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# Impact of Dietary Factors on Thyroid Function and Disorders

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## Abstract

Thyroid health is intricately linked to nutritional status, as multiple micronutrients and dietary components modulate hormone biosynthesis, metabolism, and immune function. The increasing global prevalence of autoimmune thyroid disorders, particularly Hashimoto's thyroiditis and Graves' disease, underscores the need to examine the influence of modifiable dietary factors on thyroid pathophysiology. This narrative review aimed to evaluate current evidence on the role of nutrition—especially micronutrients such as iodine, selenium, iron, zinc, and vitamin D—in thyroid function and autoimmunity. A structured literature search was conducted using PubMed and Scopus to identify relevant human studies published between January 2002 and March 2025. A total of forty-one peer-reviewed articles were selected based on predefined inclusion criteria and assessed for scientific rigor and relevance. The evidence confirms the critical involvement of iodine and selenium in thyroid hormone synthesis and immune modulation, while iron and zinc were found to support enzymatic activity and endocrine stability. Vitamin D emerged as a potential immunoregulatory agent in autoimmune thyroid disease. Furthermore, certain macronutrients—including high-quality proteins, fiber, and omega-3 fatty acids—as well as dietary patterns such as the Mediterranean diet, were associated with favorable thyroid outcomes. Conversely, the intake of goitrogenic foods and soy isoflavones may necessitate caution in predisposed individuals. These findings highlight

the relevance of individualized, evidence-based nutritional strategies as adjunctive approaches in the prevention and clinical management of thyroid disorders.

**Keywords**

Thyroid function; Autoimmune thyroid disease; Nutritional factors; Micronutrients; Dietary supplementation; Hashimoto's thyroiditis

## 1. Introduction

The thyroid gland plays a pivotal role in regulating metabolism, growth, and development through the production of thyroid hormones, thereby making its optimal function critical for overall human health [1]. Globally, thyroid disorders—including hypothyroidism, hyperthyroidism, and autoimmune conditions—represent a significant public health concern, affecting millions of individuals across varying demographics [41]. Among the well-established etiologies, iodine deficiency is historically recognized for its strong association with goiter formation and thyroid dysfunction [1,3]. However, increasing research underscores the relevance of additional micronutrients—such as selenium, iron, and zinc—as vital cofactors for thyroid hormone biosynthesis and immune regulation [4,5]. Autoimmune thyroid diseases (AITDs), particularly Hashimoto's thyroiditis and Graves' disease, are noteworthy contributors to the global burden of thyroid pathology [5,12]. Evidence suggests that nutritional factors may modulate autoimmune pathways, with vitamin D deficiency emerging as a potential risk factor for AITDs [6]. Indeed, suboptimal vitamin D status has been correlated with altered immune tolerance, supporting the hypothesis that adequate vitamin D intake may help regulate autoimmune processes [7,11]. Concurrently, iron deficiency poses additional concerns, particularly because adequate iron stores are required for proper thyroid peroxidase (TPO) activity [7,8,15]. Persistent iron deficiency may not only compromise thyroid hormone synthesis but also exacerbate existing thyroid dysfunction [8]. Beyond micronutrients, dietary composition as a whole appears to influence thyroid health. Certain dietary patterns—such as those high in fiber, specific fatty acids, or protein—have been explored for their capacity to support or hinder thyroid hormone metabolism [9,24,25,29]. Moreover, multiple studies reveal that optimizing selenium intake can help reduce thyroid autoantibody titers and potentially improve clinical outcomes in both Hashimoto's thyroiditis and Graves' disease [4,12]. In regions with adequate iodine status, excessive iodine consumption has likewise been associated with an increased risk of subclinical hypothyroidism, highlighting the delicate balance between

sufficiency and excess for this key micronutrient [3,38]. Considering the expanding body of literature on the nutritional determinants of thyroid function, there is a pressing need for a comprehensive synthesis of current findings [2]. This review seeks to comprehensively synthesize current evidence on the influence of micro- and macronutrient intake on thyroid physiology and pathology, with particular emphasis on human studies relevant to the prevention and clinical management of thyroid disorders. By critically evaluating data on essential micronutrients—such as iodine, selenium, iron, zinc, and vitamin D—as well as broader dietary patterns, this work aims to offer evidence-based perspectives to inform both clinical decision-making and future research directions in thyroid health.

## 2. Materials and Methods

A comprehensive literature search was conducted using PubMed and Scopus to identify peer-reviewed articles that examined how dietary factors—specifically micro- and macronutrients—affect thyroid function in human populations. The search spanned from January 2002 through March 2025 and involved keywords such as “thyroid function,” “thyroid diseases,” “iodine,” “selenium,” “iron,” “zinc,” “vitamin D,” “autoimmune thyroid disease,” “Hashimoto’s thyroiditis,” “Graves’ disease,” “dietary patterns,” and “nutritional factors.” Relevant synonyms and MeSH terms were also employed.

Inclusion criteria encompassed:

1. Articles published in English.
2. Studies involving human participants of any age group.
3. Focus on dietary elements, nutrient supplementation, or dietary patterns in thyroid hormone synthesis or clinical management.
4. Study types: RCTs, cohort, case-control, cross-sectional, systematic reviews.

Exclusion criteria included:

1. Animal or in vitro studies.
2. Non-original publications (editorials, abstracts).
3. Articles without detailed dietary/nutritional data.

In total, 41 eligible articles were selected and cited throughout this review using bracketed reference numbers.

## 3.Key Nutrients Affecting Thyroid Function

Thyroid hormone synthesis, activation, and regulation are highly dependent on nutritional status. A wide range of micronutrients and macronutrients are involved in these processes, either as cofactors in enzymatic reactions, as structural components of hormones, or as modulators of immune function. This section explores the most relevant nutrients in thyroid physiology and pathology, organized into thematic categories based on their mechanisms of action.

### 3.1. Iodine

Iodine is essential for the biosynthesis of thyroxine (T4) and triiodothyronine (T3). Its dietary availability is a key determinant of thyroid hormone production, and both deficiency and excess can disrupt thyroid function [1,3]. Iodine deficiency remains a leading cause of goiter and hypothyroidism worldwide, despite public health efforts to introduce iodized salt [19,28].

However, excessive iodine intake can also be harmful, particularly in regions with successful iodine fortification. Studies have shown that high iodine consumption may increase the risk of autoimmune thyroiditis and subclinical hypothyroidism [3,36]. A systematic review confirmed that populations exposed to excess iodine are more likely to experience thyroid autoimmunity, particularly those with underlying susceptibility [36].

An important consideration is the presence of goitrogens, naturally occurring substances that inhibit iodine uptake. Cruciferous vegetables such as broccoli, cabbage, and kale contain glucosinolates, which can interfere with iodine utilization when consumed in raw or excessive quantities [38]. However, cooking typically reduces their goitrogenic potential, and moderate intake is not associated with adverse thyroid outcomes in iodine-replete individuals [38].

The World Health Organization recommends a daily iodine intake of 150 µg for adults and 250 µg for pregnant and lactating women. Although iodine fortification programs have substantially reduced global iodine deficiency, emerging evidence suggests that excessive iodine intake may also pose risks, particularly in iodine-sufficient or oversupplemented populations. High iodine intake was significantly associated with subclinical hypothyroidism **[**36]. Similarly, studies in various age groups have revealed a U-shaped relationship between iodine intake and thyroid dysfunction, highlighting the need for balanced consumption [19,28].

Notably, iodine metabolism can be influenced by other micronutrients such as selenium and iron, whose deficiencies may worsen the effects of inadequate or excessive iodine intake [3,13]. Therefore, iodine sufficiency should be evaluated alongside other nutritional parameters in patients with thyroid disorders.

## 3.2. Selenium

Selenium is a trace element of critical importance in thyroid physiology, due to its integral role in both hormonal metabolism and antioxidant protection. The thyroid gland accumulates the highest selenium content per gram of tissue in the human body, reflecting its reliance on adequate selenium availability [18,23]. Low selenium status has been associated with increased prevalence of thyroid disorders, including autoimmune thyroiditis and both hypo- and hyperthyroidism, particularly in populations with marginal selenium intake [23].Selenium exerts its physiological effects via its incorporation into selenoproteins, such as glutathione peroxidases (GPx), thioredoxin reductases (TrxR), and iodothyronine deiodinases (DIOs). GPx and TrxR function as key components of the thyroid's antioxidant system, neutralizing hydrogen peroxide generated during thyroid hormone biosynthesis and thereby preventing oxidative damage to thyrocytes. Iodothyronine deiodinases, particularly types I and II, catalyze the peripheral conversion of thyroxine (T4) to the biologically active triiodothyronine (T3), a process essential for thyroid hormone homeostasis [4,18]. Interestingly, altered expression and activity of selenoenzymes such as GPx and TrxR have been documented in different types of thyroid tumors, suggesting a potential involvement of selenium in redox regulation and tumor pathophysiology [10].

In the context of autoimmune thyroid diseases (AITDs), selenium has garnered substantial research interest due to its immunomodulatory properties. A randomized controlled trial by Gärtner et al. demonstrated that selenium supplementation significantly reduced serum titers of anti-thyroid peroxidase antibodies (TPOAb) in patients with Hashimoto's thyroiditis [4]. These findings have been supported by meta-analyses, including that by Toulis et al., which confirmed a modest but statistically significant reduction in TPOAb levels following selenium intervention [5]. Additionally, the SETI (Selenium Therapy and Autoimmune Thyroiditis) study observed improvements in thyroid function markers in individuals with subclinical hypothyroidism and thyroid autoimmunity after selenium supplementation [14].

Selenium has also shown therapeutic potential in Graves' disease. A systematic review and meta-analysis by Zheng et al. concluded that selenium supplementation may facilitate earlier normalization of thyroid hormone levels, reduce TSH receptor antibody (TRAb) concentrations, and improve clinical outcomes in newly diagnosed cases [12]. Collectively, these findings suggest that selenium contributes to both redox regulation and immune modulation within the thyroid microenvironment.

Nevertheless, the clinical application of selenium remains a subject of ongoing debate. Several factors, including variability in baseline selenium status, geographical differences in soil selenium content, differences in selenium formulation and dosage, and heterogeneity in study designs, may account for inconsistent findings across trials [5,18,20]. For example, a randomized controlled trial conducted in healthy Japanese men failed to demonstrate any effect of selenium supplementation on thyroid hormone metabolism, underscoring the importance of baseline status and disease presence in determining clinical benefit [20]. As such, selenium supplementation should not be universally recommended, but rather considered on an individual basis, particularly in populations or clinical contexts with evidence of suboptimal selenium levels or autoimmune thyroid activity.

## 3.3. Iron

Iron is an essential cofactor for thyroid peroxidase (TPO), the enzyme responsible for the iodination of tyrosyl residues in thyroglobulin—a key step in thyroid hormone synthesis [7]. Iron deficiency, particularly in women of reproductive age, is prevalent globally and can impair TPO activity, leading to decreased T4 and T3 levels and compensatory increases in TSH [8].

Animal studies have demonstrated that iron-deficient states lead to altered thyroid hormone kinetics, even before clinical anemia develops [7]. In humans, subclinical hypothyroidism has been observed in iron-deficient individuals, and iron supplementation has been shown to improve TSH levels and normalize thyroid function in certain populations [15].

Importantly, iron deficiency may exacerbate the consequences of iodine insufficiency. A combined deficiency of iodine and iron has been shown to have a compounded effect on thyroid metabolism, suggesting that interventions to address one should also screen for the other [3].

## 3.4. Zinc

Zinc is another trace element essential for thyroid function. It supports the activity of deiodinases, enzymes involved in the peripheral conversion of T4 to T3, and is also necessary for maintaining receptor binding of thyroid hormones at the nuclear level [16]. Zinc is involved in immune regulation as well, which is particularly relevant in autoimmune thyroid conditions.

Lower zinc levels have been observed in patients with hypothyroidism and autoimmune thyroiditis [16,30]. Although studies on zinc supplementation in thyroid disease remain limited, zinc deficiency may be underrecognized, especially in individuals with restrictive diets, malabsorption, or chronic illness [30]. Ensuring adequate zinc intake may be particularly important in patients with nonspecific symptoms of thyroid dysfunction despite normal hormone levels.

## 3.5. Vitamin D

Vitamin D plays a well-established role in bone metabolism, but its immunomodulatory properties have drawn attention in the context of autoimmune thyroid disease [6]. Observational studies report lower serum 25-hydroxyvitamin D levels in individuals with Hashimoto's thyroiditis and Graves' disease compared to healthy controls [11,17].

Vitamin D receptors are expressed on thyroid follicular cells and various immune cells, suggesting direct effects on both thyroid function and immune regulation [6,21]. Several studies indicate that vitamin D deficiency is associated with higher levels of thyroid autoantibodies, and some trials report modest reductions in antibody titers with vitamin D supplementation in deficient individuals [26].

Vitamin D requirements are particularly important in women of reproductive age, including those who are pregnant or lactating, as thyroid dysfunction in these groups can impact both maternal and fetal outcomes [40]. Optimizing vitamin D levels in this population may serve a dual purpose of supporting endocrine balance and enhancing immune regulation.

## 3.6. Calcium and Magnesium

While calcium is not directly involved in thyroid hormone synthesis, thyroid dysfunction can significantly affect calcium homeostasis. Hyperthyroidism is known to increase bone turnover, potentially leading to bone loss and hypercalcemia, whereas hypothyroidism may impair calcium absorption and bone remodeling [26]. Co-supplementation of calcium and vitamin D in autoimmune thyroid disease has shown modest effects on reducing autoantibody levels and supporting bone health [26].

Magnesium serves as a cofactor in many enzymatic reactions, including those involved in ATP metabolism and protein synthesis, both of which are important for proper thyroid hormone function. A systematic review and meta-analysis identified associations between low magnesium status and altered thyroid function, although the clinical significance of these findings remains under debate [27].

Together, calcium and magnesium are important supportive minerals in managing the systemic effects of thyroid disorders, particularly in those with coexisting metabolic or skeletal conditions.

## 3.7. Protein and Amino Acids

Dietary protein intake may influence thyroid function both directly and indirectly. Adequate protein is necessary for the synthesis of thyroid hormone–binding globulins and for maintaining muscle mass and metabolic rate, which are often affected in both hypo- and hyperthyroidism.

A recent review suggests that low protein intake may reduce circulating thyroid hormone levels, particularly in populations with already compromised nutritional status [29]. On the other hand, excessive intake of certain amino acids, particularly in the context of protein supplementation, may affect hormone metabolism, although clinical data are limited.

Patients with hyperthyroidism, who often experience muscle wasting and increased metabolic demands, may benefit from a high-protein diet to counteract catabolism and support recovery [35]. Conversely, individuals with hypothyroidism should ensure adequate, but not excessive, protein intake to support basal metabolic functions without promoting excess weight gain.

## 3.8. Phytoestrogens and Soy-Based Foods

Soy isoflavones, classified as phytoestrogens, have been investigated for their potential impact on thyroid function, particularly due to their structural similarity to estradiol and their ability to bind estrogen receptors. There has been longstanding concern that high soy intake may interfere with thyroid hormone synthesis, especially in individuals with iodine deficiency [30].

Early in vitro and animal studies suggested that soy compounds might inhibit TPO activity, though these effects were largely dependent on iodine status. In humans, moderate soy consumption does not appear to adversely affect thyroid function in iodine-sufficient individuals. A review concluded that while soy may modestly influence thyroid hormone levels, it is unlikely to cause clinical hypothyroidism in healthy adults [31].

Nonetheless, for individuals with subclinical hypothyroidism or marginal iodine intake, monitoring soy intake may be prudent. Clinicians should ensure that patients consuming a soy-rich diet are also meeting their iodine requirements.

### 3.9. Omega-3 Fatty Acids and Dietary Fats

Omega-3 polyunsaturated fatty acids (PUFAs) possess well-documented anti-inflammatory properties and have been investigated for their potential to modulate thyroid autoimmunity. These fatty acids may influence the production of pro-inflammatory cytokines and improve the lipid profile in patients with hypothyroidism, who are often at increased cardiovascular risk [24].

Preliminary evidence suggests that omega-3 intake may reduce thyroid-specific autoantibodies in patients with Hashimoto’s thyroiditis, although more randomized controlled trials are needed to confirm these findings [24]. Additionally, diets rich in omega-3s, such as the Mediterranean diet, may offer synergistic benefits through antioxidant effects and improved immune regulation [33].

Patients with thyroid disorders are often advised to reduce their intake of trans fats and excessive saturated fats, which are linked to systemic inflammation. Replacing these with omega-3-rich foods (e.g., fatty fish, flaxseed, walnuts) may help support immune homeostasis and metabolic health.

### 3.10. Dietary Patterns and Combined Supplementation Strategies

Beyond isolated nutrients, overall dietary patterns play a crucial role in thyroid health. Diets characterized by high antioxidant content, healthy fats, and balanced macronutrient profiles—such as the Mediterranean diet—have been associated with lower inflammation and better metabolic outcomes in thyroid patients [33,39].

Certain dietary strategies, such as gluten-free or low-carbohydrate diets, may offer benefits in autoimmune thyroid disease, particularly in individuals with concurrent gluten sensitivity or celiac disease [22,35]. Fiber-rich diets also support gastrointestinal health and glycemic control but should be balanced to avoid impairing levothyroxine absorption [25].

Additionally, combined micronutrient supplementation has been explored as a strategy to enhance thyroid function. A review on nutraceutical support suggested that multi-component interventions—including iodine, selenium, iron, and vitamin D—may be more effective than single-nutrient approaches in individuals with suboptimal thyroid function [34]. While promising, such strategies require clinical validation to establish efficacy and safety.

Finally, dietary patterns may also influence thyroid cancer risk. A review of epidemiological studies suggested that diets high in fruits, vegetables, and fish may offer protective effects, whereas those high in processed meats and refined carbohydrates could be associated with increased risk [33]. Though thyroid cancer remains relatively rare, dietary interventions aimed at modulating systemic inflammation may contribute to broader endocrine protection.

## 4.Nutritional Considerations in Thyroid Disorders

Nutritional strategies can vary depending on the specific type of thyroid dysfunction. While optimal nutrient intake is essential for thyroid health in the general population, individuals diagnosed with hypothyroidism, hyperthyroidism, or autoimmune thyroid disorders may require tailored dietary approaches to address the unique pathophysiological features of each condition. This section provides an in-depth analysis of evidence-based nutritional considerations across different thyroid disorders.

### 4.1. Hypothyroidism and Hashimoto’s Thyroiditis

Hypothyroidism, characterized by insufficient production of thyroid hormones, can result from a variety of causes, with Hashimoto’s thyroiditis being the most prevalent autoimmune etiology.

Nutritional interventions in hypothyroidism aim not only to support hormone synthesis but also to mitigate immune activation in cases of autoimmune origin.

Iodine remains the cornerstone element for thyroid hormone production. However, in individuals with autoimmune thyroiditis, excessive iodine intake may aggravate immune responses and accelerate thyroid tissue destruction [3,36]. Studies emphasize the importance of avoiding both iodine deficiency and overload, as both extremes can disrupt thyroid homeostasis [3,19]. Therefore, iodine intake in hypothyroid patients—particularly those with Hashimoto's disease—should be individualized and based on regional iodine status.

Selenium plays a particularly important role in reducing thyroid autoantibody titers in Hashimoto's thyroiditis. Clinical trials have shown that selenium supplementation (typically 200 µg/day) can significantly lower levels of thyroid peroxidase antibodies (TPOAb), which are commonly elevated in Hashimoto's patients [4,12]. A meta-analysis confirmed that selenium may enhance thyroid function and quality of life in affected individuals [5,18]. However, long-term use should be monitored, as both deficiency and excess may pose health risks.

Vitamin D status is also frequently compromised in Hashimoto's thyroiditis. Deficiency has been associated with higher TSH levels and increased autoantibody titers [6,11,17]. Supplementation of vitamin D in deficient individuals may offer immune-modulating benefits and improve clinical outcomes, although further randomized controlled trials are needed to establish optimal dosing protocols [26].

Iron deficiency, especially among women of reproductive age, is common in hypothyroid populations and can compromise thyroid peroxidase activity, impeding effective hormone production [8,15]. Addressing iron deficiency through dietary strategies or supplementation has been shown to support thyroid hormone normalization in subclinical cases [15].

Beyond micronutrients, broader dietary patterns such as gluten-free diets have been explored in Hashimoto's patients due to the overlap with celiac disease and shared autoimmune pathways. Some observational data indicate that eliminating gluten may reduce thyroid autoantibodies and improve gastrointestinal symptoms in select patients [32]. However, universal recommendations for gluten elimination in Hashimoto's remain controversial and should be individualized.

Additionally, diets rich in omega-3 fatty acids and antioxidants—such as those found in the Mediterranean diet—may help modulate inflammatory responses in Hashimoto's disease [24,33]. A balanced intake of anti-inflammatory nutrients may offer supportive benefits in reducing thyroid tissue damage, though direct evidence from intervention trials is still limited.

## 4.2. Hyperthyroidism and Graves' Disease

Hyperthyroidism, especially when caused by Graves' disease, involves the overproduction of thyroid hormones and is often accompanied by weight loss, anxiety, and increased metabolic demand. Nutritional management in hyperthyroidism focuses on meeting increased energy and protein needs while reducing oxidative stress and modulating immune responses.

Increased metabolism in hyperthyroid patients can lead to the depletion of key micronutrients such as selenium, zinc, magnesium, and calcium, all of which are essential for muscle function, bone health, and hormone balance [4,8,10]. Supplementation may be necessary to restore adequate levels, particularly during periods of active disease or antithyroid drug therapy.

Selenium, in particular, has shown promise in improving clinical symptoms and quality of life in patients with Graves' disease. Supplementation may reduce oxidative damage and immune overactivity, as observed in clinical trials [12]. A meta-analysis demonstrated that selenium can contribute to faster normalization of thyroid function and decrease in TSH receptor antibodies (TRAb) in newly diagnosed Graves' disease [5].

Additionally, vitamin D insufficiency is frequently found in Graves' patients and may contribute to immune dysregulation [6]. Adequate vitamin D levels may support immunological balance and skeletal protection, particularly given the increased risk of bone demineralization in hyperthyroidism [26].

Hyperthyroid individuals often experience weight loss and muscle wasting, necessitating a high-calorie, high-protein diet to meet energy demands and prevent catabolism [29]. Including easily digestible energy sources and sufficient dietary protein is critical during the hypermetabolic phase.

As Graves' disease has an autoimmune component, anti-inflammatory diets rich in omega-3 fatty acids, fruits, and vegetables may offer additional support. Although no specific diet can cure hyperthyroidism, nutritional therapy plays a valuable adjunctive role in maintaining nutrient reserves, reducing inflammation, and supporting metabolic recovery.

### 4.3. Other Thyroid Conditions: Goiter, Subclinical Dysfunction, and Nodules

Goiter, or thyroid enlargement, often results from chronic iodine imbalance—either deficiency or excess. Epidemiological studies indicate that populations with borderline iodine intake may develop diffuse or nodular goiter, especially when coexisting selenium or iron deficiencies are present [3,13,28].

Subclinical hypothyroidism, characterized by elevated TSH with normal T3 and T4, may be influenced by nutritional factors, particularly iron and selenium status. In iron-deficient women, supplementation has been shown to improve TSH levels and prevent progression to overt hypothyroidism [15]. Similarly, selenium supplementation in individuals with thyroid autoantibodies may slow disease progression and preserve thyroid function [14].

Thyroid nodules have been associated with oxidative stress and regional iodine intake variability. Diets rich in antioxidants—such as vitamins C, E, and selenium—may offer protective benefits, although definitive clinical evidence is lacking [18,33].

Furthermore, attention should be given to dietary patterns high in goitrogens (e.g., raw cruciferous vegetables), which may interfere with iodine uptake when consumed in excess, especially in iodine-deficient individuals [32]. Cooking these vegetables typically inactivates goitrogenic compounds, making moderate consumption generally safe for most individuals.

## 5.Discussion

The literature reviewed in this article underscores the multifactorial relationship between nutrition and thyroid health. Nutritional status not only affects thyroid hormone synthesis and metabolism but may also influence the development and progression of autoimmune thyroid disorders. Key nutrients—including iodine, selenium, iron, zinc, and vitamin D—play central roles in maintaining thyroid function, and imbalances in any of these can significantly impact both biochemical and clinical outcomes.

One of the most consistently supported findings is the importance of selenium in autoimmune thyroid disease. Multiple trials and meta-analyses confirm its efficacy in lowering thyroid peroxidase antibodies (TPOAb), with beneficial effects observed in both Hashimoto's thyroiditis and Graves' disease [4,5,12,18]. However, not all studies have demonstrated uniform benefits, highlighting heterogeneity in dosing, duration, and population characteristics.

Additionally, excess selenium may pose toxicity risks, and long-term supplementation requires clinical monitoring [14].

Similarly, vitamin D deficiency is frequently observed in patients with autoimmune thyroid disease, and its immunomodulatory role is well-established [6,11,17]. While some interventional studies show that supplementation may lower autoantibody titers and improve disease markers [26], other research has failed to find consistent associations, especially in populations with borderline sufficiency. The variability in study results suggests a need for standardized trials that evaluate dosing thresholds and long-term outcomes.

The case of iodine illustrates the complex nature of nutrient-thyroid interactions. Although iodine deficiency is a well-known cause of hypothyroidism and goiter [1,3], excessive intake—especially in already iodine-sufficient populations—has been linked to subclinical hypothyroidism and increased autoimmune activity [19,36]. These findings point to a U-shaped relationship between iodine status and thyroid disease risk, reinforcing the importance of maintaining optimal, but not excessive, intake.

Iron and zinc have received comparatively less attention in thyroid research but are equally vital. Iron is required for thyroid peroxidase activity, and deficiency can impair hormone production [7,8,15]. Zinc, though often underdiagnosed as a contributor to thyroid dysfunction, plays a role in deiodinase activity and immune modulation [16]. Supplementation with these micronutrients may yield clinical benefits, particularly in populations at risk of nutritional insufficiency, such as women of reproductive age or individuals following restrictive diets [31].

Broader dietary patterns also appear to influence thyroid health. For example, gluten-free diets may reduce autoimmunity in Hashimoto's thyroiditis, though the evidence remains mixed and is currently strongest among those with coexisting celiac disease [32,37]. Similarly, diets high in omega-3 fatty acids and antioxidants may mitigate inflammatory pathways implicated in thyroid autoimmunity [24,33]. The Mediterranean diet—rich in polyunsaturated fats, fiber, and micronutrients—has shown promise in supporting endocrine and immune function, though direct studies linking it to thyroid-specific outcomes are still limited.

Emerging areas of interest include the role of gut microbiota in thyroid autoimmunity. Early studies suggest that probiotic supplementation may reduce thyroid autoantibodies and improve quality of life in patients with Hashimoto's thyroiditis [34]. While these findings are promising,

the mechanisms underlying the gut–thyroid axis are still being unraveled, and further research is needed to confirm causality and therapeutic potential.

Despite the encouraging findings in many areas, this review also highlights significant limitations in the existing body of evidence. Many studies are observational in design, limiting causal inference. Interventional studies, while more robust, often vary in methodology, population selection, and outcome measures. There is a clear need for large-scale, randomized controlled trials that evaluate long-term clinical outcomes of targeted nutritional interventions in thyroid disease.

Moreover, individual variation in nutrient absorption, genetic polymorphisms, and environmental exposures may modulate the relationship between diet and thyroid health, emphasizing the importance of personalized approaches to nutritional management.

In summary, the current evidence supports a strong link between nutritional status and thyroid function, particularly in the context of autoimmune thyroid disorders. Optimizing intake of key micronutrients, adopting anti-inflammatory dietary patterns, and correcting specific deficiencies may serve as valuable adjuncts in the prevention and management of thyroid diseases.

## 6.Conclusions

Thyroid function is intricately connected to nutritional status. A growing body of evidence supports the notion that both deficiencies and excesses of specific nutrients can significantly influence thyroid hormone synthesis, regulation, and immune tolerance. Among the most critical micronutrients are iodine, selenium, iron, zinc, and vitamin D—each playing distinct yet interrelated roles in maintaining endocrine balance and supporting metabolic and immunological processes.

Iodine remains a foundational element in thyroid physiology, but its optimal intake must be carefully balanced, as both insufficient and excessive levels can contribute to thyroid dysfunction. Selenium has emerged as a key modulator of autoimmune thyroid disease, demonstrating potential in reducing thyroid antibody levels and oxidative stress. Iron and zinc, though often less emphasized, are equally essential for enzymatic activity and hormone metabolism. Vitamin D, with its immunoregulatory functions, represents a promising adjunctive factor in managing autoimmune thyroid conditions, particularly in deficient

individuals.

Beyond individual nutrients, broader dietary patterns can influence thyroid health through their effects on inflammation, metabolism, and gut–immune interactions. Diets rich in anti-inflammatory and antioxidant components—such as the Mediterranean diet—may support overall endocrine function. Moreover, dietary interventions such as gluten-free or high-protein diets may benefit select subgroups of patients, particularly those with autoimmune or hypermetabolic thyroid states. The potential utility of combined micronutrient supplementation is also being explored, offering a more holistic approach to nutritional support in thyroid disorders.

Despite encouraging findings, it is clear that nutritional strategies should be personalized. Factors such as age, sex, physiological status, comorbidities, and regional nutrient availability must all be considered when tailoring dietary interventions. While nutrition cannot replace pharmacological treatment in overt thyroid disease, it plays a crucial complementary role in prevention, disease modulation, and quality of life enhancement.

Continued interdisciplinary research is essential to refine dietary recommendations, determine effective supplementation protocols, and better understand the complex interplay between nutrition and thyroid function. Integrating nutrition into routine thyroid care may ultimately lead to more comprehensive and individualized therapeutic approaches.

Disclosure

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