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SELENIUM AND THYROID HEALTH: THE CASE OF HASHIMOTO'S THYROIDITIS

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Abstract. Hashimoto's thyroiditis (HT) is one of the most prevalent autoimmune thyroid disorders and the leading cause of hypothyroidism worldwide. In recent years, selenium has attracted significant attention due to its essential role in thyroid hormone metabolism and its potential immunomodulatory and antioxidant properties. Selenium-dependent enzymes, such as glutathione peroxidases and iodothyronine deiodinases, are crucial for thyroid hormone synthesis and protection of thyroid tissue from oxidative damage. Several clinical studies have demonstrated that selenium supplementation can reduce thyroid autoantibody levels, particularly thyroid peroxidase antibodies (TPOAb), and improve the overall well-being of patients with HT. However, the results remain inconsistent, with some trials showing no significant benefit. This paper reviews the physiological role of selenium in thyroid health, the potential mechanisms underlying its effects in HT, and current evidence from clinical studies, highlighting both the therapeutic potential and limitations of selenium supplementation in patients with autoimmune thyroiditis.
Keywords. Selenium; Hashimoto's thyroiditis; thyroid health; autoimmunity; antioxidants; thyroid peroxidase antibodies (TPOAb)

Introduction.

Hashimoto's thyroiditis (HT), first described by Hakaru Hashimoto in 1912, is a chronic autoimmune thyroid disorder and the most common cause of hypothyroidism in iodine-sufficient regions. It predominantly affects women, with a female-to-male ratio of nearly 10:1, and its prevalence has been increasing worldwide. The pathogenesis of HT involves a complex interplay between genetic susceptibility, environmental triggers, and dysregulation of the immune response, leading to gradual destruction of thyroid follicular cells and impaired hormone production.

Among the environmental and nutritional factors implicated in HT, micronutrients such as iodine and selenium play a crucial role in thyroid physiology. Selenium, an essential trace element, is incorporated into more than 25 selenoproteins, including glutathione peroxidases, thioredoxin reductases, and iodothyronine deiodinases. These enzymes are directly involved in thyroid hormone synthesis, conversion of thyroxine (T4) to triiodothyronine (T3), and protection of the gland from oxidative stress.

Recent evidence has highlighted the potential role of selenium in modulating autoimmune processes in HT. Several randomized controlled trials have demonstrated that selenium supplementation can significantly reduce thyroid peroxidase antibody (TPOAb) titers, improve sonographic features of the thyroid gland, and enhance patients' quality of life. Nevertheless, conflicting results have also been reported, and the therapeutic value of selenium remains a subject of ongoing debate.

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Given the global burden of autoimmune thyroid disorders and the increasing interest in micronutrient-based interventions, it is important to critically assess the evidence regarding selenium and its potential role in the management of HT. This paper aims to provide an overview of the physiological significance of selenium in thyroid health, summarize findings from clinical studies, and discuss the implications and limitations of selenium supplementation as an adjunctive therapy for Hashimoto's thyroiditis.

Literature Review

Selenium and Thyroid Physiology

The thyroid gland contains the highest selenium concentration per gram of tissue in the human body. This high demand is due to the crucial role of selenium-dependent enzymes in thyroid hormone metabolism and antioxidant defense. Iodothyronine deiodinases (types I, II, and III) regulate the activation and deactivation of thyroid hormones, whereas glutathione peroxidases and thioredoxin reductases protect thyroid cells from oxidative stress generated during hormone synthesis. Deficiency of selenium has been associated with impaired thyroid hormone metabolism, increased oxidative damage, and heightened susceptibility to autoimmune thyroid disease.

Selenium Status and Autoimmune Thyroiditis

Several epidemiological studies have suggested a link between low selenium status and increased prevalence of thyroid autoimmunity. A large European cross-sectional study demonstrated that populations with marginal selenium intake had a higher incidence of thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb), both of which are characteristic of Hashimoto's thyroiditis. Similarly, selenium deficiency regions showed higher rates of goiter and hypothyroidism compared to selenium-sufficient areas.

Clinical Trials on Selenium Supplementation

Numerous randomized controlled trials (RCTs) have investigated the effect of selenium supplementation in patients with HT:

- **Gärtner et al. (2002):** Reported a significant reduction in TPOAb titers after six months of supplementation with 200 µg/day sodium selenite.
- **Negro et al. (2007):** Found that selenium supplementation during pregnancy reduced the risk of postpartum thyroid dysfunction in women with thyroid autoantibodies.
- **Winther et al. (2020):** Conducted a systematic review and meta-analysis that confirmed modest but significant antibody reduction, although clinical benefits were less consistent.
- **Other RCTs:** Some studies failed to demonstrate substantial changes in antibody levels or improvement in thyroid function, raising concerns about patient heterogeneity, baseline selenium status, and differences in supplementation protocols.

Controversies and Limitations

Despite promising findings, the role of selenium in HT remains controversial. Key limitations include:

- Variability in study design, dosage, and duration of supplementation.
- Lack of standardized biomarkers for assessing selenium status.
- Potential influence of iodine intake and genetic polymorphisms on treatment response.
- Uncertainty regarding long-term benefits and safety of high-dose supplementation.

Overall, while evidence supports a potential immunomodulatory effect of selenium, current data are insufficient to establish universal recommendations for routine supplementation in all HT patients.

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Results

The review of existing literature highlights several important findings regarding the role of selenium in thyroid health and its potential therapeutic relevance in Hashimoto's thyroiditis (HT):

1. **Selenium deficiency and thyroid autoimmunity:**
   Populations with low selenium intake demonstrate a higher prevalence of thyroid autoantibodies, goiter, and hypothyroidism. This suggests that selenium insufficiency may contribute to the development or progression of autoimmune thyroiditis.
2. **Antibody reduction with selenium supplementation:**
   Multiple clinical studies have reported that selenium supplementation, typically at doses of 200 μg/day of sodium selenite or selenomethionine, can lead to a significant reduction in thyroid peroxidase antibody (TPOAb) levels. Some studies also found a decrease in thyroglobulin antibody (TgAb) concentrations.
3. **Improvement in quality of life and thyroid ultrasound features:**
   Beyond antibody reduction, several trials noted improvements in patients' well-being, mood, and ultrasound echogenicity of the thyroid gland. These findings suggest that selenium may influence not only biochemical but also clinical outcomes in HT.
4. **Heterogeneous results across studies:**
   Despite positive findings, not all studies reported consistent benefits. In some trials, selenium supplementation did not result in significant antibody reduction or improvement in thyroid function. Such discrepancies may be attributed to variations in baseline selenium status, iodine intake, dosage, duration of supplementation, and genetic factors among study populations.
5. **Lack of long-term evidence:**
   Most available studies have short intervention periods (3–12 months), limiting conclusions about long-term efficacy and safety. It remains unclear whether selenium supplementation can modify disease progression, prevent hypothyroidism, or sustain long-term clinical benefits.

Taken together, the findings indicate that selenium has a promising but not yet fully established role in the management of HT. While antibody reduction and symptomatic improvement are frequently observed, the heterogeneity of results underscores the need for personalized approaches and further large-scale, long-term studies.

Discussion

The present review highlights the potential role of selenium as an adjunctive therapy in Hashimoto's thyroiditis (HT). Findings from several clinical studies suggest that selenium supplementation can lower thyroid peroxidase antibody (TPOAb) titers, improve thyroid ultrasound characteristics, and enhance patient-reported outcomes such as mood and quality of life. These results are consistent with the biological plausibility of selenium's function: as a key component of selenoproteins, it contributes to both antioxidant defense and regulation of thyroid hormone metabolism.

However, the clinical translation of these findings is far from straightforward. One major limitation is the heterogeneity of results across trials. While some randomized controlled studies have shown significant antibody reduction and clinical improvement, others have reported little or no benefit. This discrepancy may be explained by several factors. First, baseline selenium status appears to be critical—patients from selenium-deficient regions are more likely to benefit from supplementation than those from selenium-replete populations.

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Second, variability in study design, including differences in dosage (typically 100–300 µg/day), supplementation form (selenite vs. selenomethionine), and intervention duration (ranging from 3 to 12 months), complicates the interpretation of outcomes.

Another challenge is the lack of standardized outcome measures. While antibody titers are commonly used as surrogate markers, they do not always correlate with clinical endpoints such as thyroid function or symptom relief. Moreover, few studies have investigated the long-term safety and efficacy of selenium supplementation. Although short-term interventions appear safe, prolonged intake of high selenium doses carries the risk of selenosis, a condition characterized by gastrointestinal disturbances, hair and nail brittleness, and neurological symptoms.

The interaction between selenium and iodine is also noteworthy. Excessive iodine intake has been shown to exacerbate autoimmune thyroiditis, whereas selenium may mitigate oxidative stress induced by iodine excess. This suggests a complex interplay between micronutrients that should be considered in both research and clinical practice.

Overall, while selenium supplementation holds promise as a supportive measure in HT, current evidence does not justify its universal recommendation for all patients. Instead, it may be most beneficial in carefully selected subgroups, particularly those with selenium deficiency, high antibody titers, or persistent symptoms despite conventional therapy. Future research should focus on large-scale, long-term randomized controlled trials with standardized protocols to clarify the role of selenium in preventing disease progression and improving clinical outcomes in HT.

Conclusion

Selenium plays a fundamental role in thyroid physiology through its incorporation into selenoproteins that regulate hormone metabolism and protect against oxidative stress. Evidence from clinical studies suggests that selenium supplementation may reduce thyroid autoantibody levels, improve ultrasound features, and enhance quality of life in patients with Hashimoto's thyroiditis (HT). These findings highlight its potential as an adjunctive therapy in the management of autoimmune thyroid disease.

Nevertheless, the data remain inconsistent, with some trials showing minimal or no benefit. The effectiveness of supplementation appears to depend on factors such as baseline selenium status, iodine intake, dosage, and genetic predisposition. Furthermore, the lack of long-term studies limits the ability to draw firm conclusions regarding its safety and sustained efficacy.

Therefore, while selenium supplementation should not yet be considered a universal treatment for HT, it may be useful in selected patients, particularly those with selenium deficiency. Future large-scale, randomized controlled trials are essential to determine optimal dosing strategies, identify responsive patient subgroups, and establish evidence-based guidelines.

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