scientific reports

Check for updates

OPEN

Association between body roundness index and osteoarthritis/rheumatoid arthritis: a cross-sectional study

Tao Gao[1]✉, Zhi-Yu Chen[2], Tao Li[1], Xu Lin[1], Hai-Gang Hu[1], Jian-Dong Tang[1] & Chao Wu[1]✉

To explore the relationship between the Body Roundness Index (BRI) and the prevalence of osteoarthritis (OA) and rheumatoid arthritis (RA) among American adults, providing new insights for identifying OA and RA in adults. We analyzed data from the National Health and Nutrition Examination Survey (NHANES) 2015–2023 and conducted a large cross-sectional study. BRI was calculated based on body measurements, while OA and RA cases were identified through questionnaires. Participants under 20 years of age and those with incomplete data were excluded. Weighted multivariate logistic regression models, restricted cubic spline (RCS) functions, and stratified analyses were used to assess the relationship between BRI levels and the prevalence of OA and RA in American adults. To further evaluate BRI's diagnostic potential for OA and RA, receiver operating characteristic (ROC) curves were employed to analyze and calculate the area under the curve (AUC). After screening, 17,544 participants were included, with 2,382 cases of OA (13.58%) and 987 cases of RA (5.63%). Multivariate logistic regression analyses showed a positive correlation between BRI and OA prevalence in American adults in both the unadjusted and adjusted models. A similar correlation was observed for RA in the unadjusted and partially adjusted models ($P < 0.001$), but the fully adjusted model showed no significant association between BRI and RA ($P > 0.05$). In the unadjusted model, the prevalence of OA in the highest BRI quartile was 3.47 times than that of the lowest quartile (95% CI: 2.84, 4.24, $P < 0.001$). Even in the fully adjusted model, the prevalence of OA in the highest BRI quartile remained 1.46 times higher than that of the lowest quartile (95% CI: 1.02, 2.08, $P < 0.05$). RCS curves demonstrated a non-linear relationship between BRI and both OA and RA, with a significant increase in prevalence as BRI levels rose ($P < 0.001$). Subgroup analyses and forest plots indicated a positive correlation between BRI and OA and RA in most subgroups ($P < 0.05$). ROC curves showed that BRI had a better predictive ability for OA and RA risk compared to BMI. There is a significant positive correlation between BRI and the prevalence of OA and RA in American adults, especially OA. Maintaining a lower BRI may help prevent the onset of OA and RA.

Keywords Osteoarthritis, Rheumatoid arthritis, Body roundness index, Systemic inflammation, NHANES

Abbreviations

BRI	Body Roundness Index
OA	Osteoarthritis
RA	Rheumatoid arthritis
NHANES	National Health and Nutrition Examination Survey
RCS	Restricted cubic spline
ROC	Receiver operating characteristic
AUC	Area under the curve
OR	Odds ratios
CI	Confidence intervals
BMI	Body mass index
HSCRP	High-sensitivity C-reactive protein
WBC	White blood cell count

[1]Orthopaedics of Zigong Fourth People's Hospital, Zigong 643000, Sichuan, China. [2]Respiratory Medicine of Zigong Fourth People's Hospital, Zigong 643000, Sichuan, China. ✉email: 374648027@qq.com; flightiness@163.com

SII	Systemic inflammatory index
WC	Waist circumference

Osteoarthritis (OA) and rheumatoid arthritis (RA) are the two most common chronic joint diseases, significantly impacting patients' quality of life and posing a heavy burden on global public health systems[1,2]. According to the World Health Organization, OA is the most common form of arthritis worldwide, and its incidence increases with age. A study by Safiri et al.[3]reported that the global prevalence of OA exceeds 20%, with the condition being especially prevalent among the elderly population[3–5]. RA, on the other hand, is a chronic, systemic autoimmune disease characterized by a high disability rate and is more common among middle-aged women[6,7]. A global, regional, and national study on the burden of RA estimated that in 2020, 17.6 million people worldwide were affected by RA, with projections suggesting that by 2050, this number will rise to 31.7 million[8]. As the global population continues to age, the incidence of both OA and RA is increasing, making it crucial to understand the risk factors for these diseases in order to inform prevention and treatment strategies.

Obesity is considered a major risk factor for both OA and RA[9,10]. Obesity not only accelerates joint wear through mechanical pressure[11] but also exacerbates systemic inflammation through the secretion of pro-inflammatory cytokines by visceral fat tissue[12]. However, the traditional body mass index (BMI), commonly used to assess obesity, has certain limitations[9]. BMI merely reflects the ratio of weight to height and cannot accurately distinguish between fat and muscle mass, nor can it assess the distribution of visceral fat[13]. Recent research by Xue et al.[14] found that there may be a non-linear positive correlation between visceral fat metabolic score and OA risk, and that visceral fat metabolic score could serve as a more accurate indicator for diagnosing OA. Therefore, identifying more precise anthropometric measurements to better predict the impact of obesity on joint diseases is of significant research and clinical importance.

The Body Roundness Index (BRI) is a novel anthropometric tool that has garnered increasing attention in recent years. By combining waist circumference and height, BRI more accurately reflects body roundness and the distribution of visceral fat, effectively evaluating obesity-related health risks[15]. Previous studies have shown that BRI is closely associated with various chronic diseases, such as diabetes, cardiovascular disease, and metabolic syndrome[16–18]. However, research on the relationship between BRI and OA and RA remains limited, especially when using large, representative datasets. Exploring the potential link between BRI and arthritis could provide new perspectives for the early screening and prevention of joint diseases.

This study aims to assess the association between BRI and OA/RA using data from the National Health and Nutrition Examination Survey (NHANES) and to compare BRI with other traditional obesity measures, such as BMI, in predicting arthritis risk. The findings will provide theoretical support for the prevention and early intervention of arthritis. Additionally, this study may offer new insights for clinical practice and public health strategies. If BRI proves effective in early screening and intervention for arthritis, future healthcare providers and policymakers will be better equipped to identify high-risk individuals, reduce the prevalence and disability rates of arthritis, and ultimately alleviate the economic burden on society.

Materials and methods

Study population

This large cross-sectional study utilized data from the 2015–2023 cycles of the NHANES, conducted by the National Center for Health Statistics (NCHS). NHANES employs a multistage, stratified sampling method to collect data on various aspects of health and nutrition in the U.S. population through interviews, physical examinations, questionnaires, and laboratory testing. The NHANES study protocol was approved by the NCHS Institutional Review Board, and all participants provided informed consent. NHANES data are publicly available on its official website.

In this study, we included 37,464 individuals who participated in the 2015–2023 NHANES cycles. We excluded participants younger than 20 years of age and those with incomplete data for OA, RA, high-sensitivity C-reactive protein (HSCRP), BMI, waist circumference, height, white blood cell count (WBC), systemic inflammatory index (SII), physical activity, hypertension, smoking, alcohol consumption, or other covariates. After exclusions, 17,544 participants remained in the final analysis (Fig. 1).

Study variables

Exposure and outcomes

The main exposure of interest in this study was BRI, which was derived from the Body Measures under Examination Data. This dataset includes waist circumference (WC), height, and BMI measurements, and BRI was calculated using the following formula:

$$BRI = 364.2 - 365.5 \times \sqrt{1 - \left(\frac{wc}{2\pi}\right)^2 / \left(\frac{Height}{2}\right)^2}$$

Outcome variables were the presence of OA and RA. These outcomes were determined from self-reported data in questionnaires. Participants were asked, "Has a doctor or other health professional ever told {you/SP} that {you/she/he} had arthritis (ar-thry-tis)?" If the response was "Yes," they were further asked, "What type of arthritis?" Participants who responded "Rheumatoid arthritis" were classified as having RA, while those who responded "Osteoarthritis" were classified as having OA. Those who did not report these conditions were placed in the non-OA or non-RA groups, respectively.

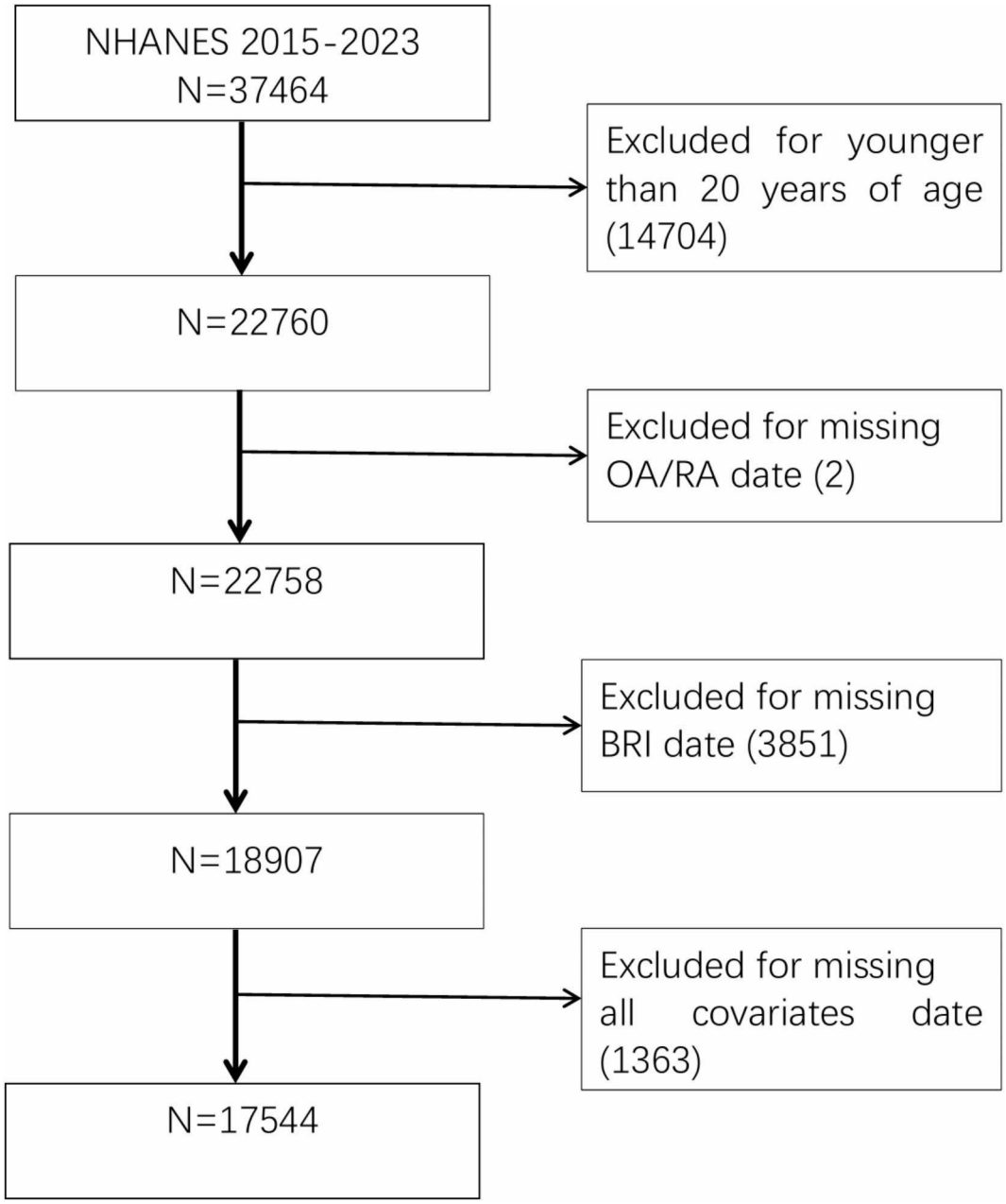

Fig. 1. Flowchart of Sample Selection. The study population consists of adults aged 20 and above.

Covariates

Several potential covariates that may influence the relationship between BRI and OA or RA were included in the analysis, such as age (y), gender, race, BMI (kg/m^2), serum HSCRP levels (mg/L), serum WBC levels (x 10^3 cells/uL), SII, hypertension, physical activity, smoking, and alcohol consumption. Age, gender, race, physical activity, smoking, and alcohol data were obtained from questionnaires. Race was categorized as Mexican American, Other Hispanic, Non-Hispanic White, Non-Hispanic Black, and Other Races (including multi-racial individuals). BMI was categorized into three groups based on obesity status (< 25, 25–30, and > 30). Smoking status was based on the question: Smoked at least 100 cigarettes in life, divided into smoking and non-smoking groups. Physical activity was classified as heavy, moderate, or insufficient based on the questionnaire responses regarding vigorous recreational or work-related activities. Hypertension was defined based on self-reported physician diagnosis or average systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 80 mmHg, following established guidelines[19]. Alcohol consumption was categorized as heavy (≥ 3 drinks/day for men or ≥ 2 drinks/day for women), moderate (1–2 drinks/day for men or 1 drink/day for women), or non-drinker[20]. Serum HSCRP and WBC levels were obtained from laboratory data, while the SII was calculated using the formula: SII = (platelet count × neutrophil count) / lymphocyte count.

Statistical analysis

All analyses were weighted according to NHANES guidelines. Continuous variables that were normally distributed were presented as weighted means ± standard deviation (SD) and compared using weighted linear regression. Categorical variables were presented as weighted percentages and compared using the weighted χ^2 test. To evaluate the relationship between BRI and OA and RA, we used weighted multivariate logistic regression models, adjusting for covariates in three models:

- Model 1: No covariate adjustment.
- Model 2: Adjusted for age, gender, and race.
- Model 3: Adjusted for model 2 variables plus HSCRP, WBC, SII, BMI, hypertension, physical activity, smoking, and alcohol consumption.

To assess the non-linear relationship between BRI and OA and RA, we used restricted cubic splines (RCS). The diagnostic performance of BRI versus BMI for OA and RA was compared using ROC curves, and the area under the curve (AUC) was used to assess the predictive ability. Weighted subgroup analyses and forest plots were used to evaluate the relationship between BRI and OA/RA in different subgroups, and P values were calculated in age-stratified analyses to assess the effect of age on the relationship between BRI and arthritis. Statistical analyses were performed using R software (version 4.2.0), with statistical significance defined as $P < 0.05$.

Results

Participant characteristics

A total of 17,544 participants met the inclusion criteria, with 2,382 individuals diagnosed with OA (prevalence 13.58%) and 987 individuals diagnosed with RA (prevalence 5.63%). Significant differences were found between OA and non-OA participants in terms of gender, age, race, BRI, BMI, HSCRP, SII, hypertension, physical activity, smoking, and alcohol consumption ($P < 0.01$), while no significant differences were found in WBC levels between the two groups (Table 1). Significant differences were also found between RA and non-RA participants in age, race, BRI, BMI, HSCRP, SII, WBC levels, hypertension, physical activity, smoking, and alcohol consumption ($P < 0.05$) (Table 2).

Association between BRI and OA/RA

Weighted multivariate logistic regression analyses indicated a positive correlation between BRI and OA in all three models ($P < 0.001$). In the unadjusted model, the prevalence of OA in the highest BRI quartile was 3.47 times than that of the lowest quartile (95% CI: 2.84, 4.24, $P < 0.001$). Even in the fully adjusted model, the prevalence of OA in the highest BRI quartile remained 1.46 times higher than that of the lowest quartile (95% CI: 1.02, 2.08, $P < 0.05$) (Table 3). The unadjusted and partially adjusted models showed a positive correlation between BRI and RA ($P < 0.001$), but this association became non-significant in the fully adjusted model ($P > 0.05$) (Table 4).

BRI and OA/RA: non-linear relationships

RCS analyses demonstrated a non-linear relationship between BRI and both OA and RA in all three models ($P < 0.05$). As BRI increased, the risk of both OA and RA also increased (Fig. 2).

Comparison of BRI and BMI for OA/RA diagnosis

ROC curve analyses revealed that BRI had a better AUC for diagnosing OA than BMI. Similarly, BRI outperformed BMI in diagnosing RA, indicating that BRI is a more accurate predictor of OA and RA risk (Fig. 3; Tables 5 and 6).

Subgroup analysis of BRI and OA/RA

The subgroup analysis of BRI and OA demonstrated a significant positive correlation across all subgroups for gender, race, smoking status, hypertension, alcohol consumption, and physical activity, as well as in the 41–60 and > 60 age groups and the BMI > 30 group (Fig. 4). Interaction tests showed significant interactions for gender, race, smoking status, and hypertension ($P < 0.05$). Similarly, in the subgroup analysis of BRI and RA, a positive correlation was observed in all subgroups except for the Insufficient Physical Activity group and the BMI < 25 group ($P < 0.05$). However, interaction tests indicated no significant interactions between subgroups for RA ($P > 0.05$) (Fig. 5).

Discussion

This study is one of the first large-scale cross-sectional analyses exploring the association between BRI and OA/RA, providing new evidence for the application of BRI in arthritis risk assessment. Our findings demonstrate that BRI outperforms BMI in predicting OA risk, supporting its potential use as a future clinical screening tool. We identify that BMI may underestimate OA risk in certain individuals, particularly those with normal BMI but elevated BRI, emphasizing the need for further research on the role of BRI. The results of this study revealed a significant positive association between BRI and the prevalence of OA and, to a lesser extent, RA in American adults. These findings highlight the potential of BRI as a novel tool for assessing obesity-related health risks, particularly in joint diseases like OA. Compared to traditional BMI, BRI offers a more accurate reflection of visceral fat distribution, making it a better predictor of disease risk. Our results showed that gender, race, smoking, hypertension, drinking status and age 41–60, > 60 group and BMI > 30 group, BRI was positively associated with OA and RA. And the results of age subgroup analysis showed that the associations between BRI and OA and RA were more consistent in different age groups, but P for interaction > 0.05, indicating

Variables	Total (*n* = 17544)	OA (*n* = 2382)	Non-OA (*n* = 15162)	*p*
Gender(%)				< 0.001
Male	8346 (48.68)	844 (34.23)	7502 (50.97)	
Female	9198 (51.32)	1538 (65.77)	7660 (49.03)	
Age(%, y)				< 0.001
20–40	5278 (35.74)	104 (4.94)	5174 (40.64)	
41–60	5672 (35.19)	631 (31.82)	5041 (35.72)	
>60	6594 (29.07)	1647 (63.23)	4947 (23.64)	
Race(%)				< 0.001
Mexican American	2115 (8.16)	137 (2.64)	1978 (9.04)	
Other Hispanic	1984 (7.92)	168 (3.87)	1816 (8.56)	
Non-Hispanic White	7421 (63.28)	1512 (79.33)	5909 (60.73)	
Non-Hispanic Black	3439 (10.41)	326 (6.63)	3113 (11.01)	
Other Race - Including Multi-Racial	2585 (10.23)	239 (7.53)	2346 (10.66)	
BMI(%, kg/m^2)				< 0.001
<25	3527 (20.86)	344 (14.51)	3183 (21.87)	
25–30	6726 (38.64)	839 (35.86)	5887 (39.08)	
>30	7291 (40.50)	1199 (49.63)	6092 (39.05)	
BRI (mean (SD))	5.597 (2.402)	6.483 (2.539)	5.456 (2.350)	< 0.001
HSCRP (mean (SD), mg/L)	3.734 (7.086)	4.260 (9.257)	3.650 (6.673)	0.009
WBC (mean (SD), x 10^3 cells/uL)	7.198 (3.062)	7.258 (2.261)	7.188 (3.170)	0.278
SII (mean (SD))	538.427 (324.111)	587.090 (406.425)	530.695 (308.331)	< 0.001
Smoke status(%)				< 0.001
Yes	7290 (41.30)	1175 (49.93)	6115 (39.92)	
No	10,254 (58.70)	1207 (50.07)	9047 (60.08)	
Hypertension status(%)				< 0.001
Yes	7246 (36.71)	1133 (45.02)	6113 (35.39)	
No	10,298 (63.29)	1249 (54.98)	9049 (64.61)	
Drinking status(%)				< 0.001
Heavy	5834 (36.96)	633 (29.17)	5201 (38.20)	
Moderate	5801 (34.57)	896 (39.01)	4905 (33.86)	
None	5909 (28.47)	853 (31.82)	5056 (27.94)	
Activity status(%)				< 0.001
Heavy	7528 (47.95)	754 (32.41)	6774 (50.42)	
Moderate	5363 (30.91)	882 (39.41)	4481 (29.56)	
Insufficient	4653 (21.14)	746 (28.17)	3907 (20.02)	

Table 1. Characteristics of included samples by OA. Categorical variables are expressed as unweighted counts (weighted percentages); continuous data are expressed as weighted means (SD). BMI, body mass index; BRI, body roundness index; SII, systemic inflammatory index.

that the interaction effect of age on the relationship between BRI and OA and RA was not significant. These results highlight the potential clinical applications of the BRI in different populations and support the need for personalized health assessment.

Osteoarthritis and BRI: a strong correlation

One of the main findings of this study is the significant correlation between higher BRI and OA. Specifically, participants in the highest BRI quartile had a 2.01 times higher risk of developing OA compared to those in the lowest quartile (95% CI: 1.58–2.55, $P < 0.001$), which is consistent with previous studies showing that obesity, especially visceral obesity, is a major risk factor for OA[14,21,22]. The pathogenesis of OA is multifactorial, but mechanical loading on the joints and the influence of metabolic and inflammatory processes are thought to be primary factors[11,23,24]. Obesity, as reflected by BRI, exacerbates both mechanical and systemic inflammatory factors, leading to the worsening of OA. Excess weight places excessive stress on weight-bearing joints, such as the knees and hips, accelerating cartilage wear and degeneration. This mechanical damage is one of the main pathways through which obesity influences OA risk[11]. Studies have shown that for every 1 kg reduction in body weight, the load on the knee joint is reduced by more than twofold, significantly decreasing the risk of joint damage[25].

In addition to mechanical stress, the role of adipose tissue in systemic inflammation has gained increasing recognition. Obesity, particularly central or visceral obesity, promotes the release of pro-inflammatory cytokines

Variables	Total (*n* = 17544)	RA (*n* = 987)	Non-RA (*n* = 16557)	*p*
Gender(%)				0.735
Male	8346 (48.68)	452 (47.91)	7894 (48.71)	
Female	9198 (51.32)	535 (52.09)	8663 (51.29)	
Age(%, y)				< 0.001
20–40	5278 (35.74)	69 (11.32)	5209 (36.79)	
41–60	5672 (35.19)	313 (38.67)	5359 (35.04)	
>60	6594 (29.07)	605 (50.02)	5989 (28.17)	
Race(%)				< 0.001
Mexican American	2115 (8.16)	116 (8.72)	1999 (8.14)	
Other Hispanic	1984 (7.92)	136 (9.29)	1848 (7.86)	
Non-Hispanic White	7421 (63.28)	358 (57.08)	7063 (63.55)	
Non-Hispanic Black	3439 (10.41)	275 (16.12)	3164 (10.16)	
Other Race - Including Multi-Racial	2585 (10.23)	102 (8.78)	2483 (10.29)	
BMI(%, kg/m^2)				< 0.001
<25	3527 (20.86)	123 (13.54)	3404 (21.18)	
25–30	6726 (38.64)	356 (35.92)	6370 (38.76)	
>30	7291 (40.50)	508 (50.54)	6783 (40.07)	
BRI (mean (SD))	5.597 (2.402)	6.425 (2.428)	5.562 (2.395)	< 0.001
HSCRP (mean (SD), mg/L)	3.734 (7.086)	5.311 (8.578)	3.666 (7.007)	< 0.001
WBC (mean (SD), x 10^3 cells/uL)	7.198 (3.062)	7.413 (2.513)	7.188 (3.083)	0.048
SII (mean (SD))	538.427 (324.111)	586.961 (435.511)	536.342 (318.300)	0.005
Smoke status(%)				< 0.001
Yes	7290 (41.28)	523 (54.38)	6766 (40.72)	
No	10,254 (58.72)	464 (45.62)	9791 (59.28)	
Hypertension status(%)				< 0.001
Yes	7246 (36.71)	499 (46.50)	6747 (36.29)	
No	10,298 (63.29)	488 (53.50)	9810 (63.71)	
Drinking status(%)				0.001
Heavy	5834 (36.96)	269 (30.59)	5565 (37.23)	
Moderate	5801 (34.57)	285 (32.54)	5516 (34.66)	
None	5909 (28.47)	433 (36.87)	5476 (28.11)	
Activity status(%)				< 0.001
Heavy	7528 (47.95)	323 (35.72)	7205 (48.47)	
Moderate	5363 (30.91)	337 (33.68)	5026 (30.79)	
Insufficient	4653 (21.14)	327 (30.60)	4326 (20.73)	

Table 2. Characteristics of included samples by RA. Categorical variables are expressed as unweighted counts (weighted percentages); continuous data are expressed as weighted means (SD). BMI, body mass index; BRI, body roundness index; SII, systemic inflammatory index.

Variables	Model 1		Model 2		Model 3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
BRI Quartiles						
Q1 (1.23, 4.02)	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q 2 (4.03, 5.38)	1.83(1.45, 2.31)	< 0.001	1.26(0.98, 1.62)	0.066	1.11(0.84, 1.48)	0.446
Q 3 (5.39, 7.08)	2.45(2.00, 3.00)	< 0.001	1.56(1.27, 1.93)	< 0.001	1.21(0.90, 1.62)	0.198
Q 4 (7.09, 22.99)	3.47(2.84, 4.24)	< 0.001	2.1(1.69, 2.62)	< 0.001	1.46(1.02, 2.08)	0.037

Table 3. The correlation between serum BRI levels and OA by weighted multivariable logistic regression. OR: Odds Ratio, CI: Confidence Interval, BRI: body roundness index. Model 1: Unadjusted. Model 2: Adjusted for gender, age, and race. Model 3: Adjusted for model 2 variables plus HSCRP, WBC, SII, BMI, hypertension, physical activity, smoking, and alcohol consumption.

Variables	Model 1		Model 2		Model 3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
BRI Quartiles						
Q1 (1.23, 4.02)	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Q 2 (4.03, 5.38)	1.67 (1.16, 2.40)	0.006	1.23 (0.85, 1.77)	0.264	1.19 (0.74, 1.90)	0.467
Q 3 (5.39, 7.08)	2.47 (1.74, 3.52)	< 0.001	1.66 (1.14, 2.43)	0.01	1.51 (0.88, 2.57)	0.128
Q 4 (7.09, 22.99)	2.8 (2.11, 3.71)	< 0.001	1.93 (1.41, 2.63)	< 0.001	1.6 (0.93, 2.74)	0.089

Table 4. The correlation between serum BRI levels and RA by weighted multivariable logistic regression. OR: Odds Ratio, CI: Confidence Interval, BRI: body roundness index. Model 1: Unadjusted. Model 2: Adjusted for gender, age, and race. Model 3: Adjusted for model 2 variables plus HSCRP, WBC, SII, BMI, hypertension, physical activity, smoking, and alcohol consumption.

such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and adipokines from adipose tissue. These cytokines not only exacerbate low-grade systemic inflammation but also directly contribute to cartilage destruction and joint inflammation[23,24]. Research has shown that the accumulation of visceral fat is positively correlated with the development of OA[14], reflecting the strong impact of visceral fat and related inflammatory processes on joint health. BRI, which better reflects these factors, has emerged as a new tool for studying the relationship between obesity and OA.

Rheumatoid arthritis and BRI: a more complex relationship

In contrast, the association between BRI and RA became non-significant after adjustment, suggesting that the pathophysiology of RA may be more complex, involving multiple immune system factors[26,27]. In our study, although a positive correlation between BRI and RA was observed in the unadjusted model, this association disappeared after multivariate adjustment. This finding aligns with other studies that emphasize the complicated relationship between RA and body weight[28–31]. Among RA patients, obesity is closely associated with decreased joint function and lower quality of life. Several studies have shown that overweight RA patients typically require more aggressive treatment and respond less effectively to therapies[10,32,33]. Marchand et al.[34], in a large prospective cohort study involving 2,583,266 individuals, examined the relationship between long-term weight changes and RA risk, finding that weight gain over time significantly increased RA risk in women, with a threefold increase in RA risk for those gaining ≥ 20 kg. Similarly, a large cross-sectional study by Ferguson et al.[35], involving 502,417 participants, demonstrated a positive association between central obesity, as measured by waist circumference, and the incidence of RA. However, compared to OA, the relationship between RA and obesity remains less understood. It likely involves a combination of factors, including inflammatory responses, immune system dysregulation, and metabolic abnormalities. Some studies suggest that obesity may exacerbate disease severity by increasing systemic inflammation[31]. Additionally, obesity may impair immune tolerance by promoting inflammation and reducing regulatory B (Breg) and regulatory T (Treg) cells, which leads to an increase in Th17 and Th1 cells, creating an environment conducive to the development of autoimmune diseases[36].

Clinical implications and preventive strategies

Traditionally, BMI has been widely used to assess obesity. However, because it does not account for fat distribution and composition, many researchers have sought more precise measurement tools. As a novel obesity assessment index, the BRI offers the advantage of more accurately reflecting visceral fat distribution, providing a more effective evaluation of health risks compared to BMI. Recent research by Xue et al.[14] indicates a potential non-linear positive correlation between visceral fat metabolic score and OA risk, suggesting that the metabolic score of visceral fat could serve as a more accurate diagnostic indicator for OA. As the prevalence of OA and RA continues to rise, adopting BRI for risk assessment could present new opportunities for early screening and intervention. Our ROC analysis results demonstrated that BRI is more accurate than BMI in predicting the risks of OA and RA. This finding suggests that clinicians could integrate BRI into routine examinations to better identify high-risk individuals.

Recent research by Zhang et al.[37] revealed a U-shaped relationship between BRI and all-cause mortality, indicating that both excessively low and high BRI levels are associated with increased mortality risk. This further suggests that BRI is not only a predictor of joint health issues but also an important metric for evaluating overall health and mortality risk. Since BRI is highly sensitive to individual fat distribution, it could be used in the future for clinical health assessments and large-scale public health initiatives as a key tool for preventing and managing obesity-related diseases[38–40].

Moreover, the widespread application of BRI could advance the practice of personalized medicine. By providing more accurate assessments of individual obesity risk, physicians can offer more targeted treatment recommendations based on BRI values, such as visceral fat reduction strategies or other lifestyle interventions, to mitigate the development of arthritis and other metabolic diseases.

Limitations and future directions

Although this study provides valuable insights, several limitations should be considered. First, the cross-sectional nature of the study prevents us from establishing a causal relationship between BRI and arthritis. Longitudinal studies are needed to confirm the temporal association between BRI and the development of OA

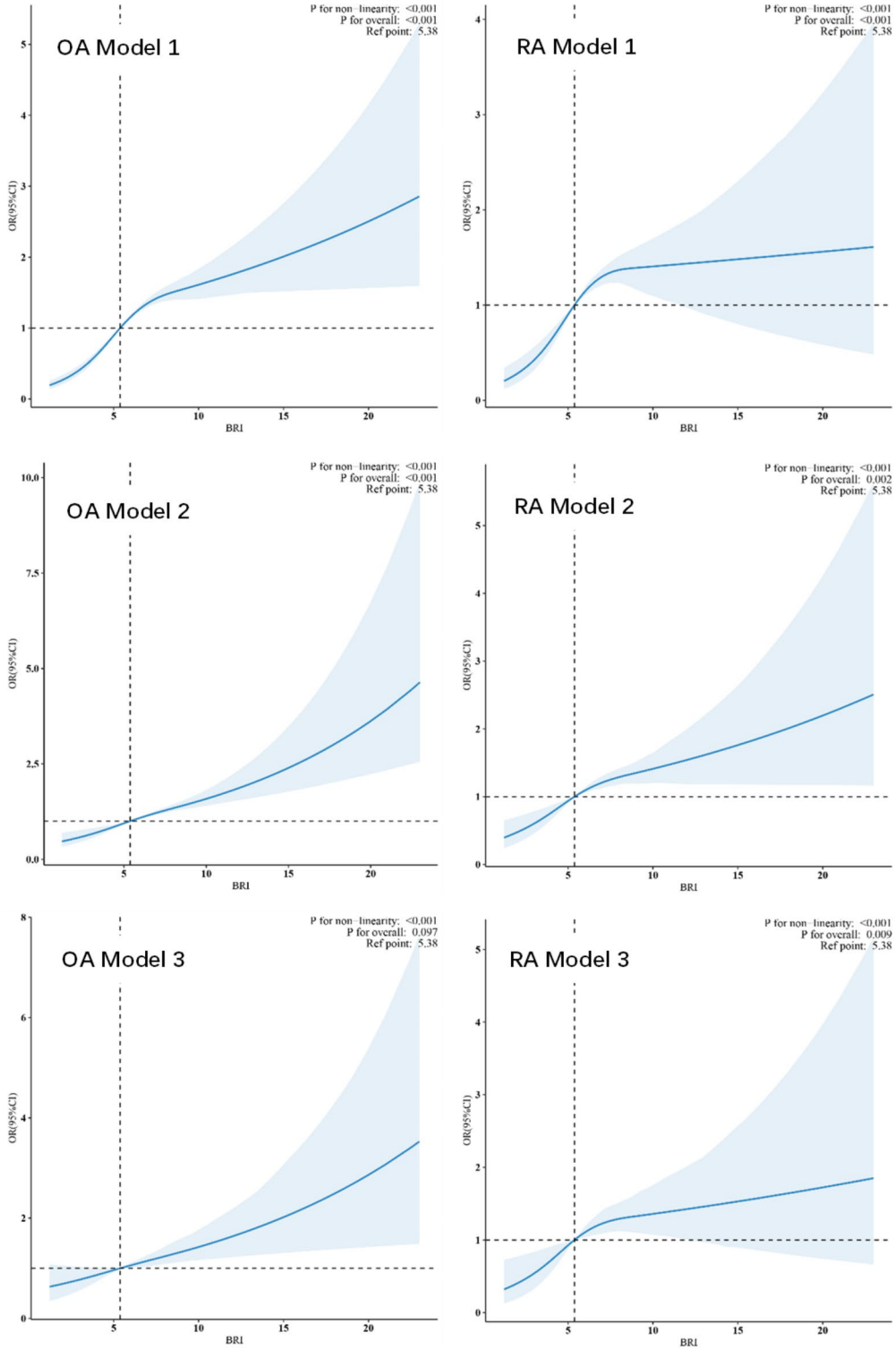

Fig. 2. Restricted cubic spline curve (RCS) plot of the relationship between BRI and OA/RA risk under different models. The solid blue line represents the odds ratio (OR) of OA/RA. The shaded area represents the 95% confidence interval.

and RA. Second, the reliance on self-reported data for OA and RA diagnosis may introduce misclassification bias. Future studies should incorporate objective diagnostic measures, such as radiographic confirmation, to improve the accuracy of findings. Finally, some participants had a combination of both OA and RA, but given the relatively low prevalence of RA compared with OA and the small number of overlapping cases, separate subgroup analyses were less statistically reliable. Future studies with larger sample sizes are needed to further

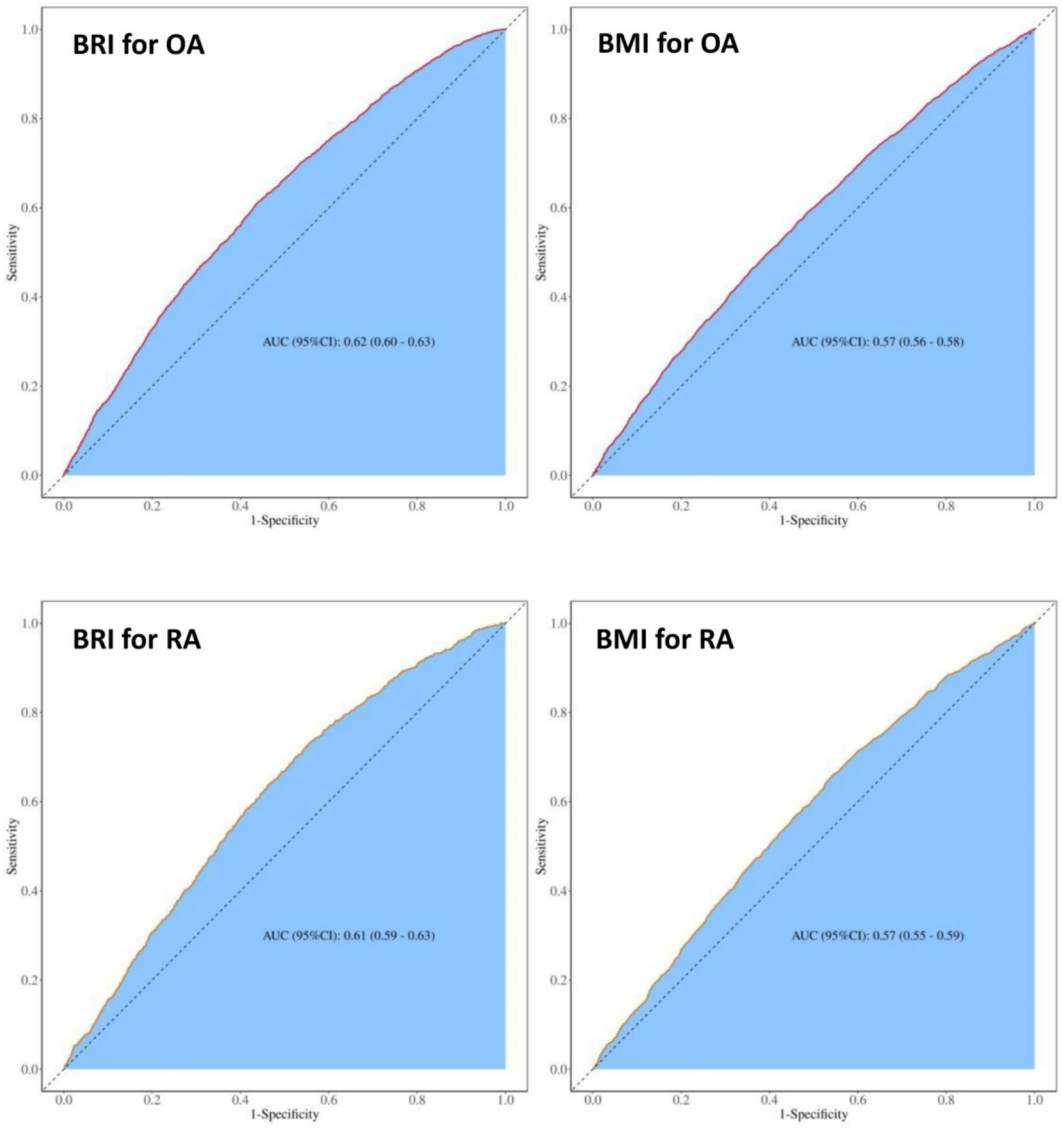

Fig. 3. ROC plot of BRI and BMI for diagnosis of OA/RA, blue shaded area represents area under the curve. ROC: receiver operating characteristic, BRI: body roundness index, BMI: body mass index.

Test	AUC (95%CI)	Sensitivity	Specificity	Cut off
BMI	0.57 (0.56, 0.58)	0.54	0.57	28.95
BRI	0.62 (0.60, 0.63)	0.56	0.61	5.615

Table 5. Sensitivity and specificity of BRI and BMI in diagnosing OA. AUC: area under the curve, BRI: body roundness index, BMI: body mass index.

Test	AUC (95%CI)	Sensitivity	Specificity	Cut off
BMI	0.57 (0.55, 0.59)	0.46	0.66	27.85
BRI	0.61 (0.59, 0.63)	0.45	0.73	5.025

Table 6. Sensitivity and specificity of BRI and BMI in diagnosing RA. AUC: area under the curve, BRI: body roundness index, BMI: body mass index.

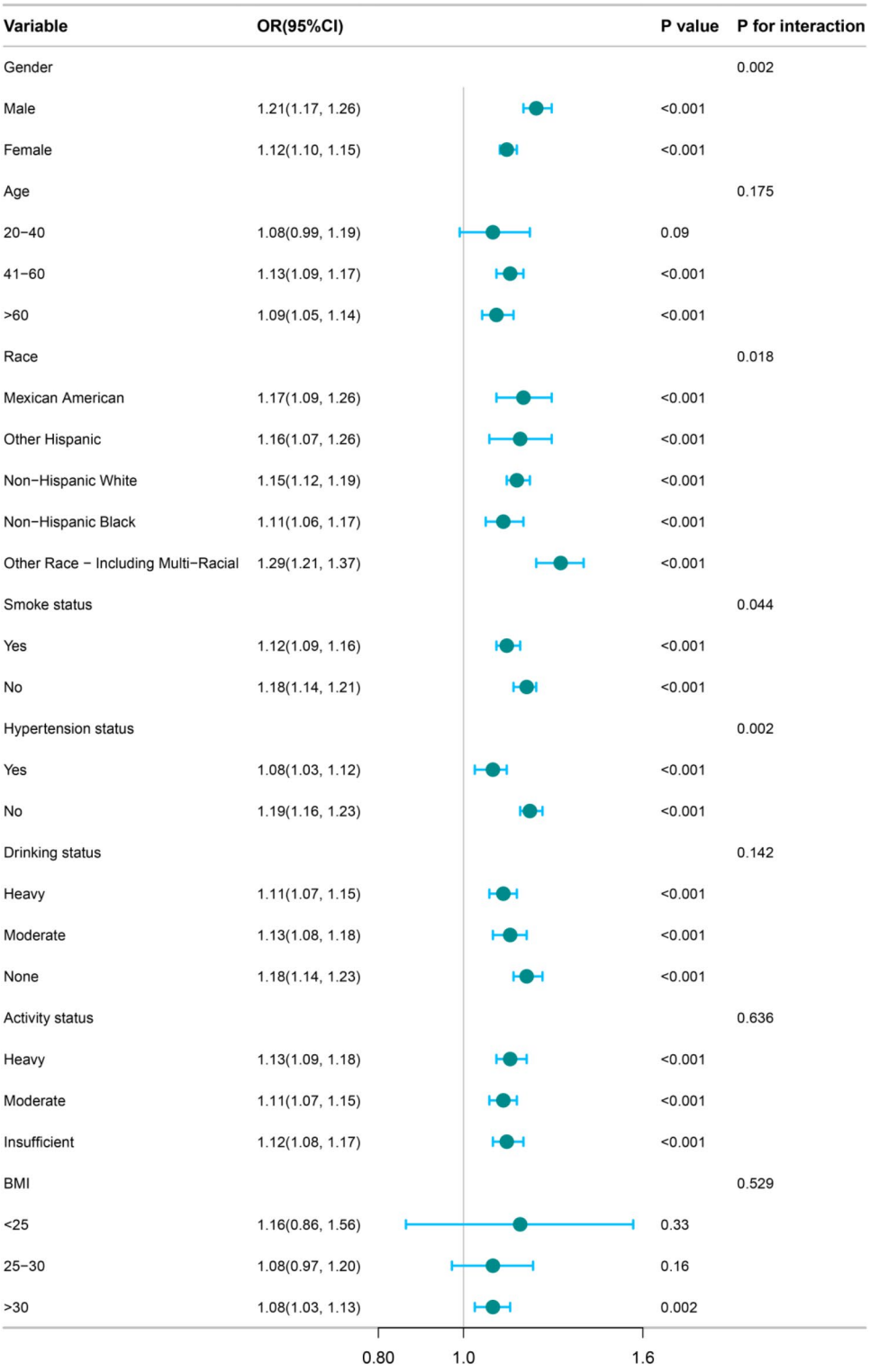

Fig. 4. Subgroup analysis of the relationship between BRI and OA. OR: Odds Ratio, CI: Confidence Interval, BMI: body mass index.

explore the implications of dual diagnosis. Additionally, while BRI shows promise as a risk assessment tool for joint diseases, further research is needed to compare its predictive ability with other anthropometric indices, such as waist-to-hip ratio and visceral fat area.

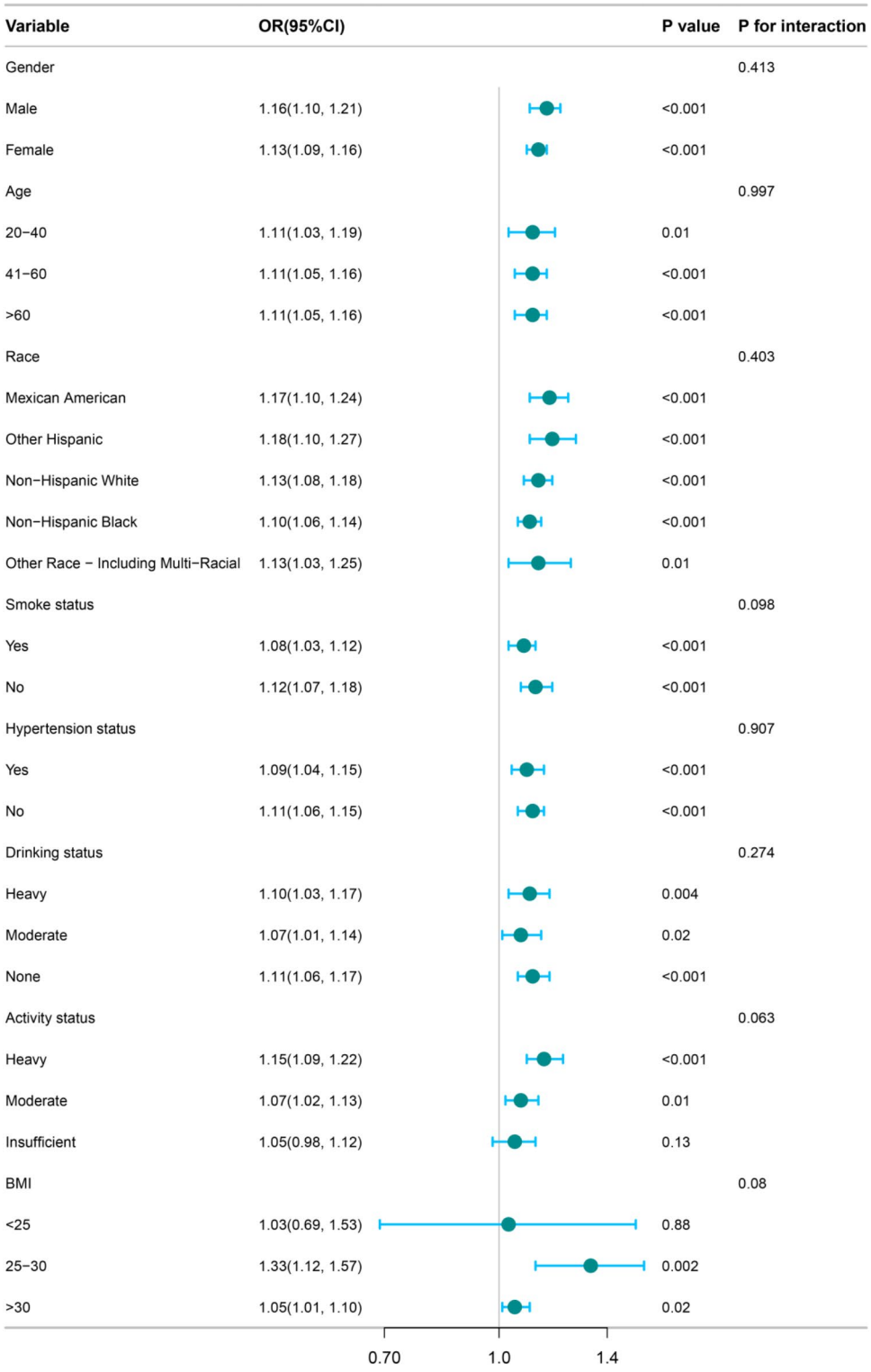

Fig. 5. Subgroup analysis of the relationship between BRI and RA. OR: Odds Ratio, CI: Confidence Interval, BMI: body mass index.

Conclusion

In conclusion, this study demonstrates a significant positive association between BRI and the prevalence of OA and, to a lesser extent, RA in American adults. BRI may serve as a valuable tool for assessing arthritis risk, particularly in populations with high levels of central obesity. Given the rising global burden of arthritis, incorporating BRI into clinical practice could help identify high-risk individuals and implement early interventions aimed at reducing obesity and inflammation. Future studies should focus on validating these

findings in diverse populations and exploring the potential for BRI to guide personalized prevention and treatment strategies for arthritis.

Data availability

All data are available in the NHANES database (www.cdc.gov/nchs/nhanes).

Received: 24 January 2025; Accepted: 14 March 2025
Published online: 20 March 2025

References

1. Pincus, T. et al. Osteoarthritis is as severe as rheumatoid arthritis: evidence over 40 years according to the same measure in each disease. *Clin. Exp. Rheumatol.* **37** (Suppl 120(5), 7–17 (2019).
2. Reginster, J. Y. The prevalence and burden of arthritis. *Rheumatology* **41** (Supp 1), 3–6 (2002).
3. Safiri, S. et al. Global, regional and National burden of osteoarthritis 1990–2017: a systematic analysis of the global burden of disease study 2017. *Ann. Rheum. Dis.* **79** (6), 819–828 (2020).
4. Sun, X. et al. Osteoarthritis in the Middle-Aged and elderly in China: prevalence and influencing factors. *Int. J. Env Res. Pub He.* **16** (23), 4701 (2019).
5. Abramoff, B., Caldera, F. E. & Osteoarthritis Pathology, diagnosis, and treatment options. *Med. Clin.* **104** (2), 293–311 (2020).
6. Almutairi, K., Nossent, J., Preen, D., Keen, H. & Inderjeeth, C. SAT0576 THE PREVALENCE OF RHEUMATOID ARTHRITIS: A SYSTEMATIC REVIEW OF POPULATION-BASED STUDIES. *Ann. Rheum. Dis.* **79** (Suppl 1), 1246 (2020).
7. Rudan, I. et al. Prevalence of rheumatoid arthritis in low- and middle-income countries: A systematic review and analysis. *J. Glob Health*. **5** (1), 10409 (2015).
8. Black, R. J. et al. Global, regional, and National burden of rheumatoid arthritis, 1990–2020, and projections to 2050: a systematic analysis of the global burden of disease study 2021. *Lancet Rheumatol.* **5** (10), e594–e610 (2023).
9. Godziuk, K. & Hawker, G. A. Obesity and body mass index: past and future considerations in osteoarthritis research. *Osteoarthr. Cartil.* **32** (4), 452–459 (2024).
10. Dubovyk, V. et al. Obesity is a risk factor for poor response to treatment in early rheumatoid arthritis: a NORD-STAR study. *Rmd Open.* **10** (2), e004227 (2024).
11. Batushansky, A. et al. Fundamentals of OA. An initiative of osteoarthritis and cartilage. Obesity and metabolic factors in OA. *Osteoarthr. Cartil.* **30** (4), 501–515 (2022).
12. Wang, T., He, C. & Yu, X. Pro-Inflammatory cytokines: new potential therapeutic targets for Obesity-Related bone disorders. *Curr. Drug Targets*. **18** (14), 1664–1675 (2017).
13. Gutin, I. Body mass index is just a number: conflating riskiness and unhealthiness in discourse on body size. *Sociol. Health Ill.* **43** (6), 1437–1453 (2021).
14. Xue, H. et al. Association of the visceral fat metabolic score with osteoarthritis risk: a cross-sectional study from NHANES 2009–2018. *Bmc Public. Health*. **24** (1), 2269 (2024).
15. Schweitzer, K. Could the body roundness index one day replace the BMI? *Jama-J Am. Med. Assoc.* **16** (332), 1317–1318 (2024).
16. Yang, M. et al. Body roundness index trajectories and the incidence of cardiovascular disease: evidence from the China health and retirement longitudinal study. *J. Am. Heart Assoc.* **13** (19), e034768 (2024).
17. Qiu, L., Xiao, Z., Fan, B., Li, L. & Sun, G. Association of body roundness index with diabetes and prediabetes in US adults from NHANES 2007–2018: a cross-sectional study. *Lipids Health Dis.* **23** (1), 252 (2024).
18. Konstantinova, E. D., Maslakova, T. A. & Ogorodnikova, S. Y. The predictive capability of several anthropometric indices for identifying the risk of metabolic syndrome and its components among industrial workers. *Sci. Rep-Uk.* **14** (1), 15327 (2024).
19. Whelton, P. K. et al. /ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2018; 71(19):e127-e248. (2017).
20. Willenbring, M. L., Massey, S. H. & Gardner, M. B. Helping patients who drink too much: an evidence-based guide for primary care clinicians. *Am. Fam Physician*. **80** (1), 44–50 (2009).
21. Sampath, S., Venkatesan, V., Ghosh, S., Kotikalapudi, N. & Obesity Metabolic syndrome, and Osteoarthritis-An updated review. *Curr. Obes. Rep.* **12** (3), 308–331 (2023).
22. Zhang, Y. et al. Visceral adipose tissue and osteoarthritis, a two-sample Mendelian randomized study. *Front. Med-Lausanne*. **10**, 1324449 (2023).
23. Kraus, V. B. et al. Direct in vivo evidence of activated macrophages in human osteoarthritis. *Osteoarthr. Cartil.* **24** (9), 1613–1621 (2016).
24. De Roover, A., Escribano-Núñez, A., Monteagudo, S. & Lories, R. Fundamentals of osteoarthritis: inflammatory mediators in osteoarthritis. *Osteoarthr. Cartil.* **31** (10), 1303–1311 (2023).
25. Aaboe, J., Bliddal, H., Messier, S. P., Alkjær, T. & Henriksen, M. Effects of an intensive weight loss program on knee joint loading in obese adults with knee osteoarthritis. *Osteoarthr. Cartil.* **19** (7), 822–828 (2011).
26. Brown, P., Pratt, A. G. & Hyrich, K. L. Therapeutic advances in rheumatoid arthritis. *Bmj-Brit Med. J.* **384**, e070856 (2024).
27. Li, R. et al. FTO-mediated RNA m(6)A methylation regulates synovial aggression and inflammation in rheumatoid arthritis. *Bba-Mol Basis Dis.* **1870** (7), 167341 (2024).
28. Qin, B. et al. Body mass index and the risk of rheumatoid arthritis: a systematic review and dose-response meta-analysis. *Arthritis Res. Ther.* **17** (1), 86 (2015).
29. Ohno, T., Aune, D. & Heath, A. K. Adiposity and the risk of rheumatoid arthritis: a systematic review and meta-analysis of cohort studies. *Sci. Rep-Uk.* **10** (1), 16006 (2020).
30. Chung, M. K. et al. DATA FROM A NATIONWIDE COHORT. *Ann. Rheum. Dis.* **82** (Suppl 1), 601 (2023).
31. Jalal, A. M., Mawlood, Z. K., Aref, S. F. & Al-Nimer, M. S. Association between the quality of life domains with anthropometric and adiposity indices in rheumatoid arthritis: A Cross-Sectional study from a single centre in Erbil-Iraq. *Mediterr. J. Rheumatol.* **35** (2), 247–254 (2024).
32. Tsoi, M. F. et al. Association between body weight and tocilizumab effectiveness in rheumatoid arthritis: results from the BSRBR-RA. Rheumatology. :keae500. (2024).
33. Baker, J. F., Reed, G., Poudel, D. R., Harrold, L. R. & Kremer, J. M. Obesity and response to advanced therapies in rheumatoid arthritis. *Arthrit Care Res.* **74** (11), 1909–1916 (2022).
34. Marchand, N. E. et al. Long-term weight changes and risk of rheumatoid arthritis among women in a prospective cohort: a marginal structural model approach. *Rheumatology* **61** (4), 1430–1439 (2022).
35. Ferguson, L. D. et al. Association of central adiposity with psoriasis, psoriatic arthritis and rheumatoid arthritis: a cross-sectional study of the UK biobank. *Rheumatology* **58** (12), 2137–2142 (2019).
36. Tsigalou, C., Vallianou, N. & Dalamaga, M. Autoantibody production in obesity: is there evidence for a link between obesity and autoimmunity?? *Curr. Obes. Rep.* **9** (3), 245–254 (2020).

37. Zhang, X. et al. Body roundness index and All-Cause mortality among US adults. *Jama Netw. Open.* **7** (6), e2415051 (2024).
38. Rico-Martín, S. et al. Effectiveness of body roundness index in predicting metabolic syndrome: A systematic review and meta-analysis. *Obes. Rev.* **21** (7), e13023 (2020).
39. Calderón-García, J. F. et al. Effectiveness of body roundness index (BRI) and a body shape index (ABSI) in predicting hypertension: A systematic review and Meta-Analysis of observational studies. *Int. J. Env Res. Pub He.* **18** (21), 11607 (2021).
40. Li, C. I. et al. Association of body indices and risk of mortality in patients with type 2 diabetes. *Bmj Open. Diab Res. Ca.* **11** (4), e003474 (2023).

Acknowledgements
Thanks to NHANES database participants and staff.

Author contributions
Tao Gao and Chao Wu analyzed and interpreted the patient data regarding the OA. Tao Gao were major contributors in writing the manuscript. Zhi-Yu Chen, Tao Li, Xu Lin, Hai-Gang Hu and Jian-Dong Tang assisted with data collection. All authors read and approved the final manuscript.

Declarations

Competing interests
The authors declare no competing interests.

Ethics approval and consent to participate
The study protocol was approved by the NCHS Ethics Review Board, and all participants provided informed consent.

Additional information
Correspondence and requests for materials should be addressed to T.G. or C.W.