **Consent for publication**

Not applicable.

#### **Competing interests**

The authors declare no competing interests.

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