International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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ARTICLE TITLE	THE ROLE OF MICRONUTRIENTS AND SUPPLEMENTS IN HYPOTHYROIDISM MANAGEMENT: A LITERATURE REVIEW
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4434
RECEIVED	03 October 2025
ACCEPTED	18 December 2025
PUBLISHED	26 December 2025
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THE ROLE OF MICRONUTRIENTS AND SUPPLEMENTS IN HYPOTHYROIDISM MANAGEMENT: A LITERATURE REVIEW

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ABSTRACT

Background: Despite standard levothyroxine (LT4) therapy, many patients with hypothyroidism continue to report persistent symptoms such as fatigue, cognitive impairment, and reduced quality of life. This has prompted growing interest in adjunctive therapies, particularly micronutrients and supplements, which may modulate immune function and thyroid gland metabolism.
Aim: To evaluate the clinical impact of vitamin D, selenium, zinc, myo-inositol, and synbiotic supplementation in hypothyroid patients—especially those with autoimmune thyroiditis and subclinical hypothyroidism—by analyzing their effects on thyroid function, antibody titers, and patient-reported outcomes.
Materials and methods: A systematic literature search was conducted predominantly in PubMed and related biomedical databases. The review included randomized controlled trials (RCTs), systematic reviews, and relevant observational studies published between 2015 and 2025. Studies were analyzed for their findings on selected micronutrients and their influence on biochemical markers (TSH, FT4, FT3, anti-TPO, anti-Tg), immune modulation, and quality of life parameters.
Results: Adjunctive supplementation with micronutrients such as selenium, vitamin D, zinc, myo-inositol, and synbiotics shows potential clinical benefits in hypothyroidism management. These include reductions in thyroid autoantibody levels, improvements in thyroid hormone profiles, and better patient-reported outcomes. Synergistic effects were noted particularly for combined regimens, though results varied by baseline nutrient status and study design.
Conclusions: Supplementation with selected micronutrients may provide meaningful support in managing hypothyroidism, particularly in patients with residual symptoms despite LT4 therapy. However, larger multi-center RCTs are needed to confirm efficacy, establish optimal dosing, and identify patient subgroups most likely to benefit.

KEYWORDS

Hypothyroidism, Autoimmune Thyroiditis, Levothyroxine Resistance, Micronutrient Supplementation

CITATION

Zuzanna Wróbel, Katarzyna Torbacka, Joanna Kaczor, Maja Torbacka, Katarzyna Rozkosz, Natalia Wróbel, Olga Jakubik, Patrycja Podlejska, Aleksandra Sosin, Wojciech Bednarz. (2025) The Role of Micronutrients and Supplements in Hypothyroidism Management: A Literature Review. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4434

Introduction

Hypothyroidism is an endocrine disorder resulting from insufficient production of thyroxine due to diminished thyroid gland function. Its reported prevalence varies from 0% to 4% in the general population, 4% to 8% in the United States, and approximately 4% to 7% in Europe [1].

Clinical manifestations of hypothyroidism include weight gain, fatigue, difficulty focusing, cognitive dysfunction, depression, and menstrual irregularities. When left untreated or inadequately treated, it can contribute to cardiovascular complications and increased mortality [2].

The prevalence of diagnosing hypothyroidism is rising, partly due to increased screening, which allows for earlier intervention in subclinical cases [3].

Nonetheless, many patients persist in experiencing symptoms such as fatigue, reduced motivation, and depressive feelings, despite standard levothyroxine (LT4) therapy. This observation suggests a need to investigate factors beyond levothyroxine dosage optimization. As hypothyroidism is a globally widespread issue, exploring treatment possibilities that enhance patients' quality of life may have significant global benefits.

Despite alternative regimens, including levothyroxine-liothyronine combinations or desiccated thyroid extract, current evidence does not show a substantial improvement in patient-reported outcomes or quality of life [4].

Moreover, the absence of established guidelines supporting the efficacy of vitamin D, selenium, zinc, myo-inositol, or symbiotic supplementation in hypothyroidism results in limited consensus among endocrinologists regarding their use. This review aims to compile and critically assess scientific evidence on vitamin D, selenium, zinc, myo-inositol, and symbiotic supplementation for improving clinical outcomes and alleviating the symptoms in hypothyroid patients.

A large-scale study published in *The Journal of Clinical Endocrinology and Metabolism* (2016) by Peterson, McAninch, and Bianco examined 9,981 individuals with normal TSH levels, including 469 participants undergoing LT4 therapy. They were found to be less physically active, had higher BMIs despite lower caloric intake, and more frequently used antidepressants compared to those without thyroid disease. Crucially, the study showed that patients with normal serum TSH levels on LT4 monotherapy had lower T3:T4 ratios than healthy controls, indicating that factors beyond serum thyroid hormone levels may affect bioavailability, potentially driving persistent symptoms and cardiometabolic risks [5].

These observations align with anecdotal patient reports of lower energy levels and reduced well-being despite normalized TSH values, suggesting additional physiological or immunological pathways may be involved.

Given the ongoing debate about micronutrient and supplement effectiveness, this review aims to assess whether deficiencies in vitamin D, selenium, zinc, myo-inositol, or a disrupted gut microbiota (addressed through synbiotics) could explain the persistently diminished quality of life in certain hypothyroid patients, even when their TSH levels fall within the normal range. Quality of life in hypothyroid patients is multidimensional, encompassing physical, emotional, and social aspects [6]. A holistic approach to hypothyroidism management thus emphasizes improving daily functioning and life satisfaction rather than focusing solely on normalizing laboratory parameters. By examining the existing body of literature, this review seeks to evaluate whether these adjunctive therapies offer a promising avenue for enhancing clinical outcomes in treatment of hypothyroidism.

Materials and Methods

A systematic literature search was performed in PubMed and other biomedical databases, guided by the references provided (1-33). Keywords included “hypothyroidism,” “autoimmune thyroiditis,” “selenium,” “vitamin D,” “myo-inositol,” “symbiotics,” “zinc,” and “micronutrient supplementation.” Studies were selected based on their relevance to thyroid function, autoimmunity, and clinical outcomes in hypothyroidism. Priority was given to studies published between 2015 and 2025 to ensure a current perspective. We selected randomized controlled trials (RCTs) and observational studies, although other sources were included when they offered unique insights. Extracted data covered study design, population characteristics, interventions, outcome measures (e.g., TSH, free T4, free T3, thyroid antibodies), and markers of patient-reported well-being (fatigue scores, quality of life). Emphasis was placed on understanding both the physiological mechanisms and practical applications of supplementation in hypothyroid management.

Results

Selenium Studies. Prevalence.

Although the European Thyroid Association recommends selenium supplementation for patients with mild Graves’ orbitopathy, there are currently no established guidelines regarding the use of selenium in hypothyroidism or Hashimoto’s thyroiditis. A survey on selenium supplementation practices in autoimmune thyroid disease, published in February 2023, reported that only 6% of Portuguese Medical Association endocrinologists frequently prescribe selenium for Hashimoto’s thyroiditis, while 30% do so occasionally and 46% never recommend it [7].

Does Selenium Supplementation Affect Thyroid Function in Healthy Individuals?

The thyroid gland contains the highest concentration of selenium relative to its weight compared to other organs in the human body [8]. A 2015 double-blinded randomized controlled trial (RCT) was conducted among a Danish population without known thyroid diseases. In total, 491 participants (men and women aged 60–74) were randomized into four groups: three different dosages of selenium-enriched yeast and one group receiving a placebo yeast tablet. The findings revealed that selenium supplementation, in a dose-dependent manner, affected thyroid function, as evidenced by decreased serum TSH and free thyroxine (FT4) levels in a population otherwise free of thyroid conditions [9].

Selenium in Hashimoto’s Hypothyroidism: Beneficially Upregulating Activated Treg Cells.

Existing literature suggests that low selenium levels correlate with impaired immune function, and even a moderate deficiency may promote the onset or progression of thyroid autoimmunity [10].

Some studies indicate that selenium supplementation helps restore euthyroidism in approximately one-third of patients with autoimmune thyroiditis and mild subclinical hypothyroidism (TSH < 10 mU/L) [11] [12]. One proposed mechanism involves selenium-driven enhancement of antioxidant activity and the upregulation of activated regulatory T (Treg) cells, which may contribute to the reduction of thyroid autoantibody levels and improve thyroid function. Although evidence points to a potential decrease in TSH and thyroid antibodies, further research is needed to confirm these correlations [13] [14].

Selenium Lowering Anti-TPO in Hypothyroid Children and Adolescents.

A study involving 71 children and adolescents has demonstrated a reduction in anti-thyroglobulin (anti-Tg) antibody levels in the selenium-supplemented group compared to placebo. Participants had symptomatic autoimmune thyroiditis, defined by anti-TPO and/or anti-Tg ≥60 IU/mL, as well as either euthyroidism or hypothyroidism (on levothyroxine treatment) or a goiter detected on thyroid ultrasound [15].

Another RCT published in April 2015 recruited patients from the Endocrinology and Metabolism Unit at the Hospital das Clínicas, University of São Paulo Medical School. Participants exhibited elevated anti-TPO levels (>100 IU/mL), normal or elevated anti-Tg levels (>100 IU/mL), thyroid hypoechogenicity, normal or elevated TSH, and normal FT4. The aim of this study was to define why do individuals with chronic autoimmune thyroiditis and hypothyroidism respond differently to Se supplementation. Patients were stratified by GPx1 polymorphisms, and outcomes included plasma selenium and GPx1 activity levels, anti-TPO titers, and ultrasound-based assessments of thyroid echogenicity. Although no correlation was found between GPx1 genotype and thyroid-related responses to selenium, the supplementation group showed decreased anti-TPO titers and increased thyroid vascularization without a rise in inflammatory markers [16].

SERENA Study in Pregnant Women.

The SERENA study aimed to evaluate whether selenium supplementation could offer a protective effect against thyroid autoimmunity during and after pregnancy. The findings indicated a significant reduction in anti-Tg and anti-TPO autoantibodies among women who received selenium. However, no differences were noted in thyroid gland volume or echogenicity, nor did selenium impact quality of life or maternal-fetal complications [17]. An additional RCT among pregnant women in the UK, all with mild-to-moderate iodine deficiency, investigated low-dose selenium's effects on thyroid autoimmunity and function. Although the sample size (25 participants) was relatively small, the findings suggest that selenium does not significantly affect TPO antibody concentrations but may influence thyroid function in women who test positive for thyroid autoantibodies [18]. Larger-scale studies are needed to clarify selenium's role in thyroid regulation during pregnancy.

Myo-Inositol and Selenium Studies.

Inositol, a carbocyclic polyol, is central to the TSH signaling pathway through its regulation of hydrogen peroxide–dependent iodination processes. A clinical study compared selenium supplementation alone to a combination of myo-inositol (MI) and selenium in patients with autoimmune thyroiditis and subclinical hypothyroidism. After six months, those receiving MI combined with Se displayed significantly lower TSH, anti-thyroid peroxidase (TPOAb), and anti-thyroglobulin (TgAb) levels. They also showed a marked increase in free T4 and improved quality of life, along with a notable decrease in the inferior thyroid artery's peak systolic velocity -indicative of reduced thyroid inflammation [19].

Zinc and Selenium Studies.

Zinc is a vital trace element that supports numerous enzymatic processes, including those related to oxidative stress control. It is also necessary for the activity of type 1 and type 2 deiodinases, which convert T4 to the biologically active hormone T3. Moreover, zinc assists in converting pre-pro thyrotropin-releasing hormone (TRH) into its active form through a carboxypeptidase enzyme. Proper T3 binding to nuclear receptors likewise depends on adequate zinc status [20]. Several RCTs have examined zinc and selenium co-supplementation in hypothyroidism. In one such trial, "Effects of Zinc and Selenium Supplementation on Thyroid Function in Overweight and Obese Hypothyroid Female Patients: A Randomized Double-Blind Controlled Trial," participants received one, both, or neither of these trace elements. Notably, FT3 levels rose in both the Zinc plus Se and Zinc-only groups, while FT4 rose and TSH fell significantly only in the Zinc plus Se group. The study concludes that while zinc alone positively influenced certain thyroid-related parameters, combined supplementation with zinc and selenium yielded the most pronounced benefits [21].

A recent case report titled "Thyroid dysfunction due to trace element deficiency—not only selenium but also zinc" underscores this synergy. In a 50-year-old patient on parenteral nutrition, a combination of selenium and zinc supplementation normalized previously abnormal thyroid function tests. At the onset of treatment, the patient exhibited elevated TSH and FT4 levels, along with a low FT3 level. Following the initiation of selenium supplementation, FT3 levels increased, and FT4 levels decreased to within the normal range. By adding zinc supplementation to the treatment plan, the doctors successfully normalized the patient's thyroid function parameters [22].

Zinc Studies.

While the studies mentioned above demonstrated the beneficial effects of zinc and selenium co-supplementation in patients with hypothyroidism, a study published in 2024 examined the effects of 25 mg of zinc alone in children and adolescents with autoimmune thyroid disease, 92.5% of whom were hypothyroid at enrollment. Over 12 weeks, investigators measured TSH, FT3, FT4, thyroid autoantibodies (TPOAb, TgAb), and oxidative stress markers. No significant changes emerged in thyroid function tests, antibody titers, or oxidative stress markers in the zinc group. Interestingly, however, levothyroxine dosage requirements significantly increased in the placebo group but not in the zinc group, raising questions about whether long-term zinc supplementation could offer more substantial clinical advantages [23].

Another 2021 study evaluated the effects of combined zinc, magnesium, and vitamin A supplementation on thyroid function, oxidative stress, and hs-CRP in hypothyroid patients. After 10 weeks, the intervention group showed a reduction in body weight and BMI relative to controls, suggesting possible adjunctive benefits for weight management [20].

Vitamin D Studies.

Vitamin D, particularly in its active form (1,25-dihydroxyvitamin D_3), exerts notable immunomodulatory effects relevant to hypothyroidism management. It can suppress Th1-mediated immune activity, inhibit B cell function (including antibody production), and promote regulatory B cells that secrete IL-10 and TGF-β. Those mechanisms may account for reductions in thyroid autoantibodies observed in recent studies [24]. A prospective RCT published in 2023 investigated cholecalciferol supplementation in newly diagnosed hypothyroid patients with vitamin D deficiency over an eight-week period. Results showed an inverse correlation between vitamin D levels and anti-TPO antibody titers. A notable decrease in serum TSH was also evident in the intervention group. Despite the limitations of this study, including the inability to assess the effects of vitamin D on serum TSH and thyroid hormone levels, as well as the exclusive inclusion of patients with severe vitamin D deficiency, it highlights an important hypothesis: vitamin D supplementation may, in some cases, eliminate the need for levothyroxine treatment in early-stage Hashimoto's thyroiditis [25]. A broader 2019 RCT examined whether vitamin D supplementation modifies anti-TPO Ab, anti-Tg Ab, and thyroid function parameters (TSH, T3, T4) in women with Hashimoto's thyroiditis. The most prominent effects included a reduction in anti-Tg Ab and TSH; however, neither anti-TPO Ab nor T3 or T4 levels changed significantly in the intervention group compared to placebo. Although these findings suggest potential benefits of vitamin D for hypothyroid patients, overall impacts on key thyroid function parameters remain inconclusive [26].

A double-blind randomized controlled trial (RCT) entitled *"Alterations in $CD4^+$ T-Cell Cytokine Profile in Female Patients with Hashimoto's Thyroiditis Following Vitamin D Supplementation"* investigated the immunomodulatory effects of vitamin D in Hashimoto's thyroiditis. During the three-month intervention, vitamin D supplementation produced a marked rise in serum 25-hydroxyvitamin D, increased expression of the transcription factor GATA3, and up-regulated transforming growth factor-β (TGF-β) gene expression. The *TGFB1* gene, which encodes transforming growth factor-beta 1 (TGF-β1), plays a key role in genetic susceptibility to autoimmune disorders, including Hashimoto's thyroiditis [27]. The intervention group also showed a significant reduction in the pro-inflammatory cytokine interleukin-17 (IL-17) compared with placebo [28]. Collectively, these findings suggest that vitamin D shifts the immune milieu toward a more regulatory profile, potentially mitigating thyroid autoimmunity. A 2023 RCT on obese women with subclinical hypothyroidism found that vitamin D supplementation did not markedly alter thyroid hormone levels but notably decreased TSH. Additional benefits included lower total cholesterol, increased irisin levels, and favorable changes in body composition after 12 weeks [29]. In another three-month study, vitamin D supplementation in women with Hashimoto's thyroiditis substantially reduced the Th17/Tr1 cell ratio, suggesting a shift toward a more regulatory immune state [30]. Additionally, a comparative trial examining the synergistic effects of vitamin D and selenium in euthyroid women with Hashimoto's thyroiditis and vitamin D deficiency found that combined treatment lowered thyroid autoantibody levels more effectively than vitamin D alone -particularly in patients with highly elevated autoantibody titers [31].

Synbiotic Intervention studies.

The intestinal microbiota is implicated in thyroid hormone homeostasis, including the enterohepatic circulation of thyroid hormones, iodothyronine metabolism, and levothyroxine absorption. Bacterial enzymes such as β-glucuronidases and sulfatases can inactivate thyroid hormones in the liver, while type 2 deiodinase in the intestinal wall may bind T3 and T4, affecting deiodinase activity. Modulating gut microbial composition via probiotics could therefore enhance levothyroxine bioavailability [32].

Synbiotic supplementation combines probiotics and prebiotics to provide a synergistic effect on gut microbiota. A recent 8-week trial explored whether improving intestinal flora in hypothyroid patients could alleviate clinical symptoms by examining anthropometric and clinical parameters. The 500 mg/day synbiotic capsules contained seven freeze-dried probiotic strains (Lactobacillus casei, L. acidophilus, L. rhamnosus, L. bulgaricus, Bifidobacterium breve, B. longum, Streptococcus thermophilus) plus 40 mg of fructooligosaccharides (prebiotics). The study concluded that synbiotics relieved hypothyroidism-related constipation but did not significantly alter anthropometric measures or appetite [33].

In RCT involving 60 hypothyroid patients, an 8-week synbiotic intervention significantly reduced TSH levels, levothyroxine dosage, and fatigue severity scale score compared to placebo [32].

A further trial assessed the impact of synbiotic supplementation on TSH, free thyroxine (FT4), depressive symptoms, quality of life, and blood pressure in 51 patients with subclinical hypothyroidism over 10 weeks. Only the intervention group showed notable improvements in functional status, self-reported well-being, and both systolic and diastolic blood pressure [33].

Conclusions

Emerging evidence indicates that targeted micronutrient and supplement strategies can complement levothyroxine therapy and may address persistent symptoms in a subset of patients with hypothyroidism.

Selenium remains one of the most extensively studied adjunct. Daily doses between 100 and 200 μg may reduce anti-TPO titers, help restore euthyroidism in up to one-third of individuals with mild subclinical hypothyroidism, and appear safe in pregnancy at 60–100 μg/day. Nonetheless, benefits vary by baseline selenium status, disease severity, and study design.

Myo-inositol plus selenium (600 mg MI + 83 μg Se twice daily in the pivotal trial) consistently lowers TSH and autoantibody levels and improves Doppler-derived markers of thyroid inflammation, suggesting a synergistic mechanism via TSH-second-messenger signaling and antioxidant protection.

Zinc, typically 25–30 mg/day, supports deiodinase activity and, when co-administered with selenium, yields larger improvements in FT3, FT4, and TSH than either element alone. Data on zinc monotherapy are mixed, but long-term supplementation may stabilize levothyroxine dose requirements.

Vitamin D supplementation (800–4000 IU/day or equivalent loading regimens) shows modest but reproducible reductions in TSH and thyroid autoantibodies -especially in patients with overt deficiency (<20 ng/mL). Immunologically, vitamin D shifts T-cell balance toward a more regulatory phenotype (↑TGF-β, ↓IL-17), though effects on FT3 and FT4 remain inconsistent.

Synbiotics at 500 mg/day (≈10^9 CFU of multi-strain probiotics + 40 mg FOS) improve gastrointestinal symptoms, fatigue scores, and in several trials lower TSH or levothyroxine dose requirements, underscoring the relevance of the gut–thyroid axis.

Across studies, heterogeneity in dosage, formulation, baseline nutrient status, and study duration limits direct comparisons and precludes universal recommendations.

Benefits can be greater in patients with documented micronutrient deficiency, high antibody titers, or residual symptoms despite biochemically adequate LT4 therapy. Routine baseline assessment of selenium, vitamin D, and zinc status and periodic safety monitoring should precede supplementation.

While levothyroxine remains the backbone of hypothyroidism management, judicious use of selenium, vitamin D, zinc, myoinositol, and synbiotics offers a promising route to mitigate autoimmunity, enhance biochemical control, and most importantly improve patients' quality of life. Large, multicenter RCTs with standardized dosing and longer followup are needed.

Disclosure:
All authors have read and agreed with the published version of the manuscript.
Funding Statement: The study did not receive special funding.
Acknowledgments: Not applicable.
Conflict of Interest Statement: No conflicts of interest to declare.

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