

# [The human microbiota in endocrinology: implications for pathophysiology, treatmen...](https://assignbuster.com/the-human-microbiota-in-endocrinology-implications-for-pathophysiology-treatment-and-prognosis-in-thyroid-diseases/)

[](https://assignbuster.com/)[Health & Medicine](https://assignbuster.com/essay-subjects/health-n-medicine/)

## Gut Microbioma

A lot of microbes, like bacteria, fungi, archaea, viruses, and protozoa, colonize our body. Many of these reside within the gastrointestinal tract and moreover on the ocular surface ( [1](#B1) ); they are predominantly bacterial, and together these microbes collectively form the gut microbiota. Gut microbiota contains trillions of microorganisms, about 66% of the human microbiota, including at least one thousand different species of known bacteria with more than 3 million genes (much more times than human genes). Thirty-three percent of these is common to all people, while 66% are explicit to every single one of us. This microbial flora has been coevolute with human being in a symbiotic relationship for millennia and, thusly, it finds itself involved in many essential activities for our organism such as digestion and nutritional absorption ( [2](#B2) – [4](#B4) ), development of the host’s immunity system ( [3](#B3) ), and pathogenesis ( [5](#B5) , [6](#B6) ). The gut microbiota was found to play an important role in maintaining the nutritional, metabolic, and immunological balance in the host ( [7](#B7) ). In addition to its intended role in maintaining gastrointestinal homeostasis, it also performs metabolic functions in digestion and nutrient absorption, detoxification, and vitamin synthesis. Furthermore, the microbiota also appears to be an important factor in the development of the lymphoid system, 70% of which resides in the intestinal mucosa. In the literature there are various examples, both on mouse models and on humans, which demonstrate an altered composition of the intestinal microbiota in a number of different pathological disorders, including obesity, compared to the “ physiological” microbiota. For this reason, a substantial part of the studies on the gut microbiota treated so far has been dedicated to this topic. Interest in the role of the microbiota in health has increased, as evidenced by the May 2016 announcement of the National Microbiome Initiative (NMI) to promote the use of microbiota science in health care, but also for environmental restoration and food production. The new genome resource increases the capability to identify gut metagenomes over 87% ( [8](#B8) , [9](#B9) ). A recent study by Huiting et al. showed that iodine treatment affected the modulation mechanisms of thyroid function and the gut microbiota in obese mice. Administration of KIO3 in these animals led to weight reduction, increased concentration of thyroid hormones, alteration in the expression of the genes involved in thyroid biosynthesis, and cause various effects on the gut microbiota, changing the composition of the intestinal microenvironment, resulting in an imbalance of gut microbes: increases in pathogenic bacteria ( *Enterococcus* , *Clostridium* , *Fusobacterium nucleatum* , *Burkholderiales* , *Helicobacter* ), decrease in beneficial ones ( *Lactobacilli, Bifidobacteria* ), quite the contrary to what has been observed in non-obese hosts (p < 0. 05). Therefore, even with iodine dosages considered safe, obesity and quantitative and qualitative changes in the microbiota can increase the risk of thyroid dysfunction ( [10](#B10) ). Frohlich and Wahl concluded in their review that the composition of the microbiota and the different representation of its individual components in the various parts of the gastrointestinal system influence the absorption of I − , also modifying the enterohepatic circulation of thyroid hormones. Moreover, minerals such as Se, Fe, and Zn seem to be involved in the interaction between microbiota and host ( [11](#B11) ). The human microbiota seems to be a key regulator of health and diseases and the relevance of its influence on human health is gradually emerging. Gut dysbiosis is an alteration of the microbiota in its physiological function in the gastrointestinal tract; this condition can lead to local phlogosis and alteration of metabolic functions ( [12](#B12) ). In the gut dysbiosis there’s a low microbial diversity ( [13](#B13) ) that is related with an extended range of human diseases, including changes in host immune status ( [14](#B14) ), asthma, allergies, inflammatory bowel disease ( [15](#B15) – [18](#B18) ), irritable bowel syndrome ( [19](#B19) ), obesity ( [20](#B20) ), chronic kidney disease ( [21](#B21) ), cardiovascular disease ( [22](#B22) ), and changes in blood pressure regulation ( [23](#B23) ). Additionally, alterations in gut microbial composition or function have been associated with age-related health impairment ( [24](#B24) , [25](#B25) ). However, it’s still difficult to establish the connections between the dysbiosis and these conditions, except for few cases. Furthermore, also changes in the viral component of microbiome (virome) can be associated with infectious and inflammatory diseases. Intestinal eukaryotic viruses have been implicated in triggering human type 1 diabetes mellitus (T1D). Changes in the intestinal virome, in particular Circoviridae-related sequences, have been found in who developed serum autoantibodies associated with T1D, suggesting that changes in the intestinal virome preceded autoimmunity ( [26](#B26) ). Some viruses establish subclinical lifelong persistent or latent infections in their host thereby becoming part of the normal microbiome. Human viral ecology is poorly understood yet. Paul G. Cantalupo et al. have developed a virus detection and discovery computational pipeline, Pickaxe, and applied it to NGS databases provided by The Cancer Genome Atlas (TCGA). They analyzed a collection of whole genome (WGS), exome (WXS), and RNA (RNA-Seq) sequencing libraries from 3, 052 participants across 22 different cancers. HBV was also detected only in one sample of thyroid cancer, but these HBV sequences were due to sample cross contamination during sequencing, so they need to be confirmed. Moreover, viruses have long been considered potential triggers of autoimmune diseases ( [27](#B27) ).

## Gut Microbiome and Autoimmunity

The relation between autoimmunity and other systemic diseases has been for a long time examined by worldwide literature ( [28](#B28) – [30](#B30) ), moreover focusing on new therapeutic implications in both surgical and medical field ( [31](#B31) – [38](#B38) ). The pathogenesis of autoimmune disorders is due to genetic ( [4](#B4) – [7](#B7) , [23](#B23) – [25](#B25) , [39](#B39) – [41](#B41) ), stochastic ( [42](#B42) ), and environmental ( [43](#B43) – [49](#B49) ) factors. In the environmental factors that have gained attention during the last decade, there is the intestinal microbiota. Several studies have supported a connection of altered microbiota composition with the beginning of several different autoimmune disorders, suggesting its role in the pathogenesis. These include Type I diabetes ( [45](#B45) , [49](#B49) ); rheumatoid arthritis ( [50](#B50) – [53](#B53) ); systemic lupus erythematous ( [54](#B54) , [55](#B55) ); inflammatory bowel disease comprising Cohn’s disease and ulcerative colitis ( [19](#B19) , [56](#B56) ); Bechet’s disease ( [57](#B57) ); autoimmune skin conditions including vitiligo ( [58](#B58) ), atopic dermatitis ( [59](#B59) , [60](#B60) ); psoriasis vulgaris ( [61](#B61) ), and autoimmune neurological diseases ( [62](#B62) , [63](#B63) ). About 5–10% of the variability of bacterial taxa ascertained among individuals should be explained by genetic. Among the bacterial taxa transmitted, most of these is linked to genes involved in innate immunity ( [64](#B64) ). It is known that gastrointestinal microbiota is in reciprocal relationship with the host’s immune system: immune system, through an equilibrium of pro- and anti-inflammatory pathways, has an important role in the modulation of the microbiome community; on the other hand microbiome has a critical role in the development of the immune system ( [65](#B65) ).

## Microbiome and Thyroid Autoimmune Diseases

Autoimmune thyroid disease (ATD) is a common autoimmune disorder. It’s an organ-specific disorder and its prevalence is higher than 5% worldwide and still increasing ( [66](#B66) ). The principal manifestation of the ATD are Graves’ Disease (GD) and Hashimoto’s Thyroiditis (HT). In HT there’s a destruction of the thyroid cells that causes hypothyroidism, often detected in a subclinical condition ( [67](#B67) ), while in GD autoantibodies bind thyreotropin receptors stimulating an excessive production of thyroid hormone leading to hyperthyroidism. The role of T-lymphocytes and their cytokines is indispensable for the immune modulation, but it’s complex and full of connections with other components of the immunity. The correlation between gastrointestinal microbiota and development and progression of ATD has not yet been fully clarified. Nowadays, the analysis of reciprocal influence between microbiome and HT is a topic of considerable interest in the literature, since HT is the most frequent autoimmune disorder worldwide. There are several studies, both in animal models and in human, suggesting a link between modification in microbiota with the origins and development of ATD showing its key role in the thyroid peripheral homeostasis. Masetti et al. conducted a randomized controlled study on mouse models of GD. They studied the gut microbiota, observing a difference in biodiversity, spatial organization, and amount between the THSR immunized treated group and the untreated control group. The THSR immunized group developed signs of ophthalmopathy and their gut microbiota had more Firmicutes and less Bacteroides when comparing controls ( [68](#B68) ). However, already in 1988, Penhale and Young ( [69](#B69) ) noticed that modulation of the intestinal microbiota, in murine models affected by autoimmune thyroiditis, consistently made them more or less sensitive to thyroid autoimmunity. Lately, Köhling et al., through use of a PCR-denaturing gradient gel electrophoresis with universal primers targeting V3 region of the 16S rRNA gene and quantitative real-time PCR, showed, in subjects affected by hyperthyroidism when compared with hypothyroid patients, an important difference of the intestinal microbiota composition, especially an overgrowth of bacteria in the small intestine, assessed by breath test with hydrogen glucose ( [70](#B70) ). Ishaq et al. found that there is a significant disparity between the gastrointestinal microbiota of patients affected by HT and healthy controls (p < 0. 05). Particularly, they showed raised level of *Actinobacteria* in HT group as compared to control. Moreover, levels of *Prevotellaceae* and *Veillonellaceae* were lower in diseased group if compared with healthy control, as well as *Bifidobacterium* and *Lactobacillus* ; *Veillonellaceae* are commensal bacteria with an important role in regulation of adaptive immunity. At last, *Enterobacteriaceae* and *Alcaligenaceae* were higher in HT group as compared to control. *Shigella* and *Escherichia* can cause a broad spectrum of severe infectious diseases from hemorrhagic colitis to septicemia. In this paper, there’s a clear demarcation of intestinal microbiota texture between HT patients and healthy group. The authors hypothesize that the raised levels of autoantibodies in these subpopulations might modify the structure of gastrointestinal microbiota, but other studies are obviously necessary to comprehend the underlying pathogenesis ( [71](#B71) ). Zhao F. et al. found similar and various concentrations of bacterial in the bowel microbiome of patients suffering from HT and controls (p = 0. 11). This study showed that levels of *Blautia* , *Roseburia* , *Ruminococcus\_torques\_group* , *Romboutsia* , *Dorea* , *Fusicatenibacter* , and *Eubacterium\_hallii\_* group were higher in patients affected by HT, while *Fecalibacterium* , *Bacteroides* , *Prevotella\_9* , and *Lachnoclostridium* genera were lower in the same patients. Additionally, through the LEfSe method the authors were able to show many differences in these 27 genera composition between the patients and the healthy control, differences that were also correlated with clinical manifestation and laboratory data ( [72](#B72) ). Both in HT and GD the first therapy strategy is represented by the recovery of euthyroidism, in the first case by hormone replacement therapy, in the second case trough specific antithyroid drugs ( [73](#B73) ). The achievement of therapeutic goal may significantly differ, in both conditions, from patients to patients. Many factors can have a role: from age, sex, changes in BMI, to other conditions such as the thyroid hormones levels and the cause itself of the hypothyroidism, or the contemporary assumption of other drugs, some sorts of food and drink, soy protein, gastrointestinal malabsorption diseases and infections, bariatric surgery, atrophic gastritis, cystic fibrosis ( [74](#B74) ). All these parallel situations may influence the dosage of L-T4 replacement therapy in case of hypothyroidism and the severity of hyperthyroidism conditions in case of GD patients under antithyroid treatments. A disadvantage of antithyroid therapy is the high frequency of disease recurrence after the drug has been interrupted; these events occur often in the first year after discontinuing therapy, mostly in the first 6 months ( [75](#B75) ). In literature we aren’t able to find many evidences about a possible correlation between gut microbiota changes and response to drug therapy. Actual studies have improved our knowledge about human microbiota, its basic functions and dynamics. The characterization of specific subset of bacterial species in ATD could help to identify new therapeutic strategy, by using probiotics, to rapidly gain therapeutic goal. Probiotics are available in many formulations, i. e. in the form of fermentable food, powders, or liquid drops. Many evidences show that probiotics, when given in the just dosage and for an adequate period of time, may lead to beneficial effects to human health; moreover they are often safer than many drugs. Probiotic impact on the immunity is important to comprehend how to therapeutically approach may interfere to the global increasing incidence of autoimmune diseases. INDIGO performed a double-blind, placebo-controlled, randomized clinical study on the effects of a LAB4 probiotic on gut microbiota composition in patients with GD. There was a significant reduction in the Firmicutes phylum count in the treated group compared to placebo (P = 0. 033) and a temporary although important reduction in circulating autoantibodies and, consequently, in relapses at 6 months after antithyroid therapy, indicating the systemic immunomodulating effect of probiotics ( [76](#B76) ).

## Microbiota and Cancer

Some studies have shown the correlation of microbiota with cancer ( [77](#B77) ). Dysbiosis could initiate inflammatory and pro-carcinogenicity; conversely gut-derived probiotics restore gut commensal bacteria and protect the host including cancer disease, returning to a condition of wellness. Vivarelli et al. found that microbiota interferes directly host’s DNA replication and its integrity. He shows ways of how pathogenic bacteria are capable to promote oncogenesis through the modulation or interfering with specifics host’s oncogenic cell pathway or by interposing either with the hormonal or the host’s immune system. Microbiota changes can release toxins that induce human DNA damage, concurring to its mutability, tumor induction and progression in gastrointestinal cancer. The author continues saying that “ gut pathogenic bacteria can also disturb with DNA damage response and repair pathways, as in the case of *Shigella flexneri* , stimulating host’s cells p53 degradation *via* the secretion of its enzymes *inositol phosphate phosphatase D* (IpgD) and *cysteine protease-like virulence gene A* .” Thus, it can increase the risk to induce mutations during the DNA damage response in infected cells. *Cytotoxin associated gene A* from *Helicobacter pylori* , causes the proteasome-mediated degradation of p53 in gastric epithelial cells, by interfering with the host’s AKT pathway and inducing the gastric cancer. It is very complicated to determine in a clear way whether dysbiosis might have effects on the genesis of cancer or not. Additionally, changes in the everyday lifestyle, diet, and immune system factors, which deeply affect the microbiota composition and activity, may affect cancer genesis ( [78](#B78) ). Currently, Thyroid Carcinoma (TC) is the fifth most common malignancy diagnosed in female gender and its incidence is United States has increased an average of 3% per year in the last 4 decades; this increase is resulted by interactions among environmental factors, lifestyle, and genetic. Bowel microbiota, maybe, is an important environmental factor in gastrointestinal and extraintestinal tumor pathogenesis. However, regarding composition of bowel microbiome in patient affected by TC, there’s limited information. Two categories include the contribution of gut microbiota to tumor pathogenesis. The first category includes the damage to DNA and apoptosis; bacteria like *E. coli* and *Bacteroides fragilis* may influence the host genomic stability leading to DNA damage and mutations, having and important role to colorectal carcinogenesis. The second category includes inflammatory reactions; many pattern recognitions receptors, comprehending *Toll-Like Receptors* (TLRs) are activated by cancer-associated microbial communities stimulating in loop *NF-kB* signaling activation in the tumor microenvironment. Moreover, tumorigenesis obviously is influenced by specific pathogens and metabolic outputs of the gut microbiota. However, it has become increasingly clear that the collective activities of resident gut microbiota, particularly their metabolic products, strongly influence the protection against and predisposition to the development of malignancies. Up to now, there are only two reports in the literature, regarding the characterization of microbiota between healthy subjects, thyroid benign nodules and thyroid cancer patients. Feng et al. evaluated relationships among gastrointestinal microbiota, fecal metabolites, and TC. Through their researches they have noticed significative differences in gastrointestinal microbial communities from patients affected by TC and not affected ones, and also to the high or low quantity of bacteria’s genera in TC, by using Ultra-performance liquid chromatography profiling of fecal samples. In fecal metabolites composition were also identified many differences, indeed 72 metabolites showed important changes. Patients affected by TC were successfully identified from a combination of eight metabolites and five genera. Moreover, the findings implicate alterations in gut microbiota and metabolites in TC pathogenesis ( [79](#B79) ). In a recent cohort study by Zhang et al., the intestinal microbiota was compared with endocrine thyroid function both in patients with thyroid cancer and in subjects with benign nodular thyroid disease. The results made evident a difference in the microbiota between the thyroid pathology, both benign and malignant, and the healthy population: in particular, it was noticed a significant higher level of *Neisseria* and *Streptococcus* in patients with thyroid diseases while *Butyricimonas* underwent a significant decrease in patients with thyroid cancer and the same happened for *Lactobacillus* in patients with thyroid nodules (p < 0. 001). This shows that both pathological situations, benign and malignant thyroid diseases, correlate with the composition of the gut microbiota and consequently poses the possibility of any future treatments with specific probiotics ( [80](#B80) ). No data are present in the literature about the characterization of intestinal virome in thyroid cancer and thyroid nodules. Data collected over the past decade have identified the gut microbiota as an important factor defining inter individual variation in diseases risk. Evidence shows that gut microbiota and metabolites alter and potentially control cancerogenesis and progression.

## Conclusion

Microbiome-wide association studies have established that numerous diseases are associated with changes in the microbiota. These studies typically generate a long list of commensals implicated as biomarkers of disease, with no clear relevance to disease pathogenesis. A major challenge is to understand which of the many diseases, including inflammatory diseases and various forms of cancer and cancer risk factors are detectable as different locations on this map. Our mini review tried to evaluate the correlation between thyroid diseases and gut dysbiosis, demonstrating that a changing in quality and quantity of intestinal commensal and pathogenic bacteria is associated with development of thyroid endocrine disease such as Hashimoto Thyroiditis and Graves’ Disease as well as thyroid carcinoma. Future investigations are needed to identify the optimal probiotic and dose for specific diseases. The future of these researches is represented by the need to find the strict and direct correlation between the microbial changes and the specific pathology in order to identify the most targeted therapy possible, customized on each patient.

## Author Contributions

All authors contributed significantly to the present research and reviewed the entire manuscript. GDoc: Participated substantially in conception and design of the manuscript and in the analysis and interpretation of the data. AC: Participated substantially in conception, design, and execution of the study and in the analysis and interpretation of the data; also participated substantially in the drafting and editing of the manuscript. RR, MF, CO, VA, MG, GDon, VN, MR, GC and DP: Participated substantially in conception and design of the manuscript and in the analysis and interpretation of the data. All the authors have read an approved the final manuscript.

## Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

## References

1. Petrillo F, Pignataro D, Lavano MA, Santella B, Folliero V, Zannella C, et al. Current Evidence on the Ocular Surface Microbiota and Related Diseases. *Microorganisms* (2020) 8(7): 1033. doi: 10. 3390/microorganisms8071033

[CrossRef Full Text](https://doi.org/10.3390/microorganisms8071033) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=F+Petrillo&author=D+Pignataro&author=MA+Lavano&author=B+Santella&author=V+Folliero&author=C+Zannella&publication_year=2020&title=Current Evidence on the Ocular Surface Microbiota and Related Diseases&journal=Microorganisms&volume=8&)

2. Hehemann JH, Correc G, Barbeyron T, Helbert W, Czjzek M, Michel G. Transfer of carbohydrate-active enzymes from marine bacteria to Japanese gut microbiota. *Nature* (2010) 464(7290): 908–12. doi: 10. 1038/nature08937

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/20376150/) | [CrossRef Full Text](https://doi.org/10.1038/nature08937) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=JH+Hehemann&author=G+Correc&author=T+Barbeyron&author=W+Helbert&author=M+Czjzek&author=G+Michel&publication_year=2010&title=Transfer of carbohydrate-active enzymes from marine bacteria to Japanese gut microbiota&journal=Nature&volume=464&pages=908-12)

3. Turnbaugh PJ, Gordon JI. The core gut microbiome, energy balance and obesity. *J Physiol* (2009) 587(Pt 17): 4153–8. doi: 10. 1113/jphysiol. 2009. 174136

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/19491241/) | [CrossRef Full Text](https://doi.org/10.1113/jphysiol.2009.174136) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=PJ+Turnbaugh&author=JI+Gordon&publication_year=2009&title=The core gut microbiome%2C energy balance and obesity&journal=J+Physiol&volume=587&pages=4153-8)

4. Relman DA. The human microbiome, ecosystem resilience and health. *Nutr Rev* (2012) 70(Suppl 1): S2–9. doi: 10. 1111/j. 1753-4887. 2012. 00489. x

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/22861804/) | [CrossRef Full Text](https://doi.org/10.1111/j.1753-4887.2012.00489.x) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=DA+Relman&publication_year=2012&title=The human microbiome%2C ecosystem resilience and health&journal=Nutr+Rev&volume=70&pages=S2-9)

5. Baldridge MT, Nice TJ, McCune BT, Yokoyama CC, Kambal A, Whea- don M, et al. Commensal microbes and interferon-lambda determine persistence of enteric murine norovirus infection. *Science* (2015) 347(6219): 266–9. doi: 10. 1126/science. 1258025

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/25431490/) | [CrossRef Full Text](https://doi.org/10.1126/science.1258025) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=MT+Baldridge&author=TJ+Nice&author=BT+McCune&author=CC+Yokoyama&author=A+Kambal&author=M+Whea- don&publication_year=2015&title=Commensal microbes and interferon-lambda determine persistence of enteric murine norovirus infection&journal=Science&volume=347&pages=266-9)

6. Geddes K, Rubino SJ, Magalhaes JG, Streutker C, Le Bourhis L, Cho JH, et al. Identification of an innate T helper type 17 response to intestinal bacterial pathogens. *Nat Med* (2011) 17(7): 837–44. doi: 10. 1038/nm. 2391

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/21666695/) | [CrossRef Full Text](https://doi.org/10.1038/nm.2391) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=K+Geddes&author=SJ+Rubino&author=JG+Magalhaes&author=C+Streutker&author=L+Le Bourhis&author=JH+Cho&publication_year=2011&title=Identification of an innate T helper type 17 response to intestinal bacterial pathogens&journal=Nat+Med&volume=17&pages=837-44)

7. Dachao L, Ross Ka-Kit L, Wenda G, William WA. Involvement of gut microbiome in human health and disease, brief overview, knowledge gaps and research opportunities. *Gut Pathog* (2018) 10: 3. doi: 10. 1186/s13099-018-0230-4

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29416567/) | [CrossRef Full Text](https://doi.org/10.1186/s13099-018-0230-4) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=L+Dachao&author=L+Ross Ka-Kit&author=G+Wenda&author=WA+William&publication_year=2018&title=Involvement of gut microbiome in human health and disease%2C brief overview%2C knowledge gaps and research opportunities&journal=Gut+Pathog&volume=10&pages=3)

8. Sinha R, Ahsan H, Blaser M, Caporaso JG, Carmical JR, Chan AT, et al. Next steps in studying the human microbiome and health in prospective studies, Bethesda. *Microbiome* (2018) 6(1): 210. doi: 10. 1186/s40168-018-0596-z

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30477563/) | [CrossRef Full Text](https://doi.org/10.1186/s40168-018-0596-z) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=R+Sinha&author=H+Ahsan&author=M+Blaser&author=JG+Caporaso&author=JR+Carmical&author=AT+Chan&publication_year=2018&title=Next steps in studying the human microbiome and health in prospective studies%2C Bethesda&journal=Microbiome&volume=6&pages=210)

9. Pasolli E, Asnicar F, Manara S, Zolfo M, Karcher N, Armanini F, et al. Extensive Unexplored Human Microbiome Diversity Revealed by Over 150, 000 Genomes from Metagenomes Spanning Age, Geography, and Lifestyle. *Cell* (2019) 176(3): 649–62. doi: 10. 1016/j. cell. 2019. 01. 001

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30661755/) | [CrossRef Full Text](https://doi.org/10.1016/j.cell.2019.01.001) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=E+Pasolli&author=F+Asnicar&author=S+Manara&author=M+Zolfo&author=N+Karcher&author=F+Armanini&publication_year=2019&title=Extensive Unexplored Human Microbiome Diversity Revealed by Over 150%2C000 Genomes from Metagenomes Spanning Age%2C Geography%2C and Lifestyle&journal=Cell&volume=176&pages=649-62)

10. Huiting S, Jiaojiao H, Yanyan L, Chenyang L, Jun Z, Ye L, et al. Different host-specific responses in thyroid function and gut microbiota modulation between diet-induced obese and normal mice given the same dose of iodine. *Appl Microbiol Biotechnol* (2019) 103(8): 3537–47. doi: 10. 1007/s00253-019-09687-1

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30850874/) | [CrossRef Full Text](https://doi.org/10.1007/s00253-019-09687-1) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+Huiting&author=H+Jiaojiao&author=L+Yanyan&author=L+Chenyang&author=Z+Jun&author=L+Ye&publication_year=2019&title=Different host-specific responses in thyroid function and gut microbiota modulation between diet-induced obese and normal mice given the same dose of iodine&journal=Appl+Microbiol+Biotechnol&volume=103&pages=3537-47)

11. Fröhlich E, Wahl R. Microbiota and Thyroid Interaction in Health and Disease. *Trends Endocrinol Metab* (2019) 30(8): 479–90. doi: 10. 1016/j. tem. 2019. 05. 008

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/31257166/) | [CrossRef Full Text](https://doi.org/10.1016/j.tem.2019.05.008) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=E+Fröhlich&author=R+Wahl&publication_year=2019&title=Microbiota and Thyroid Interaction in Health and Disease&journal=Trends+Endocrinol+Metab&volume=30&pages=479-90)

12. Petersen C, Round JL. Defining dysbiosis and its influence on host immunity and disease. *Cell Microbiol* (2014) 16(7): 1024–33. doi: 10. 1111/cmi. 12308

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/24798552/) | [CrossRef Full Text](https://doi.org/10.1111/cmi.12308) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=C+Petersen&author=JL+Round&publication_year=2014&title=Defining dysbiosis and its influence on host immunity and disease&journal=Cell+Microbiol&volume=16&pages=1024-33)

13. Kriss M, Hazleton KZ, Nusbacher NM, Martin CG, Lozupone CA. Low diversity gut microbiota dysbiosis, drivers, functional implications and recovery. *Curr Opin Microbiol* (2018) 44: 34–40. doi: 10. 1016/j. mib. 2018. 07. 003

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30036705/) | [CrossRef Full Text](https://doi.org/10.1016/j.mib.2018.07.003) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=M+Kriss&author=KZ+Hazleton&author=NM+Nusbacher&author=CG+Martin&author=CA+Lozupone&publication_year=2018&title=Low diversity gut microbiota dysbiosis%2C drivers%2C functional implications and recovery&journal=Curr+Opin+Microbiol&volume=44&pages=34)

14. Rooks MG, Garrett WS. Gut microbiota, metabolites and host immunity. *Nat Rev Immunol* (2016) 16(6): 341–52. doi: 10. 1038/nri. 2016. 42

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27231050/) | [CrossRef Full Text](https://doi.org/10.1038/nri.2016.42) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=MG+Rooks&author=WS+Garrett&publication_year=2016&title=Gut microbiota%2C metabolites and host immunity&journal=Nat+Rev+Immunol&volume=16&pages=341-52)

15. Carding S, Verbeke K, Vipond DT, Corfe BM, Owen LJ. Dysbiosis of the gut microbiota in disease. *Microb Ecol Health Dis* (2015) 26: 26191. doi: 10. 3402/mehd. v26. 26191

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/25651997/) | [CrossRef Full Text](https://doi.org/10.3402/mehd.v26.26191) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+Carding&author=K+Verbeke&author=DT+Vipond&author=BM+Corfe&author=LJ+Owen&publication_year=2015&title=Dysbiosis of the gut microbiota in disease&journal=Microb+Ecol+Health+Dis&volume=26&pages=26191)

16. Arrieta MC, Stiemsma LT, Dimitriu PA, Thorson L, Russell S, Yurist-Doutsch S, et al. Early infancy microbial and metabolic alterations affect risk of childhood asthma. *Sci Transl Med* (2015) 7(307): ra152. doi: 10. 1126/scitranslmed. aab2271

[CrossRef Full Text](https://doi.org/10.1126/scitranslmed.aab2271) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=MC+Arrieta&author=LT+Stiemsma&author=PA+Dimitriu&author=L+Thorson&author=S+Russell&author=S+Yurist-Doutsch&publication_year=2015&title=Early infancy microbial and metabolic alterations affect risk of childhood asthma&journal=Sci+Transl+Med&volume=7&pages=ra152)

17. Bunyavanich S, Shen N, Grishin A, Wood R, Burks W, Dawson P, et al. Early-life gut microbiome composition and milk allergy resolution. *J Allergy Clin Immunol* (2016) 138(4): 1122–30. doi: 10. 1016/j. jaci. 2016. 03. 041

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27292825/) | [CrossRef Full Text](https://doi.org/10.1016/j.jaci.2016.03.041) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+Bunyavanich&author=N+Shen&author=A+Grishin&author=R+Wood&author=W+Burks&author=P+Dawson&publication_year=2016&title=Early-life gut microbiome composition and milk allergy resolution&journal=J+Allergy+Clin+Immunol&volume=138&pages=1122-30)

18. Nishino K, Nishida A, Inoue R, Kawada Y, Ohno M, Sakai S, et al. Analysis of endoscopic brush samples identified mucosa-associated dysbiosis in inflammatory bowel disease. *J Gastroenterol* (2018) 53(1): 95–106. doi: 10. 1007/s00535-017-1384-4

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28852861/) | [CrossRef Full Text](https://doi.org/10.1007/s00535-017-1384-4) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=K+Nishino&author=A+Nishida&author=R+Inoue&author=Y+Kawada&author=M+Ohno&author=S+Sakai&publication_year=2018&title=Analysis of endoscopic brush samples identified mucosa-associated dysbiosis in inflammatory bowel disease&journal=J+Gastroenterol&volume=53&pages=95)

19. Cao SS. Cellular Stress Responses and Gut Microbiota in Inflammatory Bowel Disease. *Gastroenterol Res Pract* (2018) 2018: 7192646. doi: 10. 1155/2018/7192646

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30026758/) | [CrossRef Full Text](https://doi.org/10.1155/2018/7192646) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=SS+Cao&publication_year=2018&title=Cellular Stress Responses and Gut Microbiota in Inflammatory Bowel Disease&journal=Gastroenterol+Res+Pract&volume=2018&pages=7192646)

20. Schwiertz A, Taras D, Schäfer K, Beijer S, Bos NA, Donus C, et al. Microbiota and SCFA in lean and overweight healthy subjects. *Obes (Silver Spring)* (2010) 18(1): 190–5. doi: 10. 1038/oby. 2009. 167

[CrossRef Full Text](https://doi.org/10.1038/oby.2009.167) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=A+Schwiertz&author=D+Taras&author=K+Schäfer&author=S+Beijer&author=NA+Bos&author=C+Donus&publication_year=2010&title=Microbiota and SCFA in lean and overweight healthy subjects&journal=Obes+(Silver+Spring)&volume=18&pages=190-5)

21. Sircana A, De Michieli F, Parente R, Framarin L, Leone N, Berrutti M, et al. Gut Microbiota, Hypertension and Chronic kidney Disease, recent advances. *Pharmacol Res* (2018) 144: 390–408. doi: 10. 1016/j. phrs. 2018. 01. 013

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29378252/) | [CrossRef Full Text](https://doi.org/10.1016/j.phrs.2018.01.013) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=A+Sircana&author=F+De Michieli&author=R+Parente&author=L+Framarin&author=N+Leone&author=M+Berrutti&publication_year=2018&title=Gut Microbiota%2C Hypertension and Chronic kidney Disease%2C recent advances&journal=Pharmacol+Res&volume=144&pages=390)

22. Jie Z, Xia H, Zhong SL, Feng Q, Li S, Liang S, et al. The gut microbiome in atherosclerotic cardiovascular disease. *Nat Commun* (2017) 8(1): 845. doi: 10. 1038/s41467-017-00900-1

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29018189/) | [CrossRef Full Text](https://doi.org/10.1038/s41467-017-00900-1) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=Z+Jie&author=H+Xia&author=SL+Zhong&author=Q+Feng&author=S+Li&author=S+Liang&publication_year=2017&title=The gut microbiome in atherosclerotic cardiovascular disease&journal=Nat+Commun&volume=8&pages=845)

23. Marques FZ, Mackay CR, Kaye DM. Beyond gut feelings, how the gut microbiota regulates blood pressure. *Nat Rev Cardiol* (2018) 15(1): 20–32. doi: 10. 1038/nrcardio. 2017. 120

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28836619/) | [CrossRef Full Text](https://doi.org/10.1038/nrcardio.2017.120) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=FZ+Marques&author=CR+Mackay&author=DM+Kaye&publication_year=2018&title=Beyond gut feelings%2C how the gut microbiota regulates blood pressure&journal=Nat+Rev+Cardiol&volume=15&pages=20)

24. Clark RI, Walker DW. Role of gut microbiota in aging-related health decline, insights from invertebrate models. *Cell Mol Life Sci* (2018) 75(1): 93–101. doi: 10. 1007/s00018-017-2671-1

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29026921/) | [CrossRef Full Text](https://doi.org/10.1007/s00018-017-2671-1) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=RI+Clark&author=DW+Walker&publication_year=2018&title=Role of gut microbiota in aging-related health decline%2C insights from invertebrate models&journal=Cell+Mol+Life+Sci&volume=75&pages=93)

25. O’Toole PW, Jeffery IB. Microbiome-health interactions in older people. *Cell Mol Life Sci* (2018) 75(1): 119–28. doi: 10. 1007/s00018-017-2673-z

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28986601/) | [CrossRef Full Text](https://doi.org/10.1007/s00018-017-2673-z) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=PW+O’Toole&author=IB+Jeffery&publication_year=2018&title=Microbiome-health interactions in older people&journal=Cell+Mol+Life+Sci&volume=75&pages=119-28)

26. Zhao G, Vatanen T, Droit L, Park A, Kostic AD, Poon TW, et al. Intestinal virome changes precede autoimmunity in type I diabetes-susceptible children. *Proc Natl Acad Sci USA* (2017) 114(30): E6166–75. doi: 10. 1073/pnas. 1706359114

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28696303/) | [CrossRef Full Text](https://doi.org/10.1073/pnas.1706359114) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Zhao&author=T+Vatanen&author=L+Droit&author=A+Park&author=AD+Kostic&author=TW+Poon&publication_year=2017&title=Intestinal virome changes precede autoimmunity in type I diabetes-susceptible children&journal=Proc+Natl+Acad+Sci+USA&volume=114&pages=E6166-75)

27. Cantalupo PG, Katz JP, Pipas JM. Viral sequences in human cancer. *Virology* (2018) 513: 208–16. doi: 10. 1016/j. virol. 2017. 10. 017

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29107929/) | [CrossRef Full Text](https://doi.org/10.1016/j.virol.2017.10.017) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=PG+Cantalupo&author=JP+Katz&author=JM+Pipas&publication_year=2018&title=Viral sequences in human cancer&journal=Virology&volume=513&pages=208-16)

28. Pasquali D, Maiorino N II, Renzullo A, Bellastella G, Accardo G, Esposito D, et al. Female sexual dysfunction in women in thyroid disorders. *J Endocrinol Invest* (2013) 36: 729–33. doi: 10. 3275/8933

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/23580027/) | [CrossRef Full Text](https://doi.org/10.3275/8933) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=D+Pasquali&author=N+Maiorino+II&author=A+Renzullo&author=G+Bellastella&author=G+Accardo&author=D+Esposito&publication_year=2013&title=Female sexual dysfunction in women in thyroid disorders&journal=J+Endocrinol+Invest&volume=36&pages=729-33)

29. Balercia G, Bonomi M, Giagulli VA, Lanfranco F, Rochira V, Giambersio A, et al. Thyroid function in Klinefelter syndrome: a multicentre study from KING group. *J Endocrinol Invest* (2019) 42(10): 1199–204. doi: 10. 1007/s40618-019/01037-2

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30912057/) | [CrossRef Full Text](https://doi.org/10.1007/s40618-019/01037-2) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Balercia&author=M+Bonomi&author=VA+Giagulli&author=F+Lanfranco&author=V+Rochira&author=A+Giambersio&publication_year=2019&title=Thyroid function in Klinefelter syndrome%3A a multicentre study from KING group&journal=J+Endocrinol+Invest&volume=42&pages=1199-204)

30. Pasquali D, Notaro A, Bonavolontà G, Vassallo P, Bellastella A, Sinisi AA. Somatostatin receptor genes are expressed in lymphocytes from retroorbital tissues in Graves’ Disease. *J Clin Endocrinol Metab* (2002) 87(11): 5525–129. doi: 10. 1210/jc. 2002-020790

[CrossRef Full Text](https://doi.org/10.1210/jc.2002-020790) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=D+Pasquali&author=A+Notaro&author=G+Bonavolontà&author=P+Vassallo&author=A+Bellastella&author=AA+Sinisi&publication_year=2002&title=Somatostatin receptor genes are expressed in lymphocytes from retroorbital tissues in Graves’ Disease&journal=J+Clin+Endocrinol+Metab&volume=87&pages=5525-129)

31. Esposito D, Rotondi M, Accardo G, Vallone G, Conzo G, Docimo G, et al. Influcence of short-term selenium supplementation on the natural course of Hashimoto’s Thyroiditis: clinical results of a blinded placebo-controlled randomized prospective trial. *J Endocrinol Invest* (2017) 40(1): 83–9. doi: 10. 1007/s40618-016-0535-4

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27572248/) | [CrossRef Full Text](https://doi.org/10.1007/s40618-016-0535-4) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=D+Esposito&author=M+Rotondi&author=G+Accardo&author=G+Vallone&author=G+Conzo&author=G+Docimo&publication_year=2017&title=Influcence of short-term selenium supplementation on the natural course of Hashimoto’s Thyroiditis%3A clinical results of a blinded placebo-controlled randomized prospective trial&journal=J+Endocrinol+Invest&volume=40&pages=83-9)

32. Pirola I, Rotondi M, Cristiano A, Maffezzoni F, Pasquali D, Marini F, et al. Selenium supplementation in patients with subclinical hypothiroidism affected by autoimmune thyroiditis: Results of the SETI study. *Endocrinol Diabetes Nutr* (2020) 67(1): 2835. doi: 10. 1016/j. endien. 2019. 12. 002

[CrossRef Full Text](https://doi.org/10.1016/j.endien.2019.12.002) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=I+Pirola&author=M+Rotondi&author=A+Cristiano&author=F+Maffezzoni&author=D+Pasquali&author=F+Marini&publication_year=2020&title=Selenium supplementation in patients with subclinical hypothiroidism affected by autoimmune thyroiditis%3A Results of the SETI study&journal=Endocrinol+Diabetes+Nutr&volume=67&pages=2835)

33. Croce L, De Martinis L, Pinto S, Coperchini F, Dito G, Bendotti G, et al. Compared with classic Hashimoto’s Thyroiditis, chronic autoimmune serum-negative thyroiditis requires a lower substitution dose of L-thyroxine to correct hypothyroidism. *J Endocrinol Invest* (2020) 43(11): 1631–6. doi: 10. 1007/s40618-020-01249-x

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/32319047/) | [CrossRef Full Text](https://doi.org/10.1007/s40618-020-01249-x) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=L+Croce&author=L+De Martinis&author=S+Pinto&author=F+Coperchini&author=G+Dito&author=G+Bendotti&publication_year=2020&title=Compared with classic Hashimoto’s Thyroiditis%2C chronic autoimmune serum-negative thyroiditis requires a lower substitution dose of L-thyroxine to correct hypothyroidism&journal=J+Endocrinol+Invest&volume=43&pages=1631-6)

34. Ruggiero R, Gubitosi A, Conzo G, Gili S, Bosco A, Pierozzi R, et al. Sutureless thyroidectomy. *Int J Surg* (2014) 12: 189–93. doi: 10. 1016/j. ijsu. 2014. 05. 011

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/24316390/) | [CrossRef Full Text](https://doi.org/10.1016/j.ijsu.2014.05.011) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=R+Ruggiero&author=A+Gubitosi&author=G+Conzo&author=S+Gili&author=A+Bosco&author=R+Pierozzi&publication_year=2014&title=Sutureless thyroidectomy&journal=Int+J+Surg&volume=12&pages=189-93)

35. Docimo G, Tolone S, Pasquali D, Conzo G, D’Alessandro A, Casalino G, et al. Role of pre and post-operative oral calcium and vitamin D supplements in prevention of hypocalcemia after total thyroidectomy. *J Surg* (2012) 33(11-12): 974–8.

[Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Docimo&author=S+Tolone&author=D+Pasquali&author=G+Conzo&author=A+D’Alessandro&author=G+Casalino&publication_year=2012&title=Role of pre and post-operative oral calcium and vitamin D supplements in prevention of hypocalcemia after total thyroidectomy&journal=J+Surg&volume=33&pages=974-8)

36. Pezzolla A, Docimo G, Ruggiero R, Monacelli M, Cirocchi R, Parmeggiani D, et al. Incidental thyroid carcinoma: a multicentric experience. *Recenti Prog Med* (2010) 101(5): 194–8. doi: 10. 1701/493. 5851

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/20590015/) | [CrossRef Full Text](https://doi.org/10.1701/493.5851) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=A+Pezzolla&author=G+Docimo&author=R+Ruggiero&author=M+Monacelli&author=R+Cirocchi&author=D+Parmeggiani&publication_year=2010&title=Incidental thyroid carcinoma%3A a multicentric experience&journal=Recenti+Prog+Med&volume=101&pages=194-8)

37. Docimo G, Tolone S, Ruggiero R, Gubitosi A, Pasquali D, De Bellis A, et al. Total thyroidectomy without profilattic central neck dissection combined with routine oral calcium and vitamine D supplement: Is it a good option to archieve a low recurrence rate avoiding hypocalcemia? A retrospective study. *Minerva Chirurgica* (2013) 68(3): 321–8.

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/23774098/) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Docimo&author=S+Tolone&author=R+Ruggiero&author=A+Gubitosi&author=D+Pasquali&author=A+De Bellis&publication_year=2013&title=Total thyroidectomy without profilattic central neck dissection combined with routine oral calcium and vitamine D supplement%3A Is it a good option to archieve a low recurrence rate avoiding hypocalcemia%3F A retrospective study&journal=Minerva+Chirurgica&volume=68&pages=321-8)

38. Docimo G, Ruggiero R, Casalino G, Del Genio G, Docimo L, Tolone S. Risk factors for postoperative hypocalcemia. *Updates Surg* (2017) 69(2): 255–60. doi: 10. 1007/s13304-017-0452-x

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28444542/) | [CrossRef Full Text](https://doi.org/10.1007/s13304-017-0452-x) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Docimo&author=R+Ruggiero&author=G+Casalino&author=G+Del Genio&author=L+Docimo&author=S+Tolone&publication_year=2017&title=Risk factors for postoperative hypocalcemia&journal=Updates+Surg&volume=69&pages=255-60)

39. Ajayi T, Innes CL, Grimm SA, Rai P, Finethy R, Coers J, et al. Crohn’s disease IRGM risk alleles are associated with altered gene expression in human tissues. *Am J Physiol Gastrointest Liver Physiol* (2019) 316: G95–G105. doi: 10. 1152/ajpgi. 00196. 2018

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30335469/) | [CrossRef Full Text](https://doi.org/10.1152/ajpgi.00196.2018) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=T+Ajayi&author=CL+Innes&author=SA+Grimm&author=P+Rai&author=R+Finethy&author=J+Coers&publication_year=2019&title=Crohn’s disease IRGM risk alleles are associated with altered gene expression in human tissues&journal=Am+J+Physiol+Gastrointest+Liver+Physiol&volume=316&pages=G95)

40. Batura V, Muise AM. Very early onset IBD, Novel genetic aetiologies. *Curr Opin Allergy Clin Immunol* (2018) 18: 470–80. doi: 10. 1097/ACI. 0000000000000486

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30299396/) | [CrossRef Full Text](https://doi.org/10.1097/ACI.0000000000000486) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=V+Batura&author=AM+Muise&publication_year=2018&title=Very early onset IBD%2C Novel genetic aetiologies&journal=Curr+Opin+Allergy+Clin+Immunol&volume=18&pages=470-80)

41. Zhang J, Meng Y, Wu H, Wu Y, Yang B, Wang L. Association between PPP2CA polymorphisms and clinical features in southwest Chinese systemic lupus erythematosus patients. *Medicine* (2018) 97(27): e11451. doi: 10. 1097/MD. 0000000000011451

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29979448/) | [CrossRef Full Text](https://doi.org/10.1097/MD.0000000000011451) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=J+Zhang&author=Y+Meng&author=H+Wu&author=Y+Wu&author=B+Yang&author=L+Wang&publication_year=2018&title=Association between PPP2CA polymorphisms and clinical features in southwest Chinese systemic lupus erythematosus patients&journal=Medicine&volume=97&pages=e11451)

42. Wu YL, Ding YP, Gao J, Tanaka Y, Zhang W. Risk factors and primary prevention trials for type 1 diabetes. *Int J Biol Sci* (2013) 9: 666–79. doi: 10. 7150/ijbs. 6610

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/23904791/) | [CrossRef Full Text](https://doi.org/10.7150/ijbs.6610) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=YL+Wu&author=YP+Ding&author=J+Gao&author=Y+Tanaka&author=W+Zhang&publication_year=2013&title=Risk factors and primary prevention trials for type 1 diabetes&journal=Int+J+Biol+Sci&volume=9&pages=666-79)

43. Barbeau WE. What is the key environmental trigger in type 1 diabetes—Is it viruses, or wheat gluten, or both? *Autoimmun Rev* (2012) 12: 295–9. doi: 10. 1016/j. autrev. 2012. 05. 003

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/22633932/) | [CrossRef Full Text](https://doi.org/10.1016/j.autrev.2012.05.003) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=WE+Barbeau&publication_year=2012&title=What is the key environmental trigger in type 1 diabetes—Is it viruses%2C or wheat gluten%2C or both&journal=Autoimmun+Rev&volume=12&pages=295-9)

44. Lindoso L, Mondal K, Venkateswaran S, Somineni HK, Ballengee C, Walters TD, et al. The Effect of Early-Life Environmental Exposures on Disease Phenotype and Clinical Course of Crohn’s Disease in Children. *Am J Gastroenterol* (2018) 113: 1524–9. doi: 10. 1038/s41395-018-0239-9

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30267029/) | [CrossRef Full Text](https://doi.org/10.1038/s41395-018-0239-9) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=L+Lindoso&author=K+Mondal&author=S+Venkateswaran&author=HK+Somineni&author=C+Ballengee&author=TD+Walters&publication_year=2018&title=The Effect of Early-Life Environmental Exposures on Disease Phenotype and Clinical Course of Crohn’s Disease in Children&journal=Am+J+Gastroenterol&volume=113&pages=1524-9)

45. Gianchecchi E, Fierabracci A. On the pathogenesis of insulin-dependent diabetes mellitus, The role of microbiota. *Immunol Res* (2017) 65: 242–56. doi: 10. 1007/s12026-016-8832-8

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27421719/) | [CrossRef Full Text](https://doi.org/10.1007/s12026-016-8832-8) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=E+Gianchecchi&author=A+Fierabracci&publication_year=2017&title=On the pathogenesis of insulin-dependent diabetes mellitus%2C The role of microbiota&journal=Immunol+Res&volume=65&pages=242-56)

46. Gianchecchi E, Fierabracci A. Recent Advances on Microbiota Involvement in the Pathogenesis of Autoimmunity. *Int J Mol Sci* (2019) 20(2): 283. doi: 10. 3390/ijms20020283

[CrossRef Full Text](https://doi.org/10.3390/ijms20020283) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=E+Gianchecchi&author=A+Fierabracci&publication_year=2019&title=Recent Advances on Microbiota Involvement in the Pathogenesis of Autoimmunity&journal=Int+J+Mol+Sci&volume=20&pages=283)

47. Henschel AM, Cabrera SM, Kaldunski ML, Jia S, Geoffrey R, Roethle MF, et al. Modulation of the diet and gastrointestinal microbiota normalizes systemic inflammation and β-cell chemokine expression associated with autoimmune diabetes susceptibility. *PloS One* (2018) 13(1): e0190351. doi: 10. 1371/journal. pone. 0190351

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29293587/) | [CrossRef Full Text](https://doi.org/10.1371/journal.pone.0190351) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=AM+Henschel&author=SM+Cabrera&author=ML+Kaldunski&author=S+Jia&author=R+Geoffrey&author=MF+Roethle&publication_year=2018&title=Modulation of the diet and gastrointestinal microbiota normalizes systemic inflammation and β-cell chemokine expression associated with autoimmune diabetes susceptibility&journal=PloS+One&volume=13&pages=e0190351)

48. Huang Y, Li SC, Hu J, Ruan HB, Guo HM, Zhang HH, et al. Gut microbiota profiling in Han Chinese with type 1 diabetes. *Diabetes Res Clin Pract* (2018) 141: 256–63. doi: 10. 1016/j. diabres. 2018. 04. 032

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29733871/) | [CrossRef Full Text](https://doi.org/10.1016/j.diabres.2018.04.032) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=Y+Huang&author=SC+Li&author=J+Hu&author=HB+Ruan&author=HM+Guo&author=HH+Zhang&publication_year=2018&title=Gut microbiota profiling in Han Chinese with type 1 diabetes&journal=Diabetes+Res+Clin+Pract&volume=141&pages=256-63)

49. Mullaney JA, Stephens JE, Costello ME, Fong C, Geeling BE, Gavin PG, et al. Correction to, Type 1 diabetes susceptibility alleles are associated with distinct alterations in the gut microbiota. *Microbiome* (2018) 6: 51. doi: 10. 1186/s40168-018-0438-z

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29558994/) | [CrossRef Full Text](https://doi.org/10.1186/s40168-018-0438-z) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=JA+Mullaney&author=JE+Stephens&author=ME+Costello&author=C+Fong&author=BE+Geeling&author=PG+Gavin&publication_year=2018&title=Correction to%2C Type 1 diabetes susceptibility alleles are associated with distinct alterations in the gut microbiota&journal=Microbiome&volume=6&pages=51)

50. Sato K, Takahashi N, Kato T, Matsuda Y, Yokoji M, Yamada M, et al. Aggravation of collagen-induced arthritis by orally administered Porphyromonas gingivalis through modulation of the gut microbiota and gut immune system. *Sci Rep* (2017) 7: 6955. doi: 10. 1038/s41598-017-07196-7

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28761156/) | [CrossRef Full Text](https://doi.org/10.1038/s41598-017-07196-7) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=K+Sato&author=N+Takahashi&author=T+Kato&author=Y+Matsuda&author=M+Yokoji&author=M+Yamada&publication_year=2017&title=Aggravation of collagen-induced arthritis by orally administered Porphyromonas gingivalis through modulation of the gut microbiota and gut immune system&journal=Sci+Rep&volume=7&pages=6955)

51. Teng F, Felix KM, Bradley CP, Naskar D, Ma H, Raslan WA, et al. The impact of age and gut microbiota on Th17 and Tfh cells in K/BxN autoimmune arthritis. *Arthritis Res Ther* (2017) 19(1): 188. doi: 10. 1186/s13075-017-1398-6

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28810929/) | [CrossRef Full Text](https://doi.org/10.1186/s13075-017-1398-6) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=F+Teng&author=KM+Felix&author=CP+Bradley&author=D+Naskar&author=H+Ma&author=WA+Raslan&publication_year=2017&title=The impact of age and gut microbiota on Th17 and Tfh cells in K%2FBxN autoimmune arthritis&journal=Arthritis+Res+Ther&volume=19&pages=188)

52. Jubair WK, Hendrickson JD, Severs EL, Schulz HM, Adhikari S, Ir D, et al. Modulation of Inflammatory Arthritis in Mice by Gut Microbiota Through Mucosal Inflammation and Autoantibody Generation. *Arthritis Rheumatol* (2018) 70: 1220–33. doi: 10. 1002/art. 40490

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29534332/) | [CrossRef Full Text](https://doi.org/10.1002/art.40490) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=WK+Jubair&author=JD+Hendrickson&author=EL+Severs&author=HM+Schulz&author=S+Adhikari&author=D+Ir&publication_year=2018&title=Modulation of Inflammatory Arthritis in Mice by Gut Microbiota Through Mucosal Inflammation and Autoantibody Generation&journal=Arthritis+Rheumatol&volume=70&pages=1220-33)

53. Picchianti-Diamanti A, Panebianco C, Salemi S, Sorgi ML, Di Rosa R, Tropea A, et al. Analysis of Gut Microbiota in Rheumatoid Arthritis Patients, Disease-Related Dysbiosis and Modifications Induced by Etanercept. *Int J Mol Sci* (2018) 19: 2938. doi: 10. 3390/ijms19102938

[CrossRef Full Text](https://doi.org/10.3390/ijms19102938) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=A+Picchianti-Diamanti&author=C+Panebianco&author=S+Salemi&author=ML+Sorgi&author=R+Di Rosa&author=A+Tropea&publication_year=2018&title=Analysis of Gut Microbiota in Rheumatoid Arthritis Patients%2C Disease-Related Dysbiosis and Modifications Induced by Etanercept&journal=Int+J+Mol+Sci&volume=19&pages=2938)

54. Calderaro DC, Ferreira GA, de Mendonça SMS, Corrêa JD, Santos FX, Sanção JGC, et al. Há associação entre o lúpus eritematoso sistêmico e a doença periodontal? *Rev Bras Reumatol* (2016) 56: 280–4. doi: 10. 1016/j. rbr. 2015. 03. 011

[CrossRef Full Text](https://doi.org/10.1016/j.rbr.2015.03.011) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=DC+Calderaro&author=GA+Ferreira&author=SMS+de Mendonça&author=JD+Corrêa&author=FX+Santos&author=JGC+Sanção&publication_year=2016&title=Há associação entre o lúpus eritematoso sistêmico e a doença periodontal&journal=Rev+Bras+Reumatol&volume=56&pages=280-4)

55. Corrêa JD, Calderaro DC, Ferreira GA, Mendonça SM, Fernandes GR, Xiao E, et al. Subgingival microbiota dysbiosis in systemic lupus erythematosus, Association with periodontal status. *Microbiome* (2017) 5: 34.

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28320468/) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=JD+Corrêa&author=DC+Calderaro&author=GA+Ferreira&author=SM+Mendonça&author=GR+Fernandes&author=E+Xiao&publication_year=2017&title=Subgingival microbiota dysbiosis in systemic lupus erythematosus%2C Association with periodontal status&journal=Microbiome&volume=5&pages=34)

56. Ni J, Wu GD, Albenberg L, Tomov VT. Gut microbiota and IBD, Causation or correlation? *Nat Rev Gastroenterol Hepatol* (2017) 14: 573–84. doi: 10. 1038/nrgastro. 2017. 88

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28743984/) | [CrossRef Full Text](https://doi.org/10.1038/nrgastro.2017.88) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=J+Ni&author=GD+Wu&author=L+Albenberg&author=VT+Tomov&publication_year=2017&title=Gut microbiota and IBD%2C Causation or correlation&journal=Nat+Rev+Gastroenterol+Hepatol&volume=14&pages=573-84)

57. Zi Y, Ni Z, Chunyan W, Xinyuan Z, Qingfeng W, Xinyue H, et al. A metagenomic study of the gut microbiome in Behcet’s disease. *Microbiome* (2018) 6: 135.

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30077182/) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=Y+Zi&author=Z+Ni&author=W+Chunyan&author=Z+Xinyuan&author=W+Qingfeng&author=H+Xinyue&publication_year=2018&title=A metagenomic study of the gut microbiome in Behcet’s disease&journal=Microbiome&volume=6&pages=135)

58. Ganju P, Nagpal S, Mohammed MH, Nishal Kumar P, Pandey R, Natarajan VT, et al. Microbial community profiling shows dysbiosis in the lesional skin of Vitiligo subjects. *Sci Rep* (2016) 13(6): 18761. doi: 10. 1038/srep18761

[CrossRef Full Text](https://doi.org/10.1038/srep18761) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=P+Ganju&author=S+Nagpal&author=MH+Mohammed&author=P+Nishal Kumar&author=R+Pandey&author=VT+Natarajan&publication_year=2016&title=Microbial community profiling shows dysbiosis in the lesional skin of Vitiligo subjects&journal=Sci+Rep&volume=13&pages=18761)

59. Kim MH, Choi SJ, Choi HI, Choi JP, Park HK, Kim EK, et al. Lactobacillus plantarum-derived Extracellular Vesicles Protect Atopic Dermatitis Induced by Staphylococcus aureus-derived Extracellular Vesicles. *Allergy Asthma Immunol Res* (2018) 10: 516–32. doi: 10. 4168/aair. 2018. 10. 5. 516

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30088371/) | [CrossRef Full Text](https://doi.org/10.4168/aair.2018.10.5.516) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=MH+Kim&author=SJ+Choi&author=HI+Choi&author=JP+Choi&author=HK+Park&author=EK+Kim&publication_year=2018&title=Lactobacillus plantarum-derived Extracellular Vesicles Protect Atopic Dermatitis Induced by Staphylococcus aureus-derived Extracellular Vesicles&journal=Allergy+Asthma+Immunol+Res&volume=10&pages=516-32)

60. Suzuki S, Campos-Alberto E, Morita Y, Yamaguchi M, Toshimitsu T, Kimura K, et al. Low Interleukin 10 Production at Birth Is a Risk Factor for Atopic Dermatitis in Neonates with Bifidobacterium Colonization. *Int Arch Allergy Immunol* (2018) 11: 1–8. doi: 10. 1159/000492130

[CrossRef Full Text](https://doi.org/10.1159/000492130) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+Suzuki&author=E+Campos-Alberto&author=Y+Morita&author=M+Yamaguchi&author=T+Toshimitsu&author=K+Kimura&publication_year=2018&title=Low Interleukin 10 Production at Birth Is a Risk Factor for Atopic Dermatitis in Neonates with Bifidobacterium Colonization&journal=Int+Arch+Allergy+Immunol&volume=11&pages=1)

61. Chang HW, Yan D, Singh R, Liu J, Lu X, Ucmak D, et al. Alteration of the cutaneous microbiome in psoriasis and potential role in Th17 polarization. *Microbiome* (2018) 6: 154. doi: 10. 1186/s40168-018-0533-1

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30185226/) | [CrossRef Full Text](https://doi.org/10.1186/s40168-018-0533-1) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=HW+Chang&author=D+Yan&author=R+Singh&author=J+Liu&author=X+Lu&author=D+Ucmak&publication_year=2018&title=Alteration of the cutaneous microbiome in psoriasis and potential role in Th17 polarization&journal=Microbiome&volume=6&pages=154)

62. Berer K, Gerdes LA, Cekanaviciute E, Jia X, Xiao L, Xia Z, et al. Gut microbiota from multiple sclerosis patients enables spontaneous autoimmune encephalomyelitis in mice. *Proc Natl Acad Sci USA* (2017) 114: 10719–24. doi: 10. 1073/pnas. 1711233114

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28893994/) | [CrossRef Full Text](https://doi.org/10.1073/pnas.1711233114) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=K+Berer&author=LA+Gerdes&author=E+Cekanaviciute&author=X+Jia&author=L+Xiao&author=Z+Xia&publication_year=2017&title=Gut microbiota from multiple sclerosis patients enables spontaneous autoimmune encephalomyelitis in mice&journal=Proc+Natl+Acad+Sci+USA&volume=114&pages=10719-24)

63. Cekanaviciute E, Yoo BB, Runia TF, Debelius JW, Singh S, Nelson CA, et al. Gut bacteria from multiple sclerosis patients modulate human T cells and exacerbate symptoms in mouse models. *Proc Natl Acad Sci USA* (2017) 114: 10713–8. doi: 10. 1073/pnas. 1711235114

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28893978/) | [CrossRef Full Text](https://doi.org/10.1073/pnas.1711235114) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=E+Cekanaviciute&author=BB+Yoo&author=TF+Runia&author=JW+Debelius&author=S+Singh&author=CA+Nelson&publication_year=2017&title=Gut bacteria from multiple sclerosis patients modulate human T cells and exacerbate symptoms in mouse models&journal=Proc+Natl+Acad+Sci+USA&volume=114&pages=10713-8)

64. Hall AB, Tolonen AC, Xavier RJ. Human genetic variation and the gut microbiome in disease. *Nat Rev Genet* (2017) 18(11): 690–9. doi: 10. 1038/nrg. 2017. 63

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28824167/) | [CrossRef Full Text](https://doi.org/10.1038/nrg.2017.63) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=AB+Hall&author=AC+Tolonen&author=RJ+Xavier&publication_year=2017&title=Human genetic variation and the gut microbiome in disease&journal=Nat+Rev+Genet&volume=18&pages=690-9)

65. Vatanen T, Kostic AD, d’Hennezel E, Siljander H, Franzosa EA, Yassour M, et al. Variation in Microbiome LPS Immunogenicity Contributes to Autoimmunity in Humans. *Cell* (2016) 165(6): 1551. doi: 10. 1016/j. cell. 2016. 05. 056

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27259157/) | [CrossRef Full Text](https://doi.org/10.1016/j.cell.2016.05.056) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=T+Vatanen&author=AD+Kostic&author=E+d’Hennezel&author=H+Siljander&author=EA+Franzosa&author=M+Yassour&publication_year=2016&title=Variation in Microbiome LPS Immunogenicity Contributes to Autoimmunity in Humans&journal=Cell&volume=165&pages=1551)

66. Iddah MA, Macharia BN. Autoimmune thyroid disorders. *ISRN Endocrinol* (2013) 26: 509764. doi: 10. 1155/2013/509764

[CrossRef Full Text](https://doi.org/10.1155/2013/509764) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=MA+Iddah&author=BN+Macharia&publication_year=2013&title=Autoimmune thyroid disorders&journal=ISRN+Endocrinol&volume=26&pages=509764)

67. Marfella R, Ferraraccio F, Rizzo MR, Portoghese M, Barbieri M, Basilio C, et al. Innate immune activity in plaque of patients with untreated and L-thyroxine-treated subclinical hypothyroidism. *J Clin Endocrinol Metab* (2011) 96(4): 1015–20. doi: 10. 1210/jc. 2010-1382

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/21239513/) | [CrossRef Full Text](https://doi.org/10.1210/jc.2010-1382) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=R+Marfella&author=F+Ferraraccio&author=MR+Rizzo&author=M+Portoghese&author=M+Barbieri&author=C+Basilio&publication_year=2011&title=Innate immune activity in plaque of patients with untreated and L-thyroxine-treated subclinical hypothyroidism&journal=J+Clin+Endocrinol+Metab&volume=96&pages=1015-20)

68. Masetti G, Moshkelgosha S, Köhling HL, Covelli D, Banga JP, Berchner-Pfannschmidt U, et al. Gut microbiota in experimental murine model of Graves’ orbitopathy established in different environments may modulate clinical presentation of disease. *Microbiome* (2018) 6(1): 97. doi: 10. 1186/s40168-018-0478-4

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29801507/) | [CrossRef Full Text](https://doi.org/10.1186/s40168-018-0478-4) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=G+Masetti&author=S+Moshkelgosha&author=HL+Köhling&author=D+Covelli&author=JP+Banga&author=U+Berchner-Pfannschmidt&publication_year=2018&title=Gut microbiota in experimental murine model of Graves’ orbitopathy established in different environments may modulate clinical presentation of disease&journal=Microbiome&volume=6&pages=97)

69. Penhale WJ, Young PR. The influence of the normal microbial flora on the susceptibility of rats to experimental autoimmune thyroiditis. *Clin Exp Immunol* (1988) 72(2): 288–92.

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/2970354/) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=WJ+Penhale&author=PR+Young&publication_year=1988&title=The influence of the normal microbial flora on the susceptibility of rats to experimental autoimmune thyroiditis&journal=Clin+Exp+Immunol&volume=72&pages=288-92)

70. Köhling HL, Plummer SF, Marchesi JR, Davidge KS, Ludgate M. The microbiota and autoimmunity, Their role in thyroid autoimmune diseases. *Clin Immunol* (2017) 183: 63–74. doi: 10. 1016/j. clim. 2017. 07. 001

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28689782/) | [CrossRef Full Text](https://doi.org/10.1016/j.clim.2017.07.001) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=HL+Köhling&author=SF+Plummer&author=JR+Marchesi&author=KS+Davidge&author=M+Ludgate&publication_year=2017&title=The microbiota and autoimmunity%2C Their role in thyroid autoimmune diseases&journal=Clin+Immunol&volume=183&pages=63)

71. Ishaq HM, Mohammad IS, Guo H, Shahzad M, Hou YJ, Ma C, et al. Molecular estimation of alteration in intestinal microbial composition in Hashimoto’s thyroiditis patients. *BioMed Pharmacother* (2017) 95: 865–74. doi: 10. 1016/j. biopha. 2017. 08. 101

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28903182/) | [CrossRef Full Text](https://doi.org/10.1016/j.biopha.2017.08.101) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=HM+Ishaq&author=IS+Mohammad&author=H+Guo&author=M+Shahzad&author=YJ+Hou&author=C+Ma&publication_year=2017&title=Molecular estimation of alteration in intestinal microbial composition in Hashimoto’s thyroiditis patients&journal=BioMed+Pharmacother&volume=95&pages=865-74)

72. Zhao F, Feng J, Li J, Zhao L, Liu Y, Chen H, et al. Alterations of the Gut Microbiota in Hashimoto’s Thyroiditis Patients. *Thyroid* (2018) 28(2): 175–86. doi: 10. 1089/thy. 2017. 0395

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/29320965/) | [CrossRef Full Text](https://doi.org/10.1089/thy.2017.0395) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=F+Zhao&author=J+Feng&author=J+Li&author=L+Zhao&author=Y+Liu&author=H+Chen&publication_year=2018&title=Alterations of the Gut Microbiota in Hashimoto’s Thyroiditis Patients&journal=Thyroid&volume=28&pages=175-86)

73. Peirce C, Ippolito S, Lanas A, Pesce M, Pontieri G, Arpaia D, et al. Treatment of refractory and severe hypothyroidism with sublingual levothyroxine in liquid formulation. *Endocrine* (2018) 60(1): 193–6. doi: 10. 1007/s12020-017-1367-5

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/28702889/) | [CrossRef Full Text](https://doi.org/10.1007/s12020-017-1367-5) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=C+Peirce&author=S+Ippolito&author=A+Lanas&author=M+Pesce&author=G+Pontieri&author=D+Arpaia&publication_year=2018&title=Treatment of refractory and severe hypothyroidism with sublingual levothyroxine in liquid formulation&journal=Endocrine&volume=60&pages=193-6)

74. Virili C, Antonelli A, Santaguida MG, Benvenga S, Centanni M. Gastrointestinal Malabsorption of Thyroxine. *Endocr Rev* (2019) 40(1): 118–36. doi: 10. 1210/er. 2018-00168

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30476027/) | [CrossRef Full Text](https://doi.org/10.1210/er.2018-00168) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=C+Virili&author=A+Antonelli&author=MG+Santaguida&author=S+Benvenga&author=M+Centanni&publication_year=2019&title=Gastrointestinal Malabsorption of Thyroxine&journal=Endocr+Rev&volume=40&pages=118-36)

75. De Leo S, Lee SY, Braverman LE. Hyperthyroidism. *Lancet* (2016) 388(10047): 906–18. doi: 10. 1016/S0140-6736(16)00278-6

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/27038492/) | [CrossRef Full Text](https://doi.org/10.1016/S0140-6736(16)00278-6) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+De Leo&author=SY+Lee&author=LE+Braverman&publication_year=2016&title=Hyperthyroidism&journal=Lancet&volume=388&pages=906-18)

76. Salvi M, Colucci G, Masetti G, Covelli D, Muller I, Koehling H, et al. The randomised probiotic trial of indigo study (investigation of novel biomarkers and definition of role of the microbiome in Graves’ orbitopathy). *Endocr Abstracts* (2019) 63: GP71. doi: 10. 1530/endoabs. 63. GP71

[CrossRef Full Text](https://doi.org/10.1530/endoabs.63.GP71) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=M+Salvi&author=G+Colucci&author=G+Masetti&author=D+Covelli&author=I+Muller&author=H+Koehling&publication_year=2019&title=The randomised probiotic trial of indigo study (investigation of novel biomarkers and definition of role of the microbiome in Graves’ orbitopathy)&journal=Endocr+Abstracts&volume=63&)

77. Zhang YJ, Li S, Gan RY, Zhou T, Xu DP, Li HB. Impacts of gut bacteria on human health and diseases. *Int J Mol Sci* (2015) 16: 7493–519. doi: 10. 3390/ijms16047493

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/25849657/) | [CrossRef Full Text](https://doi.org/10.3390/ijms16047493) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=YJ+Zhang&author=S+Li&author=RY+Gan&author=T+Zhou&author=DP+Xu&author=HB+Li&publication_year=2015&title=Impacts of gut bacteria on human health and diseases&journal=Int+J+Mol+Sci&volume=16&pages=7493-519)

78. Vivarelli S, Salemi R, Candido S, Falzone L, Santagati M, Stefani S, et al. Gut Microbiota and Cancer, From Pathogenesis to Therapy. *Cancers* (2019) 11(1): 38. doi: 10. 3390/cancers11010038

[CrossRef Full Text](https://doi.org/10.3390/cancers11010038) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=S+Vivarelli&author=R+Salemi&author=S+Candido&author=L+Falzone&author=M+Santagati&author=S+Stefani&publication_year=2019&title=Gut Microbiota and Cancer%2C From Pathogenesis to Therapy&journal=Cancers&volume=11&pages=38)

79. Feng J, Zhao F, Sun J, Lin B, Zhao L, Liu Y, et al. Alterations in the gut microbiota and metabolite profiles of thyroid carcinoma patients. *Int J Cancer* (2019) 144(11): 2728–45. doi: 10. 1002/ijc. 32007

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30565661/) | [CrossRef Full Text](https://doi.org/10.1002/ijc.32007) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=J+Feng&author=F+Zhao&author=J+Sun&author=B+Lin&author=L+Zhao&author=Y+Liu&publication_year=2019&title=Alterations in the gut microbiota and metabolite profiles of thyroid carcinoma patients&journal=Int+J+Cancer&volume=144&pages=2728-45)

80. Zhang J, Fanghua Z, Zhang F, Zhao C, Xu Q, Liang C, et al. Dysbiosis of the gut microbiome is associated with thyroid cancer and thyroid nodules and correlated with clinical index of thyroid function. *Endocrine* (2019) 64: 564–74. doi: 10. 1007/s12020-018-1831-x

[PubMed Abstract](https://pubmed.ncbi.nlm.nih.gov/30584647/) | [CrossRef Full Text](https://doi.org/10.1007/s12020-018-1831-x) | [Google Scholar](http://scholar.google.com/scholar_lookup?author=J+Zhang&author=Z+Fanghua&author=F+Zhang&author=C+Zhao&author=Q+Xu&author=C+Liang&publication_year=2019&title=Dysbiosis of the gut microbiome is associated with thyroid cancer and thyroid nodules and correlated with clinical index of thyroid function&journal=Endocrine&volume=64&pages=564-74)