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Abdominal fat accumulation increases the risk of high blood pressure: evidence of 47,037 participants from Chinese and US national population surveys

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Abstract

Aims This study aims to ascertain whether abdominal fat accumulation evaluated by waist circumference increases the risk of hypertension using the nationwide population.

Methods We enrolled 47,037 participants from the China Health and Nutrition Survey (CHNS), China Health and Retirement Longitudinal Study (CHARLS), and National Health, and Nutrition Examination Survey (NHANES). The adjusted logistic regression model was used to examine the relationship between waist circumference and prevalent hypertension. 9445 participants without baseline hypertension from the CHNS and CHARLS were followed up to investigate the association between waist circumference and onset hypertension. The association was evaluated using a Cox regression model and restricted cubic spline. Furthermore, Mendelian randomization was employed to explore causal inferences.

Results In the baseline survey, waist circumference demonstrated a notable correlation with hypertension, presenting an odds ratio (with 95% confidence intervals) of 1.34 (1.28 ~ 1.40). After a mean follow-up of 3.8 years for participants without baseline hypertension, 2,592 (27.5%) developed hypertension. In the pooled analysis, the Cox regression showed that every 10 cm increase in waist circumference was associated with 20% (95% CI: 13% ~ 27%) elevated risk of new-onset hypertension. Restricted cubic splines indicated a pronounced linear dose–response relationship. A subgroup analysis affirmed the persisting association between waist circumference and hypertension onset even in those with normal BMI. The Mendelian randomization method revealed a significant causative association between waist circumference and hypertension.

Conclusion Elevated waist circumference stands as an independent risk factor for hypertension, even in those with normal BMI. Our results provide evidence supporting the routine measure for waist circumference.

Keywords Waist circumference, Abdominal fat accumulation, Hypertension, Body mass index

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Introduction

Over the last three decades, the prevalence of hypertension has increased markedly [1, 2], accounting for an estimated ten million global deaths annually [3, 4]. As a modifiable metabolic disorder, obesity is a major contributor to hypertension and numerous other cardiovascular diseases. Currently, the Body Mass Index (BMI) is the predominant metric for evaluating obesity and related health risks. Nonetheless, BMI alone is incapable of distinguishing variations in regional body fat distribution. Intriguingly, there is evidence suggesting that a moderately increased BMI might decrease cardiovascular events and prolong lifespan—a phenomenon termed the "obesity paradox" [5, 6]. Therefore, concerns have been raised regarding the limitation of BMI as a measure of individual metabolic risk [7].

Growing evidence suggests that waist circumference provides a more accurate reflection of abdominal fat distribution, making it a superior indicator of abdominal obesity. A recent consensus statement recommended that waist circumference should be considered as a vital sign [8]. Importantly, to fully examine the association between waist circumference and cardiovascular risk, the role of BMI cannot be overlooked [9–11]. Recent cross-sectional studies have underscored a close relationship between waist circumference and the prevalence of hypertension [12–16]. However, more prospective evidence is required to further elucidate the influence of elevated waist circumference on hypertension development, particularly in those with a normal BMI.

This study aims to use Chinese and US national population surveys to ascertain whether an elevated waist circumference, irrespective of BMI, increases hypertension risk.

Methods

Data source and study population

This study engaged different data sources, including the China Health and Nutrition Survey (CHNS), China Health and Retirement Longitudinal Study (CHARLS), and National Health and Nutrition Examination Survey (NHANES). The CHNS is an ongoing nationwide population cohort, which is designed to investigate the influence of social and economic on public health based on samples from 15 provinces and autonomous cities/districts in China [17, 18]. The CHARLS collected nationally representative middle or old-aged (≥45 years) individuals from 150 counties/districts and 450 villages/resident committees in China [19]. The NHANES survey is a cross-sectional multistage health survey on the civilian US population [20].

The flowchart for data sources of the study participants is illustrated in Fig. 1. The 2009 wave of CHNS, unique in its collection of blood tests, was established as the baseline, with subsequent 2011 and 2015 surveys serving as follow-ups. For CHARLS, the 2011 survey constituted the baseline, and the 2013 and 2015 CHARLS surveys acted as follow-up data. Seven cycles of the NHANES (2001 ~ 2018) were also incorporated. Given the absence of NHANES follow-ups, we solely used NHANES for cross-sectional correlation analysis.

Inclusion and exclusion criteria

For cross-sectional analysis, participants were acquired from the 2009 CHNS survey, the 2011 CHARLS survey,

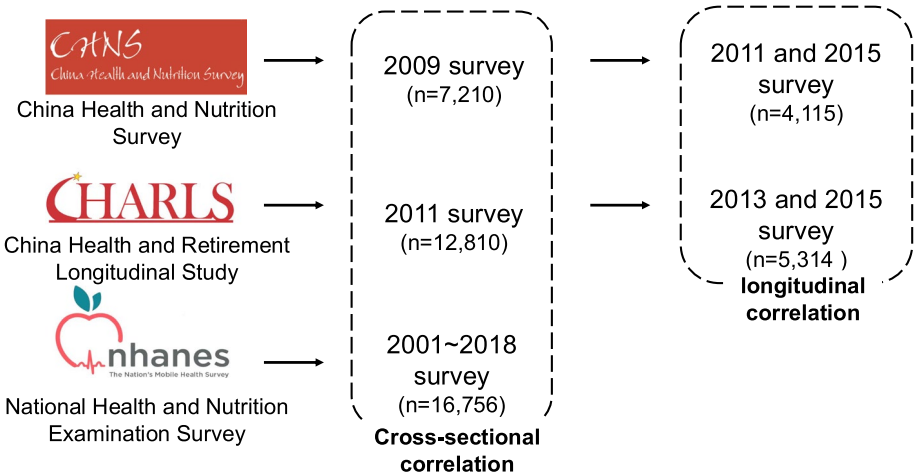


Fig. 1 Flowchart for data sources of the study participants. The 2009 CHNS survey, the 2011 CHARLS survey, and the 2001–2018 NHANES surveys were employed to explore cross-sectional relationships. To examine longitudinal associations, the 2009 CHNS and the 2011 CHARLS surveys served as baseline data, and the 2011, 2015 CHNS surveys alongside the 2013, 2015 CHARLS surveys provided the follow-up data

and the NHANES surveys between 2001 and 2018. Inclusions necessitated completed demographic interviews/questionnaires, physical examinations, fasting biochemical function tests, glycohemoglobin (HbA1c) tests, and medical interviews/questionnaires. The exclusion criteria included: (1) absence of three continuous measurements of blood pressure; (2) age < 18 or ≥ 80 years old; (3) missing measurements for waist circumference, height, or weight; (4) lack of fasting biochemical function tests; (5) pregnancy. A detailed participant flowchart, accounting for the inclusion and exclusion criteria for the baseline wave, can be found in Supplement Fig. 1. In total, the cross-sectional analysis comprised 36,776 participants: 7,210 from CHNS, 12,810 from CHARLS, and 16,756 from NHANES.

As illustrated in Fig. 1, the follow-up was based on the participants without baseline hypertension. This study employed 2011 and 2015 CHNS surveys as well as 2013 and 2015 CHARLS surveys as follow-up data. Exclusion criteria include: (1) missing three continuous measurements of blood pressure during follow-up surveys; (2) diagnosed with hypertension at baseline survey; (3) missing information in the baseline survey. Finally, this study included a total of 9,429 participants (4,115 from CHNS, and 5,314 from CHARLS) without baseline hypertension to assess the longitudinal association.

Measurement of waist circumference, height and weight

While the three surveys followed comparable protocols, nuances existed in their methods for measuring waist circumference. Participants stood upright during measurements. Using a non-elastic tape, waist circumference was gauged to the nearest 0.1 cm at the conclusion of exhalation. NHANES used the apex of the right iliac crest as its reference, whereas CHARLS took measurements at the navel level. In contrast, CNHS used the midpoint between the lowermost rib and the iliac crest. To define abdominal obesity, gender and race-specific thresholds were employed. For the U.S. cohort, abdominal obesity was designated as waist circumferences of ≥ 102 cm for males and ≥ 88 cm for females [21, 22]. The Chinese participants had cut-offs of 90 cm for males and 80 cm for females [23, 24].

Height and weight were measured by trained examiners using standard equipment. Subjects stood without shoes in light attire. Height and weight readings were precise to 0.1 cm and 0.1 kg, respectively. BMI was acquired by $\frac{Weight(kg)}{Height^2(m^2)}$.

Blood pressure measurement and hypertension diagnostic criteria

Trained examiners executed the blood pressure measurements, capturing brachial artery pressure after a 5-min rest. Both CHNS and CHARLS employed electronic blood pressure monitors (Omron, Dalian, China) for measurement, while NHANES utilized mercury sphygmomanometers (Baumanometer®, W.A. Baum, New York, USA). Examiners took three consecutive readings of systolic and diastolic pressures, with a minimum 1-min interval. The study determined the average of these readings as the office blood pressure. Participants who met any of the following criteria were diagnosed with hypertension: (1) average systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg [25]; (2) self-reported hypertension; (3) current administration of anti-hypertensive drugs [26].

Covariates

This study considered various covariates following previous studies. The self-reported covariates include age (continuous) [27], gender (male or female) [28], education level (below high school, high school, or above high school) [29], smoking history (yes or no) [30], drinking history (yes or no) [31], and history of diabetes (yes or no) [32, 33]). The measured covariates include fasting blood glucose (continuous), glycated hemoglobin (continuous), triglycerides (continuous) [34], total cholesterol-to-high-density lipoprotein ratio (continuous), and creatinine (continuous) [35]. The estimated glomerular filtration rate (eGFR) was derived using the Chronic Kidney Disease Epidemiology Collaboration equation [36]. Diabetes was defined as (1) fasting blood glucose ≥ 126 mg/dL, (2) HbA1c ≥ 6.5%, (3) self-reported diabetes, (4) current administration of antidiabetic medication.

Mendelian randomization analysis

Mendelian randomization employs genetic variants present in a population as instrumental variables to investigate the causal relationship between exposures and outcomes [37]. Given that these genetic variations remain uninfluenced by environmental or individual behavioural determinants, the association derived via Mendelian randomization is purely based on biological mechanisms, minimizing confounding interferences. Mendelian randomization operates under three core assumptions: (1) The instrumental variable is closely associated with the exposure; (2) The instrumental variable is not associated with any potential confounders; (3) The instrumental variable can only influence the outcome via the exposure, and not by any other ways.

This study applied Mendelian randomization to deduce the causal relationship between waist circumference and hypertension. We sourced genome-wide association study summary data from the IEU open GWAS Project (https://gwas.mrcieu.ac.uk/) [38]. Waist circumference from the GIANT consortium (ID: ieu-a-61) was used as

exposure, whereas hypertension from UK Biobank (ID: ukb-b-12493) was used as the outcome. Single nucleotide polymorphisms, which were reported to associate with the outcome and exposures, were used as instrumental variables. Our analysis encompassed a suite of methods, including inverse variance weighted, and MR Egger, weighted median, simple mode, and weighted mode.

Moreover, multivariable Mendelian randomization integrates genetic variations of various risk factors into one model, mitigating potential biases from multiple exposures. We also explored the causal association of waist circumference and BMI with hypertension using the multivariable Mendelian randomization method, integrating BMI data from the GIANT consortium (ID: ieu-a-835) as an additional exposure to the earlier model.

Statistical analysis

The analysis adhered to guidelines set by the American Heart Association Scientific Publication Committee [39]. We employed the multivariate multiple imputation method for handling missing covariates, enhancing the statistical power and reducing selection bias [40, 41]. The NHANES survey, having used a multi-stage stratified random sampling approach, mandated weighted statistical methods for analysis. Each NHANES participant was assigned a distinct primary sampling unit (SDMVPSU), layer identifier (SDMVSTRA), and sampling weight. The fasting subsamples from nine NHANES surveys were used, and therefore the weight is 1/9 × WTSAF2YR. All NHANES data were analyzed using weighted statistical methods.

Data distribution was examined with the Kolmogorov–Smirnov test. Normally distributed continuous variables, skewed distributed continuous variables, and categorical variables were presented as mean ± standard deviation, median (Q1, Q3), and frequencies with percentages, respectively. The difference between groups was evaluated by one-way ANOVA test, Kruskal–Wallis test, or chi-square test as appropriate.

For baseline wave cross-sectional analysis, we calculated the correlation coefficient between waist circumference and systolic/diastolic blood pressure using Pearson correlation analysis. Linear regression was applied to assess the association between waist circumference and blood pressure. We applied the adjusted logistic regression model for the association between waist circumference and the prevalence of baseline hypertension. The odds ratios (ORs) with 95% confidence intervals (CIs) were calculated accordingly. For Model 1, we adjusted for age, gender and education level; Model 2 additionally adjusted for triglycerides, total cholesterol/high-density lipoprotein, eGFR, diabetes, fasting blood glucose, HbA1c, smoking and drinking. Previous studies do not recommend adjusting for cardiovascular risk factors that have a causal relation with obesity (such as glucose metabolism, lipid metabolism variables, etc.) when exploring the correlation between waist circumference and metabolic diseases [42]. Accordingly, Model 3 only additionally adjusted for BMI, smoking, and drinking based on Model 1.

In the longitudinal correlation analysis, the trend of hypertension incidence in waist circumference quartile divisions was tested using the Mantel–Haenszel method. The Kaplan–Meier method was used to analyze the time-dependent alterations in the incidence of onset hypertension across different waist circumference groups. The overall difference was assessed by the Log-rank method, with inter-group discrepancies corrected via the Benjamini–Hochberg method. The attributable fraction was calculated to quantify the burden of baseline abdominal obesity on hypertension. Cox regression analysis was used to evaluate the longitudinal correlation between waist circumference and hypertension, and the effect size was represented by the hazard ratio (HR) and 95% CI. The covariable adjustment was consistent with the cross-sectional analysis model.

Although the CHNS and CHARLS surveys were designed for different age groups and areas in China, Cochran's Q test and I^2 heterogeneity analysis suggested a low heterogeneity between the two surveys (Cochran's $Q = 1.22$, $P = 0.27$, $I^2 = 18.0\%$). Therefore, we conducted a pooled analysis for correlation analysis. We utilized restricted cubic splines to evaluate the dose–response relationship between waist circumference and hypertension onset. The median waist circumference was selected as the reference value, and restricted cubic splines were plotted at four percentiles of waist circumference (P5, P25, P75, and P95). Subgroup analyses of Cox regression are conducted for different BMI groups and genders, and the results are presented in a forest plot. Additionally, propensity score matching (PSM) analysis was performed to examine the association waist circumference and new-onset hypertension after matching age, BMI, and lipid profile.

Statistical analysis was performed using R software 4.1.1 (R Foundation for Statistical Computing, Vienna, Austria). A two-sided *P* value less than 0.05 was considered statistically significant.

Results

Participants with baseline hypertension show high waist circumference

Participants with baseline hypertension showed a significantly higher waist circumference than the normotensive individuals in all three surveys. The waist circumference of participants with or without hypertension was 87.0 vs.

80.0 cm ($P<0.001$) in CHNS, 88.2 vs. 82.6 cm ($P<0.001$) in CHARLS and 104 vs. 93.5 cm ($P<0.001$) in NHANES. Besides, the prevalence of abdominal obesity and BMI were significantly higher in participants with hypertension. Detailed participants' characteristics are given in Supplement Table 1 ~ 3.

The Pearson correlation test indicated a significant cross-sectional correlation between the waist circumference and both systolic and diastolic blood pressures. In the CHNS survey, the observed correlation coefficient between the waist circumference and systolic blood pressure was 0.088 ($P<0.05$). Similarly, in the CHARLS survey and the NHANES study, the correlation coefficients were 0.22 ($P<0.01$) and the weighted 0.26 ($P<0.01$), respectively. As for diastolic blood pressure, the correlation coefficients were 0.35, 0.24, and 0.17 for CHNS, CHARLS, and NHANES, respectively.

Moreover, the adjusted logistic regression was used to analyze the cross-sectional correlation between waist circumference and hypertension prevalence in the CHNS, CHARLS, and NHANES surveys (Supplement Table 4). When waist circumference was analyzed as a continuous variable, we observed a significant correlation between waist circumference and hypertension prevalence. The ORs (95% CI) for CHNS, CHARLS, and NHANES surveys were 1.30 (1.19, 1.42), 1.43 (1.35, 1.53), and 1.21 (1.11, 1.31), respectively, after adjusting for age, sex, education levels, smoking, drinking and BMI (Fig. 2). Besides, the common-effect inverse-variance model was used to evaluate the overall association (OR = 1.34, 95% CI = 1.28 ~ 1.40, $I^2 = 81.2\%$).

Higher baseline waist circumference is associated with an increased incidence of hypertension

A total of 4115 participants without baseline hypertension were included in the CHNS survey for follow-up. After a mean follow-up of 4.5 years, there were 1,114 (27.1%) new cases of hypertension. The median age was 48 years old, and 45.1% were male. The incidences of hypertension in the waist circumference quartiles (from Q1 to Q4) were 16.3%, 24.8%, 29.2%, and 38.9%, respectively ($P_{\text{for trend}}<0.001$). From the CHARLS survey, a total of 5314 participants without baseline hypertension were included for follow-up. Within the mean follow-up of 3.3 years, 1,478 cases of hypertension occurred, constituting 27.8% of the total population. The participant cohort had a median age of 57 years, with males comprising 46.9% of the group and a median waist circumference of 82.4 cm. The incidences of hypertension across groups Q1 to Q4 were 22.9%, 24.5%, 28.2%, and 35.9% ($P_{\text{for trend}}<0.001$). The pooled baseline characteristics across waist circumference quartiles are given in Table 1. Additionally, baseline characteristics of participants for longitudinal analysis from CHNS and CHARLS surveys are given in Supplement Tables 5 and 6, respectively.

Table 1 The pooled baseline characteristics across waist circumference quartiles

	Q1 (*N* = 2361)	Q2 (*N* = 2354)	Q3 (*N* = 2373)	Q4 (*N* = 2341)	*P*
Age (years)	52 (42, 61)	54 (46, 62)	54 (47, 61)	55 (47, 62)	< 0.001
Gender (male, n, %)	1002 (42.4%)	1094 (46.5%)	1148 (48.4%)	1105 (47.2%)	< 0.001
Education levels (n, %)					0.024
Below high school	1946 (82.4%)	1973 (83.9%)	1982 (83.5%)	1959 (83.7%)	
High school	217 (9.2%)	237 (10.1%)	245 (10.3%)	217 (9.3%)	
Above high school	198 (8.4%)	143 (6.1%)	146 (6.2%)	165 (7.0%)	
Waist circumference (cm)	71.1 (68.0, 73.8)	78.8 (77.0, 80.0)	85.0 (83.2, 87.0)	93.4 (90.6, 97.8)	< 0.001
BMI(kg/m^2)	19.7 (18.5, 21.1)	21.7 (20.4, 23.0)	23.4 (22.1, 24.8)	26.1 (24.4, 27.9)	< 0.001
New-onset hypertension (n, %)	472 (20.0%)	574 (24.4%)	682 (28.7%)	864 (36.9%)	
SBP (mmHg)	114.0 (106.0, 122.3)	117.3 (109.0, 124.7)	119.3 (110.3, 126.3)	120.7 (113.3, 128.7)	< 0.001
DBP (mmHg)	70.7 (65.0, 78.0)	72.0 (66.0, 79.3)	73.7 (67.7, 80.0)	76.3 (70.0, 80.7)	< 0.001
TG (mg/dL)	85.0 (63.8, 117.8)	93.8 (69.0, 132.9)	106.2 (75.3, 154.7)	125.7 (87.7, 190.3)	< 0.001
TDL/HDL	3.1 (2.6, 3.7)	3.3 (2.8, 4.0)	3.7 (3.0, 4.4)	4.1 (3.3, 5.0)	< 0.001
eGFR ($ml/min/1.73m^2$)	79.3 (63.1, 96.8)	86.6 (70.1, 99.4)	88.7 (73.7, 100.4)	92.0 (76.8, 102.8)	< 0.001
Diabetes (Yes, %)	111 (4.7%)	177 (7.5%)	233 (9.8%)	369 (15.8%)	< 0.001
FBG (mg/dL)	93.2 (85.7, 102.1)	95.8 (88.2, 104.9)	97.2 (89.1, 106.7)	100.6 (92.3, 111.1)	< 0.001
HbA1c (%)	5.2 (4.9, 5.5)	5.2 (4.9, 5.5)	5.3 (5.0, 5.6)	5.3 (5.0, 5.8)	< 0.001
Smoking (Yes, %)	807 (34.2%)	858 (36.4%)	872 (36.7%)	793 (33.9%)	0.075
Drinking (Yes, %)	508 (21.5%)	626 (26.6%)	663 (27.9%)	656 (28.0%)	< 0.001

Q1, waist circumference ≤ 75.3 cm; Q2, 75.3 < waist circumference ≤ 81.9 cm, Q3; 81.9 < waist circumference ≤ 88.8 cm, Q4; waist circumference > 88.8 cm

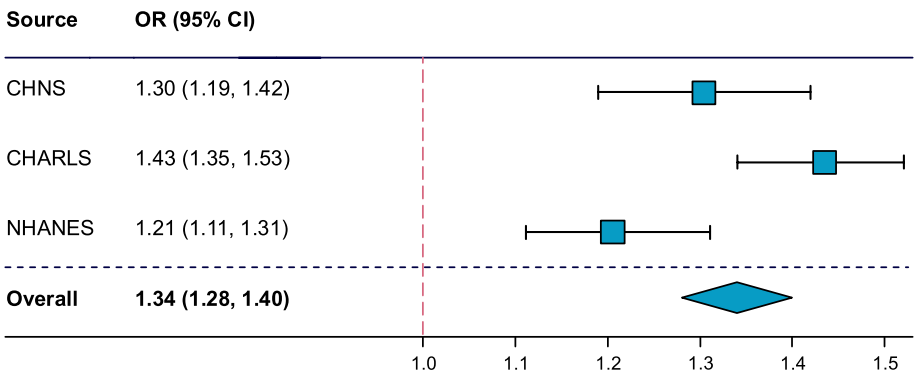


Fig. 2 The cross-sectional association between waist circumference and the prevalence of hypertension. The overall effect size of the three surveys was assessed by a common-effect inverse-variance model. Age, sex, education levels, smoking, drinking, and BMI were adjusted for

The Kaplan–Meier curves illustrating hypertension incidence across waist circumference quartiles in CHNS and CHARLS cohorts are presented in Figs. 3A and 3B, respectively. A significant difference in hypertension onset was observed across groups (Both log-rank $P<0.001$). Additionally, the cumulative incidence of hypertension during follow-ups and the inter-group discrepancies are provided in Supplement Fig. 2. Moreover, we applied Cox regression to analyze the longitudinal correlation between waist circumference and the new onset of hypertension in the two cohorts (Supplement Table 7). After adjusting for age, gender, education level, smoking, drinking, and BMI, the HRs (with 95% CI) for waist circumference (every 10 cm) were 1.12 (95% CI = 1.02 ~ 1.23) in the CHNS survey and 1.22 (95% CI = 1.12 ~ 1.32) in the CHARLS survey (Fig. 3C and 3D). Besides, was calculated the attributable fraction to quantify the burden of baseline abdominal obesity on hypertension incidence. For the CHNS survey, 14.69% (95%CI = 11.32% ~ 18.06%, $P<0.001$) new cases of hypertension were attributed to abdominal obesity at 2-year follow-up and 12.39% (95%CI = 9.57% ~ 15.20%, $P<0.001$) at 6-year follow-up. As for CHARLS, 10.94% (95%CI = 7.95% ~ 13.92%, $P<0.001$) and 10.20% (95%CI = 7.43% ~ 12.97%, $P<0.001$) of new-onset hypertension were attributable to abdominal obesity at 2-year and 4-year follow-up, respectively.

In the pooled analysis, Cox regression indicated a significant correlation between waist circumference and hypertension incidence when analyzing waist circumference as a continuous variable across all adjusted models. After adjusting for age, gender, education level, smoking, drinking, and BMI, each 10 cm increase in waist circumference led to a 1.20-fold increase in the risk of hypertension (HR = 1.20, 95% CI = 1.13 ~ 1.27, Fig. 3E). A significantly increased risk of hypertension was observed in Q3 and Q4 compared with the lowest waist circumference group. When adjusting for age, gender, education level, smoking, drinking, and BMI, the HRs (95% CI) for Q3 and Q4 were 1.22 (1.07 ~ 1.39) and 1.45 (1.26 ~ 1.68), respectively. However, there was no significant difference in hypertension risk between the Q2 and Q1 groups ($P=0.214$). Besides, given the potential heterogeneity between the two surveys, we also adjusted for the cohort source (CHNS or CHARLS) based on model 3, and the results showed that each 10 cm increased waist circumference was associated with a 1.19-fold increase in the risk of hypertension (HR = 1.19, 95% CI = 1.12 ~ 1.26).

Moreover, we observed an ascending trend in hypertension risk with an increase in waist circumference, which indicates a linear dose–response association between the variables ($P_{\text{for nonlinear}}=0.47$) (Fig. 4A). Consistently, we observed a positive linear association between waist circumference and hypertension in both males (Fig. 4B) and females (Fig. 4C).

Subgroup analysis on the association between waist circumference and new-onset hypertension

We observed a consistent trend in the subgroup analysis across BMI categories (underweight, normal weight, overweight, and obesity) and genders (male, or female) (Fig. 5A). After adjusting for age, gender, education level, smoking, drinking, and BMI, a significant correlation was observed between waist circumference and hypertension in the group with normal weight (HR = 1.16, 95% CI = 1.05 ~ 1.27) and the overweight group (HR = 1.22, 95% CI = 1.09 ~ 1.38). However, such correlation was non-significant in both the underweight group (P = 0.142) and the obese group (P = 0.071). Besides, waist circumference showed a significant correlation with hypertension in both male (HR = 1.19, 95% CI = 1.09 ~ 1.30) and female groups (HR = 1.19, 95% CI = 1.10 ~ 1.28). Besides, the sensitivity analysis on age, BMI, and lipid profiles-matched subgroup confirmed the significant association

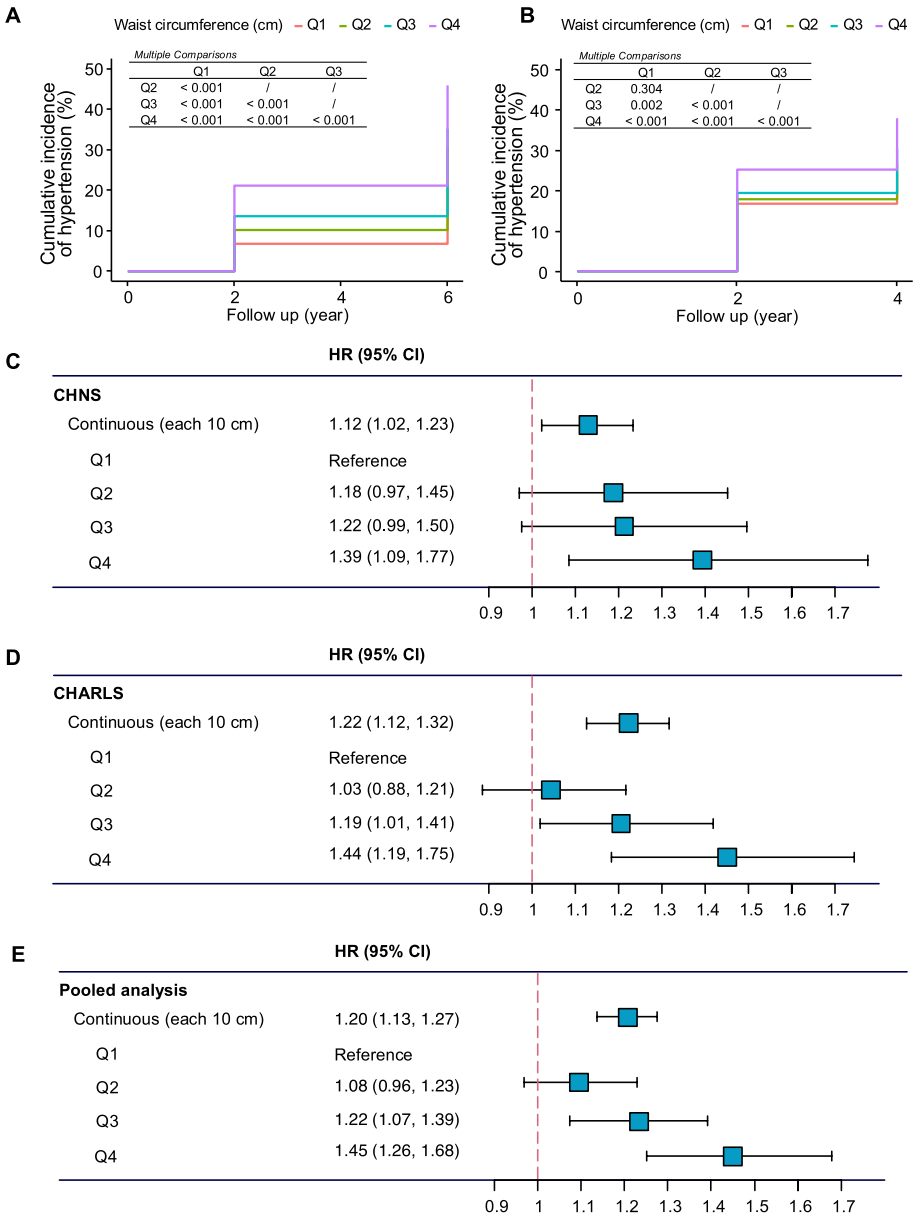


Fig. 3 High baseline waist circumference is associated with an increased incidence of hypertension. The Kaplan–Meier curves for the incidence of hypertension in (**A**) CHNS and (**B**) CHARLS. Log-rank $P < 0.001$ in both surveys. The multiple comparisons among groups are adjusted by the Benjamini–Hochberg method. The Cox regression evaluates the association between waist circumference hypertension incidence in (**C**) CHNS and (**D**) CHARLS surveys. **E** The pooled Cox regression analysis based on the two surveys. Waist circumference was analyzed by continuous variables and quartiles. Age, sex, education levels, smoking, drinking, and BMI were adjusted for. For CHNS, waist circumference quartiles were: Q1 ≤ 74.0 cm; Q2, 74.0 ~ 80.5 cm; Q3: 80.5 ~ 88.0 cm, Q4 > 88.0 cm. For CHARLS, waist circumference quartiles were: Q1 ≤ 76.2 cm; Q2, 76.2 ~ 82.4 cm; Q3: 82.4 ~ 89.2 cm, Q4 > 89.2 cm

between waist circumference and hypertension incidence (Fig. 5B).

High waist circumference is genetically associated with hypertension

We performed two-sample Mendelian randomization analyses, setting waist circumference as exposure and hypertension as the outcome (Fig. 6A). It was found that the increased waist circumference could contribute to the risk of hypertension. In the inverse variance weighted model, the increase in waist circumference was positively associated with a higher risk of hypertension (OR, 1.05; 95%CI, 1.03 ~ 1.07). As shown in Fig. 6B, the sensitivity analysis based on multiple models indicated the

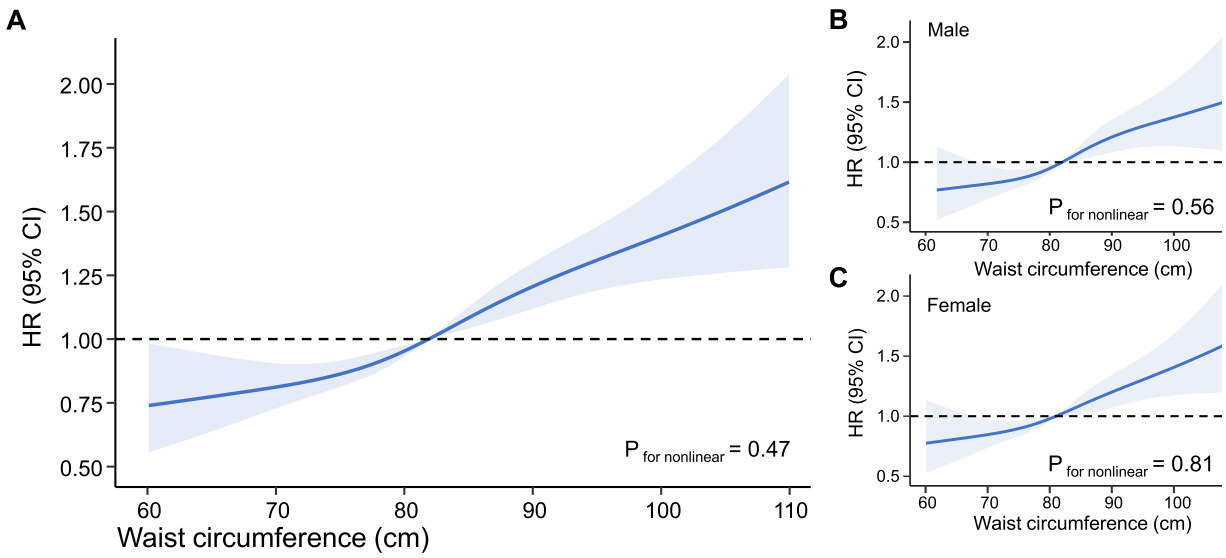


Fig. 4 Restricted cubic spline association between waist circumference and the incidence of hypertension. Age, gender, education level, and BMI were adjusted for. **A** The restricted cubic spline based on general participants. The restricted cubic spline in the subgroup of (**B**) males and (**C**) females

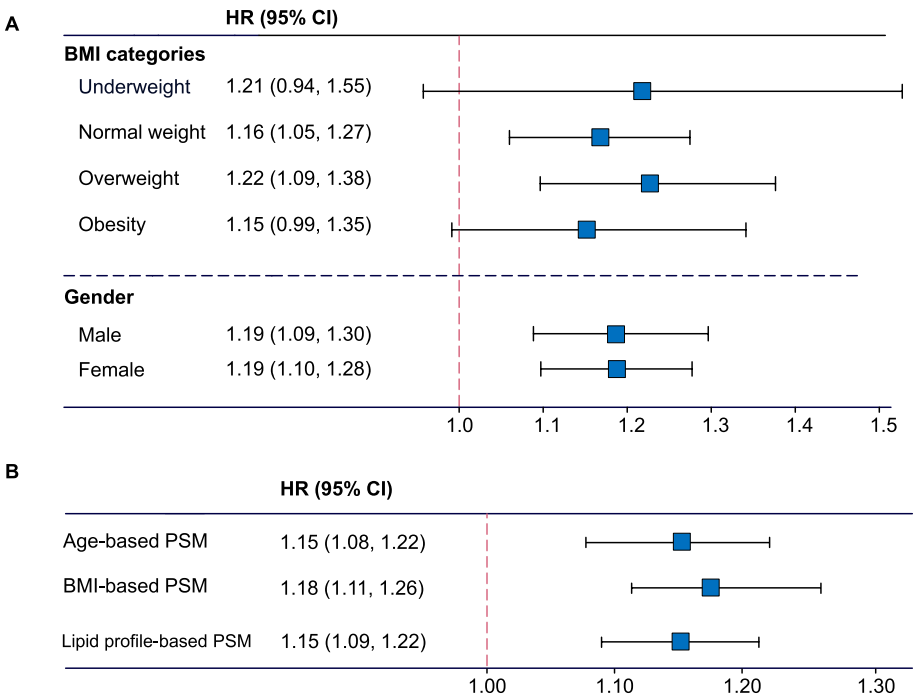
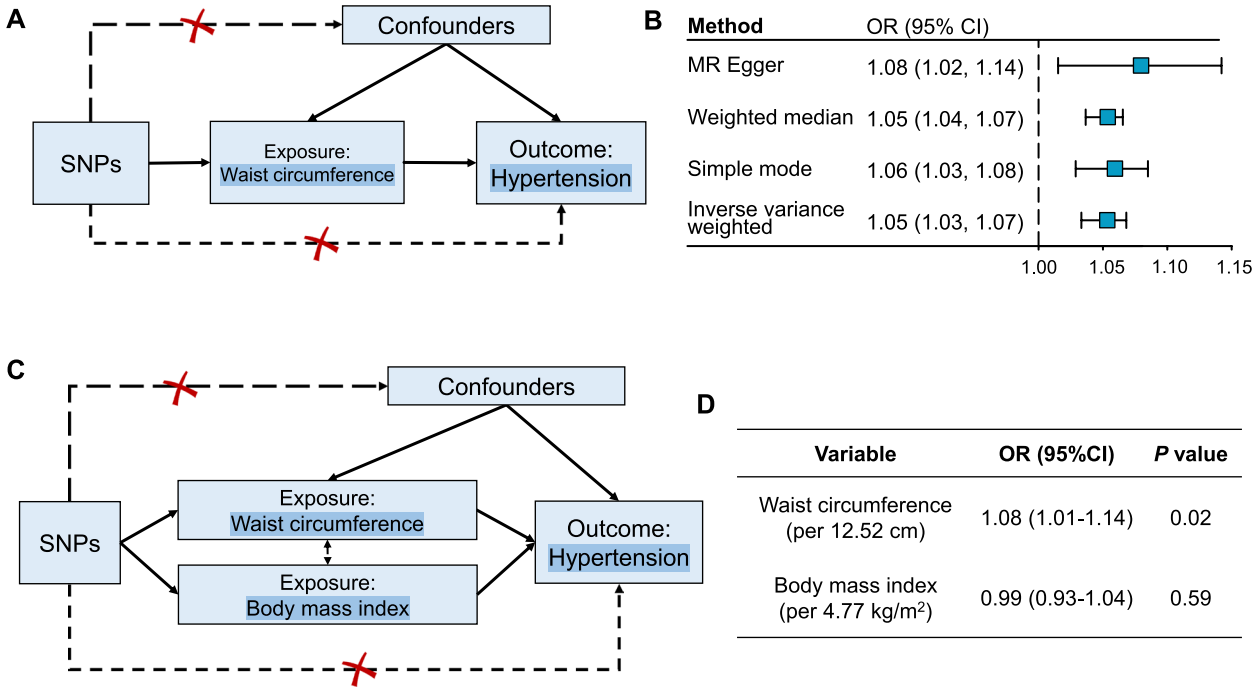


Fig. 5 Subgroup analysis on the association between waist circumference and new-onset hypertension. **A** The subgroup analysis between waist circumference and hypertension incidence across BMI categories and genders. Underweight: BMI < 18.5 kg/m^2; normal weight: BMI, 18.5–23.9 kg/m^2; overweight: BMI, 24–27.9 kg/m^2; and obesity ≥ 28 kg/m^2. **B** PSM analysis was performed on age, BMI, and lipid profile-matched subgroups

consistent genetic association between waist circumference and hypertension.

Considering the potential interaction between waist circumference and BMI, we input waist circumference and BMI collectively as exposures (Fig. 6C). In the multivariable Mendelian randomization model, high waist circumference significantly increased the risk of



Variable	OR (95%CI)	*P* value
Waist circumference (per 12.52 cm)	1.08 (1.01-1.14)	0.02
Body mass index (per 4.77 kg/m^2)	0.99 (0.93-1.04)	0.59

Fig. 6 Mendelian randomization analysis on the association between waist circumference and hypertension. **A** Two sample Mendelian randomization analyses set waist circumference as exposure and hypertension as outcome. **B** MR Egger, Weighted median, simple mode, and Inverse variance weighted models consistently indicated the genetical association between higher waist circumference and hypertension risk. **C** The waist circumference and BMI were input collectively as exposures in the multivariable Mendelian randomization model. **D** The Inverse variance weighted model showed that waist circumference was significantly associated with hypertension but not BMI

hypertension (OR, 1.08; 95% CI, 1.01 ~ 1.14) but not BMI (Fig. 6D).

Discussion

Excess abdominal fat accumulation presents a greater metabolic risk compared to subcutaneous fat. Among various methods for evaluating body composition, BMI remains a primary method for estimating body fat and assessing obesity-related cardiometabolic risk [43, 44]. However, BMI does not consider fat distribution and fails to differentiate between fat and lean mass. Consequently, BMI alone is insufficient to fully capture the cardiometabolic risks associated with obesity. In contrast with BMI, waist circumference strongly correlates with the total amount of abdominal fat and provides a more accurate assessment of metabolic risk [8, 45]. Despite its importance, routine measurement of waist circumference is not yet a standard requirement in clinical practice, and it is typically recommended only for additional measurements in the obese population [46–48]. Accordingly, a recent expert consensus highlights that waist circumference can better assess the metabolic risks associated with fat distribution, and it is important to thoroughly evaluate the association between waist circumference and cardiovascular risk [45]. This study used large-scale population-level surveys to provide robust evidence of a positive correlation between waist circumference and the incidence of hypertension. Importantly, the Mendelian randomization method confirmed a significant causative association between waist circumference and hypertension. Our findings revealed that when evaluating the risk of hypertension related to obesity, waist circumference should be considered alongside BMI to provide a comprehensive assessment of obesity-related hypertension risks.

This study demonstrated the significant cross-sectional association between waist circumference and hypertension using large-scale population data from the US and China. Similarly, Wu et al. [49] analyzed a representative sample of 45,853 Americans obtained through multistage stratified cluster random sampling. Their results suggested that the waist circumference of hypertensive patients was significantly higher than that of the normotensive participants (103.50 *vs.* 92.61 cm, $P < 0.001$). After adjusting for age, gender, race/ethnicity, education, smoking, alcohol consumption, diabetes, and eGFR, the OR was 1.71 (95% CI 1.68 ~ 1.75). Although this study employed a stratified cluster random sampling method for sample selection, it did not use a weighted statistical scheme in data analysis, which was a significant

limitation of this study [49]. Another cross-sectional study on 785 adults from Algeria reported a significant correlation between waist circumference and systolic pressure (Pearson correlation coefficient, 0.013), but not with diastolic pressure [50]. However, this study had a small sample size, and the results only focused on the effect of waist circumference on systolic/diastolic pressure without quantifying the effect of waist circumference on hypertension [50]. Additionally, all the participants in this study were patients with metabolic abnormalities and not a normal population, causing a potential bias in the conclusions [50]. Ren et al.[51]conducted a cross-sectional data analysis on 10,719 Chinese adults to explore the correlation between abdominal obesity and hypertension under normal weight (18.5 kg/m^2 ~ 24.9 kg/m2). The results suggest that, in patients with normal BMI, the hypertension risk in the group with abdominal obesity is higher than in the group with normal waist circumference (OR = 1.49, 95% CI = 1.14 ~ 1.95) [52].

The pooled analysis of follow-up data from the CHNS and CHARLS surveys indicated that every 10 cm increase in waist circumference resulted in a 20% increase in the risk of hypertension (95% CI = 13% ~ 27%). Importantly, the correlation remained in participants with normal BMI, and the risk of hypertension increased by 16% for every 10 cm increase in waist circumference. Wang et al. [36] reported that every 10 cm increase in waist circumference raised systolic pressure by 0.98 mmHg (male)/0.98 mmHg (female) and diastolic pressure by 1.13 mmHg (male) /0.74 mmHg (female) after adjusting for BMI. In a cohort enrolling 10,265 individuals without baseline hypertension from rural areas of Luoyang, Zhao et al. [51] observed 2,027 cases of new-onset hypertension after a follow-up of 6 years. Participants were divided into four groups according to the alteration in waist circumference: ≤ -2.5%, -2.5% ~ 2.5%, 2.5% ~ 5%, and > 5%. Compared with the control group (-2.5% ~ 2.5%), the > 5% increase in waist circumference made the risk of hypertension increase by 34% in males and 28% in females [51]. A prospective cohort study on 6,096 normotensive Chinese individuals showed that 26.8% developed hypertension over a 12-year follow-up [53]. Logistic regression analysis revealed that the risk of hypertension in the abdominal obesity group had increased by 79% (95% CI = 36% ~ 135%) compared to the group without abdominal obesity [53]. Together with previous studies, our results provide further evidence from population-level cohorts supporting high waist circumference as an independent risk factor for hypertension.

BMI was previously considered an alternative indicator to waist circumference, which can replace the measure of BMI. However, our results indicated a significant correlation between waist circumference and hypertension after adjusting for BMI, and the correlation remained in the population with normal BMI. Consistent with our results, the Kailuan cohort also found that in people with a BMI < 24 kg/m^2, the prevalence of hypertension increased with the waist circumference [54]. Therefore, BMI alone is insufficient to fully evaluate metabolic risks associated with fat distribution. For example, abdominal obesity under normal BMI (low BMI but high waist circumference) is a unique metabolic phenotype that implies low muscle mass, less subcutaneous fat distribution in the limbs, and excessive visceral fat [55–58]. Beyond energy storage depots, ongoing research has revealed the interaction of adipose tissue with systemic inflammation responses, the sympathetic nervous system, and the renin-angiotensin system, which thereby promotes obesity-related cardiovascular metabolic disorders and hypertension. Moreover, it should be noted that there is no significant association between waist circumference and new-onset hypertension in underweight or obese individuals. Apart from the small sample size (n = 716 in the underweight group, and n = 667 in the obesity group), it would be interesting to explore the potential mechanisms underlying this observation.

Our study investigated the association between waist circumference and hypertension based on large-scale national population surveys. Despite the representative national samples, the limitations should be mentioned. First, in the longitudinal analysis, the average follow-up of 3.8 years was insufficient, and the risk of high waist circumference to hypertension may not be fully exposed. It is necessary to investigate this correlation over a long-term follow-up time. Second, the diagnostic criteria of hypertension were based on a single average office blood pressure and self-reported hypertension. We did not distinguish primary and secondary hypertension. The loose hypertension diagnostic standards may interfere with the assessment of the risk of waist circumference. Third, despite multiple covariates being considered in this study, residual confounding still exists, such as dietary factors. It is important to explore the interactive effect of these residual confounding, waist circumference, and hypertension in the following research. Besides, the longitudinal association was only based on Chinese individuals. The association between waist circumference should be further validated in the Western population.

Conclusion

This study demonstrated that high waist circumference is a risk factor for hypertension. For every 10 cm increase in waist circumference, there is a 20% higher risk of hypertension. This correlation remains in individuals with a normal BMI. Our results provide evidence supporting

the routine measure for waist circumference regardless of BMI-defined obesity.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12937-024-01058-5.

Supplementary file 1: Supplement Figure 1. The inclusion and exclusion flowchart of participants for cross-sectional analysis from (A) CHNS, (B) CHARLS, and (C) NHANES surveys. Supplement Figure 2. The cumulative incidence of hypertension during follow-ups. The cumulative incidence of CHNS at the (A) first (follow-up time, 2 years) and (B) second follow-up (follow-up time, 6 years). The cumulative incidence of CHARLS at the (C) first (follow-up time, 2 years) and (D) second follow-up (follow-up time, 4 years). The inter-group discrepancies were evaluated by the Benjamini-Hochberg method.

Authors' contributions

(I) Conception and design: Jin-Yu Sun, Zhengyang Su, Hui Shen, Yang Hua, and Xiang-Qing Kong; (II) Administrative support: Wei Sun, and Xiang-Qing Kong; (III) Data analysis and interpretation: Jin-Yu Sun, Zhengyang Su, Hui Shen, and Yang Hua; (IV) Manuscript writing and revising: Jin-Yu Sun, Zhengyang Su, Hui Shen, Yang Hua, and Xiang-Qing Kong; (V) Final approval of manuscript: All authors.

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Data availability

All the data in this study can be accessed in the China Health and Nutrition Survey, China Health and Retirement Longitudinal Study, and National Health and Nutrition Examination Survey digital database.

Declarations

Ethics approval and consent to participate

The China Health and Nutrition Survey was approved by the institutional review committees of the University of North Carolina at Chapel Hill and National Institute for Nutrition and Health, Chinese Center for Disease Control and Prevention. The China Health and Retirement Longitudinal Study was approved by the Ethics Review Committee of Peking University. National Health and Nutrition Examination Survey was approved by the National Center for Health Statistics Research Ethics Review Board. Written informed consent was obtained from all participants.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

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