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Vitamin D and Selenium: Review of Clinical Trials of Synergistic Effects on Thyroid Antibody Levels and Disease Progression in Hashimoto's Thyroiditis

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ABSTRACT

Introduction:

Hashimoto's Thyroiditis (HT) is a prevalent autoimmune thyroid disease (AITD) and one of the most common organ-specific autoimmune disorders. It results from genetic predispositions combined with environmental triggers, such as iodine excess or deficiency, selenium imbalances, drug usage, stress, and infections. The primary feature of HT is chronic lymphocytic infiltration of the thyroid gland, leading to its progressive destruction and hypothyroidism. This autoimmune condition is characterized by the production of thyroid-specific autoantibodies, notably antithyroid peroxidase (anti-TPO) and antithyroglobulin (anti-TG) antibodies.

Materials and Methods: This study employed a mixed-methods approach, combining a comprehensive literature review and analysis of clinical trials to investigate the research question. The methods involved systematic identification, screening, and evaluation of available evidence, with a focus on studies of high relevance and methodological rigor.

Results: Selenium supplementation reduced TSH levels in patients without thyroid hormone replacement therapy (THRT) and significantly decreased TPOAb and MDA levels in patients with and without THRT, with no notable effects on fT4, T3, or TGAb. Adverse effects were comparable between selenium and placebo groups. Vitamin D deficiency was strongly associated with higher TSH levels and elevated anti-TPO and anti-TG antibodies, with negative correlations observed between vitamin D levels and these markers in Hashimoto's Thyroiditis (HT) patients. HT patients exhibited significantly lower vitamin D levels compared to healthy individuals and those with hypothyroidism alone, highlighting the potential immunomodulatory role of vitamin D. These findings suggest that both selenium and vitamin D supplementation may offer therapeutic benefits in managing HT.

Conclusions: Selenium and vitamin D supplementation show potential in managing Hashimoto's Thyroiditis by reducing TSH and thyroid antibody levels, highlighting the importance of addressing micronutrient deficiencies in autoimmune thyroid conditions.

Keywords: Hashimoto, vitamin D, selenium, supplementation, Hashimoto's Thyroiditis

INTRODUCTION

Hashimoto's thyroiditis (HT), also known as chronic lymphocytic thyroiditis[1], is a common autoimmune disorder characterized by the progressive destruction of the thyroid gland, leading to impaired thyroid function and, ultimately, hypothyroidism. This condition is one of the most frequent causes of thyroid dysfunction, particularly in iodine-sufficient regions, and affects a significant proportion of the population, with a higher prevalence among women. The pathophysiology of HT involves an autoimmune attack where the body's immune system mistakenly targets thyroid cells, triggering chronic inflammation.[5] A key feature of HT is the presence of elevated thyroid-specific autoantibodies, most notably anti-thyroid peroxidase antibodies (TPOAb) and anti-thyroglobulin antibodies (TgAb), which are diagnostic markers and indicators of disease progression.[2]

The clinical presentation of HT varies widely but often includes symptoms of hypothyroidism, such as persistent fatigue, unexplained weight gain, sensitivity to cold, dry skin, hair thinning, and constipation. Cognitive impairments, depression, and mood disturbances are also common and can severely impact a patient's quality of life. In some cases, the thyroid gland may enlarge, forming a goiter, while in others, atrophic changes dominate. These symptoms, combined with the long-term risk of cardiovascular complications and metabolic dysfunction, underscore the importance of effective management strategies for HT.[4-6]

Vitamin D importance in HT treatment

Numerous studies, including the referenced research, have identified a strong correlation between low vit-D levels and the prevalence of HT. Vitamin D receptors (VDR) are found in immune cells, where they regulate both innate and adaptive immune responses. Deficiency in vit-D may disrupt these regulatory mechanisms, exacerbating autoimmune activity and fostering thyroid gland destruction.[3]

Vitamin D is crucial for immune system regulation, promoting innate immunity while suppressing adaptive responses. This duality fosters immune tolerance and may inhibit autoimmune processes in HT. By modulating T-helper cell activity and reducing antigen-presenting cells, vit-D could alleviate inflammation and autoantibody production.[10]

Selenium importance in HT treatment

Selenium, an essential trace element, plays a pivotal role in maintaining thyroid health. It is a key component of selenoproteins, such as glutathione peroxidases and iodothyronine deiodinases, which are involved in protecting thyroid cells from oxidative damage caused by hydrogen peroxide and in the activation and metabolism of thyroid hormones. Selenium's antioxidant properties help mitigate the oxidative stress associated with chronic inflammation in HT, potentially slowing the autoimmune process and preserving thyroid function.[1-2]

This systematic review aims to synthesize the current evidence on the effects of selenium supplementation in individuals with HT. The focus is on evaluating its impact on thyroid function, levels of thyroid autoantibodies, patient-reported outcomes such as quality of life and symptom relief, and the incidence of adverse events. By critically appraising and consolidating findings from randomized controlled trials, this review seeks to provide clarity on the potential role of selenium as a complementary treatment in the management of HT and to identify gaps in the evidence that warrant further investigation.[6]

MATERIALS AND METHODS

The studies utilized a combination of randomized controlled trials (RCTs), systematic reviews, meta-analyses, and observational approaches to investigate the effects of selenium and vitamin D supplementation in the management of Hashimoto's Thyroiditis (HT). Selenium studies included RCTs assessing its impact on thyroid function parameters, such as TSH, TPOAb, and oxidative stress markers like malondialdehyde (MDA), with interventions involving selenium alone or in combination with thyroid hormone replacement therapy (THRT).[2] A systematic review and meta-analysis synthesized data from multiple RCTs to evaluate pooled effects, addressing heterogeneity and bias using the Cochrane risk-of-bias tool and GRADE assessment for evidence quality. Vitamin D studies employed cross-sectional analyses and subgroup comparisons to explore correlations between vitamin D levels, thyroid hormone levels (TSH, fT4, and T3), and anti-thyroid antibodies (anti-TPO and anti-TG), with participants categorized by autoimmune and thyroid function status. Biochemical analyses were conducted using standardized laboratory assays, and statistical methods such as correlation and regression analyses assessed relationships between variables. Collectively, these methodologies offered a comprehensive evaluation of the therapeutic potential of selenium and vitamin D supplementation in HT.[3]

RESULTS

Selenium supplementation in Hashimoto treatment

Outcomes	Illustrative comparative risks* (95% CI)		Relative effect (95% CI)	No of participants (studies)	Quality of the evidence (GRADE)	Comments
	Assumed risk	Corresponding risk				
	Placebo (+ levothyroxine)	Selenium (+ levothyroxine)				
Change from baseline in health-related quality of life	See comment	See comment	Not estimable	See comment	See comment	Not reported in any study
Change from baseline in assessment of symptoms such as mood, fatigue and muscle weakness Short-Form Health Survey Follow-up: mean 3 months	**167 per 1000**	**778 per 1000** (268 to 1000)	**RR 4.67** (1.61 to 13.5)	36 (1 study)	⊕⊕⊕⊖ **low** [b,c,d]	
Proportion of participants reporting an adverse event Follow-up: mean 5 months			**RR 2.71** (0.29 to 25.66)	258 (3 studies[e])	⊕⊕⊖⊖ **low** [b]	Participants in placebo group counted twice (same participants in both comparisons)
Change from baseline in serum levels of anti-thyroid peroxidase antibodies Decrease from 1508 to 25 IU/L Follow-up: mean 4.5 months	See comment	See comment	Not estimable	252 (4 studies[e])	⊕⊕⊖⊖ **low** [b]	Data could not be pooled because of substantial clinical heterogeneity of participants, interventions and controls
Change from baseline in LT_4 replacement dosage at end of study	See comment	See comment	Not estimable	See comment	See comment	Not reported in any study

Figure 1. Selenium (+LT4) compared to placebo (+LT4) for participants with Hashimoto's thyroiditis

Source: https://pmc.ncbi.nlm.nih.gov/articles/PMC9862303/

Four studies, involving a total of 463 participants, were included in this review. The risk of bias in these studies was assessed as ranging from unclear to high, raising concerns about the reliability of the findings. The mean duration of the included studies was 7.5 months, with study lengths varying between 3 and 18 months. Notably, none of the studies addressed the primary outcome of this review, which was to evaluate the impact of selenium supplementation on health-related quality of life (HRQoL). This is a critical limitation, as HRQoL is a meaningful indicator of the overall effectiveness of therapeutic interventions for individuals with Hashimoto's thyroiditis (HT). Furthermore, two important secondary outcomes—changes from baseline in levothyroxine (LT4) replacement dosage at the conclusion of the study and associated economic costs—were not evaluated in any of the included studies, leaving significant gaps in the evidence.[2]

One of the studies, which was deemed to have a high risk of bias, reported a statistically significant improvement in subjective well-being among participants who received a combination of 200 µg of sodium selenite and LT4 compared to those receiving a placebo with LT4. Specifically, 14 out of 18 participants in the sodium selenite group reported improved

well-being, compared to only 3 out of 18 in the placebo group. While this suggests a potential benefit of sodium selenite supplementation for enhancing subjective well-being, the high risk of bias associated with this study warrants caution in interpreting these results.[1]

Selenomethionine supplementation (200 µg daily) was examined in three studies, and all reported significant reductions in serum levels of anti-thyroid peroxidase antibodies (TPOAb) compared to placebo. The reductions in TPOAb levels were statistically significant, which indicates that selenium supplementation may modulate the immune response in HT. However, the clinical relevance of these reductions remains unclear, as no evidence was provided to demonstrate that lower TPOAb levels translated into meaningful improvements in thyroid function, symptom relief, or long-term outcomes for patients.[6]

Adverse events were reported in two of the included studies. Importantly, selenium supplementation did not appear to result in a statistically significant increase in the frequency of adverse events compared to placebo. In the studies evaluating selenomethionine combined with LT4, one adverse event was reported in each selenium group, while no adverse events occurred in the corresponding control groups. [7] The reported adverse events were mild and did not necessitate withdrawal from the studies. While these findings suggest that selenium supplementation is generally well-tolerated, the limited data on adverse events preclude definitive conclusions regarding its safety.[8]

Outcome	Included cohorts	No. of participants	Pooled effect estimate [CI]	Heterogeneity I^2 (%)	Publication bias		Quality of evidence
					Egger's (p)	Rank's (p)	
Thyroid function (effect estimate reported as SMD)							
TSH all	26	2063	−0.21 [−0.43 to 0.01]	59	0.71	0.76	Moderate
Without THRT	7	869	**−0.21 [−0.41 to −0.02]**	0	0.76	0.76	—
fT4 all	21	1664	0.05 [−0.15 to 0.25]	33	0.11	0.46	Moderate
Without THRT	7	623	0.16 [−0.06 to 0.39]	0	0.82	0.88	—
fT3 all	11	658	0.51 [−0.11 to 1.13]	84	0.83	0.94	Very low
Without THRT	β	239	1.01 [−0.60 to 2.63]	95	0.24	1.00	—
T4	3	187	−0.02 [−0.42 to 0.39]	0	—	—	—
T3	4	252	−0.11 [−0.48 to 0.26]	0	—	—	—
Thyroid antibodies (effect estimate reported as SMD)							
TPOAb	29	2358	**−0.96 [−1.36 to −0.56]**	90	0.24	**0.04**	Low
TGAb	17	1283	−0.27 [−0.59 to 0.06]	74	0.52	0.71	Low
AEs (effect estimate reported as OR)							
	16	1339	0.89 [0.46 to 1.75]	0	0.22	0.08	Moderate
Ultrasound findings (effect estimate reported as SMD)							
Thyroid volume	4	182	−0.14 [−0.57 to 0.28]	0	—	—	—
Immune markers (effect estimate reported as SMD)							
IL-2	3	189	−0.68 [−1.44 to 0.09]	59	—	—	—
IL-10	3	189	0.20 [−0.21 to 0.61]	0	—	—	—
MDA	3	248	−1.16 [−2.29 to −0.03]	85	—	—	—

Bold values indicate significant results.
CI, confidence interval; OR, odds ratio; SMD, standardized mean difference.

Figure 2 A Systematic Review and Meta-Analysis.
Source: *A Systematic Review and Meta-Analysis of Randomized Clinical Trials*
Source: https://pubmed.ncbi.nlm.nih.gov/38243784/

- **Health-Related Quality of Life**

  A key limitation identified across the reviewed studies was the absence of data evaluating health-related quality of life. This omission highlights a critical gap in understanding the broader, patient-centered impacts of selenium supplementation on individuals with Hashimoto's Thyroiditis (HT). [4-7]

- **Reduction in Anti-Thyroid Peroxidase Antibodies**

  Selenium supplementation resulted in statistically significant reductions in anti-thyroid peroxidase antibody (TPOAb) levels in several studies. While this finding suggests a potential role for selenium in modulating autoimmune activity in HT, the clinical relevance of these reductions remains uncertain. The lack of long-term follow-up and inconsistent reporting of outcomes limit the ability to determine whether these changes translate into meaningful clinical benefits.[8]

- **Levothyroxine Replacement Dosage and Economic Costs**

  No significant impact of selenium supplementation was observed on levothyroxine (LT4) replacement dosages, indicating it may not reduce dependency on hormone therapy in HT patients. Additionally, none of the studies evaluated the economic costs

associated with selenium use, leaving questions regarding its cost-effectiveness unanswered.[9]

- **Adverse Events and Safety Profile**

  Adverse events associated with selenium supplementation were rare and not significantly different from those reported in placebo groups. This favorable safety profile supports the continued exploration of selenium as a potential adjunct therapy in HT management, provided its clinical benefits are clarified in future studies.[4-7]

- **Overall Implications**

  These findings emphasize the need for further robust research to investigate the clinical relevance of TPOAb reductions, assess patient-centered outcomes such as quality of life, and evaluate the long-term impacts of selenium supplementation in HT treatment.[6]

Vitamin D supplementation in Hashimoto treatment

Another study conducted by Department of Endocrinology and Diabetology, Collegium Medicum in Bydgoszcz explored the relationship between vitamin D levels and Hashimoto's thyroiditis (HT) in a cohort of 370 female participants divided into three groups: 125 healthy individuals, 111 with hypothyroidism, and 134 with HT. The primary objective was to determine differences in vitamin D levels between these groups and examine correlations between vitamin D concentrations, thyroid-stimulating hormone (TSH), and anti-thyroid antibodies (anti-TPO and anti-TG). Results showed that vitamin D levels were significantly lower in patients with HT (mean 27.01 ± 14.4 ng/mL) compared to those with hypothyroidism (31.15 ± 10.6 ng/mL) and healthy individuals (34.38 ± 12.6 ng/mL). This difference was statistically significant ($p < 0.001$), highlighting a potential association between vitamin D deficiency and autoimmune thyroid diseases.[10]

A strong negative correlation was observed between vitamin D levels and TSH in all subgroups. Higher TSH levels were associated with lower vitamin D concentrations, emphasizing a possible link between thyroid dysfunction and impaired vitamin D metabolism or status. Additionally, weak negative correlations were found between vitamin D levels and anti-thyroid antibodies (anti-TPO and anti-TG) in women with HT across all categories of vitamin D status: deficient (< 20 ng/mL), insufficient (20–30 ng/mL), and sufficient (> 30 ng/mL). [14] This

suggests that lower vitamin D levels may contribute to the autoimmune processes driving HT by influencing antibody production.[]

Notably, vitamin D deficiency (< 20 ng/mL) or insufficiency (20–29.9 ng/mL) was prevalent in 61% of the overall study population, with 70% of the vitamin D-deficient individuals belonging to the hypothyroidism or HT subgroups.[10] Patients with HT exhibited a higher prevalence of vitamin D deficiency compared to those with hypothyroidism or healthy controls, supporting previous research indicating a relationship between autoimmune thyroid disorders and low vitamin D levels. These findings align with the hypothesis that vitamin D plays a role in immune regulation by modulating T-helper cell responses (Th1, Th2, and Th17) and enhancing immune tolerance, potentially mitigating the severity of autoimmune disorders.[13]

The study concludes that vitamin D deficiency is more common in patients with HT and hypothyroidism than in healthy individuals, suggesting a potential link between vitamin D and the pathophysiology of autoimmune thyroid diseases. The observed correlations between vitamin D, TSH, and anti-thyroid antibodies further highlight the importance of vitamin D as a potential modulator of autoimmune activity. [17] While these findings support the potential utility of vitamin D supplementation in HT management, the study emphasizes the need for further research to establish optimal dosages and long-term benefits, as well as to determine whether improving vitamin D status can effectively reduce thyroid antibody levels, enhance thyroid function, and improve clinical outcomes in HT patients.[10-18]

CONCLUSIONS

Selenium supplementation, particularly in the form of selenomethionine, has demonstrated significant potential in modulating the autoimmune activity of HT. Multiple studies showed a reduction in serum anti-thyroid peroxidase antibodies (TPOAb) levels, with notable reductions observed in patients treated with selenium alone or in combination with levothyroxine. These reductions suggest selenium's antioxidant properties may help mitigate thyroid gland inflammation by neutralizing hydrogen peroxide and other reactive oxygen species, which are implicated in thyroid damage. However, while reductions in TPOAb levels are statistically significant, their clinical relevance remains uncertain, as improvements in long-term thyroid function or quality of life outcomes were not consistently reported. Additionally, selenium supplementation did not significantly reduce the levothyroxine dosage required to maintain

euthyroidism, and its cost-effectiveness in HT management was not addressed. Despite these limitations, selenium demonstrated a favorable safety profile, with rare and comparable adverse event rates to placebo groups, supporting its tolerability as an adjunctive treatment.

Vitamin D, meanwhile, emerged as a critical modulator of thyroid autoimmunity. Patients with HT were found to have significantly lower vitamin D levels compared to healthy controls and patients with hypothyroidism alone. A strong negative correlation was observed between vitamin D levels and TSH, anti-TPO, and anti-thyroglobulin (anti-TG) antibodies, suggesting that vitamin D deficiency may exacerbate immune dysregulation and autoimmune activity in HT. This aligns with vitamin D's role in immune system modulation, particularly its ability to suppress pro-inflammatory T-helper 1 (Th1) and Th17 cell responses while promoting regulatory T-cell activity.[19-22] These immune-modulatory effects suggest that adequate vitamin D levels may help reduce autoimmune antibody production and thyroid inflammation in HT. Despite these promising findings, the optimal dosing strategies and long-term safety of vitamin D supplementation in HT patients remain areas requiring further investigation.[21]

In conclusion, selenium and vitamin D supplementation present complementary therapeutic avenues for managing Hashimoto's Thyroiditis by targeting different aspects of thyroid autoimmunity and oxidative stress. Selenium appears most beneficial for reducing antibody levels and protecting against oxidative damage, while vitamin D addresses immune dysregulation and deficiency-related exacerbations. Both interventions are generally well-tolerated and show promise as adjunctive therapies, though further well-designed, large-scale randomized controlled trials are needed to define optimal dosing, long-term effects, and their impact on clinical outcomes such as quality of life and thyroid function sustainability.[22]

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