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THE ROLE OF THE MICROBIOME IN HASHIMOTO'S DISEASE - PATHOGENESIS AND THERAPEUTIC IMPLICATIONS: NARRATIVE REVIEW

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ABSTRACT

Objective: The aim of this narrative review was to summarize the current state of knowledge regarding the role of the gut microbiome in the pathogenesis of Hashimoto's disease and to discuss potential microbiome-targeted therapeutic strategies.
Methods: A narrative literature review was conducted using publications indexed in the PubMed, Google Scholar and Scopus databases. Original research articles, systematic reviews, and meta-analyses focusing on Hashimoto's disease and gut microbiota interactions were included. Most studies were selected from the past 10-15 years, with approximately 80% of the included literature published within the last five years (2021–2025), ensuring up-to-date evidence.
Key findings: Available evidence indicates that patients with Hashimoto's disease exhibit gut dysbiosis characterized by reduced microbial diversity, decreased abundance of short-chain fatty acid-producing bacteria, and an overrepresentation of potentially pathogenic microorganisms. These alterations are associated with impaired intestinal barrier integrity, increased systemic inflammation, and dysregulation of immune responses, particularly an imbalance between regulatory T cells and Th17 lymphocytes. Furthermore, microbiome disturbances may contribute to thyroid autoimmunity through mechanisms such as molecular mimicry and translocation of bacterial antigens. Emerging studies suggest that dietary modification, probiotic and synbiotic supplementation, and fecal microbiota transplantation may positively influence thyroid function and immune regulation.
Conclusions: Although levothyroxine replacement remains the standard treatment for Hashimoto's disease, modulation of the gut microbiome represents a promising adjunctive therapeutic approach. Further well-designed clinical trials are required to clarify causal relationships and evaluate the efficacy of microbiome-targeted interventions in autoimmune thyroid disease.

KEYWORDS

Hashimoto's Disease, Hashimoto's Thyroiditis, Microbiome, Microbiota, Autoimmune Thyroid Disease

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Introduction

Hashimoto's disease (HD) is an autoimmune disease that destroys thyroid follicular cells via cell- and antibody-mediated mechanisms. This disease is also known as chronic autoimmune thyroiditis, chronic lymphocytic thyroiditis and Hashimoto's thyroiditis (HT). HD is the most common cause of hypothyroidism in developed countries. The overall global prevalence of this disease is estimated at 7.5%. It is higher in low- and middle-income areas - around 11.4% (Hu et al., 2022). This condition was initially described by a Japanese physician, Haruto Hashimoto, in 1912 as "struma lymphomatosa" after he found enlarged thyroids having lymphocytic infiltration (Antonini et al., 2025). Along with Graves' disease, it belongs to the spectrum of autoimmune thyroid diseases.

Terms microbiome and microbiota are often used interchangeably, while the meaning behind them varies. Microbiome is a collection of all microorganisms (including bacteria, archaea, viruses, fungi) and their collective genetic material that residue within human body. Whereas microbiota is strictly restricted to the organisms found in specific environment (Hou et al., 2022). The key milestone in microbiome research was naming gut microbiota as "second genome" due to its large gene content exceeding that of the human genome and therefore bringing the attention to the importance of the influence it might have on health (Grice & Segre, 2012). Interventions aimed at modifying the gut microbiome (GM) are increasingly investigated as potential adjunctive interventions for supporting endocrine and immune homeostasis. New evidence suggests that microbial dysbiosis may contribute to autoimmune thyroid diseases, including HD and Graves' disease, through immunomodulatory and metabolic pathways (Sawicka-Gutaj et al., 2022). The gut-associated lymphoid tissue (GALT), which contains approximately 70% of the body's immune cells, plays a central role in immune regulation and host-microbiome interactions. GALT comprises various types of lymphoid tissues

that store immune cells, such as T and B lymphocytes, which which mediate immune responses against antigens (Fang & Ning, 2024).

Probiotics are defined as live microorganisms that, when administered in adequate amounts, confer a health benefit on the host. Prebiotics are non-living, nondigestible food ingredients that beneficially affect host health by selectively stimulating the growth and activity of bacteria in the colon, including digestion-resistant oligosaccharides and dietary fiber (C. H. Kim, 2023). They are found naturally in many foods, such as wheat, onions, bananas, honey, garlic, or leeks. Synbiotic, a combination of probiotic and prebiotic may exert synergistic effects on microbial composition and function (Rawat et al., 2024). Dietetary interventions are increasingly investigated as potential adjunctive strategies to support immune and endocrine homeostasis in autoimmune thyroid disease.

The gut-thyroid axis is an emerging area of research that highlights the interaction between the GM and the endocrine system. This narrative review summarizes recent evidence examining the role of the GM in HD. We outline how microbiome alterations may contribute to autoimmune thyroid dysfunction and discuss the potential of microbiome-based therapies, including diet, probiotics and fecal microbiota transplantation, as emerging strategies in the management of HD.

Methodology

A narrative review was conducted based on a comprehensive search of the available scientific literature. Major biomedical databases, including PubMed, Google Scholar, and Scopus, were systematically searched to identify publications relevant to HD, HT, and the potential role of the GM in thyroid autoimmunity. Search terms included combinations of the following keywords: “Hashimoto thyroiditis”, “Hashimoto disease”, “autoimmune thyroid disease”, “gut microbiome”, “intestinal microbiota”, “thyroid autoimmunity”, “immune modulation”, and “immune tolerance”. The search strategy was designed to capture studies exploring the relationship between microbial composition, immune regulation, and the development or progression of autoimmune thyroid disorders. Preference was given to peer-reviewed original research articles, systematic reviews, and meta-analyses published within the past 10-15 years to ensure the inclusion of the most recent scientific evidence. Approximately 80% of the included sources were published within the last five years (2021-2025) to ensure the incorporation of the most up-to-date scientific evidence.

Hashimoto’s Disease

HD is the most common autoimmune thyroid disease and the leading cause of hypothyroidism in populations with adequate iodine intake. The disease occurs significantly more frequently in women, with a female-to-male ratio reaching up to 5-10:1 (Caturegli et al., 2013). The predominance of HD in women is associated with the influence of sex hormones on the regulation of the immune response as well as the presence of genetic predispositions. In addition, the disease occurs more frequently in patients with Turner syndrome and Down syndrome (Aversa et al., 2015). Currently, HD is considered a polygenic disease. Variants in genes such as CTLA4, PTPN22, CD40, FoxP3, CD25, as well as the HLA region, which are involved in the control of immune tolerance, increase the risk of disease development (Zaletel & Gaberscek, 2011). However, it should be noted that no single genetic variant is sufficient to cause the disease in the absence of environmental factors. As in many allergic and autoimmune diseases, the prevalence of HD is higher among individuals living in more hygienic environments with reduced exposure to microbial factors (Wiersinga, 2016). The combined influence of genetic and environmental factors leads to a loss of immune tolerance to thyroid antigens and infiltration of the thyroid gland by activated autoreactive T lymphocytes and B lymphocytes differentiating into plasma cells that produce antithyroid antibodies: anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) (Rydzewska et al., 2018). $CD8^+$ T lymphocytes play a key role in immune dysfunction; these cytotoxic cells infiltrate the thyroid gland, leading to inflammation and, consequently, destruction of thyroid follicular cells (Ehlers et al., 2012). $CD4^+$ T lymphocytes activate other immune cells, primarily macrophages and B lymphocytes, resulting in the production of autoreactive antibodies. Various subsets of $CD4^+$ T lymphocytes, including Th1, Th2, Th17, and Treg cells (regulatory T cells) perform distinct functions, and disturbances in their balance contribute to disease pathogenesis (Rydzewska et al., 2018) Importantly, Treg lymphocytes play a crucial role in promoting immune tolerance and preventing excessive immune responses; in HD the number of Treg cells is reduced (Pyzik et al., 2015). Through chronic lymphocytic infiltration, production of proinflammatory cytokines, and cytotoxic mechanisms, progressive fibrosis occurs, leading to gradual destruction of thyroid follicular cells and a consequent decline in thyroid hormone synthesis (Avramidou et al., 2023). HD may remain clinically asymptomatic or subclinical for a long period; however,

as thyroid hormone production decreases, symptoms such as chronic fatigue, dry skin, weight gain, and mood disturbances begin to appear. The diagnosis of HD includes assessment of thyroid hormone levels thyroid-stimulating Hormone (TSH), free triiodothyronine (fT3) and free thyroxine (fT4), evaluation of antithyroid antibodies, and ultrasonographic examination, in which a heterogeneous, hypoechoic thyroid parenchyma is characteristic. This ultrasonographic appearance reflects the underlying chronic inflammatory process.

Human Gut Microbiome

Out of all microbiomes in human body, (e.g. in uterus, skin, oral mucosa, lung) GM is the most significant (Hou et al., 2022). On average, the human body contains approximately 38 trillion bacteria, the vast majority, representing up to 3000 different species are found in gastro-intestinal system. Different studies estimate the total weight of GM to be between 0,2 to 2kg, with body mass being a key influencing factor (Cheng et al., 2022; Sender et al., 2016). Rather than static state, microbiome's composition is a constantly changing environment due to various factors such as age, diet, stress, physical activity, medications or comorbid condition (Rehner et al., 2025). Currently it is well known that GM plays a major role in homeostasis of physical as well as mental well-being (Barb & Wallen, 2025). Dominant bacteria in healthy individuals are Bacteroidetes and Firmicutes phyla, accounting for 90% of the total community, along with less abundant like Actinobacteria and Proteobacteria. It is important to take into consideration that these findings might vary greatly depending on geographical and technical differences in conducting research (Sun et al., 2024). Additionally, the composition of the gut microbiota varies across different anatomical regions of the gastrointestinal tract (Milani et al., 2017). Although components of the GM beyond bacteria - such as archaea, viruses, and fungi - are relatively few, they have a significant impact on the microbial ecosystem. The gut virome composed mainly by bacteriophages, remains a largely unexplored component of the microbiome, although its direct impact in different therapies in gastrointestinal diseases such as fecal microbial transplantation (Feng et al., 2025). Mycobiome - composed mostly of Candida, Saccharomyces and Cladosporium, help maintain intestinal barrier function and modulate the immune system while forming different relationship with bacteria. For example, fungi can prevent bacteria from growing by producing alcohol, whereas some bacterial pathogens take advantage of fungal iron carriers to boost their own growth (H. Y. Liu et al., 2025). It is believed that development of human microbiome starts at birth, with some new research suggesting that vertical transmission may occur already in uterus during pregnancy (Walker et al., 2017). Birth is a crucial element of starting of development of child's own microbiome. Human microbiota can be passed like genetics from mother to child and the method of delivery seems to have an impact. Neonates delivered vaginally exhibited a GM with higher diversity than those delivered by cesarean delivery. Intestinal colonization with Staphylococcus aureus, connected with higher level of gut inflammation, was higher in the cesarean delivery babies, with maternal skin flora being the proposed source of colonization (Pahirah et al., 2024). Infant's GM depends greatly on its mother's gut, skin, vaginal microbiome, gestational age, environmental exposure and type of feeding (Mady et al., 2023). Systematic review proved that formula-fed infants have reduced microbial diversity and lower levels of beneficial bacteria such as Lactobacillus and Bifidobacterium compared to breastfed group. This supports the thesis that diet is one of the strongest, if not the strongest - modifiable factor shaping GM (Asnicar et al., 2025). One of many studies on diet and GM showed that diet rich in animal proteins can lead to increased production of trimethylamine-N-Oxide from intestinal microbiota and increasing the risk of cardiovascular disease, while plant-based proteins could positively influence gut microbial homeostasis. Western-type diet with high intake of saturated fats and omega-6 PUFA can lead to dysbiosis, gut barrier alterations, and metabolic disorders. These revelations open the possibility of diet as a therapeutic modulator of all the diseases connected with microbiome disbalance (Rinninella et al., 2023). Microbial communities become more stable and begin to resemble the 'adult-like' GM when a child is between 1 and 2 years old (Sommer & Bäckhed, 2013). The importance of the microbiome lies in its involvement in multiple physiological pathways and functions. From early life onwards, it contributes to the development of the intestinal epithelium and immune system maturation., it is taking part in development of intestinal epithelium and immune system. It influences the characteristics of the mucus layer, supports the formation of lymphoid structures, regulates the activation and differentiation of various lymphocyte populations, and helps maintain a balance in the production of immunoglobulin A and antimicrobial peptides. Another important mechanism to mention is metabolites produced by bacteria, that can connect with receptors on immune cells of intestine and modulate the immune response. The most known being short-chain fatty acids (SCFA), produced from fermentation of fiber delivered to the intestine with food. SCFA are associated with increased immunoglobulin production. They modify gene expression, encourage neutrophil migration and can

be considered indicators of a healthy gut ecosystem (S. Kim et al., 2025,Fusco et al., 2023). Any imbalance in GM might be linked to numerous health conditions, not only within gastrointestinal system (e.g. inflammatory bowel disease, celiac disease) but also considering metabolic diseases (e.g. diabetes and obesity), psychiatry (e.g. anxiety, depression), neurodegenerative (e.g. Alzheimer's, Parkinson's) and oncology (e.g. colorectal, breast cancer) (Olvera-Rosales et al., 2021).

Importance of human microbiome in pathogenesis of Hashimoto's disease

Significant gut dysbiosis has been observed in HD. A commonly reported finding is a reduction in microbial diversity accompanied by alterations in bacterial composition. A decreased abundance of commensal bacteria such as Bifidobacterium, Lactobacillus, Megamonas, and other bacteria belonging to the order Clostridiales has been described, along with an increase in populations of potentially pathogenic strains, including Klebsiella, Bacteroides, members of the Proteobacteria group, and Desulfobacterota, as confirmed by published analyses (Kovenskiy et al., 2025). This study additionally demonstrated that Romboutsia and Haemophilus, whose abundance is associated with FT3 levels alterations, may contribute to the progression of HD, whereas Faecalibacterium and members of the Lachnospiraceae family, regulated by FT4, appear to exert a protective effect on the host (J. Liu et al., 2022). This dysbiosis consequently leads to metabolic disturbances within the gut. One of the most significant effects appears to be a reduction in the production of SCFAs. As previously mentioned, SCFAs play a crucial role in strengthening intestinal mucosal defense mechanisms and maintaining gut barrier integrity. Decreased SCFA production results in disruption of immune homeostasis, leading to impairment of the intestinal barrier and alterations in T lymphocyte subset distribution. (Jiang et al., 2025)(Gong et al., 2024) In the presence of SCFA deficiency, there is also an increased production of proinflammatory cytokines such as IFN-γ and TNF-α, along with critical disturbances in the balance between Treg and Th17 cells. (Zhu et al., 2024) Furthermore, increased intestinal permeability (leaky gut syndrome) allows for the translocation of lipopolysaccharide (LPS) and other bacterial antigens into the bloodstream, thereby amplifying systemic inflammation. This process promotes the loss of thyroid immune tolerance, manifested by the production of anti-TPO and anti-Tg autoantibodies characteristic of HD (Jiang et al., 2025). Molecular mimicry has been proposed as one of the mechanisms, whereby protein fragments of certain Lactobacillus and Bifidobacterium strains share structural similarity with thyroid antigens such as thyroid peroxidase and thyroglobulin. Additionally, some Lactobacillus species may increase the proportion of Th17 lymphocytes at the expense of a reduced number of Treg cells in the gut, thereby favoring the development of autoimmune diseases (Sessa et al., 2025). Although gut bacterial dysbiosis in HD is well documented, the role of the gut virome, including bacteriophages, remains largely unexplored and is currently limited to indirect evidence suggesting a potential modulatory effect through microbiome-immune system interactions (Fujimoto et al., 2022). Emerging evidence supports a bidirectional relationship within the gut-thyroid axis, where gut microbiota composition may influence thyroid autoimmunity, while thyroid hormone levels in turn modulate microbial diversity and metabolic activity. This dynamic interaction suggests that dysbiosis in HD may represent both a contributing factor to disease development and a consequence of altered thyroid function rather than a unidirectional process (Parveen et al., 2025).

Treatment

Currently, the treatment for HD focuses on managing hypothyroidism and is based on administering synthetic thyroid hormone (levothyroxine). The drug is administered orally, in the morning on an empty stomach for optimum absorption. No prevention or cure of the disease has been proven yet. Diet remains one of the non-invasive approaches that can provide benefits in HD. Foods rich in nutrients such as vitamin D, antioxidants, unsaturated fatty acids, magnesium, and zinc help reduce thyroid inflammation. Iodine and selenium are essential in thyroid hormone synthesis, therefore foods such as dairy, eggs, seafood might be recommended to ensure adequate intake of these elements (Danailova et al., 2022). Correction of selenium, iron, or vitamin D deficiencies appears to contribute to lower risk and more efficient treatment of HD (Antonini et al., 2025). Recent evidence suggests that selenium supplementation may reduce TSH levels in patients not receiving thyroid hormone replacement therapy. While anti-TPO levels were lower in patients supplementing selenium, with or without levothyroxine therapy (Huwiler et al., 2024). Eliminating gluten and lactose is not recommended in the diets of patients without gastrointestinal disorders, but only in groups with confirmed intolerance or celiac disease. Anti-inflammatory diets are characterized by high intake of plant food, omega-3 fatty acids, proteins, fiber and antioxidants, with limited saturated fats, sugars, and refined carbohydrates. One example is Mediterranean diet, which has been shown to lower oxidative stress parameters and as a result

might reduce inflammatory processes in thyroid (Osowiecka & Myszkowska-Ryciak, 2023) Hollywood et al. reported that paleolithic diet, which is based on lean meats, fish, fruits, vegetables, may reduce thyroid autoantibody levels and influence thyroid hormone concentrations in patients with autoimmune thyroid diseases (Hollywood et al., 2023). Probiotics are commonly used in treating antibiotic-associated diarrhea, gastrointestinal disorders, respiratory infections, bacterial vaginosis or atopic eczema. They maintain mucosal integrity by strengthening the epithelial junctions and enhance intestinal barrier function (preventing leaky gut syndrome). Recent meta-analysis results indicates that probiotics and synbiotics oral supplementation leads to a significant reduction in TSH and increased T3 and T4 levels. The mechanism behind it is strengthening the gut barrier, lowering systemic inflammation by downregulating IL-6 or TNF-α, modifying the hypothalamic-pituitary-thyroid axis and enhancing the absorption of essential micronutrients for thyroid hormone production, including selenium, zinc, and iodine (Karimi et al., 2025). A recent study proposed a comprehensive evaluation of nutritional intervention using the probiotic Lactiplantibacillus plantarum 299v (Lp299v) in patients with HD. According to this study, personalized nutrition education may improve nutritional status and quality of life, with the Lp299v strain potentially enhancing these benefits, particularly regarding quality of life (Osowiecka et al., 2025). Prebiotics have been less extensively studied as independent interventions in HD, however, they exert the effects of probiotics by selectively promoting the growth of beneficial gut bacteria, such as Faecalibacterium and Bifidobacterium, and by increasing the production of SCFAs, which possess immunomodulatory and anti-inflammatory properties. In recent years, fecal microbiota transplantation (FMT) has emerged as a potential therapeutic strategy for several diseases associated with microbiome dysbiosis. By optimizing the donor selection strategy, FMT exhibited the highest effectiveness (more than 90%) in treating intestinal infections, such as recurrent Clostridioides difficile infection (Tian et al., 2024).Experimental studies provide compelling evidence for the functional consequences of dysbiosis in HD. In one experimental model, fecal microbiota transplantation from patients with HD into healthy mice resulted in exacerbated thyroid inflammation, reflected by elevated titers of thyroid autoantibodies (notably anti-Tg) and disrupted T-cell population balance. Conversely, transplantation of microbiota from healthy donors into animal models attenuated autoimmune manifestations (Zhang et al., 2024). A randomized, double-blind, placebo-controlled trial (IMITHOT) is undertaken in the Netherlands, to test whether FMT can pause disease progression and improve thyroid function in patients with HD. Participants receive allogenic or autologous FMT with follow-up at 6, 12, and 24 months to assess thyroid hormone release. Positive results from this trial may open possibilities for the future clinical applications of microbial-targeted therapy in individuals with HD (Fenneman et al., 2023). At present, this method is still an experimental approach, but it underscores the importance of the GM in thyroiditis pathogenesis.

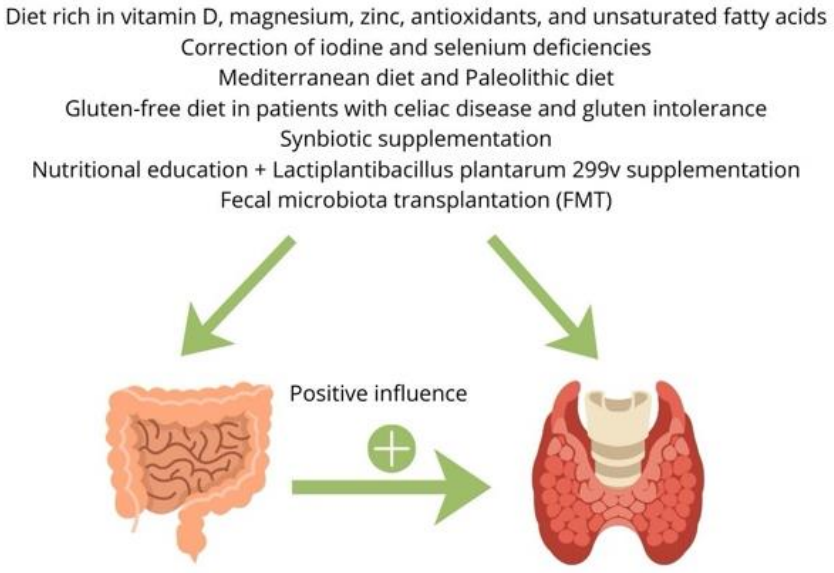

Fig. 1. *Potential microbiome-related therapeutic factors in Hashimoto's disease*

Conclusions

HD is a condition with a complex pathogenesis in which, alongside genetic and environmental factors, disturbances in immune system regulation play a significant role. Patients with HD exhibit characteristic alterations in gut microbiota composition, including reduced microbial diversity and a shift toward bacteria with pro-inflammatory potential. Gut dysbiosis may contribute to the development and progression of thyroid autoimmunity through impairment of intestinal barrier function, reduced production of SCFAs, disruption of T-lymphocyte subset balance, and amplification of inflammatory processes. Findings from experimental and clinical studies indicate that modulation of the gut microbiome may influence the course of HD, as well as thyroid function parameters. Dietary interventions, supplementation with probiotics and synbiotics, and correction of micronutrient deficiencies when present, may serve as valuable adjuncts to standard HD treatment. FMT remains an experimental approach. However, available data highlight its therapeutic potential and underscore the importance of the GM in the pathogenesis of the disease. Further well-designed clinical trials are required to clearly determine the efficacy and safety of microbiome-targeted therapies in patients with autoimmune thyroid diseases.

List of abbreviations

HD Hashimoto's disease
HT Hashimoto's thyroiditis
GM gut microbiome
Treg regulatory T cells
GALT gut-associated lymphoid tissue
anti-TPO anti-thyroid peroxidase antibodies
anti-Tg anti-thyroglobulin antibodies
TSH thyroid-stimulating hormone
fT3 triiodothyronine
fT4 thyroxine
SCFA short-chain fatty acids
FMT fecal microbiota transplantation.

REFERENCES

1. Antonini, S., Birtolo, M. F., Lania, A., & Longhi, E. V. (2025). Hashimoto thyroiditis. *Managing Psychosexual Consequences in Chronic Diseases*, 95–102. https://doi.org/10.1007/978-3-031-31307-3_9
2. Asnicar, F., Manghi, P., Fackelmann, G., Baldanzi, G., Bakker, E., Ricci, L., Piccinno, G., Piperni, E., Mladenovic, K., Amati, F., Arrè, A., Ganesh, S., Giordano, F., Davies, R., Wolf, J., Bermingham, K. M., Berry, S. E., Spector, T. D., & Segata, N. (2025). Gut micro-organisms associated with health, nutrition and dietary interventions. *Nature*, 1–9. https://doi.org/10.1038/s41586-025-09854-7
3. Aversa, T., Messina, M. F., Mazzanti, L., Salerno, M., Mussa, A., Faienza, M. F., Scarano, E., De Luca, F., & Wasniewska, M. (2015). The association with Turner syndrome significantly affects the course of Hashimoto's thyroiditis in children, irrespective of karyotype. *Endocrine, 50*(3), 777–782. https://doi.org/10.1007/S12020-014-0513-6
4. Avramidou, E., Gkantaras, A., Dermitzakis, I., Sapalidis, K., Manthou, M. E., & Theotokis, P. (2023). Histological alterations in Hashimoto's disease: A case-series ultrastructural study. *Medicines, 10*(9), 51. https://doi.org/10.3390/MEDICINES10090051
5. Barb, J. J., & Wallen, G. R. (2025). Emerging research on the relationship between diet, gut microbes, and human health. *Nutrients, 17*(16), 2627. https://doi.org/10.3390/NU17162627
6. Caturegli, P., De Remigis, A., Chuang, K., Dembele, M., Iwama, A., & Iwama, S. (2013). Hashimoto's thyroiditis: Celebrating the centennial through the lens of the Johns Hopkins hospital surgical pathology records. *Thyroid, 23*(2), 142–150. https://doi.org/10.1089/THY.2012.0554
7. Cheng, Z., Zhang, L., Yang, L., & Chu, H. (2022). The critical role of gut microbiota in obesity. *Frontiers in Endocrinology, 13*, 1025706. https://doi.org/10.3389/FENDO.2022.1025706
8. Danailova, Y., Velikova, T., Nikolaev, G., Mitova, Z., Shinkov, A., Gagov, H., & Konakchieva, R. (2022). Nutritional management of thyroiditis of Hashimoto. *International Journal of Molecular Sciences, 23*(9), 5144. https://doi.org/10.3390/IJMS23095144
9. Ehlers, M., Thiel, A., Bernecker, C., Porwol, D., Papewalis, C., Willenberg, H. S., Schinner, S., Hautzel, H., Scherbaum, W. A., & Schott, M. (2012). Evidence of a combined cytotoxic thyroglobulin and thyroperoxidase epitope-specific cellular immunity in Hashimoto's thyroiditis. *The Journal of Clinical Endocrinology and Metabolism, 97*(4), 1347–1354. https://doi.org/10.1210/JC.2011-2178

10. Fang, L., & Ning, J. (2024). Recent advances in gut microbiota and thyroid disease: Pathogenesis and therapeutics in autoimmune, neoplastic, and nodular conditions. *Frontiers in Cellular and Infection Microbiology, 14*. https://doi.org/10.3389/FCIMB.2024.1465928
11. Feng, Z., Burgermeister, E., Philips, A., Zuo, T., & Wen, W. (2025). The gut virome in association with the bacteriome in gastrointestinal diseases and beyond: Roles, mechanisms, and clinical applications. *Precision Clinical Medicine, 8*(2), 10. https://doi.org/10.1093/PCMEDI/PBAF010
12. Fenneman, A. C., Rampanelli, E., Van Der Spek, A. H., Fliers, E., & Nieuwdorp, M. (2023). Protocol for a double-blinded randomised controlled trial to assess the effect of faecal microbiota transplantations on thyroid reserve in patients with subclinical autoimmune hypothyroidism in the Netherlands: The IMITHOT trial. *BMJ Open, 13*(9). https://doi.org/10.1136/BMJOPEN-2023-073971
13. Fujimoto, K., Miyaoka, D., & Uematsu, S. (2022). Characterization of the human gut virome in metabolic and autoimmune diseases. *Inflammation and Regeneration, 42*(1), 32. https://doi.org/10.1186/S41232-022-00218-6/TABLES/2
14. Fusco, W., Lorenzo, M. B., Cintoni, M., Porcari, S., Rinninella, E., Kaitsas, F., Lener, E., Mele, M. C., Gasbarrini, A., Collado, M. C., Cammarota, G., & Ianiro, G. (2023). Short-chain fatty-acid-producing bacteria: Key components of the human gut microbiota. *Nutrients, 15*(9), 2211. https://doi.org/10.3390/NU15092211
15. Gong, B., Meng, F., Wang, X., Han, Y., Yang, W., Wang, C., & Shan, Z. (2024). Effects of iodine intake on gut microbiota and gut metabolites in Hashimoto thyroiditis-diseased humans and mice. *Communications Biology, 7*(1), 136. https://doi.org/10.1038/s42003-024-05813-6
16. Grice, E. A., & Segre, J. A. (2012). The human microbiome: Our second genome. *Annual Review of Genomics and Human Genetics, 13*, 151–170. https://doi.org/10.1146/ANNUREV-GENOM-090711-163814
17. Hollywood, J. B., Hutchinson, D., Feehery-Alpuerto, N., Whitfield, M., Davis, K., & Johnson, L. M. (2023). The effects of the paleo diet on autoimmune thyroid disease: A mixed methods review. *Journal of the American Nutrition Association, 42*(8), 727–736. https://doi.org/10.1080/27697061.2022.2159570
18. Hou, K., Wu, Z. X., Chen, X. Y., Wang, J. Q., Zhang, D., Xiao, C., Zhu, D., Koya, J. B., Wei, L., Li, J., & Chen, Z. S. (2022). Microbiota in health and diseases. *Signal Transduction and Targeted Therapy, 7*(1), 135. https://doi.org/10.1038/s41392-022-00974-4
19. Hu, X., Chen, Y., Shen, Y., Tian, R., Sheng, Y., & Que, H. (2022). Global prevalence and epidemiological trends of Hashimoto's thyroiditis in adults: A systematic review and meta-analysis. *Frontiers in Public Health, 10*, 1020709. https://doi.org/10.3389/FPUBH.2022.1020709/TEXT
20. Huwiler, V. V., Maissen-Abgottspon, S., Stanga, Z., Mühlebach, S., Trepp, R., Bally, L., & Bano, A. (2024). Selenium supplementation in patients with Hashimoto thyroiditis: A systematic review and meta-analysis of randomized clinical trials. *Thyroid, 34*(3), 295. https://doi.org/10.1089/THY.2023.0556
21. Jiang, T., Yang, X., Wu, B., Tao, R., Chen, R., Jin, L., Sun, D., & Weng, H. (2025). Gut microbiota in hypothyroidism: Pathogenic mechanisms and opportunities for precision microbiome interventions. *Frontiers in Microbiology, 16*, 1661211. https://doi.org/10.3389/FMICB.2025.1661211/XML
22. Karimi, M., Rabiei, R., Kazemi, K., Motlagh, R. B., & Asbaghi, O. (2025). Effects of probiotics and synbiotics oral supplementation on thyroid function in adults: A GRADE-assessed systematic review and meta-analysis. *Thyroid Research, 18*(1), 39. https://doi.org/10.1186/S13044-025-00257-4/TABLES/3
23. Kim, C. H. (2023). Complex regulatory effects of gut microbial short-chain fatty acids on immune tolerance and autoimmunity. *Cellular and Molecular Immunology, 20*(4), 341. https://doi.org/10.1038/S41423-023-00987-1
24. Kim, S., Ndwandwe, C., Devotta, H., Kareem, L., Yao, L., & O'Mahony, L. (2025). Role of the microbiome in regulation of the immune system. *Allergology International, 74*(2), 187–196. https://doi.org/10.1016/J.ALIT.2024.12.006
25. Kovenskiy, A., Katkenov, N., Ramazanova, A., Vinogradova, E., Jarmukhanov, Z., Mukhatayev, Z., & Kushugulova, A. (2025). *Bacteroides fragilis* and *Microbacterium* as microbial signatures in Hashimoto's thyroiditis. *International Journal of Molecular Sciences, 26*(17), 8724. https://doi.org/10.3390/IJMS26178724/S1
26. Liu, H. Y., Li, S., Ogamune, K. J., Ahmed, A. A., Kim, I. H., Zhang, Y., & Cai, D. (2025). Fungi in the gut microbiota: Interactions, homeostasis, and host physiology. *Microorganisms, 13*(1), 70. https://doi.org/10.3390/MICROORGANISMS13010070
27. Liu, J., Qin, X., Lin, B., Cui, J., Liao, J., Zhang, F., & Lin, Q. (2022). Analysis of gut microbiota diversity in Hashimoto's thyroiditis patients. *BMC Microbiology, 22*(1). https://doi.org/10.1186/S12866-022-02739-Z
28. Mady, E. A., Doghish, A. S., El-Dakroury, W. A., Elkhawaga, S. Y., Ismail, A., El-Mahdy, H. A., Elsakka, E. G. E., & El-Husseiny, H. M. (2023). Impact of the mother's gut microbiota on infant microbiome and brain development. *Neuroscience & Biobehavioral Reviews, 150*, 105195. https://doi.org/10.1016/J.NEUBIOREV.2023.105195
29. Milani, C., Duranti, S., Bottacini, F., Casey, E., Turroni, F., Mahony, J., Belzer, C., Palacio, S. D., Montes, S. A., Mancabelli, L., Lugli, G. A., Rodriguez, J. M., Bode, L., de Vos, W., Gueimonde, M., Margolles, A., van Sinderen, D., & Ventura, M. (2017). The first microbial colonizers of the human gut: Composition, activities, and health implications of the infant gut microbiota. *Microbiology and Molecular Biology Reviews, 81*(4), e00036-17. https://doi.org/10.1128/MMBR.00036-17

30. Olvera-Rosales, L. B., Cruz-Guerrero, A. E., Ramírez-Moreno, E., Quintero-Lira, A., Contreras-López, E., Jaimez-Ordaz, J., Castañeda-Ovando, A., Añorve-Morga, J., Calderón-Ramos, Z. G., Arias-Rico, J., & González-Olivares, L. G. (2021). Impact of the gut microbiota balance on the health–disease relationship: The importance of consuming probiotics and prebiotics. *Foods, 10*(6), 1261. https://doi.org/10.3390/FOODS10061261
31. Osowiecka, K., & Myszkowska-Ryciak, J. (2023). The influence of nutritional intervention in the treatment of Hashimoto's thyroiditis—A systematic review. *Nutrients, 15*(4), 1041. https://doi.org/10.3390/NU15041041
32. Osowiecka, K., Skrypnik, D., & Myszkowska-Ryciak, J. (2025). Probiotic supplementation enhances the effects of a nutritional intervention on quality of life in women with Hashimoto's thyroiditis—A double-blind randomised study. *Nutrients, 17*(21), 3387. https://doi.org/10.3390/NU17213387/S1
33. Pahirah, N., Narkwichean, A., Taweechotipatr, M., Wannaiampikul, S., Duang-Udom, C., & Laosooksathit, W. (2024). Comparison of gut microbiomes between neonates born by cesarean section and vaginal delivery: Prospective observational study. *BioMed Research International, 2024*(1), 8302361. https://doi.org/10.1155/BMRI/8302361
34. Parveen, N., Chittawar, S., & Khandelwal, D. (2025). Gut-thyroid axis and emerging role of probiotics in thyroid disorders. *Thyroid Research and Practice, 21*(1), 4–12. https://doi.org/10.4103/TRP.TRP_12_24
35. Pyzik, A., Grywalska, E., Matyjaszek-Matuszek, B., & Roliński, J. (2015). Immune disorders in Hashimoto's thyroiditis: What do we know so far? *Journal of Immunology Research, 2015*. https://doi.org/10.1155/2015/979167
36. Rawat, H. K., Nath, S., Sharma, I., & Kango, N. (2024). Recent developments in the production of prebiotic fructooligosaccharides using fungal fructosyltransferases. *Mycology, 15*(4), 564–584. https://doi.org/10.1080/21501203.2024.2323713
37. Rehner, J., Molano, L. A. G., Christodoulou, C., Holländer, S., Förster, M. O., Keller, V., Jäger, J., Volz-Willems, S., Becker, S. L., Glanemann, M., & Jelden, M. (2025). Examining spatial microbiome variations across gastrointestinal tract regions in obesity. *Scientific Reports, 15*(1), 25423. https://doi.org/10.1038/S41598-025-10931-0
38. Rinninella, E., Tohumcu, E., Raoul, P., Fiorani, M., Cintoni, M., Mele, M. C., Cammarota, G., Gasbarrini, A., & Ianiro, G. (2023). The role of diet in shaping human gut microbiota. *Best Practice & Research Clinical Gastroenterology, 62–63*, 101828. https://doi.org/10.1016/J.BPG.2023.101828
39. Rydzewska, M., Jaromin, M., Pasierowska, I. E., Stozek, K., & Bossowski, A. (2018). Role of the T and B lymphocytes in pathogenesis of autoimmune thyroid diseases. *Thyroid Research, 11*(1). https://doi.org/10.1186/S13044-018-0046-9
40. Sawicka-Gutaj, N., Gruszczyński, D., Zawalna, N., Nijakowski, K., Muller, I., Karpiński, T., Salvi, M., & Ruchała, M. (2022). Microbiota alterations in patients with autoimmune thyroid diseases: A systematic review. *International Journal of Molecular Sciences, 23*(21). https://doi.org/10.3390/IJMS232113450
41. Sender, R., Fuchs, S., & Milo, R. (2016). Revised estimates for the number of human and bacteria cells in the body. *PLOS Biology, 14*(8), e1002533. https://doi.org/10.1371/JOURNAL.PBIO.1002533
42. Sessa, L., Malavolta, E., Sodero, G., Cipolla, C., & Rigante, D. (2025). The conspiring role of gut microbiota as primer of autoimmune thyroid diseases: A scoping focus. *Autoimmunity Reviews, 24*(5). https://doi.org/10.1016/J.AUTREV.2025.103780
43. Sommer, F., & Bäckhed, F. (2013). The gut microbiota—Masters of host development and physiology. *Nature Reviews Microbiology, 11*(4), 227–238. https://doi.org/10.1038/NRMICRO2974
44. Sun, W., Zhang, Y., Guo, R., Sha, S., Chen, C., Ullah, H., Zhang, Y., Ma, J., You, W., Meng, J., Lv, Q., Cheng, L., Fan, S., Li, R., Mu, X., Li, S., & Yan, Q. (2024). A population-scale analysis of 36 gut microbiome studies reveals universal species signatures for common diseases. *npj Biofilms and Microbiomes, 10*(1), 96. https://doi.org/10.1038/s41522-024-00567-9
45. Tian, H., Wang, X., Fang, Z., Li, L., Wu, C., Bi, D., Li, N., Chen, Q., & Qin, H. (2024). Fecal microbiota transplantation in clinical practice: Present controversies and future prospects. *HLife, 2*(6), 269–283. https://doi.org/10.1016/J.HLIFE.2024.01.006
46. Walker, R. W., Clemente, J. C., Peter, I., & Loos, R. J. F. (2017). The prenatal gut microbiome: Are we colonized with bacteria in utero? *Pediatric Obesity, 12*(Suppl. 1), 3. https://doi.org/10.1111/IJPO.12217
47. Wiersinga, W. M. (2016). Clinical relevance of environmental factors in the pathogenesis of autoimmune thyroid disease. *Endocrinology and Metabolism, 31*(2), 213–222. https://doi.org/10.3803/ENM.2016.31.2.213
48. Zaletel, K., & Gaberscek, S. (2011). Hashimoto's thyroiditis: From genes to the disease. *Current Genomics, 12*(8), 576–588. https://doi.org/10.2174/138920211798120763
49. Zhang, S., Zhao, X., Wang, X., Jin, H., Chen, L., Ma, Y., Chi, Y., Zhang, J., Zhang, J., & Gao, Y. (2024). Gut microecology may be involved in the pathogenesis of Hashimoto thyroiditis by reducing production of hydrogen sulfide. *The Journal of Clinical Endocrinology and Metabolism, 109*(3), 792–801. https://doi.org/10.1210/CLINEM/DGAD588
50. Zhu, X., Zhang, C., Feng, S., He, R., & Zhang, S. (2024). Intestinal microbiota regulates the gut-thyroid axis: The new dawn of improving Hashimoto thyroiditis. *Clinical and Experimental Medicine, 24*(1). https://doi.org/10.1007/S10238-024-01304-4