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ORIGINAL RESEARCH

Influence of basal energy expenditure and body composition on bone mineral density in postmenopausal women

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Background: The aim of this study was to investigate the influence of body mass index, body weight, lean mass, fat mass, and basal energy expenditure on bone mineral density in postmenopausal women.

Methods: This was a cross-sectional, descriptive study of a sample of 50 women, with minimum time since menopause between 1 and 10 years. Bone mineral density was assessed at the lumbar spine (L2–L4), femoral neck, Ward's triangle, and trochanter using dual-energy X-ray absorptiometry. Body mass index, lean mass, fat mass, and basal energy expenditure were measured by bioimpedance.

Results: The mean age of the women was 51.49 ± 3.86 years and time since menopause was 3.50 ± 2.59 years. Significant negative correlations were found between chronological age and lumbar spine, femoral neck, Ward's triangle, and trochanteric bone mineral density. In regard to time since menopause, we also observed significant negative correlations with bone mineral density at the lumbar spine and Ward's triangle. The following significant positive correlations were recorded: body mass index with bone mineral density at the femoral neck and trochanter; fat mass with bone mineral density at the femoral neck and trochanter; lean mass with bone mineral density at the lumbar spine, femoral neck, and trochanter; and basal energy expenditure with bone mineral density at all sites assessed. On the other hand, the multiple linear regression model showed that: 20.2% of bone mineral density variability at the lumbar spine is related to lean mass and time since menopause; 22.3% of bone mineral density variability at the femoral neck is related to body weight and age; 18.9% of bone mineral density variability at Ward's triangle is related to age and basal energy expenditure; and 39% of bone mineral density variability at the trochanter is related to body mass index, age, and menarche.

Conclusion: Changes in bone mineral density, specific for each skeletal site, are influenced by age, time since menopause, body weight, body mass index, lean mass, and basal energy expenditure. Lean mass and basal energy expenditure positively influenced bone mineral density at the lumbar spine and Ward's triangle, with a predominance of trabecular bone.

Keywords: women, menopause, bone mineral density, body composition, energy expenditure

Introduction

Demographic changes predicted for the next 50 years indicate that the number of elderly people will increase worldwide, together with metabolism-related diseases.[1] Among these, osteoporosis in postmenopausal women is recognized as an important public health problem because it is associated with a high risk of fracture, elevated morbidity and mortality rates, and incurs high financial and societal costs.[2]

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Bone mineral density (BMD) increases during childhood, adolescence, and early adulthood, until reaching peak bone mineralization. It is a negative predictor of osteoporosis and risk of fracture over time, and is influenced by genetic, mechanical, nutritional, and hormonal factors.[3]

Peak bone mineralization in the entire skeleton, occurring on average at 18 years of age, varies little up to the age of 50 years, with a slight and progressive increase in BMD of around 0.2% per year in cortical bone-rich regions. However, in areas with a larger amount of trabecular bone, such as the proximal femur and the inner area of the vertebral body, an immediate decline is initiated at the age of 18 years, with an annual loss in BMD of 0.3% (trochanter), 0.4% (femoral neck), 0.6% (Ward's triangle), and 0.5% (lumbar spine).[4] This reveals a 50% loss of BMD, mainly in Ward's triangle. One study indicates that a 10% increase in peak bone mineralization could delay the development of osteoporosis by 13 years, while a 10% increase in time since menopause would delay it by only 2 years.[5]

As with BMD, lean mass in the third decade of life varies little until the fifth decade, showing a sharp decline from the sixth decade onwards.[6] The difference in muscle strength between young and elderly individuals is less when these values were adjusted for lean mass and muscle mass.[7]

Menopause is associated with diminished serum estrogen levels, which may provoke a decrease in BMD and lean mass, and a rise in body fat.[8] These alterations can affect gait and balance in the elderly,[9] reducing physical activity at work, home, during leisure time, and in sport. This causes a decline in total and basal energy expenditure, which is influenced by age, sex, body composition, and hormonal factors, including estrogen.[10–12]

Studies show that body weight has a positive influence on BMD.[13–15] However, this influence is different between skeletal sites.[15] There is no consensus regarding the effect of body composition on BMD. Some research has shown that fat mass and lean mass are correlated with lumbar spine and hip BMD, respectively.[15] However, other studies demonstrate that obesity does not protect against fracture in postmenopausal women. On the contrary, it is associated with an increased risk of ankle and femur fractures.[16] Lean mass plays a relevant role in BMD, possibly acting positively on cortical bone mass.[17]

Aging is accompanied by a decrease in lean mass and basal energy expenditure.[11] Individuals with low basal energy expenditure are predisposed to gaining weight at the expense of a proportional increase in fat mass.[10,11] Studies show a positive association between basal energy expenditure and BMD in North American women, which is much more significant than body weight.[10,11]

In order to understand the impact of body composition on BMD in the first 10 years after menopause, we studied the influence of age, time since menopause, body mass index, fat mass, and basal energy expenditure on lumbar spine, femoral neck, Ward's triangle, and trochanteric BMD.

Materials and methods

Patients and study design

This was a cross-sectional, quantitative, descriptive study performed at the Lauro Wanderley University Hospital gynecology outpatient clinic of the Universidade Federal da Paraíba, Brazil. Participants were selected from those responding to posters put up at the hospital, university campus, and family health units in nearby neighborhoods. The sample consisted of 50 women with minimum and maximum time since postmenopause of one and 10 years, respectively, and body mass index between 18.5 and 39.9 kg/m^2. A standard deviation of 6 and maximum error of estimation of 20% were used to calculate sample size, with a 5% significance level. All the women in the study were of mixed ethnicity. Exclusion criteria were: use of hormone replacement therapy; immunosuppressants, glucocorticoids, diuretics, anticonvulsants, or calcium supplementation; tobacco or alcoholic beverages; previous surgery (colostomy and oophorectomy); and history of disease (neoplasia, diabetes mellitus, liver, kidney, and thyroid disorders, and rheumatoid arthritis).

All participants gave written informed consent. The project was approved by the research ethics committee of Lauro Wanderley University Hospital, Universidade Federal da Paraíba (protocol number 335/03). After assessment, the women were referred for specialized clinical follow-up.

Instruments and data collection procedures

A form was used to record sociodemographic, clinical, and anthropometric data. Weight and height were measured while fasting and after bladder emptying. Subjects were barefoot, wearing Bermuda shorts and a t-shirt, and standing in the bipedal position, with their chin parallel to the floor. The head, buttocks, and heels were aligned with the stadiometer of a 150 kg anthropometric scale in 100 g increments and a 2 m metal rod in 1 cm increments (Filizola, Personalline E, São Paulo, Brazil). Body mass index was calculated to obtain classification of nutritional status as follows: eutrophic at 18.5–24.9 kg/m^2; overweight at 25.0–29.9 kg/m^2; and first-degree obesity at 30–34.9 kg/m^2, in accordance with World

Health Organization criteria.[18] Next, we determined basal energy expenditure, fat mass, and lean mass by bioimpedance (RJL Systems, Quantum II, Clinton Twp, MI). All care was taken to inform subjects adequately regarding bioimpedance procedures.[12,19]

Bone densitometry was conducted using dual energy x-ray absorptiometry (Lunar DPX-L; Lunar Radiation Corporation, Madison, WI), in order to measure BMD at L2–L4, femoral neck, Ward's triangle, and the trochanter. Results were calculated by bone area (cm^2) and bone mineral content (g), with BMD expressed in g/cm^2. World Health Organization criteria were used to classify BMD, as normal, osteopenic, or osteoporotic.[20]

Statistical analyses

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 16.0 (IBM Corporation, Armonk, NY). To characterize sociodemographic, anthropometric, bioimpedance, and BMD variables, descriptive statistics procedures such as central tendency (mean) and dispersion (standard deviation) measures were applied. Pearson's correlation coefficient (r) was used to determine the relationship between independent (time since menopause, age, age of menarche, body mass, body mass index, basal energy expenditure, lean mass, and fat mass) and dependent variables (lumbar spine, femoral neck, Ward's triangle, and trochanteric BMD). The Chi-squared test was used to verify the association between nutritional status (eutrophy, overweight, obesity) and diagnostic classification of BMD (normal, osteopenic, and osteoporotic). Results with $P \leq 0.05$ were considered to be statistically significant. Multiple linear regression was used to evaluate linear predictor functions.

Results

A total of 50 women completed the study, with a mean age of 51.49 ± 3.86 years and mean time since menopause of 3.50 ± 2.59 years. None of the participants were illiterate; 40 were able to read and write, 44 had secondary school education, and 16% were educated to university level. Moreover, 62% had a household income of up to three minimum monthly wages (US$795), 24% had four to six minimum wages (US$1060–US$1590) and 14% had seven to ten minimum wages (US$1860–US$2650). With respect to professional activity, 64% were economically active, 16% were retired, and 20% were homemakers.

Descriptive statistics for bioanthropometric results are shown in Table 1. The results for BMD and its diagnostic classification are shown in Table 2. There was a high occurrence of osteopenia at all skeletal sites, and 24% and 12% of osteoporosis at skeletal sites L2–L4 and Ward's triangle, respectively.

Table 1 Bioanthropometric characteristics

Variables	Mean ± SD	Minimum–maximum
Age (years)	51.49 ± 3.86	45–58
Menopause (years)	3.5 ± 2.59	1–10
Menarche (years)	13.04 ± 1.67	10–17
Body mass (kg)	63.84 ± 10.5	44–86.7
Height (m)	1.52 ± 0.06	1.43–1.65
BMI (kg/m^2)	27.49 ± 4.74	19.3–39.9
Body water (kg)	31.04 ± 3.19	26–39
LM (kg)	41.58 ± 4.9	31–51
FM	21.92 ± 6.78	11–36
BEE (BIA)	1354 ± 102.38	1159–1554
Lumbar spine BMD	1.04 ± 0.18	0.69–1.52
Femoral neck BMD	0.9 ± 0.11	0.68–1.29
Ward's triangle BMD	0.77 ± 0.17	0.44–1.2
Trochanteric BMD	0.75 ± 0.11	0.58–1.08

Abbreviations: BMI, body mass index; LM, lean mass; FM, fat mass; BEE, basal energy expenditure; BIA, bioelectrical impedance analysis; BMD, bone mineral density; SD, standard deviation.

Figure 1 shows the occurrence of normal BMD, osteopenia, and osteoporosis in women with eutrophic nutritional status, overweight, and obesity. There was a significant association between BMD at all skeletal sites and the differing nutritional status of the patients.

Table 3 shows the relationship between independent variables and BMD at all skeletal sites studied. Basal energy expenditure (bioelectrical impedance analysis) had a positive correlation with all skeletal sites studied.

Table 4 presents the multiple linear regression model, demonstrating that 20.2% of BMD variability at the lumbar spine was related to lean mass and time since menopause, 22.3% of BMD variability at the femoral neck was related to body weight and age, 18.9% of BMD variability at Ward's triangle was related to age and basal energy expenditure, and 39% of BMD variability at the trochanter was related to body mass index, age, and menarche.

Table 2 Bone mineral density (g/cm^2) and diagnostic classification of bone mineral density at four skeletal sites

Variables	Normal		Osteopenia		Osteoporosis	
	n	%	n	%	n	%
LS	18	36	20	40	12	24
FN	26	52	24	48		
WT	20	40	25	50	5	10
T	34	68	16	32		

Abbreviations: BMD, bone mineral density; LS, lumbar spine (L2–L4); FN, femoral neck; WT, Ward's triangle; T, trochanter.

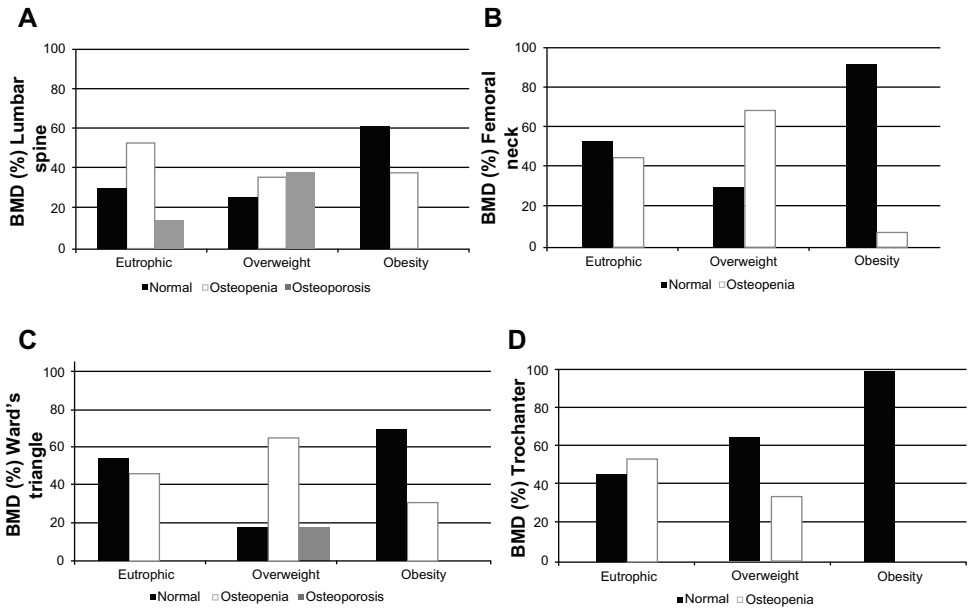

Figure 1 Relationship between BMD (normal, osteopenic, and osteoporotic) and nutritional status (eutrophia, overweight, and obesity) in menopausal patients.
Notes: Chi-square test ($P < 0.05$): association between diagnostic classification of BMD and nutritional status of lumbar spine (**A**) ($\chi^2 = 9.83$; df = 4, $P < 0.05$); in the femoral neck (**B**) ($\chi^2 = 12.77$; df = 2, $P < 0.01$); in Ward's triangle (**C**) ($\chi^2 = 12.74$; df = 4, $P < 0.05$); and in the trochanter (**D**) ($\chi^2 = 9.23$; df = 2, $P < 0.01$). Bars are shown as percentage.
Abbreviation: BMD, bone mineral density.

Discussion

In the present study, we observed osteopenia at all skeletal sites under study and osteoporosis only at L2–L4 (24%) and Ward's triangle (10%). There were significant negative correlations between age and BMD at all skeletal sites analyzed. However, with time since menopause, significant negative correlations were recorded only for bone mineral density at the lumbar spine and Ward's triangle. In this respect, Guthrie et al[21] demonstrated that the degree of bone loss at the lumbar spine and femoral neck was similar, although the rate of such loss was greater at the lumbar spine in the early postmenopausal years. This indicates significant BMD loss in the first 3 years after menopause in areas with a predominance of trabecular bone.

In an attempt to minimize these losses, studies have been conducted to describe the protective effect of body weight on BMD in menopausal women.[13,14,22] In our study, we observed significant positive correlations between body weight and BMD at all skeletal sites studied, except for Ward's triangle. Michaelsson et al[13] reported that body weight above 70 kg could be used to exclude women from an osteoporosis prevention program. This was contested by Bedogni et al,[22] who demonstrated that anthropometric measures could not be used to classify individual bone mineral status, because there was no evidence that body weight altered bone mineral status.

On the other hand, low body mass index may be associated with lower BMD at the femoral neck, with greater risk of osteoporotic fracture.[17] Elevated body mass index could be associated with ankle and femur fractures in postmenopausal women.[16] Therefore, these studies highlight the controversy concerning the effect of body weight on maintaining BMD. The protective effect of elevated weight on BMD in postmenopausal women is attributed to adipose tissue, which may be an extra-ovarian source of estrogen,[23] and the magnitude of the mechanical load to strengthen the osteogenic response.[24]

In a physiological state, mechanical overloads increase muscle strength during physical activity. This muscle strength

Table 3 Correlation coefficients (Pearson) between bioanthropometric variables and bone mineral density

Independent variables	Dependent variables			
	LS	FN	WT	T
Age (years)	–0.315*	–0.303*	–0.311*	–0.332*
Menopause (years)	–0.338*	–0.202	–0.304*	–0.194
Menarche (years)	–0.263	–0.256	–0.179	–0.454**
BW (kg)	0.267	0.368**	0.268	0.454**
BMI (kg/m^2)	0.188	0.367**	0.258	0.463**
LM (kg)	0.311*	0.343*	0.257	0.336*
FM (kg)	0.227	0.348*	0.225	0.454**
BEE (BIA)	0.281*	0.355*	0.292*	0.376**

Notes: *Statistical significance ($P < 0.05$); **statistical significance ($P < 0.01$).
Abbreviations: BW, body weight; BMI, body mass index; LM, lean mass; FM, fat mass; BEE, basal energy expenditure; BIA, bioelectrical impedance analysis; BMD, bone mineral density; LS, lumbar spine (L2–L4); FN, femoral neck; WT, Ward's triangle; T, trochanter.

Table 4 Multiple linear regression model and predictive equations

Dependent variables	R	R^2	Adjusted R^2	SE of estimate	Predictive equations*
LS	0.449	0.202	0.168	0.160	BMD = 0.678 – 0.022 (age) + 0.011 (LM)
FN	0.473	0.223	0.190	0.109	BMD = 1.096 + 0.004 (BW) – 0.009 (TSM)
WT	0.434	0.189	0.154	0.155	BMD = 0.801 – 0.014 (age) + 0.001 (BEE)
T	0.624	0.390	0.350	0.094	BMD = 1.152 + 0.009 (BMI) – 0.008 (age) – 0.018 (menarche)

Note: *All statistical variables were significant ($P < 0.05$).
Abbreviations: LS, lumbar spine; FN, femoral neck; WT, Ward's triangle; TSM, time since menopause; T, trochanter; LM, lean mass; BW, body weight; BEE, basal energy expenditure; BMD, bone mineral density; BMI, body mass index; SE, standard error.

acts on specific bone levers and modifies bone metabolism to the point of stress.[25] Thus, bone responds immediately to the mechanical loads it bears,[26] involving both cellular and tissue reactions.[27] During disuse, the metabolic activity of bone tissue is suppressed. It is normalized by brief exposure to very low mechanical stimuli,[26] responding better to dynamic than static loads.[28]

In relation to body mass index and fat mass, the present study revealed significant positive correlations (Table 3) only with BMD at the femoral neck and trochanter. When these variables were fit to the multiple linear regression model, a 0.9% reduction in femoral neck BMD was observed for each year of life and a 0.4% increase for each kg of body weight gain. For trochanteric BMD, a 0.9% increase was associated with higher body mass index. This indicated that excess weight, represented by body fat, reinforces the biomechanical theory,[24–28] given that we found strong correlations with BMD in weightbearing areas such as the femoral neck and trochanter. With respect to the theory about an extraovarian source of estrogen, attributed to adipose tissue,[23] we question whether it could prevent bone loss in these women, given that no significant positive correlations were observed between fat mass and BMD at the lumbar spine or Ward's triangle (Table 3). These areas exhibit the greatest bone density loss in the first years of menopause.[21] One study showed that fat mass was inversely correlated with bone mass, suggesting that fat mass in itself does not have a protective effect on bone mass.[24] However, lean mass shows significant positive correlations not only with BMD at the femoral neck and trochanter, but also at the lumbar spine (L2–L4, Table 3). The multiple linear regression model demonstrated a 2.2% reduction in BMD at the lumbar spine for time since menopause and a 1.1% increase of that BMD for each kg of lean body mass (Table 4). This means that lean mass is represented primarily by the large muscles, which transmit greater and more frequent mechanical loads to the skeleton.[24,29] Thus, lean mass and psoas muscle volume at L3 were associated with low loss of BMD at the lumbar spine, indicating the importance of applying muscle strength at the site where BMD is maintained.[30] In the long run, the effect of strong dorsal extensor muscles reduced the incidence of vertebral fracture in women with estrogen deficiency.[31] There is evidence that skeletal muscle is also an extraovarian source of estrogen, and the capacity to synthesize this hormone likely depends on the proportion of lean mass.[32]

Age is accompanied by an increase in fat mass, and a decrease in BMD, lean mass, and basal energy expenditure,[6,10,11,19] which may lead to disturbances in gait and balance, and increased risk of falling.[9,16] These factors may generate insecurity, contribute to a sedentary lifestyle and potentially induce changes in body composition to less lean mass and more fat mass, culminating in sarcopenia.[8,9] In postmenopausal women from the fifth decade onwards, the drop in estrogen levels has an important role in decreasing muscle mass.[8] It was also demonstrated that basal energy expenditure falls with menopause and is related to the decline in lumbar spine.[19] In our study, we observed a reduction of 1.4% in Ward's triangle BMD for each year of life and an increase of 0.1% in that BMD for every calorie of basal energy expenditure (Table 4). Therefore, in this area of predominance of trabecular bone, the basal energy expenditure improved BMD. This result corroborates the study conducted by Choi and Pai,[19] demonstrating that BMD is more strongly correlated with basal energy expenditure than are lumbar spine, fat mass, and body mass index. Additionally, basal energy expenditure was the best covariable of bone mineral content and BMD in a cohort of African-American women,[11] displaying a strong correlation with hip and whole body BMD when compared with other anthropometric measures.[10] Furthermore, the present study also shows a significant association between nutritional status and BMD. Overweight women exhibited twice as much osteoporosis at the lumbar spine and Ward's triangle compared with eutrophic women, while the femoral neck showed a 23.2% increase in osteopenia, indicating that being overweight did not increase BMD (Figure 1). A number of studies have demonstrated that obesity does not protect

menopausal women against osteoporosis, and as such, is a risk factor for fracture.[16] One study reported that visceral adiposity and low-density lipoprotein were inversely associated with BMD and that high-density lipoprotein was positively associated with BMD.[33]

Finally, the present study, using the multiple linear regression model, demonstrated BMD variability at the lumbar spine related to lean mass and time since menopause, BMD variability at the femoral neck related to body weight and age, BMD variability at Ward's triangle related to age and basal energy expenditure, and BMD variability at the trochanter related to body mass index, age, and menarche. Thus, a change in lifestyle, and consequent increase in lean mass, along with a rise in basal energy expenditure, could improve metabolic disorders related to aging, obesity, and diabetes mellitus, thereby minimizing BMD loss.

Conclusion

In conclusion, we observed the occurrence of osteopenia at all skeletal sites under study and osteoporosis only at L2–L4 and Ward's triangle. These areas are associated with lean mass and basal energy expenditure, and could prevent osteoporosis. Given that our study sample was not probabilistic, other studies with a more representative population are needed.

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Disclosure

The authors report no conflicts of interest directly relevant to the content or publication of this article.

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