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Association between obesity measurement indexes and symptomatic knee osteoarthritis among the Chinese population: analysis from a nationwide longitudinal study

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Abstract

Background The current literature lacks robust clinical data and evidence delineating the relationship between obesity measurement indexes and knee osteoarthritis (KOA). Consequently, this investigation seeks to elucidate the potential link between obesity measurement indexes and KOA among Chinese adults in a nationally representative study.

Methods Firstly, this research performed an observational study in the China Health and Retirement Longitudinal Study (CHARLS). The variables were extracted from interviews and compared between KOA and non-KOA participants. The relationship between obesity measurement indexes and KOA was analyzed by multivariate logistic regression. Restricted cubic spline (RCS) regression tests the nonlinearity of the relationship between obesity measurement indexes and KOA. Subgroup analyses were performed by sex to verify the robustness of the findings.

Results In this cross-sectional analysis, we found a positive association between obesity measurement indexes and KOA. These results did not change on multiple imputations(BMI: OR = 1.02, 95% CI, 1.01–1.04, $P < 0.05$; WHtR: OR = 2.85, 95% CI, 1.08–7.51, $P < 0.05$; BRI: OR = 1.07, 95% CI, 1.01–1.12, $P < 0.05$; BFP: OR = 1.02 95% CI, 1.00-1.03, $P < 0.05$). All the effects of obesity measurement indexes with KOA are present in females. None of the stratifying variables significantly affected the association between obesity measurement indexes and KOA. RCS regression test revealed the linear positive correlation between obesity measurement indexes and KOA.

Conclusion In this cross-sectional study, we found a significant association between obesity measurement indexes and KOA. This relationship is not affected by stratification and confounding factors.

Keywords Knee osteoarthritis, Obesity measurement indexes, CHARLS, Cross-sectional survey

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Background

Knee osteoarthritis (KOA) is a prevalent chronic joint disease characterized by clinical manifestations including joint pain, dysfunction, deformity, and muscle atrophy, significantly impacting patients' quality of life [1–3]. Epidemiological data indicates that KOA affects over 300 million individuals worldwide, imposing substantial economic burdens on patients and society [4]. Numerous established risk factors, such as advanced age, post-menopausal age in women, articular injuries, genetic predisposition, metabolic disorders, and inflammation [5–7]. Obesity is one of the significant risk factors for KOA, primarily involving increased mechanical load on the knee joints due to obesity and the participation of various adipokines and inflammatory cytokines secreted by adipose tissue in cartilage degradation, synovial membrane inflammation, and bone erosion, which subsequently lead to the development of KOA [8, 9]. The pathogenesis of KOA is multifaceted, involving intricate interactions among bone, muscle, and tendon, yet the underlying mechanisms remain incompletely elucidated [10]. For instance, the infrapatellar fat pad, as an adipose tissue that secretes adipokines, exhibits a correlation between its size and the progression of KOA, and it is considered a source and center of inflammation in KOA [11, 12]. Consequently, investigating the precise pathogenesis and identifying effective treatment modalities represent critical research priorities globally.

Obesity measurement indices are essential tools for assessing overweight and obesity in individuals. These indices encompass body mass index (BMI), waist-to-height ratio (WHtR), Body Adiposity Index (BAI), and Body Fat Percentage (BFP). They evaluate body fat content and distribution by measuring weight, height, and specific body circumferences. BMI is a widely used indicator of obesity despite a notable limitation: It cannot distinguish between individuals with similar BMI values but different body fat percentages. However, when performing correlation analyses between disease risk factors and obesity measures, it is imperative to also account for differences in body composition or body fat distribution. BRI effectively addresses this gap [13]. Specifically, BFP offers insights into body composition, whereas WHtR serves as an indicator of abdominal fat accumulation, highlighting variations in body fat distribution [14]. This information is critical for healthcare professionals and individuals managing their health, aiding in assessing health risks and guiding strategies for health improvement and disease prevention. Studies have shown the coexistence of obesity and osteoarthritis (OA) [15, 16]. For instance, animal experiments have demonstrated that disruptions in high-density lipoprotein metabolism induced by high-fat diets may increase OA incidence [17]. This underscores obesity as a significant risk factor for the onset and progression of OA, with KOA being particularly prevalent. However, Previous studies have largely been confined to utilizing BMI as the primary indicator for assessing obesity. For instance, *Long et al.*. conducted a cohort study and found that obesity (assessed by BMI) is associated with an increased risk of symptomatic KOA progression [18]. *Raud et al.*., through a cross-sectional study, revealed a dose-response relationship between the degree of obesity (BMI) and clinical outcomes of KOA [19]. *Khan et al.*, leveraging real-world data, discovered a significant correlation between obesity and KOA, with obese individuals (BMI > 25 kg/m^2) being at high risk for the progression of KOA [20]. *Larsen et al.*., through observational studies, observed that patients' Knee Injury and Osteoarthritis Outcome Scores tend to increase with rising levels of obesity (BMI) [21]. The limitation of this approach lies in its inability to assess differences among individuals with similar BMI values but varying degrees of body fat, nor can it account for variations in body composition or fat distribution. Meanwhile, there is a lack of research on the relationship between obesity indicators and symptomatic KOA in the Chinese population. Therefore, this study seeks to explore the association between KOA and multiple types of obesity measures among the Chinese population.

The China Health and Retirement Longitudinal Study (CHARLS) is a representative multi-level and multi-dimensional project, its continuity is due to the organization's annual survey of a sample of the Chinese population [22]. A survey based on CHARLS reveals that symptomatic KOA is prevalent in China and that the prevalence of symptomatic KOA varies across sociodemographic, economic, and geographic factors. The study underscores the necessity of researching the risk factors influencing the prevalence of symptomatic KOA in China [23]. Thus, CHARLS has offered a nationally representative and high-quality sample to investigate the relationship between obesity measurement indexes and symptomatic KOA.

In this research, we aim to demonstrate the relationship between obesity measurement indexes and symptomatic KOA in a national sample of the Chinese population. The results of our study will lead to further treatment and prevention of KOA. Therefore, we performed an observational study via CHARLS.

Materials and methods

Data and study population

CHARLS data were collected and screened for the cross-sectional study. CHARLS is a biannual, nationally representative longitudinal survey conducted by the China Center for Economic Research at Peking University.

With the assistance of 28 provincial Centers for Disease Control and Prevention (CDC), the CHARLS office in Beijing first collected the contact lists and information of county-level CDC liaisons. Prior to the arrival of the enumerators for mapping in the selected three villages/communities, the county-level CDC liaisons communicated with the responsible persons of the three selected villages/communities to facilitate enumerators in coordinating with local village/community leaders and residents to participate in the survey.

Participants signed the informed consent form. We combined data from the 2011–2012 and 2015–2016 waves of CHARLS. The CHARLS study protocol was approved by the Ethics Review Committee of Peking University (IRB00001052-11015), and all participants signed written informed consent.

All participants participated in a structured, face-to-face household interview utilizing a comprehensive questionnaire. In essence, the national baseline survey adopted a multi-stage probability sampling strategy, incorporating Probability Proportional to Size (PPS) techniques. This sampling methodology encompassed four distinct stages: county-level sampling, community-level sampling, household-level sampling, and respondent-level sampling, to ensure a representative national sample. During the county-level sampling phase, 150 counties, representative of 28 provinces, were meticulously chosen. In the community-level sampling, rural administrative villages and urban communities were designated as Primary Sampling Units (PSUs), with three PSUs being randomly selected from each county. At the household-level sampling stage, dwellings were selected based on detailed maps and lists provided by respective public service units. Ultimately, during the respondent-level sampling, one individual aged 45 or older was randomly picked from a designated household to serve as the key respondent [22].

Symptomatic KOA diagnosis and obesity indexes measurement of the cross-sectional study

The outcome variable of this study was whether the participants were diagnosed with symptomatic KOA. The diagnosis of symptomatic KOA was based on a CHARLs-based epidemiological survey of symptomatic KOA, symptomatic KOA was defined as physician-diagnosed arthritis combined with pain in either knee joint [23]. The diagnostic criteria for symptomatic KOA in all previous CHARls-based studies were developed according to this criterion [24, 25]. First of all, participants were asked whether he/she had been diagnosed with arthritis by a doctor. The presence of knee pain was assessed based on responses to the following question: "Do you often suffer from pain in any part of your body?" If participants answered yes, the following question was asked: "Where in your body do you feel pain? Please list all parts of body you are currently feeling pain". If the subject answered "yes" to these questions, and the knee joint was present at the pain site, we diagnosed it as symptomatic KOA.

The same doctor took anthropometry for all patients, including height, weight, waist circumference, etc., and calculated obesity measurement indexes: BMI = weight (kg)/height (m^2), WHtR = waist circumference / height, BRI=(lower body length/upper body length) × 100, Adult male: BFP = 1.20 × BMI + 0.23 × age − 16.2, Adult women: BFP = 1.20 × BMI + 0.23 × age − 5.4 [13, 26]. Height was measured using the SecaTM213 stadiometer (Medical Scales And Measuring Systems Seca (Hangzhou) Co., Ltd.), while weight was measured using the OmronTM HN-286 scale (Krell Precision (Yangzhou) Co., Ltd.). Waist circumference was measured with a flexible tape measure.

Covariate ascertainment of the cross-sectional study

Concomitant variables of this cross-sectional study included sex, age, race, education, material status, residence place, fast triglyceride (TG), fast total cholesterol (TC), LDL cholesterol, HDL cholesterol, smoking, drinking, diabetes, hypertension, hyperlipidemia, cancer, and stroke. Covariates were obtained from structured questionnaires except TG, TC, LDL cholesterol, and HDL cholesterol. Serum lipids were analyzed via enzymatic colormetric test.

Statistical analysis

Continuous variables are expressed as mean (95% confidence intervals (CI)) and categorical variables as count (percentage). The relationship between obesity measurement indexes and symptomatic KOA was analyzed via logistical regression. The confounding factors were added to the multivariate logistic model to adjust the relationship analysis. We also applied restricted cubic spline (RCS) regression to examine the nonlinearity of the association between obesity measurement indexes and symptomatic KOA. Subgroup analyses in terms of sex were conducted to examine the presence of significant interactions of these covariates with the association between obesity measurement indexes and symptomatic KOA. Odds ratios (OR) with 95% CI and P values were calculated. Statistical analysis of all data was performed by R Studio (https://ropensci.org/blog/2021/11/16/how-to-cite-r-and-r-packages/). A 2-tailed $P < 0.05$ was regarded as significant.

Results

Characteristics of the cross-sectional study participants

Among the 38,563 eligible participants, we excluded data containing null values. Finally, 7598 participants were included in the analysis. The study procedure is illustrated in Fig. 1. Among these included participants, 985 were diagnosed with symptomatic KOA, and non- symptomatic KOA were 6613. The general characteristics are shown in Table 1. Overall, symptomatic KOA individuals were older ($P<0.0001$), a high proportion of females ($P<0.0001$), rural ($P<0.0001$), and had higher obesity measurement indexes.

Relationship between obesity measurement indexes and symptomatic KOA

A logistic regression model was established to investigate the relationship between obesity measurement indexes and symptomatic KOA, as shown in Table 2. The crude model was a univariate logistic regression model that showed a positive association between obesity measurement indexes and symptomatic KOA (WHtR: OR = 8.32 95% CI, 3.41–20.31, $P<0.0001$; BRI: OR = 1.13, 95% CI, 1.08–1.18, $P<0.0001$; BFP: OR = 1.04, 95% CI, 1.03–1.05, $P<0.0001$). This association was not altered after adjusting for sex, age, education, marital status, and residence place in model 1(WHtR: OR = 3.04, 95% CI, 1.22–7.59, $P<0.05$; BRI: OR = 1.07, 95% CI, 1.02–1.12, $P<0.05$; BFP: OR = 1.02 95% CI, 1.00-1.03, $P<0.05$). Model 1 showed a positive association between BMI and KOA (OR = 1.02, 95% CI, 1.01–1.04, $P<0.05$). These associations were not altered after adjusting for several factors in model 2 and 3 (Model 2: BMI: OR = 1.02, 95% CI, 1.01–1.04, $P<0.05$; WHtR: OR = 3.26, 95% CI, 1.31–8.15, $P<0.05$; BRI: OR = 1.07, 95% CI, 1.02–1.12, $P<0.01$; BFP: OR = 1.02 95% CI, 1.00-1.03, $P<0.05$.Model 3: BMI: OR = 1.02, 95% CI, 1.01–1.04, $P<0.05$; WHtR: OR = 2.85, 95% CI, 1.08–7.51, $P<0.05$; BRI: OR = 1.07, 95% CI, 1.01–1.12, $P<0.05$; BFP: OR = 1.02 95% CI, 1.00-1.03, $P<0.05$).

The crude model is unadjusted. Model 1 is adjusted for sex, age, education, marital status, and residence place. Model 2 is adjusted for sex, age, education, marital status, residence place, drink, and smoke. Model 3 is adjusted for sex, age, education, marital status, residence place, drink, smoke, cancer, diabetes, hyperlipidemia, hypertension, stroke, TC, HDL, LDL, and TG.

Subgroup analysis

To verify whether the relationship between obesity measurement indexes and symptomatic KOA remained stable across genders, we performed subgroup analyses.

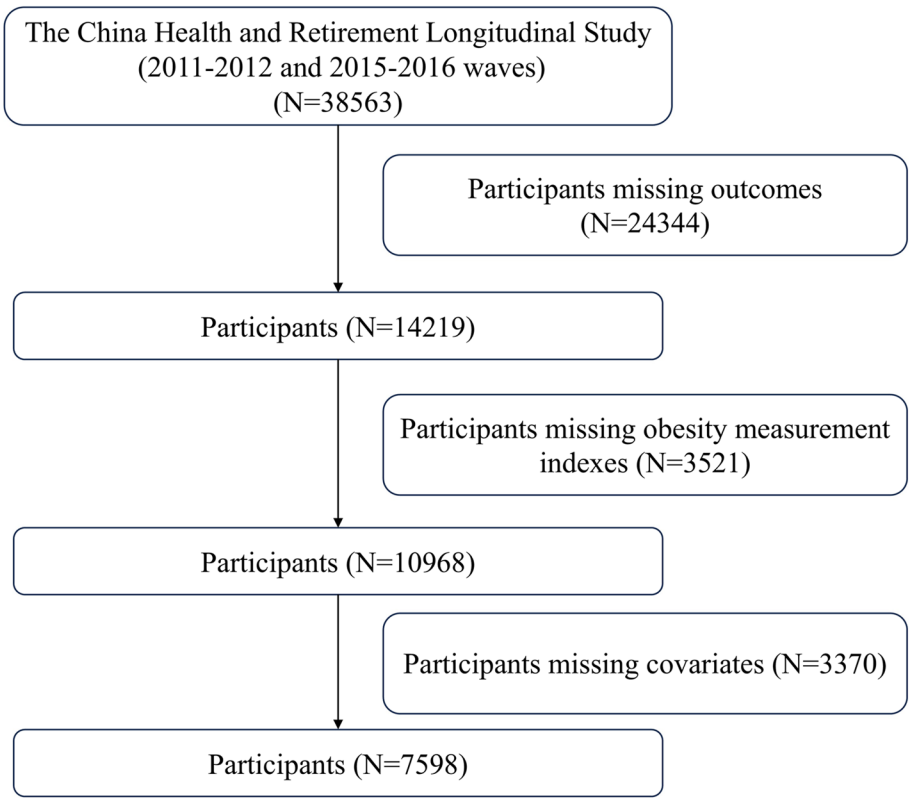

Fig. 1 Flowchart depicting the participants' selection

Table 1 General characteristics of participants

Variables	Total (*n* = 7598)	Non- symptomatic KOA (*n* = 6613)	Symptomatic KOA (*n* = 985)	Statistic	*P* value
Sex				102.594	**< 0.0001**
Female	3952(52.014)	3291(49.766)	661(67.107)		
Male	3646(47.986)	3322(50.234)	324(32.893)		
Age	59.456 ± 9.365	59.264 ± 9.418	60.747 ± 8.901	-4.842	**< 0.0001**
Marital status				18.297	**< 0.01**
Cohabitated	2(0.026)	1(0.015)	1(0.102)		
Divorced	59(0.777)	53(0.801)	6(0.609)		
Married but not living with spouse temporarily	301(3.962)	269(4.068)	32(3.249)		
Married with spouse present	6367(83.798)	5562(84.107)	805(81.726)		
Never married	46(0.605)	40(0.605)	6(0.609)		
Separated	35(0.461)	25(0.378)	10(1.015)		
Widowed	788(10.371)	663(10.026)	125(12.690)		
Education				140.380	**< 0.0001**
Bachelor's degree	40(0.526)	39(0.590)	1(0.102)		
College	87(1.145)	87(1.316)			
Did not finish primary school	1378(18.136)	1138(17.209)	240(24.365)		
High school	545(7.173)	509(7.697)	36(3.655)		
Illiterate	2119(27.889)	1753(26.508)	366(37.157)		
Master's degree	1(0.013)	1(0.015)			
Middle school	1574(20.716)	1445(21.851)	129(13.096)		
Primary school	1705(22.440)	1497(22.637)	208(21.117)		
Vocational school	149(1.961)	144(2.178)	5(0.508)		
Residence place				86.720	**< 0.0001**
Rural	4756(62.595)	4007(60.593)	749(76.041)		
Urban	2842(37.405)	2606(39.407)	236(23.959)		
TG (mg/dL)	132.618 ± 97.586	132.310 ± 96.797	134.690 ± 102.752	-0.683	0.495
TC (mg/dL)	193.532 ± 38.694	193.480 ± 38.726	193.876 ± 38.499	-0.301	0.764
HDL (mg/dL)	51.087 ± 15.321	50.981 ± 15.338	51.798 ± 15.195	-1.573	0.116
LDL (mg/dL)	116.574 ± 35.260	116.660 ± 35.347	115.994 ± 34.682	0.561	0.575
Diabetes				4.552	**0.033**
No	6426(84.575)	5616(84.924)	810(82.234)		
Yes	1172(15.425)	997(15.076)	175(17.766)		
Hyperlipidemia				0.646	0.422
No	5742(75.573)	4987(75.412)	755(76.650)		
Yes	1856(24.427)	1626(24.588)	230(23.350)		
Hypertension				10.361	**< 0.01**
No	4473(58.871)	3940(59.580)	533(54.112)		
Yes	3125(41.129)	2673(40.420)	452(45.888)		
Cancer				19.324	**< 0.0001**
No	7532(99.131)	6568(99.320)	964(97.868)		
Yes	66(0.869)	45(0.680)	21(2.132)		
Stroke				7.590	**< 0.01**
No	7455(98.118)	6500(98.291)	955(96.954)		
Yes	143(1.882)	113(1.709)	30(3.046)		
Smoke				22.215	**< 0.0001**
No	4550(59.884)	3892(58.854)	658(66.802)		
Yes	3048(40.116)	2721(41.146)	327(33.198)		
Drink				9.655	**< 0.01**

Table 1 (continued)

Variables	Total (*n* = 7598)	Non- symptomatic KOA (*n* = 6613)	Symptomatic KOA (*n* = 985)	Statistic	*P* value
No	4417(58.134)	3799(57.447)	618(62.741)		
Yes	3181(41.866)	2814(42.553)	367(37.259)		
Weigh (kg)	59.286 ± 11.783	59.563 ± 11.702	57.423 ± 12.157	5.178	**< 0.0001**
Height (cm)	158.222 ± 8.507	158.663 ± 8.453	155.265 ± 8.284	11.979	**< 0.0001**
Waist circumference (cm)	84.579 ± 12.473	84.559 ± 12.435	84.715 ± 12.732	-0.359	0.720
BMI (kg/m^2)	23.606 ± 3.913	23.588 ± 3.872	23.733 ± 4.178	-1.029	0.304
WHtR	0.536 ± 0.080	0.534 ± 0.080	0.547 ± 0.084	-4.487	**< 0.0001**
BFP	30.334 ± 8.119	30.013 ± 8.085	32.488 ± 8.026	-9.022	**< 0.0001**
BRI	4.175 ± 1.503	4.139 ± 1.481	4.411 ± 1.624	-4.952	**< 0.0001**

TG Triglyceride, *TC* Total cholesterol, *HDL* High-density lipoprotein cholesterol, *LDL* Low-density lipoprotein cholesterol, *BMI* Body Mass Index, *WHtR* Waist-to-height ratio, *BFP* Body fat percentage, *BRI* Body roundness index

Table 2 Association between obesity measurement indexes and symptomatic KOA

Variables	Crude model		Model 1		Model 2		Model 3	
	95%CI	*P*	95%CI	*P*	95%CI	*P*	95%CI	*P*
BMI	1.01(0.99,1.03)	0.28	1.02(1.01,1.04)	0.01	1.02(1.01,1.04)	0.01	1.02(1.00,1.04)	0.03
WHtR	8.32(3.41,20.31)	< 0.0001	3.04(1.22,7.59)	0.02	3.26(1.31,8.15)	0.01	2.85(1.08,7.51)	0.03
BRI	1.13(1.08,1.18)	< 0.0001	1.07(1.02,1.12)	0.01	1.07(1.02,1.12)	0.004	1.07(1.01,1.12)	0.01
BFP	1.04(1.03,1.05)	< 0.0001	1.02(1.00,1.03)	0.02	1.02(1.00,1.03)	0.01	1.02(1.00,1.03)	0.04

The crude model is unadjusted. Model 1 is adjusted for sex, age, education, marital status, and residence place. Model 2 is adjusted for sex, age, education, marital status, residence place, drink, and smoke. Model 3 is adjusted for sex, age, education, marital status, residence place, drink, smoke, cancer, diabetes, hyperlipidemia, hypertension, stroke, TC, HDL, LDL, and TG

BMI Body Mass Index, *WHtR* Waist-to-height ratio, *BFP* body fat percentage, *BRI* Body roundness index

As illustrated in Table 3, the effects of WHtR, BRI, and symptomatic KOA are present in females. None of the stratifying variables significantly affected the association between obesity measurement indexes and symptomatic KOA (all *P* for interaction > 0.05).

By constructing RCS, we further understand whether there is a nonlinear relationship between these relationships. In the relationship between obesity measurement indexes and symptomatic KOA, we observed a significant linear positive relationship (all *P* nonlinearities > 0.05) (Fig. 2).

Discussion

This cross-sectional study concluded that obesity measurement indexes were associated with symptomatic KOA. The relationship remained the same even after other factors were added (demographic factors and smoking, drinking, diabetes, hypertension, et al.).

The etiology of KOA remains incompletely understood, significantly impacting patients' quality of life with symptoms including recurrent knee pain, impaired mobility, and potential disability [27, 28]. With the escalating prevalence of adult obesity, it has emerged as a key contributor to the rising incidence of KOA [16, 29–31]. This dual burden not only imposes physical discomfort on individuals but also escalates societal economic costs [32]. Numerous studies on the correlation between obesity and KOA have confirmed that obesity can lead to the occurrence or exacerbation of KOA. Lotte Meert et al. identified a potential link between adipose tissue mass and alterations in somatosensory function in KOA patients [33]. Margreth Grotle et al. reported a substantial association between higher BMI and KOA [34]. However, these studies are largely limited to using BMI as the indicator for assessing obesity [18–21]. The limitations of this approach, as we have previously mentioned, are that it fails to evaluate differences among individuals with similar BMI values but varying degrees of body fat, and it does not account for variations in body composition or fat distribution. Although there are few studies on the relationship between WHtR, BAI, BFP and symptomatic KOA, there are some studies on the correlation between other knee diseases or discomfort symptoms and these indicators. The increase in BFP may be related to increased pain sensitivity in individuals with knee pain, and the tibial cartilage thickness decreases

Table 3 Association between obesity measurement indexes and symptomatic KOA under stratification conditions

Variables	Stratification	Crude model			Model 1			Model 2			Model 3		
		95% CI	*P*	*P* for interaction	95% CI	*P*	*P* for interaction	95% CI	*P*	*P* for interaction	95% CI	*P*	*P* for interaction
BMI	Sex			0.885			0.269			0.267			0.287
	Male	1.000(0.969,1.031)	0.996		1.037(1.004,1.069)	0.024		1.039(1.006,1.071)	0.016		1.033(0.997,1.069)	0.066	
	Female	0.997(0.977,1.018)	0.801		1.016(0.994,1.037)	0.145		1.017(0.996,1.039)	0.113		1.016(0.993,1.039)	0.164	
WHtR	Sex			0.123			0.528			0.531			0.501
	Male	0.910(0.193,4.622)	0.908		2.051(0.376,11.999)	0.417		2.257(0.408,13.404)	0.361		1.545(0.260,10.188)	0.642	
	Female	4.135(1.455,12.092)	0.009		3.245(1.124,9.661)	0.032		3.696(1.280,11.001)	0.017		3.805(1.233,12.170)	0.022	
BFP	Sex			0.700			0.811			0.747			0.883
	Male	1.005(0.980,1.029)	0.706		1.024(0.998,1.050)	0.062		1.026(1.000,1.052)	0.044		1.020(0.991,1.049)	0.175	
	Female	1.011(0.993,1.028)	0.233		1.014(0.996,1.032)	0.115		1.016(0.998,1.034)	0.087		1.017(0.997,1.037)	0.089	
BRI	Sex			0.126			0.645			0.670			0.617
	Male	0.997(0.911,1.092)	0.953		1.053(0.958,1.157)	0.283		1.059(0.963,1.165)	0.234		1.036(0.935,1.149)	0.497	
	Female	1.082(1.027,1.141)	0.003		1.068(1.012,1.127)	0.017		1.074(1.018,1.134)	0.010		1.077(1.016,1.141)	0.012	

The crude model is unadjusted. Model 1 is adjusted for age, education, marital status, and residence place. Model 2 is adjusted for age, education, marital status, residence place, drink, and smoke. Model 3 is adjusted for age, education, marital status, residence place, drink, smoke, cancer, diabetes, hyperlipidemia, hypertension, stroke, TC, HDL, LDL, and TG

BMI Body Mass Index, *WHtR* Waist-to-height ratio, *BFP* Body fat percentage, *BRI* Body roundness index

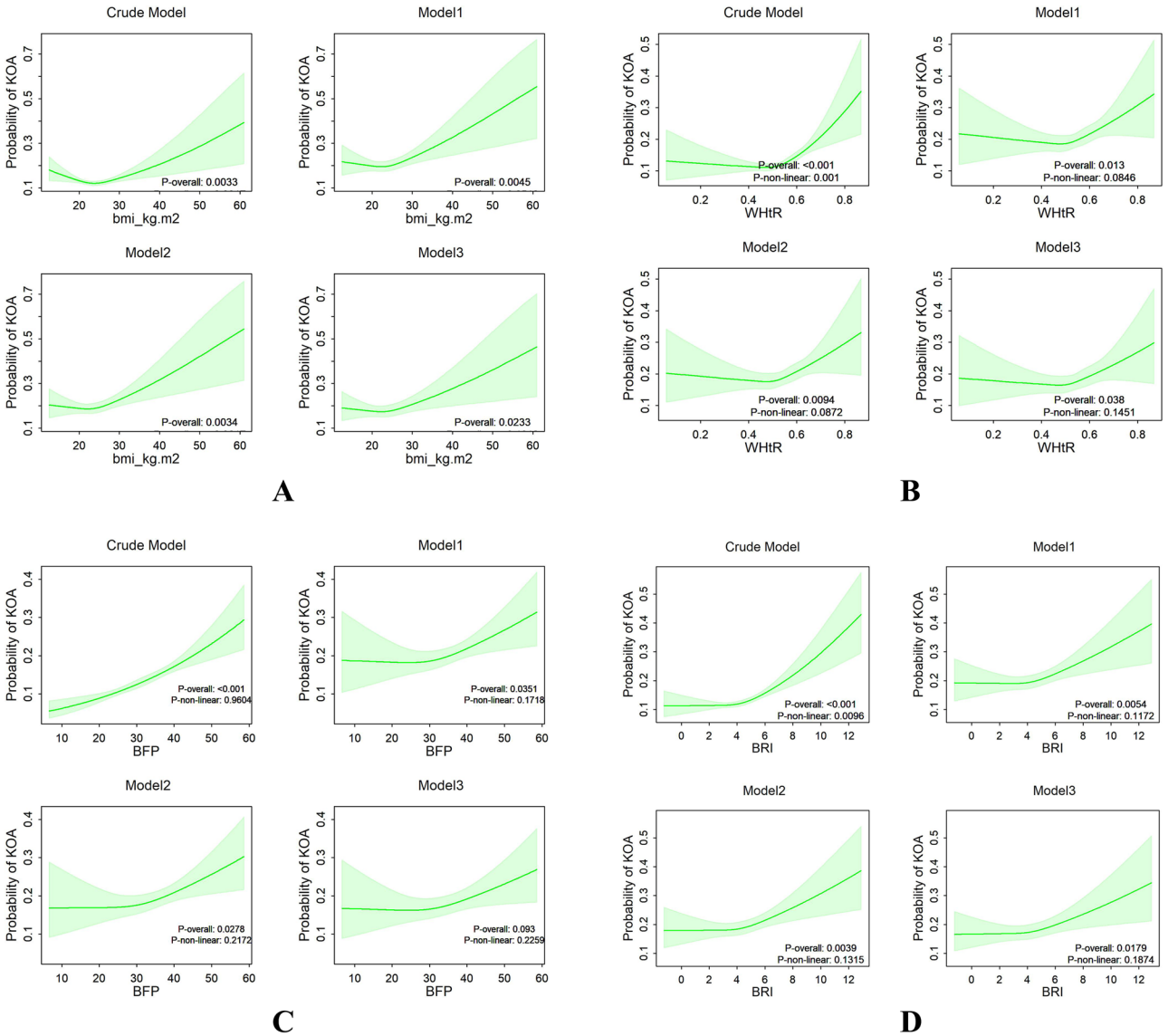

Fig. 2 The RCS curve of the association between obesity measurement indexes and symptomatic KOA. The crude model is unadjusted. Model 1 is adjusted for sex, age, education, marital status, and residence place. Model 2 is adjusted for sex, age, education, marital status, residence place, drink, and smoke. Model 3 is adjusted for sex, age, education, marital status, residence place, drink, smoke, cancer, diabetes, hyperlipidemia, hypertension, stroke, TC, HDL, LDL, and TG. **A** The RCS curve of the association between BMI and symptomatic KOA in different logistic models. **B** The RCS curve of the association between WHtR and symptomatic KOA in different logistic models. **C** The RCS curve of the association between BFP and symptomatic KOA in different logistic models. **D** The RCS curve of the association between BRI and symptomatic KOA in different logistic models

with the increase in BFP. A significant positive association was found between central obesity (WHtR) and new radiographic KOA, but the association was no longer statistically significant after adjustment for concomitant variables, and the results were similar in men and women [35, 36]. An epidemiological study based on CHARLS indicates that the prevalence of symptomatic KOA is higher among Chinese women than men [23]. However, it remains unknown whether obesity has a greater impact on the development of symptomatic KOA in Chinese women compared to men. A cross-sectional study has demonstrated that among middle-aged and elderly women in China, obesity exhibits both additive and multiplicative effects on knee joint pain and may potentially amplify the influence of reproductive and hormonal factors on KOA [37]. This aligns with the perspective of Hussain SM et al., who propose that obesity may create an inflammatory milieu, thereby facilitating

the onset and progression of OA [38]. By altering body weight, reproductive factors may contribute to underlying mechanisms and potentially mediate the relationship between endogenous hormones and joint-specific OA. Furthermore, the knee joint is more susceptible to the impact of obesity-related metabolic and inflammatory factors in women, as well as mechanical loads. Lauren M. Abbate et al. corroborated it, who emphasized BMI's strong correlation with KOA in women [39]. A study of a Dutch population suggests that moderate weight loss may reduce the risk of developing KOA in middle-aged overweight and obese women [40]. There is still a lack of systematic analysis (i.e., multi-level analysis through multiple obesity measures) of the effect of sex differences on the relationship between obesity and KOA in the Chinese population. Our study confirmed that obesity significantly affects symptomatic KOA in female population by gender stratification.

Previous studies underscored obesity as a pivotal risk factor in KOA pathogenesis. It is mainly due to biomechanical reasons, such as changes in joint load and cartilage wear [41]. Kristine Godziuk et al.'s research highlighted the biomechanical impact of increased joint loading on cartilage degradation and subchondral bone changes [42]. Apart from mechanical factors, various adipokines secreted by adipose tissue can promote cartilage degeneration through complex interactions. Additionally, obesity induces a shift in the cellular repertoire of resident macrophages, transitioning from an anti-inflammatory M2 phenotype to a pro-inflammatory M1 phenotype, thereby stimulating the production of pro-inflammatory cytokines [43]. An animal study found that rats induced by a high fat/high sucrose (HFS) diet developed KOA, and the extent of KOA lesions was controlled after exercise [44]. Our study further elucidates the robust correlation between obesity indices and KOA risk, elucidating that elevated obesity indices exacerbate knee joint wear-and-tear, precipitating cartilage degradation and arthritic changes. These findings underscore the critical importance of managing obesity indices in KOA prevention and treatment strategies.

CHARLS data are acquired and screened according to a standardized unified procedure, ensuring the accuracy and consistency of the results. The results were reliable because they were based on a large number of community samples. Comparing regression analysis to mechanism research, it is more intuitive and comprehensive to include some important confounding factors.

Nonetheless, it is crucial to recognize the inherent limitations of this study. Firstly, the CHARLS dataset relies heavily on self-reported diagnoses of certain diseases by participants, a method susceptible to misclassification stemming from memory inaccuracies. Secondly, the CHARLS study participants did not undergo radiological evaluations, and the diagnosis of symptomatic KOA was solely based on participants' self-reported knee pain and physicians' clinical diagnosis of arthritis. While the definition of symptomatic KOA has been established and validated through prior research, the risk of misclassification in the context of this study cannot be overlooked.

We accounted for numerous confounding factors in our analysis, albeit excluding those that were unmeasured. Additionally, participants with incomplete covariate data were omitted, which alongside the unmeasured factors, could potentially introduce bias into our findings. Lastly, given that the study participants were exclusively Chinese, caution is warranted when generalizing the results to other countries or ethnic groups worldwide. In the following studies, to further strengthen and verify our results, a large-scale cohort study and mechanism research are required to complete the validation.

Conclusion

In this cross-sectional study, our findings reveal a significant positive linear correlation between obesity indices (BMI, WHtR, BRI, BFP) and symptomatic KOA among middle-aged and elderly Chinese individuals. Improving these obesity indices (BMI, WHtR, BRI, BFP) may serve as an effective strategy for preventing the onset of KOA in this population.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12891-024-08009-5.

Supplementary Material 1.

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Authors' contributions

H. L., Y.W and G. Z. designed the study, analyzed the data, and wrote the manuscript. XY.W., ZM H., Y.Z., YX.Y., and QS. C., summarize and analyze data analyzed the data. JX. W., and T.J. revised the manuscript. All authors read and approved the final manuscript.

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Data availability

The raw data that support the findings of this study are available from the China Health and Retirement Longitudinal Study (CHARLS) at https://charls.pku.edu.cn/. The processed data sets used and/or analyzed during the current study are available from the corresponding authors upon reasonable request.

Declarations

Ethics approval and consent to participate
The real data is publicly available, thus ethics approval was not required.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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