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EFFECT OF SELENIUM AND ZINC CONCENTRATIONS ON THE PROGRESSION OF THYROID DISEASES: A REVIEW

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ANNOTATION: Thyroid diseases represent a significant medical problem in modern healthcare, as they affect the regulation of metabolism and the overall state of the body. Among them, hypothyroidism, hyperthyroidism, and autoimmune thyroid disorders are increasingly prevalent worldwide, leading to serious health consequences. Disruption of thyroid function may result in metabolic and mental disorders, as well as other severe complications.

One of the important factors influencing thyroid health is the deficiency of trace elements. Special attention is paid to selenium and zinc, which play a key role in thyroid hormone synthesis, antioxidant protection, and immune system regulation. Deficiency of these trace elements may contribute to the pathogenesis and progression of thyroid diseases.
The purpose of this review is to analyze the current literature on the effects of selenium and zinc concentrations on thyroid function and the development of autoimmune thyroid diseases. Summarizing available data allows us to better understand the role of these trace elements in the etiology and possible treatment of thyroid pathologies, as well as to identify directions for further research and clinical application.
Key words: selenium, zinc, thyroid gland, autoimmune thyroiditis, deficiency, pathogenesis.

ВЛИЯНИЕ КОНЦЕНТРАЦИИ СЕЛЕНА И ЦИНКА НА ПРОГРЕССИРОВАНИЕ ЗАБОЛЕВАНИЙ ЩИТОВИДНОЙ ЖЕЛЕЗЫ: ОБЗОР

АННОТАЦИЯ: Заболевания щитовидной железы представляют собой значимую медицинскую проблему современного здравоохранения, поскольку они влияют на регуляцию обмена веществ и общее состояние организма. Среди них гипотиреоз, гипертиреоз и аутоиммунные заболевания щитовидной железы, которые получают все большее распространение во всем мире, приводя к серьезным последствиям для здоровья. Нарушение функции щитовидной железы может привести к метаболическим и психическим расстройствам, а также к другим серьезным осложнениям. Одним из важных факторов, влияющих на здоровье щитовидной железы, является дефицит микроэлементов. Особое внимание уделяется селену и цинку, которые играют ключевую роль в синтезе тиреоидных гормонов, антиоксидантной защите и регуляции иммунной системы. Дефицит этих микроэлементов может способствовать патогенезу и прогрессированию заболеваний щитовидной железы. Целью данного обзора является анализ современной литературы о влиянии концентраций селена и цинка на функцию щитовидной железы и развитие аутоиммунных заболеваний щитовидной железы. Обобщение имеющихся данных позволяет лучше понять роль этих микроэлементов в этиологии и возможностях лечения тиреоидной патологии, а также определить направления дальнейших исследований и клинического применения.

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Ключевые слова: селен, цинк, щитовидная железа, аутоиммунный тиреоидит, дефицит, патогенез.

RELEVANCE: The thyroid gland plays a key role in regulating the body's metabolism and overall health. Diseases of this gland, such as hypothyroidism, hyperthyroidism, autoimmune diseases, are becoming more common in the modern world, presenting a serious medical problem. Interfering with the functioning of the thyroid gland can lead to various metabolic disorders, mental disorders, and other serious health consequences.

There are many factors that can affect thyroid health, including deficiencies in various trace elements and vitamins. Among these microelements, special attention is drawn to selenium and iodine, as well as vitamin D, which play an important role in the functioning of the thyroid gland and have the potential to influence the development and course of its diseases.

Selenium (Se), discovered by Berzelius in 1817, is essential for thyroid hormone metabolism. Three isoforms of iodothyronine deiodinases are selenoenzymes, responsible for converting thyroxine (T4) into biologically active triiodothyronine (T3) [7–10]. In thyroid tissue, Se-dependent glutathione peroxidases (GPx) also participate in the neutralization of hydrogen peroxide, thus protecting thyrocytes from oxidative stress [4–6]. Selenium deficiency disrupts these processes and contributes to thyroid dysfunction.

MATERIALS AND METHODS: This review was conducted using a systematic approach to identify, select, and analyze scientific literature related to selenium and zinc metabolism and their impact on thyroid function. Peer-reviewed articles published between 1995 and 2024 were retrieved from PubMed, Scopus, Web of Science, and Google Scholar using keywords such as “selenium,” “zinc,” “thyroid disease,” “autoimmune thyroiditis,” “micronutrient deficiency,” and “thyroid hormones.” Additional regional data from Uzbekistan, including clinical reports and epidemiological studies, were incorporated to provide context for micronutrient deficiencies in local populations. Studies involving human subjects, experimental models, or biochemical analyses of deiodinase and antioxidant enzyme activity were included. Publications lacking methodological clarity or presenting conflicting biochemical data were excluded. Extracted findings were synthesized narratively, focusing on the relationship between selenium and zinc concentrations, thyroid hormone metabolism, oxidative stress modulation, and autoimmune mechanisms. Special attention was given to interventional studies assessing supplementation outcomes and observational studies evaluating correlations between micronutrient levels and thyroid dysfunction.

RESULTS AND DISCUSSION: Zinc is likewise crucial for thyroid hormone synthesis and metabolism. It ensures the proper functioning of deiodinases, stabilizes the binding of T3 to nuclear receptors, and regulates transcription through zinc-finger proteins [20–22]. Zinc deficiency suppresses thyroid hormone synthesis and may lead to hypothyroidism, while hypothyroidism itself can worsen zinc deficiency, creating a vicious cycle [23].

Both selenium and zinc are important modulators of the immune response. Selenium enhances the activity of antioxidant enzymes, supports vitamin E function, and contributes to the regulation of inflammatory processes. Selenium deficiency is associated with impaired immune function, while supplementation stimulates lymphocyte proliferation and NK cell activity [14–17].

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Zinc is critical for both innate and adaptive immunity. Its deficiency reduces the activity of macrophages, neutrophils, and NK cells, suppresses cytokine production, and impairs phagocytosis. It also negatively affects T and B lymphocyte proliferation, shifting the immune balance from Th1 to Th2 responses, thereby increasing susceptibility to infections [Prasad, 2009; Overbeck et al., 2008].

Several clinical trials confirm the beneficial effects of selenium supplementation in patients with autoimmune thyroiditis and Graves' disease. In placebo-controlled studies, daily administration of 200 µg selenium (as sodium selenite or selenomethionine) reduced anti-thyroid peroxidase antibody levels and improved patients' well-being. Selenium therapy also increased GPx activity in plasma [11–13].

Zinc deficiency has been shown to correlate with thyroid dysfunction. Urunbaev et al. reported that only 20% of patients had normal thyroid function, while 55% had subclinical and 25% manifest hypothyroidism. Zinc deficiency was observed in 80% of patients, and lower zinc levels correlated with higher TSH, reduced free T4, and elevated autoimmune antibody titers (AT-TPO, AT-TG) [25, 27, 29]. Zinc supplementation in such patients helped restore thyroid hormone levels.

The metabolism of selenium and zinc is closely linked to other micronutrients. Deficiencies in iodine, iron, selenium, and zinc often coexist, especially in populations with plant-based diets or malabsorption syndromes. Since selenium and zinc are required for the conversion of T4 to T3, and iron is necessary for hormone synthesis, combined deficiencies reduce the effectiveness of iodine supplementation [23, 25].

Table 1. Comparison of Thyroid Function, Zinc Levels, and Correlations Across Patient Groups

Parameter	Euthyroid (n=8)	Subclinical Hypothyroidism (n=22)	Manifest Hypothyroidism (n=10)	Control Group
Percentage of patients	20%	55%	25%	–
TSH level (mU/l)	Normal	7.5 ± 1.5*	17.5 ± 2.3*	2.3 ± 1.1
Relative change in TSH	–	↑ 70%*	↑ 7.6-fold*	–
Blood zinc levels	Normal	Reduced (80% of cases)	Reduced (80% of cases)	Normal
Thyroid	Normal	↑ 48% vs.	↑ 35% vs. control	Normal

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volume		control		
Correlation: zinc vs. TSH	–	r = –0.60*	r = –0.60*	–
Correlation: zinc vs. free T4	–	r = –0.49*	r = –0.49*	–
Correlation: zinc vs. AT-TPO	–	r = –0.89***	r = –0.89***	–
Correlation: zinc vs. AT-TG	–	r = –0.76**	r = –0.76**	–

There is evidence that zinc deficiency may be involved in the pathogenesis of immunological disorders in autoimmune thyroiditis [19,23,23]

The role of selenium (Se) and zinc (Zn) in thyroid physiology is well established, but the available evidence shows both consistencies and contradictions.

Several studies clearly demonstrate that selenium supplementation reduces thyroid peroxidase antibodies in autoimmune thyroiditis and improves patient-reported outcomes [11–13]. However, these effects were most pronounced in European regions with selenium deficiency, whereas trials in selenium-replete populations showed weaker or no benefit. This raises the question of whether supplementation is universally effective or only beneficial in populations with proven deficiency.

In contrast, the role of zinc has been less extensively studied. Experimental and clinical data indicate that zinc deficiency correlates with higher TSH levels and the presence of autoantibodies [25, 27, 29]. Still, the number of controlled interventional studies is limited, and most evidence comes from observational or small-scale studies. This limits the ability to draw firm conclusions about causal relationships.

Both selenium and zinc influence not only thyroid hormone metabolism but also immune system regulation. Yet the interaction between these trace elements remains poorly defined. For instance, selenium deficiency is known to impair deiodinase activity, while zinc affects transcriptional regulation and receptor binding. Their combined deficiency may produce additive or synergistic effects, but this hypothesis has not been rigorously tested in clinical trials. Moreover, coexisting deficiencies in iodine and iron may confound the independent contributions of selenium and zinc, complicating the interpretation of current data [23, 25].

Another limitation of current research is the short duration of most interventional trials (3–6 months). Long-term outcomes, including relapse rates in autoimmune thyroiditis or prevention of

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hypothyroidism progression, remain unclear. Additionally, most studies include heterogeneous patient populations, with variations in age, comorbidities, and baseline nutritional status, which may partly explain inconsistent findings across different cohorts.

Taken together, the existing evidence supports the biological plausibility and potential clinical benefit of selenium and zinc supplementation in thyroid disorders, particularly autoimmune thyroiditis. However, more robust randomized controlled trials are required to determine:

1. the optimal dosage and duration of supplementation,
2. the specific subgroups of patients who benefit the most, and
3. the long-term safety and effectiveness of such interventions.

Future research should also investigate the combined impact of multiple micronutrient deficiencies and explore whether individualized supplementation strategies (guided by baseline nutritional status) provide better outcomes than generalized recommendations.

CONCLUSIONS: Deficiencies of selenium and zinc play a significant role in the pathogenesis of thyroid diseases, particularly autoimmune thyroiditis (AIT). These trace elements influence both the synthesis and metabolism of thyroid hormones and the regulation of immune responses.

Selenium and zinc demonstrate antioxidant and immunomodulatory properties that contribute to reducing inflammatory processes in thyroid tissues. Clinical evidence suggests that supplementation may be especially beneficial in patients with active autoimmune disease or proven micronutrient deficiency.

Despite encouraging findings, current data remain heterogeneous and limited in scale. The variability of results across populations indicates the need for individualized approaches, considering baseline nutritional status and coexisting deficiencies (e.g., iodine or iron).

Regular monitoring of selenium and zinc status, followed by targeted correction, represents a promising clinical strategy for improving thyroid function and patient outcomes. However, further large-scale, long-term randomized controlled trials are necessary to determine optimal supplementation regimens and to clarify which patient groups benefit the most.

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