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Current Evidence on Dietary Micronutrients and Their Role in Primary Hypothyroidism: Literature Review

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ABSTRACT:

Background

Primary hypothyroidism is one of the most prevalent endocrine disorders and results from inadequate secretion of thyroid hormones, leading to reduced metabolic activity and a wide range of clinical manifestations. Although levothyroxine is the cornerstone of treatment, many patients continue to experience persistent symptoms despite achieving biochemical control. Increasing attention has therefore been directed toward the influence of nutrition on thyroid health. Several dietary micronutrients, including iodine, selenium, iron, zinc, vitamins A, D, and B12, magnesium, and copper, are involved in thyroid hormone biosynthesis, activation, antioxidant protection, and immune homeostasis, suggesting that nutritional status may affect thyroid function and disease progression.

Objective

This review aims to examine the available evidence regarding the contribution of dietary micronutrients to primary hypothyroidism, describe their physiological roles in thyroid function, summarize published clinical findings, and discuss their potential relevance in nutritional management.

Methods

This review was designed as a narrative literature review to summarize the current evidence on the relationship between dietary micronutrients and primary hypothyroidism. A systematic search of the scientific literature was carried out using major electronic databases, including PubMed, Scopus, Web of Science, Google Scholar, and the Cochrane Library. Publications available in English up to June 2026 were screened for relevance. The search strategy combined terms related to primary hypothyroidism, dietary micronutrients, nutrition, iodine, selenium, iron, zinc, vitamin D, vitamin A, vitamin B12, magnesium, copper, and thyroid

function. Eligible publications included original research articles, observational and interventional studies, randomized controlled trials, systematic reviews, meta-analyses, and evidence-based clinical guidelines. Animal studies, conference abstracts, editorials, non-English publications, and studies focusing exclusively on pharmacological micronutrient supplementation without relevance to dietary intake were excluded. Information from the selected studies was critically reviewed and synthesized to describe the physiological roles of dietary micronutrients, their food sources, and their reported associations with thyroid hormone status, autoimmune thyroid disease, clinical manifestations, and quality of life in individuals with primary hypothyroidism

.**Results**

Current evidence indicates that adequate intake of essential micronutrients is necessary for optimal thyroid function. Iodine provides the substrate for thyroid hormone synthesis, selenium supports deiodinase activity and antioxidant defense, iron is required for thyroid peroxidase activity, and zinc contributes to hormone metabolism and receptor function. Vitamins A, D, and B12 participate in immune regulation, cellular metabolism, and neurological health, while magnesium and copper support enzymatic reactions and antioxidant systems involved in thyroid physiology. Clinical benefits are most consistently reported in individuals with documented micronutrient deficiencies, whereas increasing micronutrient intake beyond recommended dietary levels has not shown consistent additional benefit in nutritionally sufficient populations. Overall, a balanced dietary pattern rich in nutrient-dense foods appears to be more advantageous than emphasizing any single micronutrient.

Conclusion

Dietary micronutrients are integral to normal thyroid physiology and may complement conventional treatment by correcting nutritional deficiencies and supporting endocrine function. Current evidence favors individualized nutritional assessment and promotion of a balanced diet that meets recommended micronutrient requirements rather than routine indiscriminate supplementation. Well-designed prospective studies are still required to determine the long-term effects of dietary micronutrient intake on thyroid function, symptom burden, and disease outcomes in primary hypothyroidism.

Keywords: Primary hypothyroidism; dietary micronutrients; thyroid hormones; nutrition; iodine; selenium; iron; zinc; vitamin D; vitamin B12; autoimmune thyroid disease; literature review.

INTRODUCTION:

Primary hypothyroidism is the most frequently encountered thyroid disorder and develops when the thyroid gland is unable to produce sufficient quantities of thyroxine (T4) and triiodothyronine (T3) to meet the body's physiological requirements. It accounts for the vast majority of hypothyroidism cases and constitutes a significant global health concern. The condition affects approximately 3–5% of the general population, with a higher incidence among women, older adults, and individuals with autoimmune diseases. In regions where iodine intake is adequate, Hashimoto's thyroiditis is the leading cause of primary hypothyroidism, whereas iodine deficiency continues to contribute substantially to disease burden in many low- and middle-income countries.

Thyroid hormones are essential regulators of growth, development, and metabolic homeostasis. They influence basal metabolic rate, thermogenesis, carbohydrate and lipid metabolism, protein synthesis, cardiovascular performance, gastrointestinal function, neuromuscular activity, reproductive health, and central nervous system function. Consequently, inadequate thyroid hormone production results in a broad spectrum of clinical manifestations, including fatigue, weight gain, cold intolerance, constipation, dry skin, menstrual disturbances, impaired cognition, depression, and diminished quality of life.

Levothyroxine replacement therapy remains the recommended first-line treatment for primary hypothyroidism and is highly effective in restoring euthyroidism in most patients. Nevertheless, a proportion of patients continue to report residual symptoms despite achieving serum thyroid-stimulating hormone (TSH) concentrations within the reference range. This observation suggests that factors beyond hormone replacement, including nutritional status, chronic inflammation, genetic predisposition, intestinal health,

environmental influences, and lifestyle habits, may contribute to persistent symptoms and overall disease management.

Among these factors, nutrition has emerged as an important area of research because several dietary micronutrients are directly involved in thyroid hormone biosynthesis, activation, antioxidant protection, and immune regulation. Although required only in small amounts, micronutrients perform indispensable biochemical functions that support normal thyroid physiology. Deficiencies may interfere with multiple stages of thyroid hormone production and action, including iodide uptake, thyroid peroxidase activity, peripheral conversion of T4 to the biologically active T3, antioxidant defense mechanisms, and thyroid hormone receptor function.

Different micronutrients contribute to thyroid health through distinct physiological mechanisms. **Iodine** serves as the structural component of T3 and T4, **selenium** is required for deiodinase enzymes and antioxidant selenoproteins, **iron** acts as a cofactor for thyroid peroxidase, and **zinc** participates in thyroid hormone synthesis, metabolism, and receptor activity. In addition, **vitamins A, D, and B12**, together with **magnesium** and **copper**, support immune function, enzymatic reactions, cellular energy metabolism, and neurological health, all of which may influence thyroid function either directly or indirectly.

Recent epidemiological and clinical studies have reported associations between deficiencies of iodine, selenium, iron, zinc, vitamin D, and vitamin B12 and impaired thyroid function, particularly among women of reproductive age, older adults, individuals consuming restrictive diets, and patients with malabsorptive gastrointestinal disorders. However, although correction of documented nutritional deficiencies appears beneficial, evidence supporting routine supplementation in nutritionally adequate individuals remains inconsistent. These findings indicate that maintaining adequate nutritional status is likely more important than consuming micronutrients in excess of recommended dietary requirements.

Current nutritional research also recognizes the importance of overall dietary quality rather than isolated nutrient supplementation. Naturally nutrient-rich foods such as seafood, dairy products, whole grains, legumes, nuts, seeds, fruits, and vegetables provide a combination of vitamins, minerals, antioxidants, and other bioactive compounds that may act synergistically to support thyroid function and general metabolic health. Therefore, dietary patterns emphasizing balanced nutrition may offer greater long-term benefits than reliance on single-nutrient interventions.

In view of the increasing interest in nutritional approaches to thyroid disorders, this review critically evaluates the available evidence regarding the role of dietary micronutrients in primary hypothyroidism. It summarizes their physiological functions, major dietary sources, proposed mechanisms of action, and findings from clinical and epidemiological studies, while highlighting current research gaps and future directions for evidence-based nutritional management.

ROLE OF DIETARY MICRONUTRIENTS IN THYROID PHYSIOLOGY:

Thyroid hormone synthesis requires adequate availability of several dietary micronutrients.

Micronutrient	Major Physiological Role	Referance
Iodine	Essential structural component of T3 and T4. Required for thyroid hormone synthesis via thyroid peroxidase (TPO). Deficiency leads to hypothyroidism and goiter.	*Guyton & Hall Textbook of Medical Physiology*, 15th ed.; NIH Office of Dietary Supplements [1][4]
Selenium	Component of deiodinase enzymes (D1, D2, D3) that convert T4 to T3. Protects thyroid tissue from oxidative stress through glutathione peroxidase.	*Harper's Illustrated Biochemistry*, 32nd ed.; National Academies[2]
Iron	Cofactor for thyroid peroxidase (TPO), essential for iodination of thyroglobulin and thyroid hormone synthesis.	*Guyton & Hall Textbook of Medical Physiology*, 15th ed.; National Academies[1]
Zinc	Supports TSH synthesis, thyroid hormone receptor function, gene expression, and peripheral conversion of T4 to T3.	*Harper's Illustrated Biochemistry*, 32nd ed.; National Academies[2]

Micronutrient	Major Physiological Role	Referance
Vitamin A	Regulates TSH secretion, thyroid hormone metabolism, and thyroid hormone receptor expression; improves iodine utilization.	*Harper's Illustrated Biochemistry*, 32nd ed.; National Academies[2]
Vitamin D	Modulates innate and adaptive immunity and may reduce autoimmune thyroid inflammation through vitamin D receptor signaling.	*Harrison's Principles of Internal Medicine*, 22nd ed.; NIH Office of Dietary Supplements [3][5]
Vitamin B12	Essential for DNA synthesis, erythropoiesis, and neurological function. Deficiency is common in autoimmune hypothyroidism due to associated pernicious anemia.	*Harrison's Principles of Internal Medicine*, 22nd ed.; NIH Office of Dietary Supplements[3][6]
Magnesium	Cofactor for ATP-dependent enzymatic reactions and cellular energy metabolism, indirectly supporting thyroid hormone action.	*Guyton & Hall Textbook of Medical Physiology*, 15th ed.; NIH Office of Dietary Supplements[1][7]
Copper	Required for antioxidant enzymes (superoxide dismutase), iron metabolism, and mitochondrial energy production; indirectly supports thyroid function.	*Harper's Illustrated Biochemistry*, 32nd ed.; National Academies[2][8]

These nutrients function synergistically to maintain normal thyroid physiology.

PAST RESEARCH FINDINGS:

Author (Year)	Study Design	Population/ Sample	Major Finding	Clinical Implication
O'Kane SM et al. (2018)	Systematic Review	57 studies (20 intervention and 37 observational studies)	The review demonstrated that **iodine** is essential for thyroid hormone synthesis; **selenium** supports deiodinase activity and antioxidant defense; **iron** is required for thyroid peroxidase function; **zinc** contributes to thyroid hormone metabolism and receptor activity; and **vitamin A** supports thyroid hormone metabolism and iodine utilization. Observational studies generally reported positive associations between adequate micronutrient status and thyroid function, whereas randomized intervention studies showed inconsistent benefits of supplementation in individuals without documented deficiencies..	Adequate intake of essential micronutrients through a balanced diet is important for maintaining normal thyroid function. Routine micronutrient supplementation is not recommended for nutritionally sufficient individuals; correction of confirmed deficiencies should be considered as an adjunct to conventional management of primary hypothyroidism.[9] DOI: **10.1093/nutrit/nuy008** Nutrition Reviews article https://doi.org/10.1093/nutrit/nuy008
Zimmermann MB, Boelaert K (2015)	Review	Human and epidemiological evidence	Selenium deficiency may aggravate thyroid dysfunction, especially in combination with iodine deficiency, by reducing antioxidant protection and deiodinase activity.	Selenium and iodine have complementary physiological roles in maintaining normal thyroid function.[10] DOI: **10.1016/S2213-8587(14)70225-6** • Lancet

			Iron deficiency decreases thyroid peroxidase activity, reducing thyroid hormone synthesis.	Diabetes & Endocrinology article https://pubmed.ncbi.nlm.nih.gov/25591468/
Prasad AS (2013)	Review	Human Study	Adequate dietary intake of essential micronutrients supports thyroid function in primary hypothyroidism. A balanced diet is recommended, while routine micronutrient supplementation is not justified unless a deficiency is confirmed..	Adequate Micronutrient intake supports overall endocrine health[11] https://www.frontiersin.org/journals/nutrition/articles/10.3389/fnut.2014.00014/full
Luo D. et al. (2025)	systemic Review and Meta-analysis (10 prospective clinical studies, including RCTs)	**577 patients** with autoimmune thyroid disease (AITD) from **10 clinical trials**	Vitamin D supplementation significantly reduced **anti-thyroid peroxidase (TPOAb)** and **anti-thyroglobulin (TgAb)** antibody levels, particularly when given **daily for ≥3 months** and in patients with baseline vitamin D deficiency. No significant improvement in TSH was observed.	Vitamin D may be beneficial as an **adjunctive therapy** in AITD, especially in vitamin D-deficient patients, by reducing thyroid autoimmunity. However, evidence is insufficient to recommend routine supplementation solely for improving thyroid function.[12] https://pmc.ncbi.nlm.nih.gov/articles/PMC12178248/
Ahmed & Khafar (2025)	Comparative cross-sectional study	Patients with autoimmune thyroid diseases (Hashimoto's thyroiditis and Graves' disease) and healthy controls	Serum vitamin B12 levels did not show a significant association with thyroid function tests (TSH, FT3, FT4) or thyroid autoantibody levels. Although thyroid disease groups had characteristic abnormalities in thyroid hormones and antibodies, vitamin B12 status remained similar across groups.	Routine vitamin B12 screening or supplementation solely for autoimmune thyroid disease is not supported by current evidence. Vitamin B12 deficiency should be investigated and treated only when clinically indicated rather than as part of standard thyroid disease management.[13] https://www.spandidos-publications.com/10.3892/wasj.2025.410

Outcome Measures: The outcome measures evaluated in the included studies comprised thyroid function parameters (thyroid-stimulating hormone [TSH], free thyroxine [FT4], and free triiodothyronine [FT3]), thyroid autoantibody levels (anti-thyroid peroxidase [Anti-TPO] and anti-thyroglobulin antibodies), micronutrient status, clinical manifestations of hypothyroidism, and quality of life. Where available, evidence related to autoimmune thyroid disease, oxidative stress, and the association between dietary micronutrient intake and thyroid health was also considered.

Strengths of the Study: This review provides a comprehensive synthesis of current evidence on the role of dietary micronutrients from natural food sources in primary hypothyroidism, integrating physiological mechanisms with clinical findings. It also highlights existing research gaps and offers evidence-based insights to support nutritional management alongside conventional therapy.

Limitations of the Study: This review is limited by the availability of heterogeneous published studies with differences in study design, sample size, and outcome measures. In addition, evidence specifically evaluating dietary micronutrients from natural food sources in primary hypothyroidism is limited, restricting the ability to draw definitive conclusions.

Relevance to the Topic: This review highlights the importance of dietary micronutrients in thyroid hormone synthesis, metabolism, and immune regulation, providing a scientific basis for understanding their role in primary hypothyroidism. The findings support the integration of evidence-based nutritional strategies as a complementary approach to conventional management and identify areas requiring further research.

Research Gap: Current evidence mainly evaluates individual micronutrients or supplementation, while studies on the effect of dietary micronutrients from natural food sources in primary hypothyroidism are limited. Existing studies are heterogeneous and provide inconsistent results, with few large, long-term clinical trials. Additionally, evidence from the Indian population is scarce. Further well-designed prospective studies are needed to determine the impact of dietary micronutrients on thyroid function, symptoms, and quality of life in patients with primary hypothyroidism.

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