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Association between body roundness index and incidence of type 2 diabetes in a population-based cohort study

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There is limited national data on the association between body roundness index (BRI) and type 2 diabetes (T2D). A total of 10,785 participants from the China Health and Retirement Longitudinal Study (CHARLS) with repeated BRI measurements from 2011 to 2020 were included. We used Cox proportional hazards model and restricted cubic splines (RCS) to examine the association between BRI and T2D. During a mean follow-up of 7.72 years, 1,653 incident T2D cases were documented. Multivariable Cox proportional hazards regression model demonstrated a significant correlation between the BRI and the risk of T2D. Specifically, every 1-SD increase in BRI corresponded to a 27% heightened risk of T2D (HR: 1.27, 95% CI 1.20–1.35). The analysis also uncovered a non-linear pattern in this relationship, pinpointed by an inflection point at a BRI value of 3.96. Before the inflection point, the HR was 0.85 (95% CI 0.74–0.96), while after the inflection point, the HR increased to 1.29 (95% CI 1.18–1.41). In the middle-aged and elderly Chinese population, elevated BRI was significantly and positively associated with T2D risk. BRI could be a valuable addition to current clinical and public health strategies aimed at reducing the burden of T2D.

Keywords Body roundness index, CHARLS, Obesity, Type 2 diabetes, Visceral obesity

Type 2 diabetes (T2D) is a progressive metabolic disorder characterized by impaired β-cell insulin secretion, often accompanied by insulin resistance (IR) and excessive adiposity[1]. Approximately 500 million individuals globally are affected by T2D[2], with projections indicating a rise to 1.3 billion by 2050[3]. T2D is strongly associated with numerous severe complications, which significantly impair patients' quality of life and contribute substantially to disability and mortality[4]. Moreover, the escalating prevalence of T2D places a considerable burden on healthcare systems worldwide, with extensive economic and societal repercussions[5]. In particular, the rapid rise in T2D prevalence in China highlights the global scope of this issue, with the country now having the largest number of individuals living with T2D, accounting for approximately one-quarter of the global diabetes population[6].

Obesity is a major modifiable risk factor for T2D[7], strongly associated with disturbances in glucose metabolism, which contribute to the development of T2D[8]. Obesity, commonly assessed by body mass index (BMI), has been shown to be significantly associated with an increased risk of T2D[9]. With the extensive investigation of body composition, more attention has been paid to the association between visceral obesity and T2D[10,11]. Numerous studies have demonstrated that visceral fat is a new independent risk marker of T2D[12]. Visceral fat may contribute to IR and glucose metabolism dysregulation through the secretion of pro-inflammatory cytokines and free fatty acids, thereby increasing the risk of developing T2D[13]. However, it is widely recognized that BMI is remarkably heterogeneous[14] and that individuals with similar body weight or BMI can have substantially different fat distribution and body composition[15]. Moreover, its most notable shortcoming is its inability to distinguish between different types of weight[16]. Therefore, it is widely recognized that body fat content cannot be accurately characterized by BMI. Although recent evidence has indicated that waist circumference (WC) alone is strongly associated with the amount of intra-abdominal or visceral fat[17], it relies solely on a single measurement dimension, lacking a more comprehensive analysis of body morphology.

To better assess fat distribution, the Body Roundness Index (BRI) was introduced by Thomas et al.[18], who developed elliptical models based on human body shape to estimate body roundness, using eccentricity to approximate visceral fat and total body fat percentages. Moreover, BRI uses WC in conjunction with body shape models, which allow it to more comprehensively reflect visceral fat distribution. Preliminary studies

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have indicated that BRI is more strongly associated with the accumulation of visceral fat[18], IR[19], and metabolic syndrome[20], potentially providing more valuable predictive information for T2D risk assessment. Several studies have demonstrated the effectiveness of BRI in predicting risks for all-cause mortality[21], metabolic syndrome[20], cardiovascular disease (CVD)[22] and cancer[23]. However, there is limited national data on the association between BRI and T2D, and to our knowledge, no study has been conducted in the general populations of China. Therefore, further investigation into the potential association between BRI and the risk of developing T2D, particularly its application in early screening and personalized interventions, holds significant clinical implications.

To address this gap, we aimed to examine the association between BRI and the risk of incident T2D among adults aged 45 years and older in a nationally representative China population sample from 2011 to 2020. Through this study, we aim to provide new insights and practical evidence for the prevention, early diagnosis, and personalized intervention of T2D.

Materials and methods

Study design

In a population-based prospective cohort study, the China Health and Retirement Longitudinal Study (CHARLS) recruited 17,708 participants from 28 provinces across China. CHARLS is an ongoing cohort study that investigates the social, economic, and health conditions of the Chinese population aged 45 and older[24]. The baseline survey, employing a multi-stage probability sampling strategy, commenced in 2011 (Wave 1), with subsequent follow-up surveys conducted in 2013 (Wave 2), 2015 (Wave 3), 2018 (Wave 4), and 2020 (Wave 5). Data are available by request to CHARLS (http://charls.pku.edu.cn/index.htm). For detailed information of CHARLS, see Supplementary file 1 and Zhao et al.[24].

Study population and data source

In the present study, we selected data from all five waves (2011 to 2020) to examine the association between BRI and the incidence of T2D. The initial 2011 baseline survey included 17,708 participants. Several exclusion criteria were applied to refine the study population. First, we excluded 508 individuals aged less than 45 years and 3,920 individuals with missing data for BRI. Additionally, we excluded 1,641 participants with a previous diagnosis of T2D and 113 participants with missing T2D information. A total of 236 participants who had abnormal BRI (< 1 or > 20) were excluded, and 505 participants were lost to follow-up. Ultimately, 10,785 participants were eligible for analysis. The detailed methodology of the participant selection process is illustrated in Fig. 1.

Exposure

The exposure in the current study was BRI. BRI was calculated as $364.2 - 365.5 \times \sqrt{(1 - [\text{WC in centimeters} / 2\pi]^2 / [0.5 \times \text{height in centimeters}]^2)}$. WC and height were measured to the nearest 0.1 cm. WC was measured using a flexible tape measure at the level of the navel at the end of normal exhalation. Height was measured using a Seca 213 stadiometer (Seca GmbH, Hamburg, Germany) with participants standing upright, barefoot, on the instrument's base. Due to the lack of a reference range, BRI was categorized into 5 groups based on the 20th, 40th, 60th, and 80th quantiles, as per previous studies[21], to explore its association with T2D. Participants were also excluded if their BRI was less than 1 or greater than 20.

Outcome

The outcome of this study was based on the diagnosis of T2D at any of the follow-up visits from Wave 2 to Wave 5. In the CHARLS cohort, T2D was diagnosed based on responses to two questions: "Have you been diagnosed with T2D by a doctor?" and "Are you currently taking any treatments for diabetes?" Participants who answered affirmatively to either question were considered to have T2D. Additionally, T2D was defined as fasting plasma glucose (FPG) > 126 mg/dL, postprandial blood glucose (PBG) > 200 mg/dL, or HbA1c ≥ 6.5%.

Covariates

The following variables were considered likely confounding factors: age, gender (male/female), education (< 6 / 6 – 9/ 9 – 12 / > 12), smoking and drinking status (yes/no), sleep duration, physical activity (PA), dyslipidemia, FPG, total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), HbA1c. The confounders included in our multivariable Cox model (e.g., age, sex, smoking status, physical activity, and HbA1c) were selected if they were established risk factors for T2D in prior studies[25–27] or potential confounders of the BRI-T2D association. Age, gender, education, smoking, and drinking status were obtained from self-reported questionnaires. Sleep duration was calculated as the sum of nighttime sleep and daytime napping hours per day. Based on the questionnaire responses, daily PA duration was classified into 5 groups: 0 min, 10–29 min, 30–119 min, 120–239 min, and ≥ 240 min, using the midpoint values for analysis. PA types were further classified into vigorous PA (MET = 8.0, e.g., climbing, running, farming), moderate PA (MET = 4.0, e.g., brisk walking, Tai Chi), and light PA (MET = 3.3, e.g., casual walking) based on their corresponding MET values. MET-minutes/week = MET value × days × duration. Blood samples were collected by trained medical staff from each respondent following a standard protocol from the Chinese Center for Disease Control and Prevention.

Statistical analysis

Baseline characteristics are presented as the mean (SD) for continuous variables or number (%) for categorical variables, as appropriate. Differences between BRI groups were tested using the χ^2 test, and differences in continuous variables were analyzed using analysis of variance (ANOVA) and the Kruskal–Wallis H test.

For participants diagnosed with T2D during follow-up, person-years spanned from baseline to diagnosis date, while for those remaining diabetes-free by 2020, person-years extended from baseline to their last follow-

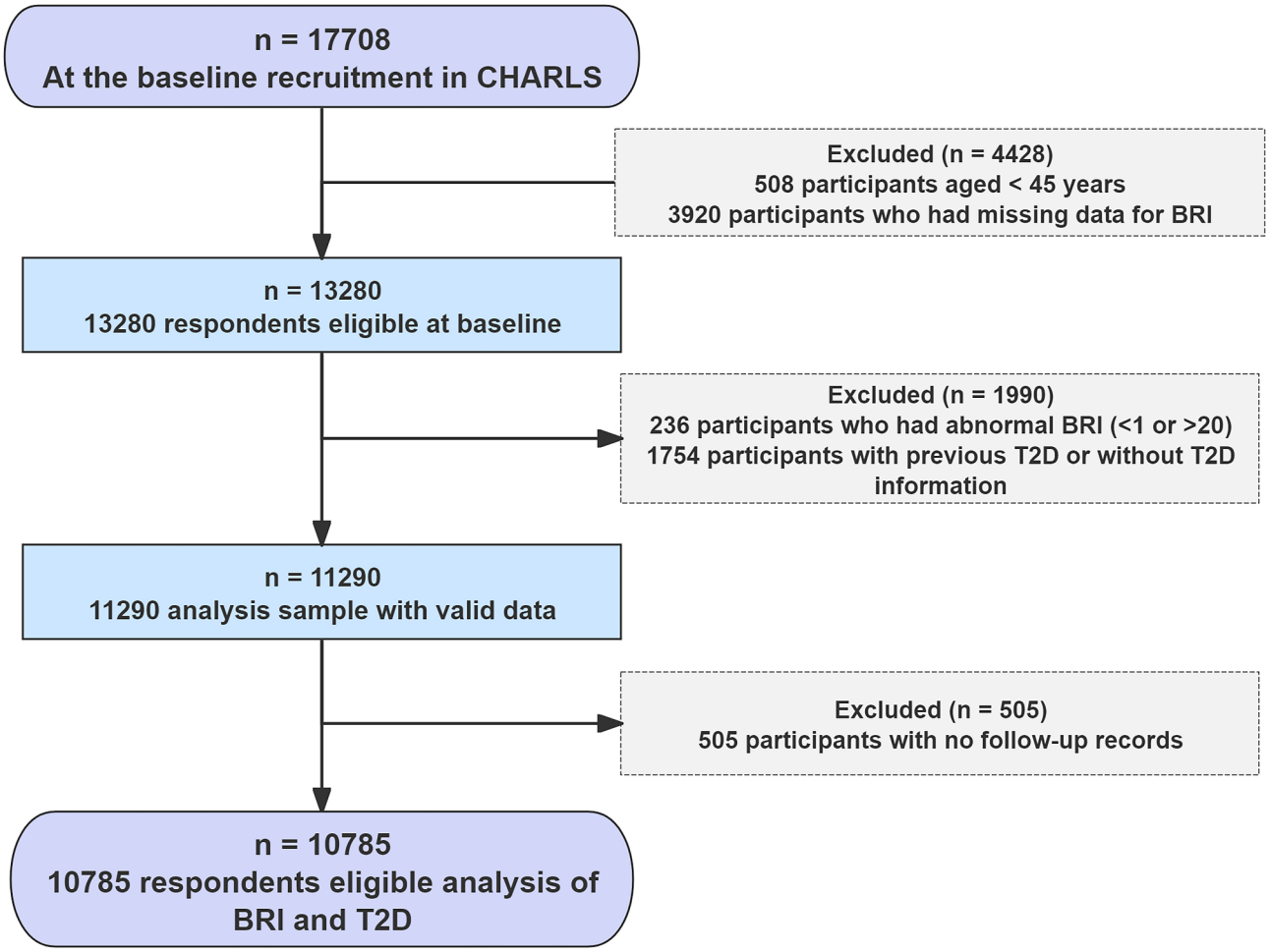

Fig. 1. Flowchart of participant enrollment. BRI: body roundness index; T2D: type 2 diabetes.

up visit, and the total person-years were summed to calculate incidence rates as $\frac{\mathrm{Numberofcases}}{\mathrm{Totalperson-years}} \times 10{,}000$. Participants who lacked follow-up data after the baseline survey were excluded due to the absence of information necessary for calculating person-years or ascertaining diabetes incidence.

We employed univariable and multivariable Cox regression models to estimate the relationship between BRI and the risk of T2D. Model I was adjusted for age and gender. Model II was adjusted for age, gender, education, drinking, smoking, sleep duration, PA. Model III was adjusted for age, gender, education, drinking, smoking, sleep duration, PA, dyslipidemia,, FPG, TG, HDL, LDL and HbA1c.The TC was excluded from the final multivariable Cox proportional hazards model due to collinearity with other predictors, as detailed in Supplementary file 1: Table S1. We checked the Cox proportional hazards assumption using Schoenfeld residuals.

Several sensitivity analyses were performed to validate the findings. First, chronic diseases were included as covariates in the model for analysis. In addition, participants with a BMI ≥ 24 kg/m^2 were excluded from the sensitivity analyses ($n = 6673$)[28]. Finally, we excluded participants who developed T2D at the second visit to minimize potential reverse causality, considering short length of follow-up may influence the outcome ($n = 10659$).

To explore potential non-linear associations between the BRI index and T2D risk, we employed Cox proportional hazards models with smooth curve fitting. We fit restricted cubic spline models with 3–5 nodes and then selected the model with the smallest AIC to determine the number of nodes. Where nonlinearity emerged, a recursive algorithm pinpointed the inflection point. We then formulated a piecewise Cox proportional hazards model on either side of these inflection points. The optimal model describing the BRI-T2D risk relationship was determined through a log-likelihood ratio test.

Subgroup analyses of different subgroups (age, gender, smoking status, alcohol consumption status, marital status, education duration, residential area, and exercise) were performed using stratified Cox proportional hazards regression models. In addition to stratification factors, we adjusted for age, gender, education, drinking, smoking, sleep duration, PA, dyslipidemia, FPG, TG, HDL, LDL and HbA1c. To assess the presence of an interaction term, we used likelihood ratio tests in models with and without an interaction term.

Results

Characteristics

A total of 10,785 participants, including 5,632 females and 5,153 males, with a mean age of 59.05 ± 9.4 years, were included in the analysis. The characteristics of participants stratified by BRI quartiles are presented in Table 1. The results showed that various parameters such as BRI, BMI, WBC, FPG, TC, TG, LDL, HbA1c, hypertension increased significantly with increasing BRI values. In contrast, HDL, BUN, and serum creatinine (Scr) showed opposite trends.

Incidence rate of T2D

During a mean follow-up of 7.72 years, 1,653 incident T2D cases (15.33%) were documented (Table 2). The overall T2D incidence rate was 198.5 cases per 10,000 person-years. T2D incidence rates across BRI quartiles were as follows: Q1: 105.67/10,000 person-years; Q2: 136.04/10,000 person-years; Q3: 175.05/10,000 person-years; Q4: 248.07/10,000 person-years; Q5: 339.08/ 10,000 person-years. The incidence of T2D also varied significantly across quartiles: Q1: 8.30%; Q2: 10.80%; Q3: 13.68%; Q4: 18.95%; Q5: 24.91% (Fig. 2). Participants with lower BRI values had a significantly lower incidence of T2D compared to those with higher T2D (p for trend < 0.001).

Variables	Total (n = 10785)	Q1 (n = 2157)	Q2 (n = 2157)	Q3 (n = 2157)	Q4 (n = 2158)	Q5 (n = 2156)	P
BRI	4.16 ± 1.38	2.50 ± 0.36	3.32 ± 0.19	3.97 ± 0.20	4.76 ± 0.26	6.24 ± 1.00	< 0.001
Age	59.05 ± 9.41	58.47 ± 9.28	58.78 ± 9.29	58.45 ± 9.15	58.92 ± 9.40	60.64 ± 9.74	< 0.001
Gender							< 0.001
Female	5632 (52.22)	683 (12.13)	861 (15.29)	1122 (19.92)	1295 (22.99)	1671 (29.67)	
Male	5153 (47.78)	1474 (28.60)	1296 (25.15)	1035 (20.09)	863 (16.75)	485 (9.41)	
BMI	23.29 ± 3.86	19.75 ± 2.34	21.49 ± 2.40	23.00 ± 2.65	24.70 ± 2.80	27.49 ± 3.59	< 0.001
Smoking	3381 (31.35)	1051 (31.09)	493 (14.58)	650 (19.23)	875 (25.88)	312 (9.23)	< 0.001
Drinking	3577 (33.17)	920 (25.72)	715 (19.99)	865 (24.18)	451 (12.61)	626 (17.50)	< 0.001
Marry							< 0.001
Yes	9426 (87.40)	1901 (20.17)	1904 (20.20)	1882 (19.97)	1914 (20.31)	1825 (19.36)	
Others	1359 (12.60)	256 (18.84)	253 (18.62)	275 (20.24)	244 (17.95)	331 (24.36)	
Rural							< 0.001
Rural	7005 (64.95)	1593 (22.74)	1472 (21.01)	1381 (19.71)	1280 (18.27)	1279 (18.26)	
Urban	3780 (35.05)	564 (14.92)	685 (18.12)	776 (20.53)	878 (23.23)	877 (23.20)	
Sleep duration	6.36 ± 1.89	6.33 ± 1.90	6.37 ± 1.89	6.37 ± 1.86	6.34 ± 1.85	6.37 ± 1.94	> 0.05
PLT (10^9/L)	213.05 ± 76.93	209.08 ± 75.08	210.85 ± 74.98	213.17 ± 72.88	211.25 ± 70.64	220.57 ± 88.78	< 0.001
WBC (10^9/L)	6.22 ± 1.88	6.07 ± 1.91	6.16 ± 1.89	6.21 ± 1.90	6.22 ± 1.77	6.42 ± 1.90	< 0.001
BUN (mg/dl)	15.69 ± 4.45	16.19 ± 4.65	15.84 ± 4.55	15.66 ± 4.41	15.41 ± 4.25	15.37 ± 4.36	< 0.001
Scr (mg/dl)	0.78 ± 0.22	0.80 ± 0.17	0.79 ± 0.20	0.78 ± 0.19	0.78 ± 0.33	0.75 ± 0.17	< 0.001
CRP (mg/L)	2.46 ± 6.72	2.25 ± 7.16	2.67 ± 9.48	2.15 ± 5.45	2.42 ± 5.50	2.77 ± 5.03	> 0.05
UA (mg/dl)	4.45 ± 1.24	4.40 ± 1.22	4.38 ± 1.20	4.44 ± 1.27	4.52 ± 1.30	4.48 ± 1.19	< 0.05
FPG (mg/dl)	101.22 ± 15.08	98.89 ± 14.05	99.84 ± 15.82	100.94 ± 14.68	102.43 ± 14.65	103.89 ± 15.58	< 0.001
TC (mg/dl)	192.47 ± 37.54	184.20 ± 35.66	188.89 ± 36.06	191.94 ± 36.68	196.50 ± 37.82	200.36 ± 39.15	< 0.001
TG (mg/dl)	123.00 ± 79.60	95.99 ± 53.87	109.61 ± 73.38	120.29 ± 74.03	137.37 ± 87.73	150.23 ± 90.59	< 0.001
HDL (mg/dl)	52.01 ± 15.12	57.57 ± 15.88	54.93 ± 15.23	52.19 ± 14.94	48.66 ± 13.91	46.98 ± 12.99	< 0.001
LDL (mg/dl)	116.78 ± 34.06	109.24 ± 31.35	113.78 ± 32.30	116.63 ± 33.11	120.63 ± 34.80	123.22 ± 36.57	< 0.001
HbA1c (%)	5.10 ± 0.40	5.06 ± 0.39	5.05 ± 0.40	5.07 ± 0.39	5.13 ± 0.39	5.18 ± 0.41	< 0.001
Cys C (mg/l)	1.02 ± 0.27	1.05 ± 0.24	1.02 ± 0.24	1.00 ± 0.25	1.01 ± 0.37	1.02 ± 0.25	< 0.001
HGB (g/l)	14.37 ± 2.22	14.39 ± 2.11	14.34 ± 2.13	14.29 ± 2.21	14.46 ± 2.30	14.39 ± 2.32	> 0.05
Hypertension	2467 (22.87)	254 (11.78)	350 (16.23)	463 (21.46)	590 (27.34)	810 (37.57)	< 0.001
Lung Disease	1046 (9.70)	244 (11.31)	227 (10.52)	191 (8.85)	176 (8.16)	208 (9.65)	≤ 0.05
CLD	353 (3.27)	81 (3.76)	75 (3.48)	69 (3.20)	62 (2.87)	66 (3.06)	> 0.05
CKD	577 (5.35)	127 (5.89)	121 (5.61)	108 (5.01)	118 (5.47)	103 (4.78)	> 0.05
Asthma	493 (4.57)	102 (4.73)	107 (4.96)	87 (4.03)	84 (3.89)	113 (5.24)	> 0.05

Table 1. Baseline characteristics of individuals by BRI quartile. BRI: body roundness index; BMI: body mass index; PLT, platelet; WBC, white blood cell count ; BUN, blood urea nitrogen; Scr, serum creatinine; CRP: C-reactive protein; UA, Uric acid; FPG, fasting plasma glucose; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoproteins; HbA1c, hemoglobin A1c; Cys C: Cystatin C; HGB, hemoglobin concentration; CLD, Chronic liver diseases; CKD, Chronic kidney diseases; HR, hazard ratio; CI confidence, Q: quartile.

BRI	Participants (*n*)	Events (*n*)	Incidence rates (95%CI)(%)	Per 10,000 person-year
Total	10,785	1653	15.33 (14.65 ~ 16.02)	198.5
Q1 (< 2.98)	2157	179	8.30 (7.17 ~ 9.54)	105.67
Q2 (2.98 ~ 3.63)	2157	233	10.80 (9.52 ~ 12.19)	136.04
Q3 (3.64 ~ 4.31)	2157	295	13.68 (12.25 ~ 15.20)	175.05
Q4 (4.32 ~ 5.24)	2158	409	18.95 (17.32 ~ 20.67)	248.07
Q5 (> 5.25)	2156	537	24.91 (23.09 ~ 26.79)	339.08
P for trend			< 0.001	

Table 2. Incidence rate of type 2 diabetes (% or per 1000 person-year). BRI: body roundness index; CI confidence, Q: quartile.

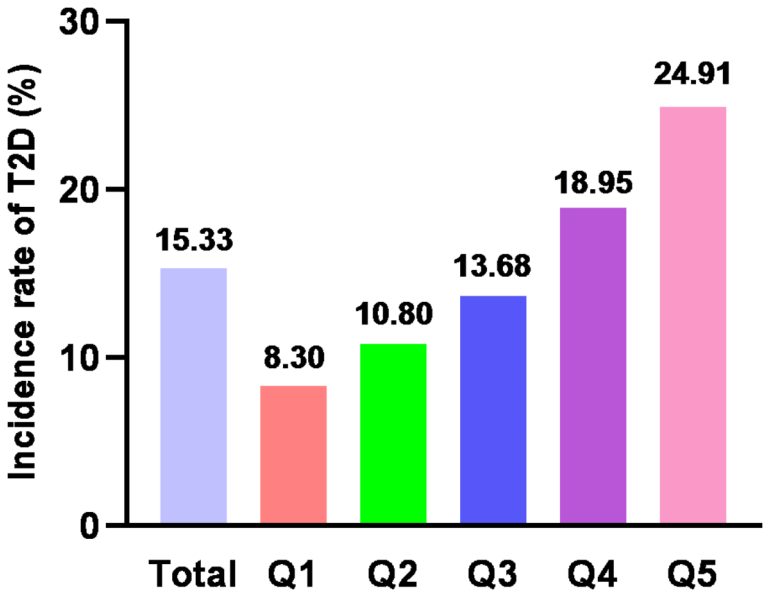

Fig. 2. Bar chart represents the incidence of T2D across different quartiles of BRI.

Factors influencing the risk of T2D analyzed by univariable Cox proportional hazards regression

Based on univariable analyses, the risk of T2D was not related to current smoking, current drinking, BUN, Scr, Cystatin C, HGB ($p > 0.05$), but was positively correlated with age, BMI, WBC, PLT, FPG, TC, TG, TyG, LDL, CRP, HbA1c, UA, Cystatin C, dyslipidemia, and hypertension, whereas it was negatively associated with HDL (all $p < 0.05$) (Supplementary file 1: Table S2).

Relationship between BRI and the risk of T2D

To examine the relationship between BRI and T2D risk, we developed three Cox proportional hazards regression models, as detailed in Table 3. In Model I, every 1-SD increase in BRI was associated with a 44% increase in T2D risk (HR: 1.44; 95% CI 1.38–1.50). Similarly, in Model II, every 1-SD increase in BRI corresponded to a 44% increase in T2D risk (HR: 1.44; 95% CI 1.39–1.50). In Model III, every 1-SD increase in BRI was linked to a 27% increase in T2D risk (HR: 1.27; 95% CI 1.20–1.35).

Additionally, we converted BRI from a continuous to a categorical variable based on quartiles. The multivariable-adjusted model showed that, with Q1 as the reference, HR for T2D risk were 1.08 (95% CI 0.86–1.37) for Q2, 1.40 (95% CI 1.12–1.76) for Q3, 1.70 (95% CI 1.37–2.12) for Q4 and 2.14 for Q5 (95% CI 1.71–2.66). This corresponds to an 8% increased risk in Q2, a 40% increased risk in Q3, a 70% increased risk in Q4 and a 140% increased risk in Q5 (Table 3).

Cox proportional hazards regression model with smooth curve fitting.

The RCS curve showed a nonlinear relationship between BRI and T2D risk ($p < 0.001$ for overall, $p < 0.05$ for nonlinear, Fig. 3). An inflection point for BRI was identified at 3.96. After identifying the inflection point, a two-piecewise Cox regression model was used to further explore the relationship on either side of it. The HR was 0.85 (95% CI 0.74–0.96, $p < 0.05$) before the inflection point and 1.29 (95% CI 1.18–1.41, $p < 0.001$) after it (Table 4).

Variables	Model I		Model II		Model III	
	HR (95%CI)	*P*	HR (95%CI)	*P*	HR (95%CI)	*P*
BRI						
Per 1-SD increase	1.44 (1.38 ~ 1.50)	< 0.001	1.44 (1.39 ~ 1.50)	< 0.001	1.27 (1.20 ~ 1.35)	< 0.001
Q1 (< 2.98)	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2 (2.98 ~ 3.63)	1.29 (1.06 ~ 1.57)	< 0.05	1.28 (1.05 ~ 1.56)	< 0.05	1.08 (0.86 ~ 1.37)	> 0.05
Q3 (3.64 ~ 4.31)	1.68 (1.40 ~ 2.03)	< 0.001	1.70 (1.41 ~ 2.05)	< 0.001	1.40 (1.12 ~ 1.76)	< 0.01
Q4 (4.32 ~ 5.24)	2.39 (2.00 ~ 2.86)	< 0.001	2.42 (2.02 ~ 2.90)	< 0.001	1.70 (1.37 ~ 2.12)	< 0.001
Q5 (> 5.25)	3.30 (2.76 ~ 3.93)	< 0.001	3.38 (2.83 ~ 4.04)	< 0.001	2.14 (1.71 ~ 2.66)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001

Table 3. Adjusted HR for the incidence of type 2diabetes across body roundness index quantiles. Abbreviations: BRI: body roundness index; HR: hazard ratio; Q: quartile. Model I: Adjusted for age and gender. Model II: Adjusted for age, gender, education, drinking, smoking, sleep duration, physical activity. Model III: Adjusted for age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c.

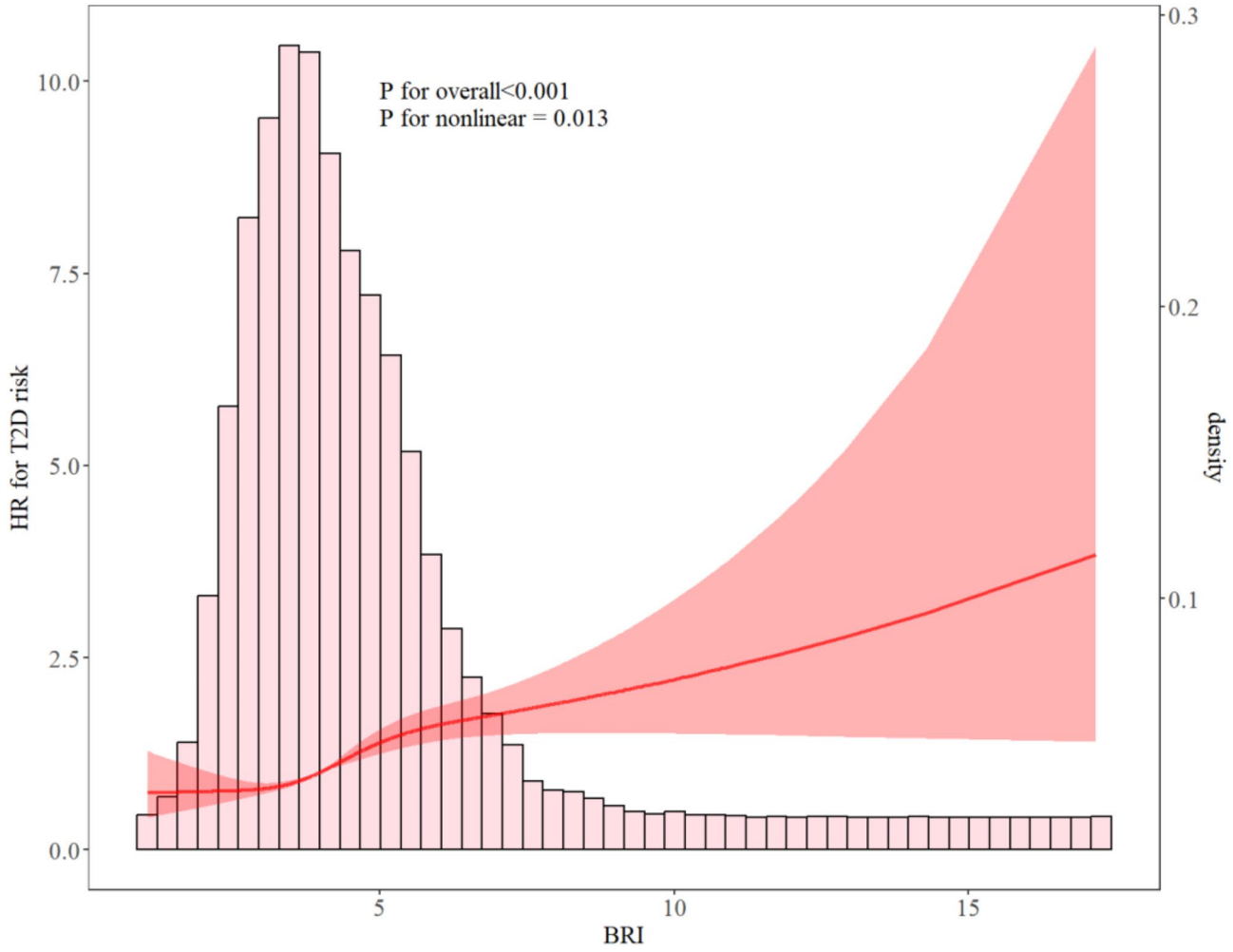

Fig. 3. Curve plot was adjusted for age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c.

Outcome: incident T2D	HR (95%CI)	*P*
Fitting model by standard linear regression	1.40 (1.14 ~ 1.25)	< 0.001
Inflection points of BRI		
< 3.96	0.85 (0.74 ~ 0.96)	< 0.05
≥ 3.96	1.29 (1.18 ~ 1.41)	< 0.001
P for likelihood ratio test		< 0.001

Table 4. Results of two-piecewise linear regression model. Adjusted for age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c.

Variables	Model I		Model II		Model III	
	HR (95%CI)	*P*	HR (95%CI)	*P*	HR (95%CI)	*P*
BRI						
Per 1-SD increase	1.24 (1.17 ~ 1.32)	< 0.001	1.21 (1.10 ~ 1.32)		1.25 (1.18 ~ 1.33)	< 0.001
Q1 (< 2.98)	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q2 (2.98 ~ 3.63)	1.05 (0.83 ~ 1.33)	> 0.05	1.08 (0.84 ~ 1.38)	> 0.05	1.06 (0.84 ~ 1.35)	> 0.05
Q3 (3.64 ~ 4.31)	1.37 (1.10 ~ 1.72)	< 0.01	1.46 (1.14 ~ 1.87)	< 0.01	1.32 (1.04 ~ 1.66)	< 0.05
Q4 (4.32 ~ 5.24)	1.63 (1.31 ~ 2.03)	< 0.001	1.71 (1.28 ~ 2.28)	< 0.001	1.67 (1.33 ~ 2.09)	< 0.001
Q5 (> 5.25)	1.98 (1.59 ~ 2.48)	< 0.001	1.86 (1.24 ~ 2.80)	< 0.01	2.04 (1.62 ~ 2.56)	< 0.01
P for trend		< 0.001		< 0.001		< 0.001

Table 5. Relationship between BRI and the risk of hypertension in different sensitive analyses. Abbreviations: BRI: body roundness index; HR: hazard ratio; Q: quartile. Model I was a sensitivity analysis that incorporates chronic conditions. Adjusted age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c, hypertension, cancer, chronic lung disease, chronic liver diseases, chronic kidney diseases, and cardiovascular diseases. Model II was a sensitivity analysis conducted on participants with BMI < 24 kg/m^2. Adjusted age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c. Model III was a sensitivity analysis that excludes participants diagnosed with T2D during the second follow-up. Adjusted age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c.

Sensitivity analysis

This study conducted three sensitive analyses to verify the strength and consistency of the relationship between BRI and the risk of T2D by focusing on specific groups and adjusting for various health factors (Table 5). Firstly, in model I of the sensitivity analysis, chronic diseases were included as confounders. This analysis still showed a significant positive association between BRI and T2D risk (every 1-SD increase, HR: 1.24, 95% CI 1.17–1.32, $p < 0.001$). Secondly, the analysis was narrowed down to participants with a BMI under 24 kg/m^2 ($n = 6,673$). The analysis revealed a significant positive association between the BRI and the risk of T2D, with a HR of 1.21 (95% CI 1.10–1.32) every 1-SD increase ($p < 0.001$). Finally, we excluded participants who were diagnosed with T2D at the second follow-up ($n = 10,659$), and the results remained robust (HR:1.25, 95% CI 1.18–1.33).

Results of subgroup analysis

The link between BRI and T2D risk was not affected by age, gender, smoking status, drinking status, marital status, education level, residence, and exercise in any of the prespecified or exploratory subgroups examined (Fig. 4). That is to say, the interaction between these variables and BRI was not statistically significant ($p > 0.05$ for interaction, Supplementary file 1: Table S3).

Discussion

This study aimed to explore the association between BRI and the risk of incident T2D among adults aged 45 years and older in a nationally representative China population sample from 2011 to 2020. Our results demonstrate that BRI, a relatively novel metric of body fat distribution, is significantly associated with an increased risk of T2D, with higher BRI values correlating with a higher incidence of T2D over an average follow-up period of 7.72 years. Additionally, an inflection point was identified, and different relationships between BRI and T2D risk were detected on both sides. These findings provide further evidence supporting the utility of BRI in assessing obesity-related metabolic risks and highlight its potential as an effective predictor of T2D risk in middle-aged and elderly populations.

Abdominal obesity, particularly the accumulation of visceral fat, has been widely recognized as a significant risk factor for the development of T2D[29]. It is noteworthy that although the prevalence of obesity is relatively lower

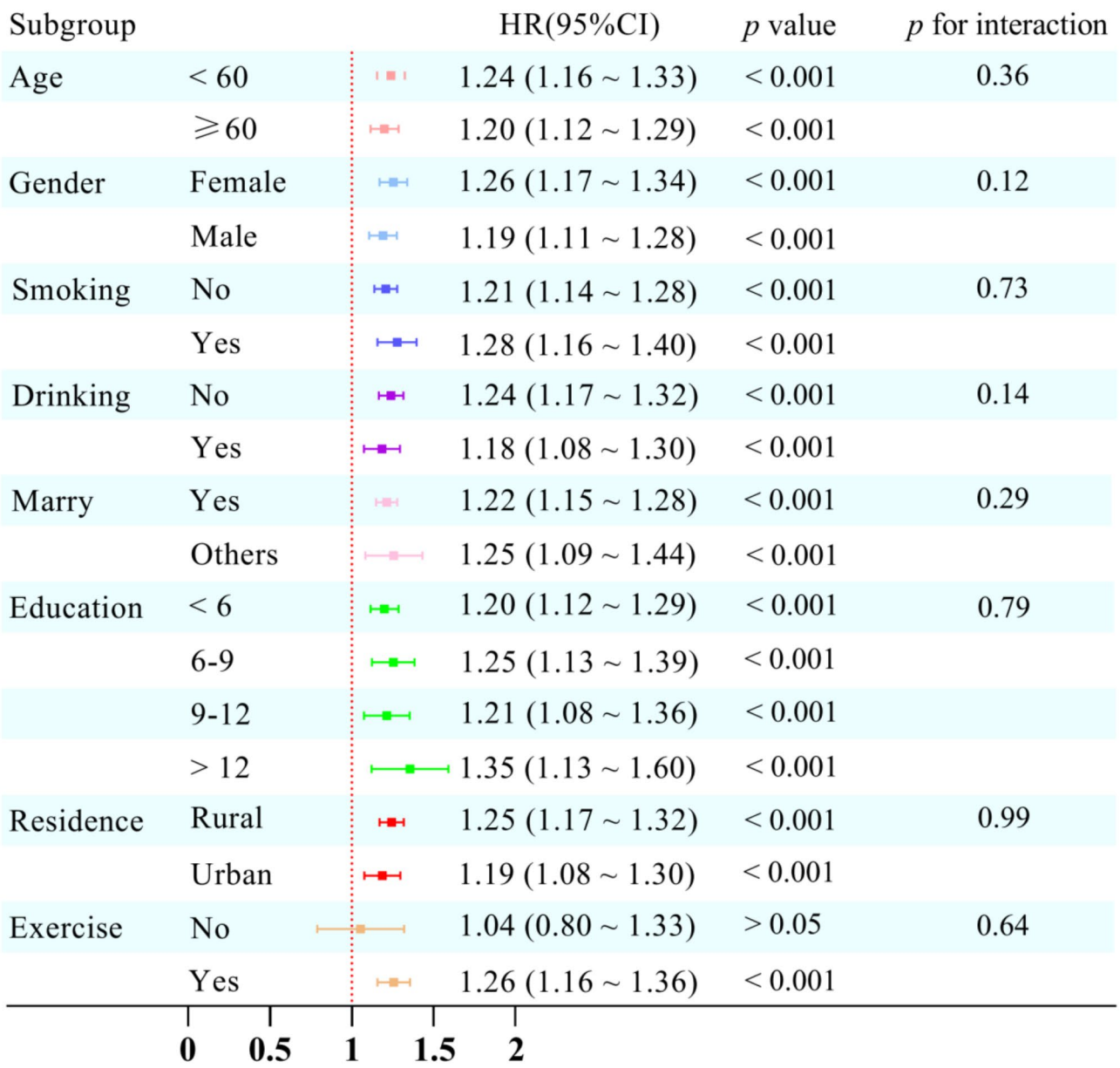

Fig. 4. Stratified associations between BRI and T2D. Adjusted age, gender, education, drinking, smoking, sleep duration, physical activity, dyslipidemia, fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, HbA1c.

in the Chinese population compared to Western countries, the distribution of body fat tends to favor abdominal accumulation, leading to a higher risk of abdominal obesity[30]. A survey of Chinese adults with T2D found that 39.7% of T2D patients had abdominal obesity, defined as a visceral fat area ≥ 100 cm^2. Among overweight individuals, 49.8% had abdominal obesity, while 83.1% of obese individuals exhibited abdominal obesity. Even among individuals with normal body weight, 11.5% had abdominal obesity[31]. Albrecht et al.[32] examined secular trends in WC across the USA (1988–2007), England (1992–2008), China (1993–2011), and Mexico (1999–2012) and reported statistically significant increases in WC relative to BMI in all the countries studied, as well as in most subpopulations. These findings are consistent with those of Tommy Visscher et al.[33], who conducted an extensive review and concluded that most of the evidence indicates a greater relative increase in WC compared to BMI. The inability of BMI to detect increases in abdominal obesity underscores its limitations in identifying the obesity phenotype that poses the greatest health risk[17]. However, a major limitation of WC is its failure to account for height, which may lead to either underestimation or overestimation of abdominal obesity in individuals of short or tall stature[34]. Accruing evidence suggests that BRI, a newer anthropometric measure, more comprehensively reflects abdominal fat distribution and visceral fat compared to conventional measures such as BMI. Theoretically, by modeling the body shape as an ellipse with height as the long axis and waist circumference as the short axis, BRI can be calculated as the ellipse's eccentricity[18]. Therefore, it is reasonable to hypothesize that BRI is a superior anthropometric measure for assessing abdominal adiposity[21].

Unfortunately, as an emerging therapeutic concept, while the idea that BRI can estimate total and regional fat percentages may be reasonable and appealing, evidence linking BRI to disease or mortality is limited. In a cohort study involving 32,995 US adults, conducted from 1999 to 2018, a U-shaped relationship was observed between BRI and all-cause mortality, with significantly increased risks of mortality in both the lowest and highest BRI groups[21]. Studies on BRI trajectories by Wu et al.[22], Yang et al.[35], and Ding et al.[36] found that higher BRI values were associated with an increased risk of CVD over time. A study by Zhang et al.[37] reported that BRI levels were positively related to an increased prevalence of depression in American adults. A study by Liu et al.[38] followed 6,990 hypertensive adults without diabetes for 3 years and found that BRI was superior to other anthropometric measures in predicting the onset of diabetes. Our conclusions are consistent with these findings. The results of the present study indicate a clear dose-response relationship between BRI and the incidence of T2D. Specifically, participants in the highest quartile of BRI (Q5) had a significantly higher incidence rate of T2D (339.08 cases per 10,000 person-years) compared to those in the lowest quartile (Q1) (105.67 cases per 10,000 person-years). The multivariable adjusted Cox regression model further confirmed these findings, revealing that each standard deviation increase in BRI was associated with a 44% increase in the risk of T2D (Model I). The magnitude of risk estimation (25%) persisted even after fully adjusting covariates (Model III). The sensitivity analyses further confirmed that these relationships persisted in this group of participants. This is consistent with the current understanding that obesity—particularly abdominal or visceral fat—is a major risk factor for the development of IR[39] and subsequent T2D[40].

Interestingly, our study identified a novel inflection point at 3.96 in the relationship between BRI and incident T2D, which has not been previously reported in the literature. While prior studies have explored associations between anthropometric indices (e.g., BMI, waist-to-height ratio) and T2D risk[41] none have specifically examined BRI in this context. When BRI was greater than 3.96, the risk of T2D increased by 29% for every 1-SD increase in BRI. On the other hand, when BRI was less than 3.96, the risk of T2D decreased by 15% for every 1-SD decrease in BRI. This suggests that the association between BRI and T2D risk may become more pronounced beyond a certain threshold, which may be indicative of the point at which fat accumulation begins to have a more detrimental effect on metabolic health. After the inflection point, the HR for T2D risk increased significantly (HR: 1.29, 95% CI 1.18–1.41). The significant increase in HR following the inflection point further underscores the importance of monitoring BRI as an early indicator for individuals at high risk for T2D. Mechanistically, excessive visceral fat accumulation disrupts adipose tissue homeostasis, leading to increased secretion of pro-inflammatory cytokine and reduced adiponectin levels, which impair insulin signaling pathways and promote β-cell dysfunction[42]. Furthermore, adipose tissue hypoxia and macrophage infiltration, triggered by adipocyte hypertrophy beyond this threshold, may exacerbate systemic inflammation and ectopic lipid deposition[43]. The ethnic specificity of this threshold aligns with evidence that Asian populations develop T2D onset at lower BMI levels than Caucasians, likely due to genetic and epigenetic differences in fat distribution and insulin sensitivity[44].

The lack of significant interactions between BRI and age, sex, or smoking status in subgroup analyses suggests that the positive association between BRI and T2D risk is robust across diverse demographic and behavioral subgroups. First, studies have shown that excess visceral fat similarly disrupts glucose homeostasis in both sexes and across age groups, as its pro-inflammatory and lipotoxic effects are largely independent of sex hormones or age-related metabolic changes[12]. Second, the homogeneity of our study population (middle-aged and older Chinese adults) may limit the detection of age-specific effects, as age-related declines in β-cell function could uniformly exacerbate T2D risk in all participants[45]. Finally, the absence of interaction with smoking status aligns with evidence that smoking primarily influences T2D risk through pathways unrelated to adiposity distribution (e.g., oxidative stress and pancreatic β-cell apoptosis)[46], leaving the BRI-T2D association unmodified. These observations reinforce BRI's potential as a universal risk stratification tool.

In summary, BRI offers a simple, cost-effective, and easily accessible method to assess visceral adiposity, which has been shown to be a strong predictor of T2D risk. Incorporating BRI into routine clinical assessments could help identify individuals at higher risk for T2D, particularly in resource-limited settings where more expensive imaging techniques (e.g., CT or MRI) are not feasible. Furthermore, BRI could complement existing strategies for lifestyle interventions, such as physical activity promotion and dietary modifications, by providing a more direct measure of visceral fat accumulation. This could help tailor more individualized prevention programs, especially in populations where traditional BMI measurements may not fully capture the risk of metabolic diseases.

Several strengths of the present study should be highlighted. First, this is the first investigation of the relationship between BRI and the onset of T2D in a nationally representative China population sample. Second, our study included a large and diverse sample of middle-aged and elderly individuals from across China, enhancing the reliability of our findings. Third, both categorical and continuous BRI were used as independent variables to evaluate their association with T2D risk. This approach minimized information loss and allowed for a more precise quantification of the relationship. Finally, the large sample size and extended follow-up period enabled a thorough investigation of the relationship between BRI and T2D progression. Besides the strengths of this study, some limitations should be acknowledged. First, the CHARLS dataset is specific to the Chinese population, raising questions about the applicability of our findings to different cultural or ethnic groups. Second, the CHARLS database is observational in nature, meaning that causality cannot be definitively established. Third, although the study adjusted for a range of potential confounders, residual confounding due to unmeasured factors may still exist. Fourth, our study may be subject to selection bias due to the exclusion of participants who were lost to follow-up and those with missing data after the baseline survey (Supplementary file 1: Table S4 and S5). Although excluding these individuals was methodologically necessary, we observed that the excluded group ($n = 6923$) differed from the analytic sample ($n = 10785$) in certain baseline characteristics. If these individuals had unmeasured risk factors (e.g., undiagnosed prediabetes or differential healthcare access) that influenced their likelihood of both study dropout and diabetes incidence, our estimates of the BRI-diabetes

association could be biased. Additionally, we tested the proportionality assumption using Schoenfeld residuals; in some instances, the assumption was violated, we therefore interpreted the HR as weighted averages of the time-varying HR over the entire follow-up period[47–49]. Future research should aim to further validate the use of BRI in predicting T2D risk in different populations and settings, including studies that incorporate direct measures of fat distribution. Longitudinal studies with repeated measurements of BRI over time could also help to better understand the temporal relationship between changes in BRI and the development of T2D. Additionally, intervention trials targeting abdominal obesity and its effects on T2D risk would provide valuable insights into the potential of BRI as a modifiable risk factor.

Conclusion

The present study demonstrates a significant association between BRI and the incidence of T2D in a middle-aged and elderly population. The findings suggest that BRI may serve as an important tool for identifying individuals at higher risk for T2D, particularly in populations with central obesity. Given its simplicity and cost-effectiveness, BRI could be a valuable addition to current clinical and public health strategies aimed at reducing the burden of T2D.

Data availability

The data utilized in this study can be accessed from the China Health and Retirement Longitudinal Study (CHARLS) database. Researchers interested in using the data can obtain it by submitting an application through the CHARLS official website: http://charls.pku.edu.cn/. Any additional information required to reanalyze the data reported in this paper is available from the first author.

Received: 13 January 2025; Accepted: 3 March 2025
Published online: 16 April 2025

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Acknowledgements

We extend our sincere gratitude to the staff of CHARLS for their invaluable efforts in data collection and participant follow-up. We are also deeply grateful to all the participants of the CHARLS survey. Their willingness to contribute their time and personal information has made this research possible.

Author contributions

Conceptualization, Zigui Zhou and Jingjing Liu; Data curation, Zigui Zhou and Jingjing Liu; Methodology, Zigui Zhou and Jingjing Liu; Resources, Zigui Zhou and Jingjing Liu; Supervision, Jingjing Liu; Writing – original draft, Zigui Zhou; Writing – review & editing, Jingjing Liu.

Funding

The study was conducted without any financial support; it did not receive funding from any grants, institutions, or other sources.

Declarations

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

CHARLS is committed to the highest ethical standards and received ethical approval from the Institutional Review Board of Peking University (IRB00001052- 11015). All participants provided informed consent at the baseline assessment.

Consent for publication

Not applicable.

Additional information

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-025-92652-y.

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