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Review Article

Hypothyroidism and Hyperthyroidism: Are they Related to the Gut Microbiome and its Metabolites?

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Abstract

Hypothyroidism and hyperthyroidism are common, more frequent in women than in men, and thyroid disease ranges from inflammatory to neoplastic lesions. Iodine deficiency is the main cause of thyroid dysfunction. Approximately 30% of the world's population suffers from iodine deficiency. Autoimmune thyroid disorders affect between 2% and 5% of the population, while thyroid cancer is the most common endocrine malignancy. The gut microbiota regulates the immune system, contributing to thyroid hormone metabolism and can generate or catabolize carcinogens, all of which are relevant to autoimmune diseases and thyroid cancer. Evidence supports a relationship between gut dysbiosis and thyroid cancer. The gut microbiome of people with thyroid disease is different from that of people without thyroid disorders. It is noted that the microbiome impacts the proper functioning of the thyroid gland, and the thyroid-gut axis exists in thyroid disease and intestinal dysbiosis. All of the above is explored in depth in this review, and there is no doubt that the relationship between thyroid disease and the gut microbiome is inextricably linked.

Keywords: *Gut Microbiome (GM); Gut Dysbiosis (GD); Intestinal Microbiota (IM); Short-Chain Fatty Acids (SCFA)*

Introduction

The gut microbiome (GM) performs diverse functions, including metabolites such as short-chain fatty acids (SCFA), secondary bile acids, modulation of immune balance, maintenance of thyroid hormone levels, and the generation or catabolism of carcinogens. SCFA are products of the anaerobic fermentation of indigestible carbohydrates from the intestinal microbiota (IM), particularly starch. There is a decreased diversity of GM, with an abundance of *Prevotella*. Studies are not straightforward, as there is a complicated interaction between the GM, thyroid hormones, and the treatments being administered [1,2]. Xie L and colleagues [3] report that, although the causal relationship between the GM and thyroid function is uncertain, the impact of its composition on thyroid function is real, detecting that the phylum Actinobacteria produces a protective effect in hypothyroidism and Deltaproteobacteria in hyperthyroidism. These microorganisms can be used as biomarkers in early diagnosis. It is essential to design studies that determine the functional potential of GM, such as whole genome sequencing and the fecal metabolome. Through a better understanding of the thyroid-gut axis, IM and its metabolites may act directly or indirectly on the thyroid by influencing intestinal micronutrient absorption, iodothyronine levels, and immune regulation [4]. The composition of IM is determined by environmental and, to a lesser extent, genetic factors. It impacts both endocrine disorders. IM

is composed of billions of bacteria and, to a lesser extent, viruses, archaea, and fungi. It has been recognized as a hidden organ system that performs trophic, metabolic, and immunological functions in the human body. Stramazzo I., *et al*. [5] confirm the existence of a bidirectional relationship between the intestine, with its microbial complex, and thyroid homeostasis, thus supporting the new entity known as the thyroid-intestine axis.

Pathophysiology

SCFA play an essential role in communication between the gastrointestinal tract and the body as a whole, participating directly or indirectly in the processes that occur. The most important are acetate, propionate, and butyrate. They primarily involve anti-inflammatory mechanisms, induction of epithelial regeneration processes, support of colonic function, as well as indirect modulation of lipid, hormonal, and energy metabolism. SCFA are considered to be related to various aspects of thyroid function, although the exact processes have not yet been described. They impact the integrity of the intestinal barrier and the regeneration of the intestinal epithelium. They protect enterocytes through nutrients; they reinforce intercellular integrity; they decrease intestinal permeability by reducing pH and cell adhesion; and they protect against pathogen invasion. Adding up their actions, we often find that SCFA s have numerous health benefits, such as immunoregulatory, anti-inflammatory, anti-obesity, antidiabetic, anticancer, cardiovascular, hepatoprotective, and neuroprotective activities [6]. Another component that influences the relationship between hypothyroidism and hyperthyroidism are secondary bile acids, with deoxycholic acid being most present in hypothyroidism, and chenodeoxycholic acid in hyperthyroidism [7]. Regarding the modulation of immune balance, thyroid hormones can affect immune cells and inflammation-related processes through complex mechanisms; T3 and T4 can modulate responses through genomic and non-genomic mechanisms [8]. How can adequate thyroid hormone function be maintained? Avoid processed foods that can disrupt thyroid function. Control iodine intake and stress. Finally, the transformation pathways in the generation or catabolism of carcinogens include nitroprocyclic hydrocarbons, polycyclic hydrocarbons and their diols; vinyl halides and dihaloalkanes [9].

Intestinal dysbiosis, hypothyroidism and hyperthyroidism. Su X and team [10], refer the difficulty in linking intestinal dysbiosis (ID) with primary hypothyroidism and analyzed the sequencing of 16S rRNA and faecal microbiota transplantation (FMT), in sick and healthy mice. There were significant differences in IM. Four intestinal bacteria *Veillonella, Paraprevotella, Neisseria* and *Rheinheimera* [11], could distinguish untreated patients with primary hypothyroidism from healthy individuals. GM imbalance in dysbiosis has been associated with Hashimoto's thyroiditis and Graves' disease.

Thyroid-gut axis in hypothyroidism and hypothyroidism

The thyroid-gut axis refers to the complex relationship between the thyroid gland, thyroid function, and homeostasis. This autoregulatory circuit includes the thyroid gland, the anterior pituitary gland, and the hypothalamus. It is involved in the production of thyroxine (T4) and triiodothyronine (T3). It helps regulate thyroid function by interacting with pituitary hormones. The gut thyroid plays a fundamental role in the development of the intestinal immune system and has a substantial impact on maintaining immune tolerance from the earliest stages of life. There is bidirectional communication between the thyroid gland and the gut thyroid, involving biochemical interactions that influence both systems. This axis generates health benefits: A healthy gut thyroid and its metabolites promote optimal thyroid function and a healthy immune system [12,13].

Intestinal barrier

The integrity of the intestinal barrier is its selective permeability to molecules of a certain size and molecular charge. The GALT (gut-associated lymphoid tissue) is activated when the intestinal barrier's ability to control antigen transport to blood vessels is lost. GALT and the proinflammatory factors produced at this time cause subclinical inflammation, initially only *in situ*. Immunocompetent cells in the intestine migrate to specific tissues and organs, which could consequently initiate persistent inflammation [14]. Immunoglobulin

A is crucial in the humoral immune response, evolutionarily selected, along with innate mucosal defenses, to protect against microbial antigens on mucosal surfaces. IgA responses are initiated in organized inductive structures, such as Peyer's patches and nasal-associated lymphoid tissues, as well as in diffuse effector tissues, such as the intestinal lamina propria and nasal mucosa. Hypermutated secretory IgAs play a significant role in regulating IM composition. Dysregulation of intestinal homeostasis in the IgA-deficient gut leads to continuous activation of immune cells and induces inflammatory processes leading to lymphangiogenesis.

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Microorganisms of the gut microbiome in hypothyroidism and hyperthyroidism

Gut bacteria undergo complex immune training, in which the host obtains a stable symbiotic environment through interdependence, reciprocal restrictions, coordination, and participation in immune processes. They can affect hypothyroidism and hyperthyroidism by influencing the absorption of iodine, selenium, and iron, altering the availability of L-thyroxine and the toxicity of PTU (propylthiouracil), and causing damage to the intestinal barrier, leading to increased permeability and activation of the immune system [15]. Faecalibacterium prausnitzii produces butyrate, which is the main energy source for colonocytes and an important epigenetic regulator of immune responses [16]. Similarly, *Prevotella* and *Oscillibacter* can reduce Th17 polarization and enhance the differentiation of anti-inflammatory regulatory T cells (Tregs) within the gut [17]. Specific IM taxa have been identified at the genetic level that are predicted to be causally related to thyroid function, which could serve as a biomarker [18].

Interactions between gut microbiota, disease, and health

Germ-free rats have smaller thyroids than normal rats, supporting the hypothesis that IM plays a prominent role in host thyroid function. The microbiota has been identified as a factor in health and numerous diseases. Microorganisms influence thyroid hormone levels by regulating iodine uptake, degradation, and enterohepatic cycling [19]. Several recent studies suggest that IM has a significant impact on human health and disease. Autoimmune thyroid disorders and metaplastic atrophic gastritis are particularly linked [20].

Mineral absorption

Minerals have a marked influence on the interactions between the host and IM, particularly iron, selenium, and zinc. These minerals are often deficient in patients with thyroid dysfunction. It is important to note that these elements are crucial for thyroid function. For example, iodine, iron, and copper are essential for the synthesis of thyroid hormones, while selenium and zinc are involved in the conversion of T4 to T3.

Iron

Iron deficiency is the most prevalent nutritional deficiency worldwide. Low serum ferritin levels could affect the thyroid gland and its function. IM promotes the absorption of trace elements that ensure normal metabolism. *In vitro* studies have shown that *Lactobacillus*

fermentum, part of the GM family, exhibits iron-reducing activity due to the excretion of p-hydroxyphenylacetic acid, which facilitates iron absorption. Furthermore, IM was observed to increase the bioavailability of dietary iron by converting ellagic acid into urolithin A, which remains active without the need to bind Fe_3. In states of iron deficiency, the expression of divalent metal transporter 1 increases, leading to increased iron absorption and release into the bloodstream, mediated by ferroportin, a transmembrane protein present in duodenal epithelial cells (enterocytes), among others. Likewise, in cases of iron overload, iron absorption decreases, and excess iron is incorporated into enterocytes, binding to ferritin, the main iron storage protein. Dietary iron deficiency leads to decreased populations of intestinal bacteria, such as *Roseburia, Bacteroides*, and *Eubacterium rectale*, and increased strains of *Lactobacillus* and *Enterobacteriaceae* [21].

Selenium

Is found mainly in the form of organic compounds, and includes the forms selenomethionine, methylselenocysteine, or gamma-glutamyl-methylselenocysteine. The absorption rate of organic selenium compounds has been shown to be approximately 85 - 90% of the supplied dose, compared to 10% of the mineral dose supplied in the form of inorganic selenates. Selenium absorption takes place in the duodenum and cecum. According to animal studies, dietary selenium increases the abundance of bacteria such as *Lactobacillus, Bacteroides, Prevotella*, and *Roseburia*, while decreasing that of *Firmicutes, Alistipes*, *Parabacteroides, Ruminococcus*, and *Helicobacter*. Once absorbed into the bloodstream, selenium binds to plasma albumin and globulin, after which it is transported to the liver, kidneys, testes, thyroid, pancreas, pituitary gland, and skeletal muscle. Selenium absorbed in the small intestine can be actively reabsorbed in the colon and metabolized by the GI tract, reducing its bioavailability [22].

Zinc

Is the second most abundant metal in the human body after iron. In 1980, Sandström and Cederblad revealed a trend according to which the higher the zinc content of a meal, the lower its fractional absorption in the intestine. After administration of 40 µmol of radiolabeled zinc, an absorption rate of 73% was achieved, compared to 46% with administration of 200 µmol. Intestinal zinc absorption in elderly individuals is lower than in young adults, regardless of dietary intake. Zinc absorption is also affected by the presence of phytates and other minerals (iron and calcium) in the intestinal lumen [23].

Gut microbiome metabolites, thyroid and other diseases

Among the numerous metabolites of the intestinal microbiome are SCFA which acquire energy, exert antioxidant effects, and can metabolize various bioactive substances. They are among the most desirable for epithelial cells and modifiers of the composition of the intestinal microbiome and its homeostasis. Both SCFA and other metabolites can affect the thyroid by influencing the intestinal absorption of trace elements and iodothyronine [24]. Knowledge of the different microorganisms can guide us toward thyroid pathology, and their determination is significant in the management of the condition. The intestinal microbiome and its metabolites play a significant role in the development of numerous diseases, such as metabolic diseases, type 2 diabetes mellitus, obesity, non-alcoholic fatty liver disease, and others [25]. The metabolites of the GM are of vital importance because they act on the host through their interactions. Effect that can occur directly or indirectly or through microbial metabolism, remembering that this interaction can be beneficial or detrimental to people. There are other metabolites such as trimethylamine N-oxide (TMAO), tryptophan, bile acids, polyamines, branched-chain amino acids and bacterial vitamins [26].

Biomarkers

There are different biomarkers that confirm the presence of hypothyroidism or hyperthyroidism, such as T3, T4, TSH, increased heat generation, resting metabolic rate, and metabolic rate. Here, we address markers based on increased microorganisms. We include *Bacteroides, Streptococcus, Faecalibacterium, Fusicatenibacter, Prevotella, Blautia, Eubacterium, Ruminococcus, Alloprevotella*, and *Roseburia*, which can serve as biomarkers for noninvasive monitoring of thyroid health. Bacteroides may not only serve as biomarkers

but also affect thyroid health and inflammation or be related to autoimmunity of the gland [27]. Linda Sessa., *et al.* [28], point out that intestinal microorganisms are essential for host metabolism, creating a mutualistic relationship. That bifidobacteria operate through molecular mimicry mechanisms due to the structural similarity of their protein sequences to thyroid peroxidase and thyroglobulin. That dietary variations and drug abuse can cause ID and imbalances in the local microbial community that could indirectly interfere with normal thyroid function.

Autoimmune thyroid diseases and gut microbiota

In recent decades, increasing attention has been paid to the field of immune microbiota. Several studies indicate that its composition is altered in patients with autoimmune thyroid disease. Autoimmune thyroid diseases and immune microbiota may be related. Studies have shown that alterations may influence development, although there is no direct evidence of a causal relationship. Lymphangiogenesis can have beneficial or detrimental effects on thyroid function. Immune responses are unique to thyroid antigens and are long-lasting, demonstrating different microbiota in patients with autoimmune processes [29]. Approximately 70% of the immune system resides in the intestinal mucosa. Microbiota plays a predominant role in autoimmune thyroid diseases. The exact etiology has not yet been determined. According to recent studies, it is assumed that microbiota may play a significant role in triggering autoimmune thyroid diseases [30].

Thyroid complications related to the gut microbiota

Altered IM increases the incidence of Graves' disease and Hashimoto's thyroiditis; it influences thyroid hormone levels by altering iodine synthesis, metabolism, regulation, uptake, degradation, and enterohepatic cycling. This effect directly or indirectly influences the progression of thyroid diseases and can be exacerbated by them. It has been observed that intestinal microorganism levels interfere with the presentation of autoantigens, inducing inflammation through metabolites. It is concluded that a deeper understanding of the subject will lead to the development of new strategies. Furthermore, IM alterations have been shown to orchestrate the development of thyroid autoimmunity, and a triggering event could be the loss of immune tolerance to gland-specific autoantigens [31].

Biotics, hypo- and hyperthyroidism

Currently, there are doubts regarding the effectiveness of biotics on thyroid function. Diverse results were determined in the studies carried out, for example, Shu Q., *et al*. [32], who carried out a meta-analysis with probiotics, prebiotics and synbiotics, on levels of free T3, T4; thyroid-stimulating hormone and thyroid-stimulating hormone receptor antibodies (TRAb) and concluded that biotic supplementation does not influence thyroid hormone levels, but may slightly reduce TRAb levels in patients with Graves' disease.

Hypothyroidism and hyperthyroidism and gut microbiota transplantation

It has been proposed that IM and its metabolites may play a significant role in modulating the immune system in diseases such as multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, type 1 diabetes mellitus, etc. [33]. Recently, scientific articles have been proposed on the potential role of microbiome transfer from healthy donors to patients with autoimmune diseases, with mixed results. Promising results have been demonstrated in patients with various autoimmune diseases after GM. IM is a promising cutting-edge intervention to restore thyroid health. IM has been studied in the context of hypothyroidism and hyperthyroidism. Recent studies have highlighted the relationship between thyroid pathophysiology and IM composition. Further studies assessing the impact of the procedure on hypothyroidism and hyperthyroidism are lacking [34].

Future

The future of the relationship between hypothyroidism and hyperthyroidism and the gut microbiota is underway, focusing on GM metabolites and the thyroid-gut axis, primarily due to their potential influence on thyroid function. FMT as a methodology has been

used, although it currently has detractors, since its systematic use should ensure clinical safety, efficacy, and true immunoregulatory actions [35]. The future of omics technologies must be analyzed, as well as digital medicine, which has the potential to impact all aspects of medicine, including the prediction, prevention, diagnosis, treatment, and subsequent management of diseases. Recent studies using artificial intelligence report reasonable performance for the classification of thyroid nodules based on ultrasound images, which can serve as a basis for other events. It is considered that digital medicine technologies will be more active [36].

Conclusion:

- The microbiome and its metabolites affect the proper functioning of the thyroid gland at various levels.
- Although not yet determined, intestinal dysbiosis may be the cause of thyroid disorders.
- Developed digital technologies will impact the management of thyroid disease.
- Artificial intelligence could overcome the limitations of conventional thyroid ultrasound.

Conflicts of Interest

The authors declare that do not have affiliation or participation in organizations with financial interests.

Ethical Approval

This report does not contain any study with human or animal subjects carried out by the authors.

Informed Consent

The authors obtained informed written consent from the patients, in order to develop this article.

Declaration on the Use of Artificial Intelligence

The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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