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Gut Microbiota Role in Thyroid Auto Immunity

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Editorial

The microbiome has been discovered as a critical role in human health and disease. Hashimoto's thyroiditis and Graves' disease are more common when the microbiota is disturbed. Microbes regulate iodine uptake, degradation, and enterohepatic cycling, all of which affect thyroid hormone levels. In addition, minerals, particularly selenium, iron, and zinc, have a significant impact on host-microbiota interactions. The microbiota may impact L-thyroxine uptake and propylthiouracil activity in people with thyroid problems. Although it is generally established that thyroid diseases are linked to microbiota composition, the relevance of certain genera and the possible utility of microbiota-targeted therapeutics are less evident. Thyroid autoimmune illnesses are the most common organ-specific autoimmune diseases, affecting 2–5% of the population [1].

Microbial compounds, especially short-chain fatty acids, can provide energy to enterocytes and, when combined with thyroid hormones, can improve enterocyte differentiation and reinforce intercellular tight connections. The generation of self-antigens by post-translational modification of proteins, lipopolysaccharide-induced Toll-like receptor 4 activation, induction of a type 1 T helper cell shift, reducing the integrity of intercellular junctions, and inducing transcriptomic, proteomic, and metabolic changes are all hypothesised mechanisms by which an altered microbiota composition in the gut promotes the development of AID. Hypothyroidism and hyperthyroidism are caused mostly by HT and GD, respectively [2].

Despite the fact that they are both AIDS, the immunological processes involved are not the same. Circulating antibodies against the TSH receptor are the major immunologic characteristics of GD. The presence of autoreactive T cells and antibodies against thyroperoxidase and thyroglobulin characterises HT, which leads to thyroid gland destruction. As a result, it's probable that microbiota plays a different role. The severity of the disease in both autoimmune thyroid disorders is unrelated to antibody levels. Furthermore, there is no relationship between anxiety and depression in GD and thyroid function or thyroid autoimmunity [3]. Thyroid hormones are unlikely to have direct effects on mood because mild hyperthyroidism is associated with a better mood than euthyroid people. In all mood disorders, Actinobacter and Enterobacteriacae were found in abundance, although Faecalibacterium spp was found in lower numbers when compared to the distribution of phyla. Microbial metabolites are known to have an effect on the central nervous system, and microbial products can cause a variety of symptoms [4]. Transfer of microbiota from conventional to specific pathogen-free rats increased their sensitivity to HT, suggesting that the microbiota may play a role in AITD. Lactobacillus spp. and Bifidobacterium spp. may cause antibodies to react with thyroperoxidase and thyroglobulin due to molecular mimicry. The microbiota's composition appears to play a crucial impact in mouse strain sensitivity to GD. In comparison to the less-susceptible strain, susceptible mice had higher levels of Paludibacter and Allobaculum, Limibacter, Anaerophaga, and Ureaplasma [5].

Anti-gliadin, anti-transglutaminase, and anti-yeast antibodies are produced by patients with GD or HT. In patients, Prevotellaceae and Pasteurellaceae were substantially higher than in controls, although Enterobacteriaceae, Veillonellaceae, and Rikenellaceae were significantly lower. In hyperthyroid individuals, there was a decrease in Bifidobacteria and Lactobacillaceae, as well as an increase in Enterococcus spp., as compared to healthy controls [6]. When compared to healthy guts, GD patients' intestines had higher levels of antibodies against Yersinia enterocolica and Helicobacter pylori, more yeast colonization, and less Bacteroides colonization. Hypothyroid individuals had a greater diversity of microbiota species than healthy controls. This could be explained by consequences related to hypothyroid patients' prolonged gastrointestinal transit times. The higher variety of bacteria in the colon has been attributed to low cell turnover, low redox potential, and long transit durations [7].

Although high diversity is thought to be beneficial to human health, it can also have negative consequences, such as increased protein catabolism, decreased polyphenol conversion and mucus secretion, and reduced epithelial turnover. Increased tryptophan metabolism, on the other hand, stimulates the synthesis of anti-inflammatory indole derivatives [8]. For HT patients, levels of Bacteroides richness and diversity were comparable to those of healthy participants, and variations in 27 genera were linked to clinical symptoms. The disease's progression and course are also influenced by the microbiome. In the intestines of mice with Graves' ophthalmopathy, Bacteroidetes levels were lower and Firmicutes levels were higher [9]. This conclusion is consistent with what has been observed in GD patients. Supplementing mice with Lactobacillus reuteri increased their thyroid function by raising free thyroxine, thyroid mass and physiological markers like slimness and skin structure. Lactic acid bacteria feeding raised thyroid hormone levels in broiler hens. These data suggest that, despite significant interspecies changes in taxonomic profiles, specific microbial species perform similar tasks in different species [10].

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Conflict of Interest

None

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