REVIEW | OPEN ACCESS

Medical Science

To Cite:
Erazmus K, Kjałocha K, Świtała K, Pysz P, Hrapkowicz R, Mroczka M, Czernecka A, Borecki M, Tomczak D, Kuciel J. The Association of Vitamin D with Rheumatic Diseases: A Review. *Medical Science* 2025; 29: e102ms3586
doi: https://doi.org/10.54905/dissi.v29i161.e102ms3586

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Peer-Review History
Received: 07 May2025
Reviewed & Revised: 16/May2025 to 27/June/2025
Accepted: 03 July 2025
Published: 09 July 2025

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

The Association of Vitamin D with Rheumatic Diseases: A Review

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ABSTRACT

Vitamin D is a steroid hormone that exerts multidirectional effects due to the widespread expression of the vitamin D receptor (VDR) across various tissues. One of its most notable extra-skeletal roles is its ability to modulate immune system function. The objective of this review is to summarize current scientific evidence regarding the connection between vitamin D deficiency in the body and the onset or exacerbation of rheumatic diseases. Observational studies have established an association between low serum 25-hydroxyvitamin D levels and increased severity of rheumatic diseases. However, the benefits of vitamin D supplements in treating these conditions are still unclear. Some evidence suggests that vitamin D may help alleviate symptoms. It may also help prevent complications, especially those related to bone health. A review of the available data indicates that vitamin D may alleviate symptoms of the underlying conditions. Furthermore, it appears to be beneficial in preventing systemic manifestations of rheumatic diseases, particularly those affecting the skeletal system. Due to observed interindividual differences in response to vitamin D supplementation, the optimal serum 25(OH)D level remains a subject of ongoing debate.

Keywords: rheumatic diseases, vitamin D, 25-hydroxyvitamin D

1. INTRODUCTION

Vitamin D, a secosteroid hormone, is fundamental in maintaining bone integrity and regulating calcium–phosphate metabolism. Its biological influence, however, extends well beyond skeletal health due to the ubiquitous expression of vitamin D receptors (VDRs) across a wide array of human tissues. Thanks to this, vitamin D can influence several body systems. One of its key roles outside the bones is immune system regulation (Pike et al., 2017).

Rheumatic diseases are a wide range of autoimmune and inflammatory disorders primarily affecting the joints, muscles, and bones. Nonetheless, they can cause dysfunction in important organs such as the heart, lungs, and kidneys. In rheumatology, vitamin D supplementation is commonly recommended not only to treat or prevent osteoporosis—especially that caused by glucocorticoids—but also to reduce the risk of fractures in affected individuals (Buckley et al., 2017).

Besides protecting bones, vitamin D may help control immune activity and reduce inflammation. Consequently, it may suppress disease activity and manage chronic pain in autoimmune diseases (Charoenngam & Holick, 2020).

This review synthesizes current scientific knowledge regarding the physiological functions of vitamin D while exploring the connection between circulating vitamin D level and the development or exacerbation of several rheumatic conditions. These include rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), osteoarthritis (OA), spondyloarthropathies (SpA), and systemic sclerosis (SSc). The discussion focuses on recent evidence supporting vitamin D's multifaceted influence on immune regulation and the growing recognition of its deficiency as a contributing factor in autoimmune pathophysiology.

2. REVIEW METHODS

We conducted this review by searching for several databases, including PubMed, Google Scholar, and the Cochrane Library. The literature search covered studies published from January 2020 to April 2025. Predefined search terms included "vitamin D," "rheumatic diseases," and "25-hydroxyvitamin D." The inclusion criteria covered randomized controlled trials, observational studies, meta-analyses, and systematic reviews. We first screened the titles and abstracts of the retrieved records. We then reviewed the full-text articles that were considered relevant. We included only English-language publications and excluded studies that didn't directly examine the link between vitamin D levels and rheumatic disease activity. Each study was independently assessed for methodological quality and thematic relevance. In total, we included 29 studies that met all criteria.

3. RESULTS & DISCUSSION

Vitamin D – Metabolism in the Body

The classical function of vitamin D involves its role, alongside calcitonin and parathyroid hormone (PTH), in regulating calcium-phosphate homeostasis. The active form of vitamin D, calcitriol (1,25-dihydroxyvitamin D_3), exerts its effects by activating the vitamin D receptor (VDR), thereby contributing to the regulation of ionized calcium and phosphate levels through actions on the gastrointestinal tract, kidneys, and skeletal system.

When serum calcium levels drop, the body releases PTH, promoting the production of calcitriol. $1,25(OH)_2D_3$ increases the production of calcium-binding protein in the small intestine, which enhances intestinal calcium absorption—this process occurs independently of direct PTH action. PTH and calcitriol stimulate renal calcium reabsorption and promote calcium mobilization from bone. Calcitriol's effect on bone tissue is mediated through the RANK/RANKL signaling pathway by upregulating RANKL expression in osteoblasts, which secondarily activates RANK receptors on osteoclast precursors. This results in increased bone resorption and the subsequent release of phosphate and calcium into the circulation. The pleiotropic actions of vitamin D stem from the widespread expression of VDR in various tissue types. Furthermore, vitamin D influences the transcription of genes involved in the pathogenesis of rheumatic diseases (Charoenngam et al., 2019; Martens et al., 2020).

Pleiotropic Effects of Vitamin D

Vitamin D has significant immunomodulatory properties. The vitamin D receptor (VDR) is found in nearly all immune cells, allowing vitamin D to regulate both innate and adaptive immune responses. Inflammatory signals stimulate immune cells like phagocytes (e.g., interferon-γ and lipopolysaccharide), they convert circulating 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D, which exerts anti-inflammatory effects. It increases interleukin-10 (IL-10) secretion and reduces pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α (Colotta et al., 2017). Vitamin D also modulates the activity of adaptive immune cells, promoting a more tolerogenic phenotype in antigen-presenting cells (Martens et al., 2020).

However, research shows individualized responses to vitamin D supplementation. A trial by Shirvani et al., (2019) found that 60% of adults with vitamin D deficiency responded robustly to 10,000 IU of vitamin D_3 daily for six months, while 40% showed only mild changes despite reaching similar serum 25(OH)D levels. This indicates that genetic and epigenetic variables may influence how vitamin D affects the immune system in different people.

Vitamin D and Rheumatic Diseases

Epidemiological evidence indicates a strong correlation between vitamin D deficiency and the increased incidence and severity of rheumatic diseases. As previously mentioned, researches primarily attributed the association to the immunomodulatory and anti-in ammatory properties of active vitamin D, 1,25-dihydroxyvitamin D_3 ($1,25(OH)_2D_3$).

Researchers discovered considerably lower amounts of vitamin D in a variety of autoimmune rheumatic illnesses, including RA, SLE, and SSc. Vitamin D supplementation is investigated as a preventive and therapeutic strategy for various diseases, though its efficacy remains under scientific debate. Confounding factors like reduced outdoor physical activity and limited sun exposure, common in patients with chronic illnesses, may partly explain the observed associations (Buckley et al., 2017). This section reviews the relationship between vitamin D and rheumatic diseases, along with clinical evidence regarding the effects of vitamin D supplementation.

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune disorder that develops against complex and poorly understood immune system dysregulation, leading to inflammatory processes affecting multiple tissues and organs. Clinical manifestations include joint pain, cutaneous rashes, glomerulonephritis, and systemic symptoms. The disease exhibits considerable variability in both severity and natural course.

Symptoms related to a single organ may predominate for extended periods, with intermittent partial remissions and exacerbations. As the disease progresses, involvement of additional organs may occur; however, previously existing symptoms typically do not resolve (Predescu et al., 2025). Studies have shown that low concentrations of 25-hydroxyvitamin D [25(OH)D] in the blood are associated with both the onset and presence of systemic lupus erythematosus (SLE) (Islam et al., 2019). Individuals with SLE typically exhibit significant immune system imbalances, including increased activity of TH17 cells, impaired function of regulatory T cells (Tregs), fluctuating responses in TH1 and TH2 pathways, and the production of numerous autoantibodies, such as antinuclear antibodies (ANA), anti-Sm/RNP, anti-Ro/La, and anti-dsDNA (Predescu et al., 2025).

Moreover, studies have demonstrated that VDR expression in renal biopsy samples from 20 patients inversely correlates with both the Systemic Lupus International Collaborating Clinics (SLICC) renal activity scores and the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) scores (Sun et al., 2019). Thus, we may hypothesize that stimulation of the vitamin D signaling pathway could contribute to the attenuation of the autoinflammatory response in systemic lupus erythematosus (SLE) and lupus nephritis, primarily through its immunomodulatory actions on TH17 cells, regulatory T cells (Treg), and B lymphocytes, as well as through potential direct effects on renal tissues.

The findings indicate that vitamin D supplementation at daily doses of 4000 IU and 8000 IU significantly increases serum vitamin D concentrations and complement levels, while concurrently reducing fatigue, as assessed by the FACIT-Fatigue and FSS scales. Although a modest reduction in disease activity, as measured by the SELENA-SLEDAI index, was observed among the supplemented groups, these changes did not reach statistical significance. Furthermore, there are no significant differences in disease activity between the treated and untreated groups after six months of follow-up. These results indicate that vitamin D supplementation can effectively alleviate fatigue and enhance specific immunological parameters in individuals with systemic lupus erythematosus (SLE) (Predescu et al., 2025).

In summary, substantial evidence suggests a correlation between decreased serum 25(OH)D levels and the severity of symptoms in systemic lupus erythematosus (SLE). However, further research is needed to determine whether and to what extent vitamin D levels could influence the reduction of SLE risk.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is an autoimmune disease that mostly affects connective tissue. It is characterized by nonspecific, symmetric polyarthritis that affects small and medium-sized joints. Symptoms of the disease include extra-articular signs as well as systemic problems affecting numerous organ systems.

RA follows a relapsing-remitting course, with progressive joint damage leading to cartilage and bone destruction, deformities, joint contractures, and irreversible loss of function. Over time, the cumulative effects of the disease result in substantial physical disability, progressive functional impairment, and contribute significantly to premature mortality among affected individuals (Giannini et al. 2020). Through its immunomodulatory effects on the adaptive immune system, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D] plays a key role in the modulation of rheumatoid arthritis (RA) etiology and disease activity. This active form of vitamin D has been shown to inhibit the proliferation and function of T helper 1 and T helper 17 cells, while simultaneously promoting the activity of regulatory T cells (Tregs) (Aslam et al., 2019). Epidemiological studies have demonstrated a notable correlation between circulating 25-hydroxyvitamin D concentrations or dietary vitamin D consumption and the intensity of clinical symptoms (Lee & Bae, 2016).

Lin et al., (2016) conducted a meta-analysis of 24 papers published up to 2015 to investigate the connection between vitamin D status and RA. The results showed that patients with RA had notably lower serum 25(OH)D concentration than healthy individuals. Additionally, it was observed that vitamin D levels are inversely correlated with disease activity measured using the Disease Activity Score in 28 joints (DAS28). This correlation was more pronounced in patients residing in regions with lower latitudes and developing countries. Furthermore, another inverse relationship was identified between vitamin D levels and serum C-reactive protein (CRP) concentrations.

Furthermore, several studies have indicated that supplementation with 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] may contribute to improved clinical outcomes in rheumatoid arthritis (RA). In an open-label randomized clinical trial conducted by Gopinath & Danda (2011), 59 patients with RA who received $1,25(OH)_2D_3$ in combination with disease-modifying anti-rheumatic drugs (DMARDs) and calcium supplementation experienced significantly greater pain relief compared to 62 patients treated with DMARDs and calcium alone.

Merlino et al., (2004) demonstrated that increased vitamin D intake, assessed through dietary questionnaires, was associated with a lower incidence of rheumatoid arthritis (RA). However, subsequent large-scale studies did not confirm these findings. Nielen et al., (2006) investigated a smaller cohort of RA patients for whom pre-diagnostic serum samples were available. The results of retrospective vitamin D level measurements did not show significant differences compared to the control group.

In summary, evidence from clinical trials indicates that vitamin D supplementation may help lower disease activity in RA patients, even though the observed association between vitamin D status and the incidence and severity of RA may partially reflect confounding factors. Thus, the available data do not allow for a definitive assessment of the role of vitamin D in the modulation of the inflammatory process in rheumatoid arthritis (RA). Additional large-scale randomized clinical trials are necessary to establish whether vitamin D or $1,25(OH)_2D$ supplementation can be recommended as an adjunctive therapy for rheumatoid arthritis (RA) in clinical practice.

Osteoarthritis

Osteoarthritis is a disorder caused by the interaction of biological and mechanical factors, which disturb the balance between cartilage breakdown and repair, and alter subchondral bone remodeling, eventually affecting all joint tissues. It is primarily characterized by joint pain, reduced range of motion, crepitus, and varying degrees of local inflammatory changes, without systemic symptoms (Chen et al., 2017).

Considering that studies have demonstrated an association between low serum 25(OH)D concentrations and both the presence and severity of knee osteoarthritis across different age groups, it is hypothesized that vitamin D may influence the onset and progression of osteoarthritis (Bergink et al., 2016). This potential effect is attributed not only to its role in maintaining bone quality but also to its capacity to alleviate pain through the attenuation of inflammation and the enhancement of skeletal muscle function in the lower limbs.

Sanghi et al., (2013) included 107 patients with knee osteoarthritis in a randomized controlled trial. The numerical data summary is presented in Table 1.

Table 1. Data Summary Based on the Study

Study / Data	Value	Unit / Notes
Number of patients in Sanghi et al., (2013) study	107	Patients with knee osteoarthritis
Vitamin D_3 supplementation dose	60,000	IU/day
Duration of supplementation	12	Months
Pain reduction (VAS) – mean difference (MD)	−0.39	95% CI: −0.71 to −0.08
Improvement in symptom severity (WOMAC) – MD	−3.53	95% CI: −4.39 to −2.71

They received oral vitamin D_3 supplementation at a dose of 60,000 IU daily for 12 months. The treatment led to a relevant reduction in pain compared to the placebo group, with pain severity assessed with the Visual Analog Scale (VAS) (mean difference between groups [MD] −0.39, 95% confidence interval [CI]: −0.71 to −0.08). Furthermore, there was a notable improvement in overall symptom severity, as measured by the Western Ontario and McMaster Universities Arthritis Index (WOMAC) (MD −3.53, 95% CI: −4.39 to −2.71), compared to the placebo group (Sanghi et al., 2013). In conclusion, while vitamin D supplementation may confer a slight improvement in pain reduction and functional capacity among patients with knee osteoarthritis, it does not alter or reverse the fundamental pathophysiological progression of the disease.

Spondyloarthropathies

Spondyloarthropathies (SpA) comprise a heterogeneous group of autoimmune disorders unified by common genetic risk factors and overlapping clinical manifestations. This category includes, among others, reactive arthritis (ReA), ankylosing spondylitis (AS), psoriatic arthritis (PsA), enteropathic arthritis, and undifferentiated spondyloarthropathy. A distinguishing feature of these disorders is their high relationship with the human leukocyte antigen B27 (HLA-B27) and the frequent presence of enthesitis, indicating a similar pathophysiological route (McCullough et al., 2019).

$1,25(OH)_2D$ plays a regulatory role in modulating immune responses in individuals with psoriatic arthritis (PsA) (De Martinis et al., 2020). Research has shown that it reduces both the production of pro-inflammatory cytokines and the capacity of immune cells to promote osteoclast formation. These effects may help explain the noted inverse connection between serum 25(OH)D concentrations and both PsA incidence (Petho et al., 2015) and C-reactive protein (CRP) concentrations (Sağ et al., 2018). In addition, researchers have linked high-dose oral vitamin D_3 supplementation (50,000 IU daily) to marked clinical improvements in patients who have psoriasis (McCullough et al., 2019).

In addition to PsA, reduced serum 25(OH)D concentrations have also been reported in individuals with ankylosing spondylitis (AS) (De Martinis et al., 2020) and inflammatory bowel disease (IBD) (Del Pinto et al., 2015) when compared to healthy controls. In a population-based study involving 919 Israeli patients with AS, both 25(OH)D insufficiency (< 30 ng/mL) and deficiency (< 20 ng/mL) were identified as predictors of all-cause mortality (Ben-Shabat et al., 2020). Thus, vitamin D contributes to the modulation of disease severity in spondyloarthropathies (SpA) through its regulatory effects on both immune cell function and the gut microbiota, with the latter playing an increasingly important role in SpA pathogenesis.

Systemic Sclerosis

Systemic sclerosis is a long-term autoimmune connective tissue disease marked by ongoing fibrosis affecting the skin and internal organs. The disease frequently presents with vascular abnormalities and impaired function of multiple systems, including the lungs and digestive tract. It is considered a severe disorder due to its potential to cause progressive physical disability. Emerging research highlights a possible link between systemic sclerosis and insufficient levels of vitamin D (Runowska et al., 2021).

In a pilot clinical trial involving 20 patients with localized scleroderma, a 9-month regimen of oral calcitriol administration (0.75 μg/day for the initial 6 months, followed by 1.25 μg/day for the subsequent 3 months) did not demonstrate superior efficacy compared to placebo in improving skin scores (Hulshof et al., 2000).

Moreover, conventional vitamin D supplementation does not effectively correct deficiency in patients with systemic sclerosis (SSc). A potential explanation for this observation may concern the impaired intestinal absorption capacity resulting from the thickened intestinal wall, a characteristic quality of the disorder (Lin et al., 2016).

4. CONCLUSIONS

Low levels of calcidiol may lead to more serious symptoms in rheumatic diseases. Some studies have small sample sizes and limited statistical power. Confounding factors include variations in study populations, ethnicity, and diet. Most studies use retrospective data, which reduces reliability. Still, meta-analyses and in vitro studies suggest vitamin D has therapeutic potential and benefits for the immune system. Genetic studies show that individual genetic factors may affect the response to supplementation. To clearly determine the effect of vitamin D on the course of rheumatic diseases, more randomized clinical trials (RCTs) are needed, involving different vitamin D doses, well-defined dosing regimens, and precise patient selection. Consensus on defining vitamin D deficiency may facilitate more standardized research results. Despite its limitations, vitamin D remains a promising, safe, and cost-effective treatment option for patients with rheumatic diseases, even in those who use corticosteroids.

Author's Contributions

Kinga Erazmus - Conceptualization, review and editing, investigation, methodology
Karolina Jałocha - Methodology, investigation, visualization, supervision
Kinga Świtała - Conceptualization, visualization, resources,
Agnieszka Czernecka - Review, data curation, investigation
Marek Borecki - Resources, writing- rough preparation, data curation
Patrycja Pysz - Visualization, data curation, investigation
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Justyna Kuciel - Supervision, writing- rough preparation, data curation
Dominik Tomczak - Review and editing, formal analis, supervision
Roksana Hrapkowicz - Resources, writing- rough preparation, formal analis
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Acknowledgments

No acknowledgments.

Informed consent

Not applicable.

Ethical approval

Not applicable.

Funding

This study has not received any external funding.

Conflict of interest

The authors declare that there is no conflict of interest.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES

1. Aslam MM, John P, Bhatti A, Jahangir S, Kamboh MI. Vitamin D as a principal factor in mediating rheumatoid arthritis-derived immune response. Biomed Res Int. 2019;2019:3494937. doi:10.1155/2019/3494937
2. Ben-Shabat N, Watad A, Shabat A, Bragazzi NL, Comaneshter D, Cohen AD, Amital H. Low vitamin D levels predict mortality in ankylosing spondylitis patients: A nationwide population-based cohort study. Nutrients. 2020;12(5):1400. doi:10.3390/nu12051400
3. Bergink AP, Zillikens MC, van Leeuwen JPTM, Hofman A, Uitterlinden AG, van Meurs JBJ. 25-Hydroxyvitamin D and osteoarthritis: A meta-analysis including new data. Semin Arthritis Rheum. 2016;45(5):539–546. doi:10.1016/j.semarthrit.2015.09.010
4. Buckley L, Guyatt G, Fink HA, Cannon M, Grossman J, Hansen KE, Lane NE, Ma NS, Magrey M, McAlindon T, Robinson AB, Saha S, Womack C, Abdulhadi B, Charles JF, Cheah JTL, Chou S, Goyal I, Haseltine K, Jackson L, Mirza R, Moledina I, Punni E, Rinden T, Turgunbaev M, Wysham K, Turner AS, Uhl S. 2017 American College of Rheumatology guideline for the prevention and treatment of glucocorticoid-induced osteoporosis. Arthritis Rheumatol. 2017;69(8):1521–1537. doi:10.1002/art.40137
5. Charoenngam N, Holick MF. Immunologic effects of vitamin D on human health and disease. Nutrients. 2020;12(7):2097. doi:10.3390/nu12072097
6. Charoenngam N, Shirvani A, Holick MF. Vitamin D for skeletal and non-skeletal health: What we should know. J Clin

Orthop Trauma. 2019;10(6):1082–1093. doi:10.1016/j.jcot.2019.07.004
7. Chen D, Shen J, Zhao W, Wang T, Han L, Hamilton JL, Im HJ. Osteoarthritis: Toward a comprehensive understanding of pathological mechanism. Bone Res. 2017;5(1):16044. doi:10.1038/boneres.2016.44
8. Colotta F, Jansson B, Bonelli F. Modulation of inflammatory and immune responses by vitamin D. J Autoimmun. 2017;85:78–97. doi:10.1016/j.jaut.2017.07.007
9. De Martinis M, Ginaldi L, Sirufo MM, Bassino EM, De Pietro F, Pioggia G, Gangeni S. IL-33/vitamin D crosstalk in psoriasis-associated osteoporosis. Front Immunol. 2020;11:604055. doi:10.3389/fimmu.2020.604055
10. Del Pinto R, Pietropaoli D, Chandar AK, Ferri C, Cominelli F. Association between inflammatory bowel disease and vitamin D deficiency: A systematic review and meta-analysis. Inflamm Bowel Dis. 2015;21(11):2708–2717. doi:10.1097/MIB.0000000000000546
11. Giannini D, Antonucci M, Petrelli F, Bilia S, Alunno A, Puxeddu I. One year in review 2020: Pathogenesis of rheumatoid arthritis. Clin Exp Rheumatol. 2020;38(3):387–397.
12. Gopinath K, Danda D. Supplementation of 1,25 dihydroxy vitamin D3 in patients with treatment naive early rheumatoid arthritis: A randomised controlled trial. Int J Rheum Dis. 2011;14(4):332–339. doi:10.1111/j.1756-185X.2011.01684.x
13. Hulshof MM, Bouwes Bavinck JN, Bergman W, Masclee AA, Heickendorff L, Breedveld FC, Dijkmans BAC. Double-blind, placebo-controlled study of oral calcitriol for the treatment of localized and systemic scleroderma. J Am Acad Dermatol. 2000;43(6):1017–1023. doi:10.1067/mjd.2000.108369
14. Islam MA, Khandker SS, Alam SS, Kotyla P, Hassan R. Vitamin D status in patients with systemic lupus erythematosus (SLE): A systematic review and meta-analysis. Autoimmun Rev. 2019;18(11):102392. doi:10.1016/j.autrev.2019.102392
15. Lee YH, Bae SC. Vitamin D level in rheumatoid arthritis and its correlation with the disease activity: A meta-analysis. Clin Exp Rheumatol. 2016;34(5):827–833.
16. Lin J, Liu J, Davies ML, Chen W. Serum vitamin D level and rheumatoid arthritis disease activity: Review and meta-analysis. PLoS One. 2016;11(1):e0146351. doi:10.1371/journal.pone.0146351
17. Martens PJ, Gysemans C, Verstuyf A, Mathieu AC. Vitamin D's effect on immune function. Nutrients. 2020;12(5):1248. doi:10.3390/nu12051248
18. McCullough PJ, Lehrer DS, Amend J. Daily oral dosing of vitamin D3 using 5000 to 50,000 international units a day in long-term hospitalized patients: Insights from a seven year experience. J Steroid Biochem Mol Biol. 2019;189:228–239. doi:10.1016/j.jsbmb.2018.12.010
19. Merlino LA, Curtis J, Mikuls TR, Cerhan JR, Criswell LA, Saag KG. Vitamin D intake is inversely associated with rheumatoid arthritis: Results from the Iowa Women's Health Study. Arthritis Rheum. 2004;50(1):72–77. doi:10.1002/art.11434
20. Nielen MMJ, van Schaardenburg D, Lems WF, van de Stadt RJ, de Koning MHMT, Reesink HW, Habibuw MR, van der Horst-Bruinsma IE, Twisk JWR, Dijkmans BAC. Vitamin D deficiency does not increase the risk of rheumatoid arthritis: Comment on the article by Merlino et al. Arthritis Rheum. 2006;54(11):3719–3720. doi:10.1002/art.22191
21. Petho Z, Kulcsar-Jakab E, Kalina E, Balogh A, Pusztai A, Gulyas K, Horvath A, Szekanecz Z, Bhattoa HP. Vitamin D status in men with psoriatic arthritis: A case-control study. Osteoporos Int. 2015;26(7):1965–1970. doi:10.1007/s00198-015-3069-2
22. Pike JW, Meyer MB, Lee SM, Onal M, Benkusky NA. The vitamin D receptor: Contemporary genomic approaches reveal new basic and translational insights. J Clin Invest. 2017;127(4):1146–1154. doi:10.1172/JCI88887
23. Predescu OR, Dinescu SC, Bita CE, Florescu A, Musetescu AE, Vreju F, Ciurea PL. The immunomodulatory potential of vitamin D on Th17 lymphocytes in systemic lupus erythematosus: A literature review. Med Pharm Rep. 2025;98(1):13–20. doi:10.15386/mpr-2752
24. Predescu OR, Vreju FA, Musetescu AE, Florescu A, Dinescu SC, Bita CE, Ciurea PL. Effects of vitamin D supplementation on fatigue and disease activity in systemic lupus erythematosus. Cureus. 2025;17(2):e78830. doi:10.7759/cureus.78830
25. Runowska M, Majewski D, Majewska K, Puszczewicz M. Vitamin D supply in patients with rheumatic diseases in Poland: A pilot study. Reumatologia. 2021;59(3):146–152. doi:10.5114/reum.2021.107430
26. Sağ MS, Sağ S, Tekeoğlu İ, Solak B, Kamanlı A, Nas K, Halman H, Kantar M. Comparison of 25-hydroxy vitamin D serum concentrations in patients with psoriasis and psoriatic arthritis. J Back Musculoskelet Rehabil. 2018;31(1):37–43. doi:10.3233/BMR-169617
27. Sanghi D, Mishra A, Sharma AC, Singh A, Natu SM, Agarwal S, Srivastava RN. Does vitamin D improve osteoarthritis of the knee: A randomized controlled pilot trial. Clin Orthop Relat Res. 2013;471(11):3556–3562. doi:10.1007/s11999-013-3201-6
28. Shirvani A, Kalajian TA, Song A, Holick MF. Disassociation of vitamin D's calcemic activity and non-calcemic genomic activity and individual responsiveness: A randomized

controlled double-blind clinical trial. Sci Rep. 2019;9(1):17685. doi:10.1038/s41598-019-53864-1

29. Sun J, Zhang S, Liu JS, Gui M, Zhang H. Expression of vitamin D receptor in renal tissue of lupus nephritis and its association with renal injury activity. Lupus. 2019;28(3):290–294. doi:10.1177/0961203319826704