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The effect of vitamin D supplementation on immune and hormonal parameters in patients with Hashimoto's disease: a review of the literature

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ABSTRACT

Introduction: Hashimoto's disease is the most common cause of autoimmune hypothyroidism. Accumulating research highlights the association between vitamin D deficiency and the pathogenesis of thyroid diseases. This is related to its immunomodulatory effect, through which it can influence the innate and acquired immune responses. This study aims is to evaluate the effectiveness of vitamin D supplementation in patients with Hashimoto's disease based on current clinical trials and meta-analyses. *Methods:* We reviewed selected literature available in the PubMed. Clinical trials, meta-analyses, systematic reviews and non-interventional studies published between 2017 and 2026 were included. We analysed the effect of supplementation on immunological and hormonal parameters. *Results:* Analysis of the collected data showed that vitamin D supplementation leads to a significant reduction in anti-thyroid antibody titers (TPO-Ab, TG-Ab). These changes are particularly noticeable when therapy lasts longer than 3 months. The effect on thyroid secretory function remains unclear. Some studies indicate improvements in hormonal parameters (TSH, FT3, FT4) while meta-analyses do not confirm this effect. Vitamin D suppresses inflammation by reducing pro-inflammatory cytokines. It also restores the balance between Th17 and Treg lymphocytes and affects B lymphocytes. Genetic factors, such as vitamin D receptor (VDR) polymorphisms, may modify the response to therapy. *Conclusions:* Vitamin D supplementation supports the reduction of inflammatory activity in Hashimoto's disease. Therefore, monitoring and balancing its concentration in patients is essential. Further research is needed to determine its impact on clinically relevant parameters and to establish therapeutic doses depending on the patient's genetic profile.

Keywords: Hashimoto's disease, autoimmune thyroiditis, anti-thyroid antibodies, immunomodulation, vitamin D supplementation

1. INTRODUCTION

Hashimoto's thyroiditis (HT) is a chronic autoimmune disease. The current prevalence of HT is estimated at 0.3-1.5 cases per 1,000 people, significantly more often in women (Durá-Travé et al., 2024). Environmental, genetic, immunological, and even nutritional factors play an important role in the etiopathogenesis of HT. The immune system malfunctions in an autoimmune manner, attacking and destroying thyrocytes. Both the humoral and cellular responses play a role here (Jin et al., 2022). Characteristic antibodies serve as helpful markers in the HT diagnosis. These include antibodies against thyroid peroxidase (TPO-Ab) and antibodies against thyroglobulin (TG-Ab) (Li et al., 2024). The consequence of these actions is chronic inflammation and fibrosis (Jin et al.,2022). These changes are visible on ultrasound as areas of reduced echogenicity (Li et al., 2024). Gradually, the thyroid gland becomes increasingly damaged. Ultimately, hypothyroidism may develop, resulting in a decrease in thyroid hormone levels in the blood. Patients may also develop goiter (Siddiq et al., 2023). Standard treatment requires lifelong thyroid hormones replacement therapy with L-thyroxine. According to the latest studies, 5-10% of patients with HT who have achieved normal thyrotropin (TSH) and free thyroxine (FT4) levels as a result of treatment continue to experience symptoms. The most common symptoms include fatigue, weight gain, dry skin, feeling cold, constipation, insomnia and depression. Additionally, symptom severity correlates noticeably with antibody levels (Zhang et al., 2025). Although HT is an incurable disease, dietary changes and appropriate supplementation can have improve the clinical picture and immunological parameters (Lebiedziński and Lisowska, 2023).

Vitamin D is a fat-soluble vitamin. Its two main steroid forms are vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol) (Szulc et al.,2023). Ultraviolet B (UVB) radiation stimulates its production in the skin. It can also be obtained from food (oily fish, egg yolks, yellow cheeses) and supplements (Sun et al., 2025). Currently, its deficiency constitutes a worldwide epidemic (Abaza et al., 2025). The conversion of vitamin D in the body is a process consisting of several stages carried out by cytochrome P450 oxidases. First, both vitamin D synthesized in the skin and that derived from the diet undergoes hydroxylation in the liver, converting into 25-hydroxyvitamin D (25(OH)D). This compound travels to the kidneys. There, another hydroxylation process takes place, resulting in the formation of its active form, 1,25-dihydroxyvitamin D (1,25(OH)2D). The resulting compound, also known as calcitriol, is transported to target tissues mainly by vitamin D-binding protein (DBP). By binding to vitamin D receptors (VDR), it influences calcium and phosphate metabolism and bone metabolism (Vieira et al., 2020; Sun et al., 2025; Pakosiński et al., 2025). Studies indicate that vitamin D has pleiotropic effects. Adequate levels of vitamin D have beneficial effects on the immune, nervous, and muscular systems, heart function and cancer risk.

Numerous analyses have noted a link between vitamin D deficiency and autoimmune thyroid diseases (Lebiedziński and Lisowska, 2023). This study assesses the impact of vitamin D levels and supplementation on the course of autoimmune thyroiditis based on recent research.

2. REVIEW METHODS

A literature review was conducted in the PubMed databases. The analysis included clinical trials, meta-analyses, systematic reviews and non-interventional studies on vitamin D supplementation in patients with HT. Publications from 2017 to 2026 in English were considered. Exclusion criteria: case reports, studies conducted exclusively in vitro, and lack of a full text. Relevant titles were identified using keywords such as "Hashimoto's disease," "autoimmune thyroiditis," "vitamin D," "anti-thyroid antibodies," "immunomodulation," "vitamin D supplementation," and "VDR receptor." The authors analyzed the selected publications and presented the results in the text. The article screening process followed the PRISMA guidelines (Figure 1).

3. RESULTS & DISCUSSION

Immunomodulatory mechanisms of vitamin D

Vitamin D has immunomodulatory effects, and some reports indicate that it plays an important role in the development of autoimmune thyroiditis. It affects both the innate and acquired immune responses. Recent studies point to potential mechanisms of its action. Immune system cells have nuclear vitamin D receptors (VDR) (Sun et al., 2025). Through these receptors, vitamin D modulates immune cell proliferation and differentiation, thus lessening thyroid gland damage (Zhao et al., 2021). It leads to a change in the phenotype from Th1 lymphocytes to Th2 lymphocytes. It inhibits the conversion of naive T lymphocytes into Th17 lymphocytes, which are responsible for tissue damage. It also supports the activity of Treg lymphocytes that maintain autotolerance. Consequently, this modulation alters the Th17/Treg ratio. This affects on the amount of cytokines produced. Vitamin D inhibits the production of pro-

inflammatory cytokines such as IL-2, IL-12, IL-17, IFN-γ, and TFN-α. At the same time, the anti-inflammatory cytokines IL-4, IL-5, IL-10, and TGF-β are produced in greater quantities (Durá-Travé et al., 2024; Soda et al., 2024; Bendotti et al., 2026).

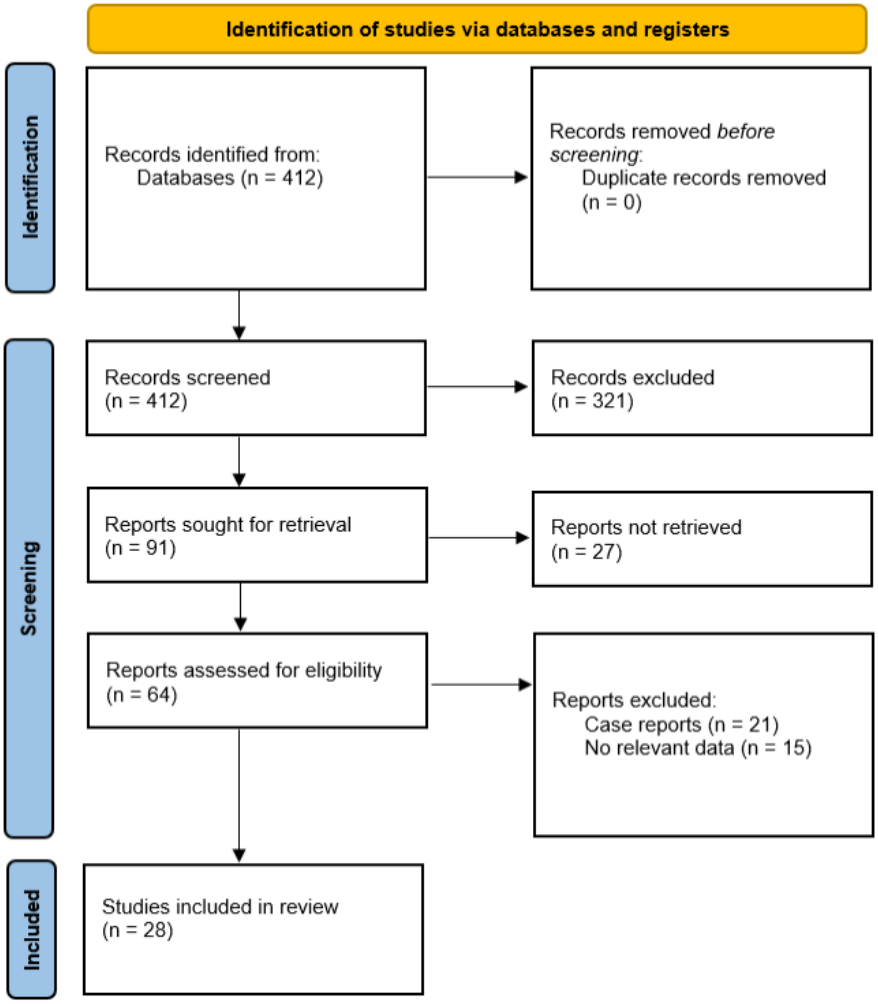

Figure 1. PRISMA flow diagram.

In addition, vitamin D can reduce MHC class II expression on the surface of thyroid follicular cells. This mechanism directly reduces the ability of antigen-presenting cells (APCs) to present T lymphocytes with thyroid autoantigen. It is also worth noting that vitamin D affects the activation, proliferation and apoptosis of B lymphocytes and their differentiation into plasma cells. This regulation leads to a reduction of the secretion of IgG and IgM. All these mechanisms promote immune tolerance and weaken the autoimmune attack on thyroid tissue (Szulc et al., 2023). Table 1 summarises the mechanisms by which vitamin D affects thyroid function in HT.

Table 1. Summary of the effects of vitamin D immunomodulatory mechanisms on immunological parameters and thyroid function in Hashimoto's disease.

Immune parameter	Effect of vitamin D	Effect on thyroid function in Hashimoto's disease
Th (helper) lymphocytes	Shifts the response from a pro-inflammatory Th1 profile to an anti-inflammatory Th2 profile	Reduction in the secretion of cytokines that destroy the gland and cause inflammation
Th17/Treg ratio	Restores balance	Inhibition of thyroid parenchyma destruction by lymphocytic infiltrates

MHC class II expression	Reduces the expression of MHC class II molecules on the surface of dendritic cells and thyrocytes	Reduction of thyroid autoantigen presentation to T lymphocytes
B lymphocytes	Inhibits activation, proliferation and differentiation into plasma cells	Reduction in the production of autoreactive antibodies

Vitamin D levels in patients with Hashimoto's disease

Currently, vitamin D deficiency is becoming increasingly common in the general population (Gierach and Junik, 2023). Measuring serum calcidiol (25(OH)D) concentration determines systemic vitamin D levels. The levels of this metabolite are considered the best indicator due to its long half-life and diagnostic reliability. The American Endocrine Society guidelines state that calcidiol concentrations below 20 ng/ml (<50 nmol/l) indicate vitamin D deficiency. Values between 20 and 29 ng/ml (50 – 75 nmol/l) suggest a relative deficiency. Normal vitamin D levels range from 30 to 50 ng/ml (75 – 125 nmol/l) (Durá-Travé et al., 2024).

Numerous observational studies and meta-analyses point out that patients with HT have significantly lower serum 25-hydroxyvitamin D [25(OH)D] concentrations compared to healthy individuals in the control group. In addition, there is a noticeable correlation between reduced vitamin D levels and the severity of HT. A 2023 study conducte in Poland by Gierach and Junik involved 370 women, categorized into three subgroups. A comparison of the results showed differences in vitamin D levels depending on thyroid function. In the control group (healthy women), the concentration was 34.38 ± 12.6 ng/ml. In patients with hypothyroidism, it was 31.15 ± 10.6 ng/ml, while patients with HT demonstrated the lowest values, 27.01 ± 14.4 ng/ml. Vitamin D concentration in women with HT was significantly lower ($p < 0.001$) compared to both healthy individuals and patients with hypothyroidism of other aetiologies. Vitamin D deficiency or relative deficiency affected 61% of patients studied most of whom had thyroid disease.

A retrospective analysis of 112 patients with newly diagnosed HT and 178 healthy women demonstrated similar results. It showed that patients had significantly lower vitamin D levels ($p = 0.007$). The percentage of patients with vitamin D deficiency was significantly higher in the euthyroid and HT group than in the healthy group (90.2% vs. 74.7%; $p = 0.001$) (Genç et al., 2025). In another retrospective observational study of the Mexican population, vitamin D deficiency (<20 ng/ml) affected 49.5% of patients with overt hypothyroidism in the course of HT and 42.9% of patients with subclinical HT (Copari-Vargas et al., 2025).

Different conclusions can be drawn from the multicentre study by Karakaya et al., (2025). This research evaluated a very large cohort of 81, 180 patients and shown that vitamin D levels were statistically higher in the HT group (41.43 nmol/l vs. 39.44 nmol/l, $p < 0.001$). In addition, vitamin D deficiency affected 65.5% of participants in the non-HT group and 62.1% in participants with HT ($p < 0.001$). These differences were not clinically significant. This discrepancy suggests a more complex role for vitamin D as a regulator rather than a direct cause of HT. Although most studies show lower vitamin D levels in HT, large population studies point out that territorial factors and lifestyle may modify this difference (Karakaya et al., 2025). Table 2 compares vitamin D concentrations in patients with Hashimoto's disease and in control groups.

Table 2. Comparison of vitamin D concentrations in patients with Hashimoto's disease and control groups.

Author and year of study	Vitamin D concentration in the study group [ng/ml]	Vitamin D concentration in the control group [ng/ml]	Conclusions
Gierach and Junik, 2023	27.01 ± 14.4	34.38 ± 12.6	Significantly lower concentrations in patients with HT ($p < 0.001$)
Genç et al., 2025	11.43 ± 5.19	14.53 ± 8.25	Significant deficiency in the HT group ($p < 0.001$)
Karakaya et al., 2025	16.57 ± 9.28	15.78 ± 8.88	Statistically higher concentrations in HT, but without clinical significance

Impact on immunological parameters

Vitamin D plays an essential role in preserving immune tolerance through immunomodulatory mechanisms. Its low concentration or deficiency in the body may exacerbate the autoimmune reactions characteristic of HT, while aiding in the production of autoantibodies against the thyroid gland (Aygün et al., 2025).

One study examined 144 participants, including 118 women and 26 men. The analysis showed that in patients with HT, TPO-Ab and TG-Ab levels were significantly higher, with simultaneously low vitamin D concentrations. The correlation between TPO-Ab and vitamin D levels was significant compared to the control group, and the level of these antibodies increased to 500 mmol/IU. The same relationship emerged for TG-Ab. They also assessed pro-inflammatory and anti-inflammatory cytokine concentrations finding that individuals with HT had elevated serum levels of IL-1β, IL-8, IL-10, and TNF-α ($p < 0.05$). IL-6 and IL-12 concentrations were not statistically different from those of healthy individuals ($p > 0.05$) (Siddiq et al., 2023).

These results were confirmed in another study which demonstrated a clear negative correlation between vitamin D concentration and antibody titer. In the group of patients with vitamin D deficiency, TPO-Ab antibody levels were significantly elevated (731.8 ± 991.0 U/ml) compared to individuals with optimal vitamin D concentrations (539.1 ± 313.4 U/ml, p=0.01). Therefore, there may be a relationship between low vitamin D levels and increased autoimmune activity of the thyroid gland (Copari-Vargas et al., 2025). In turn, one of the aforementioned studies observed a weak but noticeable negative correlation for both TPO-Ab and TG-Ab, regardless of the vitamin D status of the patients: < 20 ng/ml, 20–30 ng/ml, and > 30 ng/ml (Gierach and Junik, 2023). The study by Genç et al., (2025) confirmed a significant negative correlation only for TPO-Ab antibodies ($r = -0.133$, $p = 0.023$). However, no significant relationship was found between vitamin D concentration and TG-Ab levels ($r = -0.109$, $p = 0.063$).

Effect on hormonal parameters

The correlation between vitamin D and thyroid secretory function is clearly demonstrated in the results of some clinical studies. Copari-Vargas et al., (2025) indicate that lower vitamin D levels correlate with higher TSH values. Patients with vitamin D deficiency had the highest TSH concentration (11.5 ± 22.0 mIU/l). The results were compared to those recorded in patients with relative vitamin D deficiency (6.9 ± 7.9 mIU/l, $p = 0.01$) and in patients with normal levels (4.5 ± 1.9 mIU/l, $p = 0.01$). In turn, FT4 levels remained stable in all subgroups ($p = 0.44$-0.93). Additionally, researchers note the possible effect of vitamin D on the FT4/FT3 ratio. This hormonal shift may represent a form of compensatory adaptation of the body to deficiency (Vieira et al., 2020). The effect on FT4 is less clear but some studies show lower FT4 levels in patients with HT and low vitamin D compared to the healthy group (Genç et al., 2025; Siddiq et al., 2023).

A study conducted in the Jordanian population found a strong negative correlation between 25(OH)D and TSH ($r = 0.7493$, $p < 0.0001$ in the HT group). Lower vitamin D levels were associated with higher TSH values, regardless of age and gender. The TSH level in patients with HT (5.97 ± 0.18) was significantly higher compared to the control group (1.61 ± 0.1) ($p < 0.001$). In contrast vitamin D concentration was lower in the HT group (40.5 ± 0.72) than in the control group (57.3 ± 0.72) ($p < 0.001$) (Abaza et al., 2025).

Another retrospective study of 1,295 individuals showed a particularly strong association between vitamin D and TSH in premenopausal women (under 45 years of age). In the group of male subjects, the statistical analysis revealed no significant relationship between 25(OH)D levels and the thyroid parameters studied. The results showed a significant negative correlation between TSH levels and vitamin D levels in the entire female population ($p = 0.01$) and in individuals under 45 years of age ($p = 0.036$). This relationship was not statistically significant in women aged > 45 years ($p = 0.232$) or in men ($p = 0.465$). The statistical analysis failed to demonstrate a relationship between FT4 and vitamin D in any of the study groups. In addition, the authors analyzed ultrasound examinations. Low vitamin D levels were associated with heterogeneous thyroid parenchyma. This image shows lymphocytic infiltration in HT (Turashvili et al., 2021).

Proven effectiveness of supplementation

The therapeutic goal in HT is to inhibit the chronic inflammatory process. Its main diagnostic marker is elevated levels of anti-thyroid autoantibodies. Analysis of the results of studies on vitamin D supplementation in HT patients provides conclusions about the role of cholecalciferol in suppressing the autoimmune response.

The effectiveness of supplementation is supported by a meta-analysis conducted by Tang et al., (2023). It included 12 randomised clinical trials (RCTs) with a total of 862 participants. The authors observed that supplementation leads to reduced antibody levels. It has a significant effect on lowering TPO-Ab titres (SMD = -1.084, 95% CI = -1.624 to -0.545) and TG-Ab (SMD = -0.996, 95% CI = -1.579 to -

0.413) in patients with HT. At the same time, it contributes to improving thyroid function by influencing hormonal parameters. It leads to a reduction in TSH levels (SMD = -0.167, 95% CI = -0.302 to 0.031) and increases FT3 (SMD = 0.549, 95% CI = 0.077-1.020) and FT4 (SMD = 0.734, 95% CI = 0.184-1.285) levels. The meta-analysis also assessed the type of preparation administered and the duration of therapy. The active form of vitamin D (calcitriol) showed higher efficacy in reducing TPO-Ab than native forms (vitamin D2 or D3). Vitamin D supplementation for more than 12 weeks is significantly more effective in reducing antibodies and increasing FT4 and FT3 levels than shorter interventions (≤12 weeks).

Another meta-analysis, which included 8 studies (n = 652), shows similar effects. Vitamin D supplementation significantly reduced both TPO-Ab (SMD = -1.11, 95% CI = 1-1.92, -0.29) and TG-Ab (SMD = -1.12; 95% CI = -1.96, -0.28 p = 0.009). Additional analysis of the study subgroups revealed that these effects were only obtained after the use of vitamin D3. Vitamin D supplementation for >3 months led to a reduction in TPO-Ab titer (SMD = -1.66, 95% CI = -2.91, -0.41; p = 0.009). However, treatment lasting ≤3 months was not effective (SMD = –0.16; 95% CI = –0.51, 0.20; p = 0.392) (Zhang et al., 2021).

The study by Jiang et al., (2023) conducted in China involved 179 patients aged 12 to 75. It aimed to assess the effects of 6 months of treatment with vitamin D alone or in combination with L-thyroxine, and to compare them with a control group receiving no treatment. Some of the subjects in the treatment group received 800 IU/day of vitamin D, while the rest received 800 IU/day of vitamin D and 25-50 μg/day of L-thyroxine. After 6 months, the results between the groups were compared. In the group that used vitamin D, a decrease in TPO-Ab was observed compared to the control group (351.70 ± 183.25 vs. 246.37 ± 157.39, $p < 0.001$). FT3, FT4, and TSH concentrations were also compared. In the group treated with vitamin D, increases in FT3 and FT4 concentrations were observed (FT3: 4.30 ± 0.64 vs. 4.84 ± 0.9, $p < 0.001$; FT4: 15.15 ± 1.93 vs. 17.38 ± 2.97, $p < 0.001$) with a decrease in TSH concentration (3.58 ± 1.78 vs. 2.25 ± 1.22, $p < 0.001$). All of the above comparisons were made in relation to the control group. The addition of vitamin D to standard L-T4 treatment accelerated the normalization of TSH levels and allowed for faster achievement of euthyroidism compared to hormone monotherapy.

Controversy surrounding the effects of supplementation

The effect of vitamin D on parameters in patients with HT is unclear. Some researchers have shown a reduction in anti-thyroid autoantibody titers. Others, however, have not observed any effects (Behera et al., 2020).

A study involving seven cohorts of patients from six clinical trials with 258 patients with HT examined the effect of supplementation not only on immunological parameters, but also on hormonal parameters. It confirmed a statistically significant reduction in TPO-Ab levels (WMD = -158.18, 95% CI = -301.92, -14.45, p = 0.031; I2 = 68.8%, p heterogeneity = 0.007) in patients taking vitamin D compared to the control group. No significant differences in TSH, FT3, or FT4 levels were found (Jiang et al., 2022). Similarly, in a double-anonymized, randomized clinical trial, no significant changes in metabolic parameters were found after 12 weeks of oral vitamin D3 administration (50,000 IU per week) (Anaraki et al., 2017).

Table 3. Summary of the effects of vitamin D supplementation in Hashimoto's disease.

Author and year of publication	Number of participants	Vitamin D dose	Duration	Effects
Anaraki et al., 2017	65	50,000 IU/week	12 weeks	No significant effect on hormonal parameters
Chahardoli et al., 2019	42	50,000 IU/week	3 months	Reduction in TG-Ab titre and TSH level
Behera et al., 2020	23	60,000 IU/week (8 weeks), then once a month	6 months	Decrease in TSH, increase in FT4, increase in TPO-Ab titre
Zhang et al., 2021	652 (8 studies)	Varies (depending on the study)	Various (significant at >3 months)	Significant decrease in TPO-Ab and TG-Ab titres
Jiang et al., 2022	421 (6 studies)	Varies (depending on the study)	Various	Significant reduction in TPO-Ab titre No effect on TSH, FT3 and FT4
Tang et al., 2023	862 (12	Varies (depending on	Various	Significant reduction in TPO-Ab and TG-

	studies)	the study)		Ab titres, decrease in TSH levels, increase in FT3 and FT4
Jiang et al., 2023	179	800 IU/day	6 months	Decrease in TPO-Ab and TSH, increase in FT3 and FT4 levels

Another randomized controlled trial involved 42 women with HT. Some of them received vitamin D at a dose of 50,000 IU/week, while the others received a placebo. The concentrations of the parameters studied were measured at the beginning and end of the 3-month therapy. Analysis of the results showed that vitamin D had an effect on reducing serum TG-Ab ($p = 0.009$) and TSH ($p = 0.027$) levels compared to the values at the beginning of the study. At the same time, there was no significant reduction in TPO-Ab or in FT3 and FT4 hormone concentrations compared with the placebo group (Chahardoli et al., 2019).

Completely different results were obtained in India, where 23 patients with HT were given vitamin D at a dose of 60,000 IU/week for 8 weeks, followed by once-monthly doses for 4 months. The level of vitamin D increased significantly compared to the period before supplementation (15.33 ± 5.71 vs. 41.22 ± 12.24 ng/ml). Paradoxically, this led to a significant increase in TPO-Ab (from 746.8 ± 332.2 to 954.1 ± 459.8 IU/ml; $p = 0.006$). TSH levels decreased (from 7.23 ± 3.16 to 3.04 ± 2.62 (mIU/l); $p = 0.01$) (Behera et al., 2020). Table 3 summarises the effects of vitamin D supplementation in Hashimoto's disease.

Several publications suggest that supplementation is most effective in the early stages of the disease (euthyroidism or subclinical hypothyroidism). When the thyroid gland is almost destroyed (in the late stage of HT) vitamin D supplementation may not produce visible changes in hormone levels. However, it may still improve the overall response of the patient's immune system (Tang et al., 2023; Zhang et al., 2021).

Genetic factors

Recently, there have also been publications addressing the aspect of genetic factors and their impact on the effectiveness of therapy. Their authors point out that the body's response to the administered dose of vitamin D is influenced by environmental and epigenetic factors, as well as the presence of VDR receptor polymorphisms.

VDR gene polymorphisms can modify the structure and function of the receptor. This modulation occurs through their influence on mRNA stability. Their different effects depend on the region of occurrence. Two variants of polymorphisms are particularly important: FokI (rs2228570) and TaqI (rs731236). Meta-analyses indicate that their presence affects the way the body utilizes available vitamin D. They may also reduce the risk of developing HT. The presence of these variants is associated with a more effective reduction in antibody titers after supplementation. Interindividual variability is explained by DNA methylation of VDR promoters in immune system cells. The effect of polymorphisms is characteristic of a given population.

All this translates into individual patient sensitivity to vitamin D supplementation and allows researchers to understand differences in response to supplementation. Standard doses may be insufficient to produce a therapeutic effect on the immune system, even if blood levels of 25(OH)D are normal. Patients with specific genotypes may require higher doses or longer treatment times to achieve a therapeutic effect (Pakosiński et al., 2025; Sun et al., 2025).

4. CONCLUSION

Many studies show that vitamin D supplementation can have a beneficial effect on the course of the disease in patients with HT. In some studies, especially in patients with HT and baseline vitamin D deficiency, it seems to be effective mainly in reducing anti-thyroid antibody titers. Treatment lasting more than 3 months with a preparation of proven efficacy is more beneficial. However, the data are heterogeneous and do not always translate into improved thyroid function, slowed disease progression, prevention of recurrence or clinical benefits.

Vitamin D is a promising modifiable risk marker. However, its role as an independent therapeutic target in the treatment of HT has not been confirmed. It acts as an adjunctive therapy, influencing inflammatory activity. However, it does not replace L-thyroxine replacement therapy. This indicates the need for further research into its effect on clinically relevant parameters. Long-term, comprehensive studies in large cohorts are needed to determine the role of vitamin D supplementation in suppressing the excessive immune response in HT.

At this point, it is suggested that vitamin D levels be screened in patients during diagnosis or in cases of ineffective standard treatment. If a deficiency is found, individually tailored supplementation should be implemented.

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Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

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