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Osteoporosis – the silent epidemic of the 21st century

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ABSTRACT

Osteoporosis is the most prevalent bone tissue disorder worldwide. Its incidence is closely linked to the aging of the global population – a demographic shift observed since the late 19th century. The disease progresses asymptomatically, with the first clinical manifestation often being a low-energy fracture, which typically indicates an advanced stage of the condition. As a result, osteoporosis is frequently underestimated by both patients and healthcare professionals. The aim of this study was to analyze the available scientific literature indexed in databases such as PubMed, Google Scholar, and ScienceDirect. The article presents a historical overview, pathophysiology, epidemiology, diagnostic criteria, and risk factors associated with the disease. It also discusses current strategies for prevention and pharmacological treatment. Further research is essential to identify optimal diagnostic and therapeutic approaches and to elevate public awareness regarding the seriousness of osteoporosis.

Keywords: metabolic bone diseases; FRAX; FRAXplus; DEXA; low-energy fracture; postmenopausal osteoporosis; BMD.

INTRODUCTION

Osteoporosis is a metabolic disease of bone tissue. While it is most commonly associated with postmenopausal women, the aging of the global population has made it an increasingly significant public health concern [1]. The disease progresses painlessly, leading to gradual bone mass deterioration. A noticeable symptom is typically a fracture of long bones or vertebrae, which indicates an advanced stage of the condition [2]. Although references to osteoporosis date back over 250 years, epidemiological and clinical data have only been systematically collected for the past 70 years [3]. The term "osteoporosis" was likely first introduced in 1830 by the French pathologist Jean Lobstein "the Younger". He coined the term using 2 Greek words – "osteon" meaning bone, and "poros" meaning small hole. Initially, the term described bone lesions observed during autopsies and was subsequently adopted into medical literature [4, 5]. It wasn't until the 20th century that experts recognized the need to update the definition. In 1993, researchers from the WHO in Hong Kong proposed a new definition, describing osteoporosis as a systemic skeletal disease characterized by low bone mass, microarchitectural deterioration, and increased bone fragility [6, 7]. Later, in 2001, the National Osteoporosis Foundation (NOF) and the National Institutes of Health (NIH) in the United States offered their own definition, describing osteoporosis as a skeletal disorder marked by compromised bone strength, leading to an increased risk of fracture [8]. Both definitions remain in use today, although the NOF/NIH definition is considered more current [9]. Given the rising incidence of osteoporosis in both the Polish and global populations, this review aims to synthesize the latest scientific findings on the disease.

MATERIALS AND METHODS

The authors conducted a detailed analysis of the available scientific literature using the PubMed, Google Scholar, and ScienceDirect databases. To narrow the scope of the search, specific keywords were applied, including: "osteoporosis", "definition of osteoporosis", "pathophysiology of osteoporosis", "classification of osteoporosis", "epidemiology of osteoporosis", "osteoporosis risk factors", "prevention and management of osteoporosis" and "pharmacological treatment of osteoporosis". Boolean operators (AND, OR) were used to refine the search results and improve the precision of the review. The inclusion criteria focused on original research articles and review papers published in peer-reviewed scientific journals, in both English and Polish. Publications without full-text access or lacking scientific merit were excluded. Source selection was based on publication date, with preference given to articles published within the last 10 years. Initially, the search strategy identified 48,520 articles. After removing duplicates and screening titles and abstracts, 1,674 articles were selected for further analysis. Following full-text review and application of inclusion and exclusion criteria, a total of 95 studies were ultimately included in this publication (Fig. 1).

RESULTS

Pathophysiology

Understanding the changes in bone metabolism is key to grasping the pathogenesis of osteoporosis. Bone is composed of a dense outer cortical layer and an inner trabecular (spongy) structure [10]. Throughout life, bone tissue undergoes continuous remodeling – a process of replacing old bone with new. This

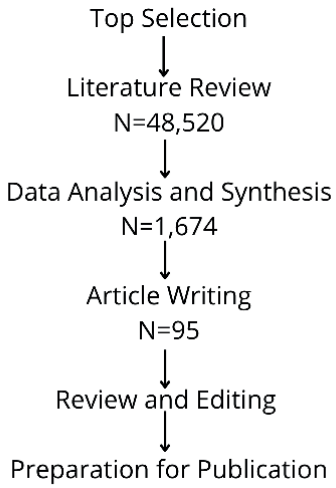

FIGURE 1. Flowchart (own elaboration)

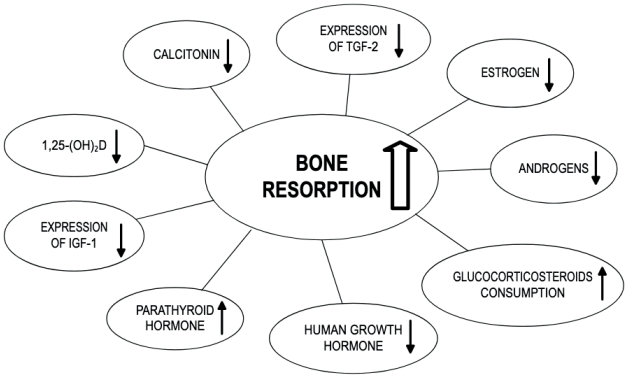

FIGURE 2. Risk factors for osteoporosis (own elaboration based on reference [11])

remodeling is regulated by vitamin D ($1.25(OH)_2D_3$), hormones, growth factors and prolonged use of glucocorticoids [11, 12] – Figure 2. Bone remodeling serves several crucial functions: adapting bone architecture to mechanical demands, repairing microdamage and removing excess mineralized (aged) bone [13]. The physiological process involves several cell types: osteoclasts (bone-resorbing), osteoblasts (bone-forming), osteocytes, chondrocytes (cartilage cells) and endothelial cells within the bone microenvironment [14]. Osteoclasts are large, multinucleated cells responsible for bone resorption, preparing the matrix for regeneration. Osteoblasts, derived from undifferentiated mesenchymal cells, are involved in bone formation and growth. Osteocytes, embedded within the bone matrix, act as mechanoreceptors, converting mechanical stimuli into biochemical signals to maintain bone homeostasis [15]. In osteoporosis, bone resorption exceeds bone formation, leading to progressive degeneration of bone tissue [16]. This results in the loss of bone mass and dissolution of hydroxyapatite crystals synthesized by osteoblasts – crystals that form the mineral scaffold essential for bone structure [17]. Consequently, the cortical bone becomes more porous and trabecular bone undergoes increased perforation, reducing the biomechanical strength of the skeleton. This compromised bone integrity predisposes individuals to fractures [18]. These are typically low-energy fractures, where minimal trauma causes a break in bone continuity – most commonly due to a fall from standing height. Typical fracture sites include vertebrae, the distal radius (Colles fracture), the proximal humerus and the proximal femur (hip fractures) [19].

Classification

The primary classification of osteoporosis is based on its etiology, dividing the condition into primary and secondary forms. Primary osteoporosis includes 2 distinct types [20]. Type I (Postmenopausal Osteoporosis): This form typically affects women over the age of 60 and is associated with a decline in estrogen levels. Estrogen plays a protective role in bone metabolism, and its reduction during menopause increases osteoclast activity, accelerating bone mass loss [21]. Type II (Senile Osteoporosis): This type is linked to the natural aging process and occurs in both sexes, usually after the age of 70. It involves a decrease in both the mass and quality of trabecular and cortical bone, significantly increasing the risk of fractures [22]. Secondary osteoporosis arises due to other medical conditions or their treatments and is more commonly observed in men. It may develop in the context of hyperparathyroidism therapy, chronic kidney disease, or conditions requiring prolonged immobilization. Medications that elevate the risk of secondary osteoporosis include glucocorticoids and antiepileptic drugs [23].

Idiopathic juvenile osteoporosis

Osteoporosis is rare in childhood. Its idiopathic juvenile form remains poorly understood and presents a diagnostic challenge for clinicians [24]. Idiopathic juvenile form typically affects children before puberty and is characterized by pain in the vertebrae (especially the lower spine), hips and feet, along with mild myopathy and mobility difficulties [25]. To date, no genetic mutations or inheritance patterns have been identified as underlying causes. Diagnosis is based on excluding a family history of pediatric osteoporosis, collagen structural defects, and other causes of bone loss. Radiological imaging confirms the presence of osteoporosis [26]. Some cases of idiopathic juvenile osteoporosis may resolve after puberty, while others may progress, leading to disability and increased skeletal fragility [27].

Epidemiology

Until recently, osteoporosis was believed to be a condition affecting only older women, particularly postmenopausal women living in Northern Europe. However, it is now well established, that the disease also affects men and advancements in diagnostic methods have revealed its global prevalence [28]. The incidence of osteoporosis varies significantly depending on geographic location. The highest number of cases is reported in Africa and Europe, while in Asia, the condition occurs more frequently than in the United States and Australia [29]. These differences are likely due to genetic factors and lower intake of calcium, vitamin D, and protein in traditional diets [30]. Age is another critical factor. The ongoing global aging process has exponentially increased the risk of osteoporotic fractures [31]. This trend is reflected in the numbers: approx. 10 mln Americans over the age of 50 currently suffer from osteoporosis and an additional

34 mln are at risk. Each year, around 1.5 mln fragility fractures are diagnosed in the USA population [32]. In Poland, osteoporosis affects approx. 1.9 mln individuals [33]. Back in 1990, projections indicated that by 2050, the global risk of hip fractures would increase by 310% in men and 240% in women. In 2010, it was estimated that 158 mln patients were at high risk of osteoporotic fractures, and due to demographic changes, this number is expected to double by 2040 [34].

Diagnostic criteria

Diagnosing osteoporosis presents a significant challenge for healthcare systems, primarily because the disease is asymptomatic. Fractures – often the first visible sign – typically indicate an advanced stage of the condition. Therefore, early detection before the occurrence of fractures is crucial [35]. The diagnostic process begins with a thorough medical history and physical examination. The history helps identify risk factors such as age, sex, body weight, physical activity level, presence of chronic diseases, and dietary habits. The physical exam may reveal overt signs of osteoporosis, such as vertebral fractures or compression deformities [36]. The gold standard for diagnosing osteoporosis is the measurement of bone mineral density (BMD) using dual-energy X-ray absorptiometry (DEXA). This test is typically performed at the proximal femur and lumbar spine [37]. The results are compared with population-based reference values and expressed as standard deviations from peak bone mass in healthy individuals of the same sex (T-score) and from the average bone density of individuals of the same age (Z-score) [38, 39]. According to the WHO, the interpretation of the T-score is as follows:

- > –1 SD – normal,
- from –1 to –2.5 SD – osteopenia,
- ≤ –2.5 SD – osteoporosis,
- ≤ –2.5 SD and 1 or more osteoporotic fractures – advanced osteoporosis [40].

To assess the 10-year risk of osteoporotic fractures, clinicians commonly use the Fracture Risk Assessment Tool (FRAX). This algorithm can be applied with or without BMD measurements, allowing risk evaluation even when densitometry is unavailable [41]. Fracture Risk Assessment Tool analyzes 7 dichotomous clinical risk factors to estimate fracture probability. Despite its utility, FRAX is not intended to serve as a standalone decision-making tool. It does not account for several variables that may significantly influence fracture risk over the next decade [42]. To address these limitations, a beta version of a new calculator – FRAXplus – was introduced in 2023. Compared to its predecessor, FRAXplus incorporates additional parameters, including: number of falls in the previous year, site of prior osteoporotic fracture, dosage of glucocorticoids used by the patient, presence and duration of type 2 diabetes, trabecular bone score (TBS), hip axis length (HAL) and discrepancy between BMD values at the hip and lumbar spine. These enhancements allow for a more precise assessment of fracture risk [43, 44].

Risk factors

Osteoporosis is a multifactorial disease. The clinician's role is to identify risk factors early and implement appropriate therapeutic strategies. The following factors are known to increase the risk of developing osteoporosis:

- female sex,
- age over 50,
- Asian ethnicity,
- coexisting conditions such as: hypogonadism, type 2 diabetes, rheumatoid arthritis, inflammatory bowel disease, primary biliary cholangitis, non-alcoholic fatty liver disease, systemic lupus erythematosus or asthma,
- impaired renal function,
- history of fragility fractures or parental history of such fractures,
- elevated levels of sex hormone-binding globulin,
- low body mass index,
- sedentary lifestyle,
- low dietary intake of vitamin d and calcium,
- excessive alcohol consumption,
- smoking,
- use of medications such as: thiazolidinediones, sodium-glucose co-transporter 2 inhibitors, glucocorticoids other immunosuppressive agents [45, 46, 47, 48, 49, 50, 51, 52].

Prevention

Osteoporosis prevention is primarily based on lifestyle modifications and the implementation of appropriate dietary recommendations [53]. Physical activity plays a crucial role in preventing this condition. According to the piezoelectric theory, mechanical pressure generates electrical potential differences within bone tissue, thereby stimulating osteogenesis [54]. For this reason, physical exercises should be performed in an upright position rather than a horizontal one. The intensity, frequency, and duration of exercise should be tailored to the individual. A review study by Daly et al. questioned the effectiveness of walking and aerobic exercise in improving or maintaining bone mass. Instead, the authors emphasized the benefits of progressive resistance training for bone health [55]. These findings are supported by Italian researchers. In their review, Benedetti et al. concluded, that strength training more effectively increases bone mineral density, compared to non-weight-bearing activities. They noted that athletes participating in jumping sports, such as basketball, soccer, and volleyball tend to have higher BMD than those engaged in swimming or cycling [56]. One of the most notable clinical programs evaluating the impact of resistance training on osteoporosis prevention is the LIFTMOR study, conducted in 2015 [57]. The study involved 101 postmenopausal women over the age of 58, divided into an intervention group and a control group. Over an 8-month period, participants engaged in prescribed activities twice a week, with each session lasting 30 min. The intervention group performed supervised resistance exercises at a clinic under the guidance of a physiotherapist, while the control group practiced balance and mobility exercises independently at home. After 8 months, DEXA scans were performed to assess changes in bone metabolism. In the intervention group, lumbar spine and femoral neck BMD increased by 2.9% and 0.3%, respectively. In contrast, the control group experienced a decrease in BMD of 1.2% and 2.0% at the same sites. These

results highlight the significant impact of resistance training on bone remodeling. Moreover, resistance training offers an additional advantage over traditional preventive methods: it enhances muscle mass and strength, thereby reducing the risk of falls – one of the leading causes of osteoporotic fractures [58].

A properly balanced diet is a crucial element in the prevention of osteoporosis. Its primary goal is to ensure adequate intake of calcium, vitamin D, and protein [59]. These nutrients influence peak bone mass, which is typically achieved the ages of 20–30. Therefore, it is essential that a balanced diet, along with possible supplementation, begins in early childhood. The higher the peak cortical bone density at the completion of skeletal development, the lower the risk of fractures and the onset of osteoporosis [60]. Calcium and vitamin D, in the form of calcium phosphate, contribute to the formation of hydroxyapatite crystals, which are responsible for bone strength. The most important dietary sources of calcium include dairy products, fish (especially sardines), legumes, nuts, and certain fruits and vegetables [61]. According to the National Institute of Public Health (PZH, 2024), the recommended daily intake of calcium is 1000 mg for women under 50 years of age and 1200 mg for older women [62, 63]. Vitamin D enhances calcium absorption in the human body. It is found in breakfast cereals, fortified milk, saltwater fish (such as tuna, mackerel, and salmon), and cod liver oil. It is also synthesized in the keratinocytes of the epidermal basal layer under UV radiation. According to guidelines from The Bone Health and Osteoporosis Foundation, serum levels of 25(OH)D3 should be maintained at 30 ng/mL [64, 65]. Polish recommendations suggest daily vitamin D intake for individuals aged 19–65 should range 800–2000 IU. For pregnant women, the recommended dose is 2000 IU/day, and for individuals over 75 years of age, it ranges 2000–4000 IU/day [62]. Protein intake positively affects bone mineral density. This has been confirmed for animal-derived protein, particularly from dairy products, but not for plant-based protein [65]. The recommended daily intake for men and women over 19 years of age is 0.9 g/kg of body weight. For pregnant and breastfeeding women, the recommended intake is 1.20 g/kg and 1.45 g/kg of body weight per day, respectively [66]. In recent years, increasing evidence suggests that phosphorus, magnesium, zinc, selenium, manganese, copper, and vitamins K2, C, A, and B may also play a role in osteoporosis prevention. However, current data are insufficient to confirm these findings, and further research is needed in this area [67, 68].

Preventive strategies in Poland

In the Polish population, educational and informational programs concerning osteoporosis prevention have been gaining increasing significance. One such initiative is the health policy program implemented between 2023–2025, entitled Niezłomne Warszawianki. This program has been co-financed by the Ministry of Health and the Capital City of Warsaw. Its primary objective is the early detection of osteoporosis among women over the age of 65 residing in Warsaw. Within the program, participants may voluntarily undergo bone densitometry, a 10-year fracture risk assessment using the FRAX algorithm, as well as a medical consultation. During these visits, participants are also provided with practical guidance on preventive strategies that can be implemented to reduce the progression of the disease. The program is delivered in selected healthcare facilities in Warsaw, under the scientific and methodological supervision of the National Institute of Geriatrics, Rheumatology, and Rehabilitation [69]. In addition, a program for the early detection of osteoporosis is being implemented in the Mazovian Voivodeship during 2024–2026. Its aim is to educate both patients and healthcare professionals in order to reduce the incidence of osteoporotic fractures. The program is targeted at women over 65 years of age, as well as women aged 40–64 who meet at least one of the medical criteria defined in the program. It also includes men over the age of 75. The program provides bone densitometry, FRAX risk assessment, and specialist consultations [70]. These initiatives are designed not only to enhance public awareness of osteoporosis but also to enable systematic screening and early diagnosis among individuals at increased risk of developing the disease.

Pharmacological treatment

Osteoporosis is an incurable disease. Its treatment focuses on protecting patients who are at high or very high risk of fragility fractures. Pharmacotherapy plays a crucial role in this context [71]. It is undertaken in cases where osteoporosis is diagnosed by means of bone densitometry and/or the FRAX algorithm, accompanied by the occurrence of a low-energy fracture, which constitutes an absolute indication for initiating treatment. The decision regarding the choice of specific pharmacological agents is made on the basis of registration guidelines and clinical trial results, taking into account the indications, potential adverse effects, and expected therapeutic benefits for the individual patient [72]. Pharmacotherapy is based on the use of antiresorptive agents, anabolic drugs, or a combination of both, with the aim of slowing disease progression, reducing fracture risk and lowering mortality [73]. The most commonly used agents in Europe for the treatment of osteoporosis include bisphosphonates, raloxifene, parathyroid hormone derivatives and denosumab [74].

Bisphosphonates

Bisphosphonates are considered first-line drugs in osteoporosis therapy. These include alendronate, risedronate, ibandronate and zoledronic acid. Their mechanism of action involves inhibition of farnesyl diphosphate synthase in the cholesterol biosynthesis pathway and binding to hydroxyapatite crystals. This leads to reduced bone resorption by osteoclasts and promotes the formation of new bone tissue [75]. All bisphosphonates increase bone mineral density. Alendronate, risedronate and zoledronic acid reduce the risk of vertebral fractures, hip fractures and other non-vertebral fractures. In contrast, ibandronate does not reduce the risk of fractures at the proximal femur [76]. These drugs are available in oral formulations (alendronate, risedronate) and intravenous forms (ibandronate, zoledronic acid). They differ in their potency to inhibit osteoclasts and their affinity for hydroxyapatite. It is worth emphasizing that their effects

do not cease immediately after discontinuation, as they bind strongly to bone minerals and may remain active in bone tissue and circulation for years [77]. This property also allows for the implementation of so-called "drug holidays", meaning a temporary discontinuation of bisphosphonate therapy. To date, there is insufficient evidence to fully support the rationale and effectiveness of such an approach, as well as the efficacy of resuming treatment after the break. Therefore, the duration and timing of a drug holiday should be tailored to the individual patient, taking into account their specific risk profile and the potential benefits of this strategy [78]. Like all drug classes, bisphosphonates are associated with certain adverse effects, depending on the route of administration. Short-term use of oral formulations may cause gastrointestinal discomfort, particularly in patients with esophageal narrowing or reflux disease. Other complications include musculoskeletal pain and atrial fibrillation. Hypocalcemia and flu-like symptoms are more commonly linked to intravenous administration [79]. Long-term use of bisphosphonates has been associated with rare but serious adverse events such as osteonecrosis of the jaw (ONJ) and atypical femoral fractures (AFF). These events occur relatively infrequently, and the overall benefits of treatment outweigh the risks over the long term [80].

Denosumab

It has been demonstrated that the introduction of denosumab following long-term treatment with oral bisphosphonates increases BMD at the proximal femur and lumbar spine [81]. Denosumab is a human monoclonal antibody that acts as an inhibitor of the receptor activator of nuclear factor κB ligand (RANKL). By blocking the interaction between RANKL and its receptor RANK, it prevents the differentiation of osteoclasts from precursor cells and promotes apoptosis of mature osteoclasts. Additionally, this compound facilitates the filling of resorption cavities by osteoblasts without affecting overall bone turnover [82]. Discontinuation of denosumab therapy is associated with a decline in BMD and an increased risk of osteoporotic fractures, particularly in the vertebrae. Therefore, the benefits and risks of such an approach should be carefully considered in each individual case [83]. In addition to ONJ and AFF, the most common adverse effects of denosumab include back and joint pain, skin rash and hypocalcemia – especially in patients with severe renal impairment. There are reports indicating that the drug may trigger severe anaphylactic reactions and DRESS syndrome, in which case immediate discontinuation of therapy should be considered [84].

Parathyroid hormone derivatives

A study conducted by Leder et al. suggests that monotherapy with teriparatide yields better outcomes than combination therapy with teriparatide and denosumab. Although both therapeutic approaches increase BMD at measured sites, the gain is greater with teriparatide alone [85]. Teriparatide is a recombinant fragment of parathyroid hormone, consisting of the first 34 N-terminal amino acids. It is a potent osteoanabolic agent [86]. It binds with high affinity to receptors located on osteoblasts and renal tubular cells, thereby stimulating osteoblasts to produce new bone tissue. This leads to the formation of the so-called "anabolic window", during which bone formation markers increase more rapidly than bone resorption markers. These changes are believed to correlate with improvements in bone quality and bone mineral density [87]. Currently, its indications are limited to patients with severe osteoporosis who have experienced one or more vertebral or non-vertebral fractures. The drug is generally well tolerated by patients [88].

Selective estrogen receptor modulator

Raloxifene is a selective estrogen receptor modulator. It acts as an agonist on estrogen receptors located in bone tissue. It improves BMD and bone strength, while inhibiting excessive bone resorption in osteoporosis [89] This agent reduces the risk of vertebral fractures by approx. 30–50%, but does not affect the risk of hip or non-vertebral fractures. It is recommended for postmenopausal osteoporosis and for reducing the risk of invasive breast cancer [90]. Unlike the aforementioned drugs, raloxifene appears to be well tolerated in patients with end-stage renal disease [91, 92, 93, 94]. The most common adverse effects include hot flashes and muscle cramps. It is contraindicated in immobilized patients, due to an increased risk of thromboembolic events [95].

CONCLUSIONS

Osteoporosis remains a significant challenge for healthcare systems. As a multifactorial disease, it is difficult to eliminate exposure to all potential risk factors. For most of its course, the condition is asymptomatic, meaning that patients are often unaware of the threat until a fracture occurs. In recent years, there has been notable progress in the development of tools for fracture risk assessment, as well as an expansion in the range of substances used for prevention and treatment. These advancements offer the potential for earlier diagnosis and improved quality of life for affected individuals. It is also essential to implement effective educational strategies – both among at-risk populations and healthcare professionals – to raise awareness and understanding of this condition.

REFERENCES

1. Lane NE. Epidemiology, etiology, and diagnosis of osteoporosis. Am J Obstet Gynecol 2006;194(2):S3-11. doi: 10.1016/j.ajog.2005.08.047.
2. Janiszewska M, Kulik T, Dziedzic M, Żołnierczuk-Kieliszek D, Barańska A. Osteoporoza jako problem społeczny – patogeneza, objawy i czynniki ryzyka osteoporozy pomenopauzalnej. Probl Hig Epidemiol 2015:96(1):106-14.
3. Curate F. Osteoporosis and paleopathology: a review. J Anthropol Sci 2014;92:119-46. doi: 10.4436/JASS.92003.
4. Schapira D, Schapira C. Osteoporosis: the evolution of a scientific term. Osteoporos Int 1992;2(4):164-7. doi: 10.1007/BF01623921.
5. Bijlsma AY, Meskers CG, Westendorp RG, Maier AB. Chronology of age-related disease definitions: osteoporosis and sarcopenia. Ageing Res Rev 2012;11(2):320-4. doi: 10.1016/j.arr.2012.01.001.
6. Consensus development conference: Diagnosis, prophylaxis and treatment of osteoporosis. Am J Med 1993;94:646-50. doi: 10.1016/0002-9343(93)90218-e.
7. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Technical Report Series 843. World Health

Organization; 1994. https://iris.who.int/bitstream/handle/10665/39142/WHO_TRS_843_eng.pdf (24.08.2025).
8. NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Therapy. Osteoporosis prevention, diagnosis, and therapy. JAMA 2001;285(6);785-94. doi: 10.1001/jama.285.6.785.
9. Rajska-Neumann A. Osteoporoza – definicja, epidemiologia, rozpoznawanie, leczenie i profilaktyka. Farmacja Współczesna 2008;1:47-53.
10. Liang B, Burley G, Lin S, Shi YC. Osteoporosis pathogenesis and treatment: existing and emerging avenues. Cell Mol Biol Lett 2022;27:72. doi: 10.1186/s11658-022-00371-3.
11. Siddiqui JA, Partridge NC. Physiological Bone Remodeling: Systemic Regulation and Growth Factor Involvement. Physiology (Bethesda) 2016;31(3):233-45. doi: 10.1152/physiol.00061.2014.
12. Bone Remodeling. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK499863/ (24.08.2025).
13. Hadjidakis DJ, Androulakis II. Bone Remodeling. Ann NY Acad Sci 2006;1092:385-96. doi: 10.1196/annals.1365.035.
14. Ansari N, Sims NA. The Cells of Bone and Their Interactions. Handb Exp Pharmacol 2020;262:1-25. doi: 10.1007/164_2019_343.
15. Liu L, Luo P, Wen P, Xu P. The role of magnesium in the pathogenesis of osteoporosis. Front Endocrinol 2024;15:1-11. doi: 10.3389/fendo.2024.1406248.
16. Al Saedi A, Stupka N, Duque G. Pathogenesis of Osteoporosis. Handb Exp Pharmacol 2020;262:353-67. doi: 10.1007/164_2020_358.
17. Smektała A, Dobosz A. Osteoporoza – patofizjologia, objawy, profilaktyka i leczenie. Farm Pol 2020;76 (6):344-52. doi: 10.32383/farmpol/125762.
18. Barszczewski K, Karaś R, Kępczyńska A, Górecki K, Lepich T. Osteoporoza starcza – poważny problem starzejącego się społeczeństwa. Gerontol Pol 2024;32:105-11.
19. Malachowski K, Materkowski M. Osteoporotic Fractures During and after Pandemic Time – Always Required Treatment. Approach of the Physician, Comments of the Professor. Ortop Traumatol Rehabil 2020;22(4):281-5. doi: 10.5604/01.3001.0014.4151.
20. Amarnath SS, Kumar V, Das SL. Classification of Osteoporosis. Indian J Orthop 2023;57(1):49-54. doi: 10.1007/s43465-023-01058-3.
21. Stromsnes K, Fajardo CM, Soto-Rodriguez S, Kajander ERU, Lupu RI, Pozo-Rodriguez M, et al. Osteoporosis: Causes, Mechanisms, Treatment and Prevention: Role of Dietary Compounds. Pharmaceuticals 2024;17:1697. doi: 10.3390/ph17121697.
22. Dhruvee P, Bhagawati S. Decoding osteoporosis: Understanding the disease, exploring current and new therapies and emerging targets. J Orthop Rep 2025;4(4):100472. doi: 10.1016/j.jorep.2024.100472.
23. Osteoporosis. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK441901/ (24.08.2025).
24. Shimazaki S, Sato J. Idiopathic Juvenile Osteoporosis: A Case Report and Literature Review. Cureus 2024;16(9):e68361. doi: 10.7759/cureus.68361.
25. Wade E, Mulholland K, Shaw I, Cundy T, Robertson S. Idiopathic juvenile osteoporosis-a polygenic disorder? JBMR Plus 2024;8(9):ziae099. doi: 10.1093/jbmrpl/ziae099.
26. Imerci A, Canbek U, Haghari S, Sürer L, Kocak M. Idiopathic juvenile osteoporosis: A case report and review of the literature. Int J Surg Case Rep 2015;9:127-9. doi: 10.1016/j.ijscr.2015.02.043.
27. Akhvlediani G, Nakaidze N, Dzodzuashvili E, Gabidzashvili N, Dopidze E, Mikaberidze A, et al. Idiopathic Juvenile Osteoporosis Diagnosed in Adulthood: The First Documented Case in Georgia. Cureus 2025;17(5):e83707. doi: 10.7759/cureus.83707.
28. The Epidemiology and Pathogenesis of Osteoporosis. Endotext. 2020. https://www.ncbi.nlm.nih.gov/books/NBK279134/ (24.08.2025).
29. Salari N, Ghasemi H, Mohammadi L, Behzadi MH, Rabieenia E, Shohaimi S, et al. The global prevalence of osteoporosis in the world: a comprehensive systematic review and meta-analysis. J Orthop Surg Res 2021;16:609. doi: 10.1186/s13018-021-02772-0.
30. Wang L, Yu W, Yin X, Cui L, Tang S, Jiang N, et al. Prevalence of Osteoporosis and Fracture in China: The China Osteoporosis Prevalence Study. JAMA Netw Open 2021;4(8):e2121106. doi: 10.1001/jamanetworkopen.2021.21106.
31. Epidemiology of osteoporosis. Balloon Kyphoplasty. Springer; 2008. https://link.springer.com/chapter/10.1007/978-3-211-74221-1_1 (24.08.2025).
32. Clynes MA, Harvey NC, Curtis EM, Fuggle NR, Dennison EM, Cooper C. The epidemiology of osteoporosis. Br Med Bull 2020;133(1):105-17. doi: 10.1093/bmb/ldaa005.
33. Program polityki zdrowotnej profilaktyki i wczesnego wykrywania osteoporozy dla mieszkańców województwa wielkopolskiego na lata 2025–2026. Załącznik do Uchwały Zarządu Województwa Wielkopolskiego nr 1285/2025 z 29 stycznia 2025 r. https://bip.umww.pl/artykuly/2831947pliki/20250129123537_programprofilaktykiiwczesnegowykrywaniaosteoporozy.pdf (24.08.2025).
34. Fragility fractures. International Osteoporosis Foundation; 2025. https://www.osteoporosis.foundation/health-professionals/fragility-fractures/epidemiology (24.08.2025).
35. Kuczera W, Pluskiewicz W. Osteoporoza starcza. Ann Acad Med Siles 2012;66(6):57-65.
36. Marcinowska-Suchowierska E, Czerwiński E, Badurski J, Walicka M, Tałałaj M. Osteoporoza – diagnostyka i terapia u osób starszych. Postępy Nauk Medycznych 2011;24(5):410-23.
37. Głuszko P, Sewerynek E, Misiorowski W, Konstantynowicz J, Marcinowska-Suchowierska E, Blicharski T, et al. Zalecenia postępowania diagnostycznego i leczniczego w osteoporozie w Polsce. Aktualizacja 2022. Endokrynol Pol 2023;74(1). doi: 10.5603/EP.a2023.0012.
38. Ćwirlej-Sozańska A. Ocena ryzyka złamań osteoporotycznych. Prz Med Uniw Rzesz Inst Leków 2013;4:515-24.
39. Pusz-Sapa A, Sobczyk J. Gęstość tkanki kostnej młodych osób w zależności od aktywności fizycznej. Inżynier i Fizyk Medyczny 2022;11(3):253-9.
40. Brown JP, Fortier M, Frame H, Lalonde A, Papaioannou A, Senikas V, et al. Osteoporosis Guidelines Committee. Canadian Consensus Conference on osteoporosis, 2006 update. J Obstet Gynaecol Can 2006;28(2):95-112. doi: 10.1016/s1701-2163(16)32087-4.
41. Schini M, Johansson H, Harvey NC, Lorentzon M, Kanis JA, McCloskey EV. An overview of the use of the fracture risk assessment tool (FRAX) in osteoporosis. J Endocrinol Invest 2024;47(3):501-11. doi: 10.1007/s40618-023-02219-9.
42. Leszczyński P, Korkosz M, Pawlak-Buś K, Bykowska M, Gruszecka K, Górska A, et al. Diagnostyka i leczenie osteoporozy – zalecenia Polskiego Towarzystwa Reumatologicznego 2015. Forum Reumatol 2015;1(1):12-24.
43. Zerikly R, Demetriou EW. Use of Fracture Risk Assessment Tool in clinical practice and Fracture Risk Assessment Tool future directions. Womens Health 2024:17455057241231387. doi: 10.1177/17455057241231387.
44. Discover the advantages of FRAXplus. 2025. https://fraxplus.org/pl/frax-plus (24.08.2025).
45. Kim JY, Kong GM. Age- and Gender-Related Femoral Bowing Analysis in the Korean Population and Features for Clinical Applications. Med Kaunas 2024;60(12):1930. doi: 10.3390/medicina60121930.
46. Bijelic R, Milicevic S, Balaban J. Risk Factors for Osteoporosis in Postmenopausal Women. Med Arch 2017;71(1):25-8. doi: 10.5455/medarh.2017.71.25-28.
47. Ji W, Pan B, Chen X, Lao Z, Yang W, Qian Y. Mendelian randomization studies of risk and protective factors for osteoporosis: a systematic review and meta-analysis. Front Endocrinol 2025;15:1486188. doi: 10.3389/fendo.2024.1486188.
48. Pandey D, Basnet S, Pradhananga S, Shrestha S, Rijal B, Neupane A, et al. Prevalence and Risk Factors of Osteoporosis among Postmenopausal Women Visiting a District Hospital of Nepal: An Observational Study. J Nepal Med Assoc 2024;62(279):744-9. doi: 10.31729/jnma.8800.
49. Özmen S, Kurt S, Timur HT, Yavuz O, Kula H, Demir AY, et al. Prevalence and Risk Factors of Osteoporosis: A Cross-Sectional Study in a Tertiary Center. Med Kaunas 2024;60(12):2109. doi: 10.3390/medicina60122109.
50. Su W, Jia H, Yang L, Zhang J, Wei Z, Tsikwa P, et al. Risk factors for osteoporosis in elderly patients with type 2 diabetes: A protocol for systematic review and meta-analysis. PLoS One 2025;20(2):e0319602. doi: 10.1371/journal.pone.0319602.
51. Florez H, Carrasco JL, Barberá M, Hernández-Rodríguez J, Muxi A, Mocritcaia A. et al. Risk factors for glucocorticoid induced osteoporosis in young adults. Front Endocrinol 2025;16:1528962. doi: 10.3389/fendo.2025.1528962.
52. Kuang C, Shang J, Ma M, Huang S, Yan B, Zhong Y, et al. Risk factors and clinical prediction models for osteoporosis in pre-dialysis chronic kidney disease patients. Ren Fail 2024;46(2):2361802. doi: 10.1080/0886022X.2024.2361802.
53. Kanis JA, Cooper C, Rizzoli R, Reginster JY. Scientific Advisory Board of the European Society for Clinical and Economic Aspects of Osteoporosis and Osteoarthritis (ESCEO) and the Committees of Scientific Advisors and National Societies of the International Osteoporosis Foundation

(IOF). Executive summary of the European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Calcif Tissue Int 2019;104(3):235-8. doi: 10.1007/s00223-018-00512-x.
54. Weber-Rajek M, Ciechanowska K. Physical treatment in postmenopausal osteoporosis – A review of research. Ginekol Poloznictwo 2015;35(1):53-8.
55. Daly RM, Dalla Via J, Duckham RL, Fraser SF, Helge EW. Exercise for the prevention of osteoporosis in postmenopausal women: an evidence-based guide to the optimal prescription. Braz J Phys Ther 2019;23(2):170-80. doi: 10.1016/j.bjpt.2018.11.011.
56. Benedetti MG, Furlini G, Zati A, Letizia Mauro G. The Effectiveness of Physical Exercise on Bone Density in Osteoporotic Patients. Biomed Res Int 2018;2018:4840531. doi: 10.1155/2018/4840531.
57. Watson SL, Weeks BK, Weis LJ, Harding AT, Horan SA, Beck BR. High-Intensity Resistance and Impact Training Improves Bone Mineral Density and Physical Function in Postmenopausal Women With Osteopenia and Osteoporosis: The LIFTMOR Randomized Controlled Trial. J Bone Miner Res 2018;33(2):211-20. doi: 10.1002/jbmr.3284.
58. Layne JE, Nelson ME. The effects of progressive resistance training on bone density: a review. Med Sci Sports Exercise 1999;31(1):25-30.
59. Rizzoli R, Biver E, Brennan-Speranza TC. Nutritional intake and bone health. Lancet Diabetes Endocrinol 2021;9(9):606-21. doi: 10.1016/S2213-8587(21)00119-4.
60. Mędrela-Kuder E, Bis H. Porównanie odżywiana kobiet z prawidłową i obniżoną masą kostną. Bromat Chem Toksykol 2011;44(3);415-9.
61. Muñoz-Garach A, García-Fontana B, Muñoz-Torres M. Nutrients and Dietary Patterns Related to Osteoporosis. Nutrients 2020;12(7):1986. doi: 10.3390/nu12071986.
62. Suplementy diety, czy potrzebujesz? Instytut Żywności i Żywienia. 2025. https://ncez.pzh.gov.pl/wp-content/uploads/2021/03/broszura_suplementy.pdf (24.08.2025).
63. Normy żywienia dla populacji Polski. Narodowy Instytut Zdrowia Publicznego PZH – Państwowy Instytut Badawczy; 2025. https://ncez.pzh.gov.pl/wp-content/uploads/2025/02/Normy-zywienia-dla-populacji-Polski-07-2025-2.pdf (24.08.2025).
64. LeBoff MS, Greenspan SL, Insogna KL, Lewiecki EM, Saag KG, Singer AJ, et al. The clinician's guide to prevention and treatment of osteoporosis. Osteoporos Int 2022;33(10):2049-102. doi: 10.1007/s00198-021-05900-y.
65. Gawryś J, Gawryś K, Doroszko A. Sezonowe zmiany stężenia witaminy D i ich wpływ na jej suplementację. Kosmetologia Estetyczna 2016;5(6):561-6.
66. Rizzoli R, Chevalley T. Nutrition and Osteoporosis Prevention. Curr Osteoporos Rep 2024;22(6):515-22. doi: 10.1007/s11914-024-00892-0.
67. Martiniakova M, Babikova M, Mondockova V, Blahova J, Kovacova V, Omelka R. The Role of Macronutrients, Micronutrients and Flavonoid Polyphenols in the Prevention and Treatment of Osteoporosis. Nutrients 2022;14(3):523. doi: 10.3390/nu14030523.
68. Papadopoulou SK, Papadimitriou K, Voulgaridou G, Georgaki E, Tsotidou E, Zantidou O, et al. Exercise and Nutrition Impact on Osteoporosis and Sarcopenia-The Incidence of Osteosarcopenia: A Narrative Review. Nutrients 2021;13(12):4499. doi: 10.3390/nu13124499.
69. Niezłomne warszawianki. Profilaktyka osteoporozy wśród seniorek. Zdrowie. Miasto Stołeczne Warszawa. 2025. https://zdrowie.um.warszawa.pl/niezlomne-warszawianki (1.10.2025).
70. Program polityki zdrowotnej w zakresie profilaktyki i wczesnego wykrywania osteoporozy wśród mieszkańców województwa mazowieckiego na lata 2024–2026. Urząd Marszałkowski Województwa Mazowieckiego w Warszawie. 2025. https://mazovia.pl/pl/bip/zalatw-sprawe/zdrowie-i-polityka-spoleczna/priorytetowe-dzialania-w-obszarze-zdrowia/programy-polityki-zdrowotnej/program-polityki-zdrowotnej-w-zakresie-profilaktyki-i-wczesnego-wykrywania-osteoporozy-wsrod-mieszkancow-wojewodztwa-mazowieckiego-na-lata-2024-2026.html (1.10.2025).
71. Brown JP. Long-Term Treatment of Postmenopausal Osteoporosis. Endocrinol Metab 2021;36(3):544-52. doi: 10.3803/EnM.2021.301.
72. Głuszko P. Osteoporoza. Reumatologia 2016;(1):124-8. doi: 10.5114/reum.2016.60014.
73. Vandenbroucke A, Luyten FP, Flamaing J, Gielen E. Pharmacological treatment of osteoporosis in the oldest old. Clin Interv Aging 2017;12:1065-77. doi: 10.2147/CIA.S131023.
74. Kanis JA, Cooper C, Rizzoli R, Reginster JY. Scientific Advisory Board of the European Society for Clinical and Economic Aspects of Osteoporosis (ESCEO) and the Committees of Scientific Advisors and National Societies of the International Osteoporosis Foundation (IOF). European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int 2019;30(1):3-44. doi: 10.1007/s00198-018-4704-5.
75. Amin U, McPartland A, O'Sullivan M, Silke C. An overview of the management of osteoporosis in the aging female population. Womens Health 2023;19:17455057231176655. doi: 10.1177/17455057231176655.
76. Bisphosphonate. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK470248/ (24.08.2025).
77. Hayes KN, Winter EM, Cadarette SM, Burden AM. Duration of Bisphosphonate Drug Holidays in Osteoporosis Patients: A Narrative Review of the Evidence and Considerations for Decision-Making. J Clin Med 2021;10(5):1140. doi: 10.3390/jcm10051140.
78. Diab DL, Watts NB. Bisphosphonate drug holiday: who, when and how long. Ther Adv Musculoskelet Dis 2013;5(3):107-11. doi: 10.1177/1759720X13477714.
79. Gehrke B, Alves Coelho MC, Brasil d'Alva C, Madeira M. Long-term consequences of osteoporosis therapy with bisphosphonates. Arch Endocrinol Metab 2023;68:e220334. doi: 10.20945/2359-4292-2022-0334.
80. Eastell R, Rosen CJ, Black DM, Cheung AM, Murad MH, Shoback D. Pharmacological Management of Osteoporosis in Postmenopausal Women: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab 2019;104(5):1595-622. doi: 10.1210/jc.2019-00221.
81. 8Miller PD, Pannacciulli N, Brown JP, Czerwinski E, Nedergaard BS, Bolognese MA, et al. Denosumab or Zoledronic Acid in Postmenopausal Women With Osteoporosis Previously Treated With Oral Bisphosphonates. J Clin Endocrinol Metab 2016;101(8):3163-70. doi: 10.1210/jc.2016-1801.
82. Kendler DL, Cosman F, Stad RK, Ferrari S. Denosumab in the Treatment of Osteoporosis: 10 Years Later: A Narrative Review. Adv Ther 2022;39(1):58-74. doi: 10.1007/s12325-021-01936-y.
83. McClung MR, Wagman RB, Miller PD, Wang A, Lewiecki EM. Observations following discontinuation of long-term denosumab therapy. Osteoporos Int 2017;28(5):1723-32. doi: 10.1007/s00198-017-3919-1.
84. Denosumab. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK535388/ (24.08.2025).
85. Leder BZ, Tsai JN, Uihlein AV, Wallace PM, Lee H, Neer RM, et al. Denosumab and teriparatide transitions in postmenopausal osteoporosis (the DATA-Switch study): extension of a randomised controlled trial. Lancet 2015;386(9999):1147-55. doi: 10.1016/S0140-6736(15)61120-5.
86. Teriparatide. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK559248/ (24.08.2025).
87. Blick SKA, Dhillon S, Keam SJ. Teriparatide. Drugs 2008;68:2709-37. doi: 10.2165/0003495-200868180-00012.
88. Hauser B, Alonso N, Riches PL. Review of Current Real-World Experience with Teriparatide as Treatment of Osteoporosis in Different Patient Groups. J Clin Med 2021;10(7):1403. doi: 10.3390/jcm10071403.
89. Raloxifene. StatPearls Publishing; 2025. https://www.ncbi.nlm.nih.gov/books/NBK544233/ (24.08.2025).
90. Hansdóttir H. Raloxifene for older women: a review of the literature. Clin Interv Aging 2008;3(1):45-50. doi: 10.2147/cia.s224.
91. Ma HY, Chen S, Lu LL, Gong W, Zhang AH. Raloxifene in the Treatment of Osteoporosis in Postmenopausal Women with End-Stage Renal Disease: A Systematic Review and Meta-Analysis. Horm Metab Res 2021;53(11):730-7. doi: 10.1055/a-1655-4362.
92. Khairallah P, Nickolas TL. Effectiveness of Pharmacological Interventions for Treatment of Osteoporosis in Patients With CKD 3-5D: No Clear Choices. Am J Kidney Dis 2022;80(6):797-800. doi: 10.1053/j.ajkd.2022.08.002.
93. Nitta K, Yajima A, Tsuchiya K. Management of Osteoporosis in Chronic Kidney Disease. Intern Med 2017;56(24):3271-6. doi: 10.2169/internalmedicine.8618-16.
94. Haghverdi F, Farbodara T, Mortaji S, Soltani P, Saidi N. Effect of raloxifene on parathyroid hormone in osteopenic and osteoporotic postmenopausal women with chronic kidney disease stage 5. Iran J Kidney Dis 2014;8(6):461-6.
95. Kukowka A, Pluskota M, Arciszewski M, Sroczyński T. Obecna farmakoterapia osteoporozy oraz jej przyszłość. Farm Pol 2021;77(11):683-9. doi: 10.32383/farmpol/145401.