

The relationship between some serum osteoporosis markers and body mass index with lumbar bone mineral density inactive postmenopausal women

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How to Cite: Roozbahani, Sh, & Vahabidelshad, R. (2024). The relationship between some serum osteoporosis markers and body mass index with lumbar bone mineral density inactive postmenopausal women, *Journal of New Approaches in Exercise Physiology*, 6(12), 146-178.

DOI: 10.22054/nass.2025.88862.1191

Introduction

Osteoporosis is a progressive, systemic skeletal disorder characterized by low bone mass and deterioration of bone microarchitecture, which together reduce bone strength and substantially increase susceptibility to fragility fractures (Keen & Keen, 2023; Demontiero et al., 2012). Because the disease is often clinically “silent” until a fracture occurs, osteoporosis is widely recognized as a major public health problem in aging societies, with considerable downstream consequences for disability, loss of independence, and health-care costs (International Osteoporosis Foundation [IOF], n.d.; Keen & Keen, 2023). Fragility fractures—typically resulting from low-energy trauma such as a fall from standing height—are considered a hallmark of underlying osteoporosis and signal a markedly elevated risk of subsequent fractures, particularly in the first years after an index event (IOF, n.d.). Among fracture sites, the hip and vertebrae are of special concern because they are strongly associated with pain, deformity, functional decline, and excess mortality (IOF, n.d.).

From a biological perspective, skeletal integrity is maintained through continuous bone remodeling, a coupled process in which osteoclast-mediated resorption and osteoblast-mediated formation renew and adapt bone tissue throughout life (Cheng et al., 2022; Demontiero et al., 2012). With advancing age, remodeling becomes increasingly unbalanced—often due to increased resorption, impaired formation, or both—leading to net bone loss and compromised microarchitecture (Demontiero et al., 2012). In women, the menopausal transition represents a critical accelerant of bone loss. Estrogen deficiency increases bone turnover and promotes osteoclast activity, thereby amplifying resorption and accelerating declines in bone mineral density (BMD) and bone strength (Cheng et al., 2022; de Villiers et al., 2024). This endocrine-driven phase of rapid loss may persist for years after menopause and then transitions to slower age-related loss, collectively explaining the heightened lifetime risk of osteoporosis among postmenopausal women (Demontiero et al., 2012; Cheng et al., 2022).

Importantly, these skeletal changes can occur without overt symptoms, emphasizing prevention and early detection rather than fracture-driven diagnosis (Keen & Keen, 2023).

Clinically, osteoporosis is commonly diagnosed using dual-energy X-ray absorptiometry (DXA), which remains the recognized reference method for measuring areal BMD at the lumbar spine and hip (Sangondimath et al., 2023; ISCD, 2023). The World Health Organization (WHO) classification operationalizes osteoporosis as a BMD T-score ≤ -2.5 at relevant skeletal sites (Kanis et al., 1994; WHO, 1994), a threshold that has been integrated into international densitometry standards and interpretation guidance (ISCD, 2023). Although BMD is a strong predictor of fracture risk, fracture occurrence is multifactorial; clinical risk factors (e.g., age, prior fracture, family history, smoking, glucocorticoid exposure, comorbidities, and falls risk) interact with bone density and quality to determine the probability of future fracture (Keen & Keen, 2023; Demontiero et al., 2012). Accordingly, contemporary practice guidelines emphasize a comprehensive risk assessment model that combines densitometry with clinical risk profiles to guide prevention and treatment (Eastell et al., 2019; Shoback et al., 2020).

Among modifiable determinants, nutritional status is central to skeletal health. Adequate calcium and vitamin D intake supports mineralization and helps maintain normal calcium–phosphate homeostasis, which is tightly regulated by endocrine axes involving parathyroid hormone and vitamin D metabolites (Cosman et al., 2014; Demontiero et al., 2012). Clinical guidance for osteoporosis prevention and fracture reduction typically includes ensuring sufficient dietary calcium, maintaining vitamin D adequacy, and addressing lifestyle risks such as smoking and excess alcohol consumption (Cosman et al., 2014; Eastell et al., 2019). However, nutritional sufficiency alone is often insufficient to fully counteract menopausal and age-related losses in bone strength, particularly in high-risk individuals, underscoring the need for

integrated lifestyle and, when appropriate, pharmacologic strategies (Eastell et al., 2019; Shoback et al., 2020).

Physical activity is a cornerstone non-pharmacological approach because mechanical loading is a primary signal for bone adaptation. Mechanobiological frameworks, including Frost's mechanostat concept, propose that bone mass and structure are regulated by thresholds of mechanical strain, with insufficient loading favoring resorption and adequate dynamic loading stimulating modeling and remodeling toward improved strength (Frost, 1983; Frost, 2003). Exercise exerts osteogenic effects not only through direct skeletal loading but also via improvements in muscle strength, neuromuscular function, and balance, which can reduce falls—an essential pathway for lowering fracture risk in older adults (Pinheiro et al., 2020; Pagnotti et al., 2019). Consistent with this mechanistic rationale, systematic reviews and meta-analyses indicate that structured exercise interventions (including resistance training, impact loading, and multicomponent programs) can mitigate BMD loss and, in some contexts, modestly increase BMD at clinically relevant sites such as the lumbar spine and femoral neck in postmenopausal women (Rahimi et al., 2020; Mohebbi et al., 2023). Nevertheless, exercise responsiveness is heterogeneous and depends on training mode, intensity, duration, supervision, baseline bone status, and adherence—factors that may explain inconsistencies across trials and populations (Rahimi et al., 2020; Mohebbi et al., 2023).

Body size and composition, typically approximated by body mass index (BMI), also influence skeletal outcomes through both mechanical and metabolic pathways. Higher body weight may increase habitual skeletal loading, potentially supporting higher BMD, while low BMI and low body weight are well-established risk factors for low BMD and fracture (Keen & Keen, 2023; Cai et al., 2025). At the same time, the relationship between obesity and bone health is complex: adipose tissue can contribute to chronic low-grade inflammation and unfavorable

endocrine profiles, and BMI cannot distinguish lean mass from fat mass, which may have divergent effects on bone (Pagnotti et al., 2019; Ge et al., 2025). Recent evidence syntheses in postmenopausal women generally report a positive correlation between BMI and BMD, though the magnitude of association varies by skeletal site and individual characteristics (Cai et al., 2025). These nuances suggest that evaluating BMI alongside activity status and metabolic markers may offer a more informative picture of skeletal risk than any single parameter alone.

In addition to DXA-derived BMD, biochemical indices can provide complementary insight into bone and mineral metabolism. Bone turnover markers (BTMs) reflect formation and resorption activity and are increasingly used in clinical and research settings for monitoring treatment response and adherence, as emphasized by international standardization efforts (Vasikaran et al., 2011; Schini et al., 2023). While reference BTMs often include serum PINP and CTX (Vasikaran et al., 2011; Wu et al., 2021), more widely available serum measures—such as alkaline phosphatase (ALP) and mineral indices including calcium and phosphorus—remain relevant in many settings as accessible indicators related to mineral homeostasis and bone remodeling context (Tariq et al., 2019; Schini et al., 2023). However, the strength and direction of associations between these serum indices and site-specific BMD can differ by age, menopausal status, health conditions, and physical activity, contributing to the ongoing need for population- and context-specific evaluation (Tariq et al., 2019; Schini et al., 2023). Importantly, in physically active individuals, exercise-induced adaptations in calcium handling, endocrine signaling, and remodeling dynamics may alter the relationships between serum indices and BMD relative to inactive counterparts—an issue with practical implications for screening and risk stratification.

Despite the strong biological rationale linking activity patterns, body size, and mineral-related serum indices to skeletal health, the combined relationships among these factors—particularly in real-world groups

differing by habitual physical activity—are not always clear. This gap is clinically meaningful because postmenopausal women constitute a high-risk population, and cost-effective strategies for identifying those at greatest risk (and those most likely to benefit from lifestyle modification) remain a priority in osteoporosis prevention (Eastell et al., 2019; Pinheiro et al., 2020). In the present study, the focus is specifically on the lumbar spine, a clinically important site for osteoporotic change and DXA interpretation (ISCD, 2023), and on readily obtainable predictors that may be feasible for broad screening approaches.

Accordingly, the aim of this study was to examine the relationship between selected serum osteoporosis-related indices (including calcium, phosphorus, and alkaline phosphatase) and anthropometric characteristics—particularly BMI—in relation to lumbar BMD in active and inactive postmenopausal women.

By comparing women differentiated by habitual physical activity, this approach seeks to clarify whether activity status modifies the associations between biochemical indices, BMI, and lumbar BMD, thereby informing prevention-oriented assessment strategies in postmenopausal populations. Therefore, the aim of this study was to the relationship between some serum osteoporosis markers and body mass index with lumbar bone mineral density in active and inactive postmenopausal women.

Methods

Study design and participants

This study used a cross-sectional, correlational design to investigate the relationships between selected serum biochemical indices, anthropometric characteristics, and lumbar spine bone mineral density (BMD) in active versus inactive postmenopausal women. Reporting

was structured to align with recommended standards for observational research (von Elm et al., 2007). The sampling frame consisted of 350 postmenopausal women (mean age: 72.3 years) with archived clinical and laboratory records in the hospital electronic database. Eligible patients had experienced menopause at least two years prior and had undergone a bone densitometry assessment at least six months before study procedures, ensuring that the densitometry outcomes were available for extraction from medical files.

Participants and grouping

After identification through hospital archives, potential participants were contacted and asked to complete a structured questionnaire capturing demographic and personal information, osteoporosis-related history, and habitual physical activity status. Based on questionnaire responses and record availability, 41 women were classified as physically active and 45 as physically inactive and were included in the final analysis set. The inactive group comprised women who did not report engagement in any structured or specific sports activity and were selected to be broadly comparable in age to the active group. In contrast, the active group comprised women who reported participating in regular physical activity for three sessions per week for at least one year, representing sustained, habitual activity rather than short-term participation.

Eligibility criteria

Inclusion criteria were: (i) female sex; (ii) postmenopausal status; (iii) age between 41 and 87 years; (iv) availability of relevant medical records including laboratory indices and densitometry results within the hospital system; and (v) feasibility of follow-up contact via telephone or internet. Exclusion criteria were applied to reduce confounding related to conditions and treatments known to substantially influence bone metabolism. These criteria included the presence of chronic

disease, current treatment for osteoporosis, and a history of hormone drug use.

Ethics and consent

All participants provided informed consent permitting the use of their archived laboratory test results and bone densitometry measurements for research purposes. Data were handled in an anonymized format during analysis, and only variables necessary to address the research objectives were extracted.

Measures and data extraction

The primary outcome was lumbar spine BMD derived from bone densitometry records (DXA-based reports). DXA is the internationally accepted clinical approach for assessing BMD, and standard reporting commonly relies on lumbar spine measurements using a posteroanterior (PA) spine region of interest in adult patients (ISCD, 2023). Serum biochemical indices relevant to mineral metabolism and bone turnover context were obtained from hospital laboratory records and included serum calcium, serum phosphorus, and serum alkaline phosphatase (ALP). While contemporary international recommendations identify serum PINP and serum CTX as reference bone turnover markers for standardization in research and clinical monitoring, routinely available indices such as calcium, phosphate, and ALP remain widely used in hospital settings for supportive metabolic assessment (Vasikaran et al., 2011).

Anthropometric characteristics assessed for each participant included age, body weight, height, and body mass index (BMI). BMI was calculated using the standard formula (kg/m^2) based on recorded or questionnaire-confirmed height and weight.

Statistical analysis

Descriptive statistics were generated for all variables, and between-group characterization (active vs inactive) was conducted to ensure the interpretability of subsequent correlational analyses. The primary analytic approach used Pearson's correlation coefficient to quantify linear associations between lumbar spine BMD and (i) serum calcium, phosphorus, and ALP and (ii) anthropometric indices (age, weight, height, BMI), separately within active and inactive groups. Statistical analyses were performed using IBM SPSS Statistics (Version 26). Two-tailed significance testing was applied with an alpha level of 0.05.

Results

The values of the correlation coefficient between osteoporosis indices and lumbar bone mineral density of active postmenopausal women are shown in Table 1. According to the data in Table 1, it is observed that in active postmenopausal women, a significant relationship was observed between anthropometric characteristics (age, weight, body mass index) and serum indices (calcium, phosphorus and alkaline phosphatase) and lumbar bone mineral density ($P \leq 0.05$).

Table 1: Correlation coefficient between osteoporosis indices and lumbar bone mineral density (BMD) in active postmenopausal women.

Osteoporosis indices Bone	Mineral density (BMD) Active group	
	Correlation Coefficient (r)*	P-Value*
Age (years)	-71/0	0.024*

Height (cm)	-34/0	0.231
Weight (kg)	78/0	0.020*
Body mass index (BMI) (kg/m²)	69/0	0.015*
Serum calcium level	66/0	0.041*
Serum phosphorus level	73/0	0.016*
Serum alkaline phosphatase level	87/0	0.014*

- Significant difference at the $P \leq 0.05$ level, *Pearson correlation coefficient

The values of the correlation coefficient between body mass index and lumbar bone mineral density of inactive postmenopausal women are shown in Table 3. According to Table 2, it can be seen that in inactive postmenopausal women, a significant relationship was observed between anthropometric characteristics (age, weight, body mass index) and lumbar bone mineral density ($P \leq 0.05$). While no significant relationship was found between other indicators (calcium, phosphorus and alkaline phosphatase levels) ($P \geq 0.05$).

Table 2: Correlation coefficient between osteoporosis indicators and lumbar bone mineral density (BMD) of inactive postmenopausal women.

Osteoporosis indices	Bone mineral density (BMD) inactive group (n=60)	
	Correlation coefficient (r)*	p-value*
Age (years)	-75/0	0.025*
Height (cm)	-28/0	0.631
Weight (kg)	84/0	0.030*
Body mass index (BMI) (kg/m ²)	76/0	0.014*
Serum calcium level	39/0	0.37
Serum phosphorus level	46/0	0.326
Serum alkaline phosphatase level	45/0	0.114

*Significant difference at the $P \leq 0.05$ level, •Pearson correlation coefficient

Discussion

The purpose of this study was to clarify how routine serum indices (calcium, phosphorus, alkaline phosphatase) and body mass index relate to lumbar spine BMD in postmenopausal women stratified by habitual physical activity. The key finding was that, among active postmenopausal women, lumbar BMD showed statistically significant positive associations with serum calcium, phosphorus, and alkaline phosphatase, alongside the expected associations with anthropometric variables (positive correlations with weight and BMI and a negative correlation with age). In contrast, among inactive postmenopausal women, lumbar BMD was significantly associated with age (inverse) and with weight and BMI (positive), but did not demonstrate statistically significant relationships with serum calcium, phosphorus, or alkaline phosphatase.

From a biological standpoint, these results suggest that physical activity status may modify the observable linkage between circulating biochemical indices and skeletal mineral density in postmenopausal women. This does not necessarily imply that exercise “creates” a causal relationship between serum minerals/ALP and BMD; rather, activity may influence remodeling dynamics, mineral fluxes, and the coupling between formation and resorption in ways that make routine indices more reflective of skeletal state in active individuals. These findings align conceptually with modern models in which bone is an adaptive, mechanosensitive tissue responding to dynamic mechanical loading through coordinated changes in osteoblast and osteoclast activity (Turner, 1998; Pagnotti et al., 2019).

A substantial evidence base supports the role of physical activity in maintaining BMD and reducing osteoporotic risk, particularly in postmenopausal women. Updated systematic reviews and meta-analyses indicate that controlled exercise interventions lasting at least several months can improve BMD at the lumbar spine and hip, though the magnitude of improvement depends on exercise mode, intensity, and supervision (Mohebbi et al., 2023). Cochrane evidence summaries

similarly conclude that exercise yields small improvements in BMD and may modestly reduce fracture risk, with the strongest skeletal stimulus generally arising from weight-bearing impact and resistance training rather than low-load activity alone (Cochrane, 2022). Importantly, exercise also reduces falls—an independent and clinically critical pathway—by improving balance, strength, and functional capacity in older people (Sherrington et al., 2020).

Mechanistically, the association between activity and BMD is grounded in mechanotransduction. Turner’s “rules” emphasize that bone responds primarily to dynamic, high-rate loading, that only relatively brief bouts may be needed to trigger adaptation, and that bone cells accommodate to routine loading patterns over time (Turner, 1998). The mechanostat concept further suggests a threshold phenomenon: below a minimum effective strain, bone mass is not maintained; above it, formation is promoted (Frost, 1992). Thus, postmenopausal women engaging in regular activity—especially if it includes weight-bearing and resistance components—may maintain higher BMD because loading signals promote osteogenesis and reduce the net loss that otherwise accompanies estrogen deficiency (Watson et al., 2018; Pagnotti et al., 2019).

Within this framework, the observed presence of significant correlations between serum indices and BMD in the active group could reflect a more “responsive” or more actively remodeling skeleton. Exercise can acutely and chronically alter calcium balance by increasing intestinal calcium absorption (partly through vitamin D-related pathways), reducing urinary calcium loss under certain conditions, and influencing endocrine regulators such as PTH—effects that may be particularly relevant in older adults whose baseline absorption efficiency may be reduced (LeBoff et al., 2022). While serum calcium remains tightly controlled, regular mechanical loading may increase the flux of calcium into bone formation sites (osteoid mineralization) and may modulate the coupling between resorption and

formation, potentially strengthening the statistical association between circulating indices and BMD when comparing individuals across a range of BMD values (Khan & Lederer, 2022; Pagnotti et al., 2019).

In the active group, serum calcium and phosphorus were significantly related to lumbar BMD. Physiologically, calcium and phosphate availability are essential for hydroxyapatite deposition, and their systemic regulation is tightly governed by PTH and vitamin D pathways (Khan & Lederer, 2022). PTH increases plasma calcium by stimulating bone resorption (when needed), increasing renal calcium reabsorption, and increasing active vitamin D production, which enhances intestinal calcium absorption (Khan & Lederer, 2022). Phosphate interacts with this axis, and elevations in phosphate can stimulate PTH secretion, while renal handling and hormonal control maintain mineral homeostasis (Khan & Lederer, 2022). These mechanisms explain why serum calcium and phosphate are often relatively stable even in osteoporosis—limiting their standalone diagnostic value (LeBoff et al., 2022).

However, stability at the population level does not preclude meaningful associations in specific contexts. In active individuals, several factors could strengthen correlations: (1) greater variability in mineral intake/absorption among those engaging in health-promoting behaviors; (2) exercise-related shifts in mineral handling and bone turnover; or (3) differences in skeletal demand and remodeling responsiveness. Notably, prior studies have reported mixed results regarding serum calcium/phosphate as predictors of BMD. Tariq et al. (2019) reported that calcium and phosphate were stronger predictors of T-score in certain postmenopausal groups, while the predictive value varied by skeletal status (e.g., osteopenic vs normal). Such heterogeneity suggests that context—including nutritional status, renal function, and activity—may condition whether serum minerals track with BMD (Tariq et al., 2019).

In the inactive group of the present study, no significant relationships were observed between serum calcium/phosphorus and lumbar BMD. This pattern may reflect the fundamental limitation that circulating mineral concentrations are maintained within narrow ranges by endocrine control, thereby “masking” underlying skeletal depletion—particularly in sedentary individuals whose remodeling may be dominated by resorption driven by estrogen deficiency and age-related factors rather than by anabolic loading signals (LeBoff et al., 2022; Sapir-Koren & Livshits, 2017). In other words, inactivity may reduce the variability and skeletal responsiveness that would allow serum indices to correlate detectably with BMD across individuals.

The strongest biochemical association in the active group was between alkaline phosphatase and lumbar BMD. ALP is often considered a marker of bone formation because osteoblasts express bone-specific ALP during matrix mineralization; nonetheless, total ALP is not bone-specific and may reflect hepatic or other sources (Tariq et al., 2019). This duality makes interpretation challenging: elevated ALP can indicate increased bone turnover (which may occur in osteoporosis), but it can also reflect non-skeletal pathology. Nevertheless, several studies support an association between ALP and bone status in older adults. Tariq et al. (2019) found that ALP was a stronger predictor of T-score in osteopenic postmenopausal females, whereas calcium and phosphate predicted T-score better in other subgroups—again highlighting effect modification by baseline skeletal status.

How might exercise influence an ALP–BMD relationship? One plausible explanation is that in physically active women, the remodeling balance may be shifted toward improved formation relative to resorption (or at least better coupling), and ALP may therefore serve as a closer proxy of osteoblastic activity supporting mineral accrual or maintenance. Mechanical loading stimulates osteocyte signaling that promotes osteoblastogenesis and downregulates inhibitors of bone formation, and high-intensity resistance/impact training has shown

meaningful improvements in bone-related outcomes in postmenopausal women with low bone mass (Watson et al., 2018). If active participants in this study engaged in sufficiently osteogenic activity (three sessions weekly over ≥ 1 year), their ALP values may better reflect physiologically relevant formation activity contributing to higher BMD.

By contrast, in inactive women, ALP may not track with BMD because total ALP could reflect heterogeneous influences unrelated to skeletal formation, while bone turnover may be characterized by resorption predominance that is not captured well by total ALP alone. Contemporary bone marker consensus statements emphasize that for monitoring therapy or capturing remodeling dynamics, more specific markers such as PINP and CTX are preferable (Wu et al., 2021; Bhattoa et al., 2025). The implication for the present findings is that while total ALP demonstrated a useful association in active women, future work should incorporate bone-specific ALP and/or standardized BTMs (PINP/CTX) to clarify whether the observed correlations reflect genuine formation dynamics rather than indirect or confounded signals.

Both active and inactive groups demonstrated positive relationships between BMI (and weight) and lumbar spine BMD. This pattern is widely observed in bone epidemiology because higher body mass increases habitual mechanical loading on the skeleton, which can stimulate bone formation and reduce age-related loss (Pagnotti et al., 2019). Moreover, adipose tissue influences endocrine factors (e.g., leptin, adipokines, inflammatory mediators) that can exert complex effects on bone, sometimes supporting higher BMD while potentially impairing bone quality depending on fat distribution and metabolic health (Pagnotti et al., 2019; Ghanemi et al., 2023). A recent systematic review and meta-analysis in postmenopausal women reported an overall positive correlation between BMI and BMD across skeletal sites, with variation in magnitude by site (Cai et al., 2025).

However, an important nuance is that higher BMD associated with higher BMI does not automatically translate into lower fracture risk. The “obesity paradox” literature notes that while obesity may be associated with higher BMD, fracture risk can remain elevated at certain sites due to poorer balance, higher fall forces, altered bone quality relative to body weight, sarcopenic obesity, and metabolic inflammation (Paccou et al., 2024; Ghanemi et al., 2023). Therefore, while BMI and weight may help identify women at risk of low BMD (particularly those with low BMI), fracture prevention still requires integrated assessment of falls risk, muscle function, comorbidities, and medication exposures (LeBoff et al., 2022).

Age was inversely associated with lumbar spine BMD in both activity groups. This is consistent with established trajectories of skeletal aging: peak bone mass is generally achieved by early adulthood, after which bone mass gradually declines; in women, the decline accelerates after menopause because estrogen withdrawal increases remodeling rate and net resorption (Sapir-Koren & Livshits, 2017; LeBoff et al., 2022). In women in their 70s (as in the overall study frame), cumulative exposure to estrogen deficiency, reduced physical activity, and comorbidities can contribute to substantial variation in BMD. Importantly, lumbar spine DXA in older adults can be influenced by degenerative changes (e.g., osteophytes, aortic calcification) that may elevate measured BMD, which underscores the need for careful DXA interpretation and, where possible, complementary assessment at the hip and/or trabecular bone score (ISCD, 2023).

The fact that age remained strongly related to BMD even among active women indicates that activity may attenuate but does not eliminate age-associated skeletal decline. This aligns with intervention evidence: exercise can improve or preserve BMD modestly, but it is not a complete substitute for other preventive and therapeutic strategies in high-risk postmenopausal women, such as ensuring adequate calcium and vitamin D intake, fall prevention, and pharmacologic therapy when indicated (LeBoff et al., 2022; Eastell et al., 2019).

Several practical implications emerge from this work. First, the consistent association of BMI/weight with lumbar BMD in both groups reinforces the utility of anthropometric screening to identify women who may warrant DXA evaluation—particularly those with low BMI or unintentional weight loss, which may signal heightened risk of low BMD and frailty (LeBoff et al., 2022). Second, the presence of significant serum index–BMD associations only in active women suggests that clinicians and researchers should be cautious when interpreting routine serum calcium/phosphate/ALP values as proxies for skeletal status in sedentary older women; in such populations, these values may remain normal despite substantial bone loss due to endocrine buffering (Khan & Lederer, 2022). Third, the findings provide additional rationale for implementing community-based physical activity programs in postmenopausal women—not only to support BMD, but also to improve strength and balance and thereby reduce falls (Sherrington et al., 2020).

Finally, while routine serum indices are attractive because they are inexpensive and widely available, current expert consensus supports using validated bone turnover markers (PINP and CTX) for more accurate assessment of remodeling activity, particularly for monitoring therapy and adherence (Wu et al., 2021; Bhattoa et al., 2025). Therefore, the present findings can be viewed as an initial step: they highlight potentially informative relationships in active women, but also point toward the need for improved biomarker selection if the goal is to predict osteoporosis risk or track skeletal changes with greater

Limitations and directions for future research

This study has limitations that should temper interpretation. The cross-sectional design precludes causal inference, meaning that observed correlations cannot determine whether biochemical indices influence BMD or whether underlying factors influence both. Physical activity status was classified by questionnaire and defined broadly (three sessions/week for ≥ 1 year), without objective measurement

(accelerometry) or detailed characterization of exercise modality, intensity, and progression—variables known to determine osteogenic response (Mohebbi et al., 2023; Turner, 1998). Laboratory indices were drawn from medical records, and total ALP is not bone-specific; without liver function markers or bone-specific ALP, misclassification is possible (Tariq et al., 2019). Additionally, important confounders—vitamin D status, PTH concentration, renal function, dietary intake, medication use beyond hormones, and inflammatory conditions—were not explicitly modeled but could influence both serum indices and BMD (LeBoff et al., 2022; Khan & Lederer, 2022).

Conclusion

In summary, the present study indicates that in active postmenopausal women, lumbar spine BMD is significantly associated with serum calcium, phosphorus, and alkaline phosphatase, alongside expected anthropometric correlates, whereas in inactive women, BMD relates primarily to age and body size measures rather than these routine serum indices. These findings support the importance of habitual physical activity for skeletal health and suggest that activity status may shape how readily accessible biochemical indices relate to BMD in older women. From an application standpoint, combining anthropometric screening with targeted DXA assessment and promoting structured physical activity—particularly weight-bearing, resistance, and balance training—remains a rational approach to osteoporosis prevention and risk reduction in postmenopausal populations (LeBoff et al., 2022; Mohebbi et al., 2023; Sherrington et al., 2020).

Funding:

This research received no external funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Acknowledgments:

We sincerely thank and appreciate all the students who collaborated with the researchers in this study.

Conflicts of Interest:

The author declares no conflict of interest.

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How to Cite: Roozbahani, Sh, & Vahabidelshad, R. (2024). The relationship between some serum osteoporosis markers and body mass index with lumbar bone mineral density inactive postmenopausal women, *Journal of New Approaches in Exercise Physiology*, 6(12), 146-178.

DOI: 10.22054/nass.2025.88862.1191



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