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The Gut–Thyroid Axis: Emerging Insights into the Role of Intestinal Microbiota in Thyroid Physiology and Disease

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Abstract

Background:

The human gut microbiota has emerged as a key regulator of host metabolism, immune responses, and endocrine signaling. Recent research suggests that interactions between intestinal microorganisms and thyroid physiology form a complex regulatory network referred to as the gut–thyroid axis.

Aim:

This review aims to summarize current evidence regarding the relationship between gut microbiota composition and thyroid function, with particular emphasis on mechanisms linking microbial metabolites, immune regulation, and micronutrient metabolism with thyroid hormone homeostasis.

Methods:

A narrative review of recent literature was conducted, focusing on studies investigating associations between gut microbiota and thyroid disorders, including autoimmune thyroid diseases, hypothyroidism, Graves' disease, and thyroid cancer.

Results:

Accumulating evidence indicates that intestinal microbiota may influence thyroid function through several mechanisms, including modulation of micronutrient absorption (iodine, selenium, and iron), regulation of immune responses, and production of microbial metabolites such as short-chain fatty acids. Dysbiosis has been consistently associated with autoimmune thyroid diseases and may contribute to the pathogenesis of thyroid cancer and other thyroid

dysfunctions. Emerging studies also suggest that probiotic supplementation and dietary interventions targeting the microbiome may improve thyroid-related outcomes.

Conclusion:

The gut–thyroid axis represents a promising area of research in endocrine physiology. Understanding the complex interactions between intestinal microbiota and thyroid function may provide new insights into the pathogenesis of thyroid diseases and open novel therapeutic opportunities.

Keywords

gut microbiota, thyroid hormones, gut–thyroid axis, autoimmune thyroid disease, Hashimoto thyroiditis, Graves’ disease, thyroid cancer, microbiome, endocrinology

Introduction

The human gastrointestinal tract hosts a highly complex and dynamic community of microorganisms collectively known as the gut microbiota. This microbial ecosystem plays a fundamental role in maintaining host metabolic balance, immune homeostasis, and overall physiological function. Advances in sequencing technologies over the past decade have significantly expanded our understanding of the human microbiome and its involvement in multiple regulatory pathways within the host organism. Increasing evidence indicates that intestinal microbiota contributes to numerous biological processes including nutrient metabolism, immune regulation, maintenance of intestinal barrier integrity, and endocrine signaling [17,26]. Consequently, disturbances in microbial composition, commonly referred to as dysbiosis, have been associated with a wide range of metabolic, inflammatory, and autoimmune disorders.

Among endocrine organs potentially influenced by microbial activity, the thyroid gland has recently attracted growing scientific attention. Thyroid hormones, including triiodothyronine (T3) and thyroxine (T4), are essential regulators of metabolic rate, thermogenesis, growth, and energy homeostasis. The synthesis and metabolism of thyroid hormones depend on the availability of several micronutrients, including iodine, selenium, iron, and zinc. These nutrients are absorbed primarily in the gastrointestinal tract, suggesting that intestinal microbiota may indirectly influence thyroid hormone production and metabolism through modulation of nutrient bioavailability and intestinal absorption processes [4,7].

In addition to nutrient metabolism, the gut microbiota plays a crucial role in regulating immune responses. The intestinal immune system constantly interacts with microbial communities, maintaining a balance between immune tolerance and inflammatory responses. Disruption of this balance may contribute to systemic immune dysregulation and the development of autoimmune diseases. Increasing evidence suggests that microbial dysbiosis may influence immune pathways involved in the pathogenesis of autoimmune thyroid diseases, including Hashimoto thyroiditis and Graves' disease [20,27]. Changes in microbial diversity and composition observed in patients with these conditions further support the hypothesis that intestinal microbiota may play a role in thyroid autoimmunity.

The concept of the gut–thyroid axis has been proposed to describe the bidirectional interaction between intestinal microbiota and thyroid physiology. On one hand, microbial communities may influence thyroid function through several mechanisms including modulation of micronutrient absorption, production of microbial metabolites, and regulation of immune responses. On the other hand, thyroid hormones may affect gastrointestinal motility, intestinal permeability, and microbial composition, suggesting a complex feedback relationship between endocrine regulation and intestinal microbial ecosystems [17,24].

Recent studies have also suggested that gut microbiota may be involved in other thyroid related conditions beyond autoimmune diseases. Alterations in microbial composition have been reported in patients with hypothyroidism and thyroid cancer, indicating that microbial communities may influence thyroid pathology through metabolic, inflammatory, and immunological mechanisms [10,21]. Moreover, emerging research has demonstrated potential causal associations between specific microbial taxa and thyroid cancer risk, highlighting the growing importance of microbiome research in endocrine oncology [15].

Dietary factors represent one of the most important determinants of gut microbiota composition and may therefore influence thyroid physiology indirectly through microbiome dependent mechanisms. Nutritional patterns, micronutrient intake, and probiotic supplementation have been shown to modulate microbial composition and metabolic activity, which may in turn affect thyroid hormone metabolism and endocrine regulation [4,30]. These findings highlight the potential clinical relevance of microbiota targeted interventions in the management of thyroid disorders.

Despite the increasing number of studies exploring the relationship between gut microbiota and thyroid diseases, many aspects of the gut thyroid axis remain incompletely understood. Most available evidence is derived from observational studies, and the underlying molecular mechanisms linking microbiota with thyroid physiology require further investigation.

Therefore, the aim of this review is to summarize current evidence regarding interactions between intestinal microbiota and thyroid function, with particular emphasis on mechanisms underlying the gut thyroid axis and their potential clinical implications.

Materials and Methods

A literature review was conducted to identify studies examining the relationship between gut microbiota and thyroid function. Electronic databases including PubMed, Scopus, and Web of Science were searched for articles published up to 2026. The search strategy included combinations of the following keywords: gut microbiota, microbiome, gut thyroid axis, thyroid function, autoimmune thyroid disease, Hashimoto thyroiditis, Graves' disease, and thyroid cancer. Boolean operators AND and OR were used to refine the search results.

Studies were included if they investigated interactions between intestinal microbiota and thyroid physiology or thyroid disorders. Human studies, animal experiments, mechanistic studies, and relevant review articles published in peer reviewed journals and available in English were considered eligible. Conference abstracts without full text and studies not directly addressing microbiota thyroid interactions were excluded. Titles and abstracts were initially screened, followed by full text evaluation of potentially relevant publications. Due to heterogeneity among study designs and outcomes, a qualitative narrative synthesis of the literature was performed.

Results

Composition of Gut Microbiota and Thyroid Homeostasis

Accumulating evidence indicates that the intestinal microbiota plays an important role in maintaining thyroid homeostasis through complex metabolic and immunological interactions. The concept of the gut–thyroid axis describes a bidirectional relationship between microbial communities inhabiting the gastrointestinal tract and thyroid hormone metabolism. Several studies have demonstrated that gut microbiota may influence thyroid physiology by regulating

nutrient absorption, immune signaling, and metabolic pathways that affect thyroid hormone synthesis and activation [16,17,24].

One of the key mechanisms linking gut microbiota and thyroid function involves the metabolism of micronutrients essential for thyroid hormone synthesis. Nutrients such as iodine, selenium, iron, and zinc play a crucial role in thyroid hormone production and activation. The intestinal microbiota can influence the bioavailability of these micronutrients by affecting intestinal absorption and metabolic processes. Experimental studies have demonstrated that selenium supplementation may alter microbial composition and influence endocrine pathways associated with thyroid hormone metabolism, supporting the existence of a functional gut–thyroid interaction [1]. Nutritional factors more broadly may influence thyroid physiology through microbiota-dependent mechanisms that affect metabolic regulation and inflammatory responses [4].

In addition to micronutrient metabolism, microbial metabolites produced in the gut also appear to play a key role in endocrine regulation. Short-chain fatty acids generated through bacterial fermentation of dietary fibers have been shown to regulate immune responses, intestinal barrier function, and systemic metabolic pathways. These metabolites may indirectly influence thyroid hormone metabolism by modulating inflammatory signaling pathways and immune regulation [7,32]. Other microbiota-derived molecules may interact with neuroendocrine signaling systems and influence host hormonal regulation, suggesting that microbial activity in the gut may affect endocrine pathways beyond direct metabolic interactions [11].

Gut Dysbiosis and Hypothyroidism

Several clinical studies have reported alterations in gut microbial composition in patients with hypothyroidism. Changes in microbial diversity and abundance have been observed in individuals with primary hypothyroidism, suggesting that dysbiosis may influence thyroid hormone metabolism and endocrine homeostasis [10]. The mechanisms underlying this association may involve microbial regulation of immune responses, intestinal permeability, and systemic inflammatory processes.

Disruption of the intestinal barrier caused by microbial imbalance may allow microbial metabolites and inflammatory mediators to enter systemic circulation. These factors may contribute to systemic immune activation and inflammatory signaling pathways that affect

thyroid hormone metabolism. Such interactions highlight the complex relationship between gut microbial communities and endocrine regulation [41].

Dietary patterns may further influence the relationship between microbiota and thyroid dysfunction. Nutritional interventions targeting microbial composition have been proposed as potential supportive strategies for thyroid disorders. Studies investigating dietary patterns associated with gut microbiota diversity have demonstrated correlations between diet-induced microbial changes and thyroid hormone levels [30]. Nutritional management strategies may therefore influence thyroid function by modulating gut microbial composition and metabolic activity [34].

Gut Microbiota and Autoimmune Thyroid Diseases

A substantial body of evidence suggests that gut microbiota plays an important role in autoimmune thyroid diseases, including Hashimoto thyroiditis and Graves' disease. Several systematic reviews and meta-analyses have reported significant alterations in microbial diversity and composition in patients with autoimmune thyroid disorders compared with healthy individuals [20,28,35]. Reduced microbial diversity and increased abundance of pro-inflammatory bacterial taxa have been observed in multiple studies investigating gut microbiota in autoimmune thyroid diseases.

Immune-mediated mechanisms appear to play a central role in these interactions. The intestinal microbiota regulates immune tolerance and inflammatory responses through interactions with immune cells and signaling pathways. Dysbiosis may disrupt immune homeostasis and promote autoimmune responses targeting thyroid tissue [12,39]. Additionally, microbial dysbiosis may increase intestinal permeability, allowing bacterial antigens to enter systemic circulation and stimulate inflammatory responses that contribute to autoimmune disease development.

Several studies have also suggested that microbial antigens may trigger autoimmune responses through molecular mimicry. Structural similarities between microbial antigens and thyroid proteins may lead to cross-reactive immune responses that contribute to autoimmune thyroid disease development [39]. These mechanisms support the hypothesis that intestinal microbiota may act as a trigger for autoimmune processes affecting thyroid function.

Recent Mendelian randomization studies provide additional evidence supporting a causal relationship between gut microbiota and autoimmune thyroid disease. Genetic analyses have

suggested that specific microbial taxa may influence the risk of autoimmune thyroid disorders, further highlighting the potential role of gut microbiota in disease pathogenesis [2,29].

Gastrointestinal disorders may also influence thyroid autoimmunity through microbiota-related mechanisms. For example, celiac disease has been associated with altered gut microbiota composition and increased prevalence of autoimmune thyroid diseases, suggesting that intestinal immune dysregulation may contribute to thyroid autoimmunity [6].

Gut Microbiota and Graves' Disease

Several studies have specifically investigated microbial alterations in patients with Graves' disease. Altered gut microbiota composition has been reported in individuals with Graves disease and Graves' orbitopathy, suggesting that microbial dysbiosis may influence disease progression through immune and inflammatory pathways [13]. The interaction between microbial communities and host immune responses may contribute to inflammatory processes involved in the pathogenesis of Graves' disease.

Therapeutic interventions targeting microbiota have also been explored in patients with Graves' disease. Clinical studies have demonstrated that probiotic supplementation combined with standard antithyroid therapy may improve thyroid hormone levels and immune responses in patients with Graves' disease [14]. These findings suggest that modulation of gut microbiota may represent a promising therapeutic strategy for autoimmune thyroid diseases.

Gut Microbiota and Thyroid Cancer

Emerging evidence indicates that gut microbiota may also influence thyroid carcinogenesis. Several studies have reported differences in gut microbial composition between patients with thyroid carcinoma and healthy individuals [21]. Microbial dysbiosis has also been associated with euthyroid thyroid cancer, suggesting that microbial imbalance may contribute to tumor development through inflammatory and metabolic mechanisms [3].

In addition to intestinal microbiota, microbial communities have also been identified within tumor tissues. Intratumoral microbiota may influence tumor development through interactions with the immune system and tumor microenvironment [26]. These microbial communities may regulate inflammatory signaling pathways, immune responses, and metabolic interactions that contribute to tumor progression.

Recent research has further explored the relationship between gut microbiota and thyroid cancer through genetic epidemiological approaches. Mendelian randomization studies have suggested potential causal associations between specific microbial taxa and thyroid cancer risk, supporting the hypothesis that microbiota may influence tumor development [15].

Microbiota alterations may also occur during thyroid cancer treatment. Changes in oral microbiota composition have been reported in patients undergoing radioiodine therapy, particularly in individuals experiencing xerostomia following treatment [31]. These findings suggest that microbiota may influence treatment-related side effects and may represent a potential target for supportive interventions in cancer therapy.

Microbiota-Related Mechanisms in Endocrine and Immune Regulation

The interactions between gut microbiota and thyroid physiology are closely linked with broader mechanisms of immune and metabolic regulation. The gut microbiome influences host metabolism, immune signaling, and inflammatory responses involved in various diseases [41]. Microbial metabolites and signaling molecules may influence endocrine pathways and contribute to systemic metabolic regulation.

Recent studies have also emphasized the role of microbiota in cancer immunity and host immune responses to tumors. Microbial communities may influence immune cell activity and inflammatory pathways involved in cancer progression and immunotherapy responses [40]. These findings highlight the potential role of microbiota as a regulator of immune and endocrine interactions in thyroid diseases.

Overall, the available evidence suggests that gut microbiota may influence thyroid physiology and disease through multiple interconnected mechanisms including nutrient metabolism, immune regulation, microbial metabolites, and inflammatory signaling pathways. The growing body of research on the gut–thyroid axis highlights the importance of intestinal microbial communities in endocrine regulation and suggests that microbiota-targeted strategies may represent promising therapeutic approaches for thyroid disorders.

Discussion

The results summarized in this review highlight the growing evidence supporting the concept of the gut–thyroid axis, a complex bidirectional interaction between intestinal microbial communities and thyroid physiology. The intestinal microbiota plays an important role in

metabolic regulation, immune homeostasis, and endocrine signaling, all of which may influence thyroid hormone metabolism and thyroid-related diseases [16–18,23].

One of the most important mechanisms linking gut microbiota and thyroid function involves the regulation of immune responses. The intestinal microbiome plays a critical role in maintaining immune tolerance and preventing excessive inflammatory responses. Dysbiosis may disrupt intestinal barrier integrity and promote systemic immune activation, which may contribute to autoimmune reactions targeting thyroid tissue [12,41]. Several systematic reviews and meta-analyses have demonstrated that patients with autoimmune thyroid diseases exhibit significant alterations in gut microbial diversity and composition compared with healthy individuals [20,28,35]. In particular, reduced microbial diversity and increased abundance of pro-inflammatory bacterial taxa have been consistently reported in patients with Hashimoto thyroiditis and Graves' disease.

Recent studies have further highlighted the potential role of intestinal microbiota in the development of autoimmune thyroid diseases. Microbial dysbiosis may influence inflammatory signaling pathways and immune regulation mechanisms involved in thyroid autoimmunity [5,25]. Molecular mimicry between microbial antigens and thyroid proteins has also been proposed as a mechanism linking gut microbiota and autoimmune thyroid disorders [39]. In addition, Mendelian randomization studies have suggested that specific microbial taxa may be causally associated with autoimmune thyroid disease risk, further supporting the role of microbiota in disease pathogenesis [2,29].

Dietary factors appear to play a crucial role in shaping gut microbial composition and influencing thyroid physiology. Nutritional patterns may modulate microbial diversity and metabolic activity in the gastrointestinal tract, thereby affecting endocrine regulation. Studies investigating diet and thyroid function suggest that dietary interventions targeting microbial composition may improve metabolic and inflammatory pathways associated with thyroid disorders [4,30,34]. Anti-inflammatory dietary patterns such as the Mediterranean diet may provide protective effects in autoimmune thyroid diseases by promoting beneficial microbial diversity and reducing inflammatory responses [38].

Another important pathway connecting gut microbiota and thyroid function involves microbial metabolites. Short-chain fatty acids produced by intestinal bacteria during fermentation of dietary fibers have been shown to regulate immune responses, intestinal barrier integrity, and

metabolic signaling pathways that may influence thyroid hormone metabolism [7,32]. Microbiota-derived molecules may also interact with neuroendocrine pathways and inflammatory signaling processes involved in endocrine regulation [11].

In addition to autoimmune thyroid diseases, emerging evidence suggests that gut microbiota may also influence thyroid carcinogenesis. Several studies have demonstrated significant differences in microbial composition between patients with thyroid carcinoma and healthy individuals [21]. Dysbiosis has also been observed in euthyroid thyroid cancer patients, suggesting that microbial imbalance may contribute to tumor development even in the absence of overt thyroid dysfunction [3]. Furthermore, recent studies have identified microbial communities within thyroid tumor tissues, indicating that intratumoral microbiota may play a role in tumor progression through immune modulation and metabolic interactions within the tumor microenvironment [26].

Genetic and epidemiological studies provide additional support for the relationship between gut microbiota and thyroid cancer. Mendelian randomization analyses have identified potential associations between specific microbial taxa and thyroid cancer risk [15]. Microbiota may also influence immune responses involved in cancer progression and immunotherapy effectiveness, highlighting its potential role in oncological outcomes [40].

Several studies have also suggested that microbiota alterations may occur in patients undergoing treatment for thyroid cancer. For example, changes in oral microbiota composition have been observed in patients receiving radioiodine therapy, particularly in individuals experiencing xerostomia following treatment [31]. These findings suggest that microbiota may contribute to treatment-related complications and may represent a potential target for supportive therapies.

Beyond disease mechanisms, microbiota-targeted therapeutic strategies are increasingly being investigated. Probiotic supplementation has been shown to improve thyroid function parameters in patients with Graves' disease when combined with conventional antithyroid therapy [14]. These findings highlight the potential clinical importance of microbiota modulation in endocrine diseases.

Recent studies also emphasize the broader role of gut microbiota in host metabolic and immune regulation. The gut microbiome participates in numerous physiological processes including nutrient metabolism, immune signaling, and inflammatory regulation, all of which may

influence endocrine homeostasis and disease development [41]. Consequently, the gut microbiota may represent an important therapeutic target in endocrine disorders, including thyroid diseases.

Despite the growing interest in the gut–thyroid axis, several limitations should be acknowledged. Many available studies remain observational and therefore cannot establish causal relationships between microbiota alterations and thyroid diseases. Differences in study design, sequencing technologies, and study populations contribute to heterogeneity among studies, which complicates direct comparisons of results.

Future research should focus on elucidating the molecular mechanisms underlying microbiota–thyroid interactions and identifying microbiota-based therapeutic strategies. A deeper understanding of the gut–thyroid axis may provide new insights into thyroid disease pathogenesis and support the development of novel microbiome-targeted interventions.

Conclusions

The growing body of evidence highlights the important role of intestinal microbiota in thyroid physiology and disease. The concept of the **gut–thyroid axis** describes a complex bidirectional interaction between gut microbial communities and thyroid function involving immune regulation, microbial metabolites, and nutrient metabolism essential for thyroid hormone synthesis and activation [16–18]. Increasing evidence suggests that alterations in gut microbiota composition may contribute to thyroid disease development through mechanisms involving immune dysregulation, inflammatory signaling pathways, and changes in metabolic activity.

Microbial dysbiosis has been consistently associated with autoimmune thyroid diseases such as Hashimoto thyroiditis and Graves' disease, as well as with hypothyroidism and thyroid cancer [20,21,35]. Microbiota-derived metabolites, particularly short-chain fatty acids, may further influence endocrine regulation by modulating immune responses and metabolic pathways involved in thyroid hormone metabolism [7,32].

Recent research also indicates that microbiota-targeted strategies, including dietary interventions, probiotic supplementation, and modulation of microbial diversity, may represent promising approaches for improving thyroid-related outcomes and supporting endocrine health [14,30,38].

Although current findings provide valuable insights into microbiota–thyroid interactions, further experimental and clinical studies are required to clarify causal mechanisms and evaluate the therapeutic potential of microbiome-based interventions in thyroid diseases.

Disclosure

Supplementary Materials

There are no supplementary data connected with this article.

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The authors declare no conflicts of interest.

Declaration of the Use of Generative AI and AI-Assisted Technologies in the Writing

Process

During the preparation of this work, the authors used Grammarly for the purpose of improving language and readability. After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the substantive content of the publication

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