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The impact of vitamin D on muscle function and bone health in postmenopausal women

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Abstract:
The role of vitamin D in preserving muscle function and bone health in postmenopausal women has gained significant scientific interest. Menopause, characterized by a decline in estrogen levels, heightens the risk of osteoporosis and sarcopenia, significantly affecting mobility and quality of life. It is also associated with various physiological changes, such as hot flashes, mood disturbances, sleep irregularities, and an increased likelihood of musculoskeletal decline [1,2,3]. Vitamin D plays a pivotal role in calcium homeostasis, bone remodelling, and muscle function. However, its deficiency is widespread among postmenopausal women and has been linked to decreased bone mineral density, heightened fracture susceptibility, and diminished muscle performance. Although vitamin D supplementation is frequently recommended to mitigate these effects, its efficacy in enhancing musculoskeletal health remains a subject of debate. Recent studies have identified vitamin D receptors (VDR) in human muscle tissue, supporting the hypothesis that vitamin D contributes to muscle cell proliferation, differentiation, and function [11]. Despite these findings, inconsistencies in research outcomes and gaps in understanding the physiological mechanisms of vitamin D's action highlight the need for further investigation into how different levels of vitamin D influence musculoskeletal health and overall well-being in menopausal women.

This review evaluates evidence on vitamin D's impact on muscle strength, bone density, and fracture risk in postmenopausal women, focusing on its therapeutic potential.

Keywords: Vitamin D; Postmenopausal Women; Muscle Function; Bone Health; Sarcopenia; Osteoporosis

Introduction

Menopause is defined as the permanent cessation of menstrual cycles. According to the World Health Organization (WHO), it is characterized by the last menstrual period, followed by at least 12 consecutive months without bleeding. This transition must not be the result of surgical intervention, pharmacotherapy, or other factors such as illness or significant weight loss [39]. It is estimated that postmenopausal women currently account for over 10% of the world's population, which equates to approximately 700 million, with projections indicating that by 2030, this figure will exceed 20%. The age of menopause varies among individuals, typically occurring between 40 and 54 years, with a median age of 51 in developed nations [1,14,15]. According to the collected data, there is a link between vitamin D and menopause-related symptoms as well as postmenopausal illnesses.

Vitamin D is a lipid-soluble vitamin that plays a crucial role in various physiological processes such as calcium and phosphate metabolism. It is primarily synthesized in the skin upon exposure to ultraviolet B (UVB) radiation, which facilitates its conversion into its active form. Additionally, vitamin D can be obtained from dietary sources such as fish oil, fatty fish, egg yolks, meats, and dairy products. Its metabolism requires the coordinated function of several organs, including the skin, liver, kidneys, and intestines [4, 6, 7]. The primary circulating form of vitamin D is 25-hydroxyvitamin D (25(OH)D), which has a half-life of approximately 2–3 weeks and serves as the most reliable indicator of vitamin D reserves in the body. In contrast, the active metabolite, 1,25-dihydroxyvitamin D (1,25(OH)2D), is present in much lower concentrations and has a significantly shorter half-life of around 4 hours. Therefore, it is not commonly used as a marker of vitamin D status in the body [4,5,6].

Cardiovascular disease is the leading cause of death among postmenopausal women, followed closely by osteoporosis, neurodegenerative disorders, and hormone-sensitive cancers such as breast, ovarian, and endometrial cancer, with growing evidence suggesting that vitamin D deficiency is associated with their development and progression [3, 8].

Low vitamin D levels contribute to osteoporosis through decreased total fractional calcium absorption (TFCA), secondary hyperparathyroidism, increased bone resorption, and decreased bone mineral density (BMD). Furthermore, vitamin D deficiency is associated with accelerated bone loss, with studies indicating that while premenopausal women experience an annual bone loss of approximately 0.2% in the spine, this rate increases to 0.75% in postmenopausal individuals [7, 17]. Longitudinal studies have demonstrated that women with optimal vitamin D levels (46–98 nmol/L) have a 50% lower risk of premature mortality compared to those with levels below 40 nmol/L [16].

Given the high prevalence of vitamin D deficiency among postmenopausal women and its potential implications for both muscle function and bone health, this systematic review aims to evaluate the impact of vitamin D on musculoskeletal outcomes, with a particular focus on bone density, muscle strength, and the risk of fractures and falls in postmenopausal women.
The analysis is based on a comprehensive literature search of studies published between 2010 and 2025 using PubMed, Scopus, and Google Scholar. By synthesizing data from clinical trials and observational studies, this review aims to provide a thorough assessment of vitamin D's role in postmenopausal musculoskeletal health, identify gaps in existing research, and explore optimal supplementation strategies for this at-risk population.

Discussion

Osteoporosis

Natural menopause leads to significant endocrinological changes, particularly affecting bone and mineral metabolism. The decline in ovarian follicular function results in reduced estradiol production, which increases osteoclast activity, thereby accelerating bone resorption and leading to decreased mineral density, ultimately contributing to osteoporosis. Bone loss accelerates during the postmenopausal years, with a 1–2.3% decrease occurring within the first five years and an increase of 7–10% thereafter, greatly heightening the risk of osteoporotic fractures [41,17]. Osteoporosis affects over 30% of women aged 60-70, and the incidence rises to 70% for those aged 80 and older, largely due to estrogen deficiency during menopause (type 1 primary osteoporosis) [40,17]. It has been estimated that at least 1 in 3 women over age 50 will experience osteoporotic fractures, often requiring hospitalisation and long-term care, causing a large financial burden to health insurance systems [5].
Vitamin D is essential for maintaining calcium levels in the bones and regulating parathyroid hormone (PTH) levels. Low vitamin D levels are associated with osteoporosis, osteopenia, and increased fracture risk [17].
G Siregar MF et al.in a cross-sectional study, showed a significant positive correlation between serum vitamin D levels and bone mineral density (BMD) in postmenopausal women, as measured by radiofrequency echographic multi-spectrometry (REMS) [17]. Bone densitometry assessment utilizing radiofrequency echographic multi-spectrometry (REMS) is a novel ultrasound-based technique that provides reliable evaluations of BMD in the lumbar spine, femoral neck, and hip. The use of REMS has been validated in postmenopausal osteoporosis and has been recognized as a potential alternative to bone density scan by dualenergy x-ray absorptiometry (DXA) [17].
Shu-Bao Zhang et. al. in retrospective case-control study examined the relationship between pre-operative serum 25-hydroxyvitamin D [25(OH)D] levels and the occurrence of new vertebral fractures after percutaneous vertebral augmentation (PVA) in postmenopausal women [13]. The study found that lower 25(OH)D levels were significantly associated with a higher risk of both osteoporotic vertebral refractures (OVRFs) and cascade vertebral fractures (CVFs). Multivariate analysis confirmed 25(OH)D as an independent risk factor, even after adjusting for other factors. The study suggests that maintaining adequate vitamin D levels through supplementation may reduce the risk of new vertebral fractures [13].

Moschonis G et. Al. study was to evaluate the effects of vitamin D-enriched Gouda cheese consumption on serum concentrations of parathyroid hormone (PTH) and certain bone remodelling biomarkers in postmenopausal women in Greece [19]. A total of 79 women (aged 55–75 years) were randomly assigned to either a control group or an intervention group that consumed 60 g of Gouda cheese enriched with vitamin D3 (5.7 µg) daily for eight weeks during the winter. Results showed that consuming vitamin D-enriched Gouda cheese increased serum 25(OH)D levels, higher serum levels of both bone formation markers (i.e., osteocalcin and P1NP) reduced PTH levels and decreased bone resorption markers such as TRAP-5b. TRAP-5b seems to have an advantage against other bone resorption biomarkers, since it is a lysosomal enzyme specific only to osteoclast activity [19].

In a randomized, double-blind, placebo-controlled trial conducted by Nahas-Neto J et al., the effects of isolated vitamin D supplementation on bone turnover markers in younger postmenopausal women were investigated [20]. In this study, 160 women aged 50–65 years, who had amenorrhea for at least 12 months and normal bone mineral density, were divided into two groups: one receiving 1000 IU of vitamin D3 daily and the other receiving a placebo. The intervention lasted for 9 months. Results indicated that supplementation with vitamin D significantly increased serum 25-hydroxyvitamin D (25(OH)D) levels from, while levels decreased in the placebo group. There was a notable decrease in PTH levels and significant reductions in bone turnover markers, such as s-CTX and P1NP, in the vitamin D group. However, no significant differences in bone turnover markers were observed between the two groups, suggesting that further research is needed to clarify the effects of vitamin D on bone health [20].

The prospective cohort study conducted by Cauley JA investigated the association between serum 25-hydroxyvitamin D [25(OH)D] levels, bone mineral density (BMD), and fracture risk in women during the menopausal transition [43]. The study followed 1756 women across five clinical centres in the United States, measuring 25(OH)D levels and assessing incident fractures over an average of 9.5 years. The results indicated that higher 25(OH)D levels were inversely associated with nontraumatic fracture risk in midlife women. Specifically, women with 25(OH)D levels ≥20 ng/mL had a significantly reduced risk of nontraumatic fractures compared to those with lower levels. However, no significant association was found between 25(OH)D levels and traumatic fractures or changes in BMD across the menopausal transition. The study concludes that maintaining adequate serum 25(OH)D levels is important for reducing nontraumatic fracture risk in midlife women, suggesting that vitamin D supplementation may be warranted for those with levels <20 ng/mL [43].

The prospective observational study conducted by Raptis K et.al. focused on the impact of vitamin D levels on volumetric bone mineral density (vBMD) and bone architecture in postmenopausal women after distal radial fractures (DRF) treated conservatively [21]. A total of 39 participants were classified based on their serum 25(OH)D levels into two groups: Group A (≥15 ng/ml) and Group B (<15 ng/ml). Patients were followed for 12 weeks with peripheral quantitative computed tomography (pQCT) measurements taken at baseline, 6 weeks, and 12 weeks. Findings indicated that vitamin D deficiency did not significantly influence changes in trabecular or cortical bone metrics during the early healing period.

Trabecular bone mineral content (BMC) and vBMD showed significant increases at 6 weeks compared to baseline, reflecting early fracture healing. However, cortical BMC, vBMD, and cross-sectional area (CSA) progressively decreased over the 12 weeks, suggesting the effects of immobilization independent of vitamin D levels. Advanced age and higher bone turnover markers were associated with greater cortical bone loss. Overall, the study concluded that while vitamin D deficiency does not adversely affect early changes in vBMD and bone architecture after a DRF, factors such as age and increased remodelling are significant contributors to cortical bone loss [21].

Lastly, Reid IR et.al. study aimed to investigate the impact of vitamin D supplementation on bone mineral density (BMD) in adults [18]. The analysis was based on a systematic review and meta-analysis of 23 randomized trials that met specific criteria, involving 4,082 participants (92% women, average age 59 years). Most participants had serum 25-hydroxyvitamin D levels below 50 nmol/L, and many studies used doses of vitamin D below 800 IU per day. The meta-analysis revealed a small, statistically significant increase in bone mineral density at the femoral neck (mean difference 0.8%), but no effect was observed at other measured sites, including the total hip. The study suggests that while vitamin D may confer some benefits, particularly in the femoral neck, further investigation is warranted to better understand its effects on bone health [18].

Sacropenia

The term sarcopenia, derived from the Greek words “sarx” (flesh) and “penia” (loss), was first introduced by Rosenberg in 1989, who described the age-associated loss in muscle mass [12]. Nowadays, sarcopenia is defined as the loss of both muscle mass and strength and has been formally recognized as a muscle disease in the International Classification of Disease [44]. Sarcopenia has an estimated prevalence of 5–13% in 60–70 year olds and 11–50% in persons older than 80 years [25]. Vitamin D regulates the calcium-mediated functions of muscle, such as contraction, mitochondrial function and insulin sensitivity [12]. Hypovitaminosis D is usually asymptomatic, but subjects with low levels of circulating Vitamin D may present proximal muscle weakness, diffuse muscle pain, and difficulty in walking. Nevertheless, there is no consensus on a possible association between circulating levels of Vitamin D and walking speed, and only a few studies have assessed the association among serum Vitamin D levels and muscle strength and function in post-menopausal women [28].

Iolascon G et.al. in a prospective cohort study of postmenopausal women showed that six months of calcifediol treatment significantly increased serum 25(OH)D3 levels, improved appendicular muscle strength (measured by the Isometric Hand Grip Strength Test and the Knee Isometric Extension Strength Test) and physical performance (as measured by Short Physical Performance Battery (SPPB) and the 4-m gait speed (4MGS) [22]. At 6 months, the percentage of fallers was lower, although not significantly, whereas there was a significant reduction both in percentage of recurrent fallers and in the mean number of falls. The study concludes that calcifediol is effective in improving vitamin D levels, muscle function, and reducing fall risk in this population [22].

The Iolascon G et.al. retrospective study investigated the relationship between vitamin D deficiency and muscle performance in 401 postmenopausal women. Researchers compared women with hypovitaminosis D (serum 25-hydroxyvitamin D3 [25(OH)D3] <30 ng/mL) to those with sufficient levels. Outcome measures were: appendicular lean mass (ALM); ALM-to-BMI ratio (ALMBMI); total fat mass (FM); visceral adipose tissue (VAT); Hand Grip Strength (HGS); Knee Isometric Extension Strength (KES); Short Physical Performance Battery (SPPB); 4-meter gait speed (4MGS). The hypovitaminosis D group showed significantly lower scores across all measured indicators. Statistically significant correlations existed between 25(OH)D3 levels and muscle strength and physical performance measures.
The study concludes that vitamin D deficiency is associated with reduced muscle strength and physical performance in postmenopausal women. However, the retrospective design limits the ability to establish causality [11].
Current evidence demonstrating the impact of vitamin D supplementation on lower extremity muscle strength and function are controversial. While some trials have confirmed a significant effect of vitamin D administration on improving lower extremity muscle strength and function in older population others did not [23].
Bislev LS et.al. randomized, double-blind, placebo-controlled trial investigated the effects of vitamin D3 supplementation (70 µg/day, 2800 IU) on muscle strength, mass, physical performance, postural stability, well-being, and quality of life in 81 community-dwelling postmenopausal women with vitamin D insufficiency (<50 nmol/L 25(OH)D) and hyperparathyroidism [24]. The three-month intervention took place during the winter to minimize cutaneous vitamin D synthesis. Vitamin D3 supplementation significantly increased 25(OH)D and 1,25(OH)2D levels and decreased parathyroid hormone (PTH) levels. However, contrary to the hypothesis, vitamin D3 supplementation did not improve muscle strength (handgrip and knee flexion strength were significantly reduced), physical performance (Timed Up and Go test time increased), or other outcomes. Body composition (lean mass, fat mass index), postural stability, well-being, and quality of life remained unchanged. Analyses stratified by quartiles of 25(OH)D levels at the end of the study further revealed negative correlations between higher 25(OH)D levels and muscle strength and performance. The authors conclude that a relatively high daily dose of vitamin D3 supplementation (70 µg) provided no benefits and even showed adverse effects on muscle strength and physical performance in this population [24].
Rosendahl-Riise H et. al. systematic review and meta-analysis aimed to assess the effects of vitamin D supplementation, with or without calcium, on muscle strength and mobility in community-dwelling older adults [25]. A literature search conducted in April 2016 identified 15 relevant studies from 2408 articles, including 2866 participants aged 65 and older. Most studies reported no significant improvement in muscle strength or mobility following vitamin D supplementation. The meta-analysis found a nonsignificant change in hand grip strength across seven studies and a small but significant improvement in the timed-up-and-go test across five studies. However, a high degree of heterogeneity was observed among the studies. In conclusion, vitamin D supplementation did not enhance muscle strength but showed a minor positive effect on mobility, though further research with larger sample sizes is needed [25].

The aim of the systematic review with meta-analysis led by Tomlinson PB was to examine the effects of vitamin D supplementation on muscle strength in healthy adults [26]. The analysis included seven studies (six randomized controlled trials and one controlled trial) of high methodological quality, involving 310 participants (67% female) aged 21.5–31.5 years. The intervention duration ranged from 4 weeks to 6 months, with vitamin D doses varying from 4000 IU per day to 60,000 IU per week. The meta-analysis revealed a significant increase in upper and lower limb muscle strength in the supplemented group. These findings suggest that vitamin D supplementation positively affects muscle strength. However, further research should explore its effects on muscle power, endurance, and maximal strength [26].

Carranza-Lira S et.al. study examined the relationship between vitamin D levels, muscle mass, and cognitive function in 99 postmenopausal women aged 50 and older [27]. Measurements included calf circumference, skinfolds, muscle mass calculations, grip strength, the Short Physical Performance Battery (SPPB), the Sarcopenia Rapid Diagnostic Questionnaire (SARC-F), and the Mini-Mental State Examination (MMSE). Results showed a negative correlation between vitamin D levels and grip strength, SPPB scores, and MMSE scores; higher age correlated with higher vitamin D levels and SARC-F scores. Contrary to expectations, vitamin D did not positively impact muscle mass, and better cognitive performance (higher MMSE scores) was observed in women with lower vitamin D levels. These findings suggest a more complex relationship between vitamin D and both muscle mass and cognitive function in postmenopausal women than previously understood [27].

Ceglia L et.al. study investigated the effects of vitamin D3 (3200 IU/day), calcifediol (HyD, 20 mcg/day), or placebo on muscle tissue in postmenopausal women. Muscle biopsies (vastus lateralis) were analysed at baseline and 6 months using immunofluorescence to assess intramyonuclear vitamin D receptor (VDR) concentration, muscle fiber cross-sectional area (FCSA) for type I and II fibers, and PAX7 (satellite cell marker) levels. After 6 months, serum 25-hydroxyvitamin D (25OHD) levels increased significantly in both VD3 and HyD groups compared to placebo. . However, there were no significant group differences in VDR concentration, type II muscle fiber size or PAX7 markers. Importantly, only the vitamin D3 group showed a significant increase in type I muscle fiber cross-sectional area (FCSA). The authors conclude that while both vitamin D3 and HyD effectively raised 25OHD levels, only vitamin D3 resulted in increased type I FCSA, suggesting a potential benefit for muscle endurance. Simply increasing circulating 25OHD may not be sufficient to induce muscle benefits and the type of vitamin D supplementation may matter [28].

Cangussu LM et.al. in a double-blind, placebo-controlled clinical trial aimed to evaluate the effect of vitamin D supplementation on muscle function in younger postmenopausal women. 160 Brazilian women aged 50-65, with a history of falls in the previous 12 months, were assigned to receive either 1000 IU/day of vitamin D3 or a placebo for 9 months. After the intervention, the treatment group showed a significant increase in plasma 25-hydroxyvitamin D [25(OH)D] levels, while the placebo group had a decrease. In the treatment group, muscle strength of the lower limbs significantly improved as measured by the chair rising test.

The placebo group experienced a significant loss of lean mass measured by total-body dual-energy X-ray absorptiometry (DXA). The study concludes that vitamin D supplementation provides a significant protective effect against sarcopenia in postmenopausal women, improving muscle strength and preventing the progressive loss of lean mass [29].

In a randomized, double-blind, placebo-controlled conducted by Zhu et al., 1,000 IU/day vitamin D supplementation for 1 year significantly improved muscle strength in subjects who had low baseline muscle strength and whose serum 25(OH)D levels were below 24 ng/mL. Lower limb muscle strength and mobility were measured by Timed Up and Go test at baseline and after one year. Vitamin D supplementation significantly improved hip extensor and adductor strength and TUAG test performance, only in the lowest tertile of baseline muscle strength and mobility. This indicates that the benefit of vitamin D was most pronounced in the initially weakest and least mobile participants. The study concludes that vitamin D supplementation, combined with calcium, is beneficial for improving muscle strength and mobility in frail older women, but primarily those with the greatest pre-existing functional deficits [35].

The meta-analysis conducted by Beaudart et al. revealed a small but statistically significant positive effect of vitamin D supplementation on overall muscle strength. However, this effect was more pronounced in individuals with baseline 25-hydroxyvitamin D levels below 30 nmol/L and in those aged 65 years or older. No significant effects were observed on muscle mass or muscle power. Subgroup analyses explored potential effect modifiers (baseline 25(OH)D, age, treatment type, sex, study duration, vitamin D dose and study quality). The authors conclude that vitamin D supplementation has a modest positive effect on muscle strength, particularly in older adults and those with vitamin D insufficiency. However, they emphasize the need for further research to determine optimal treatment parameters (dose, duration, administration route) and to investigate the effects on muscle mass and power more thoroughly, due to limited data in these areas.[36]

Vitamin D plays a crucial role in musculoskeletal health, but its association with walking speed in older adults remains unclear. This systematic review and meta-analysis examined the relationship between circulating 25-hydroxyvitamin D (25OHD) levels and walking speed. A Medline search identified 22 observational studies (17 cross-sectional, 5 longitudinal) with participant numbers ranging from 54 to 4,100. The analysis found that individuals with vitamin D deficiency (VDD) or insufficiency (VDI) had significantly slower usual and fast walking speeds, as well as poorer performance on the Timed Up and Go (TUG) test, compared to those with normal vitamin D levels (NVD). The risk of slow walking speed was also significantly higher in participants with severe vitamin D deficiency (SVDD), VDD, and VDI [37].

Different Supplementation Methods and Their Effects

The commonest form of Vitamin D supplementation is represented by cholecalciferol (Vitamin D3), and most healthy adults reach the target of 20 ng/mL with 600 to 800 IU Vitamin D per day, whereas the cut off level of 30 ng/mL may require from 1800 IU to 4000 IU vitamin D3 per day [10]. The 25 hydroxylated Vitamin D metabolite (calcifediol) has been suggested as a therapeutic alternative; it has much shorter half-life compared to cholecalciferol and causes a rapid and sustained increase in plasma 25(OH)D concentration [10].

The rapidity and the extent of 25(OH)D circulating levels increase depend on the dosage, frequency, and kind of Vitamin D metabolite administration. Several randomized clinical trials showed that calcifediol is more effective and rapid than cholecalciferol in increasing circulating levels of 25(OH) Vitamin D. Furthermore, several reports suggest that different frequencies of supplementation with cholecalciferol act with different potency and rapidity in increasing 25(OH) Vitamin D levels [10].

The study led by Corrado A et. al. compared the effectiveness of calcifediol and different cholecalciferol supplementation regimens on serum 25(OH)D levels and lower limb muscle function in 107 postmenopausal women with vitamin D insufficiency. Participants were randomized into four groups: a single high-dose cholecalciferol (300,000 IU), cholecalciferol administered monthly (100,000 IU), cholecalciferol administered weekly (7000 IU), and calcifediol administered weekly (7000 IU). Serum 25(OH)D levels and muscle function (Sit-to-Stand test and Timed Up and Go test) were assessed at baseline and over a 6-month period. Calcifediol and weekly cholecalciferol led to faster and greater increases in serum 25(OH)D compared to monthly or single-dose cholecalciferol. These higher 25(OH)D levels were associated with improved lower limb muscle function. The most significant improvements were seen in the calcifediol group. The authors conclude that calcifediol is a more effective and rapid way to increase 25(OH)D levels and improve muscle function than the cholecalciferol regimens tested, offering a potential therapeutic advantage in treating vitamin D insufficiency in postmenopausal women. They also found that more frequent cholecalciferol dosing was superior to less frequent dosing [10].

The study by Yoon-Sok Chung et. al. evaluated the effectiveness of vitamin D supplementation in repleting serum 25-hydroxyvitamin D [25(OH)D] levels to ≥50 nmol/L in Korean postmenopausal women with osteoporosis. Of 371 screened women, 191 (52%) required vitamin D supplementation, and 88% (168 of 191) were successfully repleted. The majority (58%) of those successfully repleted received a daily dose of 2000 IU. The mean time to successful repletion was 31 days (standard deviation 8.4 days; range 11-48 days). Supplementation with a daily median dose of 2000 IU vitamin D successfully repleted 88% of Korean postmenopausal women with osteoporosis within 48 days to a serum vitamin D level of 50 nmol/L [42].

Hansen KE et.al. investigated whether high-dose cholecalciferol supplementation (aiming for 25(OH)D levels ≥30 ng/mL) would be more beneficial than low-dose cholecalciferol or placebo for postmenopausal women with vitamin D insufficiency. The randomized, double-blind, placebo-controlled trial included 230 participants. High-dose cholecalciferol resulted in a small increase in calcium absorption (1%), but there were no significant differences between treatment groups in bone mineral density, muscle mass, physical function, falls, or other measured outcomes after one year. The researchers concluded that there is no evidence to support maintaining serum 25(OH)D levels at 30 ng/mL or higher in this population, as both low and high doses of cholecalciferol were comparable to placebo in their effects [7].

The randomized controlled trial (RCT) conducted by Glendenning P et.al. investigated the effects of 3-monthly, supervised oral cholecalciferol supplementation (150,000 IU) versus placebo on falls, muscle strength, and mobility in 686 community-dwelling women over 70. After 9 months, there was no significant difference in fall rates between the groups (29% vs. 27%), nor were there significant differences in muscle strength or Timed Up and Go test results. While serum 25-hydroxyvitamin D levels were significantly higher in the cholecalciferol group, this did not translate into improved physical function or fall prevention. The study concludes that this intermittent, high-dose vitamin D regimen is not an effective strategy for reducing falls or improving physical function in older women, even considering potential adherence issues with daily vitamin D supplementation [9].

Pérez-Castrillón JL et.al. 1-year, double-blind, randomized, controlled, multicentre clinical trial evaluated the efficacy and safety of calcifediol (25OHD3) compared to cholecalciferol (vitamin D3) in postmenopausal women with vitamin D deficiency. A total of 303 participants were randomized into three groups: one receiving calcifediol 0.266 mg/month for 12 months, another receiving calcifediol 0.266 mg/month for 4 months followed by placebo, and a third receiving cholecalciferol 25,000 IU/month for 12 months. After 4 months, significantly more participants in the calcifediol group (35%) achieved serum 25(OH)D levels above 30 ng/mL compared to the cholecalciferol group (8.2%). Calcifediol demonstrated a faster and more potent increase in serum 25(OH)D levels, with the most significant differences observed in the first month of treatment. The study concluded that calcifediol was significantly more effective and faster than cholecalciferol in raising serum 25(OH)D levels in postmenopausal women with vitamin D deficiency. It provided a steady increase in vitamin D levels, making it a valuable alternative for supplementation, particularly in individuals requiring rapid correction of vitamin D deficiency [34]. Another study conducted by the same author, proved that long-term treatment with calcifediol produces stable and sustained 25(OH)D concentrations, with no associated safety concerns. When discontinued, it has been proved detrimental, with a sharp decrease in levels previously obtained indicating the need of maintaining vitamin D supplementation. Calcifediol is superior to cholecalciferol in improving vitamin D deficiency in postmenopausal patients with and without osteoporosis, with a faster onset of action [38].

International Osteoporosis Foundation (IOF) recommends that older adults aged 60 years and over should take a vitamin D supplement at a dose of 800 to 1000 IU/day, to achieve a serum 25(OH)D level of 75 nmol/L (30 ng/ml), as this is associated with greater muscle strength and improved bone health. Higher doses may be needed for individuals with obesity, osteoporosis, limited sun exposure, or malabsorption [30].

The recommendations of the Institute of Medicine (IOM) panel released on November 30, 2010 are largely based on bone health and call for 600 IU of vitamin D daily for all ages up to age 70 and 800 IU after age 71 [31].

In 2024, the Endocrine Society updated its guidelines, recommending that healthy adults under the age of 75 should not exceed the daily vitamin D intake levels established by the Institute of Medicine (IOM), which are 600 IU per day for individuals aged 50-70 years and 800 IU per day for those over 70 years old [32].

The study conducted by Chao YS et.al. examines the independent effects of vitamin D supplementation dose, frequency, and duration on plasma 25-hydroxyvitamin D (25(OH)D) levels in a large population-based sample. Data from 2,714 participants across 4,224 visits were analysed using multilevel regression. The findings indicate that a minimal regimen of 1,000–2,000 IU once or twice per week for one month was ineffective in significantly raising 25(OH)D levels. Higher doses (e.g., 5,000 IU or more), more frequent intake (e.g., daily supplementation), and longer duration (e.g., five months or more) were associated with significantly higher plasma 25(OH)D levels. Other factors, such as age, body weight, physical activity, smoking, and seasonality, also contributed to variations in 25(OH)D levels. The study confirms that higher doses, increased frequency, and longer duration of vitamin D supplementation significantly enhance plasma 25(OH)D levels. A daily intake of higher doses (e.g., 5,000 IU or more) for an extended period (five months or longer) leads to substantial increases in vitamin D status. Individual characteristics and lifestyle factors also influence vitamin D levels, suggesting that personalized supplementation strategies may be necessary to optimize vitamin D status [33].

Conclusion

In summary, the studies we reviewed confirm the importance of vitamin D in musculoskeletal function. Vitamin D insufficiency is a common but often neglected health problem. Evidence consistently demonstrates that vitamin D deficiency is linked to decreased bone mineral density, heightened fracture risk, and impaired muscle function. However, the effectiveness of vitamin D supplementation in reversing these adverse outcomes remains contentious, with studies yielding mixed results regarding its impact on muscle strength and bone density.

Vitamin D's role in muscle health appears to be complex and may depend on baseline levels of the vitamin, individual muscle characteristics, and the specific populations studied. The optimal dosage, frequency, and formulation of vitamin D supplements (comparing cholecalciferol and calcifediol) necessitate further exploration.

Our findings highlight the need for personalized approaches to vitamin D supplementation in postmenopausal women, tailored to individual needs and characteristics. While maintaining adequate vitamin D levels is crucial for overall health, further research is necessary to establish optimal supplementation strategies that maximize positive effects on bone health, muscle function, and overall well-being, while minimizing potential adverse effects. Future studies should focus on larger sample sizes, well-defined inclusion/exclusion criteria, and consistent measurement of relevant biomarkers to clarify the optimal approach to vitamin D supplementation in this population.

Disclosure

Author's contribution

Conceptualization: Katarzyna Agopsowicz and Katarzyna Blicharz; methodology: Michalina Piwowar; software: Anna Bieniasz; check: Anna Zdziebło, Martyna Biernacka and Dominika Stolarczyk; formal analysis: Katarzyna Zdziebło; investigation: Katarzyna Agopsowicz and Piotr Mojżesz; resources: Igor Biernacki; data curation: Katarzyna Blicharz; writing-rough preparation: Anna Bieniasz; writing- review and editing: Michalina Piwowar and Katarzyna Zdziebło ; visualization: Anna Zdziebło; supervision: Dominika Stolarczyk; project administration: Martyna Biernacka;
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Conflict of interest

The authors deny any conflict of interest.

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