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Review Article

Gut microbiota changes and its potential relations with thyroid disorders

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ABSTRACT

The human intestinal flora is composed of more than 1200 species of anaerobic and aerobic bacteria, along with bacteriophages, viruses and fungi, essential for several processes including digestive and non-digestive, being of vital importance for health, including digestive balance and immunological, hormonal and metabolic homeostasis. Micronutrients, generally trace elements (copper, iodine, iron, selenium, zinc) and vitamins (A, C, D and E), interact with the bacterial flora to generate an adequate immunological metabolism of the host. Multiple studies on the functioning of the gut microbiota (GM) have revealed an association between microbiota alterations and various pathological disorders, such as encephalitis due to antibodies against the N-methyl-D-aspartate receptor (NMDAR), anxiety, depression, early-onset cancer, type 1 diabetes, and type 2 diabetes. According to recent studies, the thyroid microbiota (TM) could play a fundamental role in the triggering of thyroid gland diseases, among which autoimmune diseases play an important role. Not only environmental triggers and predisposing genetic background cause autoaggressive damage, which affects the cellular and humoral networks of the immune system, but the GM interacts with distant organs through signals that may be part of the bacteria themselves or their metabolites. The objective of this review is to describe the current knowledge about the microbiota in the metabolism of thyroid hormones and the pathogenesis of thyroid diseases, as well as its participation in the appearance of benign nodules. and papillary cancer.

Keywords: Gut microbiota, Thyroid diseas, Hashimoto's thyroiditis, Thyroid nodules, Thyroid cancer

INTRODUCTION

The intestinal microbiota constitutes a complex ecosystem composed of bacteria, viruses, and fungi that dynamically interact with the human body. Its role in systemic homeostasis has been associated with metabolic, immunological, and neuroendocrine processes, which has led to reconsidering its relevance in pathologies beyond the digestive system.[1]

In this regard, thyroid disorders have emerged as a field of special interest, considering that thyroid function depends on the adequate bioavailability of essential micronutrients and immune modulation, both processes in which the microbiota plays a decisive role.[2]

Various studies have shown that intestinal dysbiosis, understood as the qualitative or quantitative alteration of the microbial composition, could influence the pathophysiology of the thyroid gland. On the one hand, the microbiota participates in the absorption and metabolism of iodine, iron, and selenium, essential elements for the synthesis of thyroid hormones (T3 and T4).[3] On the other hand, microbial imbalance has been linked to the activation of abnormal immune responses, favoring the onset of autoimmune diseases such as Hashimoto's thyroiditis or Graves' disease. Likewise, the production of bacterial metabolites, such as short-chain fatty acids (SCFAs), could intervene in the regulation of systemic inflammation, indirectly affecting thyroid function.[4]

Micronutrients and trace elements such as copper, iodine, iron, selenium, zinc, and vitamins (vitamin A, C, D and E) interact directly with the GM to influence host immune metabolism. They can also alter the host-microbiome symbiosis, thereby influencing micronutrient bioavailability.[5]

The growing evidence on the microbiota-thyroid axis raises the need to further characterize these interactions, not only to understand the multifactorial etiology of thyroid disorders but also to develop innovative therapeutic strategies aimed at modulating the microbiome.[6]

Within this framework, the study of the relationship between intestinal dysbiosis and thyroid dysfunction is projected as an interdisciplinary approach that integrates endocrinology, microbiology, and translational medicine, opening new diagnostic and preventive perspectives in the field of metabolic and autoimmune health. Figure 1 summarizes the relationship between GM dysbiosis and thyroid dysfunction.

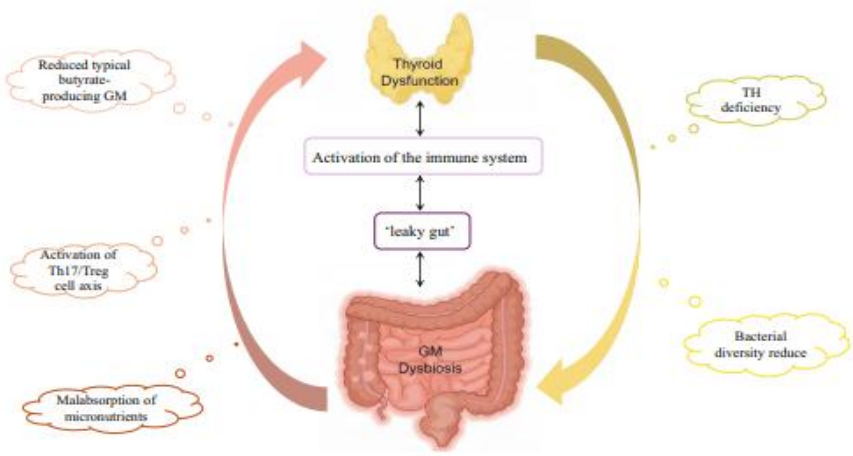

Figure 1: The interrelationship between GM dysbiosis and thyroid dysfunction.

Under physiological conditions, the thyroid gland and GM have a mutually beneficial association. When thyroid function or GM homeostasis changes, the other party would be influenced. In other words, the differences in GM can lead to the development of thyroid dysfunction, and the development of the thyroid gland can cause a series of endocrine changes, leading to the differences in GM.[3]

LITERATURE SEARCH

It is a descriptive-exploratory study type of bibliographic review. The literature search period is from 2018 to 2024 in electronic databases such as PubMed, Elsevier, and Web of Science. The keywords used in the MesH search were: GM; thyroid diseas; Hashimoto's thyroiditis; thyroid nodules; thyroid cancer.Inclusion criteria: search terms, level of evidence, summaries and keywords, exclusion criteria: not related to the topic, outside the year limit, not available; They will be classified by year, type of study and level of evidence. For eligibility, a critical reading is carried out, level of evidence, documents available for analysis and according to the topic. A total of 32 sources were obtained for analysis and synthesis.

ALTERATIONS IN THE GM IN PATIENTS WITH THYROID NODULES

Gastric mucosal cells and thyroid follicular cells originate from the same embryonic source, as the thyroid gland develops from basic intestinal cells. The gut flora contributes to the generation of thyroid hormone and the hydrolysis of thyroid hormone conjugates.[7] Although studies on the direct connection between thyroid nodules and the GM are still preliminary, some research has indicated a notable interaction between the two. Table 1 presents the most frequently analyzed characteristics based on nodule categorization by ultrasound. Some mechanisms that could affect the development or progression of thyroid nodules are described below:

The GM plays an essential role in controlling the immune system. Variations in microbiota composition can influence the immune system's response, promoting autoimmune processes that could lead to the development of thyroid disorders, such as autoimmune thyroiditis, which could be linked to the appearance of thyroid nodules.

It has been shown that certain gut bacteria have the ability to alter thyroid hormone metabolism, transforming thyroxine (T4) into its active form, triiodothyronine (T3). An unbalanced microbiota could alter this process, impacting thyroid hormone levels and promoting thyroid dysfunction. Gut dysbiosis can cause increased gut permeability, triggering the release of endotoxins and proinflammatory cytokines into the bloodstream.

This systemic inflammation could affect the thyroid gland and promote the appearance of thyroid nodules, particularly in the context of autoimmune diseases.

The butyrate pathway: It can disrupt the growth of normal cells and enhance iodine uptake in follicular thyroid carcinoma cells by restoring sodium-iodide import (NIS) function in humans.

Table 1: Characteristics found in patients with thyroid nodules classified by ultrasound.

Variables	TIRADS 1 and 2	TIRADS 3 and 4
GM species found	Unclassified *Butyrivibrio*, *Bacteroides plebeius*, *Coprococcus comes*, *Coprococcus catus*, *Roseburia hominis*, *Eubacterium eligens*, unclassified *Anaerotruncus*, *Faecalibacterium prausnitzii and Barnesiella intestinihominis*	14.25 and 23.99% respectively *Bacteroides ovatus* and *Eggerthella* unclassified unclassified *Butyrivibrio*, *Bacteroides plebeius*, *Coprococcus comes*, *Coprococcus catus*, *Roseburia hominis*, *Eubacterium eligens*, unclassified *Anaerotruncus*, *Faecalibacterium prausnitzii and Barnesiella intestinihominis*
Activated metabolic pathways	Butyrate production pathways Pyruvate fermentation Acetyl-CoA fermentation Pyruvate fermentation Amino acid biosynthesis	Cofactor or vitamin biosynthesis Cell structure biosynthesis Nucleoside and nucleotide biosynthesis and glycolysis

HUMAN GM AND HYPOTHYROIDISM

Human GM plays a critical role in body protection through metabolic, trophic and protective function.[9] Previous studies have shown gut microbiome is associated with thyroid diseases such as Graves' disease, Hashimoto's disease, thyroid nodules, and thyroid cancer but the association between intestinal flora and primary hypothyroidism remains elusive.[10] There are few new studies contemplating the association between gut microbiome and hypothyroidism.

There are different ways being studied in how gut microbiome plays a possible role in the development of hypothyroidism like amount available of short chain fatty acids, increased or decreased number of immunoglobulins and blood serum complement proteins, bacterial components such as LPS and through activation of certain signaling pathways.

The studies review share results of which of the bacteria family overgrow such as *Actinobacteria, Escherichia-Shiegella Prevotella* and a decrease amount of *Bacteroidetes* making the ratio of *Firmicutes/Bacteroides* increased.[10] There are four bacteria that can be used to classify primary hypothyroidism, *Veillonella*, *Paraprevotella*, *Neisseria*, and *Rheinheimera* with high accuracy, where *Paraprevotella* and *Veiloneella* are reduced and *Neisseria* and *Rheinheimera* are increased. But it should not be used as a diagnostic method since measuring T3 and TSH is easier. It may be possible that correcting *Neisseria* abundance could improve primary hypothyroidism symptoms.[11]

Short chain fatty acids (SCFA) can regulate the endocrine function in different organs, including the anterior pituitary gland, where they inhibit growth hormone (GH) secretion and enhance T3-induced stimulation of prolactin expression.[11] Also, regulation of colonic motility and blood flow, regulation of gastrointestinal pH, which can influence electrolytes and nutrients uptake and absorption. SCFA have been found to been downregulated in patients with primary hypothyroidism, explained due to a reduced number of bacteria such as *Veillonella* and *Paraprevotella*, that are able to produce propionate and butyrate (SCFA) that may be associated with some of the symptoms of T3 deficiency.

Decreased SCFAs may explain the gastrointestinal symptoms of primary hypothyroidism. As SCFAs increase the barrier function of the gut, their reduction in primary hypothyroidism patients allows many endotoxins to enter the body, triggering a wide range of symptoms.

Besides affecting the availability of SCFA, the composition of the intestinal microbiota affects the absorption of essential trance elements important for the thyroid gland such as iondine, iron, copper, selenium and zinc, which are crucial for the synthesis of thyroid hormones and are required for the conversion of T4 to T3, these elements are often reduced in pactients with thyroid disease.[12]

An example of how the bacteria itself can affect thyroid hormones is due to bacterial lipopolysaccharides that has been demonstrated, that can inhibit the iodothyronine deiodinase activity and decrease the level of T3 in the circulation and lead to hypothermia.[13]

In experiments with hypothyroid rats, it has been shown that intestinal microbiota dysbiosis may cause the reduced metabolism and enhance systemic inflammation through the activation of LPS-TLR4 and peptidoglycan-nod/Pglyrp 1 signaling pathway.[14] According to Sirchack et al it has been found that the immunologic status of obese and hypothyroid patients shows increased levels of immunoglobins such as A, M and G and a decrease in blood serum complement C3 and C4.

Levels of IgA and IgG depends on the reduction of *Bifidocterium*, *Lactobacillus* and increase of

Staphylococcus, *Clostridium* and *Klebsiella* that are more evident in patients with obesity and hypothyroidism.[14]

RELATIONSHIP BETWEEN GM ALTERATIONS AND THYROID CANCER

The thyroid neoplasia with increasing incidence worldwide is carcinoma. Predisposing factors have been documented to be genetic and hormonal, although recent studies show that GM can have an invisible effect on immunological and metabolic mechanisms that enhance carcinogenesis. In addition to increased thyroid function, variability and variety of GM (α-diversity) has been observed in people with malignant thyroid disease versus patients with Hashimoto's thyroiditis and hyperthyroidism.[9,10] A marker of intestinal eubiosis, the *Firmicutes/Bacteroidetes* (F/B) ratio, has been reported to be increased in patients with thyroid neoplasia, a pattern visible in malignant neoplasias such as colorectal cancer and breast cancer.[11,12]

Proteobacteria, *Klebsiella*, and *Escherichia coli* were found in certain bacterial strains that were enriched in individuals with thyroid cancer. These bacteria have been linked to inflammatory responses, DNA damage, and metabolic dysfunction, all of which could have carcinogenic effects.[13,14] In contrast, there was a decrease in genera involved in the development of SCFAs, such as *Bacteroides* and *Megamonas*, which are SCFA that regulate energy metabolism and intestinal homeostasis.[15]A study of fecal metabolites in individuals with thyroid neoplasia showed a significant change in the lipid profile, with an increase in triacylglycerols and sphingolipids. These molecules are related to the progression of neoplasia by synthesizing processes such as cell proliferation, inflammation, and apoptosis.[16]

Studies have shown that T3 and T4 directly influence lipid metabolism. In hyperthyroidism, serum triglyceride and LDL levels decrease due to increased cholesterol excretion, while in hypothyroidism, they increase. However, patients with thyroid neoplasia had normal thyroid function, suggesting that lipid abnormalities are related to mechanisms independent of thyroid hormone and the GM.[17,18]

Patients with thyroid neoplasia demonstrated not only lipid abnormalities but also changes in metabolites such as benzoic acids and flavonoids. Flavonoids act by inhibiting thyroid hormone synthesis by being alternative substrates for TPO. This could lead to increased thyroid cell proliferation and compensatory elevation of TSH.[18-20]

Phthalic acid is an endocrine disruptor, and its elevation is a key factor. It promotes cell proliferation by stimulating estrogen receptors, a process evident in breast cancer and may be important in thyroid neoplasia.Thyroid neoplasia is influenced by the intestinal microbiota, and metabolic alterations play an important role in its pathogenesis. Lipid metabolism can help us understand the molecular mechanisms for the development of therapeutic strategies.[21,22]

DISCUSION

Impact of micronutrients on the intestinal microbiota

Recent findings on the interaction between GM, micronutrient metabolism, and thyroid function suggest the existence of a bidirectional axis that may significantly influence the pathophysiology of thyroid disorders.[23,24]Although their required intake is lower than that of macronutrients, typically ranging from 1 to 100 mg per day in adults, their importance remains profound.[25] This symbiotic relationship acquires clinical relevance given that nutritional deficiencies are known risk factors for thyroid dysfunction, while alterations in the microbiota could exacerbate this risk.[25]

In particular, selenium and zinc play critical roles as enzymatic cofactors in the conversion of thyroxine (T4) to triiodothyronine (T3), a process essential for maintaining metabolic homeostasis. Studies have shown that intestinal dysbiosis compromises the bioavailability of these micronutrients, increasing susceptibility to subclinical hypothyroidism and autoimmune thyroid diseases. In turn, iron is essential for thyroid peroxidase activity, and its intestinal absorption is modulated by microbial balance; an imbalance in bacterial composition can alter this process, with direct consequences on hormone synthesis.[23]

Furthermore, iodine, a key element in the production of thyroid hormones, is also indirectly influenced by the microbiota, given that intestinal inflammation associated with dysbiosis can reduce its absorption. In addition, the production of bacterial metabolites, such as SCFAs, modulates the integrity of the intestinal mucosa and regulates immunological processes that could explain, at least in part, the association between dysbiosis and autoimmune thyroid diseases such as Hashimoto's thyroiditis.[25]

Phosphorus supplementation has been shown to increase the abundance of *Fecalibacterium* and *Pseudoflavonifractor* in the cecal digesta, as well as enhance the concentration of SCFAs. Conversely, prolonged magnesium deficiency may lead to an increase in the intestinal content of *Bifidobacteria* and *Lactobacilli*. However, further studies are needed to elucidate the association between these micronutrients and the GM. [26]

In this context, evidence suggests that microbiota manipulation through nutritional, probiotic, or prebiotic strategies could constitute a complementary therapeutic approach in patients with thyroid disorders. However, the heterogeneity of available studies and the absence of longitudinal clinical trials still limit the possibility of establishing the firm causal relationships. Therefore, an interdisciplinary approach integrating endocrinology, microbiology, as well as the nutritional sciences is

required to elucidate the molecular as well as the clinical mechanisms underlying this microbiota-micronutrient-thyroid axis.[25]

Flavonoids, found in various sources such as blueberries, can reduce the *Firmicutes/Bacteroidetes* ratio and decrease the abundance of *Proteobacteria* at the phylum level, while increasing fecal microbial diversity. Additionally, both alpha and beta diversity are altered at the phylum and genus levels, with a decrease in *Verrucomicrobia*, *Tenericutes*, and *Deferribacteres.*[23]

Effects of selenium, zinc, vitamins A, B, C, D, E and K on the intestinal microbiota

Selenium

Selenium exerts direct effects on the microbiota by influencing the expression of bacterial selenoproteins and regulating intestinal oxidative stress. Selenium deficiency has been associated with a reduced abundance of beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*, favoring a proinflammatory environment.[27]

Similarly, zinc plays a critical role in the integrity of the intestinal barrier and in modulating the immune response. Clinical studies have shown that zinc supplementation increases microbial richness and reduces the prevalence of pathobionts, suggesting therapeutic potential in intestinal dysbiosis.[27]

In addition, there is a relationship between selenium deficiency and inflammatory bowel disease, where it is present in 30.9% of cases. This produces a dysbiosis in the intestinal microbiota, alteration in motility and secretion. There may also be presence of visceral hypersensitivity and a failure in the communication between the intestine and the cerebral response.[27]

Vitamin A

Among fat-soluble vitamins, vitamin A, through its active metabolite retinoic acid, regulates T cell differentiation and IgA production, thus modulating intestinal mucosal immunity. This effect impacts the composition of the microbiota, promoting a more tolerogenic environment and less prone to inflammation.[29]

Vitamin B

Regarding water-soluble vitamins, the B vitamin family plays a particularly important role, as many intestinal bacterial species are capable of synthesizing compounds such as biotin, folate, and vitamin B12. The microbiota not only contributes to the production of these vitamins but also depends on them for growth and energy metabolism. Recent studies have shown that supplementation with B vitamins can alter the relative abundance of intestinal bacteria, modulating the production of SCFAs, metabolites essential for intestinal and systemic health.

Vitamin C

Vitamin C, for its part, acts as an antioxidant and modulator of the intestinal environment; its supplementation appears to promote the proliferation of beneficial bacteria and reduce intestinal inflammation. However, high doses could generate a paradoxical effect, adversely modifying the microbial composition and altering intestinal homeostasis.[29]

Vitamin D

Vitamin D, by interacting with the VDR receptor present in intestinal epithelial cells, influences the expression of antimicrobial peptides and the regulation of innate immunity. Deficiencies in this vitamin have been linked to lower microbial diversity and an increase in pro-inflammatory bacteria, which could explain its association with autoimmune and inflammatory bowel diseases.[31]

Vitamin E

Vitamin E, recognized for its antioxidant function, contributes to the protection of the intestinal epithelium and may promote the stability of bacteria sensitive to oxidative stress; however, human clinical studies are still limited and the results inconclusive.

Vitamin K

Vitamin K, particularly menaquinone (K2), is produced in the gut by bacteria such as *Bacteroides* and *Escherichia coli*, reflecting the symbiotic relationship between host and microbiota. Alterations in bacterial composition can compromise its endogenous synthesis and, consequently, influence coagulation processes and bone health.[32]

CONCLUSION

Human intestinal microbiota is immensely diverse, composed by various microbes like viruses, fungi and parasites, but mainly bacteria, predominantly found in the colon. The main dominant microbes found were *Firmicutes* and *Bacteroidetes*. The microbiota and its metabolites preform various functions including, nutrition, substance metabolism, immunoregulation, maintenance of structural integrity of the mucosal barrier, and protection against pathogens. It has become very clear that the genes from our normal flora can alter the expression of our genes, and so alterations of the microbial intestinal flora, may influence distant organs like the thyroid.

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