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The association between body roundness index and mortality in diabetes

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Abstract

Background The association between body roundness index (BRI) and all-cause mortality and the occurrence of cardiovascular disease (CVD) in patients with diabetes is unknown. This study aimed to determine the association between BRI and all-cause/CVD mortality in a diabetic cohort.

Methods A total of 8227 individuals with diabetes from the 1999–2018 National Health and Nutrition Examination Survey (NHANES) database were included. Multifactorial Cox regression models were used to analyze the association between BRI and mortality in patients with diabetes. Multivariate-adjusted restricted cubic spline (RCS) regression was used to test for nonlinearity.

Results During a median follow-up of 7.25 years, a total of 27.22% died, with 9.18% of these deaths due to CVD. After fully adjusting for potential confounders, BRI remained significantly associated with all-cause/CVD mortality in the diabetic population. The restricted cubic splines revealed no significant nonlinear relationship between BRI and all-cause mortality ($P=0.29$) or CVD mortality ($P=0.73$). BRI was better associated with all-cause/CVD mortality in patients with diabetes compared to other body metabolic indices.

Conclusions In patients with diabetes, we found an association between BRI and all-cause/CVD mortality.

Keywords Body roundness index, Diabetes, All-cause mortality, Cardiovascular disease

Background

Diabetes is becoming a significant worldwide health concern. 4.2 million fatalities worldwide were attributed to diabetes in 2019; by 2045, 700 million people are predicted to have the disease [1]. Cardiovascular disease (CVD) afflict over 32.2% of Type 2 diabetes mellitus (T2DM) patients worldwide, and CVD is the main cause of mortality for T2DM patients, accounting for roughly half of all fatalities [2]. Because of this, diabetes has a significant role in the worldwide burden of CVD.

The Body Roundness Index (BRI), introduced by Thomas et al. in 2013, is a useful tool for determining body fat and visceral fat percentages since it can be computed using height and waist circumference [3]. BRI is a better predictor of the risk of metabolic diseases than body mass index (BMI), particularly in individuals with normal BMI but uneven body fat distribution. It is more strongly correlated with metabolic diseases such as nonalcoholic fatty liver disease, diabetes, metabolic syndrome, and hypertension [4–7]. Additionally, BRI is crucial in predicting the course of a disease. Several studies have demonstrated a U-shaped relationship between

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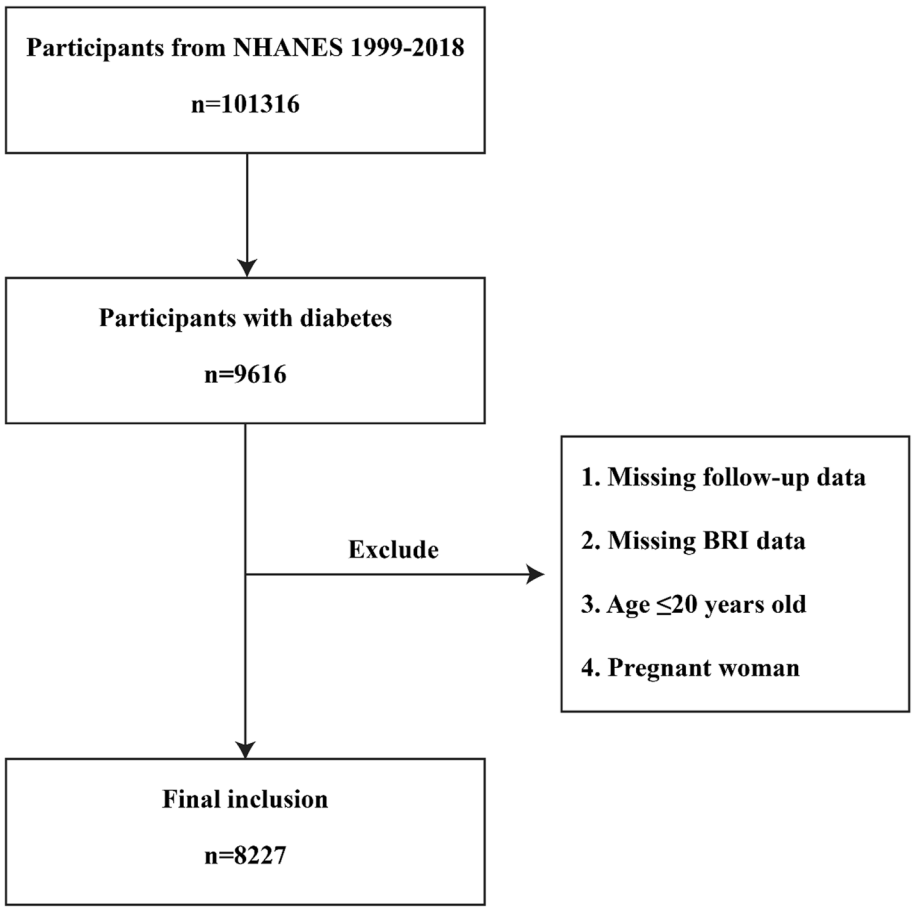

Fig. 1 The flow chart of this study

BRI and overall mortality in the general population [8, 9]. However, the effect of BRI on overall mortality in certain communities has received less research. As a follow-up to earlier correlation studies, the current study looked at the relationship between BRI and all-cause/CVD mortality in a diabetic population.

Methods

Study population

The population data were obtained from the National Health and Nutrition Examination Survey (NHANES). This database, conducted by the National Center for Health Statistics (NCHS), is a nationally representative cross-sectional study designed to assess the health and nutritional status of adults and children in the United States. All participants provided written informed consent, and the protocol was approved by the NCHS Ethics Review Board. Our study flow is illustrated in Fig. 1. From the 1999–2018 NHANES database spanning 10 cycles, we selected 9616 individuals with diabetes. After excluding those with missing follow-up data, missing BRI data, those aged ≤ 20 years, and pregnant women, 8227 participants were finally included.

Identification of diabetes

According to the American Diabetes Association (ADA) guidelines, participants were defined as having diabetes if they met any of the following criteria: self-reported diagnosis, use of oral hypoglycemic agents or insulin, fasting blood glucose (FBG) ≥ 7.0 mmol/L, random blood glucose ≥ 11.1 mmol/L, glycated hemoglobin (HbA1c) ≥ 6.5%, or an oral glucose tolerance test 2-h (OGTT 2 h) blood glucose ≥ 11.1 mmol/L.

Identification of hypertension and hyperlipidemia

Determination of hypertension was based on any of the following three criteria: 1) The participant was diagnosed with hypertension by a physician. 2) The participant was taking antihypertensive medication. 3) The participant's mean systolic blood pressure (SBP) was ≥ 140 mmHg or mean diastolic blood pressure (DBP) was ≥ 90 mmHg. Hyperlipidemia was determined based on either of the following two criteria: 1) Elevated lipid levels, such as

elevated total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), or decreased high-density lipoprotein cholesterol (HDL-C). 2) Participants self-reported that they were using lipid-lowering medications.

Calculation of BRI and other metabolic indices

Waist circumference and height data were obtained from the questionnaire, while HDL-C, triglycerides (TG), and FBG were sourced from laboratory tests. The formulas for calculating BRI [9], BMI [10],atherogenic index of plasma (AIP) [11], lipid accumulation product (LAP) [12], visceral adiposity index (VAI) [13], triglyceride-glucose index (TyG) [14], and cardiometabolic index (CMI) [15] are as follows:

$$\mathrm{BRI} = 364.2 - 365.5 \times \sqrt{1 - \left(\frac{\mathrm{WC(cm)}/2\pi}{0.5 \times \mathrm{Height\ (cm)}}\right)^2} \quad (1)$$

Waist Circumference (cm): Reflects abdominal fat accumulation and serves as a key indicator of central obesity.

Height (m): Normalizes the proportion of waist circumference to avoid bias caused by height differences.

Waist Circumference/(2π×Height): Simulates the hypothetical cross-sectional radius of the human torso under the assumption of a cylindrical shape.

Square Root: Applied to linearize nonlinear relationships and adjust distribution.

Constants 364.2 and 365.5: Calibrated through statistical methods to standardize the BRI range (0–12), facilitating health risk stratification and ensuring a linear association between BRI and disease risk.

$$\mathrm{BMI} = \frac{\mathrm{Weight\ (kg)}}{\mathrm{Height\ (cm)}^2} \quad (2)$$

Weight (kg): The numerator in BMI calculation, representing absolute body mass.

Height Squared (m^2): Adjusts the proportionality between weight and height. Weight correlates with body volume (a three-dimensional measure), while height is a linear measurement (one-dimensional). Squaring height allows BMI to better reflect the ratio of weight to body volume.

Division Operation: Dividing weight by height squared yields a dimensionless value to assess whether body weight falls within a healthy range.

$$\mathrm{AIP} = \log\left(\frac{\mathrm{TG\ (mmol/L)}}{\mathrm{HDL\ (mmol/L)}}\right) \quad (3)$$

Triglycerides (TG, mmol/L): Plasma triglyceride concentration, reflecting lipid metabolism status.

High-Density Lipoprotein Cholesterol (HDL-C, mmol/L): Plasma HDL-C concentration.

Logarithmic Operation ($\log_{10}$): Converts the TG/HDL-C ratio into a more interpretable value. Higher AIP values indicate greater risks of atherosclerosis and cardiovascular diseases.

$$\begin{aligned}\text{Male}\quad \mathrm{LAP} &= (\mathrm{WC\ (cm)} - 65) \times \mathrm{TG\ (mmol/L)}\\ \text{Female}\ \mathrm{LAP} &= (\mathrm{WC\ (cm)} - 58) \times \mathrm{TG\ (mmol/L)}\end{aligned} \quad (4)$$

Waist Circumference (cm): Reflects abdominal fat accumulation and central obesity.

Gender-Specific Constants (Male: 65, Female: 58): Statistically calibrated parameters to differentiate baseline waist circumference thresholds between genders, based on population studies, to more accurately reflect visceral fat thresholds.

Triglycerides (TG, mmol/L): Plasma triglyceride concentration, reflecting lipid metabolism status.

Multiplicative Relationship: LAP combines anthropometric (fat distribution) and biochemical (lipid metabolism) indicators via the product of waist circumference and TG, providing a comprehensive assessment of visceral fat accumulation and metabolic risk.

$$\begin{aligned}\text{Male}\quad \mathrm{VAI} &= \frac{\mathrm{WC\ (cm)}}{39.68+(1.88\times\mathrm{BMI\ (kg/m^2)})} \times \frac{\mathrm{TG\ (mmol/L)}}{1.03} \times \frac{1.31}{\mathrm{HDL\ (mmol/L)}}\\ \text{Female}\ \mathrm{VAI} &= \frac{\mathrm{WC\ (cm)}}{36.58+(1.89\times\mathrm{BMI\ (kg/m^2)})} \times \frac{\mathrm{TG\ (mmol/L)}}{0.81} \times \frac{1.52}{\mathrm{HDL\ (mmol/L)}}\end{aligned} \quad (5)$$

Waist Circumference (cm): Reflects abdominal fat accumulation and central obesity.

Body Mass Index (BMI): Measures overall obesity.

Triglycerides (TG, mmol/L): Plasma triglyceride concentration.

High-Density Lipoprotein Cholesterol (HDL-C, mmol/L): Plasma HDL-C concentration.

Gender-Specific Constants (e.g., 39.68, 1.88, 1.03, 1.31): Derived from large-scale population studies and statistical analyses to optimize the assessment of visceral adipose function and metabolic risk. Distinct parameters for males and females account for gender differences in fat distribution and metabolic profiles.

$$\mathrm{TyG} = \ln\left(\frac{\mathrm{TG\ (mg/dL)} \times \mathrm{FPG\ (mg/dL)}}{2}\right) \quad (6)$$

Triglycerides (TG, mmol/L): Plasma triglyceride concentration.

Fasting Plasma Glucose (FPG, mg/dL): Blood glucose concentration under fasting conditions, reflecting glucose metabolism.

Natural Logarithm (ln): Log-transforms the product of TG and FPG to approximate a normal distribution for statistical analysis.

Division 2: Adjusts the scale for easier interpretation and comparison.

$$\text{CMI} = \frac{\text{WC (cm)}}{\text{Height (cm)}} \times \frac{\text{TG (mmol/L)}}{\text{HDL (mmol/L)}} \quad (7)$$

Waist-to-Height Ratio (WHtR): Calculated as waist circumference (cm) divided by height (cm). Evaluates abdominal obesity; higher WHtR correlates with increased cardiovascular risk.

Triglycerides (TG, mmol/L): Plasma triglyceride concentration.

High-Density Lipoprotein Cholesterol (HDL-C, mmol/L): Plasma HDL-C concentration.

Determination of mortality

Mortality was determined using data from the National Death Index (NDI) up to December 31, 2019, which is linked to the NHANES dataset. Cause-specific mortality was identified using the 10th revision of the International Classification of Diseases (ICD-10). CVD mortality was defined by ICD-10 codes I00-I09, I11, I13, and I20-I51.

Covariate assessment

We included demographic information, lifestyle factors, and health status as covariates. Race was categorized as Mexican American, non-Hispanic white, non-Hispanic black, and other. Education level was classified as less than high school, high school or equivalent, and college or above. Family poverty ratio (PIR) was divided into <1.30, 1.30–3.49, and ≥ 3.50. BMI was grouped as <25.0 kg/m^2, 25.0–29.9 kg/m^2, and ≥ 30.0 kg/m^2. Individuals with a lifetime smoking history of <100 cigarettes were categorized as never smokers, those with ≥ 100 cigarettes but quit were past smokers, and those currently smoking were current smokers. Alcohol consumption was classified as non-drinkers, light/moderate drinkers, and heavy drinkers. Physical activity was categorized based on metabolic equivalent (MET) minutes per week, with <600 min/week as inactive and ≥ 600 min/week as active. The duration of diabetes was calculated as current age minus the age at first diagnosis. Treatment was defined by the use of insulin or oral hypoglycemic agents. Comorbidities such as hypertension, hyperlipidemia, history of CVD, and cancer were obtained from questionnaire data, with CVD including congestive heart failure, coronary heart disease, angina, heart attack, and stroke.

Statistical analysis

Due to the complex sampling design of NHANES, all analyses incorporated sample weights, clustering, and stratification. Continuous variables were presented as mean and standard deviation (SD), and categorical variables as frequency and percentage. BRI was modeled as both a continuous variable and a categorical variable (quartiles). We used three Cox proportional hazards regression models to analyze the relationship between BRI and mortality in patients with diabetes. The effect sizes were estimated as hazard ratios (HR) with corresponding 95% confidence intervals (CI). Model 1 was unadjusted, Model 2 adjusted for age, sex, and race, and Model 3 adjusted for all covariates.

To explore the relationship between BRI and mortality in patients with diabetes, we employed multivariable-adjusted restricted cubic spline (RCS) regression to test for nonlinearity. Subgroup analyses were conducted by sex, age, BMI, diabetes duration, treatment status, hypertension, hyperlipidemia, and CVD history to further clarify the association. Additionally, sensitivity analyses excluded participants who died within the first two years of follow-up to rule out potential reverse causality. In this study, a two-sided p-value <0.05 was considered statistically significant, and all analyses were performed using R version 4.4.1.

Results

Baseline characteristics of study participants

A total of 8,227 patients with diabetes were included in this study, with a mean age of 61.24 ± 13.44 years and 51.49% male. During a median follow-up of 7.25 years, 27.22% of participants died from all causes, and 9.18% died from CVD. Higher mortality was observed among males, non-Hispanic whites, individuals with less than high school education, current smokers, active individuals, those with a diabetes duration of ≥ 10 years, and those with a history of CVD. Detailed baseline characteristics are presented in Tables 1 and 2.

Association between BRI and mortality in diabetes

As shown in Table 3, in Model 2, higher BRI was associated with increased all-cause mortality (HR = 1.04, 95% CI = 1.01–1.07) and CVD mortality (HR = 1.06, 95% CI = 1.00–1.11). After adjusting for all covariates in Model 3, the association between BRI and all-cause mortality (HR = 1.16, 95% CI = 1.07–1.27) and CVD mortality (HR = 1.27, 95% CI = 1.13–1.42) became more significant. Compared to the lowest quartile of BRI, the highest quartile was strongly associated with increased all-cause mortality (HR = 2.52, 95% CI = 1.27–5.00) and CVD

Table 1 The baseline characteristics of all-cause mortality in diabetes

		All-cause mortality		
	All	**Alive**	**Death**	***P*-value**
	***N* = 8227**	***N* = 5988 (72.78%)**	***N* = 2239 (27.22%)**	
BRI	6.86 (2.40)	6.93 (2.43)	6.68 (2.32)	< 0.001
Gender				< 0.001
Male	4236 (51.49%)	2969 (49.58%)	1267 (56.59%)	
Female	3991 (48.51%)	3019 (50.42%)	972 (43.41%)	
Age (years old)	61.24 (13.44)	58.22 (13.07)	69.31 (10.86)	< 0.001
Race				< 0.001
Mexican American	1663 (20.21%)	1280 (21.38%)	383 (17.11%)	
Non-Hispanic White	2966 (36.05%)	1863 (31.11%)	1103 (49.26%)	
Non-Hispanic Black	2056 (24.99%)	1513 (25.27%)	543 (24.25%)	
Other Race	1542 (18.74%)	1332 (22.24%)	210 (9.38%)	
Education				< 0.001
Less than high school	3028 (36.86%)	1991 (33.28%)	1037 (46.44%)	
High school or equivalent	1889 (22.99%)	1364 (22.80%)	525 (23.51%)	
College or above	3298 (40.15%)	2627 (43.92%)	671 (30.05%)	
PIR				< 0.001
< 1.30	2601 (34.97%)	1827 (33.82%)	774 (38.03%)	
1.30–3.49	3036 (40.82%)	2126 (39.36%)	910 (44.72%)	
≥ 3.50	1800 (24.20%)	1449 (26.82%)	351 (17.25%)	
BMI (kg/m^2)				< 0.001
< 25.0	1178 (14.37%)	752 (12.58%)	426 (19.20%)	
25.0–29.9	2514 (30.67%)	1742 (29.15%)	772 (34.79%)	
≥ 30.0	4504 (54.95%)	3483 (58.27%)	1021 (46.01%)	
Smoke				< 0.001
Never smoker	4071 (49.52%)	3168 (52.96%)	903 (40.33%)	
Ever smoker	1373 (16.70%)	971 (16.23%)	402 (17.95%)	
Current smoker	2777 (33.78%)	1843 (30.81%)	934 (41.72%)	
Drinking				< 0.001
Nondrinker	3097 (48.43%)	2273 (47.01%)	824 (52.82%)	
Low-to-moderate drinker	1790 (27.99%)	1344 (27.80%)	446 (28.59%)	
Heavy drinker	1508 (23.58%)	1218 (25.19%)	290 (18.59%)	
Physical activity				< 0.001
Inactivity	1606 (33.90%)	1176 (30.79%)	430 (46.84%)	
activity	3132 (66.10%)	2644 (69.21%)	488 (53.16%)	
Duration (years)				< 0.001
< 10	2932 (51.89%)	2254 (55.82%)	678 (42.06%)	
≥ 10	2718 (48.11%)	1784 (44.18%)	934 (57.94%)	
Medication				< 0.001
No	4437 (80.37%)	3200 (78.43%)	1237 (85.84%)	
Yes	1084 (19.63%)	880 (21.57%)	204 (14.16%)	
Hypertension				< 0.001
No	2938 (35.82%)	2293 (38.38%)	645 (28.95%)	
Yes	5265 (64.18%)	3682 (61.62%)	1583 (71.05%)	
Hypercholesteremia				0.405
No	3128 (41.61%)	2288 (41.31%)	840 (42.42%)	
Yes	4390 (58.39%)	3250 (58.69%)	1140 (57.58%)	
CVD				< 0.001
No	6130 (75.17%)	4839 (81.41%)	1291 (58.39%)	

Table 1 (continued)

		All-cause mortality		
	All	**Alive**	**Death**	***P*-value**
	***N* = 8227**	***N* = 5988 (72.78%)**	***N* = 2239 (27.22%)**	
Yes	2025 (24.83%)	1105 (18.59%)	920 (41.61%)	
Cancer				< 0.001
No	7084 (86.25%)	5307 (88.78%)	1777 (79.51%)	
Yes	1129 (13.75%)	671 (11.22%)	458 (20.49%)	

mortality (HR = 6.04, 95% CI = 2.08–17.58) after multivariable adjustment. We further performed sensitivity analyses on Model 3 and showed that the highest quartile of BRI was strongly associated with increased all-cause mortality (HR = 2.72, 95% CI = 1.27-5.83) and CVD mortality (HR = 6.62, 95% CI = 2.01-21.82) (Table 4).

To further elucidate the relationship between BRI and mortality in patients with diabetes, we used RCS to analyze the nonlinear relationship. As shown in Fig. 2, there was no significant nonlinear relationship between BRI and all-cause mortality ($P = 0.29$) or CVD mortality ($P = 0.73$).

Subgroup and sensitivity analyses

As shown in Fig. 3, the positive association between BRI and all-cause and CVD mortality remained consistent across subgroups stratified by sex, age, BMI, diabetes duration, treatment status, hypertension, hyperlipidemia, and CVD history. The association was more pronounced among females, individuals aged ≥ 60 years, those with BMI ≥ 30.0 kg/m^2, diabetes duration ≥ 10 years, those not receiving treatment, and those with hypertension, hyperlipidemia, or CVD history. An interaction was observed between BRI and CVD history ($P = 0.04$).

Sensitivity analysis showed that excluding participants who died within the first two years of follow-up did not alter the relationship between BRI and all-cause mortality (HR = 1.15, 95% CI = 1.05–1.26) and CVD mortality (HR = 1.23, 95% CI = 1.05–1.43), regardless of whether BRI was treated as a continuous variable or a categorical variable (Q4 all-cause mortality: HR = 2.72, 95% CI = 1.27–5.83; Q4 CVD mortality: HR = 6.62, 95% CI = 2.01–21.82).

Association between other metabolic indices and mortality in diabetes

We also analyzed other metabolic indices, such as BMI, CMI, AIP, LAP, VAI, TyG and WC. As shown in Table 5, BMI was associated with mortality in patients with diabetes (all-cause mortality: HR = 1.03, 95% CI = 1.01–1.06; CVD mortality: HR = 1.05, 95% CI = 1.00–1.10), but the association was not as strong as BRI. Other metabolic indices were not significantly associated with mortality in patients with diabetes ($P > 0.05$).

Discussion

This study characterized the association between BRI and all-cause/CVD mortality in the US diabetic population from 1999 to 2018 and compared it with other body metabolic indicators. This study includes a total of 8227 diabetic individuals from the NHANES database. Interestingly, BRI remained substantially linked to all-cause mortality in the diabetic group even after controlling for several variables, including age, sex, and race. In comparison to other body metabolism indicators, there was a considerable improvement in the correlations between BRI and all-cause/CVD mortality in the diabetic group. To our knowledge, this is the first study to assess how BRI affects all-cause/CVD mortality in a population with diabetes. Obesity is an independent risk factor associated with all-cause/CVD mortality. Currently, an increasing number of studies have concluded that the accumulation of visceral fat is more dangerous than subcutaneous fat and significantly increases the risk of CVD [16]. BRI was proposed by Thomas et al. in 2013. BRI is more accurate in predicting the percentage of body fat and visceral adipose tissue than the classic BMI because it considers waist circumference, a measurement that is sensitive to visceral fat [3]. There have been fewer studies on the evidence of association between BRI and disease and mortality. Zhang et al. found a U-shaped association between BRI and all-cause/CVD mortality in the general population [9]. And a study by Wang et al. found a significant linear dose–response relationship between BRI and heart failure, with higher BRI associated with an increased risk of heart failure [17]. We included diabetic populations from the NHANES database between 1999 and 2018 as supplemental research of prevalent populations. Additionally, we analyzed 10-year follow-up data for these participants. The sample size of our study

Table 2 The baseline characteristics of CVD mortality in diabetes

	All	CVD mortality		*P*-value
	N = 8227	No *N* = 7472 (90.82%)	Yes *N* = 755 (9.18%)	
BRI	6.86 (2.40)	6.88 (2.42)	6.67 (2.26)	0.017
Gender				0.002
Male	4236 (51.49%)	3806 (50.94%)	430 (56.95%)	
Female	3991 (48.51%)	3666 (49.06%)	325 (43.05%)	
Age	61.24 (13.44)	60.33 (13.35)	70.22 (10.79)	< 0.001
Race				< 0.001
Mexican American	1663 (20.21%)	1548 (20.72%)	115 (15.23%)	
Non-Hispanic White	2966 (36.05%)	2583 (34.57%)	383 (50.73%)	
Non-Hispanic Black	2056 (24.99%)	1864 (24.95%)	192 (25.43%)	
Other Race	1542 (18.74%)	1477 (19.77%)	65 (8.61%)	
Education				< 0.001
Less than high school	3028 (36.86%)	2660 (35.64%)	368 (48.94%)	
High school or equivalent	1889 (22.99%)	1732 (23.21%)	157 (20.88%)	
College or above	3298 (40.15%)	3071 (41.15%)	227 (30.19%)	
PIR				< 0.001
< 1.30	2601 (34.97%)	2342 (34.69%)	259 (37.76%)	
1.30–3.49	3036 (40.82%)	2733 (40.48%)	303 (44.17%)	
≥ 3.50	1800 (24.20%)	1676 (24.83%)	124 (18.08%)	
BMI (kg/m^2)				< 0.001
< 25.0	1178 (14.37%)	1035 (13.89%)	143 (19.17%)	
25.0–29.9	2514 (30.67%)	2257 (30.30%)	257 (34.45%)	
≥ 30.0	4504 (54.95%)	4158 (55.81%)	346 (46.38%)	
Smoke				0.001
Never smoker	4071 (49.52%)	3742 (50.12%)	329 (43.58%)	
Ever smoker	1373 (16.70%)	1248 (16.72%)	125 (16.56%)	
Current smoker	2777 (33.78%)	2476 (33.16%)	301 (39.87%)	
Drinking				< 0.001
Nondrinker	3097 (48.43%)	2801 (47.81%)	296 (55.12%)	
Low-to-moderate drinker	1790 (27.99%)	1638 (27.96%)	152 (28.31%)	
Heavy drinker	1508 (23.58%)	1419 (24.22%)	89 (16.57%)	
PA				< 0.001
Inactivity	1606 (33.90%)	1474 (33.18%)	132 (44.59%)	
activity	3132 (66.10%)	2968 (66.82%)	164 (55.41%)	
Duration(years)				< 0.001
< 10	2932 (51.89%)	2693 (52.85%)	239 (43.14%)	
≥ 10	2718 (48.11%)	2403 (47.15%)	315 (56.86%)	
Medication				0.002
No	4437 (80.37%)	4011 (79.84%)	426 (85.71%)	
Yes	1084 (19.63%)	1013 (20.16%)	71 (14.29%)	
HBP				< 0.001
No	2938 (35.82%)	2732 (36.67%)	206 (27.36%)	
Yes	5265 (64.18%)	4718 (63.33%)	547 (72.64%)	
Hypercholesteremia				0.972
No	3128 (41.61%)	2851 (41.62%)	277 (41.47%)	
Yes	4390 (58.39%)	3999 (58.38%)	391 (58.53%)	
CVD				< 0.001
No	6130 (75.17%)	5758 (77.70%)	372 (50.00%)	
Yes	2025 (24.83%)	1653 (22.30%)	372 (50.00%)	
Cancer				0.001
No	7084 (86.25%)	6464 (86.66%)	620 (82.23%)	
Yes	1129 (13.75%)	995 (13.34%)	134 (17.77%)	

CVD included congestive heart failure, coronary heart disease, angina, heart attack and stroke

cohort was larger than that of previous investigations. We found that BRI was strongly correlated with all-cause/CVD mortality in both continuous and quartile variables. Even after excluding participants who passed away within two years, this association remained.

Apart from BRI, other novel indicators have been developed in response to energy metabolism, combining anthropometry and lipid data. LAP is a better indicator of diabetes and insulin resistance in the obese population than standard anthropometric indices, and it can also predict CVD in the general population [18, 19]. Proposed by Dobiásová and Frohlich in 2001, AIP is a lipid measure linearly correlated with insulin resistance and the onset of type 2 diabetes in the general population [20, 21]. Ichiro Wakabayashi et al. presented the CMI as a new metabolic indicator. In addition to being an independent risk factor and a reliable indicator of the onset of diabetes, it can more accurately reflect the degree of obesity and lipid levels [22]. As a novel indicator for evaluating insulin resistance, TyG index is currently commonly regarded as a replacement for the HOMA-IR [23, 24]. VAI, which combines ergometric data and lipid profiles, is a reliable indicator for assessing visceral dysfunction and has been strongly associated with cardiometabolic risk [25]. The Waist-to-Height Ratio (WHtR) is similar to the BRI in that both utilize the parameters of waist circumference and height to assess fat distribution. A meta-analysis has confirmed that, when evaluating metabolic syndrome, the BRI demonstrates a stronger correlation compared to WHtR [6]. Additionally, other studies have shown that, compared to BMI and WHtR, BRI performs better in predicting cardiometabolic abnormalities [26]. Therefore, this paper does not delve further into the exploration of WHtR. We also analyzed the association

Table 3 The association between BRI and mortality in diabetes

	Model 1 HR (95%CI) *P*-value	Model 2 HR (95%CI) *P*-value	Model 3 HR (95%CI) *P*-value
All-cause mortality			
BRI	1.00 (0.97–1.02) 0.82	1.04 (1.01–1.07) 0.01	1.16 (1.07–1.27) < 0.001
Q1 (≤ 5.15)	1.00	1.00	1.00
Q2 (> 5.15 to ≤ 6.48)	1.03 (0.86–1.22) 0.77	0.90 (0.77–1.06) 0.21	1.09 (0.64–1.88) 0.75
Q3 (> 6.48 to ≤ 8.15)	0.95 (0.79–1.14) 0.60	0.88 (0.74–1.05) 0.16	1.38 (0.77–2.48) 0.27
Q4 (> 8.15)	1.01 (0.85–1.20) 0.92	1.20 (1.01–1.43) 0.04	2.52 (1.27–5.00) 0.01
CVD mortality			
BRI	1.00 (0.96–1.04) 0.99	1.06 (1.00–1.11) 0.03	1.27 (1.13–1.42) < 0.001
Q1 (≤ 5.15)	1.00	1.00	1.00
Q2 (> 5.15 to ≤ 6.48)	1.19 (0.94–1.50) 0.15	1.04 (0.82–1.33) 0.73	1.95 (0.77–4.94) 0.16
Q3 (> 6.48 to ≤ 8.15)	1.02 (0.77–1.35) 0.90	0.95 (0.72–1.27) 0.75	3.60 (1.28–10.09) 0.01
Q4 (> 8.15)	1.04 (0.80–1.35) 0.77	1.31 (0.996–1.72) 0.053	6.04 (2.08–17.58) < 0.001

Model 1: Unadjusted

Model 2: Adjusted for age, gender and race

Model 3: Adjusted for age, gender, race, education, BMI, smoke, drinking, duration, medication, physical activity, duration, medication, hypertension, hypercholesteremia and cancer

Table 4 Sensitive analysis

	Model 3 HR (95%CI) *P*-value
All-cause mortality	
BRI	1.15 (1.05–1.26) 0.003
Groups	
Q2	1.26 (0.69–2.31) 0.45
Q3	1.48 (0.74–2.95) 0.27
Q4	2.72 (1.27–5.83) 0.01
CVD mortality	
BRI	1.23 (1.05–1.43) 0.01
Groups	
Q2	2.12 (0.78–5.75) 0.14
Q3	3.86 (1.21–12.29) 0.02
Q4	6.62 (2.01–21.82) 0.002

Excluding less than 2 years of follow-up

Model 3: Adjusted for age, gender, race, education, BMI, smoke, drinking, duration, medication, physical activity, duration, medication, hypertension, hypercholesteremia and cancer

of these metabolic indicators with all-cause/CVD mortality in patients with diabetes. Previous research has predominantly focused on the association between a single metabolic indicator related to energy metabolism and all-cause mortality and cardiovascular mortality, without assessing the strength of the correlation among these indicators. As an extension of this research, the present study compared the associations of these metabolic indicators with the BRI in relation to all-cause mortality and cardiovascular disease mortality among patients with diabetes. In our study, we found that BRI was more highly associated with all-cause/CVD mortality in the diabetic population compared to these novel metabolic indicators of energy metabolism. This is probably because the BRI measures waist circumference, which is a better indicator of the level of visceral fat buildup. The study by Torgny Karlsson et al. points out that the accumulation of visceral fat is a high-risk factor for CVD [27]. Visceral fat is more important and should be detected, according to BRI, than indications of total body lipid metabolism. This also emphasizes more on the management of waist circumference in patients with diabetes due to minimal changes in height.

In this cohort study, we note that the association between BRI and all-cause mortality was higher in people > 60 years of age, BMI ≥ 30 kg/m^2, disease duration > 10 years, unmedicated, and comorbid with hypertension and hyperlipidemia. This may be due to the increased risk of death as the duration of diabetes increases and when other metabolic diseases are comorbid. These findings were also obtained in the subgroup analysis of BRI and CVD, but it is worth noting that BRI and CVD remained correlated even in those with a BMI < 30 kg/m^2. BMI is a commonly used measure of body mass that shows a U-shaped relationship with death [28]. It has been observed that BRI is a better predictor of T2DM than BMI and that BMI has not been examined in conjunction with lipid metabolism, particularly in the case of abdominal obesity [29]. In this case, BRI continued to have a substantial correlation with CVD in

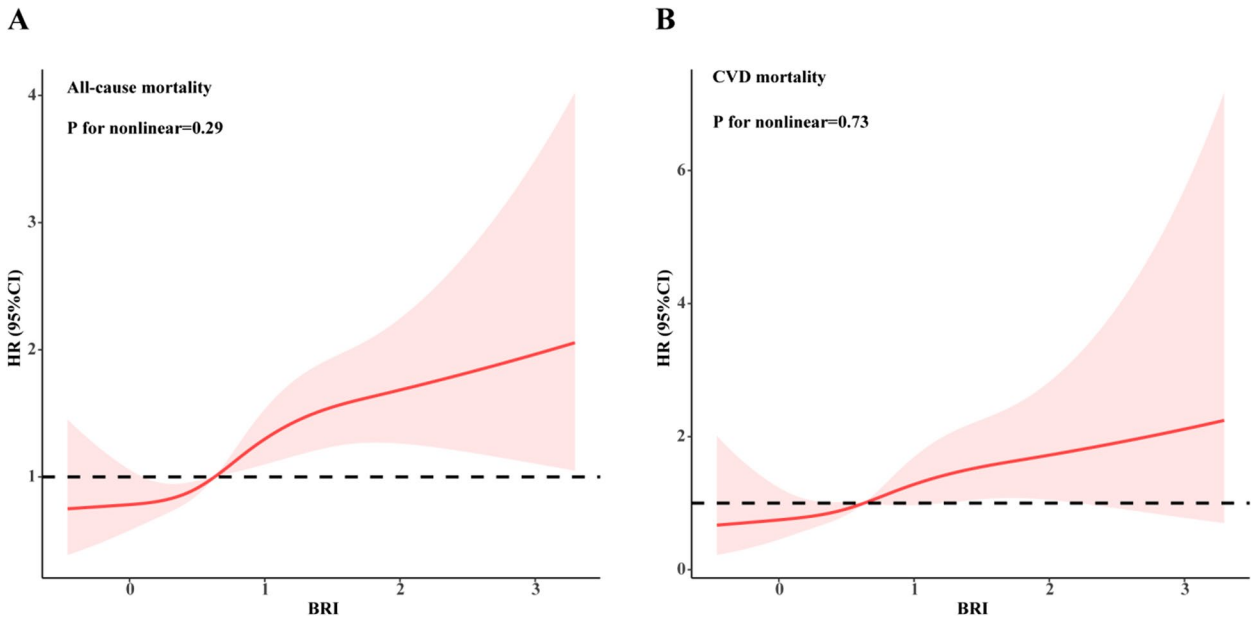

Fig. 2 The nonlinear relationship between BRI and mortality in diabetes

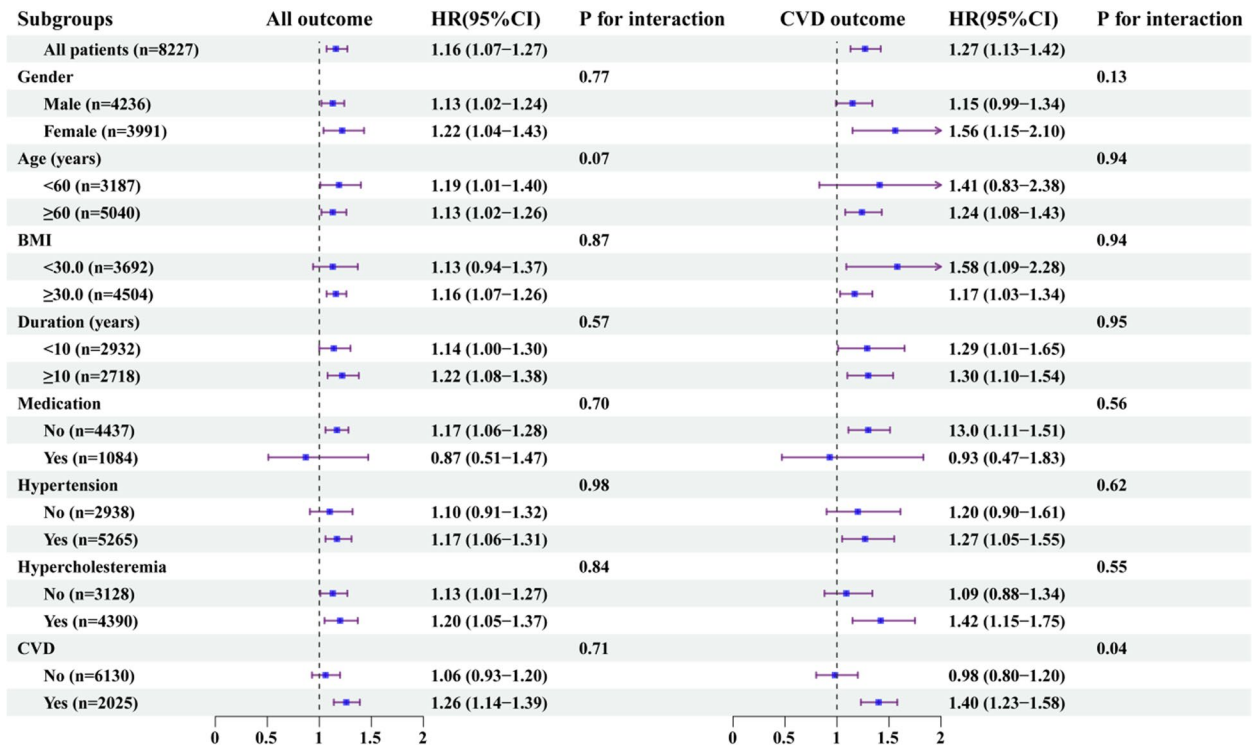

Fig. 3 Subgroups analysis

Table 5 Relationship between other metabolism-related indices and mortality in patients with diabetes

	Model 3 HR (95%CI) *P*-value
All-cause mortality	
BMI	1.03 (1.01–1.06) 0.02
CMI	0.98 (0.87–1.11) 0.77
AIP	1.18 (0.58–2.38) 0.65
LAP	1.00 (0.99–1.01) 0.86
VAI	0.99 (0.95–1.03) 0.53
TyG	1.23 (0.91–1.67) 0.19
WC	1.03 (1.00–1.05) 0.06
CVD mortality	
BMI	1.05 (1.00–1.10) 0.04
CMI	0.86 (0.70–1.05) 0.14
AIP	0.53 (0.13–2.11) 0.37
LAP	1.01 (1.00–1.03) 0.13
AVI	0.95 (0.88–1.02) 0.13
TyG	0.75 (0.41–1.37) 0.36
WC	1.01 (0.96–1.07) 0.69

The model adjusted for age, gender, race, education, BMI, smoke, drinking, duration, medication, physical activity, hypertension, hypercholesteremia and cancer

AIP atherogenic index of plasma, *TyG* triglyceride-glucose index, *CMI* cardiometabolic index, *LAP* lipid accumulation product, *VAI* visceral adiposity index, *WC* waist circumference

Model 3: Adjusted for age, gender, race, education, BMI, smoke, drinking, duration, medication, physical activity, duration, medication, hypertension, hypercholesteremia and cancer

the BMI < 30 kg/m^2 cohort. Additionally, BRI was associated with CVD in the female group compared to the male. This could be because males and females are primarily distinguished by the formation of abdominal fat and subcutaneous fat, respectively [30]. Therefore, once abdominal fat accumulation develops, females may be more susceptible to the incidence of CVD.

Significant ramifications of this study exist for both public health and therapeutic practice. Firstly, BRI is more straightforward and practical than computed tomography for determining fat content; it can be calculated using height and waist circumference, and it can be applied more broadly. Consequently, BRI can be used as a screening technique to notify medical practitioners of patients with diabetes who pose a high risk. Furthermore, by identifying BRI, doctors may be better able to tailor intervention plans to lower the risk of CVD in patients with diabetes. According to our research, reducing BRI is crucial for public health initiatives that aim to minimize all-cause/CVD death in diabetic individuals.

The present study acknowledges several limitations. First, given the retrospective nature of the study design, it is not possible to establish a definitive causative relationship between BRI and all-cause/CVD mortality among patients with diabetes. Thus, prospective studies are warranted in the future to elucidate the causal associations. Second, despite the adjustment for multiple covariates, it is not feasible to account for all potential confounders, which may influence the observed results.

Conclusions

In the diabetic population, we found an association between BRI and all-cause/CVD mortality.The association between BRI and all-cause mortality was higher in people > 60 years of age, BMI ≥ 30 kg/m^2, disease duration > 10 years, unmedicated, and comorbid with hypertension and hyperlipidemia. Further studies are necessary to confirm these findings.

Abbreviations

BRI	Body roundness index
CVD	Cardiovascular disease
FBG	Fasting blood glucose
HbA1c	Glycated hemoglobin
OGTT 2h	Oral glucose tolerance test 2-h
HDL-C	High-density lipoprotein cholesterol
TG	Triglycerides
AIP	Atherogenic index of plasma
LAP	Lipid accumulation product
VAI	Visceral adiposity index
TyG	Triglyceride-glucose index
CMI	Cardiometabolic index
PIR	Family poverty ratio
MET	Metabolic equivalent

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Clinical trial number

Not applicable.

Authors' contributions

H L, HW Y and XF Z conceptualized the study and designed the experiments. LY W, Y W and JX W participated in the statistical analyses. X Y, MX Y, CX M and YT Z contributed to the data analysis. LH W supervised the work and is the guarantor of this work. All authors read and approved the final manuscript.

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Data availability

The data were obtained from the National Health and Nutrition Examination Survey (NHANES), a publicly available source.

Declarations

Ethics approval and consent to participate
The National Center for Health Statistics and Ethics Review Board approved the protocol for NHANES, and all participants provided written informed consent. The authors have disclosed no conflicts of interest. Our study is in line with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Demir S, Nawroth PP, Herzig S, Ekim Üstünel B. Emerging targets in type 2 diabetes and diabetic complications. Adv Sci (Weinh). 2021;8:2100275.
2. Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017. Cardiovasc Diabetol. 2018;17:83.
3. Thomas DM, et al. Relationships between body roundness with body fat and visceral adipose tissue emerging from a new geometrical model. Obesity (Silver Spring). 2013;21:2264–71.
4. Zhao E, Wen X, Qiu W, Zhang C. Association between body roundness index and risk of ultrasound-defined non-alcoholic fatty liver disease. Heliyon. 2024;10: e23429.
5. Wu L, Pu H, Zhang M, Hu H, Wan Q. Non-linear relationship between the body roundness index and incident type 2 diabetes in Japan: a secondary retrospective analysis. J Transl Med. 2022;20:110.
6. Rico-Martín S, et al. Effectiveness of body roundness index in predicting metabolic syndrome: a systematic review and meta-analysis. Obes Rev. 2020;21: e13023.
7. Liu XZ, et al. Body roundness index is significantly associated with prehypertension and hypertension in Nonobese Chinese subjects. Biomed Environ Sci. 2019;32:854–9.
8. Zhou D, Liu X, Huang Y, Feng Y. A nonlinear association between body roundness index and all-cause mortality and cardiovascular mortality in general population. Public Health Nutr. 2022;25:3008–15.
9. Zhang X, et al. Body roundness index and all-cause mortality among US adults. JAMA Netw Open. 2024;7: e2415051.
10. He K, Pang T, Huang H. The relationship between depressive symptoms and BMI: 2005–2018 NHANES data. J Affect Disord. 2022;313:151–7.
11. Shi Y, Wen M. Sex-specific differences in the effect of the atherogenic index of plasma on prediabetes and diabetes in the NHANES 2011–2018 population. Cardiovasc Diabetol. 2023;22:19.
12. Kahn HS. The 'lipid accumulation product' performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. BMC Cardiovasc Disord. 2005;5:26.
13. Amato MC, et al. Visceral Adiposity Index: a reliable indicator of visceral fat function associated with cardiometabolic risk. Diabetes Care. 2010;33:920–2.
14. Zhang Q, Xiao S, Jiao X, Shen Y. The triglyceride-glucose index is a predictor for cardiovascular and all-cause mortality in CVD patients with diabetes or pre-diabetes: evidence from NHANES 2001–2018. Cardiovasc Diabetol. 2023;22:279.
15. Wakabayashi I, Daimon T. The 'cardiometabolic index' as a new marker determined by adiposity and blood lipids for discrimination of diabetes mellitus. Clin Chim Acta. 2015;438:274–8.
16. Fox CS, et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. Circulation. 2007;116:39–48.
17. Wang J, Wu M, Wu S, Tian Y. Relationship between body roundness index and the risk of heart failure in Chinese adults: the Kailuan cohort study. ESC Heart Fail. 2022;9:1328–37.
18. Sun K, et al. Assessment of adiposity distribution and its association with diabetes and insulin resistance: a population-based study. Diabetol Metab Syndr. 2019;11:51.
19. Kyrou I, et al. Lipid accumulation product in relation to 10-year cardiovascular disease incidence in Caucasian adults: The ATTICA study. Atherosclerosis. 2018;279:10–6.
20. Yin B, et al. Non-linear association of atherogenic index of plasma with insulin resistance and type 2 diabetes: a cross-sectional study. Cardiovasc Diabetol. 2023;22:157.
21. Dobiásová M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER(HDL)). Clin Biochem. 2001;34:583–8.
22. Shi W-R, et al. Estimate of prevalent diabetes from cardiometabolic index in general Chinese population: a community-based study. Lipids Health Dis. 2018;17:236.
23. Simental-Mendía LE, et al. The triglycerides and glucose index is strongly associated with hepatic steatosis in children with overweight or obesity. Eur J Pediatr. 2021;180:1755–60.
24. Gilmore GC, Wenk HE, Naylor LA, Stuve TA. Motion perception and aging. Psychol Aging. 1992;7:654–60.
25. Kang YM, et al. Visceral adiposity index predicts the conversion of metabolically healthy obesity to an unhealthy phenotype. PLoS ONE. 2017;12: e0179635.
26. Tian S, Zhang X, Xu Y, Dong H. Feasibility of body roundness index for identifying a clustering of cardiometabolic abnormalities compared to BMI, waist circumference and other anthropometric indices: the China Health and Nutrition Survey, 2008 to 2009. Medicine (Baltimore). 2016;95:e4642.
27. Karlsson T, et al. Contribution of genetics to visceral adiposity and its relation to cardiovascular and metabolic disease. Nat Med. 2019;25:1390–5.
28. Davey Smith G, et al. The association between BMI and mortality using offspring BMI as an indicator of own BMI: large intergenerational mortality study. BMJ. 2009;339: b5043.
29. Sadeghi E, et al. Novel anthropometric indices for predicting type 2 diabetes mellitus. BMC Public Health. 2024;24:1033.
30. Pan R, Chen Y. Fat biology and metabolic balance: On the significance of sex. Mol Cell Endocrinol. 2021;533: 111336.

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