CHOJNACKA, Aleksandra Beata, BORAWSKI, Jacek, BURDON-SAJNÓG, Julia, SZYMONA-KUCIEWICZ, Zofia, BARTELA, Klaudia Katarzyna, DUSIEL, Julia Katarzyna, WAŁACHOWSKA, Anna, TABIAN, Alicja, KOZŁOWSKA, Paulina and SMĘT, Jakub. The role of vitamin D in the prevention and treatment of selected autoimmune diseases. ***Journal of Education, Health and Sport*****. 2025;85:66902. eISSN 2391-8306.**
https://doi.org/10.12775/JEHS.2025.85.66902
https://apcz.umk.pl/JEHS/article/view/66902

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 26.11.2025. Revised: 03.12.2025. Accepted: 03.12.2025. Published: 05.12.2025.

The role of vitamin D in the prevention and treatment of selected autoimmune diseases

Aleksandra Beata Chojnacka (corresponding author)
ORCID: https://orcid.org/0009-0009-9940-2264
Central Clinical Hospital of UCC WUM, Warsaw, Poland

Jacek Borawski
ORCID: https://orcid.org/0009-0003-4051-9990
St. Anne's Hospital in Piaseczno, Piaseczno, Poland

Julia Burdon-Sajnóg
ORCID: https://orcid.org/0009-0002-3883-9330
Independent Public Healthcare Complex in Lipsko

Zofia Szymona-Kuciewicz
ORCID:https://orcid.org/0009-0003-7403-6938
Międzyleski Specialist Hospital in Warsaw, Poland

Klaudia Katarzyna Bartela
ORCID: https://orcid.org/0009-0003-1233-9423
Cardinal Stefan Wyszyński University, Faculty of Medicine, Warsaw, Poland

Julia Katarzyna Dusiel
ORCID: https://orcid.org/0009-0007-0695-8964
Cardinal Stefan Wyszyński University, Faculty of Medicine, Warsaw, Poland

Anna Wałachowska
ORCID: https://orcid.org/0009-0008-1096-3827
Bielański Hospital named after Father Jerzy Popiełuszko, Warsaw, Poland

Alicja Tabian
ORCID: https://orcid.org/0009-0009-9485-5483
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland

Paulina Kozłowska
ORCID: https://orcid.org/0009-0004-8037-9862
Międzyleski Specialist Hospital in Warsaw, Poland

Jakub Smęt
ORCID: https://orcid.org/0000-0001-8532-4044
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland

Abstract:
Background. Autoimmune diseases affect a large number of people worldwide and currently pose a serious challenge to public health. Growing knowledge about their epidemiology, risk factors, as well as the processes leading to their development and the mechanisms responsible for their onset, opens new possibilities for the prevention and treatment of these diseases.
Aim. The article aims to draw attention to vitamin D and its powerful immunomodulatory effect, which is attracting increasing interest in the potential regulation of the immune response and its use in the prevention and treatment of autoimmune diseases.
Material and Methods. A literature review has been conducted using databases such as PubMed. Particular attention was paid to the most recent years of publication.
Conclusions. Vitamin D supplementation in people with autoimmune disease can provide numerous benefits, such as pain control, fatigue reduction, lower disease activity scores, remission induction, and reduced need for other medications. According to many premises, the active form of vitamin D may be a useful parameter in monitoring inflammation, a therapeutic biomarker, and even an indicator of disease progression and treatment effectiveness. However, further research is needed to assess the long-term effects and to gain a more complete understanding of the role, mechanisms of action and safety profile of vitamin D supplementation. Until more conclusive evidence is available, vitamin D should be considered only as a potentially helpful adjunct in the prevention and treatment of autoimmune diseases.

Keywords: vitamin D, autoimmune diseases, pathogenesis, rheumatoid arthritis (RA), multiple sclerosis (MS), systemic lupus erythematosus (SLE), supplementation, treatment

Introduction:

Mechanisms of action of vitamin D in the immune system

Vitamin D is essential for maintaining normal bone mineralization and also plays an important role in regulating the immune system. Its mechanisms of action are particularly important in the context of autoimmune diseases [4]. The vitamin D receptor (VDR) is found in many cells of the immune system, such as T lymphocytes, B lymphocytes, dendritic cells and macrophages [5]. The binding of vitamin D to the VDR initiates genomic and non-genomic signaling pathways that affect the functioning of the immune system [6].

In addition, vitamin D promotes the development of regulatory T cells (Tregs), which help maintain immune tolerance and prevent autoimmune reactions by inhibiting autoreactive T cells [6,7]. It can also inhibit the production of pro-inflammatory cytokines while increasing the production of anti- inflammatory cytokines, which contributes to a reduction in systemic inflammation [4].
Vitamin D supplementation may reduce the severity of autoimmune diseases by controlling the release of various inflammatory mediators and is therefore considered beneficial in reducing the activity of these diseases [8].

Pathogenesis of autoimmune diseases

Autoimmune diseases represent a diverse group of diseases characterized by an imbalance in the immune system, loss of immune tolerance to autoantigens, and impaired mechanisms for recognizing the body's own structures. This dysfunction leads to excessive proliferation and activation of autoreactive T or B cells, which causes the immune response to be directed against the body's own healthy tissues.
The resulting clinical symptoms resulting from this are heterogeneous and related to the specific site of involvement. Therefore, they can be divided into organ-specific, e.g. type 1 diabetes and systemic autoimmune diseases, e.g. lupus [9].
Autoimmune diseases tend to run in families, and their pathogenesis is mainly driven by genetic factors. Environmental and epigenetic factors also play an indispensable role in their development [9]. Many infectious agents also play a major role in the development of autoimmune diseases. For example, the Epstein-Barr virus (EBV) stimulates innate and adaptive immune responses through the structure of its proteins, which is associated with many diseases such as MS, SLE and RA [1].
In some Western countries, the increase in the incidence of autoimmune diseases is associated with changes in nutrition, which is explained by interactions between diet, gut microbiota, metabolites and immune cells. Despite its unclear mechanism of action, smoking also has an impact on these conditions [1].
It is assumed that most autoimmune diseases have a common etiology and pathogenesis, including a noticeable tendency to coexist with vitamin D deficiency in the body [3,9].

Rheumatoid arthritis (RA)

Rheumatoid arthritis (RA) is an autoimmune disease classified as a chronic rheumatic disease [10]. The prevalence of RA is estimated at approximately 1% of the global population. Although the exact etiology of the disease is unknown, there is evidence pointing to the interaction of genetic predisposition and environmental factors. The main area affected by the disease is the joints, which manifests itself in pain, stiffness and swelling. However, RA can also affect other organs, leading to serious complications, including cardiovascular and pulmonary diseases [4].

The basic mechanism of RA, as in other autoimmune diseases, is an abnormal attack by immune system cells on the body's own tissues [4]. Treatment of RA usually involves a combination of different therapeutic methods aimed at reducing inflammation, alleviating symptoms and preventing joint damage [11]. There is growing evidence that complementary therapies can also alleviate symptoms and improve the overall well-being of patients with rheumatoid arthritis [4].
The literature indicates that vitamin D plays a key role in the development and treatment of RA [7]. Vitamin D supplementation, analyzed in terms of clinical results for disease activity and joint damage assessment (DAS-28) and RA progression, shows promising results compared to the results of immunological studies [12].
The role of vitamin D in modulating immune function and inflammatory processes is well documented. Some studies have also shown that high doses of vitamin D have a beneficial effect on disease activity and pain control in patients with active RA and vitamin D deficiency [12]. Nevertheless, many results regarding its effect on the course of RA remain contradictory and inconclusive [13,14].
In addition, studies indicate that the active form of vitamin D may be a useful parameter in monitoring inflammation, a therapeutic biomarker, and even an indicator of disease progression and treatment efficacy in patients with RA [12].
Higher vitamin D concentrations are associated with a lower risk of RA, but the overall effect of vitamin D on the immunomodulation of pathophysiology cannot be clearly determined. Further research is needed to evaluate the long-term effects and to gain
a more complete understanding of the role, mechanisms of action and safety profile of vitamin D supplementation in patients with RA with varying disease activity as assessed by the DAS-28 index [12,15].

Multiple sclerosis (MS)

Multiple sclerosis (MS) is the most common demyelinating disease with multifactorial pathogenesis affecting the central nervous system. Many researchers have demonstrated a link between vitamin D deficiency and an increased risk of developing MS.
This disease is also an increasingly common cause of disability worldwide, especially among young adults [16]. MS is considered an immune-mediated disease leading to demyelination, axonal damage and oligodendrocyte damage [17]. The most common clinical form is relapsing-remitting MS, which occurs in approximately 80% of patients. Clinical symptoms are varied and include visual, motor and sensory disturbances, fatigue, coordination and cognitive impairment [16]. Currently, disease-modifying therapies (DMTs) aim to reduce the number of relapses and the progression of demyelinating lesions, thereby reducing the risk of permanent disability [18].
Vitamin D3 inhibits myelin basic protein (MBP)-specific T lymphocytes, increases the number of CD4+CD25+ regulatory lymphocytes, and reduces the activation of microglia and astrocytes [19,20]. Studies indicate that high or normal vitamin D intake may prevent the development of MS or reduce the risk of its occurrence [16].
Many studies have observed a significant reduction in the EDSS (Expanded Disability Status Scale – used to qualify treatment and evaluate its effectiveness) and the number of relapses in groups receiving vitamin D. However, analyses have shown that a significant reduction in the frequency of relapses was observed mainly in patients taking vitamin D for more than 12 months. No significant effect on fatigue or quality of life was found [16].

In addition, each 10 ng/ml increase in vitamin D levels may be associated with a 15% lower risk of new T2 lesions at a later date [21]. Interestingly, higher vitamin D levels may also reduce the severity of COVID-19 in people with multiple sclerosis [22].
In order for it to be considered an effective disease-modifying therapy, larger, dose- ranging clinical trials with clinically relevant endpoints are needed. Therefore, until more conclusive evidence is obtained, vitamin D should be considered only as a potentially helpful adjunctive therapy [16].

Systemic lupus erythematosus (SLE)

Systemic lupus erythematosus (SLE) is a chronic inflammatory connective tissue disease that affects the joints and numerous organs, including the kidneys, heart, skin, lungs and central nervous system. The disease manifests itself through a variety of symptoms and most commonly affects women of childbearing age [23]. Dysfunction and deposition of immune complexes activate the complement system and inflammatory cells, causing local and systemic inflammation, leading to damage to the body's own tissues and, as a consequence, disrupting the proper functioning of many organs [24]. The causes of this immune system dysregulation are not fully understood [23].
SLE is a disease with an unpredictable course, characterised by periods of remission and exacerbation, with a wide range of clinical symptoms, including general symptoms such as fatigue (in up to 100% of patients) and fever, as well as symptoms related to organ involvement [25].
Increasing serum vitamin D levels reduces inflammation, improves hemostasis indicators and reduces fatigue. In addition, it leads to a significant reduction in the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) and is associated with clinical benefits reflecting reduced disease activity [23]. Recent studies suggest that vitamin D supplementation not only lowers SLEDAI scores but may also promote remission or low disease activity (LDAS), reducing the need for glucocorticosteroids and modulating the immune response [26,27].
Vitamin D supplementation, thanks to its immunomodulatory and favorable safety profile, is a simple and accessible strategy to support the treatment of patients with systemic lupus erythematosus in clinical practice [23].

Vitamin D supplementation in selected autoimmune diseases

Vitamin D plays an important role in the musculoskeletal system and in the prevention of nutritional rickets, osteomalacia and osteoporosis as a mediator in the regulation of calcium-phosphate metabolism. Its deficiency (25(OH)D < 20 ng/ml) accelerates bone turnover, bone loss and is an associated common factor in osteoporotic fractures. Therefore, in the case of secondary osteoporosis caused by chronic treatment with glucocorticosteroids (GCS), it is important to maintain optimal concentrations of 25- hydroxyvitamin D, i.e. 25(OH)D (Table 1) [3].

Table 1. Blood 25(OH)D concentration thresholds [3].

25(OH)D concentration	Vitamin D status
<20 ng/ml (<50 nmol/L)	Deficiency
20-30 ng/ml (50-75 nmol/L)	Decreased level
30-50 ng/ml (75-125 nmol/L)	Optimal
50-60 ng/ml (125-150 nmol/L)	Safe
60-100 ng/ml (150-250 nmol/L)	Area of uncertainty; with potential benefits and risks
>100 ng/ml (>250 nmol/L)	Potential toxicity

The action of vitamin D is not limited to bones, as evidenced by the presence of vitamin D receptors (VDR) in every cell and tissue of the body, including immune cells, skin, brain, gonads, stomach, heart and pancreas. As a result, vitamin D deficiency can also affect the functioning of these organs, disrupt their functioning and, consequently, contribute to the development of chronic diseases [3].

Table 2. Recommended vitamin D dosage [3].

Patient group	Recommended dosage regimen (IU)
>18 years of age	1000-2000/day OR 7,000-14,000/week OR 30,000-60,000/month
>75 years of age	2000-4000/day OR 14,000-30,000/week
High-risk group (including autoimmune diseases)	**4,000/day OR Up to 30,000/week OR Up to 120,000/month for 3 months**

Due to the lipophilic nature of vitamin D, daily dosing is not necessary. An equal efficacy and safety profile is provided by weekly and monthly administration of the daily equivalent of 1000 IU of vitamin D3 [3].
Taking 2000 IU of vitamin D daily can reduce the risk of developing new autoimmune diseases by up to 22%. However, in vitamin D-deficient patients suffering from serious diseases, including autoimmune diseases such as RA, MS or SLE, dosing regimens should be more aggressive than in healthy individuals and sufficient to achieve and maintain higher 25(OH)D levels throughout the year, i.e. 55 to 70 ng/ml. Therefore, for patients at risk, vitamin D3 dosages of 4,000 IU/day or up to 30,000 IU weekly or up to 120,000 IU monthly for 3 months may be considered (Table 2.) [3].

In RA, supplementation at 60,000 IU per week or even 4,000 IU per day can potentially improve disease activity and pain control in patients after six months of therapy as part of stable basic treatment [13,28].
In patients with SLE, taking 400 IU daily for 12 weeks can significantly reduce IL-6 and TGF-β1 levels. In addition, changes in serum vitamin D concentrations may affect the proportions of Treg and Th17 lymphocytes, as well as the levels of cytokines associated with these subpopulations [29,30].
In patients with early MS taking 100,000 IU of vitamin D every 2 weeks for 2 years, this may be associated with reduced disease activity, number of relapses and new lesions on MRI [31].

Summary

- Numerous autoimmune diseases share similar etiological and pathogenetic factors, which are often accompanied by vitamin D deficiency.
- Vitamin D plays an important role in regulating the immune system, helping to maintain immune tolerance and prevent autoimmune reactions.
- Vitamin D supplementation may reduce the severity of the disease by controlling the release of various inflammatory mediators.
- Taking 2000 IU of vitamin D daily can reduce the risk of developing new autoimmune diseases by up to 22%.
- Due to the lipophilic nature of vitamin D, daily dosing is not necessary. Therefore, for patients at risk, vitamin D can be taken at a dose of 4000 IU/day or up to 30,000 IU per week or up to 120,000 IU per month.
- Higher concentrations of vitamin D in the body are associated with a lower risk of RA, may reduce the severity of the disease, and may be a useful parameter in monitoring inflammation, a therapeutic biomarker, and even an indicator of disease progression and treatment effectiveness
- High vitamin D intake may prevent the development of MS or reduce the risk of its occurrence, lead to a reduction in the Expanded Disability Status Scale (EDSS) score and the risk of new T2 lesions in patients with MS
- Increasing serum vitamin D levels reduces inflammation, improves hemostasis indicators and reduces fatigue, leads to a significant reduction in the SLEDAI disease activity index, and may also promote remission and reduce the need for glucocorticosteroids.
- More clinical trials with different doses and relevant endpoints are needed, and until more conclusive evidence is available, vitamin D should only be considered a potentially helpful adjunct.

Disclosures

Author's contribution:

Conceptualisation: Aleksandra Beata Chojnacka, Jacek Borawski
Methodology: Aleksandra Beata Chojnacka, Jacek Borawski
Software: Julia Burdon-Sajnóg, Zofia Szymona-Kuciewicz
Check: Julia Brudon-Sajnóg, Zofia Szymona-Kuciewicz
Formal analysis: Klaudia Bartela, Julia Dusiel
Investigation: Klaudia Bartela, Julia Dusiel
Resources: Alicja Tabian
Data curation: Alicja Tabian, Paulina Kozłowska
Writing-rough preparation: Aleksandra Beata Chojnacka, Jacek Borawski
Writing review and editing: Aleksandra Beata Chojnacka, Anna Wałachowska, Jacek Borawski
Visualisation: Anna Wałachowska, Jakub Smęt
Project administration: Aleksandra Beata Chojnacka

All authors have read and agreed with the published version of the manuscript.

Funding Statement:
The study did not receive special funding.

Institutional Review Board Statement:
Not applicable.

Informed Consent Statement:
Not applicable.

Data Availability Statement:
Not applicable.

Acknowledgments:
Not applicable.

Conflict Of Interest:
The author declare no conflict of interest.

References:

[1] Song, Y., Li, J., C Wu, Y. (2024). Evolving understanding of autoimmune mechanisms and new therapeutic strategies of autoimmune disorders. *Signal transduction and targeted therapy*, *S*(1), 263. https://doi.org/10.1038/s41392-024-01952-8
[2] Athanassiou, L., Kostoglou-Athanassiou, I., Koutsilieris, M., C Shoenfeld, Y. (2023). Vitamin D and autoimmune rheumatic diseases. *Biomolecules, 13(*4), 709. https://doi.org/10.3390/biom13040709
[3] Pludowski P. (2023). Vitamin D supplementation in different patient groups to reduce deficiencies. *Nutrients*, *15*(17), 3725. https://doi.org/10.3390/nu15173725

[4] Ranjbar M, Rahimlou M, Fallah M, Djafarian K, Mohammadi H. Effects of vitamin D supplementation in patients with rheumatoid arthritis: A systematic review and meta-analysis. *Heliyon*. 2025;11(3):e42463. Published 2025 Feb 4. https://doi.org/10.1016/j.heliyon.2025.e42463
[5] Skrobot A, Demkow U, Wachowska M. Immunomodulatory Role of Vitamin D: A Review. *Adv Exp Med Biol*. 2018;1108:13-23. https://doi.org/10.1007/5584_2018_246
[6] Chambers ES, Hawrylowicz CM. The impact of vitamin D on regulatory T cells. *Curr Allergy Asthma Rep*. 2011;11(1):29-36. https://doi.org/10.1007/s11882-010-0161-8
[7] Harrison SR, Li D, Jeffery LE, Raza K, Hewison M. Vitamin D, Autoimmune Disease and Rheumatoid Arthritis. *Calcif Tissue Int*. 2020;106(1):58-75. https://doi.org/10.1007/s00223-019-00577-2
[8] Fassio A, Gatti D, Rossini M, et al. Effects on Serum Inflammatory Cytokines of Cholecalciferol Supplementation in Healthy Subjects with Vitamin D Deficiency. *Nutrients*. 2022;14(22):4823. Published 2022 Nov 14. https://doi.org/10.3390/nu14224823
[9] Mu S, Wang W, Liu Q, et al. Autoimmune disease: a view of epigenetics and therapeutic targeting. *Front Immunol*. 2024;15:1482728. Published 2024 Nov 13. https://doi.org/10.3389/fimmu.2024.1482728
[10]Hasan, Ahmed & Raheem, Hasan & K. Hameed, Alaa. (2022). Rheumatic autoimmune diseases (focus on RA) : prevalence, types, causes and diagnosis.
[11]Radu AF, Bungau SG. Management of Rheumatoid Arthritis: An Overview. *Cells*. 2021;10(11):2857. Published 2021 Oct 23. https://doi.org/10.3390/cells10112857
[12]Rexhepi M, Krasniqi B, Hoti K, Daci A, Rexhepi-Kelmendi B, Krasniqi S. Impact of vitamin D supplementation on disease activity and pain management in rheumatoid arthritis: a randomized double-blinded controlled study. *BMC Rheumatol*. 2025;9(1):87. Published 2025 Jul 11. https://doi.org/10.1186/s41927-025-00543-6
[13]Al-Saoodi H, Kolahdooz F, Andersen JR, Jalili M. Effect of vitamin D on inflammatory and clinical outcomes in patients with rheumatoid arthritis: a systematic review and dose-response meta-analysis of randomized controlled trials. *Nutr Rev*. 2024;82(5):600-611. https://doi.org/10.1093/nutrit/nuad083
[14]Low CE, Loke S, Chew NSM, Lee ARYB, Tay SH. Vitamin, antioxidant and micronutrient supplementation and the risk of developing incident autoimmune diseases: a systematic review and meta-analysis. *Front Immunol*. 2024;15:1453703. Published 2024 Dec 9. https://doi.org/10.3389/fimmu.2024.1453703
[15] Song GG, Bae SC, Lee YH. Association between vitamin D intake and the risk of rheumatoid arthritis: a meta-analysis. Clin Rheumatol. 2012;31(12):1733–9. https://doi.org/10.1007/S10067-012-2080-7.
[16]Serag I, Abouzid M, Alsaadany KR, et al. Role of vitamin D as adjuvant therapy on multiple sclerosis: an updated systematic review and meta-analysis of randomized controlled trials. *Eur J Med Res*. 2025;30(1):736. Published 2025 Aug 12. https://doi.org/10.1186/s40001-025-02981-x
[17]Thompson AJ, Baranzini SE, Geurts J, Hemmer B, Ciccarelli O. Multiple sclerosis. *Lancet*. 2018;391(10130):1622-1636. https://doi.org/10.1016/S0140-6736(18)30481-1

[18]Haki M, Al-Biati HA, Al-Tameemi ZS, Ali IS, Al-Hussaniy HA. Review of multiple sclerosis: Epidemiology, etiology, pathophysiology, and treatment. *Medicine (Baltimore)*. 2024;103(8):e37297. https://doi.org/10.1097/MD.0000000000037297
[19]Correale J, Ysrraelit MC, Gaitán MI. Immunomodulatory effects of Vitamin D in multiple sclerosis. *Brain*. 2009;132(Pt 5):1146-1160. doi:10.1093/brain/awp033
[20]Galoppin M, Kari S, Soldati S, et al. Full spectrum of vitamin D immunomodulation in multiple sclerosis: mechanisms and therapeutic implications. *Brain Commun*. 2022;4(4):fcac171. Published 2022 Jun 30. https://doi.org/10.1093/braincomms/fcac171
[21]Mowry EM, Waubant E, McCulloch CE, et al. Vitamin D status predicts new brain magnetic resonance imaging activity in multiple sclerosis. *Ann Neurol*. 2012;72(2):234-240. https://doi.org/10.1002/ana.23591
[22]Montini F, Nozzolillo A, Tedone N, et al. COVID-19 has no impact on disease activity, progression and cognitive performance in people with multiple sclerosis: a 2-year study. *J Neurol Neurosurg Psychiatry*. 2024;95(4):342-347. Published 2024 Mar 13. https://doi.org/10.1136/jnnp-2023-332073
[23]El Kababi S, El Ouali EM, Kartibou J, Lamiri A, Deblij S, Supriya R, Saiedi A, Del Coso J, Laher I, Zouhal H. A Systematic Review and Meta-Analysis of the Effects of Vitamin D on Systemic Lupus Erythematosus. Nutrients. 2025; 17(17):2794. https://doi.org/10.3390/nu17172794
[24]Accapezzato D, Caccavale R, Paroli MP, et al. Advances in the Pathogenesis and Treatment of Systemic Lupus Erythematosus. *Int J Mol Sci*. 2023;24(7):6578. Published 2023 Mar 31. https://doi.org/10.3390/ijms24076578
[25]Schilirò D, Silvagni E, Ciribè B, et al. Systemic lupus erythematosus: one year in review 2024. *Clin Exp Rheumatol*. 2024;42(3):583-592. https://doi.org/10.55563/clinexprheumatol/mnvmvo
[26]Irfan SA, Ali AA, Shabbir N, et al. Effects of Vitamin D on Systemic Lupus Erythematosus Disease Activity and Autoimmunity: A Systematic Review and Meta-Analysis. *Cureus*. 2022;14(6):e25896. Published 2022 Jun 13. https://doi.org/10.7759/cureus.25896
[27]Arshad A, Mahmood SBZ, Ayaz A, Al Karim Manji A, Ahuja AK. Association of vitamin D deficiency and disease activity in systemic lupus erythematosus patients: Two-year follow-up study. *Arch Rheumatol*. 2020;36(1):101-106. Published 2020 Dec 10. https://doi.org/10.46497/ArchRheumatol.2021.8178
[28]Mukherjee D, Lahiry S, Thakur S, Chakraborty DS. Effect of 1,25 dihydroxy vitamin D3 supplementation on pain relief in early rheumatoid arthritis. *J Family Med Prim Care*. 2019;8(2):517-522. https://doi.org/10.4103/jfmpc.jfmpc_446_18
[29]Singgih Wahono C, Diah Setyorini C, Kalim H, Nurdiana N, Handono K. Effect of *Curcuma xanthorrhiza* Supplementation on Systemic Lupus Erythematosus Patients with Hypovitamin D Which Were Given Vitamin D_3 towards Disease Activity (SLEDAI), IL-6, and TGF-β1 Serum. *Int J Rheumatol*. 2017;2017:7687053. https://doi.org/10.1155/2017/7687053

[30]Jiang LJ, Rong ZH, Zhang HF. The changes of Treg and Th17 cells relate to serum 25(OH)D in patients with initial-onset childhood systemic lupus erythematosus. *Front Pediatr*. 2023;11:1228112. Published 2023 Aug 23. https://doi.org/10.3389/fped.2023.1228112
[31]Thouvenot E, Laplaud D, Lebrun-Frenay C, et al. High-Dose Vitamin D in Clinically Isolated Syndrome Typical of Multiple Sclerosis: The D-Lay MS Randomized Clinical Trial. *JAMA*. 2025;333(16):1413-1422. https://doi.org/10.1001/jama.2025.1604