Archives of Osteoporosis (2021) 16:92
https://doi.org/10.1007/s11657-021-00953-2

ORIGINAL ARTICLE

Bone characteristics and metabolic phenotypes of obesity in an Iranian Elderly population: Bushehr Elderly Health Program (BEHP)

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Received: 11 January 2021 / Accepted: 19 April 2021

Abstract

Introduction Obesity and osteoporosis are health problems with high impact on the morbidity and mortality rate. While the association between BMI and bone density is known, the combined effects of obesity and metabolic components on bone health have not yet been revealed. The objectives of this study were to determine the association between bone health and different phenotypes of obesity in an elderly population.

Methods This cross-sectional study was conducted on the data collected in the Bushehr Elderly Health Program (BEHP). The participants were classified in four groups based on the metabolic phenotypes of obesity (metabolic healthy obese (MHO), metabolic non-healthy non-obese (MNHNO), metabolic non-healthy obese (MNHO), and metabolic healthy non-obese (MHNO)). The association between osteoporosis and TBS and the metabolic phenotypes of obesity were assessed using multiple variable logistic regression models.

Results Totally, 2378 people (1227 women) were considered for analyses. The prevalence of MHNO, MHO, MNHNO, and MNHO were 902 (39.9%), 138 (6.1%), 758 (33.5%), and 464 (20.5%), respectively. In the multivariate logistic regression models, those with MHO (OR 0.22; 95% CI 0.12–0.36), MNHNO (OR 0.52; 95% CI 0.4–0.66), and MNHO phenotypes (OR 0.22; 95% CI 0.16–0.3) had a significantly lower risk of osteoporosis. Likewise, those having MHO (OR 2.38; 95% CI 1.51–3.76), MNHNO (OR 1.49; 95% CI 1.11–2), and MNHO (OR 2.50; 95% CI 1.82–3.42) phenotypes were found to had higher risk of low bone quality as confirmed by TBS.

Conclusions The obese subjects have lower bone quality, regardless of their obesity phenotype.

Keywords Abdominal obesity · General obesity · Metabolic syndrome · Obesity · Osteoporosis · Metabolic phenotype

Introduction

Obesity and osteoporosis are two major health problems with high impact on the global morbidity and mortality rate. Obesity has various adverse effects on health. According to the 2016 WHO report, 13% and 39% of the adult population were obese and overweight worldwide, respectively [1]. National studies have reported the prevalence of obesity among Iranian adults to range from 12.6 to 25.9% [2]. BMI is commonly used to indicate obesity. However, it cannot distinguish fat from lean body mass. Accordingly, further obesity phenotypes such as lean mass, fat mass, and percentage fat mass have been suggested to overcome this shortcoming [3].

"Osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, increased bone fragility and susceptibility to fracture" [4]. According to a meta-analysis conducted in 2013 in Iran, the general prevalence of osteoporosis was 12% among men, while it was 3% and 19% in pre- and postmenopausal women, respectively [5]. The diagnosis of osteoporosis is generally made based on bone mineral density (BMD) measurement using dual-energy X-ray absorptiometry

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Published online: 08 June 2021

(DXA) [6]. Most individuals with fragility fractures are reported to have a normal or osteopenic BMD values [7], suggesting that areal BMD (aBMD) fails to fully capture the fragility fracture risk. Therefore, trabecular bone score (TBS) was developed to reflect bone microarchitecture [8]. TBS is an indirect measurement of bone microarchitecture that can be extracted from the two-dimensional lumbar spine DXA images [9]. A high TBS indicates higher connectivity between bone cells, greater number of trabeculae cells, and lower trabecular spacing; all of which results in better resistance to fragility fracture [10].

Obesity was traditionally believed to have a positive effect on bone health due to the positive correlation of mechanical loading and bone formation [11]. Adipose tissue-derived hormones are also believed to strengthen this relationship [12]. On this basis, low BMI is considered as a negative risk factor in the FRAX algorithm, used to calculate an individual's 10-year fracture risk probability [13]. Recent studies, however, indicate that obesity, particularly abdominal obesity, is inversely related with BMD [3, 14]. Some investigations have shown that visceral adipose tissue causes low-grade chronic inflammation that has a negative effect on bone metabolism. Inflammation may also increase bone absorption by osteoclasts [15–17].

Previous studies on the association of other metabolic diseases such as diabetes and bone health have had conflicting results [18–20]. This disagreement also applies to obesity [21]. For instance, the Osteoporotic Fractures in Men (MrOS) study showed obesity to be associated with a higher incidence of hip and other non-spine fractures [22]. This is while a meta-analysis of 60,000 men and women from 12 prospective, population-based cohorts concluded a negative correlation between BMI and total and osteoporotic fractures in both genders [23].

This study was therefore carried out to assess the association of different obesity phenotypes and bone quality indices (BMD and TBS) in a group of elderly from a community-based study, Bushehr Elderly Health (BEH) Program.

Material and methods

Setting and sampling The data of this study (anthropometric measurements and bone densitometry results) belong to the participants of BEH Program. This on-going longitudinal population-based cohort study is discussed elsewhere [24, 25].

Data collection Demographic data were collected through interviewing the participants. Lifestyle information was collected using a standard questionnaire (Monica questionnaire) and physical activity was assessed using Metabolic Equivalent of Task questionnaire. Anthropometric data were measured according to the National Health and Nutrition Examination Survey III Protocol with precision of 0.1 kg and 0.1 cm, respectively [26]. All anthropometric measurements were carried out in the morning and after about 12 h fasting. BMI was calculated by dividing weight by square of height. Obesity, overweight, and normal body weight were defined based on the WHO definition (BMI ≥ 30 kg/m^2 defined as obesity, 25 kg/m^2 ≤ BMI < 30 kg/m^2 defined as overweight, 18.5 kg/m^2 ≤ BMI < 25 kg/m^2 defined as normal body weight, and BMI < 18.5 kg/m^2 defined as underweight). The BMD of lumbar spine (L1–L4), neck of femur, and total hip were measured using dual-energy X-ray absorptiometry DXA (Discovery WI, Hologic, USA). Osteoporosis was defined as T-score of − 2.50 or lower at the lumbar spine, femoral neck, or total hip [27]. Also, low bone quality was defined as TBS ≤ 1.231 in men and TBS ≤ 1.287 in women [28].

The participants were classified as having metabolic syndrome (MetS) if they had three or more of the corresponding criteria according to the Adult Treatment Panel III (ATP III).

1. Abdominal obesity: cut-off of 102 cm in men and 88 cm in women
2. Fasting blood sugar (FBG) ≥ 100 mg/dL
3. Triglyceride (TG) ≥ 150 mg/dL
4. HDL-C ≤ 40 mg/dL
5. Systolic blood pressure (SBP) ≥ 130 mmHg and/or diastolic blood pressure (DBP) ≥ 85 mmHg [29]

The participants were classified into four different metabolic phenotypes of obesity according to their obesity and metabolic status: metabolically healthy obese (MHO) defined as obese (BMI > 30) individuals who did not have metabolic syndrome, metabolically non-healthy non-obese (MNHNO) as non-obese (18.5 < BMI < 25) individuals with metabolic syndrome, metabolically non-healthy obese (MNHO) as being both obese and having metabolic syndrome, and metabolically healthy non-obese (MHNO) as non-obese individuals without metabolic syndrome.

Ethical consideration All the participants signed an informed consent. This study was approved by the Ethical Research Committee of Endocrinology and Metabolism Research Institute affiliated to Tehran University of Medical Sciences (Ethical Code: IR.TUMS.EMRI.REC.1394.0036).

Statistical analysis The association between TBS, BMD, degraded bone structure, and osteoporosis at lumbar spine and at neck of femur and BMI were assessed using univarate and multiple variable linear and logistic regression models. Adjustments were performed based on variables with P < 0.20 in univariate regression models or significant associations were reported by previous studies. In the final

multivariable regression models, associations adjusted for age, gender, smoking, low physical activity as dummy variable, and waist circumference. All analyses were conducted using Stata 12 (StataCorp, TX, USA).

Results

The information of 2378 individuals (1227 women and 1151men) aged > 60 years were analyzed. From all the participants, 42.64% (38.96% and of men 46.57%) were overweight and 26.49% (36.51% of women and 15.81% of men) of them were obese (Table 1). Abdominal obesity was reported 57.65% (83.86% of women and 29.71% of men). The prevalence of abdominal obesity in osteoporosis at spinal, hip, and femoral neck was 29.91%, 10.36%, and 34.65%, respectively. As for abdominal obesity and cumulative osteoporosis, the rate was as high as 44.13%.

The MHNO, MHO, MNHNO, and MNHO phenotypes were reported in 40.16%, 6.18%, 33.31%, and 20.35%, respectively. These phenotypes were seen in association with abdominal obesity in 21.78%, 94.56%, 68.94%, and 98.76% of cases, respectively.

The mean BMD values at lumbar spine, hip, and neck of femur were 0.81 (±0. 0.14) g/cm^2, 0.75 (±0.13) g/m^2, and 0.59 (±0. 0.11) in women and 0.99 (±0.17) g/cm^2, 0.94 (±0.14) g/m^2, and 0.73 (±0.13) g/m^2 in men, respectively. There was a significant correlation between BMI and BMD at lumbar spine in both gender (r = 0.48 for female and r = 0.32 for men). This correlation was weaker for BMI and BMD at neck of femur (r = 0.34 in female and r = 0.26 in male). The prevalence of lumbar spine, neck of femur, and cumulative osteoporosis increased significantly from the underweight to overweight group (p < 0.001) (Table 2).

In univariate linear regression model, a positive association was noted between BMD at spine and femoral neck with BMI (β = 0.21 for lumbar spine and β = 0.12 for neck of femur, P values < 0.001). In final multivariable linear regression model, also, relationship between BMDs and BMI was observed (β = 0.29 for lumbar spine' BMD and β = 0.18 for neck of femur's BMD, P values < 0.001). Moreover, lumbar spine's TBS had a negative relationship with abdominal obesity (β = −0.167) based on the univariate linear regression. In final multivariable linear regression model, this negative association remained significant (β = −0.2) (Table 3).

A negative association was observed between BMD at spine (β = −0.23), hip (β = −0.33), and femoral neck (β = −0.29) and spinal TBS (β = −0.49) with body fat percentage (p value < 0.001). In final models, β coefficients between BMDs and body fat percentage were calculated as the following: lumbar spine' BMD β = −0.08, neck of femur's BMD β = −0.14, and lumbar spine' TBS β = −0.16 (Table 4).

The association of spinal, neck of femur, and cumulative osteoporosis with obesity in univariate and multivariable logistic regression models, adjusted models for age, sex, physical activity, and smoking, are presented in Table 5. Decreasing trends of odds ratio in overweight and obese participants in comparison to normal body weight subjects were observed in spinal, neck of femur, and cumulative osteoporosis in both univariate and multivariable models (p values of trends were < 0.001) (Table 5). Compared with those with low body weight, the risk of osteoporosis at different sites was significantly lower in obese subjects (91% for spine (OR 0.09; 95% CI 0.04–0.23), 84% for neck of femur (OR 0.16; 95% CI 0.06–0.38), 91% for hip (OR 0.09; 95% CI 0.03–0.31), and 92% for at any site (OR 0.08; 95% CI 0.03–0.22)).

Table 6 illustrates the association of spinal, neck of femur, and cumulative osteoporosis with different metabolic phenotypes of obesity using logistic regression models. In the final multivariate logistic regression model, MHO (OR 0.25; 95% CI 0.15–0.40), MNHNO (OR 0.54; 95% CI 0.43–0.69), and MNHO (OR 0.21; 95% CI 0.15–0.28) phenotypes were at a significantly lower risk of cumulative osteoporosis compared with the MHNO phenotype. MHO (OR 2.40; 95% CI 1.52–3.81), MNHNO (OR 1.43; 95% CI 1.10–1.86), and MNHO (OR 2.30; 95% CI 1.71–3.09) phenotypes were at a significantly higher risk of having low bone quality based on TBS cutoff as compared with the MHNO group. With each unit of increase in TBS increased the odds of having the MHO phenotype rather than MHNO increased by 2.40 times. Compared with the MHNO group, this increase was by 1.43 and 2.30 times for MNHNO and MNHO (adjusted for age, sex, physical activity, and smoking).

Discussion

This study established that while there was an association between BMI, mechanical loading, and BMD, the case is totally in opposition for the body fat composition which led to the conclusion that it decreased both the BMD and TBS for the selected bones. The same goes for age and smoking as both decrease bone health. Furthermore, we have illustrated that obese subjects have a lower risk of osteoporosis than their low body weight counterparts. Another subtle fact that was elicited was that people with MHNO phenotype had a higher cumulative risk of osteoporosis than other phenotypes but they were found to have the highest bone quality base on TBS cutoff.

The generally accepted mechanism by which obesity may protect bones against osteoporosis is mechanical loading. The more frequent mechanical loading stimulates proliferation and differentiation of osteoblasts, therefore, it increases bone formation [16, 23]**.** On the other hand, obesity is associated with low-grade chronic inflammation that has a negative

Table 1 Demographic and health characteristics of the participants

	Female participants N = 1227						Male participants N = 1151				
	LBW N = 13	NW N = 288	OW N = 478	Obese N = 448	P value		LBW N = 32	NW N = 401	OW N = 536	Obese N = 182	P value
Age year mean (SD)	73.30 (8.67)	71.35 (7.36)	68.94 (6.21)	67.78 (5.02)	< 0.001		70.12 (6.37)	70.92 (7.09)	68.61 (5.87)	68.83 (5.66)	< 0.001
Smoking n (%)	6 (46.15)	60 (20.83)	91 (19.08)	71 (15.88)	0.021		19 (59.38)	113 (28.18)	113 (21.12)	28 (15.38)	< 0.001
Physical inactivity n (%)	12 (92.31)	224 (78.87)	353 (74.32)	346 (77.93)	0.221		28 (90.32)	290 (73.42)	397 (74.91)	148 (81.32)	0.045
BMI kg/m^2 mean (SD)	17.22 (0.88)	22.70 (1.60)	27.51 (1.40)	34.22 (3.81)	< 0.001		16.97 (1.41)	22.77 (1.60)	27.25 (1.39)	32.52 (2.40)	< 0.001
WC cm mean (SD)	70.96 (7.79)	87.90 (7.89)	99.04 (7.27)	110.34 (10.10)	< 0.001		71.59 (6.62)	88.98 (7.17)	99.45 (6.42)	112.15 (7.42)	< 0.001
HTN n (%)	9 (69.23)	185 (64.24)	346 (72.38)	367 (81.92)	< 0.001		11 (34.38)	249 (62.09)	371 (69.22)	151 (82.97)	< 0.001
DM n (%)	2 (15.38)	81 (28.13)	179 (37.45)	171 (38.17)	0.010		0 (0.0)	95 (23.69)	164 (30.60)	67 (36.81)	< 0.001
	Female participants N = 1227						Male participants N = 1151				
	MHNO = 307	MHO = 93	MNHNO = 471	MNHO = 356	P value		MHNO = 648	MHO = 54	MNHNO = 321	\MNHO = 128	P value
Age year mean (SD)	70.60 (7.50)	67.81 (5.30)	69.46 (6.28)	67.76 (4.95)	< 0.001		69.91 (6.63)	68.27 (4.70)	69.01 (6.23)	67.07 (6.03)	< 0.001
Smoking n (%)	68 (22.22)	14 (15.05)	88 (18.68)	58 (16.34)	< 0.001		176 (27.20)	4 (7.41)	69 (21.50)	24 (18.75)	< 0.001
Physical inactivity n (%)	227 (74.92)	81 (89.01)	361 (77.14)	266 (75.14)	0.032		469 (73.40)	40 (74.07)	246 (77.60)	108 (84.38)	0.050
BMI kg/m^2 mean (SD)	24.46 (3.31)	33.78 (2.93)	26.26 (2.45)	34.32 (4.01)	< 0.001		24.40 (3.13)	32.14 (2.15)	26.37 (2.32)	32.68 (2.49)	< 0.001
WC cm mean (SD)	89.81 (10.73)	109 43 (8.54)	97.45 (7.59)	110.57 (10.46)	< 0.001		91.85 (9.54)	110 05 (6.23)	98.95 (7.15)	113.04 (7.72)	< 0.001
HTN n (%)	159 (51.79)	55 (59.14)	381 (80.89)	312 (87.64)	< 0.001		386 (59.57)	41 (75.93)	245 (76.32)	110 (85.94)	< 0.001
DM n (%)	29 (9.45)	3 (3.23)	233 (49.47)	168 (47.19)	< 0.001		97 (14.97)	3 (5.56)	162 (50.47)	64 (50.00)	< 0.001

LBW low body weight, *NW* normal weight, *OW* overweight, *BMI* body mass index, *WC* waist circumference

Underweight BMI < 18.5 kg/m^2, normal weight 18.5 kg/m^2 ≤ BMI < 25 kg/m^2, overweight 25 kg/m^2 ≤ BMI < 30 kg/m^2, obese BMI ≥ 30 kg/m^2

effect on bone metabolism. Pro-inflammatory cytokines are elevated in blood circulation of the subjects with obesity; this promotes osteoclast differentiation and bone resorption [16]. Furthermore, obesity is associated with dysregulation of the GH/IGF-I axis, and may adversely affect bone formation by decreasing GH secretion [15].

The effect of obesity on gonadal hormones is usually different between men and women. Testosterone is a positive determinant of bone mineral density (BMD) and muscle mass, which is reduced in men with obesity, whereas estrogen production is increased with increment of body fat [12]. Another potential mechanism by which obesity can have an impact on the bone quality is related to overall decrease in vitamin D levels that is inversely associated with abdominal adiposity. Moreover, a diet with high fat content that is often observed in obese subjects may interfere with calcium absorption [12, 16].

The current study demonstrates higher prevalence of osteoporosis in non-obese elderly men and women in comparison with obese ones. There is also a plummeting trend in the osteoporosis prevalence as body composition changes from low bodyweight to the obese group. This effect was still observed even after adjustment for age, sex, physical activity, smoking, and waist circumference. In other words, the increase of BMI had an independent protective effect on osteoporosis. We observed that less than one-tenth of subjects with obesity were prone to spinal osteoporosis. This protective effect was more noticeable in spinal osteoporosis in women. The same phenomenon happens in men though in terms of femoral neck osteoporosis.

Moreover, we found that the prevalence of lumbar spine, neck of femur, and cumulative osteoporosis in the MHNO phenotype was higher than other phenotypes in both sexes.

Also, previous studies, for instance a study conducted in Chinese healthy men (N = 228, aged from 38 to 89 years), had shown the positive correlation between BMD and BMI [30].

In addition, we found a negative correlation between TBS and BMI in both sexes that remained significant after adjustment for age and sex. Previous studies have also shown the same results, one of such studies was the National Health and Nutrition Examination Survey(NHANES) cohort study, conducted in American adults [31].

Another similar result was manifested by a study in South Korea that showed a positive correlation between BMI and BMD while presenting a negative correlation between BMI and TBS [32]. A study conducted in Ukraine found a significant higher BMD but a lower TBS in obese group in comparison with non-obese ones [33], and in another study on Ukrainian men, obese men had a significantly higher BMD while their TBS on L1–L4 vertebrae was significantly lower than non-obese group [34]. On the other hand, Mazetti et al. illustrated that when TBS was measured with Hologic densitometers, there is a negative correlation between TBS and BMI in both men and women and no significant correlation was observed when TBS measurement was done on GE Lunar densitometers [35]. In another study, Ayoub et al. showed that there were no significant differences between the TBS in obese, overweight, and normal weight young men [36]. These different results may be due to the effect of various races, ethnicities, and ages in subjects as well as different densitometer devices manufactured by different companies.

Furthermore, in our study, a negative association between spinal BMD, femoral BMD, spinal TBS, and body fat composition was observed. Several studies have been conducted to evaluate the effect of fat mass on osteoporosis; for instance in a study, Zhao et al. showed that the positive correlation between fat mass and bone mass changes to a negative relationship after adjustment for mechanical loading effect of body weight [3]. Moreover, in a large community-based, cross-sectional study performed in China, it was observed that subjects with a higher body fat percentage had higher risk of osteoporosis independent of body weight, physical activity, and age [37].

A negative association between lumbar spinal TBS with abdominal obesity was observed in our study. Likewise, Romagnoli explored the relationship between abdominal obesity and TBS, and demonstrated that waist circumference could negatively affect TBS values in overweight/obese men [38]. Bredella et al. measured distal radius microarchitecture by three-dimensional high-resolution peripheral quantitative computed tomography in thirty-five obese men and found that high visceral adipose tissue (VAT) and bone marrow fat are negative predictors of cortical microarchitecture in obese men [12].

Our study was strong in certain aspects that are defined in the following: it was conducted on a large sample size consisting of both male and female individuals, who were representative of the elderly population in the country. As a result, not only did we evaluate bone health parameters of old people that are at a higher risk of osteoporotic fracture but also our findings in this study can be generalized to the whole country. Moreover, we evaluated the association between different metabolic phenotypes of obesity and osteoporosis.

This study suffered from some limitations including the fact that we could not consider anti-osteoporosis medication consumption as an adjusted factor. Also, our study was a cross-sectional study and we did not have access to osteoporotic fracture data as a major outcome of osteoporosis. So, additional studies are suggested to evaluate the impact of BMI and other bone quality parameters on outcomes such as fractures.

Table 2 Bone mineral density and osteoporosis condition among different BMI conditions

	Female participants N = 1227					Male participants N = 1151				
	LBW N = 13	NW N = 288	OW N = 478	Obese N = 448	P value*	LBW N = 32	NW N = 401	OW N = 536	Obese N = 182	P value*
Lumbar spines BMD mean (SD)	0.62 (0.10)	0.73 (0.13)	0.80 (0.12)	0.88 (0.14)	< 0.001	0.80 (0.14)	0.95 (0.16)	1.00 (0.17)	1.07 (0.17)	< 0.001
Total hip BMD mean (SD)	0.6 (0.10)	0.68 (0.12)	0.75 (0.11)	0.80 (0.12)	< 0.001	0.78 (0.11)	0.90 (0.13)	0.96 (0.13)	1.00 (0.15)	< 0.001
Lumbar spines TBS mean (SD)	1.24 (0.06)	1.26 (0.08)	1.25 (0.08)	1.24 (0.09)	< 0.001	1.34 (0.06)	1.38 (0.07)	1.37 (0.09)	1.34 (0.11)	< 0.001
Neck of femur BMD mean (SD)	0.49 (0.08)	0.53 (0.10)	0.59 (0.10)	0.62 (0.10)	< 0.001	0.60 (0.08)	0.69 (0.12)	0.75 (0.13)	0.77 (0.13)	< 0.001
Spinal osteoporosis n (%)	12 (92.31)	192 (66.67)	200 (41.84)	102 (22.77)	< 0.001	21 (65.63)	78 (19.45)	65 (12.13)	13 (7.14)	< 0.001
Femoral neck osteoporosis n (%)	9 (69.23)	103 (35.76)	71 (14.58)	36 (8.04)	< 0.001	5 (15.63)	9 (2.24)	4 (0.75)	2 (1.10)	< 0.001
Total hip osteoporosis n (%)	9 (69.23)	103 (35.76)	71 (44.91)	36 (8.04)	< 0.001	5 (15.63)	9 (2.24)	4 (0.75)	2 (1.10)	< 0.001
Cumulative osteoporosis n (%)	13 (100.00)	232 (80.56)	280 (58.58)	194 (43.30)	< 0.001	25 (78.13)	115 (28.68)	104 (19.40)	21 (11.54)	< 0.001

LBW low body weight, *NW* normal weight, *OW* overweight, *BMD* bone mineral density

*P for ANOVA tests

Table 3 Bone health and osteoporosis condition among different metabolic obesity phenotype

	Female participants N = 1227					Male participants N = 1151				
	MHNO = 307	MHO = 93	MNHNO = 471	MNHO = 356	P value*	MHNO = 648	MHO = 54	MNHNO = 321	MNHO = 128	P value*
Lumbar spines BMD mean (SD)	0.74 (0.14)	0.86 (0.12)	0.79 (0.12)	0.88 (0.14)	< 0.001	0.95 (0.16)	1.06 (0.16)	1.02 (0.17)	1.08 (0.17)	< 0.001
Total hip BMD mean (SD)	0.70 (0.13)	0.80 (0.11)	0.74 (0.12)	0.81 (0.12)	< 0.001	0.91 (0.14)	0.98 (0.13)	0.97 (0.13)	1.01 (0.15)	< 0.001
Lumbar spines TBS mean (SD)	1.26 (0.08)	1.25 (0.08)	1.25 (0.08)	1.24 (0.1)	0.03	1.37 (0.08)	1.35 (0.12)	1.38 (0.09)	1.33 (0.11)	< 0.001
Neck of femur BMD mean (SD)	0.55 (0.11)	0.62 (0.11)	0.58 (0.10)	0.62 (0.11)	< 0.001	0.71 (0.13)	0.77 (0.11)	0.76 (0.14)	0.77 (0.14)	< 0.001
Spinal osteoporosis n (%)	186 (60.59)	23 (24.73)	218 (46.28)	79 (22.19)	< 0.001	132 (20.37)	5 (9.26)	32 (9.97)	8 (6.56)	< 0.001
Femoral neck osteoporosis n (%)	194 (63.19)	38 (40.86)	242 (51.38)	114 (32.02)	< 0.001	122 (18.83)	5 (9.26)	30 (9.35)	10 (7.81)	< 0.001
Total hip osteoporosis n (%)	96 (31.27)	5 (5.38)	87 (18.47)	31 (8.71)	< 0.001	17 (2.62)	1 (1.85)	1 (0.31)	1 (0.78)	< 0.001
Cumulative osteoporosis n (%)	228 (74.27)	47 (50.54)	296 (62.85)	148 (41.57)	< 0.001	196 (30.25)	7 (12.96)	48 (14.95)	14 (10.94)	< 0.001

MHNO metabolically healthy non-obese, *MHO* metabolically healthy obese, *MNHNO* metabolically non-healthy non-obese, *MNHO* metabolically non-healthy obese, *BMD* bone mineral density, *TBS* trabecular bone score

*P for ANOVA tests

Table 4 Association between lumbar spines, femoral neck BMD, and lumbar spines TBS with Body Fat Percentage in linear regression models

	Lumbar Spine BMD			Neck of Femur BMD			Total hip BMD			Lumbar spine TBS		
	B	β standardized	P value	B	β standardized	P value	B	β standardized	P value	B	β standardized	P value
Univariate model												
Body fat percentage	− 0.005	− 0.235	< 0.001	− 0.005	− 0.294	< 0.001	− 0.006	− 0.328	< 0.001	− 0.006	− 0.4907	< 0.001
Multivariable model												
Body fat percentage	− 0.002	− 0.0819	0.021	− 0.003	− 0.1476	< 0.001	− 0.003	− 0.1689	< 0.001	− 0.002	− 0.1689	< 0.001
Age (year)	− 0.002	− 0.0740	< 0.001	− 0.005	− 0.2271	< 0.001	− 0.006	− 0.1558	< 0.001	− 0.003	− 0.1558	< 0.001
Gender (male/female)	0.192	0.5318	< 0.001	0.132	0.4724	< 0.001	0.182	0.3975	< 0.001	0.088	0.3975	< 0.001
Physical inactivity (yes/no)	0.001	0.0172	0.858	− 0.017	0.0802	< 0.002	− 0.013	0.0365	0.024	− 0.002	0.0365	0.615
Smoking (yes/no)	− 0.031	− 0.0346	< 0.001	− 0.018	− 0.0116	0.001	− 0.024	− 0.0231	< 0.001	− 0.023	− 0.0231	< 0.001
BMI (kg/m^2)	0.014	0.3707	< 0.001	0.009	0.3015	< 0.001	0.012	− 0.0506	< 0.001	− 0.001	− 0.0506	0.015

BMD bone mineral density, *TBS* trabecular bone score, *BMI* body mass index

Table 5 Association between osteoporosis and obesity in logistic models

	Lumbar spine osteoporosis			Neck of femur osteoporosis			Total hip osteoporosis			Cumulative osteoporosis			Lumbar spine TBS		
	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value
First model															
Low body weight	Reference group			Reference group			Reference group			Reference group			Reference group		
Normal weight	0.23	0.11–e0.46	< 0.001	0.47	0.25–0.87	0.016	0.43	0.22–0.83	0.013	0.19	0.08–0.42	< 0.001	1.09	0.54–2.20	0.803
Over-weight	0.13	0.06–0.25	< 0.001	0.25	0.13–0.46	< 0.001	0.18	0.09–0.35	< 0.001	0.11	0.05–0.25	< 0.001	1.54	0.77–3.09	0.218
Obese	0.08	0.04–0.16	< 0.001	0.24	0.12–0.44	< 0.001	0.14	0.07–0.29	< 0.001	0.09	0.04–0.22	< 0.001	3.58	1.78–7.19	< 0.001
Final multivariable model															
Low body weight	Reference group			Reference group			Reference group			Reference group			Reference group		
Normal weight	0.24	0.11–0.52	< 0.001	0.36	0.17–0.77	0.008	0.24	0.09–0.64	0.005	0.16	0.06–0.40	< 0.001	0.36	0.14–0.95	0.039
Over-weight	0.16	0.07–0.36	< 0.001	0.21	0.09–0.46	< 0.001	0.12	0.04–0.35	< 0.001	0.11	0.04–0.29	< 0.001	0.38	0.14–1.03	0.058
Obese	0.09	0.04–0.23	< 0.001	0.16	0.06–0.38	< 0.001	0.09	0.03–0.31	< 0.001	0.08	0.03–0.22	< 0.001	0.38	0.13–1.11	0.078
Age (year)	1.04	1.02–1.06	< 0.001	1.09	1.07–1.11	< 0.001	1.14	1.11–1.16	< 0.001	1.07	1.05–1.08	< 0.001	1.07	1.05–1.09	< 0.001
Gender (male/female)	0.16	0.13–0.20	< 0.001	0.11	0.08–0.13	< 0.001	0.04	0.02–0.06	< 0.001	0.13	0.11–0.16	< 0.001	0.04	0.03–0.05	< 0.001
Low physical activity (yes/no)	1.22	0.96–1.56	0.109	1.31	1.02–1.67	0.032	1.06	0.71–1.59	0.770	1.26	1.01–1.58	0.042	1.04	0.81–1.35	0.740
Smoking (yes/no)	1.33	1.05–1.70	0.019	1.26	0.99–1.61	0.062	1.35	0.92–1.99	0.120	1.35	1.07–1.70	0.011	1.49	1.13–1.96	0.004
Waist circum-ference (cm)	0.97	0.96–0.98	< 0.001	0.98	0.97–0.99	0.007	0.98	0.96–0.99	0.009	0.97	0.96–0.99	< 0.001	1.04	1.02–1.06	< 0.001

TBS trabecular bone score, *CI* confidence interval

Table 6 Association between osteoporosis and metabolic obesity phenotype in logistic models

	Lumbar spine osteoporosis			Neck of femur osteoporosis			Total hip osteoporosis			Cumulative osteoporosis			Lumbar spine TBS		
	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value	Odds Ratio	95% CI odds ratio	P value	Odds ratio	95% CI odds ratio	P value
First Model															
MHNO	Reference group			Reference group			Reference group			Reference group			Reference group		
MHO	0.47	0.30–0.73	0.001	0.84	0.57–1.22	0.356	0.32	0.14–0.73	0.007	0.73	0.51–1.04	0.082	3.41	2.26–5.16	< 0.001
MNHNO	0.92	0.75–1.13	0.442	1.06	0.87–1.29	0.581	0.93	0.69–1.25	0.638	0.96	0.79–1.16	0.686	2.13	1.63–2.79	< 0.001
MNHO	0.44	0.33–0.57	< 0.001	0.69	0.54–0.89	0.004	0.53	0.35–0.79	0.002	0.63	0.50–0.79	< 0.001	3.8	2.86–5.04	< 0.001
Final multivariable model															
MHNO	Reference group			Reference group			Reference group			Reference group			Reference group		
MHO	0.25	0.15–0.40	< 0.001	0.47	0.31–0.74	0.001	0.18	0.07–0.45	< 0.001	0.37	0.25–0.57	< 0.001	2.4	1.52–3.81	< 0.001
MNHNO	0.54	0.43–0.69	< 0.001	0.58	0.45–0.74	< 0.001	0.51	0.35–0.72	< 0.001	0.52	0.41–0.66	< 0.001	1.43	1.10–1.86	0.007
MNHO	0.21	0.15–0.28	< 0.001	0.31	0.23–0.42	< 0.001	0.28	0.18–0.44	< 0.001	0.26	0.20–0.35	< 0.001	2.30	1.71–3.09	< 0.001
Age (year)	1.05	1.03–1.06	< 0.001	1.10	1.08–1.12	< 0.001	1.14	1.11–1.17	< 0.001	1.07	1.05–1.09	< 0.001	1.06	1.04–1.08	< 0.001
Gender (male/female)	0.16	0.12–0.20	< 0.001	0.10	0.08–0.13	< 0.001	0.04	0.02–0.07	< 0.001	0.12	0.09–0.15	< 0.001	0.04	0.03–0.06	< 0.001
Low physical activity (yes/no)	1.25	0.98–1.59	0.069	1.32	1.03–1.68	0.026	1.13	0.76–1.69	0.535	1.28	1.03–1.60	0.029	1.07	0.83–1.38	0.616
Smoking (yes/no)	1.45	1.15–1.83	0.002	1.35	1.06–1.72	0.014	1.49	1.03–2.15	0.035	1.45	1.16–1.82	0.001	1.51	1.15–1.97	0.003

TBS trabecular bone score, *CI* confidence interval

Conclusion

To this day, the mechanism by which higher BMI values and mechanical loading have stimulated osteoblasts and thus protected bones against osteoporosis is generally accepted. But what they have failed to notice is the microarchitecture that was hampered by obesity. This study tried to have a multi-perspective view on the matter.

Although a fairly large number of analyses have established that bone density increases with mechanical loading, metabolic health and weight control can lead to a higher bone quality.

The importance of TBS along with other markers such as BMD and metabolic profile has led us to believe that even though higher BMI increases bone density, it alters the bone microarchitecture.

Advanced imaging and measurement modules will further elaborate the delicacies of bone quality and thus will accelerate identification of osteoporosis; an evaluation of bone health studies and metabolic profiles from different perspectives will help promote clinical application and will also help mitigate mortality and morbidity in high-risk groups.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11657-021-00953-2.

Acknowledgements The authors would like to express their gratefulness to the staff and researchers of the Bushehr Elderly Health Program for their thoughtful contribution.

Funding This article was a part of a larger project which was granted by the Endocrinology and Metabolism Research Institute (EMRI).

Declarations

Conflicts of interest None.

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