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Review

The role of vitamin D, calcium, and phosphorus in the primary prevention of osteoporosis

Maksymilian Dobosz[1], Bartłomiej Palmowski[2], Wiktoria Ficoń[2], Beata Całyniuk[3]

[1]Students' Scientific Club, Department of Human Nutrition, Faculty of Public Health in Bytom, Medical University of Silesia, Katowice, Poland

[2]Doctoral School, Medical University of Silesia in Katowice, Poland

[3]Department of Human Nutrition, Faculty of Public Health in Bytom, Medical University of Silesia, Katowice, Poland

Address for correspondence:
Maksymilian Dobosz
Studenckie Koło Naukowe
Zakład Żywienia Człowieka
ul. Jordana 19, 41-808 Zabrze
tel. +48 32 275 51 95
e-mail: maksymiliandobosz582@gmail.com

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ABSTRACT

Most current scientific research focuses primarily on pharmacological methods of treating osteoporosis. Few researchers pay attention to the need to systematize preventive measures. One such measure is an appropriate diet. The intake of adequate nutrients can significantly improve bone mineral density and reduce the risk of developing the disease. The aim of this article was to analyze the available scientific literature on the efficacy and safety of the use of specific nutrients in patients with osteoporosis. This paper is a narrative review based on electronic databases (PubMed, ScienceDirect, Google Scholar), covering mainly literature from 2016–2026. A total of 87 carefully selected publications were included in the detailed analysis. The greatest body of evidence points to the potential positive effects of combined vitamin D and calcium supplementation. When calcium supplements are used as monotherapy, the possible negative effects of these compounds on the cardiovascular system should be taken into account. Excessive dietary phosphorus intake may have adverse effects on the skeletal system.

KEYWORDS

osteoporosis, vitamin D, calcium, phosphorus, nutrition, prophylaxis

Introduction

Osteoporosis is a metabolic disease of bone tissue associated with a decrease in bone mineral density (BMD) and increased susceptibility to fractures [1]. The underlying cause of this condition is a disruption of bone tissue remodeling. Under physiological conditions, bone formation and resorption processes remain in balance. As a result of fluctuations in intracellular signaling pathways, the activity of bone-resorbing cells, known as osteoclasts, which are responsible for bone resorption, increases, while the activity of osteoblasts, which are responsible for bone formation, decreases. This leads to a predominance of resorption processes over bone renewal processes [2]. The first visible symptom of progressive disease is a low-energy fracture involving the wrist, shoulder, pelvis, or hip, or a compression fracture of a vertebral body. Due to the substantial financial resources required to treat the consequences of osteoporosis, appropriate preventive strategies should be implemented in patients at risk [3]. Such measures primarily include changes to existing lifestyle habits. In addition to giving up stimulants and engaging in regular resistance training, patients should follow an appropriate diet and use supplementation [4]. Diet is the foundation of primary, secondary, and tertiary prevention of osteoporosis. It affects bone metabolism, microstructure, and bone mineral density, while also coordinating proper muscle function. Following an appropriate diet contributes to increased bone tissue strength and determines its reduced susceptibility to fractures [5]. The aim of this review article is to synthesize

scientific evidence from the years 2016–2026 and update knowledge on key nutrients, with particular emphasis on calcium, vitamin D, and phosphorus in the prevention and treatment of osteoporosis. The added value of this paper lies in compiling current intake recommendations, discussing official guidelines issued by scientific societies, and systematizing existing reports on the effects of these nutrients on bone tissue function.

Methods

The authors conducted a review of the scientific literature available in electronic databases, with particular emphasis on PubMed, ScienceDirect, and Google Scholar. To narrow the scope of the analysis, Boolean combinations using the AND and OR operators were employed: "diet" OR "diet pattern" OR "food patterns" OR "nutrition" AND "osteoporosis", along with individual keywords incorporating Medical Subject Headings (MeSH), including: "vitamin D", "role of vitamin D in bone health", "sources of vitamin D in food", "vitamin D deficiency", "calcium and vitamin D", "hypervitaminosis D", "excess of vitamin D", "calcium", "role of calcium in bone health", "sources of calcium in food", "hypocalcemia", "calcium deficiency", "excess of calcium", "hypercalcemia", "calcium and cardiovascular disease", "phosphorus", "role of phosphorus in bone health", "sources of phosphorus in food", "hypophosphatemia", "phosphorus deficiency", "hyperphosphatemia", and "excess of phosphorus". The inclusion criteria comprised review articles, systematic reviews, meta-analyses, original research papers, as well as a position statement issued by a non-governmental organization (The International Osteoporosis Foundation) and position statements issued by scientific societies, published in Polish and English. Additionally, animal studies were included in the review. The review covered articles published after 2016. In justified cases, i.e., when no recent guidelines regulating the recommended intake of a given nutrient were available, earlier key source publications or official guidelines issued by recognized scientific societies were included, such as the National Osteoporosis Guideline Group (NOGG), The Endocrine Society, The Italian Society for Osteoporosis, Mineral Metabolism and Bone Diseases (SIOMMMS), and the European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO). The literature selection process proceeded as follows: search results obtained using the specified keywords in the aforementioned scientific databases were identified, and duplicates were removed; the thematic relevance of abstracts to the aim of this article was assessed, and papers that did not meet the inclusion criteria were excluded; subsequently, a critical assessment of all remaining articles was conducted in terms of their substantive and methodological value. Ultimately, 87 references were selected for the preparation of this review article. The most recent search of the electronic databases was conducted on October 22, 2026. The sources used enabled a comprehensive presentation of the current state of

knowledge regarding the nutrients discussed, as well as the prevention and treatment of osteoporosis.

The basic definitions, pathophysiology, classification, epidemiology, diagnostic criteria, risk factors, and non-pharmacological and pharmacological treatment methods for osteoporosis fall outside the scope of this review and were presented in a previous publication by the authors [6].

Prevention

Primary prevention of osteoporosis focuses on limiting modifiable risk factors. These include, among others, alcohol consumption, smoking, excessive salt intake, physical activity, and vitamin D and calcium deficiencies in plasma [7]. An important element of prevention is diet, whose components, such as vitamin D, calcium, and phosphorus, are involved in osteogenesis. Randomized controlled trials have shown that an adequate combined intake of calcium and vitamin D reduces the risk of bone fractures [8]. Particular attention should be paid to the reduced absorption of calcium through the gastrointestinal tract in older adults. Additionally, older adults are at risk of vitamin D deficiency. In postmenopausal women, the effect of dietary calcium intake on reducing the risk of fractures remains not fully understood. Physical activity tailored to the individual needs of patients is also important. Particularly important components include functional training, resistance training, and balance exercises [5,9].

Vitamin D

Vitamin D belongs to the group of fat-soluble vitamins. In nature, it occurs mainly in two forms: ergocalciferol (vitamin D_2) and cholecalciferol (vitamin D_3). Vitamin D_2 is supplied to the body exogenously through food, whereas vitamin D_3 is predominantly synthesized endogenously in the skin [10]. Both forms of vitamin D can be supplemented through the diet. The main sources of cholecalciferol are fish, meat, eggs, and dairy products, whereas ergocalciferol is found in mushrooms and yeast exposed to UV radiation (Table I) [11]. Foods of animal origin are characterized by greater bioavailability than those of plant origin. Additionally, the bioavailability of vitamin D_3 is higher than that of vitamin D_2 [12].

Table I. Vitamin D content in selected food products (based on [11])

Product	Content (µg/100 g of product)
Beef liver	1.7
Mackerel	16
Egg yolk	5.6
Milk powder	20
Butter	1.2
Maitake mushrooms	30
Fortified margarine	7.5
Dark chocolate	4.0
Soy drink	0.8
Tomato	<0.1

Vitamin D plays a key role in bone remodeling in the body. By participating in the regulation of calcium-phosphate homeostasis, it increases the absorption of calcium and phosphates in the intestines. Normal concentrations of these macronutrients ensure proper mineralization and quality of bone tissue [13,14]. Factors influencing optimal vitamin D levels in the human body include age, sex, and physiological status. The initiation of supplementation should be correlated with the patient's biochemical parameters [15].

Vitamin D deficiency is a global public health problem. In children, it prevents the achievement of peak bone mass and genetically determined growth, contributing to the development of rickets in this age group. In adults, vitamin D deficiency leads to osteomalacia, osteoporosis, and the consequences of secondary hyperparathyroidism [16,17]. Factors contributing to reduced vitamin D intake and synthesis in the body include age, sex, genetics, skin pigmentation, exposure to UVB radiation, geographical latitude, season, use of sunscreen, diet, socioeconomic status, and cultural and religious practices [18,19]. The International Osteoporosis Foundation defines vitamin D deficiency as a 25(OH)D concentration below 25 nmol/L [20]. In its latest guidelines, The Endocrine Society advises against using specific values to define vitamin D deficiency, adequacy, and excess [21]. In contrast, the national dietary recommendations for the Polish population consider a 25(OH)D concentration of ≤20 ng/ml to indicate vitamin D deficiency in the body [22]. The discrepancy between the threshold values for vitamin D deficiency results from the different purposes for which they were established. Attention should be paid to the use of different measurement units. The threshold established by the International Osteoporosis Foundation is

half the value considered indicative of deficiency in the recommendations for the Polish population. The standard established by the International Osteoporosis Foundation focuses on reducing the risk of developing osteoporosis, preventing possible fractures, and maintaining proper bone health. Polish recommendations are population-based and preventive in nature; therefore, their primary objective is to maintain adequate vitamin D concentrations in the body and ensure proper calcium-phosphate homeostasis across the entire population.

Treatment of vitamin D deficiency involves increasing its dietary intake and initiating supplementation with oral vitamin preparations [23]. Cholecalciferol is the first-line treatment. It is assumed that a dose of 1000 IU/day allows the desired 25(OH)D concentration of ≥30 ng/ml to 50 ng/ml (75–125 nmol/L) to be achieved in most adults in the general population, although the response to supplementation shows significant interindividual variability. Calcifediol (25(OH)D) is a second-line treatment for patients in at-risk groups. Concurrent calcium intake is very important during treatment with cholecalciferol or calcifediol [24]. Providing approximately 1000 mg/day of this element through the diet or 800–1000 mg/day through calcium supplements allows measurable benefits from the treatment to be achieved [25]. The results of a review presented by Italian experts indicate that simultaneous supplementation with vitamin D and calcium reduces the risk of low-energy fractures [26]. However, the scientific evidence presented appears to be inconsistent with the position of the European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO) Working Group. The society emphasizes that the evidence supporting the role of vitamin D, calcium, and combined preparations in reducing the risk of hip fractures, fractures of other types, minimizing the risk of falls, and preventing the development of osteoarthritis remains inconclusive and should be a focus of future research [27]. Nevertheless, many authors emphasize the importance of vitamin D, calcium, and especially combined preparations in the primary prevention of osteoporosis and during treatment with anabolic and antiresorptive drugs [4,28,29].

According to the 2011 Endocrine Society guidelines, excess vitamin D in the human body is defined as a serum 25(OH)D concentration above 50 ng/ml (125 nmol/L) [30]. The 2023 Polish update of the guidelines defines a 25(OH)D concentration above 100 ng/ml (250 nmol/L) as a threshold associated with an increased risk of vitamin D toxicity [24]. The discrepancy between the cited values results from changing expert positions in response to newly published studies on the safety of high vitamin D concentrations in the body. Incorrect vitamin D dosing, excessive consumption of vitamin D-fortified foods, errors made by physicians when prescribing vitamin D preparations, improper dispensing of prescriptions by pharmacists, and excessive use of tanning beds are the

main causes of elevated vitamin D concentrations in the body [31]. The symptoms of hypervitaminosis D are associated with subsequent hypercalcemia. They are often nonspecific and pose diagnostic difficulties. Chronic misuse of vitamin D preparations predisposes individuals to neurological, gastrointestinal, and renal symptoms, including acute kidney failure and kidney stones [32]. Treatment of the toxic effects of hypervitaminosis requires limiting vitamin D and calcium supplementation and exposure to UVB radiation. Intravenous administration of isotonic fluids, loop diuretics, and glucocorticoids should be considered, and hemodialysis should be initiated if no alternative treatment methods are available [33].

Calcium

Calcium is an element essential for maintaining proper bone health. The only natural source of calcium for humans is the diet. Foods with a high calcium content include dairy products and certain canned fish products, such as sardines in oil. Many food products are also fortified with this macronutrient (Table II) [34,35]. In most cases, the diet meets the body's calcium requirements. In the presence of persistent deficiencies, supplementation with calcium preparations is necessary. The most popular of these include calcium citrate and calcium carbonate, with the former characterized by better bioavailability [36]. Total daily calcium intake includes calcium supplied through food and supplements. The diet should be the primary means of providing this macronutrient. The requirement for this element depends, among other factors, on sex, age, and physiological status. Particular attention should be paid to periods of growth in children and adolescents, pregnant and breastfeeding women, and older adults. Pharmacological supplementation should be used when it is not possible to meet the recommended requirements through diet. Additional calcium preparations should serve to supplement the missing amount of calcium to the level recommended for a given group [37,38].

Table II. Calcium content in selected food products (based on [35])

Product	Content (mg/100 g of product)
Skimmed milk	122
Coconut milk	27
Plain yogurt	138
Feta cheese	450
Egg	54
Muesli	42
Apple	5
Almonds	250
Tofu	105
Canned tuna	28

99% of calcium reserves in the body are found in the bones, mainly in the form of hydroxyapatite crystals. This element, like vitamin D, is involved in the process of bone tissue mineralization, ensuring the maintenance of the structural integrity and mechanical strength of the skeleton [39]. In addition to its role in bone remodeling, calcium is responsible for regulating muscle contraction, nerve conduction, and cell proliferation and differentiation [40]. Insufficient daily dietary calcium intake is a problem in both high-income and low-income countries. Chronic deficiency of this macronutrient in the body predisposes children to the development of rickets and older adults to osteopenia and osteoporosis, increasing the risk of low-energy fractures [41]. Hypocalcemia may result from iatrogenic or autoimmune hypoparathyroidism, chronic kidney disease, acute pancreatitis, prematurity, pharmacological treatment, as well as inadequate intake and disturbances in vitamin D metabolism [42,43]. It is defined as a serum divalent calcium cation (Ca^{2+}) concentration below 8.8 mg/dl (2.2 mmol/l or 4.4 mEq/l) [44]. The treatment of hypocalcemia depends on its nature. In severe cases, intravenous administration of 1 ampoule of calcium gluconate (90 mg of calcium in 10 ml of solution) over a 10-minute infusion is recommended. In contrast, the aim of long-term therapy is to replenish the element's deficiency to achieve a concentration of 8.0–9.0 mg/dl [45]. Appropriate calcium supplementation through the diet may reduce the risk of hypocalcemia. According to the 2011 Endocrine Society guidelines and the 2024 dietary recommendations for the Polish population, daily intake of this macronutrient should range from 700 to 1300 mg/day [22,30]. However, higher values, above 1000 mg/day, are preferred in the 2026 document issued by the Polish Society of Rheumatology [46]. The indicated differences in recommended calcium intake result from the different target

populations. The dietary recommendations for the Polish population and the Endocrine Society guidelines are intended for healthy individuals. In contrast, the recommendations of the Polish Society of Rheumatology concern patients with musculoskeletal disorders. The value above 1000 mg/day is intended to reduce the risk of fractures and maintain proper skeletal mineralization.

The use of excessively high doses of vitamin D, dairy products containing calcium carbonate, prolonged immobilization, the use of certain anti-osteoporosis medications, primary or secondary adrenal insufficiency, and primary hyperparathyroidism are factors that may predispose individuals to the development of hypercalcemia [47]. Hypercalcemia, or excess calcium in the body, is a condition in which the serum concentration of this element exceeds 10.5 mg/dl. Depending on its severity, it can be classified as mild hypercalcemia, moderate hypercalcemia, and hypercalcemic crisis. Symptoms resulting from electrolyte disturbances most commonly occur when calcium concentrations exceed 12 mg/dl [48]. The most common symptoms of hypercalcemia include depressed mood, musculoskeletal pain, constipation, polydipsia and polyuria, intellectual impairment, as well as shortening of the QT interval, which may result in cardiac arrest [49]. Given that this condition may pose a potential threat to life, appropriate therapeutic strategies must be implemented. The treatment of mild hypercalcemia of choice is the intravenous administration of 0.9% normal saline at a rate of 200–300 ml/h. In moderate and severe cases, pamidronate or zoledronic acid should be added to ensure adequate hydration. Glucocorticoids and denosumab have been approved as treatment strategies for hypercalcemia associated with malignancy. In contrast, hemodialysis should be used to rapidly reduce calcium concentrations in the body [50].

Potential cardiovascular risk associated with the use of calcium preparations

The randomized trials most frequently cited in meta-analyses are inconclusive in their assessment of the impact of calcium supplementation on cardiovascular risk [51,52,53,54,55]. In the analysis by Bolland et al. [51], conducted among 1471 healthy postmenopausal women, myocardial infarction occurred more than twice as frequently in the group receiving calcium citrate. Similar results were obtained in the Multi-Ethnic Study of Atherosclerosis (MESA) [52]. During a 10-year follow-up of 2742 participants, a 22% increase in the risk of coronary artery atherosclerosis was documented among patients taking calcium supplements. Furthermore, an article published in the Journal of the American Heart Association demonstrated that calcium supplementation is associated with an increased risk of recurrent myocardial infarction, stroke, recurrence of coronary artery disease, and subsequent hospitalization for cardiovascular causes [53]. Interestingly, no statistically significant risk was observed with combined vitamin D and calcium

supplementation [53,54,55,56]. Additionally, only in the 5-year study by Prince et al. [57] was the administration of 600 mg of calcium carbonate twice daily not associated with a significant increase in cardiovascular risk compared with the placebo group. A summary comparison of the discussed publications is presented below (Table III).

Table III. Comparison of the cited scientific articles

Study	Conclusion
Bolland et al. [51]	The risk of myocardial infarction was more than twice as high among individuals taking calcium citrate.
Anderson et al. [52]	Calcium supplementation was associated with a 22% increase in the risk of coronary artery atherosclerosis.
Zhang et al. [53]	Calcium supplementation is associated with an increased risk of subsequent cardiovascular events.
Zhang et al. [53] Hsia et al. [54] Larsen et al. [55] Cauley et al. [56]	No statistically significant increase in risk was observed with concurrent calcium and vitamin D supplementation.
Prince et al. [57]	The use of 600 mg of calcium carbonate twice daily was not associated with a significantly higher cardiovascular risk.

Phosphorus

In recent years, an increase in daily dietary phosphorus intake has been observed [58,59]. This trend also applies to Europe [60]. When analyzing the impact of phosphorus intake on bone mineral metabolism, it is important to distinguish between its chemical forms due to differences in their absorption by the human body [61]. This element occurs in food in two forms: organic and inorganic [62]. The inorganic form is found mainly in food additives in the form of phosphoric acid (E338), phosphate salts (E339-341, E343, E450-451), and polyphosphate (E452), and is more widely distributed in food [63]. It does not require digestion by digestive enzymes, resulting in rapid dissociation in the acidic environment of the stomach. Absorption from the gastrointestinal tract occurs rapidly, with total absorption reaching 80–100%. Naturally occurring organic phosphates in food are absorbed from the gastrointestinal tract at a rate of 40–60%. Their lower bioavailability is due to the presence of phosphorus in plant products in the form of phytates, which human digestive enzymes are unable to break down [64,65,66]. Bioavailability is also affected by the degree of activation of vitamin D receptors in the gastrointestinal tract, which

influences both phosphorus and calcium, as well as the presence of compounds such as carbonate, acetate, and citrate anions [67,68]. Phosphorus present in animal-derived products is bound to organic molecules, resulting in greater bioavailability than in plant-derived products, but considerably lower bioavailability than that of inorganic phosphorus [69]. Foods with a high phosphorus content include dairy products, meat, eggs, whole grains, nuts, and legumes [63]. Selected foods containing high amounts of organic phosphorus are presented in Table IV.

Table IV. Organic phosphorus content in selected food products (based on [63])

Product	Content (mg/100 g of product)
Blue poppy seeds	1022
Skimmed milk powder	1010
Parmesan cheese	810
Feta cheese	360
Egg yolk	590
Milk chocolate	302
Fresh cod fillet	145
Smoked mackerel	240
Peanuts	385
Toast bread	100

Phosphorus, like calcium and vitamin D, is involved in the process of bone tissue mineralization. Approximately 85% of the body's phosphorus stores are stored in bones in the form of hydroxyapatite crystals. In addition, this macronutrient is a component of biological membranes and high-energy compounds, and is also the main intracellular anion in the body [70]. Phosphorus deficiency in the body is relatively rare due to its substantial dietary intake. Impaired phosphate absorption in the small intestine, excessive movement of phosphorus into cells, and abnormal renal reabsorption of this macronutrient are conditions that predispose individuals to hypophosphatemia [71]. Hypophosphatemia occurs when the serum phosphate concentration is ≤2.5 mg/dL. Chronic hypophosphatemia contributes to the development of rickets, osteomalacia, and valgus deformity of the lower limbs [72]. The development of these disorders and hypophosphatemia can be limited by ensuring an adequate dietary intake of phosphorus. In a 2015 document published by the European Food Safety Authority (EFSA), a group of experts highlighted significant gaps in the available published data regarding the possibility of establishing a Recommended Dietary Allowance (RDA) for phosphorus [73]. The Agency recommends

correcting phosphorus deficiencies in the body depending on the molar calcium-to-phosphorus ratio, which should be 1.4:1. Furthermore, in this publication, EFSA established an Adequate Intake (AI) for phosphorus, which should be 550 mg/day in patients over 18 years of age [73]. The RDA for this age group was established in 1997 by the Institute of Medicine (IOM). The IOM Committee recommended a daily phosphorus intake of 700 mg [74]. This value is also currently recommended by the dietary guidelines for the Polish population [22].

Previous analyses have not explained how dietary phosphorus intake affects bone tissue. The results of the NHANES study, conducted between 2005 and 2010, indicate that a dietary phosphorus intake above the recommended daily allowance, provided that calcium intake is adequate, may have a positive effect on bones [75]. In both white men and women aged 20–99 years, higher phosphorus intake was associated with increased femoral Bone Mineral Content (BMC) and Bone Mineral Density (BMD), as well as a 45% reduction in the risk of developing osteoporosis. An increase in BMC in both sexes was also reported by Itkonen et al. [76]. However, this effect was observed at sex-specific anatomical sites. Finnish researchers also documented that increased dietary phosphorus intake in women was correlated with reduced P1NP and CTX concentrations, leading to decreased bone turnover. A subsequent NHANES analysis conducted between 2015 and 2018, involving 5589 participants, showed that higher serum phosphorus concentrations in white women were associated with lower pelvic BMD and an increased risk of fractures [77]. Restricting dietary phosphorus intake appears to be more beneficial than increasing it. In a study by Peng et al. [78] involving 30 patients undergoing hemodialysis, reducing dietary phosphorus intake to ≤800 mg was associated with a 0.9% increase in BAP, a 2.8% increase in P1NP, and a 1.7% increase in TRACP-5b. Thus, a low-phosphorus diet led to accelerated bone turnover. Similar conclusions were drawn from an animal study. In a 20-week analysis by Arnst et al. [79] involving 40 male C57BL/6J mice, it was demonstrated that a low-phosphorus diet (LPD) increased the BV/TV ratio, trabecular number, and trabecular thickness in the third lumbar vertebra, as well as cortical thickness and cortical area in the femur. This suggests that LPD improves the microarchitecture of mouse bone tissue.

When discussing individual population-based and clinical studies, reference was occasionally made to the results of experimental studies using animal models and studies conducted in specific patient groups, such as individuals with chronic kidney disease (CKD) requiring hemodialysis. These data should be interpreted with particular caution due to significant limitations in their comparability with the general population.

Excessive dietary phosphorus intake predisposes individuals to a condition known as hyperphosphatemia. It is observed in patients with impaired renal phosphorus excretion, resulting in the formation of insoluble calcium salts [80]. It is diagnosed when the serum phosphorus concentration exceeds 4.5 mg/dL [81]. Chronic hyperphosphatemia leads to the development of numerous adverse conditions in the body. It causes remodeling of vascular smooth muscle cells, impairing their ability to relax and increasing the risk of atherosclerosis. Furthermore, it induces changes in the kidneys, promoting the development of chronic kidney disease [82]. Hyperphosphatemia also contributes to an increased mortality rate among affected individuals [83]. Reduced renal phosphorus excretion in patients with chronic kidney disease stimulates the synthesis of parathyroid hormone (PTH) and fibroblast growth factor 23 (FGF-23), which are involved in the development of chronic kidney disease–mineral and bone disorder (CKD-MBD) [84]. In view of the above, appropriate treatment regimens should be implemented as soon as possible. In patients with normal kidney function, excess phosphorus in the body can be eliminated through the infusion of saline and the administration of diuretics. Another strategy involves supplementation with phosphate-binding compounds or medications that block phosphorus transporters in the intestines [85]. In contrast, hemodialysis or peritoneal dialysis is indicated in individuals with impaired kidney function [86]. Phosphorus supplementation should be individually tailored to the patient's current health status, comorbidities, and latest laboratory test results.

Limitations of the study

The publications included in the review differ in terms of the methodologies used, the study populations with respect to age, sex, and health status, as well as the duration of supplementation with individual components and their intake. The heterogeneity of the studies therefore prevents direct comparison of the results obtained in individual studies and the formulation of universal conclusions. No standardized tools were used in this study to formally assess the methodological quality of the cited studies or the risk of bias. The narrative nature of the review, based on the intentional selection of the most important source papers and scientific society guidelines, entails a risk of author subjectivity in the selection and interpretation of data.

Summary

In summary, the available scientific evidence regarding the benefits of vitamin D, calcium, and phosphorus supplementation in the primary prevention of osteoporosis remains highly limited. The greatest body of evidence points to the potential positive effects of combined vitamin D and calcium supplementation. When calcium preparations are used as monotherapy, the possible

adverse effects of these compounds on the cardiovascular system should be taken into account. Excessive dietary phosphorus intake may have negative effects on the skeletal system. Although data in this area have been confirmed in both human and animal models, some publications suggest that a high-phosphate diet has a positive effect on bone mineral density. Establishing appropriate preventive regimens based on the aforementioned nutrients should be a public health priority, as effective osteoporosis prevention may significantly reduce the financial expenditure required to treat its complications.

The recommended intake of vitamin D, calcium, and phosphorus for individual population groups living in Poland, as established by the National Institute of Public Health NIH - National Research Institute, is presented in the table below (Table V) [22]. The reference values for vitamin D were established at the level of Adequate Intake (AI), whereas those for calcium and phosphorus were established based on the Recommended Dietary Allowance (RDA). The reference values for infants aged 6–11 months have been deliberately omitted from the table due to the specific characteristics of this age group and the fact that breast milk or infant formula remains the primary source of nutrients.

Table V. Recommended intake of vitamin D, calcium, and phosphorus in selected population groups (based on [22])

Group/age	Vitamin D (µg of cholecalciferol/person/day)	Calcium (mg/day)	Phosphorus (mg/day)
Children			
1-3 years	15	700	460
4-6 years	15	1000	500
7-9 years	15	1000	600
Boys			
10-12 years	15	1300	1250
13-15 years	15	1300	1250
16-18 years	15	1300	1250
Girls			
10-12 years	15	1300	1250
13-15 years	15	1300	1250
16-18 years	15	1300	1250

Men			
19-30 years	15	1000	700
31-50 years	15	1000	700
51-65 years	15	1000	700
66-75 years	15	1200	700
> 75 years	15	1200	700
Women			
19-30 years	15	1000	700
31-50 years	15	1000	700
51-65 years	15	1200	700
66-75 years	15	1200	700
> 75 years	15	1200	700
Pregnant women			
< 19 years	15	1300	1250
≥ 19 years	15	1000	700
Breastfeeding women			
< 19 years	15	1300	1250
≥ 19 years	15	1000	700

Current scientific evidence confirms the significant role of calcium, vitamin D, and phosphorus in osteoporosis prevention. However, the strength of the conclusions that can be drawn from most available publications is limited by their methodological heterogeneity. The mechanisms regulating calcium and phosphorus homeostasis provide a key theoretical background for understanding bone loss. However, it should be noted that observations concerning severe metabolic disorders, for example in the aforementioned patients undergoing hemodialysis or those with chronic kidney disease, constitute only a mechanistic model and cannot be directly compared with the general population with normal kidney function. Recommendations regarding nutrient intake constitute an indispensable non-pharmacological component of comprehensive osteoporosis prevention; therefore, formulating clear and universal conclusions requires further well-designed, multicenter clinical trials with randomized sampling. Ensuring adequate intake of calcium, phosphorus, and vitamin D, together with a well-balanced diet, forms the foundation of primary prevention, supporting normal bone tissue metabolism and preventing compensatory bone resorption. Targeted supplementation is effective in individuals with a confirmed serum deficiency or as an adjunct to pharmacological osteoporosis treatment. Routine use of dietary supplements cannot replace an appropriately balanced diet.

Conflict of interest

The authors report there are no competing interests to declare.

Use of AI tools statement

ChatGPT was used to translate this manuscript. The entire research process, including the review of the available scientific literature and the selection of key information related to the topic, was conducted independently by the authors.

Authors' contribution

Study design – M. Dobosz, B. Całyniuk

Data collection – M. Dobosz, B. Palmowski, W. Ficoń

Manuscript preparation – M. Dobosz, B. Palmowski, W. Ficoń, B. Całyniuk

Literature research – M. Dobosz, B. Palmowski, W. Ficoń

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