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The Gut Microbiome as a Modifiable Factor in Autoimmune Thyroid Disease: Addressing Gaps in Nutrition-Based Strategies for Supporting Thyroid and Gut Microbiome Health

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Abstract: Autoimmune thyroid diseases (AITD), including Hashimoto's Thyroiditis and Graves' Disease, are prevalent endocrine disorders characterized by immune dysregulation, chronic inflammation, and altered thyroid hormone metabolism. Emerging evidence links the gut-thyroid axis as a potentially modifiable contributor to AITD: gut microbiome composition influences intestinal permeability, micronutrient absorption, immune signaling, and thyroid hormone regulation. Gut dysbiosis has been associated with reduced availability of iodine, selenium, zinc, and iron, as well as impaired intestinal barrier function, factors that may promote autoimmune activation and symptom persistence. Abbott et al. (2019) reported that a multidisciplinary lifestyle intervention incorporating the Autoimmune Protocol diet was associated with improvements in quality of life, symptom burden, and inflammatory markers among women with Hashimoto's thyroiditis. However, no statistically significant improvements were observed in thyroid function tests or thyroid antibody levels, and because the intervention combined dietary modification with lifestyle coaching, the independent contribution of diet could not be isolated. This literature review synthesizes current findings on the microbiome, nutrient status, immune regulation, and dietary modification in AITD, identifies evidence-informed clinical and nutritional strategies, and highlights critical gaps warranting rigorous human studies to clarify the roles of targeted nutrition and microbiome-focused interventions in adult AITD.

Keywords: Autoimmune Thyroid Disease; Gut-Thyroid Axis; Microbiome; Micronutrients; Dietary Interventions, Graves' Disease, Hashimoto's Thyroiditis, Gut Dysbiosis, Immune Health, Gut Bacteria, Thyroid Hormone

Introduction

The thyroid gland is a very important part of our endocrine system. It is a butterfly-shaped organ that is located anterior to our trachea, just inferior to the larynx (Betts et al., 2022, p. 680). Betts et al. note that the thyroid hormones, T3 and T4, are often referred to as metabolic hormones because their levels influence the body's basal metabolic rate, the amount of energy used by the body at rest. When the T3 and T4 hormones bind to the intracellular receptors located on the mitochondria, they cause an increase in nutrient breakdown and use of oxygen to produce ATP (Betts et al., 2022, p. 683). The thyroid requires key nutrients to function properly; nutrient imbalances can increase the risk of autoimmune thyroid disease.

Autoimmune thyroid diseases (AITD), including Hashimoto's Thyroiditis and Graves' Disease, represent some of the most common endocrine disorders worldwide and are characterized by chronic immune dysregulation, thyroid dysfunction, and significant impacts on daily functioning (Ovčariček et al., 2024). Current estimates suggest that Hashimoto's Thyroiditis affects approximately 10-12% of the population, while Graves' Disease affects an additional 1-1.5%, underscoring the substantial public health burden associated with these conditions (Fröhlich & Wahl, 2019). Autoimmune thyroid diseases are common endocrine disorders that create a significant health burden, and recent research has increased interest in the gut microbiome as a possible contributor to disease development and symptom persistence.

Although pharmacological and surgical treatments remain central to autoimmune thyroid disease management, some patients continue to experience persistent symptoms despite standard care. As individuals with AITD often experience persistent symptoms, fluctuating thyroid hormone levels, and ongoing medication needs, there is growing interest in complementary strategies that may complement conventional medical treatment and improve patient-reported outcomes (Larsen et al., 2022). This creates interest in evidence-informed complementary strategies, including dietary interventions that may influence inflammation, immune regulation, and quality of life. Long-term medication treatments can also affect organ health when not appropriately supported by nutrition therapy (Mohn ES et al., 2018). For patients who continue to experience symptoms despite standard treatment, evidence-informed complementary interventions may be clinically relevant, particularly when they target inflammation, nutrient deficiencies, and quality-of-life outcomes.

The gut-thyroid axis is relevant to autoimmune thyroid disease because the gut microbiome may influence immune regulation, intestinal permeability, micronutrient absorption, and inflammatory pathways that affect thyroid function (Knezevic et al., 2020). Stress, ultra-processed foods, antibiotics, environmental toxins, and sedentary lifestyles can all contribute to gut microbiome dysbiosis (Spiller et al., 2025; Min'an Zhao et al., 2023; Monda et al., 2017). The gastrointestinal tract often plays a distinct role in the development of autoimmune thyroid disease (Legakis et al., 2023). The gut is home to 70% of the immune system (Legakis et al., 2023). Twenty percent of T4 conversion to T3 takes place in the gut (Kellman, 2021, p. 87). Gut dysbiosis has been associated with altered nutrient absorption and thyroid hormone metabolism, although causal relationships have not been conclusively established in human autoimmune thyroid disease populations.

Shulhai et al. note that nutrition is central to thyroid health, with key micronutrients like iodine, selenium, iron, zinc, and Vitamin B12 directly influencing hormone synthesis, regulation, and immune function. A balanced, nutrient-dense diet prevents dysfunction, while specific deficiencies can lead to autoimmune diseases, hypothyroidism, or hyperthyroidism (Shulhai et al., 2024). When the thyroid is starved of one or more essential nutrients, thyroid disease can develop. An example is iodine; it is required for the synthesis of T3 and T4 (Betts et al., 2022, p. 684). Other factors outside of micronutrients can influence a patient's thyroid antibodies. Evidence examining gluten-free diets in Hashimoto's thyroiditis remains limited. Some studies have reported reductions in thyroid antibody levels among selected patient populations, particularly those with concurrent gluten sensitivity or celiac disease; however, evidence remains insufficient to recommend a gluten-free diet universally for all patients with Hashimoto's thyroiditis. Raphael Kellman, MD, states that he has seen many patients successfully manage their thyroid function by changing only their food choices. (Kellman, 2021, p. 123).

While many studies acknowledge the existence of a thyroid-gut axis, few investigate which nutrition strategies are most effective, how they influence the microbial composition of AITD patients, or whether individualized nutrition plans based on microbiome testing can improve outcomes for individuals with thyroid dysfunction or autoimmune thyroid disease. Future adequately powered human studies are necessary to evaluate the impact of the gut microbiota on thyroid function and disease (Knezevic et al., 2020). This paper argues that nutrition-based interventions may complement conventional treatment for autoimmune thyroid disease by supporting thyroid hormone regulation, reducing inflammatory burden, correcting nutrient deficiencies, and promoting healthier gut microbial balance. However, the current evidence remains limited by inconsistent dietary protocols, insufficient human trials, and uncertainty regarding the clinical usefulness of individualized microbiome testing. By evaluating nutrient needs, gut microbial patterns, and existing dietary strategies, this paper identifies evidence-informed approaches and key gaps in personalized nutrition interventions for autoimmune thyroid disease.

Problem/Thesis Statement

Autoimmune thyroid diseases (AITD), including Hashimoto's Thyroiditis and Graves' Disease, are common endocrine disorders associated with immune dysregulation, thyroid dysfunction, and reduced quality of life (Ovčariček et al., 2024). Hashimoto's Thyroiditis affects an estimated 10-12% of the population, and Graves' Disease affects an additional 1-1.5%, demonstrating the substantial public health burden posed by these conditions (Fröhlich & Wahl, 2019). Because individuals with AITD often experience persistent symptoms, fluctuating hormone levels, and ongoing medication needs, there is growing interest in supportive strategies that may complement standard medical care (Larsen et al., 2022).

The specific problem is that the role of the gut microbiome in autoimmune thyroid disease remains incompletely understood, particularly in relation to nutrition-based and microbiome-targeted approaches. Emerging literature suggests that gut microbiota composition may be associated with immune regulation, inflammatory signaling, intestinal barrier function, and thyroid hormone metabolism in individuals with autoimmune thyroid disease (Fröhlich & Wahl, 2019). However, existing studies primarily describe associations between gut dysbiosis and autoimmune thyroid disease rather than establishing whether dietary modification, probiotic or prebiotic use, micronutrient correction, or microbiome-informed nutrition strategies improve thyroid, immune, gut, or overall patient health.

This gap makes it difficult to determine what the literature currently supports regarding nutrition and microbiome-focused approaches for adults with autoimmune thyroid disease. Therefore, this narrative literature review will examine current evidence on the gut–thyroid axis, gut microbiome composition, immune regulation, dietary modification, microbial metabolite production, and microbiome-targeted nutrition strategies in autoimmune thyroid disease. There is evidence that dysbiosis has been consistently associated with autoimmune thyroid diseases (Bronikowski et al., 2026). The review will focus on outcomes including thyroid autoantibodies, thyroid hormone markers, inflammation, gastrointestinal symptoms, and patient-reported symptom burden, while identifying gaps in the evidence for individualized microbiome testing and personalized nutrition approaches.

Purpose Statement

The purpose of this narrative literature review is to discuss the current literature on the association of diet and gut microbiome composition in patients with autoimmune thyroid disease (AITD). Examining current evidence on the diet and gut microbiome in adults with AITD, including Hashimoto's Thyroiditis and Graves' Disease, can help discuss how proper nutrition, or lack thereof, can play a driving factor in AITD patients. Although AITD is common, the specific role of gut dysbiosis in immune regulation, inflammation, thyroid function, and the potential impact of nutrition-based or microbiome-targeted interventions remains unclear. This review will cover research on the gut-thyroid axis, dietary modification, microbial metabolites, and microbiome-focused nutrition strategies to identify major themes in the literature and evaluate theory-based and evidence-based recommendations for improving thyroid-related biomarkers, inflammation, gastrointestinal symptoms, and patient-reported outcomes.

Research Question

What does current literature suggest about the role of gut dysbiosis in autoimmune thyroid disease, and how may dietary or microbiome-focused interventions support thyroid function, immune regulation, and symptom burden in adults?

Search Strategies

This paper provides an overview of thyroid health, autoimmune thyroid disease (AITD), the importance of gut health in autoimmune conditions, and dietary strategies to support thyroid function. The literature search was conducted across major academic databases, including PubMed, Google Scholar, ScienceDirect, Frontiers, MDPI, Nature, and Elsevier journals. Additional peer-reviewed and authoritative sources, such as OpenStax and professional medical organizations (e.g., the British Thyroid Foundation), were included when relevant to clinical guidance. A total of 31 references were reviewed. Incorporating research published between 2005 and 2026 provided substantial evidence supporting the association between gut microbiome dysbiosis and autoimmune thyroid disease in U.S. adults. Two books were also referenced to provide conceptual and clinical context. Priority was given to recent literature (2017–2026) to capture emerging research on the microbiome and thyroid, although earlier foundational work was included when appropriate.

The review focused on human studies involving adults with autoimmune thyroid disorders (Hashimoto's Thyroiditis and Graves' Disease), hypothyroidism, hyperthyroidism, or thyroid-related metabolic dysfunction. Included articles examined relationships between the gut microbiome and thyroid function, immune mechanisms, intestinal barrier integrity, dietary patterns, micronutrient intake, and nutrition-based therapies. Some animal studies were incorporated when mechanistic insights were directly relevant to human physiology. Approximately 65 records were initially identified through database searches. Titles, abstracts, and introductions were screened for relevance to the gut–thyroid axis, autoimmune thyroid disease, intestinal microbiota, or nutrition-related interventions. Articles meeting inclusion criteria were reviewed in full, and data were extracted regarding study design, population characteristics, microbiome composition, immune health, dietary factors, intestinal permeability, and clinical outcomes. Clinical findings across studies were compared to identify converging evidence and gaps in the literature.

The final set of sources included clinical studies in humans and animals, observational studies, and expert reviews. Studies focused exclusively on children were

excluded to maintain an adult population focus. Research on thyroid cancer was excluded, as the review centered specifically on autoimmune thyroid disease. Weight or gender were not included as criteria, and studies involving all adult demographics were considered. Articles examining gut microbiota in unrelated diseases without connecting findings to thyroid function were excluded, as were opinion pieces and non-scholarly web content. Key search terms included *gut microbiota*, *intestinal microbiome*, *gut–thyroid axis*, *autoimmune thyroid disease*, *Hashimoto's Thyroiditis*, *Graves' Disease*, *intestinal permeability*, *dysbiosis*, *nutrition*, *micronutrients*, *selenium*, *iodine*, *gluten*, *ultra-processed foods*, *immune mechanisms*, and *thyroid physiology*.

Theoretical Framework

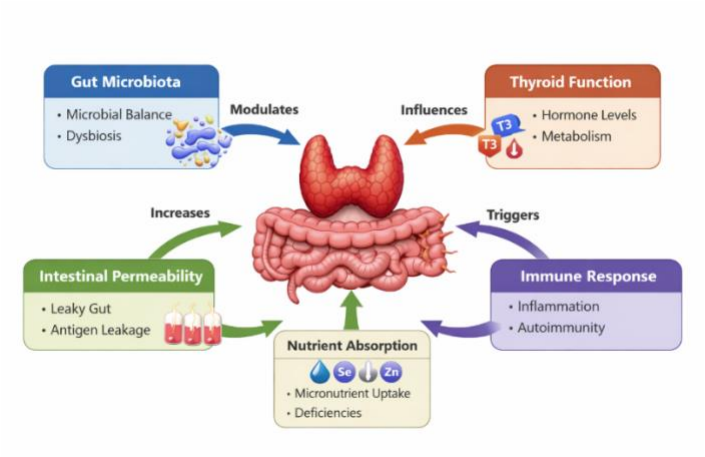

Figure 1. Gut-Thyroid Axis Framework

The gut-thyroid axis framework (Figure 1) illustrates how the gut microbiota, intestinal permeability, nutrient absorption, immune response, and thyroid function interact to maintain endocrine and immune homeostasis. In this model, the thyroid gland and intestines are positioned centrally to represent the bidirectional relationship between gastrointestinal health and thyroid regulation. Five interrelated physiological components are emphasized within this framework. First, the gut microbiota plays a critical role in modulating intestinal integrity and influencing nutrient bioavailability. Second, intestinal permeability, often increased in states of dysbiosis, permits antigens to enter systemic circulation, thereby triggering inflammation and contributing to autoimmune activity. Third, nutrient absorption directly affects thyroid hormone synthesis and metabolism; deficiencies in essential micronutrients such as iodine, selenium, iron, and zinc can impair thyroid function. Fourth, the immune response interacts with these processes, as inflammation and autoimmunity can further disrupt thyroid regulation. Finally, thyroid function influences hormone levels and metabolic activity by affecting cellular processes, mitochondrial function, and overall energy utilization.

Taken together, this framework demonstrates that gut health and thyroid function operate as an interconnected system. Proposed models suggest that dysbiosis and impaired intestinal barrier function may contribute to immune dysregulation associated with autoimmune thyroid disease, but most available evidence remains associative. Nutrition-based and microbiome-focused interventions are therefore conceptualized as modifiable factors with the potential to improve thyroid, immune, and gastrointestinal outcomes.

Review of Literature

Dietary Interventions	Influence microbiome composition and immune tone.	Anti-inflammatory, fiber-rich diets improve antibodies and quality of life.	Moderate.	Protocols vary; probiotic strain data inconsistent.
Medication–Nutrient Interactions	Gut health affects levothyroxine absorption.	Adequate micronutrients enhance medication efficacy.	Moderate.	Requires individualized monitoring; limited comparative data.
Evidence Gaps	–	Need for large, controlled human trials; personalized microbiome testing remains investigational.	–	Personalized nutrition still experimental; avoid over-claiming clinical utility.
Dietary Interventions	Influence microbiome composition and immune tone.	Anti-inflammatory, fiber-rich diets improve antibodies and quality of life.	Moderate.	Protocols vary; probiotic strain data inconsistent.
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Medication–Nutrient Interactions	Gut health affects levothyroxine absorption.	Adequate micronutrients enhance medication efficacy.	Moderate.	Requires individualized monitoring; limited comparative data.
Evidence Gaps	–	Need for large, controlled human trials; personalized microbiome testing remains investigational.	–	Personalized nutrition still experimental; avoid over-claiming clinical utility.

Figure 2. Nutritional and Gut-Related Mechanisms in Autoimmune Thyroid Disease

Micronutrients and Thyroid Function

Adequate intake of micronutrients is essential for maintaining normal thyroid hormone synthesis, conversion, and immune regulation. Nutritional factors are modifiable contributors to thyroid health, and deficiencies in key nutrients can impair thyroid function or increase susceptibility to autoimmune thyroid disease (AITD) (Leung et al., 2018; Shulhai et al., 2024). Macronutrients support energy production and hormone synthesis, while micronutrients, including iodine, selenium, iron, zinc, copper,

magnesium, vitamin A, vitamin B12, and vitamin D, are required for enzymatic reactions, cellular protection, and metabolic regulation (Espinosa-Salas et al., 2026).

Iodine is central to thyroid hormone production, with the thyroid storing 70-80% of the body's iodine (Chung et al., 2014). As noted in the document, *"Without an adequate intake of iodine from nutrition, thyroid function can diminish."* Excessive iodine intake may also temporarily suppress hormone synthesis (Shulhai et al., 2024). Selenium deficiency is associated with hypothyroidism, thyroid cancer, and both Hashimoto's Thyroiditis (HT) and Graves' Disease (GD) (Wang et al., 2023). Iron is required for thyroid peroxidase activity, and iron deficiency reduces hormone production (Shulhai et al., 2024; Bassi, 2025). Vitamin D influences immune modulation, and low levels may reflect malabsorption or limited sun exposure rather than causation (Palanca et al., 2022). Because gut dysbiosis can impair nutrient absorption, micronutrient deficiencies may both contribute to and result from disrupted gut terrain, linking nutrition directly to thyroid dysfunction. Because each patient is unique, the nutrition intervention should cater to that patient's individual needs.

Gut Dysbiosis and Microbial Composition in AITD

The gut microbiome plays a central role in gastrointestinal homeostasis and may influence thyroid disorders through nutrient absorption, immune signaling, and intestinal barrier integrity (Legakis et al., 2023; Fröhlich & Wahl, 2019). The gut contains approximately 39 trillion bacteria, more than the number of human cells, highlighting its physiological significance (Fröhlich & Wahl, 2019). Four major bacterial phyla dominate the intestinal flora: *Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria* (Arumugam et al., 2011). Dysbiosis alters the balance of beneficial bacteria that support tight junction integrity and produce metabolites essential for thyroid health (Vancamelbeke & Vermeire, 2017). In HT, studies report increased *levels of Bacteroides and Prevotella*, and decreased levels of *Lactobacillus* and *Bifidobacterium* (Chen et al., 2024). In GD, dysbiosis is characterized by increased *Bacteroidetes* and *Actinobacteria* and a higher *Firmicutes*-to-*Bacteroidetes* ratio (Zhu et al., 2024). Reduced short-chain fatty acid (SCFA) producing bacteria may contribute to GD development (Deng et al., 2023). Gut bacteria also influence micronutrient availability. Selenium correlates positively with *Bifidobacterium adolescentis*, while iron shows negative associations with *Lactobacillaceae* and *Bifidobacterium spp*. (Fröhlich & Wahl, 2019).

In HT and GD patients, most experience related symptoms, but the symptoms can act in opposite ways. Gut microbiome changes are noticed in both. The observed bacterial balance varies between HT and GD. Gastrointestinal symptoms are present in both GD and HT. GD patients will experience frequent or loose bowel movements, while HT patients will experience bloating and constipation. Rapid weight loss is common in some GD patients, whereas weight gain is common in HT patients. Mood can be affected as well, with GD patients feeling anxious and irritable, while HT patients feel depressed and sluggish. Research shows that patients with GD and HT frequently suffer from a disrupted intestinal barrier (often termed "leaky gut") and microbiome imbalances (Zheng et al., 2021).

Intestinal Permeability and Autoimmune Activation

Intestinal barrier integrity is a critical component of the gut-thyroid axis. Tight junctions prevent antigens and toxins from entering systemic circulation, but dysbiosis can weaken these junctions, increasing intestinal permeability (Vancamelbeke & Vermeire, 2017). When tight junctions are compromised, pathogens and undigested particles

penetrate the sub-epithelium, triggering inflammation and autoimmunity (Fasano & Shea-Donohue, 2005). Molecular mimicry may occur when leaked particles resemble thyroid tissue, prompting the immune system to mistakenly attack the thyroid (Knezevic et al., 2020). This can trigger AITD. Increased permeability also reduces absorption of essential minerals such as selenium, zinc, and iron, further impairing thyroid hormone synthesis and conversion. This mechanistic pathway is strongly supported by biological evidence, though large human trials remain limited.

Immune Regulation and Inflammatory Pathways

Because 70% of the immune system resides in the intestinal mucosa, gut microbiome composition directly influences immune development, tolerance, and inflammatory signaling (Legakis et al., 2023). Dysbiosis disrupts immune maturation and promotes inflammatory dysregulation, increasing the risk of autoimmune diseases such as HT and GD (Knezevic et al., 2020). Compromised intestinal barriers expose immune cells to foreign antigens, amplifying inflammation and perpetuating autoimmunity. Chronic inflammation further increases intestinal permeability, creating a self-reinforcing cycle of dysbiosis and immune activation (Fasano & Shea-Donohue, 2005). HT and GD involve distinct immune pathways. HT is typically Th1-dominant, while GD is more Th2-dominant, yet both are influenced by gut-mediated immune dysregulation.

Antibodies in GD and HT patients can differ significantly. Both AITD involve antibodies of thyroperoxidase and thyroglobulin. In GD patients, TRAb (Thyrotropin Receptor Antibodies) are present, continuously stimulating the thyroid and leading to overproduction of thyroid hormones (De Bruin et al., 1990). In HT, TPOAb (anti-TPO) and TgAb (anti-Tg) levels remain elevated for years, if not permanently (Liu et al., 2023). Microbial metabolites act as vital chemical messengers that regulate immune tolerance and inflammation. Gong et al. (2021) note that specific byproducts, such as Short-Chain Fatty Acids (SCFAs), butyrate, acetate, and propionate, help balance AITD antibodies by promoting the differentiation of Regulatory T (Treg) cells. These Treg cells suppress the overactive B cells that produce autoantibodies, such as TPOAb, TgAb, or TRAb (Gong et al., 2021). Treg cells act as peacekeepers, preventing B cells from mistakenly attacking the thyroid.

Microbial Metabolites and Thyroid Function

Microbial metabolites, particularly short-chain fatty acids (SCFAs), play an important role in maintaining epithelial integrity, regulating inflammation, and supporting metabolic processes relevant to thyroid function. Reduced SCFA-producing bacteria have been observed in GD, suggesting a potential role in disease development (Deng et al., 2023). SCFAs also influence nutrient absorption and immune tolerance, linking microbial metabolism to thyroid hormone regulation and autoimmune activity.

Dietary Interventions and Nutrition-Based Strategies

Diet is a modifiable factor that influences both gut microbiome composition and thyroid function. Aziz et al. (2024) reviewed evidence supporting the role of dietary patterns in maintaining gut health and, more broadly, reducing inflammation. While these findings support the biological plausibility of nutrition-based approaches, they do not constitute direct evidence of intervention for autoimmune thyroid disease outcomes. Some nutrition strategies that should be included involve increasing dietary fiber and plant diversity, reducing ultra-processed foods, incorporating fermented foods, correcting micronutrient deficiencies, and supporting medication absorption. Probiotics and

prebiotics may be beneficial when evidence supports specific strains, but their use remains promising rather than definitive (Skrypnik & Suliburska, 2018; Deng et al., 2023). Nutrition also influences the effectiveness of thyroid medications, as deficiencies impair hormone conversion and absorption (Shulhai et al., 2024; Ludgate & Hayes, 2026).

Evidence Gaps

Despite growing interest in nutrition-based and microbiome-focused interventions, several gaps limit clinical application. Additional large, controlled human trials are needed to further strengthen the data. Dietary protocols that combine varied, diverse, or mixed eating plans rather than a single, uniform approach need to be studied. The limited evidence for personalized microbiome testing needs further advancement. More studies are needed to address inconsistencies in probiotic strain data. There are too few studies comparing HT versus GD outcomes. Research is also limited on patient-reported outcomes on fatigue levels, mood, GI symptoms, etc. Current evidence supports nutrition as a complementary strategy, but stronger clinical trials are needed to clarify the effectiveness of targeted dietary and microbiome interventions.

Recommendations from the Literature

The literature supports integrating nutrition and clinical care for patients with autoimmune thyroid disease by promoting whole food, anti inflammatory dietary patterns, increasing dietary fiber and plant diversity, and using microbiome targeted therapies cautiously while maintaining standard medical monitoring. Specifically, clinicians and dietitians should encourage Mediterranean style or other anti inflammatory eating patterns that reduce ultra processed foods and emphasize diverse plant sources and fermented foods to support microbial diversity and intestinal barrier function (Abbott et al., 2019; Aziz et al., 2024). Abott et al. (2019) noted in their study that sixteen women (n = 16) completed the SF-36 and MSQ before and after the 10-week program. Participants showed significant improvements in HRQL across all SF 36 subscales, with the largest gains in physical role functioning, emotional role functioning, vitality, and general health. MSQ scores dropped sharply, from 92 (SD 25) to 29 (SD 20). Thyroid function markers (TSH, free/total T4, free/total T3; n = 12) and thyroid antibodies (n = 14) showed no significant change. Inflammation decreased, with hs CRP falling 29% ($p = 0.0219$) from 1.63 mg/L (SD 1.72) to 1.15 mg/L (SD 1.31). Assessment of micronutrient status may be appropriate when clinical risk factors, symptoms, laboratory findings, or dietary histories suggest potential deficiencies. When deficiencies are identified, correction may support thyroid function and overall health (Chung et al., 2014; Shulhai et al., 2024; Wang et al., 2023).

Probiotics and prebiotics may be considered when evidence supports specific strains or clinical indications, but their use remains promising rather than definitive and should be documented with strain, dose, and duration and followed by outcome assessment (Skrypnik & Suliburska, 2018; Deng et al., 2023). Ongoing medical monitoring (TSH, free T4/T3, antibodies) and attention to medication-nutrient interactions are essential to avoid overtreatment and optimize levothyroxine absorption and efficacy (Ochani et al., 2022; Mohn et al., 2018). Finally, individualized microbiome testing should not be presented to patients as a diagnostic or therapeutic solution; current evidence is largely associative and preliminary, so clinicians should avoid overclaiming its clinical utility and instead frame such testing as investigational until stronger, reproducible clinical data are available (Fröhlich & Wahl, 2019; Knezevic et al., 2020).

Conclusion

Current evidence demonstrates that the gut microbiome plays a meaningful and biologically plausible role in AITD, influencing intestinal permeability, micronutrient absorption, immune regulation, and thyroid hormone metabolism. Across the literature, consistent patterns of dysbiosis, such as reduced *Lactobacillus* and *Bifidobacterium* in HT and altered *Firmicutes: Bacteroidetes* ratios in GD suggest that gut imbalance may contribute to both the onset and persistence of thyroid dysfunction. Micronutrient deficiencies in iodine, selenium, iron, zinc, and vitamin D further connect gut health to thyroid regulation, as impaired absorption and chronic inflammation can exacerbate autoimmune activity. Although some dietary interventions show promise in improving patient-reported outcomes and inflammatory measures, evidence on their effects on thyroid antibodies and thyroid function remains inconclusive.

However, the evidence base remains limited by small sample sizes, diverse dietary protocols, inconsistent probiotic findings, and a lack of adequately powered human trials. Much of the current research is associative rather than causal, and the clinical utility of personalized microbiome testing is still uncertain. These limitations underscore the need for rigorous, controlled studies that directly evaluate how nutrition-based and microbiome-focused interventions influence thyroid biomarkers, immune pathways, and patient-reported outcomes in adults with AITD.

In answering the research question, this review finds that gut dysbiosis is consistently associated with AITD, and emerging evidence suggests that dietary and microbiome-supportive interventions may help regulate thyroid function, reduce inflammation, and alleviate symptom burden. The practical significance of these findings is substantial: nutrition represents a modifiable, accessible, and potentially impactful adjunct to conventional thyroid treatment. While not a replacement for medical therapy, evidence-informed dietary strategies may enhance medication efficacy, support immune balance, and improve overall well-being for individuals with HT or GD. Continued research will be essential to refine these approaches and establish clear, clinically validated guidelines for integrating nutrition and gut-focused care into AITD management.

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