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Correlation of Fat Mass, Muscle Mass, and Basal Metabolic Rate with Bone Mass in Postmenopausal Women

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Abstract

Background: Bone health is an important aspect of aging, particularly in postmenopausal women who experience estrogen deficiency that increases the risk of osteoporosis and related complications. Although components of body composition have individually been associated with bone health, their relative contributions remain incompletely understood. This study aimed to examine the correlations of fat mass, muscle mass, and basal metabolic rate (BMR) with bone mass in postmenopausal women.

Methods: A cross-sectional analytic study was conducted in March 2025 using data from 40 postmenopausal women obtained from the Bandung Sport and Health Nabati Nutrition (B-SHENN) Project. Spearman's rho correlations analysis was used to assess relationships between fat mass, muscle mass, BMR and bone mass. Multiple linear regression analysis was performed to identify the most influential predictor of bone mass.

Results: Fat mass, muscle mass, and BMR were all significantly and positively correlated with bone mass ($p<0.001$). Among these variables, muscle mass showed the strongest correlation with bone mass ($r=0.991$). Multiple linear regression analysis demonstrated that muscle mass was the only independent predictor significantly associated with bone mass ($B=0.069$; $p=0.022$).

Conclusions: Fat mass, muscle mass, and BMR are associated with bone mass in postmenopausal women, with muscle mass emerging as the strongest predictor. These findings underscore the importance of maintaining or increasing muscle mass as a key modifiable factor in strategies to preserve bone health and reduce osteoporosis risk in postmenopausal women.

Keywords: Basal metabolic rate, bone mass, fat mass, muscle mass

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Introduction

Estrogen deficiency disrupts the balance between bone formation and bone resorption, favoring osteoclastic activity and resulting in progressive bone loss. When sustained, this process substantially increases the risk of osteoporosis and fragility fractures, which are associated with increased morbidity, mortality, and reduced quality of life in older women.[1] Bone mass is influenced by a complex interaction of hormonal, mechanical, metabolic, and nutritional factors.[2] Among these, body composition has received increasing attention as a potentially modifiable determinant of bone health. Fat mass exerts dual effects on bone. On one hand, adipose tissue contributes to peripheral estrogen production and increases mechanical loading on the skeleton, both of which may support bone formation. On the other hand, excessive fat mass is associated with chronic low-grade inflammation and increased secretion of pro-inflammatory cytokines, which may accelerate bone resorption and impair bone quality.[3–5]

Muscle mass plays a fundamental role in skeletal integrity through mechanotransduction. Skeletal muscle

contractions generate mechanical forces that are transmitted to bone, stimulating osteocytes and promoting osteoblast activity. Consequently, individuals with greater muscle mass tend to have higher bone mass and bone strength. In postmenopausal women, age-related sarcopenia may therefore exacerbate bone loss and further increase fracture risk.[6–8] Interestingly, basal metabolic rate (BMR) reflects the minimum energy expenditure required to maintain essential physiological functions at rest and is largely determined by body composition, particularly muscle mass. A higher BMR may indicate greater metabolic activity and energy availability for bone remodeling processes. However, BMR declines with aging, and its independent contribution to bone mass remains unclear.[9,10]

Given the rising prevalence of osteoporosis in aging populations, identifying modifiable factors related to bone mass is of paramount importance. Understanding the relative contributions of fat mass, muscle mass, and BMR may inform targeted interventions aimed at preventing osteoporosis and fractures in postmenopausal women. Therefore, this study aimed to examine the correlation between fat mass, muscle mass, and BMR with bone mass in postmenopausal women and to determine which of these variables is the strongest predictor of bone mass.

Methods

This study employed a cross-sectional descriptive analytic design using secondary data obtained from the 'Bandung Sports and Health Nabati Nutrition (B-SHENN) Project', conducted in Bandung, Indonesia, in January 2024. The B-SHENN Project was designed to evaluate nutritional status, body composition, and health indicators among adults adhering to plant-based dietary patterns. Ethical approval for the use of secondary data was granted by the Research Ethics Committee of Universitas Padjadjaran (No. 299/UN6.KEP/EC/2025).

The present analysis included data from postmenopausal women enrolled in the B-SHENN Project. Postmenopausal status was defined as the permanent cessation of menstruation for at least 12 consecutive months. Exclusion criteria were incomplete data on fat mass, muscle mass, BMR, or bone mass; history of hyperparathyroidism; current use of systemic corticosteroids; and the presence of bone injuries or conditions that could independently affect bone mass.

Sample size was determined using the Cochran formula with finite population correction. A total of 40 datasets met the inclusion and exclusion criteria and were included in the analysis.

Body composition parameters, including fat mass, muscle mass, BMR, and bone mass, were assessed using bioelectrical impedance analysis (BIA) following standardized measurement procedures. All measurements were recorded in kilograms (kg) for mass variables and kilocalories (kcal) for BMR.

Statistical analyses were performed using IBM SPSS version 26. Data distribution was assessed using the Shapiro–Wilk test. Descriptive statistics were used to summarize participant characteristics. Spearman's rho correlation analysis was applied to examine the relationships between fat mass, muscle mass, BMR, and bone mass. Multiple linear regression analysis was conducted to identify independent predictors of bone mass. Statistical significance was set at $p<0.05$.

Results

A total of 40 postmenopausal women were included in the analysis. Most participants were classified as elderly (≥60 years, 55%) and obese (47.5%) as presented in Table 1.

Fat mass, muscle mass, and BMR were

Table 1 Characteristics of Postmenopausal Women (n=40)

Variable	Frequency (n)	Percentage (%)
Age (years)		
Middle age: 40–59	18	45
Elderly: ≥60	22	55
BMI (kg/m^2)		
Underweight	1	2.5
Normal	11	27.5
Overweight	9	22.5
Obese	19	47.5

Note: BMI= body mass index

Table 2 Correlation Analysis and Coefficient of Determination Between Body Composition Parameters Bone Mass

Variable	r	p-value	R2
Fat mass (kg)	0.666	<0.001	0.426
Muscle mass (kg)	0.991	<0.001	0.985
BMR (kcal)	0.955	<0.001	0.914

Note: BMR= Basal metabolic rate

Table 3 Multiple Linear Regression Analysis of Factors Associated with Bone Mass

Variable	β (Standardized)	B (Unstandardized)	Std. Error	P value
Fat mass (kg)	–0.152	–0.006	0.005	0.294
Muscle mass (kg)	0.730	0.069	0.29	0.022
BMR (kcal)	0.377	0.001	0.001	0.367

Note: β= Standardized coefficient; B=Unstandardized coefficient; BMR= Basal metabolic rate

all positively and significantly correlated with bone mass (p<0.001). Muscle mass demonstrated the strongest correlation with bone mass (r=0.991), followed by BMR (r=0.955) and fat mass (r=0.666). The coefficient of determination indicated that muscle mass explained 98.5% of the variance in bone mass, suggesting that it is the main contributor to bone mass among postmenopausal women (Table 2).

Multiple linear regression analysis revealed that muscle mass was the only variable independently associated with bone mass (B=0.069; p=0.022). Fat mass and BMR were not significant predictors in the multivariable model. The coefficient of determination (R^2=0.985) indicated that muscle mass accounted for approximately 98.5% of the variance in bone mass (Table 3).

Discussion

This study demonstrates that fat mass, muscle mass, and BMR are positively correlated with bone mass in postmenopausal women, with muscle mass emerging as the strongest and only independent predictor. These findings reinforce the central role of skeletal muscle in maintaining bone health through mechanical loading and mechanotransduction mechanism.[8]

Fat mass showed a moderate positive correlation with bone mass, supporting evidence that increased body weight can enhance skeletal loading and stimulate bone formation.[2] However, excessive fat mass tends to have a more negative impact on bone, promoting bone resorption due to increased inflammatory cytokines.[5] In postmenopausal women, estrogen deficiency induces a chronic low-grade inflammatory state characterized by increased levels of pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6. Estrogen normally suppresses these cytokines and maintains the balance between bone formation and resorption. After menopause, elevated cytokine levels enhance RANKL expression and reduce osteoprotegerin (OPG), thereby promoting osteoclast differentiation and accelerating bone resorption.[15,19] Adipose tissue further contributes to cytokine production in postmenopausal women. Although higher fat mass can increase the mechanical loading on bone, excessive adiposity is associated with increased inflammatory cytokines that may counteract its protective effects.[2,5] This may explain why fat mass showed a positive correlation with bone mass but did not emerge as an independent predictor in the regression analysis. Estrogen deficiency also increases oxidative stress and cytokine-mediated osteocyte apoptosis, thereby impairing bone mechanosensing ability and bone quality.[13,19] In contrast, muscle mass exerts a protective effect through mechanical loading and the release of myokines with anti-inflammatory properties. Together, these mechanisms highlight the central role of estrogen–cytokine interactions in bone loss among postmenopausal women.[8,17]

The strong association between muscle mass and bone mass is consistent with previous studies showing that increased mechanical stress from muscle contractions stimulates osteogenesis and improves bone strength. In postmenopausal women, preservation of muscle mass may therefore

mitigate age- and estrogen-related bone loss.[9,10] Muscle mass contributes to bone mass through a mechanism called mechanical loading. Mechanical loading increases bone formation by stimulating osteoblasts.[11] This causes bone matrix deformation, resulting in apoptosis of osteocytes.[12,13] Osteocytes, which are mechanosensing cells, detect mechanical loading through bone matrix deformation.[14] Osteocytes' apoptosis causes the adjacent osteocytes that have not yet undergone apoptosis to increase the production of receptor activator of nuclear factor kappa-B ligand (RANKL). RANKL production results in osteoclast activation, leading to bone resorption. Bone resorption begins with the binding of parathyroid hormone (PTH) to receptors found on osteoblasts.[15] Osteoblasts will form activating receptors for RANKL and release macrophage-colony stimulating factor (M-CSF). RANKL will bind to receptor activator of nuclear factor-κβ (RANK), while M-CSF binds to its receptors on preosteoclast cells, which causes preosteoclast cells to differentiate into osteoclasts. Osteoclasts will then dissolve bone matrix for about 3 weeks, resulting in a tunnel formation with 0.2–1 mm in diameters. In this phase, osteoclasts will undergo apoptosis, and the tunnel will be filled with osteoblasts, which are responsible for synthesizing, transporting, and assembling the matrix and regulating bone mineralization.[16] Osteoblasts secrete collagen molecules and ground substances. Collagen monomers polymerize rapidly to form collagen fibers, and the generated tissue becomes osteoid. As osteoid forms, some osteoblasts are trapped within the osteoid and become quiescent. The trapped osteoblasts are called osteocytes, which make up 95% of the bone matrix. In other words, an increase in osteocyte concentration leads to increased bone mass. In addition, muscle myokines also contribute to bone formation.[17] Myokine secretion increases during exercise. Irisin, secreted during skeletal muscles contraction, promotes osteocytes' survival and sclerostin synthesis, thereby reducing bone resorption. L-β-aminoisobutyric acid (L-BAIBA), which enhances muscle contraction and strength, helps maintain bone mass by inhibiting bone cell apoptosis caused by Reactive Oxygen Species (ROS).[17]

Basal metabolic rate (BMR) has been found to correlate strongly with bone mass, however, BMR does not independently predict bone mass after adjustment for other variables. As BMR is largely determined by muscle mass, its association with bone mass is likely mediated primarily through muscle-related mechanisms rather than representing an independent effect. The BMR is the minimum total energy expenditure at rest, which is essential for maintaining vital physiological functions.[18] Furthermore, BMR is determined by body composition, with muscle mass being a main contributor. Therefore, higher muscle mass leads to a higher BMR because of increased energy expenditure at rest. Several other factors affect BMR, incllluding age, sex, genetics, and hormonal regulation.[19] The multiple factors influencing BMR may explain why BMR is not a strong predictor of bone mass.

Understanding key factors influencing bone mass in postmenopausal women is essential for developing strategies to minimize the risk of osteoporosis and its complications. The significant influence of muscle mass highlights the need to increase muscle mass through resistance training, such as high-intensity and high-impact exercises and physical activity as key interventions for maintaining bone health.[20] Additionally, although higher fat mass may contribute to higher bone mass in postmenopausal women, excessive fat accumulation may increase the risk of cardiovascular disease and other metabolic disorders. Therefore, further studies should explore the safe limit of fat mass in postmenopausal women so as not to increase the risk of cardiovascular disease and metabolic disorders.

Several limitations should be considered. In this study, physical activity levels, osteoporosis status, and duration since menopause were not available, which may influence bone mass. Furthermore, the cross-sectional design precludes causal inference. Although participants were categorized into two age groups, this classification was used for descriptive purposes only. Comparative analyses between age groups were not performed due to limited sample size. In addition, bone mass was estimated using bioelectrical impedance analysis (BIA), which, although practical and non-invasive, has lower accuracy for bone mass assessment compared with dual-energy X-ray absorptiometry (DXA), the gold standard for bone evaluation. Differences in measurement principles may have influenced the precision of bone mass estimates and should be considered when interpreting the results. Therefore, future studies with larger sample sizes with longitudinal designs are needed to clarify causal relationships between

body composition and bone health. Detailed information on menopausal onset and duration is also essential to enable meaningful age-group comparisons and a more comprehensive understanding of age- and menopause-related differences in bone health. The inclusion of gold-standard bone assessment methods, such as dual-energy X-ray absorptiometry (DXA), along with detailed data on physical activity, inflammatory markers, hormonal profiles, and osteoporosis status will help to better elucidate the mechanisms linking muscle mass, inflammation, metabolic factors, and bone health in postmenopausal women.

In conclusion, the findings of this study demonstrate that fat mass, muscle mass, and BMR are associated with bone mass in postmenopausal women with muscle mass emerging as the strongest predictor. These findings underscore the importance of maintaining or increasing skeletal muscle resistance training and regular physical activity as an effective strategy for preserving bone health and reducing the risk of osteoporosis. By identifying muscle mass as a modifiable factor, this study contributes to aging-related diseases prevention and support healthy skeletal aging and improved quality of life in postmenopausal women.

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Authors' Contributions

LL conceived and designed the study, performed data analysis and interpretation, drafted the manuscript, and served as the corresponding author. RAP contributed to the conceptual framework, critical revision of the manuscript for important intellectual content, and supervision of the research process. GS contributed to data acquisition, data curation, and assisted in manuscript preparation and revision. All authors read and approved the final version of the manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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