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THE ASSOCIATION BETWEEN PLATELET-TO-LYMPHOCYTE RATIO AND BONE MINERAL DENSITY IN REPRODUCTIVE-AGED WOMEN

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ABSTRACT

Background: Bone Mineral Density (BMD) decreases in women aged 40–49 years. Platelet-Lymphocyte Ratio (PLR) is an inflammatory marker involved in the pathophysiology of osteoporosis. **Objective:** This study aimed to analyze the relationship between PLR and BMD in productive-age women for early osteoporosis risk detection. **Method**: This analytic observational study with a cross-sectional design included 107 medical records of productive-age women at Diponegoro National Hospital from March to September 2024, selected by consecutive sampling. The relationship between PLR and BMD was analyzed using the Spearman correlation test. **Results**: The analysis of Spearman correlation test showed that there was no significant relationship between PLR value and BMD value of spine L1 - L4 ($p = 0.713$; $r = -0.036$), PLR value and BMD value of left femur neck ($p = 0.883$; $r = 0.014$), and PLR value and BMD value of left femur total ($p = 0.532$; $r = 0.061$). **Conclusion:** There was no association between PLR and BMD of the L1–L4 spine, left femur neck, or left femur total; thus, it is not a marker of inflammation.

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BACKGROUND

Women of reproductive age, defined as those aged 15–49 years regardless of marital status, represent a critical period in skeletal health[1]. During this phase, peak bone mass is typically achieved, particularly between the ages of 20–29, as a result of a balanced interaction between bone formation and resorption, which is highly influenced by hormonal regulation, especially estrogen[2].

Low-grade inflammation contributes to osteoporosis by enhancing osteoclast activity and impairing bone formation via cytokines such as TNF-α and IL-6[3,4]. This chronic inflammatory state, intensified by aging and estrogen deficiency, correlates with reduced bone mineral density and increased fracture risk[5–7]. Targeting inflammatory pathways may offer novel strategies for osteoporosis prevention[8].

Inflammatory and hormonal pathways are closely intertwined in bone regulation, particularly through the modulatory effects of estrogen on immune function and bone cell activity[9]. Its deficiency such as during menopause leads to increased bone turnover and loss, while other reproductive hormones (e.g., FSH, prolactin, oxytocin) contribute to bone regulation through the hypothalamic-pituitary-gonadal axis[10,11]. Hormonal imbalance significantly elevates osteoporosis risk, highlighting the importance of endocrine regulation in bone health[12,13].

With increasing age, the risk of skeletal fragility rises. Approximately 29% of women over the age of 45 experience bone loss, which contributes to falls and fractures due to diminished bone mass and quality[14]. Data from Indonesia's Basic Health Research (Riskesdas) between 2007–2018 show an upward trend in fracture prevalence across age groups: 5.3% (15–24 years), 6.2% (25–34 years), 6.5% (35–44 years), and 7.9% (45–54 years) [15]. Notably, more than half of women with fragile bones (51.47%) still

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menstruate, indicating that bone density disturbances can occur even before menopause[16].

International studies emphasize that bone health in premenopausal women significantly influences future osteoporosis risk, with early fractures and reproductive factors (e.g., menarche and menopause timing) linked to long-term BMD outcomes[17–19]. Although systemic inflammation shows weaker associations with BMD in premenopausal women, hormonal changes and estrogen exposure remain critical modulators of bone integrity across the reproductive lifespan[18,20]. These findings highlight the importance of early risk assessment to inform preventive strategies before menopause[21].

Osteoporosis is an age-related skeletal disease characterized by reduced bone mass and microarchitectural deterioration, which may also affect women of reproductive age as they approach menopause[22]. Individuals with low BMD are at increased risk for fractures and long-term skeletal complications[23]. Evidence suggests that low bone density is already present among women aged 40–49, supporting the need for early screening in this age group[22].

Dual-energy X-ray absorptiometry (DXA) is a widely used method for measuring BMD, providing assessments of the whole skeleton and specific fracture-prone areas[24]. The World Health Organization (WHO) recognizes DXA as the global "gold standard" for BMD measurement[25]. Common DXA scan sites include the lumbar spine, femoral neck, trochanteric region, and distal forearm, with BMD expressed in grams per square centimeter (g/cm^2)[26].

In recent years, attention has shifted toward hematological markers such as the Platelet-Lymphocyte Ratio (PLR), which offers a stable, cost-effective, and widely accessible indicator of systemic inflammation compared to cytokines like IL-6 or TNF-α[27,28]. PLR has been associated with several chronic inflammatory conditions and osteoporosis risk, particularly in postmenopausal women [27]. Chen et al. (2023) found a negative correlation between PLR and BMD at the lumbar spine, total femur, and femoral neck in postmenopausal women[27]. Similarly, Tang et al. (2022) reported PLR as a predictive marker for osteoporosis risk [23].

Given these findings, women of reproductive age with low BMD may be at risk for worsening skeletal health as they transition into menopause. Considering the established association between systemic inflammation and bone loss in postmenopausal populations, this study aims to investigate the relationship between Platelet-Lymphocyte Ratio (PLR) and Bone Mineral Density (BMD) in women of reproductive age a population that remains understudied yet holds significant potential for early preventive intervention.

METHODS

This cross-sectional study was conducted on 107 women of reproductive age at Diponegoro National Hospital, Semarang, from March to September 2024. Eligible participants were women aged 15–49 years, not menopausal, and with regular menstruation within the past three months [29]. Exclusion criteria included a history of diabetes mellitus, cardiovascular disease, autoimmune disorders, malignancy, chronic infectious diseases, corticosteroid use, or current use of hormonal contraceptives. The study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Diponegoro University (No.266/EC/KEPK/FK-UNDIP/V/2024). Statistical analyses were performed using SPSS version 26.0. Correlations between Platelet-Lymphocyte Ratio (PLR) and Bone Mineral Density (BMD) were evaluated using Spearman's or Pearson's correlation tests, depending on data distribution. The analysis of Spearman correlation test showed that there was no significant relationship between PLR value and BMD value of spine L1 – L4 ($p = 0.713$; $r = -0.036$), PLR value and BMD value of left femur neck ($p = 0.883$; $r = 0.014$), and PLR value and BMD value of left femur total ($p = 0.532$; $r = 0.061$).

RESULTS

A total of 107 women of reproductive age who met the inclusion and exclusion criteria were enrolled in this study. For each participant, routine hematological parameters and Bone Mineral Density (BMD) measurements were obtained.

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Table 1. Characteristics of study participants

Variable	Mean ± SD	Median (min–max)
Age (year)	34 ± 5,81	33 (25 – 48)
Body Weight (kg)	76,31 ± 14,93	72 (49,9 – 137,6)
Height (cm)	156,06 ± 5,19	156 (145 – 169,5)
Body Mass Index/BMI (kg/m^2)	31,41 ± 5,66	30,1 (20,20 – 53,1)
BMD		
Spine L1 – L4 (g/cm^2)	1,20 ± 0,125	1,19 (0,92 – 1,52)
Left femur Neck (g/cm^2)	0,99 ± 0,12	25,00,98 (0,74 – 1,47)
Left femur Total (g/cm^2)	1,06 ± 0,199	1,007 (0,79 – 1,98)
Score-Z BMD		
Spine L1 – L4	0,79 ± 1,04	0,70 (-1,60 – 3,40)
Left femur Neck	0,67 ± 0,98	0,50 (-1,50 – 4,60)
Left femur Total	0,80 ± 1,02	0,50 (-1,30 – 4,80)
Hemoglobin (g/dL)	13,12 ± 1,25	13,2 (8,6 – 16,1)
Platelet (10^3/µl)	319,50 ± 63,38	307 (168 – 522)
Limfosit (10^3/µl)	2,22 ± 0,66	32 (18 – 45)
PLR	155,28 ± 52,46	151,07 (66,33 – 314,57)

Note: BMD = Bone Mineral Density, PLR = Platelet-Lymphocyte Ratio

Table 1 summarizes the characteristics of the 107 included participants. Both mean ± SD and median (min–max) were presented to provide a clearer description of the data distribution and to account for potential skewness. The variables included age, BMD, hemoglobin, platelet count, lymphocyte count, and PLR, with PLR calculated as the ratio between the absolute platelet count and absolute lymphocyte count.

Table 1 presents the mean values of age, BMD, hemoglobin, platelet count, lymphocyte count, and PLR among the 107 included participants. PLR was calculated as the ratio between the absolute platelet count and absolute lymphocyte count.

The study population consisted of women of reproductive age, ranging from 25 to 48 years. The mean BMI was 31.41 kg/m², with the distribution as follows: 7 subjects (6.54%) had normal BMI, 45 (42.05%) were overweight, 31 (28.97%) had obesity class I, 18 (16.82%) had obesity class II, and 6 (5.61%) had obesity class III. Thrombocytosis was identified in 3 subjects (2.80%), while 104 (97.2%) had normal platelet counts. In terms of bone mineral density, 3 subjects (2.80%) had low BMD, and 104 subjects (97.2%) had normal BMD.

Table 2. Correlation between PLR and BMD

BMD	PLR	
	p	r
Spine L1 – L4	0,634	-0,047
Left femur Neck	0,669	-0,042
Left femur Total	0,526	-0,062

Based on the Spearman non-parametric correlation test, Table 2 shows no significant correlation between PLR and BMD at the spine L1–L4, left femur neck, or left femur total, as all p-values exceed 0.05. The correlation coefficients are negative and very weak, indicating no meaningful association between PLR and BMD in this population.

DISCUSSION

Based on the results of the Spearman correlation test, there was no significant association between PLR and BMD at the L1–L4 spine ($p = 0.634$; $r = –0.036$). This finding is consistent with a previous study by Lee et al. (2019), which reported no significant relationship between PLR and BMD. Unlike the neutrophil-to-lymphocyte ratio (NLR), which has shown significant correlations with BMD, PLR may be less sensitive because platelets are not directly involved in bone resorption pathways such as those mediated by neutrophils via the RANK/RANKL system. Thus, NLR appears to be more strongly associated with BMD than PLR[30]. However, this contrasts with a study by Akbas et al. (2016), which demonstrated that vitamin D levels decreased with increasing PLR. Nonetheless, that study did not directly examine the relationship between PLR and BMD; therefore, the findings are not directly comparable to our results[31].

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The correlation analysis between PLR and BMD at the left femoral neck also revealed no significant relationship (p = 0.669; r = –0.042). A study by Onalan et al. (2020) reported that PLR and lymphocyte counts were significantly higher in patients with osteoporosis, particularly among geriatric individuals[24]. Aging, often associated with a chronic low-grade inflammatory state termed inflammaging, contributes to the pathogenesis of various chronic diseases, including osteoporosis[32,33]. Inflammation promotes the release of proinflammatory mediators such as interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), C-reactive protein (CRP), and interleukin-6 (IL-6), which reflect tissue damage and degeneration[34] Estrogen deficiency, as a primary etiological factor in osteoporosis, enhances the release of proinflammatory cytokines, particularly TNF-α, leading to osteoclastogenesis and bone resorption. However, in premenopausal women with physiologically adequate estrogen levels, these inflammatory effects are likely suppressed[27,35]. Therefore, given that the participants in this study were premenopausal women with optimal estrogen status, the impact of systemic inflammation on BMD may not be as pronounced as in postmenopausal or elderly populations.

Similarly, no significant association was found between PLR and total left femur BMD (p = 0.526; r = –0.062). This finding differs from a study by Chen et al. (2023), which reported a significant relationship between PLR and total femur BMD in postmenopausal women. However, their study excluded participants with a history of malignancy and also accounted for confounding factors such as physical activity, diabetes, medication use, and acute infections, which were not controlled for in our analysis. These differences may explain the contrasting results and highlight the potential influence of unmeasured confounders such as physical activity levels and metabolic status in the present study.

Although the current study included many women with overweight or obese BMI, body fat distribution was not assessed, making it unclear whether fat accumulation was predominantly visceral or subcutaneous. Subcutaneous fat produces more leptin than visceral fat, which may partly explain why leptin levels are typically higher in women than in men, as both fat types are capable of expressing leptin[36]. Recent studies suggest that adipokines are associated with metabolic diseases such as obesity, insulin resistance, and diabetes, and play an important role in bone health. Leptin has been shown to locally increase bone mass and may mediate the balance between bone formation and resorption[37].

In addition to PLR, body composition and metabolic mediators such as leptin should also be considered, as they may modulate the relationship between chronic inflammation and bone health. Elevated leptin levels are associated with poor metabolic profiles and are a hallmark of obesity[38]. Leptin has proinflammatory properties and stimulates the secretion of proinflammatory cytokines[36]. The systemic inflammatory state in obesity may impair bone remodeling, disrupt bone homeostasis, and increase the risk of bone loss. Two major mechanisms underlie this process: the release of proinflammatory mediators from adipose tissue in obesity, which reduces osteoblast activity and promotes osteoclast differentiation and resorption; and the suppression of osteogenic differentiation of mesenchymal stem cells (MSCs) in favor of increased adipogenic differentiation[39].

CONCLUSION

This study found no significant correlation between the platelet-to-lymphocyte ratio (PLR) and bone mineral density (BMD) across the examined anatomical sites in women of reproductive age. These findings suggest that PLR may not serve as a reliable inflammatory marker associated with BMD in this population.

Future studies are warranted to explore the association between BMD and other systemic inflammatory markers beyond PLR in women of reproductive age. In addition, further investigation should consider stratifying subjects into more specific subgroups based on age, body composition, hormonal status, or metabolic profile to better understand the potential relationship between PLR and BMD.

ETHICAL APPROVAL

The study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Diponegoro University (No.266/EC/KEPK/FK-UNDIP/V/2024)

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization, MH; methodology, NPO and EKSL; software, EKSL; validation, NPO, EKSL, and FHN; formal analysis, NPO; investigation, NPO and FHN; resources, MH; data curation, NPO and EKSL; writing original draft preparation, NPO, MH; writing review and editing, MH and EKSL; visualization, FHN; supervision, MH; project administration, MH; funding acquisition, – (no funding)

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REFERENCES

1. Wirata RB, Istianti DW. Faktor-Faktor Yang Mempengaruhi Pemanfaatan Posbindu Kesehatan Wanita Usia Subur. Jurnal Kesehatan 2020;8:25–32. https://doi.org/10.35913/jk.v8i1.196.
2. Larijani B, Hossein-Nezhad A, Mojtahedi A, Pajouhi M, Bastanhagh MH, Soltani A, et al. Normative data of bone Mineral Density in healthy population of Tehran, Iran: A Cross sectional study. BMC Musculoskelet Disord 2005;6:38. https://doi.org/10.1186/1471-2474-6-38.
3. Ginaldi L, Di Benedetto MC, De Martinis M. Osteoporosis, inflammation and ageing. Immunity & Ageing 2005;2:14. https://doi.org/10.1186/1742-4933-2-14.
4. Torres HM, Arnold KM, Oviedo M, Westendorf JJ, Weaver SR. Inflammatory Processes Affecting Bone Health and Repair. Curr Osteoporos Rep 2023;21:842–53. https://doi.org/10.1007/s11914-023-00824-4.
5. Sponholtz TR, Zhang X, Fontes JDT, Meigs JB, Cupples LA, Kiel DP, et al. Association Between Inflammatory Biomarkers and Bone Mineral Density in a Community-Based Cohort of Men and Women. Arthritis Care Res (Hoboken) 2014;66:1233–40. https://doi.org/10.1002/acr.22270.
6. Iantomasi T, Romagnoli C, Palmini G, Donati S, Falsetti I, Miglietta F, et al. Oxidative Stress and Inflammation in Osteoporosis: Molecular Mechanisms Involved and the Relationship with microRNAs. Int J Mol Sci 2023;24:3772. https://doi.org/10.3390/ijms24043772.
7. Rotta D, Fassio A, Rossini M, Giollo A, Viapiana O, Orsolini G, et al. Osteoporosis in Inflammatory Arthritides: New Perspective on Pathogenesis and Treatment. Front Med (Lausanne) 2020;7. https://doi.org/10.3389/fmed.2020.613720.
8. Livshits G, Kalinkovich A. Targeting chronic inflammation as a potential adjuvant therapy for osteoporosis. Life Sci 2022;306:120847. https://doi.org/10.1016/j.lfs.2022.120847.
9. Börjesson AE, Lagerquist MK, Windahl SH, Ohlsson C. The role of estrogen receptor α in the regulation of bone and growth plate cartilage. Cellular and Molecular Life Sciences 2013;70:4023–37. https://doi.org/10.1007/s00018-013-1317-1.
10. Mills EG, Yang L, Nielsen MF, Kassem M, Dhillo WS, Comninos AN. The Relationship Between Bone and Reproductive Hormones Beyond Estrogens and Androgens. Endocr Rev 2021;42:691–719. https://doi.org/10.1210/endrev/bnab015.
11. Nicks KM, Fowler TW, Gaddy D. Reproductive Hormones and Bone. Curr Osteoporos Rep 2010;8:60–7. https://doi.org/10.1007/s11914-010-0014-3.
12. Streicher C, Heyny A, Andrukhova O, Haigl B, Slavic S, Schüler C, et al. Estrogen Regulates Bone Turnover by Targeting RANKL Expression in Bone Lining Cells. Sci Rep 2017;7:6460. https://doi.org/10.1038/s41598-017-06614-0.
13. Khosla S, Oursler MJ, Monroe DG. Estrogen and the skeleton. Trends in Endocrinology & Metabolism 2012;23:576–81. https://doi.org/10.1016/j.tem.2012.03.008.
14. Martini FH NJBEF. Fundamentals of Anatomy and Physiology 11th edition. Pearson 2018.
15. Kemenkes RI. Riskesdas Nasional. Badan Penelitian Dan Pengembangan Kesehatan Kementerian Kesehatan RI 2018.
16. Unni J, Garg R, Pawar R. Bone mineral density in women above 40 years. J Midlife Health

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JKD (DMJ), Volume 15, Number 6, November 2026 : 537-543

2010;1:19. https://doi.org/10.4103/0976-7800.66989.
17. Abraham A, Cohen A, Shane E. Premenopausal Bone Health. Clin Obstet Gynecol 2013;56:722–9. https://doi.org/10.1097/GRF.0b013e3182a8ae55.
18. Chen Y, Yu J, Shi L, Han S, Chen J, Sheng Z, et al. Systemic Inflammation Markers Associated with Bone Mineral Density in perimenopausal and Postmenopausal Women. J Inflamm Res 2023;Volume 16:297–309. https://doi.org/10.2147/JIR.S385220.
19. Lin X, Zuo Y, Hu H, Zhou J. Causal relationship between reproductive factors and female bone density: a univariate and multivariate mendelian randomization study. Front Genet 2024;15. https://doi.org/10.3389/fgene.2024.1393106.
20. Abdi F, Mobedi H, Bayat F, Mosaffa N, Dolatian M, Ramezani Tehrani F. The Effects of Transdermal Estrogen Delivery on Bone Mineral Density in Postmenopausal Women: A Meta-analysis. Iran J Pharm Res 2017;16:380–9.
21. Lo JC, Burnett-Bowie S-AM, Finkelstein JS. Bone and the Perimenopause. Obstet Gynecol Clin North Am 2011;38:503–17. https://doi.org/10.1016/j.ogc.2011.07.001.
22. Sözen T, Özışık L, Çalık Başaran N. An overview and management of osteoporosis. Eur J Rheumatol 2019;4:46–56. https://doi.org/10.5152/eurjrheum.2016.048.
23. Tang Y, Peng B, Liu J, Liu Z, Xia Y, Geng B. Systemic immune-inflammation index and bone mineral density in postmenopausal women: A cross-sectional study of the national health and nutrition examination survey (NHANES) 2007-2018. Front Immunol 2022;13. https://doi.org/10.3389/fimmu.2022.975400.
24. Önalan E, Gokalp Y. The relationship between bone mineral density and hematological parameters in the geriatric age group. Family Practice and Palliative Care 2020;5:1–5. https://doi.org/10.22391/fppc.634197.
25. Oliveira MA de, Moraes R, Castanha EB, Prevedello AS, Vieira Filho J, Bussolaro FA, et al. Osteoporosis Screening: Applied Methods and Technological Trends. Med Eng Phys 2022;108:103887. https://doi.org/10.1016/j.medengphy.2022.103887.
26. Shevroja E, Cafarelli FP, Guglielmi G, Hans D. DXA parameters, Trabecular Bone Score (TBS) and Bone Mineral Density (BMD), in fracture risk prediction in endocrine-mediated secondary osteoporosis. Endocrine 2021;74:20–8. https://doi.org/10.1007/s12020-021-02806-x.
27. Fulop T, Larbi A, Pawelec G, Khalil A, Cohen AA, Hirokawa K, et al. Immunology of Aging: the Birth of Inflammaging. Clin Rev Allergy Immunol 2021;64:109–22. https://doi.org/10.1007/s12016-021-08899-6.
28. Hijjawi NS, Al-Radaideh AM, Al-Fayomi KI, Nimer NA, Alabadi HA, Al-Zu'bi RM, et al. Relationship of serum leptin with some biochemical, anthropometric parameters and abdominal fat volumes as measured by magnetic resonance imaging. Diabetes & Metabolic Syndrome: Clinical Research & Reviews 2018;12:207–13. https://doi.org/10.1016/j.dsx.2017.09.004.
29. Allshouse AA, Polotsky A, Crawford S, Chen H-Y, El Khoudary SR, Santoro N. Consistent ovulation may not be enough to make women healthy when approaching menopause. Menopause 2015;22:267–74. https://doi.org/10.1097/GME.0000000000000314.
30. Lee S-H, Ryu S-Y, Park J, Shin M-H, Han M-A, Choi S-W. The Relationship of Neutrophil-Lymphocyte Ratio and Platelet-Lymphocyte Ratio with Bone Mineral Density in Korean Postmenopausal Women. Chonnam Med J 2019;55:150. https://doi.org/10.4068/cmj.2019.55.3.150.
31. Akbas EM, Gungor A, Ozcicek A, Akbas N, Askin S, Polat M. Vitamin D and inflammation: evaluation with neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio. Archives of Medical Science 2016;4:721–7. https://doi.org/10.5114/aoms.2015.50625.
32. Franceschi C, Garagnani P, Parini P, Giuliani C, Santoro A. Inflammaging: a new immune–metabolic viewpoint for age-related diseases. Nat Rev Endocrinol 2018;14:576–90. https://doi.org/10.1038/s41574-018-0059-4.

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JKD (DMJ), Volume 15, Number 6, November 2026 : 537-543

33. Franceschi C, Garagnani P, Vitale G, Capri M, Salvioli S. Inflammaging and 'Garb-aging.' Trends in Endocrinology & Metabolism 2017;28:199–212. https://doi.org/10.1016/j.tem.2016.09.005.
34. Koelman L, Pivovarova-Ramich O, Pfeiffer AFH, Grune T, Aleksandrova K. Cytokines for evaluation of chronic inflammatory status in ageing research: reliability and phenotypic characterisation. Immunity & Ageing 2019;16:11. https://doi.org/10.1186/s12979-019-0151-1.
35. Nie Y, Yan Z, Yin H, Shan L, Wang J, Wu Q. Osteosarcopenic obesity and its components—osteoporosis, sarcopenia, and obesity—are associated with blood cell count-derived inflammation indices in older Chinese people. BMC Geriatr 2022;22:532. https://doi.org/10.1186/s12877-022-03225-x.
36. Picó C, Palou M, Pomar CA, Rodríguez AM, Palou A. Leptin as a key regulator of the adipose organ. Rev Endocr Metab Disord 2022;23:13–30. https://doi.org/10.1007/s11154-021-09687-5.
37. Hou J, He C, He W, Yang M, Luo X, Li C. Obesity and Bone Health: A Complex Link. Front Cell Dev Biol 2020;8. https://doi.org/10.3389/fcell.2020.600181.
38. Fischer J, Völzke H, Kassubek J, Müller H, Kühn J, Nauck M, et al. Associations of a Panel of Adipokines with Fat Deposits and Metabolic Phenotypes in a General Population. Obesity 2020;28:1550–9. https://doi.org/10.1002/oby.22871.
39. Forte YS, Renovato-Martins M, Barja-Fidalgo C. Cellular and Molecular Mechanisms Associating Obesity to Bone Loss. Cells 2023;12:521. https://doi.org/10.3390/cells12040521.