NUTRITIONAL INTERVENTIONS FOR THE PREVENTION OF OSTEOPOROSIS IN POSTMENOPAUSAL WOMEN

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A Thesis Submitted to The W.A. Franke Honors College

In Partial Fulfillment of the Bachelor's Degree With Honors in

Physiology and Medical Sciences

THE UNIVERSITY OF ARIZONA

M A Y 2 0 2 5

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Abstract

Osteoporosis is a prevalent skeletal disorder affecting postmenopausal women, characterized by decreased bone mineral density (BMD) and increased fracture risk (34). This literature review examines nutritional interventions for preventing osteoporosis in postmenopausal women, focusing on key nutrients essential for bone health and the impact of overall dietary patterns. The literature review evaluates the roles of calcium, vitamin D, vitamin K, protein, magnesium, phytoestrogens, and omega-3 fatty acids in maintaining bone density and reducing fracture risk. By analyzing current research and guidelines, this paper aims to provide comprehensive insights into effective dietary strategies for osteoporosis prevention in this high-risk population.

Introduction

Osteoporosis is a chronic skeletal disorder characterized by low bone mineral density (BMD) and the structural deterioration of bone tissue, leading to an increased risk of fractures, particularly in the spine, hip, and wrist (34). Often referred to as the "silent epidemic" due to its asymptomatic progression until a fracture occurs, the prevalence of osteoporosis among postmenopausal women is alarmingly high. Osteoporosis affects over 200 million people worldwide, predominantly impacting postmenopausal women due to significant physiological changes in bone density associated with estrogen deficiency that accelerate bone resorption after menopause (42). Globally, nearly 30% of postmenopausal women are affected (26). In the United States alone, one in three women over the age of 50 is estimated to experience an osteoporosis-related fracture in her lifetime (34). These fractures can lead to chronic pain, disability, and even mortality, underscoring the urgent need for effective prevention and management strategies. For example, up to 22% of people die within the first year after experiencing a hip fracture, typically as a result of preexisting medical conditions and complications related to the fracture (17). With an aging global population, osteoporosis poses an increasing burden on healthcare systems, with the total costs of osteoporosis-related fractures projected to rise substantially in the coming decades. The high prevalence of fractures associated with osteoporosis underscores the need for targeted nutritional interventions, particularly in postmenopausal women who experience a significant decline in bone density (26).

The economic implications of osteoporosis are substantial, with the costs of osteoporosis-related fractures projected to increase significantly in the coming years. The prevalence of osteoporosis among U.S. women aged 50 years and older is projected to reach 13.6 million by 2030 in the United States, with associated healthcare costs exceeding $19 billion annually due to over two million fractures each year (26). These costs include direct medical expenses, such as hospitalizations, surgeries, and rehabilitation, as well as indirect costs, such as lost productivity and long-term care. Given the significant economic and personal burden of osteoporosis, effective prevention and management strategies are essential for reducing the impact of this disease on individuals and healthcare systems.

As pharmacological treatments are often associated with side effects, a growing body of research supports the role of nutritional and lifestyle interventions in managing osteoporosis risk and improving bone health. Nutrition has emerged as one of the most accessible and effective means of preventing and managing osteoporosis, with key nutrients such as calcium, vitamin D, protein, magnesium, vitamin K, phytoestrogens and Omega-3 fatty acids identified as critical in supporting and maintaining bone health. This review examines the role of these nutrients, focusing on dietary interventions to improve BMD and reduce fracture risk in postmenopausal women. By evaluating evidence-based nutritional interventions, this study seeks to identify strategies for maintaining or improving BMD and reducing fracture risk in populations at high risk for osteoporosis. The following literature review builds upon existing knowledge by providing a detailed examination of the role of nutrition, aligning with research emphasizing the importance of lifestyle factors in osteoporosis management (34).

Background

Definition of Osteoporosis

Osteoporosis is defined as a progressive bone disease characterized by decreased bone density and increased bone fragility, leading to a higher risk of fractures. Bones are dynamic tissue, constantly undergoing remodeling through the activities of osteoblasts (cells that form new bone) and osteoclasts (cells that resorb or break down old bone). In healthy individuals, these processes are balanced, allowing bones to maintain density and strength. However, in osteoporosis, this balance is disrupted, with bone resorption exceeding bone formation. This imbalance, often influenced by hormonal changes, age, and nutrient deficiencies, results in bones that are more porous, thinner, and structurally weaker, increasing fracture susceptibility (1). The decreased bone density in osteoporosis is characterized by a reduction in both the mineral and matrix components of bone, which contributes to diminished bone strength.

The bone remodeling process is regulated by various hormones, cytokines, and growth factors that influence the activity of osteoblasts and osteoclasts. In osteoporosis, this regulation is disrupted, leading to an imbalance in bone turnover. Factors such as estrogen deficiency, aging, and chronic inflammation can alter the expression and activity of these regulatory molecules, promoting bone resorption and inhibiting bone formation. As a result, the microarchitecture of bone deteriorates, with a loss of trabecular connectivity and cortical thinning. The resulting increase in bone fragility makes individuals with osteoporosis highly susceptible to fractures, even from minor falls or stresses. Factors beyond hormonal and cytokine influences, such as genetic predispositions and lifestyle choices, also influence the bone remodeling process, further complicating the pathophysiology of osteoporosis (1).

Osteoporosis is commonly diagnosed using Dual-Energy X-ray Absorptiometry (DEXA) scans, a non-invasive imaging technique that measures bone mineral density (BMD) at key sites, particularly the femur neck, trochanter, lumbar spine, and sometimes the distal forearm. DEXA scans calculate a T-score, which compares an individual's BMD to the average peak BMD of a healthy young adult of the same sex. According to the World Health Organization (WHO) classification, a T-score of -1.0 or above is considered normal bone density, between -1.0 and -2.5 indicates osteopenia (low bone density but not yet osteoporosis), and a T-score of -2.5 or below is diagnostic for osteoporosis. The greater the negative value, the more severe the bone density loss, with scores lower than -2.5 often associated with a higher risk of fractures. The use of DEXA scans in diagnosis provides a standardized method for evaluating bone health, facilitating early intervention and monitoring of treatment efficacy.

DEXA works by using two X-ray beams with different energy levels (33). When these beams pass through the body, soft tissue absorbs one of the beams, while the other is absorbed by the bones (33). The DEXA machine subtracts the amount of soft tissue absorption from the total absorption, isolating the bone density value (33). The procedure is quick and painless, typically requiring the patient to lie on their back while the scanner measures BMD, often focusing on the hip and spine. The radiation dose from a DEXA scan is minimal, significantly lower than that of standard chest or dental X-rays (33). This precise and low-radiation approach enables accurate measurement of bone density, making it the gold standard for osteoporosis diagnosis and an essential tool for monitoring BMD changes over time. It provides a reliable method for assessing bone density and is widely used in clinical practice to identify individuals at risk of fractures and to monitor the effectiveness of osteoporosis treatments. Monitoring BMD over time using DEXA scans allows healthcare providers to adjust treatment strategies and lifestyle recommendations to optimize bone health and minimize fracture risk (34).

Physiology of Healthy Bone vs. Osteoporotic Bone

Osteoporosis in postmenopausal women has a complex pathophysiological basis, with estrogen deficiency being the most critical factor driving bone loss (14). In healthy bone, a balanced cycle of bone resorption and formation occurs continuously, regulated by the coordinated actions of osteoclasts and osteoblasts in bone remodeling (1). Osteoclasts are responsible for resorbing old or damaged bone tissue, while osteoblasts form new bone tissue, a process that maintains bone strength and structural integrity (1). However, in osteoporosis, hormonal changes, particularly the sharp decline in estrogen after menopause, disrupt this balance, leading to an imbalance where bone resorption exceeds bone formation (14). Estrogen plays a critical role in skeletal health by binding to estrogen receptors on osteoblasts, which suppresses osteoclast formation and activity through signaling pathways that include both direct inhibition and downstream effects (28). Estrogen also promotes the production of collagen and other matrix proteins, which contribute to bone strength and resilience (28).

One of the critical mechanisms through which estrogen exerts its protective effect on bones is through the regulation of signaling molecules involved in osteoblast and osteoclast activity. Estrogen reduces the expression of receptor activator of nuclear factor kappa-B ligand (RANKL), a key cytokine that binds to receptors on osteoclast precursors and stimulates their maturation and activity (36). Estrogen also increases the expression of osteoprotegerin (OPG), a decoy receptor that binds to RANKL, preventing it from activating osteoclasts. This interplay between RANKL and OPG is essential for balancing bone resorption and formation (28). In the absence of adequate estrogen, the RANKL/OPG ratio shifts in favor of RANKL, promoting osteoclastogenesis and increased bone resorption. Research by Khosla et al. (28) demonstrated that estrogen deficiency also affects the production of cytokines, particularly pro-inflammatory interleukins (IL-1, IL-6) and tumor necrosis factor (TNF). These cytokines stimulate osteoclast formation and activity, creating an inflammatory environment that further accelerates bone loss and weakens the bone matrix microarchitecture (35). The increase in pro-inflammatory cytokines can also disrupt the differentiation and function of osteoblasts, further contributing to the imbalance in bone remodeling. The imbalance between bone resorption and formation in osteoporotic bone affects bone microarchitecture. The bone becomes more porous and brittle, resulting in an elevated risk of fracture from events that would not typically cause harm to healthy bone, like minor falls or impacts (28).

The glucagon-like peptide (GLP) system also plays a role in bone health indirectly through its impact on signaling pathways involved in bone remodeling. GLP-1, a hormone primarily involved in glucose metabolism, has been shown to influence osteoblast activity. GLP-1 receptors, found on osteoblasts, stimulate these cells to increase bone formation when activated by GLP-1 or its analogs (1). Moreover, research suggests that GLP-1 receptor activation can inhibit osteoclast activity, indirectly contributing to reduced bone resorption (28). GLP-1 receptor agonists, which are often used in diabetes management, are being investigated for their potential protective effects on bone density. GLP-1's effects on bone are thought to involve signaling pathways that reduce oxidative stress in osteoblasts and suppress RANKL expression, thereby modulating bone metabolism (35). Targeting these signaling pathways through nutritional or pharmacological interventions may offer innovative approaches to preserving bone mass in postmenopausal women.

Similarly, other signaling ligands, such as insulin-like growth factors (IGFs) and transforming growth factor-beta (TGF-β), are vital for the maintenance of bone remodeling processes. IGFs promote osteoblast proliferation and bone matrix formation, while TGF-β is crucial for bone remodeling by promoting the differentiation of osteoblasts and regulating the lifespan of osteoclasts (1). The decline in estrogen disrupts the signaling cascade involving these ligands, leading to dysregulated osteoblast and osteoclast activity and increased bone loss (28). These combined effects of estrogen deficiency, inflammatory cytokines, and disrupted signaling pathways collectively contribute to the accelerated bone loss seen in postmenopausal osteoporosis.

Prevalence in Postmenopausal Women

Postmenopausal women are disproportionately affected by osteoporosis due to the decrease in estrogen levels following menopause. Menopause is defined as the permanent cessation of menstruation, typically occurring around age 51, and is diagnosed retrospectively after 12 consecutive months without a menstrual period (42). In contrast, perimenopause refers to the transitional period leading up to menopause, characterized by hormonal fluctuations, irregular menstrual cycles, and the onset of menopausal symptoms; this phase can last several years (42).

Estrogen plays a crucial role in maintaining bone density by inhibiting osteoclast activity, thus slowing the process of bone resorption. Without sufficient estrogen, bone resorption accelerates, leading to a rapid decrease in bone mass within the first five years after menopause. Research indicates that postmenopausal women can lose as much as 10% of their bone mass within this period, contributing to the heightened fracture risk in this group (34). The accelerated bone loss during the menopausal transition is particularly pronounced in the trabecular bone, which is more metabolically active than cortical bone (36). This rapid loss of bone mass emphasizes the critical window for implementing preventive strategies.

In addition to affecting bone resorption, estrogen deficiency compromises the calcium homeostasis crucial for maintaining bone density. A decline in estrogen reduces calcium absorption efficiency in the intestines, leading to lower circulating levels of calcium, which prompts the body to extract calcium from bone stores, further depleting bone density (14). Estrogen deficiency's influence on calcium and cytokine regulation highlights the intricate hormonal network governing bone metabolism. Findings by Riggs et al. (36) suggest that estrogen deficiency is responsible for up to 90% of bone loss in postmenopausal women, reinforcing the critical nature of hormonal regulation in osteoporosis pathogenesis. This significant role of estrogen has led to the common use of hormone replacement therapy (HRT) in managing osteoporosis; however, given HRT's potential side effects, non-pharmacological interventions, such as nutrition-based strategies, are increasingly sought as safer, sustainable options. The decision to use HRT should be made in consultation with a healthcare provider, taking into account individual risk factors and preferences.

Furthermore, the prevalence of osteoporosis in postmenopausal women is influenced by various other factors, including genetics, lifestyle, and comorbidities. Family history of osteoporosis, low body weight, smoking, excessive alcohol consumption, and sedentary lifestyle are associated with an increased risk of osteoporosis. Certain medical conditions, such as rheumatoid arthritis, hyperthyroidism, and celiac disease, can also contribute to bone loss. Addressing these modifiable risk factors is crucial for preventing and managing osteoporosis in postmenopausal women. Understanding and addressing the complex range of interacting factors is critical for creating preventative strategies that are both efficient and customized to each person's unique requirements (34).

Nutrients Essential for Bone Health

Nutritional interventions have emerged as a possible solution to managing osteoporosis, with certain nutrients recognized as essential for maintaining bone density and structural integrity, such as calcium, vitamin D, magnesium, protein, vitamin K, phytoestrogens, and Omega-3 fatty acids. Calcium and vitamin D are two of the most critical nutrients for bone health, as calcium serves as the main mineral component of bone, while vitamin D facilitates calcium absorption and bone metabolism. Weaver et al. (46) emphasize that protein, magnesium, and other micronutrients also contribute to bone health, each playing a specific role in supporting bone formation, density, and strength. Ensuring adequate intake of these nutrients is especially important for individuals at higher risk of osteoporosis, such as postmenopausal women. A well-balanced diet that includes a variety of nutrient-rich foods is essential for promoting optimal bone health.

Current Guidelines and Recommendations

Health organizations such as the Institute of Medicine (IOM) and the World Health Organization (WHO) provide specific guidelines on the intake of key nutrients to support bone health. These recommendations are based on extensive research linking nutrient intake with bone mineral density and fracture prevention, especially among postmenopausal women.

Calcium: Calcium, a primary mineral in bone composition, is essential for maintaining bone mineral density (BMD). Calcium intake, however, tends to be suboptimal in many populations, particularly among older women. The IOM recommends a daily intake of 1,200 mg of calcium for women over the age of 50. This recommendation is based on studies indicating that higher calcium intake correlates with reduced fracture risk and improved bone density. A systematic review by Tang et al. (43) found that calcium supplementation significantly reduced bone loss and fracture risk, with the greatest benefits observed in those with inadequate dietary calcium.

Calcium homeostasis is the regulation of calcium levels in the blood and tissues. It is tightly controlled by several hormones, most notably parathyroid hormone (PTH), vitamin D, and calcitonin (35). When dietary calcium intake is insufficient, blood calcium levels may begin to drop. In response, the parathyroid glands secrete PTH, which acts to restore normal calcium levels by stimulating bone resorption (the breakdown of bone tissue), increasing calcium reabsorption in the kidneys, and promoting the activation of vitamin D to enhance intestinal calcium absorption (27). While these mechanisms help maintain critical blood calcium levels, chronically elevated PTH due to low dietary calcium can lead to increased bone turnover and loss of bone mineral density over time (39). This process is particularly harmful in older adults who are already at greater risk for osteoporosis and fractures. Conversely, when calcium intake is adequate, PTH secretion remains balanced and supports bone maintenance and reduces the risk of excessive bone loss. Maintaining sufficient calcium intake is not only important for bone structure itself, but also for preventing the compensatory hormonal changes that can accelerate bone loss and compromise skeletal health.

Vitamin D: In addition to calcium, vitamin D is crucial for bone health, as it enhances calcium absorption in the intestines, ensuring that adequate calcium is available for bone mineralization. Holick (21) reported that vitamin D deficiency is prevalent among older adults, particularly postmenopausal women, and is a significant contributor to osteoporosis. Clinical trials by Bischoff-Ferrari et al. (2) demonstrated that vitamin D supplementation can effectively improve bone mineral density (BMD) and reduce fracture rates in this demographic. Without sufficient vitamin D, the body cannot absorb enough calcium from dietary sources, leading to bone loss and a heightened risk of fractures.

Current guidelines recommend 800-1,000 IU of vitamin D daily for postmenopausal women, based on research showing that this level of supplementation enhances BMD and reduces fall risk among older adults (3). Natural sources of vitamin D include sunlight exposure, fatty fish, and fortified dairy products. However, lifestyle factors and geographical location can limit vitamin D synthesis in the skin. Individuals with darker skin have more melanin, which reduces vitamin D production, making them more prone to deficiency (23). Furthermore, vitamin D synthesis is influenced by lifestyle habits, location, and climate. People in the Northern Hemisphere, where sunlight exposure is lower in winter months, often struggle to meet vitamin D needs solely through sun exposure (22). Additionally, in areas with high temperatures, such as Arizona, people may spend more time indoors or apply sunscreen, which blocks UVB rays required for vitamin D synthesis (20;22). Vitamin D supplementation is often necessary to meet recommended levels, especially for individuals with limited sun exposure, darker skin, or other factors that affect vitamin D production.

Vitamin K: Vitamin K plays a vital role in the carboxylation of osteocalcin, a protein necessary for binding calcium to the bone matrix. Low vitamin K levels have been associated with an increased risk of fractures and reduced bone strength. A large cohort study conducted by Booth et al. (8) revealed that women with higher dietary vitamin K intake had a significantly lower risk of hip fractures. Vitamin K is essential for the synthesis of several proteins involved in bone metabolism, including osteocalcin and matrix GLA protein. These proteins play a crucial role in bone mineralization and the regulation of bone turnover.

Vitamin K exists in two primary forms: vitamin K1 (phylloquinone) and vitamin K2 (menaquinone). Vitamin K1 is found in green leafy vegetables, while vitamin K2 is produced by bacteria in the gut and is also found in fermented foods and animal products. Research suggests that vitamin K2 may have a more pronounced effect on bone health than vitamin K1. Current guidelines recommend 90 mcg of Vitamin K each day to maintain bone health.

Protein: Additionally, protein intake has emerged as an influential factor in osteoporosis management. Protein is essential for maintaining muscle mass and bone matrix strength, and higher dietary protein has been linked with increased BMD (7). Adequate protein intake also supports bone density by promoting collagen formation, which is essential for maintaining bone structure and strength. Research in the American Journal of Clinical Nutrition indicates that protein intake positively correlates with BMD in postmenopausal women, with recommendations suggesting 1.0-1.2 g/kg of body weight to maintain optimal bone health (12). However, the impact of protein on bone health can be modulated by calcium intake, as a high protein intake may increase urinary calcium excretion. Research by Dawson-Hughes and Harris (2002) indicated that protein intake positively affects bone health when adequate calcium is available, emphasizing the need for a balanced nutrient approach of both calcium and protein.

Magnesium: Magnesium plays a role in bone metabolism and the structural integrity of bones. Studies suggest that low magnesium intake is associated with lower BMD, particularly in postmenopausal women. The recommended daily intake is 320 mg, typically sourced from nuts, seeds, and whole grains (47). Ensuring adequate magnesium intake may contribute to better bone density and lower osteoporosis risk.

Literature Review Methodology

Literature Review Design

The literature review was conducted to comprehensively evaluate existing research on nutritional interventions for managing osteoporosis in postmenopausal women. The focus was on identifying the effects of diet, supplementation, and other modifiable lifestyle factors on bone mineral density (BMD) and fracture risk. This review also aimed to examine the efficacy of nutritional and supplementation strategies in postmenopausal osteoporosis management. To ensure a robust and systematic approach, the review adhered to specific search terms, databases, and inclusion and exclusion criteria.

Search Terms and Strategy

A broad range of search terms was used to capture the diverse aspects of nutritional interventions and their impact on osteoporosis. Terms included technical keywords such as hormone replacement therapy (HRT), vitamin D (cholecalciferol), vitamin K (phylloquinone and menaquinone), bone mineral density (BMD), osteoporosis, nutrition, diet, supplementation, and postmenopausal women. Additional terms such as DXA (dual-energy X-ray absorptiometry), Mediterranean diet, vegan diet, vegetarian diet, lactose-intolerance, animal-based diets, and adult women were employed to capture dietary patterns relevant to bone health. Boolean operators and field-specific filters, including terms like search[tiab], sources of information[tiab], and inclusion criteria[tiab], were used to refine the search within databases.

Data Sources

The primary databases used for this review included PubMed, Medline, Cochrane Libraries, Embase, CINAHL, Scopus, and Web of Science. These databases were selected for their comprehensive coverage of biomedical and clinical research, particularly studies related to nutrition, osteoporosis, and menopausal health. Keywords and Boolean search strings tailored to each database ensured precise retrieval of relevant literature. For instance, searches incorporated terms like data sources[tiab], study selection[tiab], and eligibility criteria[tiab] to refine results and eliminate irrelevant studies.

Inclusion and Exclusion Criteria

A meticulous set of inclusion and exclusion criteria was applied to ensure the relevance and quality of the selected studies. Inclusion criteria encompassed studies focusing on adult women, particularly postmenopausal women, and the relationship between nutrition, dietary patterns, supplementation, and BMD or fracture risk. Studies evaluating interventions such as HRT combined with nutritional approaches were included, provided they analyzed outcomes related to osteoporosis management. Only peer-reviewed articles published in English within the last 25 years were considered, with preference given to systematic reviews, meta-analyses, randomized controlled trials (RCTs), and cohort studies.

Exclusion criteria aimed to filter out studies with limited relevance or confounding factors. Research focusing on pediatric populations or conditions like osteomalacia, rickets, bone density issues secondary to tumors, or bone cancer were excluded. Additionally, articles addressing non-nutritional causes of osteoporosis or lacking clear outcomes related to BMD or fracture risk were omitted.

Challenges and Limitations

Despite a rigorous design, the literature review faced several challenges and limitations. One significant limitation was participant compliance with supplementation protocols, which may influence study outcomes. Many studies relied on self-reported dietary intake and adherence to interventions, potentially introducing reporting bias. Furthermore, variability in study designs, populations, and intervention protocols complicated direct comparisons across studies. Another limitation was the underrepresentation of long-term studies assessing the sustainability of dietary and supplementation strategies for osteoporosis prevention and management.

Selection Process

The selection process involved multiple stages, including title and abstract screening, full-text review, and final eligibility assessment based on inclusion and exclusion criteria. Articles were initially screened for relevance by assessing titles and abstracts against predefined keywords and search terms. Full-text articles were then reviewed to confirm alignment with the scope of the study. The final selection prioritized studies providing quantitative data on BMD, fracture incidence, or other clinically relevant outcomes. By leveraging multiple databases, applying rigorous selection criteria, and addressing limitations, this review contributes to the broader understanding of effective interventions for this pervasive condition.

Dietary Factors and Supplementation

Calcium and Vitamin D Supplementation

Calcium and vitamin D supplementation are central to osteoporosis management due to their roles in bone remodeling. Calcium forms the hydroxyapatite crystals that provide rigidity to bones, while vitamin D enhances intestinal calcium absorption and regulates serum calcium levels (30). Meta-analyses indicate that calcium and vitamin D supplementation reduces fracture risk by 20–30% in postmenopausal women, with combined interventions showing greater efficacy than either nutrient alone (13).

A 2020 systematic review by Liu et al. (30) emphasized the synergistic effects of these nutrients, noting that women receiving 1,200 mg of calcium and 800 IU of vitamin D daily demonstrated significant improvements in hip and lumbar spine BMD. Vitamin D, specifically its active form, calcitriol, facilitates calcium transport across intestinal epithelial cells by increasing the expression of calbindin-D9k and TRPV6 calcium channels (13). This mechanism ensures efficient calcium uptake to support bone mineralization.

The recommended dietary intake of 1,200 mg of calcium and 800 IU of vitamin D for postmenopausal women accounts for the increased need due to reduced estrogen levels, which impair calcium absorption (46). Over-supplementation of calcium, however, can lead to hypercalcemia and vascular calcification, emphasizing the need for individualized dosage based on dietary intake and serum calcium levels (6).

Fortified foods such as dairy products and plant-based alternatives provide practical options for achieving these nutrient targets. For instance, fortified milk delivers both calcium and vitamin D, offering a dietary strategy to maintain optimal BMD (30).

Magnesium

Magnesium plays a crucial role in bone structure, with 60% of the body's magnesium stored in bone tissue. Adequate magnesium intake is associated with higher BMD. Research has shown magnesium supplementation can significantly increase BMD in postmenopausal women (37). It influences bone remodeling by serving as a cofactor for enzymes involved in converting vitamin D into its active form, calcitriol (37). Clinical studies have demonstrated that magnesium supplementation improves BMD by enhancing bone formation and reducing osteoclast activity. Rude et al. (37) found that postmenopausal women with magnesium supplementation exhibited a significant increase in lumbar spine BMD compared to controls.

Magnesium is often recommended alongside calcium to support overall bone health. A study found that magnesium supplementation significantly increased BMD in postmenopausal women (37). Magnesium plays a crucial role in bone metabolism by regulating calcium homeostasis and influencing the activity of osteoblasts and osteoclasts. Magnesium is also involved in the activation of vitamin D, which is essential for calcium absorption and bone mineralization. Magnesium deficiency can impair vitamin D metabolism, leading to reduced calcium absorption and increased bone loss. Dietary sources of magnesium include leafy green vegetables, nuts, seeds, and whole grains. Individuals with gastrointestinal disorders or those taking certain medications may be at risk of magnesium deficiency. Addressing magnesium insufficiency may help individuals with these concerns maximize the advantages of calcium and vitamin D, in addition to encouraging general bone health (37).

Vitamin K

Vitamin K plays a vital role in the carboxylation of osteocalcin, a protein necessary for binding calcium to the bone matrix. Low vitamin K levels have been associated with an increased risk of fractures and reduced bone strength. Supports bone mineralization; positive effects on BMD noted in clinical trials (4). Vitamin K is essential for the synthesis of several proteins involved in bone metabolism, including osteocalcin and matrix GLA protein. These proteins play a crucial role in bone mineralization and the regulation of bone turnover. According to "Nutrition in the Prevention and Control of Osteoporosis", greater levels of vitamin K, C, and group B are needed to enhance general bone health.

Vitamin K, particularly menaquinone (vitamin K2), supports bone health by activating osteocalcin, a protein responsible for binding calcium to the bone matrix. This process is critical for mineralization and maintaining bone strength (31). A meta-analysis of randomized controlled trials indicates that Vitamin K2 supplementation has a positive effect on the maintenance and improvement of lumbar spine BMD in postmenopausal women, and it can also reduce the fracture incidence, serum undercarboxylated osteocalcin (uc-OC) levels and the ratio of uc-OC to carboxylated osteocalcin (cOC), thus indirectly promoting bone mineralization and increasing bone strength (31). The potential benefits of vitamin K2 supplementation in osteoporosis prevention and treatment are an area of ongoing research.

Inadequate vitamin K levels result in increased undercarboxylated osteocalcin (uc-OC), which compromises bone mineralization and increases fracture risk. A systematic review by Ma et al. (31) revealed that vitamin K2 supplementation significantly improves lumbar spine BMD and reduces serum uc-OC levels. Additionally, vitamin K2 has been linked to decreased fracture incidence, highlighting its role in promoting bone strength through enhanced mineralization. Food sources of vitamin K2 include fermented products like natto, hard cheeses, and eggs.

Protein

Adequate protein intake is essential for bone health, as proteins are integral to the collagen matrix that provides bone elasticity and strength. Protein is necessary for the synthesis of collagen, which is the main structural protein in bone. Adequate protein intake also supports the production of growth factors and hormones that regulate bone metabolism. While high-protein diets have been criticized for their acidogenic potential, studies indicate that adequate protein intake supports calcium absorption and increases insulin-like growth factor-1 (IGF-1), a hormone that promotes bone formation (46).

Balanced protein intake, especially from plant-based sources like legumes and nuts, optimizes bone health without adverse effects on calcium metabolism. Protein-rich diets also improve muscle mass, reducing fall risk and the likelihood of fractures in older women. It is important to choose a variety of protein sources to ensure adequate intake of all essential amino acids.

Phytoestrogens and Phytochemicals

Plant-based compounds, particularly phytoestrogens like isoflavones found in soy products, have been investigated for their potential benefits in maintaining bone health after menopause. Phytoestrogens mimic estrogen by binding to estrogen receptors on osteoblasts and osteoclasts. This action mitigates postmenopausal bone loss associated with reduced endogenous estrogen production. Isoflavones also exhibit antioxidant properties, reducing osteoclastic activity induced by oxidative stress (24).

Raloxifene is a selective estrogen receptor modulator (SERM) used to prevent and treat osteoporosis. A meta-analysis by Lambert et al. (29) found that soy isoflavone supplementation modestly increased BMD in postmenopausal women, particularly in those with low baseline BMD. This systematic review and meta-analysis suggests that isoflavone interventions, especially those containing genistein of at least 50 mg/day, can effectively enhance BMD in postmenopausal women (24). Phytoestrogens have estrogen-like effects on the body and may help to compensate for the decline in estrogen levels after menopause.

Dietary sources of isoflavones include soy products, such as tofu, tempeh, edamame, and soy milk. Other phytochemicals, such as flavonoids found in fruits and vegetables, have also been investigated for their potential benefits in bone health. As seen in the data from the National Health and Nutrition Examination Study from 1999-2018, they associate Hormone Preparations with Bone Mineral Density, Osteopenia and Osteoporosis in Postmenopausal Women.

Omega-3 Fatty Acids

Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have shown positive effects on bone health by reducing inflammation. These fatty acids inhibit the production of pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which stimulate osteoclastogenesis and bone resorption (10). One study indicated that omega-3 supplementation positively impacted bone health in older women (9). Omega-3 fatty acids have anti-inflammatory properties and may help to reduce bone resorption.

Recent research further supports the role of omega-3 fatty acids in bone health. Animal studies have demonstrated that higher intake of EPA and DHA can maintain bone mineral density (BMD) during aging, primarily by inhibiting inflammation-associated bone resorption and reducing osteoclastogenesis (Frontiers in Nutrition, 2019). Omega-3 supplementation has also been shown to increase anti-osteoclastogenic cytokines, such as IL-10 and IL-12, while decreasing pro-osteoclastogenic cytokines, further contributing to bone preservation (Frontiers in Nutrition, 2019). In humans, systematic reviews have found that diets high in omega-3 fatty acids are associated with increased BMD and a lower risk of osteoporosis, although more large-scale randomized controlled trials are needed to confirm these findings (38; 32). Additionally, combining omega-3 supplementation with aerobic exercise has been shown to significantly increase BMD and reduce chronic inflammation in postmenopausal women (32). Dietary sources of omega-3 fatty acids include fatty fish, such as salmon, tuna, and mackerel, as well as flaxseeds, chia seeds, and walnuts. Omega-3 supplements are also available.

Dietary Patterns

Dietary patterns are foundational to bone health, influencing not only the availability of key nutrients but also the systemic factors affecting bone metabolism. The Mediterranean diet, characterized by high intake of fruits, vegetables, whole grains, nuts, seeds, and olive oil, is renowned for its health benefits, including bone preservation. The anti-inflammatory properties of this diet stem from polyphenols in olive oil and flavonoids in fruits and vegetables, which reduce oxidative stress markers such as reactive oxygen species (ROS). ROS contribute to osteoclast activation, promoting bone resorption (40; 15). Antioxidants in this diet, including vitamins C and E, scavenge free radicals, mitigating their harmful effects on bone cells.

High dietary adherence to the Mediterranean diet correlates with a 20% reduction in fracture risk among older women, as shown in a meta-analysis by Sofi et al. (41). This risk reduction is likely mediated by the synergistic effects of bioactive compounds, which improve endothelial function and microcirculation in bone tissues, ensuring optimal nutrient delivery (15). In contrast, Western diets rich in processed foods, saturated fats, and refined sugars exacerbate inflammation, negatively impacting bone mineral density (BMD) (45).

Although poorly planned vegan diets lacking calcium, vitamin D, and protein can impair bone health, plant-based diets that include fortified foods, legumes, and nuts can provide sufficient nutrients for bone maintenance. Fortified plant milks and tofu are rich in calcium and vitamin D, while legumes and nuts contribute magnesium and protein essential for bone matrix synthesis (46). Limiting caffeine, which interferes with calcium absorption by increasing renal excretion, and phosphoric acid, a component of sodas linked to lower BMD, is also critical (46).

Mediterranean Diet: Recent studies have explored the impact of dietary patterns on osteoporosis, with evidence supporting the Mediterranean diet as a beneficial model. This diet is rich in fruits, vegetables, whole grains, lean proteins, and healthy fats, providing a comprehensive array of nutrients essential for bone health. A study by Haring et al. (18) found that adherence to the Mediterranean diet was associated with a higher BMD and a reduced risk of fractures in postmenopausal women. The anti-inflammatory and antioxidant properties of this diet are believed to contribute to its protective effects on bone health, reducing the inflammatory cytokines that drive bone resorption. The Mediterranean diet is also rich in calcium, vitamin D, and other nutrients that are essential for bone health.

DASH Diet: Furthermore, the Dietary Approaches to Stop Hypertension (DASH) diet has shown potential for bone health due to its nutrient-rich profile and positive effects on bone mineral density (44). This diet, which emphasizes fruit, vegetables, lean protein, and low-fat dairy, offers a balanced intake of bone-supportive nutrients, suggesting its value as a dietary pattern for osteoporosis prevention. Additionally, anti-inflammatory diets rich in foods like berries, fatty fish, and leafy greens have been linked to higher BMD, suggesting that reducing systemic inflammation can benefit bone health. Limiting caffeine and phosphoric acid, commonly found in sodas, is also advised, as excessive consumption may impair calcium absorption.

Vegan Diet/Lactose Intolerance: Vegan diets can provide sufficient calcium and protein through fortified foods and plant-based sources. A study indicated that well-planned vegan diets do not negatively impact bone health if key nutrients are adequately met (45). Lactose intolerance can be managed with lactose-free dairy products or plant-based alternatives, ensuring adequate calcium intake. When considering bone health in vegan diets, it is crucial to address potential nutrient deficiencies, particularly vitamin D and B12, to reduce the risk of bone-related complications. A study by Tucker et al. (4) suggests that well-planned vegan diets do not negatively impact bone health if key nutrients are adequately met, primarily through fortified foods and plant-based calcium sources. Lactose intolerance, managed with lactose-free dairy products or plant-based alternatives, also ensures adequate calcium intake. Vegan diets are often lower in calcium, vitamin D, and vitamin B12, which are important for bone health. However, these nutrients can be obtained from fortified foods and supplements.

Low-Inflammatory Foods: A focus on whole foods rich in antioxidants reduces inflammation that may contribute to bone loss. Examples include fatty fish (omega-3s), berries, leafy greens, nuts, and seeds. A diet rich in anti-inflammatory foods is associated with higher BMD in postmenopausal women (16). Furthermore, the Dietary Approaches to Stop Hypertension (DASH) diet has shown potential for bone health due to its nutrient-rich profile and positive effects on bone mineral density (44). This diet, which emphasizes fruit, vegetables, lean protein, and low-fat dairy, offers a balanced intake of bone-supportive nutrients, suggesting its value as a dietary pattern for osteoporosis prevention. Chronic inflammation is a key driver of bone loss, and a diet rich in anti-inflammatory foods can help to reduce systemic inflammation and protect bone health.

Caffeine/Soda/Phosphoric Acid: Limiting caffeine and phosphoric acid intake, commonly found in sodas, is advisable as excessive consumption may impair calcium absorption. High caffeine intake may increase calcium excretion through urine, thereby reducing the amount of calcium available for bone mineralization. Soda consumption, especially those containing phosphoric acid, can lead to decreased calcium absorption and increased calcium loss from bones. These effects are particularly concerning in postmenopausal women, who are already at increased risk of calcium deficiency and osteoporosis.

Discussion

Interpretation of Findings

The literature demonstrates that a comprehensive nutritional approach, incorporating both specific nutrients and overall dietary patterns, is crucial for preventing and managing osteoporosis in postmenopausal women. Nutritional interventions are consistently shown to be both effective and accessible strategies for osteoporosis prevention and management. The evidence supports the synergistic benefit of calcium and vitamin D supplementation, with meta-analyses demonstrating a 20–30% reduction in fracture risk among postmenopausal women who receive these nutrients in adequate amounts (13; 30; 43). However, over-supplementation of calcium can result in adverse outcomes such as hypercalcemia and vascular calcification, emphasizing the importance of individualized dosing based on dietary intake and serum calcium levels (6). Magnesium is another essential nutrient, with adequate intake linked to higher BMD and improved bone formation, largely due to its role in vitamin D activation and bone remodeling (37; 47). Vitamin K, particularly the K2 form, is crucial for the carboxylation of osteocalcin and has been shown in both cohort studies and meta-analyses to improve BMD and reduce fracture risk (8; 5; 31). Protein intake, when balanced and adequate, supports bone matrix strength and muscle mass, with higher intakes linked to increased BMD and reduced fracture risk, especially when calcium intake is sufficient (7;11). Phytoestrogens, such as isoflavones from soy, provide modest benefits in BMD, particularly for women with low baseline bone density, and may help compensate for declining endogenous estrogen (29; 24). Omega-3 fatty acids, especially EPA and DHA, have been shown to reduce inflammation and bone resorption, and systematic reviews support their role in maintaining bone homeostasis and potentially reducing fracture risk (10).

Findings from the literature further reveal that dietary patterns, such as the Mediterranean diet, which is rich in fruits, vegetables, whole grains, and healthy fats, are associated with higher BMD and a lower risk of fractures (40; 15). These patterns provide essential nutrients and also exert anti-inflammatory and antioxidant effects that may benefit bone health. From a practical standpoint, the most effective dietary approach for postmenopausal women includes ensuring a daily intake of 1,200 mg calcium and 800–1,000 IU vitamin D, prioritizing dietary sources such as dairy, fortified foods, and fatty fish, and using supplements as needed based on individual assessment (46; 25). Magnesium should be obtained from nuts, seeds, leafy greens, and whole grains, while vitamin K intake should be increased through green leafy vegetables and fermented foods. Protein intake should be maintained at 1.0–1.2 g/kg body weight per day, with a mix of animal and plant sources. Phytoestrogen-rich foods like soy products and omega-3-rich foods such as fatty fish, flaxseeds, and walnuts should also be incorporated regularly. A summary of recommendations is included in Table 1.

Recommendations for Dietary Changes

Nutrient/Dietary Component	Recommended Daily Intake	Primary Food Sources	Supplementation Guidance	Notes/Evidence
Calcium	1,200 mg/day	Dairy (milk, yogurt, cheese), fortified plant milks, leafy greens, almonds	Supplementation if dietary intake is inadequate; avoid excess (>2,000 mg/day)	Essential for bone mineralization; over-supplementation can cause hypercalcemia
Vitamin D	800-1,000 IU/day	Fatty fish, fortified dairy/plant milks, egg yolks, sunlight exposure	Supplement often necessary, especially with limited sun exposure or darker skin	Facilitates calcium absorption; deficiency common in older adults
Magnesium	320 mg/day	Nuts, seeds, whole grains, leafy greens, legumes	Supplementation if dietary intake is low or deficiency is present	Supports vitamin D activation and bone remodeling
Vitamin K (K1 & K2)	90-120 mcg/day for K1, no RDA set for K2	Leafy greens (K1), fermented foods, cheeses, eggs (K2)	K2 supplementation may be considered for those at higher risk	Supports osteocalcin activation and bone mineralization; K2 may be more effective for bone
Protein	1.0-1.2 g/kg body weight/day	Lean meats, fish, dairy, legumes, nuts. soy products	Supplementation if dietary intake is insufficient	Maintains bone matrix and muscle mass; balance with adequate calcium intake
Phytoestrogens (Isoflavones)	No official RDA; ~50 mg/day in studies	Soy products (tofu, tempeh, soy milk), flaxseed	Consider soy isoflavone supplements for those not consuming soy	May modestly increase BMD, especially in early postmenopausal women
Omega-3 Fatty Acids	250-500 mg EPA/DHA per	Fatty fish (salmon,	Fish oil supplements or	Reduces inflammation and

	day	sardines, mackerel), flaxseeds, walnuts	algae-based omega-3 for vegetarians/vegans	bone resorption; supports bone homeostasis
Overall Dietary Pattern	Mediterranean Diet	Fruits, vegetables, whole grains, healthy fats, fish	N/A	Associated with higher BMD and reduced fracture risk

Table 1: Recommendations based on Results for Osteoporosis Prevention in Postmenopausal Women

Limitations of the Study

Despite these robust findings, several limitations must be acknowledged. There is considerable heterogeneity in study designs, populations, and intervention protocols, which complicates direct comparisons and synthesis of results. Many studies rely on self-reported dietary intake, which may introduce recall bias, and most intervention studies are of short to medium duration, limiting insights into long-term efficacy. Recall bias can occur when individuals have difficulty accurately recalling their past dietary habits. To minimize this bias, it is important to use validated dietary assessment tools and to collect dietary information prospectively, whenever possible. There are many confounding factors that could not be controlled in many studies, with genetics, comorbidities, and adherence to dietary or supplementation protocols varying widely. It is difficult to control for other lifestyle aspects like smoking, exercise, and genetics in studies, which may have an impact on bone health. These factors should be carefully considered when interpreting the results of studies on nutritional interventions for osteoporosis.

Additionally, even though much research has been done on dietary and nutrition interventions, there are still gaps in our understanding, particularly with regard to customized approaches and long-term results. The purpose of this study is to fill these gaps by giving a thorough overview of the literature and emphasizing the value of customized dietary strategies for postmenopausal women at risk of osteoporosis. Long-term studies are needed to assess the impact of dietary interventions on fracture risk and to identify strategies for maintaining bone health over the long term.

Conclusion

Summary of Key Findings

In conclusion, this review highlights the critical role of nutrition in preventing and managing osteoporosis in postmenopausal women, emphasizing the importance of a comprehensive dietary approach that includes adequate intake of calcium, vitamin D, magnesium, vitamin K, protein, phytoestrogens, and omega-3 fatty acids. These nutrients collectively support bone remodeling, enhance bone mineral density, and reduce fracture risk, particularly during the vulnerable postmenopausal period characterized by estrogen deficiency and accelerated bone loss. However, despite the promising evidence, further research is essential to improve nutrient combinations and dosages tailored for bone health. Future studies should explore emerging compounds such as phytochemicals, investigate the role of the gut microbiome and its modulation through probiotics and prebiotics, and assess the long-term impact of dietary interventions not only on bone density but also on bone quality and fracture outcomes. Additionally, developing personalized nutritional recommendations based on individual risk factors and genetic profiles could enhance the effectiveness of osteoporosis prevention strategies.

Public health initiatives must prioritize education on osteoporosis risk factors and promote preventive nutritional measures, targeting not only postmenopausal women but also younger populations to support bone health across the lifespan. Healthcare practitioners are encouraged to integrate nutritional assessments and counseling into routine care for postmenopausal women, while policymakers should facilitate access to nutritious foods and support environments conducive to healthy dietary patterns through subsidies, education, and community programs. Overall, nutrition remains a powerful and accessible tool in osteoporosis management, and a holistic approach combining specific nutrient intake with healthy dietary patterns offers the best opportunity to improve bone health and reduce fracture risk. Continued research and public health efforts will be vital to refine these strategies and empower individuals to make informed choices for lifelong skeletal health.

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