Association between visceral adiposity index and bone mineral density: an NHANES study

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Abstract

Objectives:

With the improvement of living standards and changes in dietary structure, the number of individuals with central obesity is increasing. Recent studies have indicated that visceral fat accumulation is closely linked to osteoporosis. Therefore, we aimed to explore the relationship between obesity status and bone density mineral (BMD) from a novel perspective of visceral adiposity.

Methods

The study included 9,016 participants from the National Health and Nutrition Examination Survey (NHANES) 2007-2010. The visceral adiposity index (VAI) was calculated by anthropometry parameters (body mass index and waist circumference) and functional parameters (triglycerides and high-density lipoprotein cholesterol). Considering the complex survey design and sample weights, we employed multivariate linear regression models and smooth curve fittings to evaluate the correlation between VAI and BMD. For addressing the nonlinear relationship, we employed a two-piecewise regression to calculate the inflection points.

Results

After adjusting for multiple covariates, there was a significant positive correlation between VAI and lumbar spine, total femur, and femoral neck BMD. After converting VAI to a categorical variable (quartiles), participants in the highest VAI quartile had a 0.049g/cm^2 higher BMD than those in the lowest VAI quartile (β =0.049, 95%CI: 0.040-0.057, *P*<0.001). In subgroup analyses stratified by age, the relationship between VAI and lumbar BMD was an inverted U-shaped curve in the age ≤18 groups, with a point of inflection at 4.86.

Conclusions

VAI was significantly positively correlated with lumbar BMD, demonstrating the complex relationship between visceral fat and bone metabolism. In addition, VAI may be used as a useful predictor of osteoporosis and provide new ideas for the evaluation and screening of osteoporosis.

Introduction

Osteoporosis is a systemic bone metabolic disease characterized by decreased bone mass, degeneration of bone tissue structure, which subsequently results in bone fragility and susceptibility to fractures [1–2]. The incidence of osteoporosis rises rapidly as the population ages, with approximately 1.5 million typical osteoporotic fractures (OF) of the spine, hip, and wrist occurring each year in the United States [3]. Medical expenditures related to osteoporosis are on the rise in the global medical expenditure [4]. The costs associated with osteoporotic fractures will more than double by 2050 if effective measures are not taken to control it [5–6]. In addition to financial costs, osteoporosis also brings tremendous physical and

emotional burdens to patients. Thus, early identification and management of modifiable risk factors are critical for decreasing the prevalence of osteoporosis.

The risk factors for osteoporosis are classified as uncontrollable and controllable factors, with the latter including low body weight, excessive drinking and smoking, and unhealthy lifestyles [7]. According to recent research, osteoporosis and obesity share a common biological basis and visceral fat accumulation is important for the pathogenesis of osteoporosis [8–9]. Body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR) are traditional indicators of obesity, but they are unable to distinguish between visceral and subcutaneous fat, thus accurately reflecting the degree of body fat accumulation [10–11]. Therefore, some scholars proposed VAI based on WC, BMI, triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) to reflect the individual fat distribution and visceral fat metabolism [12].

In comparison to computed tomography (CT), magnetic resonance imaging (MRI), and other tools for assessing visceral fat content, VAI has the advantages of simple calculation, simple data acquisition, and no ionizing radiation [13]. Emerging consensus demonstrated that VAI can not only assess visceral fat content but also indirectly reflect visceral fat function and insulin resistance [14–15]. In addition, VAI has recently been proved to be a reliable predictor of insulin resistance, cardiometabolic risk, and disorders of glucose and lipid metabolism [16–17]. However, studies on the association between VAI and the risk of osteoporosis are still very sparse and the results are inconclusive. Therefore, we analyzed NHANES (2007–2010) data to explore the relationship between VAI and BMD to evaluate the risk factors related to BMD, which is helpful for the early prevention and screening of osteoporosis.

Methods

Study design and population

The NAHNES is a research project of the National Center for Health Statistics that collects the health and nutrition statistics of the United States population. The organization implemented a stratified multistage probability sampling design to ensure the study participants are representative. And all participants signed informed consent before the start of the investigation.

Our research data were selected from physical measurements, examination data, demographic data, and questionnaire data in NHANES 2007-2010. We included participants (n= 20,686) who had completed nutrition investigations, medical examinations, and BMD measurements. The exclusion criteria were as follows (Figure 1): (1) Missing bone mineral density data (n= 9,544); (2) Missing total cholesterol (TC), WC, TG and BMI data (n= 2,083); (3) Participants with history of cancer diseases (n= 43). As a result, 9,016 participants were eventually included in this analysis.

Assessment of BMD

As the exposure variable of this study, total femur, femoral neck, and lumbar spine BMD were estimated by TBS software (Med-Imap SA TBS Calculator version 2.1.0.2). All measurements were performed by well-trained and certified radiology technicians from NHANES. The NHANES DXA bone measurement component provides nationally representative data on bone mineral content (BMC, g), bone area (cm^2), and BMD (g/cm^2). We defined osteopenia as −2.5 < T score < -1.0 and osteoporosis as T score ≤ -2.5.

Assessment of Thyroid Function

The body measurement data of BMI (kg/m^2), WC (cm), Weight (kg), and Height (cm) were based on Anthropometric Standardization Reference Manual. The body measurement rooms were identical in terms of layout and equipment in each of the mobile examination centers. The calibration of scheduled equipment was performed by health technicians and verified by supervisory personnel.

Blood specimens were processed, stored, and shipped to the University of Minnesota and the National Center for Environmental Health for analysis. All HDL cholesterol (mmol/L) samples were analyzed using the direct HDL cholesterol immunoassay method. The concentration of TG (mmol/L) in serum or plasma was measured by a timed-endpoint method.

The following is the calculation method of obesity-related indicators:

Waist-to-height ratio (WHtR) = WC (cm) / height (cm).

VAI was calculated using the following gender-specific equations:

Male: VAI=(WC/(39.68+(1.88*BMI))) *(TG/1.03) * (1.31/HDL)

Female: VAI=(WC/(36.58+(1.89*BMI))) * (TG/0.81) * (1.52/HDL)

Covariates

The covariates are demographic information, laboratory data, and questionnaire data. Demographic information included age, gender, race, and education level. Laboratory data included HbA1c (%), total cholesterol (TC, mmol/L), total calcium (mmol/L), creatinine (μmol/L), uric acid (μmol/L), phosphorus (P, mmol/L), high-density lipoprotein cholesterol (HDL-C, mmol/L). Finally, questionnaire data covered alcohol consumption, smoking behavior, and physical activity. Smoking behavior was defined as Never (smoked less than 100 cigarettes in the lifetime), Past (smoked more than 100 cigarettes in the lifetime, but did not currently smoke), and Current (smoked more than 100 cigarettes in the lifetime and currently still smoked). Alcohol consumption was classified as Non-drinker, Low to moderate drinker (< 1 drink/day in women and < 2 drinks/day in men), and Heavy drinker (≥ 1 drink/day in women and ≥2 drinks/day in men). Physical activity was defined as Low (≤ twice a week), Moderate (< once a day and > twice a week), High (≥ once a day).

Statistical analyses

The analysis of the NHANES was calculated by using the sample weight according to the analytical guideline regulated by NCHS. All continuous data conforming to normal or skewed distributions were presented as mean ± standard deviation, while categorical variables were shown as the number and percentage of subjects. We stratified participants into quartiles according to BMD levels and used univariate analysis and chi-square test to explore the correlation between various indexes of the population and BMD. The statistically significant covariates were then incorporated into the multiple linear regression model. By adjusting covariates, we further analyzed the correlation between VAI and BMD, and calculated the effect value β and its 95% confidence intervals (CI). Furthermore, the linear relationship between VAI and lumbar spine BMD was demonstrated by smoothing curve fitting. For addressing the nonlinear relationship, we employed a two-piecewise regression to calculate the inflection points. Finally, we performed subgroup analyses stratified by gender and age to strengthen the confidence of the data. In this study, SPSS 26.0 software, R software, and EmpowerStats were used for data analysis, and *P*□0.05 was considered statistically significant. Missing values often occurred during the data extraction and data collection processes. For avoiding potential survival bias, we used multiple interpolations to replace the missing values in our study.

Results

The demographic characteristics of the participants

The demographic characteristics of the 9,016 participants based on lumbar spine BMD quartiles are presented in Table 1. Participants in the highest quartile of BMD were more likely to be male, Mexican American, more educated, nondrinkers, and nonsmokers. Among the subgroups, no significant correlation was found between lumbar BMD and physical activity or TC ($P > 0.05$). Besides, compared to those with lower BMD levels, those with higher BMD levels had higher levels of HbA1c, TG, creatinine, uric acid, BMI, weight, WC, WHTR, and VAI, whereas were less likely to have higher levels of total calcium, phosphorus, and HDL-C ($P < 0.001$).

Table 1

Baseline characteristics of the research population based on lumbar spine BMD quartiles.

Characteristics	Quartiles of lumbar-spine BMD				
	Q1 (≤ 0.919)	Q2 (0.919-1.019)	Q3 (1.019-1.115)	Q4 (≥ 1.15)	*P* value
Age (years)	41.16 ± 20.99	38.96 ± 17.04	37.69 ± 15.79	40.22 ± 15.44	<0.001
Gender					
Male	1037 (46.42%)	1089 (48.16%)	1153 (51.20%)	1220 (53.73%)	<0.001
Female	1196 (53.58%)	1172 (51.84%)	1109 (48.80%)	1051 (46.27%)	
Race/ethnicity (%)					<0.001
Non-Hispanic white	277 (12.41%)	264 (11.65%)	213 (9.46%)	154 (6.77%)	
Non-Hispanic black	361 (16.17%)	313 (13.84%)	233 (10.35%)	222 (9.74%)	
Mexican American	1461 (65.40%)	1508 (66.71%)	1556 (69.14%)	1512 (66.64%)	
Other race	134 (6.02%)	176 (7.81%)	249 (11.06%)	383 (16.85%)	
Level of education (%)					<0.001
Less Than High School Grade	339 (15.17%)	410 (18.15%)	340 (15.11%)	311 (13.70%)	
High School Grad	410 (18.37%)	416 (18.45%)	406 (18.05%)	506 (22.29%)	
More Than High School Grade	891 (39.92%)	1073 (47.75%)	1184 (52.54%)	1266 (55.74%)	
Missing	593 (26.54%)	362 (15.97%)	321 (14.30%)	188 (8.28%)	
Alcohol consumption (%)					<0.001
Nondrinker	1108 (49.61%)	1378 (60.94%)	1499 (66.60%)	1625 (71.55%)	
Low to moderate drinker	454 (20.32%)	417 (18.44%)	373 (16.55%)	362 (15.96%)	
Heavy drinker	671 (30.07%)	466 (20.62%)	379 (16.85%)	284 (12.49%)	
Smoking behavior (%)					<0.001

Current	968 (43.33%)	488 (21.57%)	392 (17.41%)	233 (10.28%)	
Past	556 (24.90%)	733 (32.43%)	884 (39.26%)	969 (42.64%)	
Never	709 (31.77%)	1040 (46.00%)	975 (43.33%)	1069 (47.08%)	
Physical activity (%)					0.156
Low	344 (15.41%)	423 (18.73%)	398 (17.66%)	440 (19.39%)	
Moderate	445 (19.95%)	344 (15.20%)	449 (19.95%)	422 (18.58%)	
High	151 (6.74%)	126 (5.56%)	152 (6.74%)	141 (6.19%)	
Not recorded	1293 (57.91%)	1368 (60.52%)	1252 (55.65%)	1268 (55.84%)	
HbA1c (%)	5.48 ± 0.74	5.43 ± 0.76	5.45 ± 0.79	5.53 ± 0.86	<0.001
Total cholesterol (mmol/L)	4.95 ± 1.17	4.97 ± 1.05	4.91 ± 1.05	4.94 ± 1.02	0.348
TG (mmol/L)	1.54 ± 1.26	1.60 ± 1.34	1.59 ± 1.45	1.76 ± 1.62	<0.001
Total calcium (mmol/L)	2.38 ± 0.09	2.37 ± 0.09	2.36 ± 0.09	2.35 ± 0.09	<0.001
Creatinine (μmol/L)	70.24 ± 28.46	74.24 ± 23.80	75.80 ± 29.19	78.72 ± 27.58	<0.001
Uric acid (μmol/L)	300.34 ± 74.24	318.05 ± 80.91	319.31 ± 80.24	329.82 ± 81.44	<0.001
Phosphorus (mmol/L)	1.31 ± 0.83	1.24 ± 0.19	1.23 ± 0.19	1.21 ± 0.18	<0.001
HDL-C (mmol/L)	1.44 ± 0.43	1.38 ± 0.43	1.34 ± 0.38	1.31 ± 0.39	<0.001
BMI (kg/m^2)	24.48 ± 5.27	26.36 ± 5.33	27.64 ± 5.61	29.16 ± 5.52	<0.001
Weight (kg)	66.66 ± 16.95	74.55 ± 17.20	80.25 ± 17.82	86.03 ± 17.57	<0.001
Height (cm)	164.56 ± 9.78	167.92 ± 9.52	170.27 ± 9.70	171.71 ± 17.57	<0.001
WC (cm)	87.21 ± 14.85	91.66 ± 14.59	94.71 ± 14.88	98.28 ± 14.16	<0.001
WHTR	0.53 ± 0.09	0.55 ± 0.09	0.56 ± 0.09	0.57 ± 0.09	<0.001
VAI	3.05 ± 4.13	3.61 ± 5.53	3.72 ± 6.56	4.38 ± 6.23	<0.001

Mean ± SD for continuous variables: the *P* value was calculated by the weighted linear regression model. (%) for categorical variables: the *P* value was calculated by the weighted chi-square test.

BMD, bone density mineral; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; BMI, body mass index; WC, waist circumference; WHTR, waist-to-height ratio; VAI, visceral adiposity index.

Correlation between VAI and BMD in all parts of the body

The adjusted correlations between VAI and BMD in all parts of the body were presented in Table 2. We found that VAI was significantly positively correlated with BMD levels in the unadjusted model (β = 0.002, 95%CI: 0.002–0.003, *P*<0.001), and the positive association was still present in model 2 (β = 0.002, 95%CI: 0.002–0.003, *P*<0.001) and model 3 (β = 0.002, 95%CI: 0.001–0.002, *P*<0.001) after adjusting for confounders. In the multivariable model 3, every 1 increase in VAI was associated with a 0.002 increase in lumbar BMD. Furthermore, this positive correlation remained significant when the BMD measurement site was changed to the total femur, femoral neck. After transforming VAI into a categorical variable, participants in the highest VAI quartile had a 0.049g/cm^2 higher BMD than those in the lowest VAI quartile. And the trend remained significant among different BMD quartile groups (*P* < 0.01). The subgroup analyses stratified by gender and age are reported in Table 3. In both genders, participants with higher VAI levels had significantly higher BMD than those with lower levels. Meanwhile, BMD was increased with the increase of VAI levels in different age subgroups (*P* < 0.01).

Table 2

Associations between VAI and BMD in all parts of the body.

	Lumbar-spine BMD	Total femur BMD	Femoral neck BMD
Model 1 **β (95% CI)**	0.002 (0.002, 0.003) <0.001	0.001 (0.001, 0.002) <0.001	0.002 (0.002, 0.003) <0.001
Model 2 **β (95% CI)**	0.002 (0.002, 0.003) <0.001	0.001 (0.001, 0.002) <0.001	0.001 (0.001, 0.002) <0.001
Model 3 **β (95% CI)**	0.002 (0.001, 0.002) <0.001	0.001 (0.001, 0.002) <0.001	0.001 (0.001, 0.002) <0.001

Logistic regression models:

Model 1: no covariates were adjusted.

Model 2 was adjusted for demographic factors, including gender, age, race.

Model 3 was adjusted for gender, age, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, triglycerides, total calcium, creatinine, phosphorus, uric acid.

Table 3

Subgroup analyses stratified by gender and race.

	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)
VAI	0.002 (0.002, 0.003) <0.001	0.002 (0.002, 0.003) <0.001	0.002 (0.001, 0.002) <0.001
VAI (quartile)			
Q1	Reference	Reference	Reference
Q2	0.017 (0.008, 0.025) <0.001	0.022 (0.014, 0.031) <0.001	0.014 (0.006, 0.021) <0.001
Q3	0.035 (0.026, 0.043) <0.001	0.044 (0.036, 0.053) <0.001	0.030 (0.022, 0.038) <0.001
Q4	0.052 (0.044, 0.061) <0.001	0.065 (0.056, 0.074) <0.001	0.049 (0.040, 0.057) <0.001
***P* for trend**	<0.001	<0.001	<0.001
Subgroup analysis stratified by gender			
Male	0.002 (0.001, 0.003) <0.001	0.002 (0.001, 0.003) <0.001	0.001 (0.000, 0.002) <0.001
Female	0.002 (0.001, 0.003) <0.001	0.003 (0.002, 0.004) <0.001	0.003 (0.002, 0.004) <0.001
Subgroup analysis stratified by age			
Age ≤18	0.006 (0.002, 0.009) 0.002	0.010 (0.006, 0.013) <0.001	0.002 (0.001, 0.002) <0.001
Age 19-45	0.001 (-0.000, 0.001) 0.122	0.001 (0.001, 0.002) <0.001	0.001 (0.000, 0.002) 0.033
Age 46-59	0.002 (0.001, 0.003) <0.001	0.002 (0.001, 0.003) <0.001	0.002 (0.001, 0.003) <0.001
Age ≥60	0.005 (0.003, 0.006) <0.001	0.003 (0.001, 0.004) <0.001	0.006 (0.001, 0.010) 0.010

Logistic regression models:

Model 1: no covariates were adjusted.

Model 2 was adjusted for demographic factors, including gender, age, race.

Model 3 was adjusted for gender, age, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, triglycerides, total calcium, creatinine, phosphorus, uric acid.

The subgroup analyses stratified by gender and age

Through multiple linear regression analysis, we demonstrated a significant relationship between VAI and lumbar BMD. Therefore, we used smooth curve fittings and generalized additive models to further investigate the correlation between VAI and lumbar BMD. As shown in Figure 2-4, VAI was positively correlated with lumbar BMD. The correlation between VAI and lumbar BMD was an inverted U-shaped curve in the age ≤18 groups, with a point of inflection at 4.86. As shown in Table 4, for individuals with a VAI <4.86, every 1 unit increase in VAI was associated with a 0.025 g/cm^2 higher lumbar BMD (95%CI: 0.015–0.035); In contrast, for individuals with a VAI >4.86, a 1 unit increase in VAI was associated with a 0.027 g/cm^2 decrease in lumbar BMD (95%CI: -0.040– -0.014).

Table 4

Threshold effect analysis of VAI on lumbar BMD in the age ≤18 groups using the two-piecewise linear regression model.

Lumbar bone mineral density	Adjusted β (95% CI), *P* value
Age ≤18	
Fitting by standard linear model	0.006 (0.002, 0.009) 0.002
Fitting by two-piecewise linear model	
Inflection point	4.86
VAI <4.86	0.025 (0.015, 0.035) <0.001
VAI >4.86	-0.027 (-0.040, -0.014) <0.001
Log likelihood ratio	<0.001

Gender, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, total calcium, creatinine, phosphorus, and uric acid were adjusted.

VAI, visceral adiposity index; CI, confidence interval.

Discussion

In this study, we examined the association between VAI and bone loss based on NHANES 2007–2010 data. We demonstrated that VAI was negatively correlated with lumbar BMD. In subgroup analyses stratified by gender and age, the strong correlations between VAI and lumbar BMD were observed in both genders and the correlation between VAI and lumbar BMD was an inverted U-shaped curve in the age ≤ 18 groups, with a point of inflection at 4.86. The innovation of this study is that we used visceral

adiposity index rather than BMI, WC, and WHR as predictors of bone loss. We directly linked visceral fat accumulation with bone metabolism, which provides a novel hypothesis for the evaluation and prevention of osteoporosis.

With the increasing prevalence of osteoporosis and obesity, people have increasingly recognized the importance of exploring the relationship between them. However, there are currently conflicting views on the relationship between obesity and osteoporosis. The majority of studies using BMI as an indicator of obesity discovered that obesity has a protective effect on osteoporosis [18–20]. A prospective study in 1999 showed a significant correlation between BMI and BMD among Turkish women, regardless of changes in the location of BMD measurements or age [21]. Ma et al. reported that there was a positive relationship between BMI and lumbar BMD in older adults, and a BMI saturation value (26.13 kg/m^2) was observed in the lumbar spine [22]. According to relevant mechanism studies, on the one hand, weight gain was commonly accompanied by increased fat mass, which inhibits the transformation of androgen into estrogen, decreases bone absorption and prevents bone mass loss [23]. On the other hand, the abnormal production of adipokines and activation of certain pro-inflammatory signaling pathways in obese patients could contribute to the production of TNF-α, IL-1, IL-6, and other inflammations, thereby stimulating osteoclast activity and increasing bone resorption [24–26]. However, recent reports have indicated that excess fat mass may not protect against osteoporosis and may even accelerate bone loss. According to a large population-based cohort study, obesity was associated with an elevated risk of ankle, leg, and forearm fractures [27]. Hind et al. observed that high levels of visceral fat were associated with prevalent vertebral fracture (VF) in women but not in men [28]. A large cohort study in Puerto Rican found that increased abdominal and total fat mass were correlated with lower BMD, with a 10–16% increase in the risk of osteoporosis or osteopenia for every 100 g increase in abdominal fat mass (AFM) [29]. In contrast, our study found that those with higher BMD levels are more likely to have higher levels of BMI. Therefore, we hypothesized that obesity may be a protective factor for osteoporosis, but once the fat content exceeds the saturation threshold, the bone loss would accelerate. However, we failed to achieve similar results due to differences in study design, study population, methods for BMD quantification and site of measurement, and control of confounding variables. Therefore, we need further large-sample prospective studies to confirm this conclusion.

Compared with subcutaneous fat, visceral fat exerts different actions on the bone [30], so a better understanding of the link between visceral fat and bone tissue might help to uncover new molecular therapeutic targets. VAI has previously been proved to indirectly show visceral fat accumulation and fat distribution [31], and latest mechanistic researches have revealed a potential link between VAI and osteoporosis. Osteoblasts and adipocytes shared the same stem cell precursors, and extracellular vesicles from adipose tissue-derived stem cells inhibited osteoporosis through osteoprotegerin and miR-21-5p [32–33]. Furthermore, activation of the nuclear peroxisome proliferator activated receptor γ(PPARγ) played an crucial role in the initiation of adipocyte differentiation, and PPARγ could inhibit osteoblast differentiation through runt-related transcription factor 2 (Runx2) reduction [34]. Increasing evidence suggested that abnormal expansion of marrow adipose tissue (MAT) also is crucial for the occurrence

and development of osteoporosis [35]. For example, high VAI was associated with the high lumbar spine, femoral neck, and total hip T-scores in patients with metabolic syndrome (MetS), according to Wung et al. [36]. In a recent cross-sectional study in a Mexican population, Palacios et al. identified VAI to be a significant predictor of lumbar BMD and showed the serum metabolite profile associated with VAI and low BMD [37]. Similarly, our findings demonstrated a positive relationship between VAI and the BMD of the lumbar spine, femoral neck, and total hip. Therefore, it further indicated that visceral fat is positively correlated with BMD and VAI might be an independent protective factor of osteoporosis.

Considering the influence of gender on body fat distribution, we used different VAI calculation formulas for males and females to ensure the accuracy the of data. In stratified analysis by sex, we observed a significant positive correlation between VAI levels and BMD in both genders. After adjustment for multiple confounders, the correlation remained significant, which indicates this relationship is independent of gender. Moreover, in stratified analysis by age, we found that the correlation between VAI and lumbar BMD was an inverted U-shaped curve in the age ≤ 18 groups. We considered that the differences were due to differences in body composition and fat distribution with age [38]. Frank et al. discovered that age is a factor influencing the distribution of total and regional fat location and distribution, and lipids are redistributed to muscles, bone marrow, and other tissues with aging [39–41]. Visceral fat increased with age, and adipose tissue was more likely to accumulate in the viscera and abdomen with increasing age according to Amdanee et al. [42]. In addition, we found that VAI was not associated with lumbar BMD in participants in the age range 19 to 45 in the unadjusted model. This may be due to differences in genetic risk factors, age distribution, and adjusted confounding factors.

In our research, we used nationally representative NHANES data and combined sample weights for statistical analysis to ensure the authenticity and objectivity of the data. And we provided solid evidence of an independent association between VAI and BMD by employing multiple logistic regression and stratified analyses. However, there were also some limitations in our study. Firstly, the anti-osteoporotic treatment history of our participants was absent, which affects the objectivity of bone mineral density measurements. In addition, this study only included patients from the United States, so we might not guarantee that the results of this study can be generalized to other countries. Finally, we used a cross-sectional approach in this study, thus we could not collect patient follow-up data to explore the causal relationship between VAI and osteoporosis.

Conclusions

In conclusion, our present study demonstrates that VAI is significantly positively associated with lumbar BMD. Our findings indicate the importance of VAI in the early screening and evaluation of osteoporosis and that VAI may be a new predictor of osteoporosis in the future.

Abbreviations

BMD Bone density mineral

BMI Body mass index

VAI Visceral adiposity index

NHANES The National Health and Nutrition Examination Survey

WC Waist circumference

WHtR Waist-to-height ratio

CT Computed tomography

MRI Magnetic resonance imaging

TC Total cholesterol

P Phosphorus

HDL-C High-density lipoprotein cholesterol

AFM Abdominal fat mass

PPARγ Peroxisome proliferator activated receptor γ

Runx2 Runt-related transcription factor 2

MAT Marrow adipose tissue

MetS Metabolic syndrome

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. All analyses were based on data of the National Health andNutrition Examination Survey (NHANES). The study was approved by the ethics review board of the National Center for Health Statistics. The detailed information located on the NHANES website.

Consent for publication

Not applicable.

Availability of data and materials

The survey data are publicly available on the Internet for data users and researchers throughout the world http://www.cdc.gov/nchs/nhanes/.

Competing interests

Shuai Chen, Guowei Zhou, Xiaohe Sun, Jie Jin and Zhiwei Li declare that they have no conflict of interest.

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Author Contributions

S Chen and XH Sun collected data. GW Zhou organized the study and performed the statistical analysis. J Jin and ZW Li drafted the manuscript, to which all authors contributed, and approved the final version for publication.

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Figures

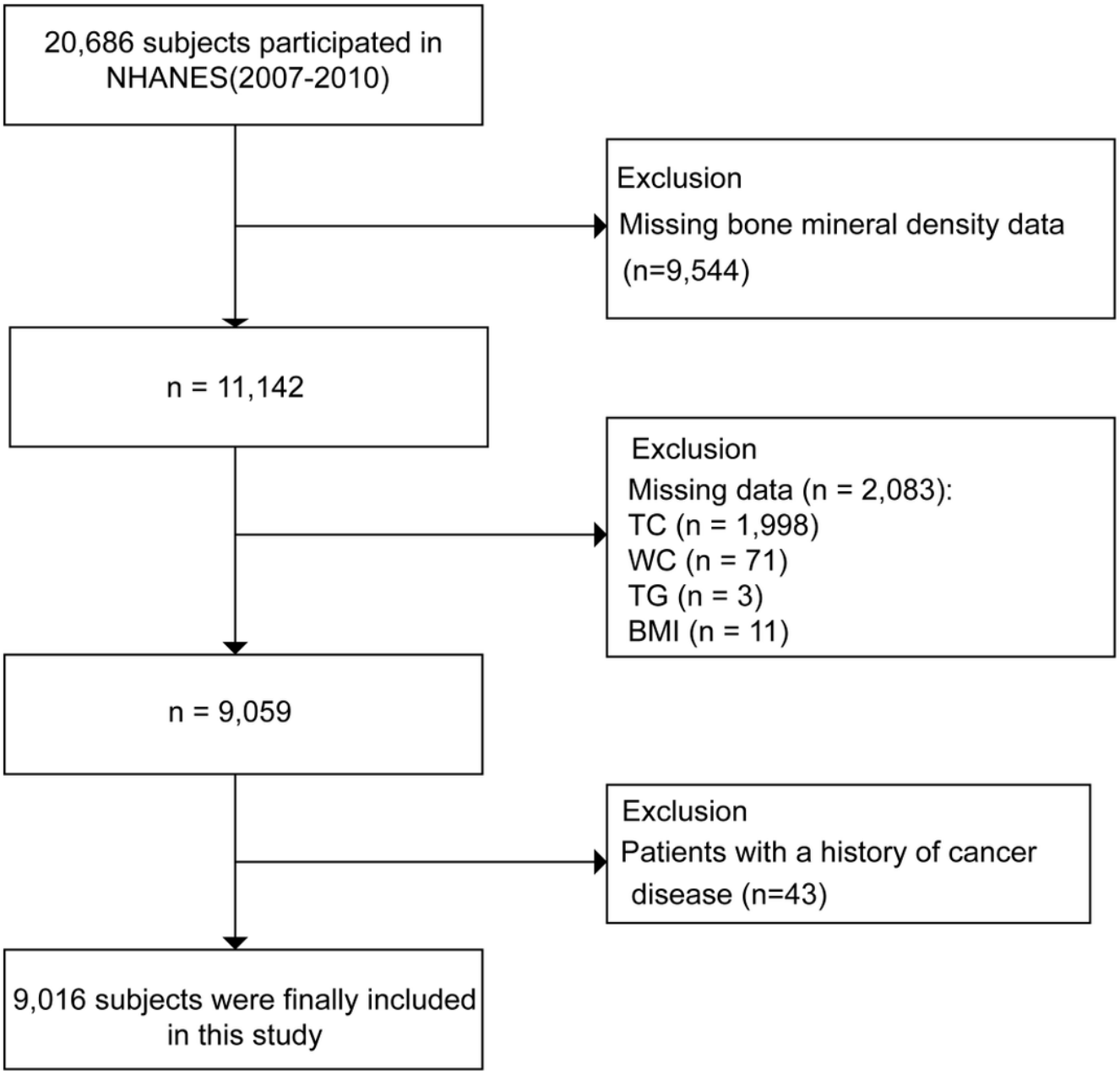

Figure 1

flow chart

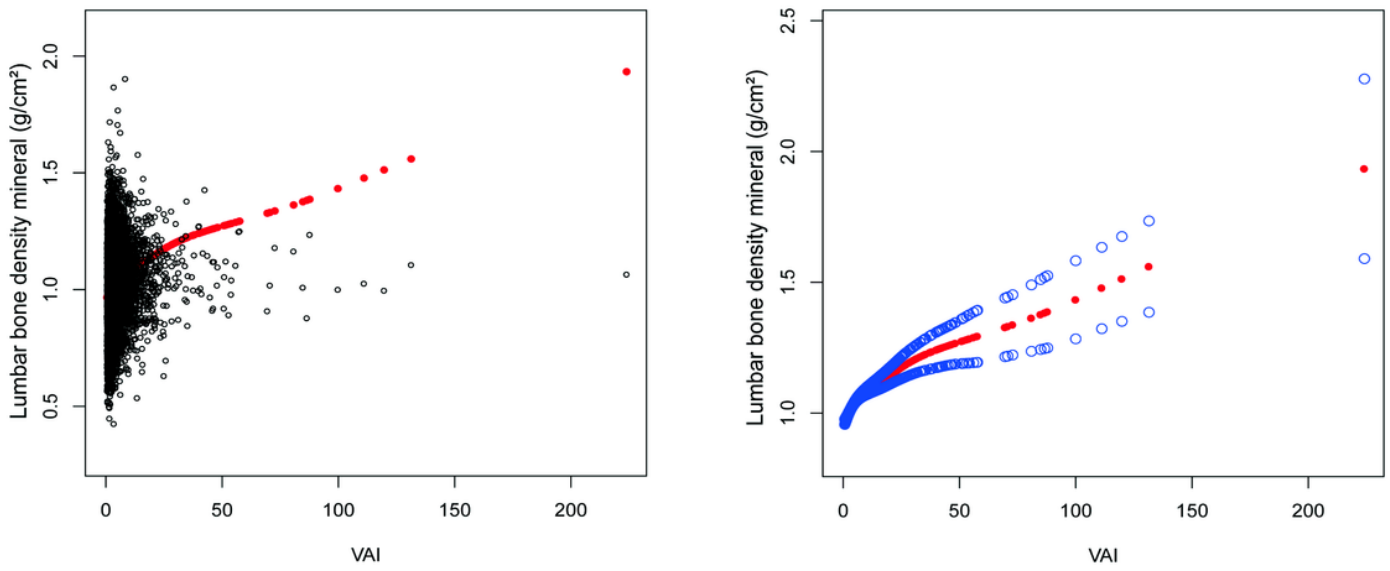

Figure 2

The association VAI between and lumbar BMD. (a) Each black point represents a sample. (b) Solid rad line represents the smooth curve fit between variables. Blue bands represent the 95% of confidence interval from the fit. Age, gender, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, total calcium, creatinine, phosphorus, and uric acid were adjusted.

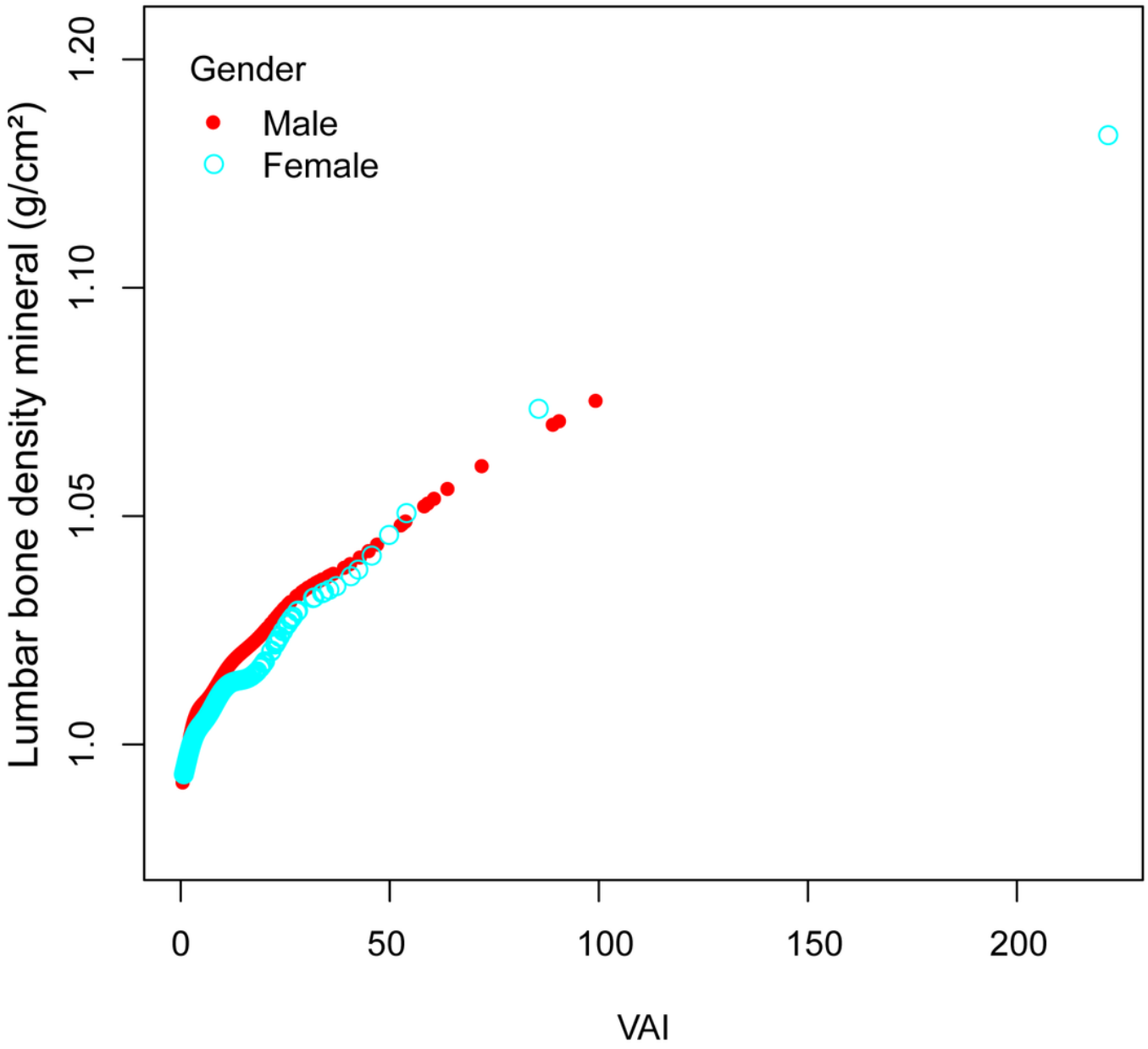

Figure 3

The association between VAI and lumbar bone mineral density stratified by gender. Age, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, total calcium, creatinine, phosphorus, and uric acid were adjusted.

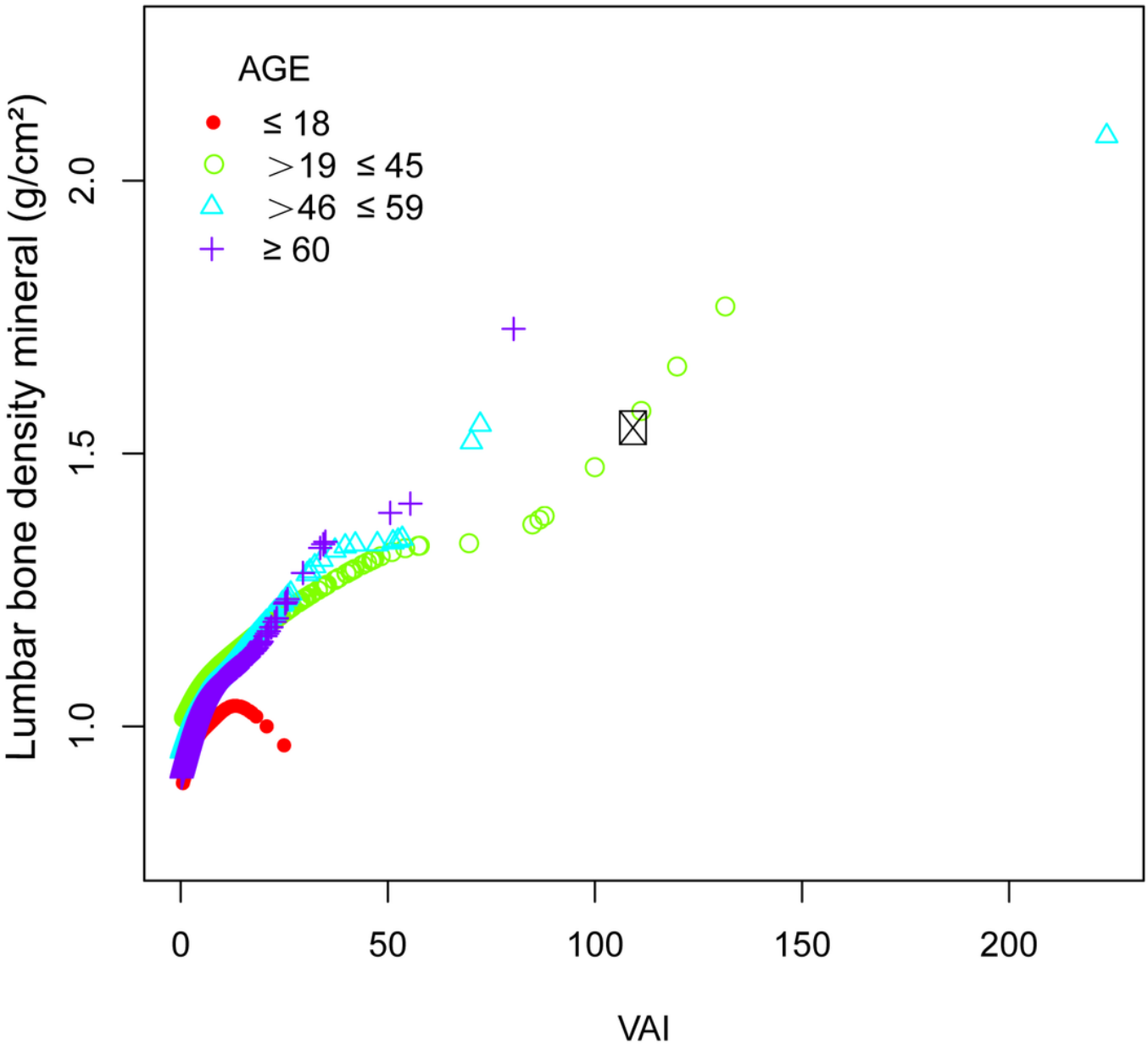

Figure 4

The association between VAI and lumbar bone mineral density stratified by age. Gender, race, education, physical activity, smoking behavior, alcohol consumption, HbA1c, total cholesterol, total calcium, creatinine, phosphorus, and uric acid were adjusted.