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Review Article

Autoimmune Diseases and their Correlation with the Intestinal Microbiome and Fecal Microbiota Transplantation

Álvaro Zamudio Tiburcio[1]*, Héctor Bermúdez Ruiz[2], Silverio Alonso López[3] and Pedro Antonio Reyes López[4]

[1]*Department of Gastroenterology, Intestinal Microbiota Transplantation, Medical Specialties, Naples Unit, Mexico*

[2]*Endoscopy Service, Oncology Hospital, National Medical Center, XXI Century, Mexican Social Security Institute, Mexico*

[3]*Department of Urologist, Chairman Medical Specialties Naples in Mexico City, Mexico*

[4]*Immunologist, Rheumatologist, National Institute of Cardiology "I. Chávez" Mexico City, Mexico*

***Corresponding Author:** Álvaro Zamudio Tiburcio, Department of Gastroenterology, Intestinal Microbiota Transplantation, Medical Specialties, Naples Unit, Mexico.

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Abstract

Autoimmune diseases are complex and diverse processes that arise from a lack of recognition by the immune system. They affect cytokines, immune cells, and protein kinases, generating monoclonal antibodies and small molecule inhibitors targeting these molecules. The purpose of this review is to analyze the impact of the Gut Microbiome (GI) on different autoimmune diseases and to leverage it in the management of these complex processes. A significant number of these processes are analyzed, but not all, since they total approximately more than 80.

Keywords: *Autoimmune Diseases (AD); Gut Microbiome (GM); Short-Chain Fatty Acids (SCFA); Intestinal Dysbiosis (ID); Fecal Microbiota Transplant (FMT)*

Introduction

We find similarities in the various autoimmune diseases (ADs), which are often numerous, and this similarity can be determined through the antigen-associated immune complex profile. This analysis can determine patterns of antigen-associated immune complex profiles [1]. We begin the analysis with the most common conditions and conclude with rare disorders, adding part of the diagnosis and treatment of these processes. The symptoms and treatments are usually as follows.

Most common symptoms [2]:

- Difficulty concentrating
- Muscle pain
- Numbness or tingling in the hands or feet
- Skin rashes
- Fatigue
- Swelling and redness

- Hair loss
- Low temperature.

Most common treatments [3]:

- Monoclonal antibodies
- Biological therapies
- Non-steroid anti-inflammatory drugs (NSAID)
- Immunosuppressant drugs
- Relieving symptoms such as pain, swelling, fatigue, and skin rashes
- A well-balanced diet and regular exercise.

Most common AD [4-7]:

- **Rheumatoid arthritis (RA):** The immune system attacks the joints, causing redness, warmth, pain, and stiffness.
- **Inflammatory bowel disease (IBD):** A term used to describe autoimmune conditions that cause inflammation in the lining of the intestinal wall.
- **Crohn's disease (CD):** It can inflame any part of the digestive tract. Common symptoms include frequent urination, up to more than 10 times during the acute phase of the disease.
- **Ulcerative colitis (UC):** Another type of autoimmune bowel disease that affects the large intestine, with chronic inflammation. Its symptoms include fatigue, fever, bleeding during bowel movements, and rectal pain.
- **Celiac disease:** Celiac Sprout or Gluten-Sensitive Enteropathy. People with celiac disease cannot eat foods containing gluten, a protein found in wheat, rye, and grains. When gluten is in the small intestine, the immune system attacks this part of the gastrointestinal tract and causes inflammation. Gluten sensitivity is not an autoimmune disease, but it can have similar symptoms such as diarrhea and abdominal pain.
- **Psoriasis/Psoriatic arthritis:** Skin cells normally grow and then shed when they are no longer needed. Psoriasis causes skin cells to multiply too rapidly. They accumulate and form inflamed red patches, commonly with silvery-white plaque-like scales on the skin. Up to 30 percent of people with psoriasis also develop joint swelling, stiffness, and pain. This form of the disease is called psoriatic arthritis.
- **Multiple sclerosis (MS):** MS damages the myelin sheath, the protective layer that surrounds nerve cells, in the central nervous system. Damage to the sheath reduces the speed at which messages are transmitted between the brain and spinal cord to and from the rest of the body. This damage can cause numbness, weakness, balance problems, and difficulty walking.
- **Systemic lupus erythematosus:** A skin disease caused by the rash it often produces; the systemic form is the most common and affects joints, kidneys, brain, and heart.
- **Type 1 diabetes mellitus:** The immune system attacks and destroys the insulin-producing cells in the pancreas. The resulting high blood sugar levels can cause damage to the blood vessels, heart, kidneys, eyes, and nerves.
- **Addison's disease:** Affects the adrenal glands, which produce cortisol, aldosterone, and androgens. A lack of cortisol can affect how the body uses and stores carbohydrates and sugar. Aldosterone deficiency causes sodium loss and excess potassium in the bloodstream.

- **Graves' disease:** Disrupts the thyroid gland, causing excessive hormone production. Excesses of these hormones accelerate the body's activities, causing nervousness, rapid heartbeat, heat intolerance, and weight loss. Bulging eyes (exophthalmos) are a possible sign of this disease. It can occur as part of what is called Graves' ophthalmopathy, which affects about 30 percent of people with Graves' disease.
- **Sjogren's syndrome:** This condition affects the glands that lubricate the eyes and mouth. The characteristic symptoms of Sjogren's syndrome are dry eyes and mouth. It can affect the joints or skin.
- **Hashimoto's thyroiditis:** Thyroid hormone production is reduced to the point of deficiency. Symptoms include weight gain, sensitivity to cold, fatigue, hair loss, and thyroid inflammation.
- **Myasthenia gravis:** This condition affects the nerve impulses that help the brain control muscles. When communication from nerves to muscles is impaired, muscles fail to contract due to ineffective signals. The most common symptom is muscle weakness that worsens with activity and improves with rest.
- **Autoimmune vasculitis (Behcet's vasculitis):** This condition affects the blood vessels, causing inflammation known as systemic vasculitis. The mouth and genitals are the most affected areas. Painful, deep ulcers with a white or yellow base and a red halo appear.
- **Pernicious anemia:** There is a deficiency of intrinsic factor, which is necessary for the small intestine to absorb vitamin B-12 from food. When this vitamin is deficient, anemia develops and the body's ability to synthesize DNA properly is impaired.

Some other autoimmune diseases

Many of them are classified as rare diseases, and the WHO states: A disease should be classified as "rare" or "uncommon" if it affects at least 5 in every 10,000 inhabitants (0.05%). However, most of these diseases are even less common. We summarize a good number of them [8]:

- **Sarcoidosis:** A systemic granulomatous disease of unknown etiology. It can affect patients of all latitudes and ages, being most common between the third and fourth decades of life with a second peak around age 50 in Scandinavian and Japanese populations.
- **Youth idiopathic arthritis:** A rheumatic disease that affects children under 16 years of age. It is characterized by persistent joint inflammation, which can last more than six weeks.
- **Scleroderma:** Also known as systemic sclerosis, this is a group of rare diseases that involve hardening and tightening of the skin. Scleroderma can also cause problems with blood vessels, internal organs, and the digestive tract.
- **Polymyositis (PM) and dermatomyositis (DM):** Inflammatory myopathies. Muscle diseases caused by the body's immune response. They cause muscle weakness as the main symptom; dermatomyositis also causes a rash.
- **Inclusion body myopathy (IBM):** One of the four major inflammatory myopathies, along with dermatomyositis (DM), polymyositis (PM), and necrotizing myopathy.
- **Guillain-Barré syndrome:** A condition in which the body's immune system attacks the nerves. It can cause weakness, numbness, or paralysis. The first symptoms are usually weakness and tingling in the hands and feet. These sensations can spread rapidly and often lead to paralysis.
- **Isolated agammaglobulinemia:** A genetic autoimmune disorder that affects antibody production, resulting in immunoglobulin deficiency. This disorder is passed from parents to children and is caused by a genetic abnormality that blocks the growth of mature, normal immune system cells (B lymphocytes).
- **IGG4 disease:** Its etiology is unknown; advances have been made in understanding its pathophysiological and immunological bases, as well as the role of inflammatory cells in the development of target organ. There is no international consensus on its diagnosis.
- **Severe combined immunodeficiency:** An autoimmune disease that involves the immune system, causing failure of the humoral and tumor responses. It is one of the most serious primary immunodeficiency conditions.

- **Common variable immunodeficiency:** An autoimmune disease with a low antibody concentration, which generates susceptibility to recurrent infections, autoimmune diseases, and neoplasia.
- **Ankylosing spondylitis:** Also known as Bechterew's disease. A type of arthritis focused at the end of the spine. It causes joint inflammation and is characterized by an immune system attack on the body's healthy tissues. It alternates periods of inactivity with flare-ups of inflammation and pain.
- **Reiter's syndrome:** It causes urinary tract infections and joint inflammation, affecting mobility. It causes frequent uveitis and conjunctivitis.
- **Ulcerative autoimmune hepatitis:** The organ of this condition is the liver, causing inflammation and cell death. A chronic degenerative disease with latency periods. Symptoms include fever and jaundice, as well as nausea and abdominal swelling.
- **Graves-Basedow's disease:** An autoimmune thyroiditis of undefined etiology; it commonly causes thyrotoxicosis and is characterized by diffuse thyroid hyperplasia, which progresses to goiter and hyperthyroidism.
- **Kawasaki disease (KD):** Acute, self-limiting systemic vasculitis with potentially dangerous complications, primarily affecting infants and young children.
- **Retroperitoneal fibrosis:** A rare disorder in which the ureters become blocked.
- **Paroxysmal nocturnal hemoglobinuria:** A rare disorder characterized by intravascular hemolysis with hemoglobinuria, leukopenia, thrombocytopenia, arterial and venous thrombosis, and common crises.
- **Poems syndrome:** This is rare. The blood damages the nerves, affecting other areas of the body.
- **Ocular thyroid ocular:** Also known as Graves' eye disease. It occurs when the immune system is overactive. It can damage the thyroid, eyes, and other parts of the body.
- **Eosinophilic granulomatosis with polyangiitis (Wegener's disease):** Systemic and necrotizing vasculitis of small and medium-sized vessels, characterized by extravascular granulomas, eosinophilia, and tissue infiltration by eosinophils.
- **Mixed connective tissue disease:** A rare disorder that combines signs and symptoms of systemic lupus erythematosus, scleroderma, and polymyositis.
- **Undifferentiated connective tissue disease (UCTD):** A systemic autoimmune disease. Although it shares characteristics with other diseases, such as lupus, scleroderma, and rheumatoid arthritis, it does not meet the established diagnostic criteria for any of them.
- **Antiphospholipid syndrome:** An autoimmune disorder in which the immune system produces antibodies that can cause blood clots in arteries and veins. This syndrome is characterized by persistently elevated antiphospholipid antibodies, which is associated with frequent thrombosis and pregnancy-related morbidity.
- **Cogan syndrome:** A rare autoimmune syndrome affecting the ocular and vestibuloauditory systems. Fatal aortitis sometimes occurs. If left untreated, permanent vision and hearing loss can occur.
- **Giant cell arteritis (Temporal arteritis):** Inflammation of the arteries, especially those in the head, is often referred to as temporal arteritis. It affects large and medium-sized arteries and can have significant consequences if not diagnosed and treated promptly. It is considered a medical emergency, and early diagnosis can significantly improve the patient's prognosis.
- **Panarteritis nodosa:** Necrotizing vasculitis that affects medium- and small-caliber arteries, causing inflammation and damage to the blood vessels. This disease usually occurs in middle-aged or older people and can lead to serious complications due to arterial inflammation.
- **Takayasu arteritis:** A rare type of vasculitis that causes inflammation of the blood vessels, especially the aorta and its main branches. It occurs most frequently in young women and can cause symptoms such as claudication, visual disturbances, fainting, or angina.

- **Microscopic polyangiitis (MPA):** A rare small-vessel vasculitis. Manifestations are variable and may include alveolar hemorrhage, multiple mononeuropathy, and glomerulonephritis.
- **Essential mixed cryoglobulinemia:** A disease characterized by the presence of abnormal proteins in the blood that can cause inflammation and tissue damage. It is often associated with chronic infections, especially hepatitis C, and may be linked to autoimmune diseases. Symptoms can include skin rashes, joint pain, and fatigue.
- **Henoch-Schönlein purpura (IGA vasculitis):** Autoimmune disorder characterized by inflammation and bleeding in small blood vessels, primarily affecting the skin, joints, intestines, and kidneys.
- **Leukocytoclastic vasculitis:** Immune-mediated process that affects small blood vessels, primarily in the skin, causing inflammation and necrosis. Common symptoms include palpable purpura and other dermatological manifestations.
- **Immune-mediated necrotizing myopathy:** A subtype of inflammatory myopathy characterized by: Symmetrical proximal muscle weakness that may be acute or subacute. Elevated creatine kinase levels and muscle fiber necrosis with a sparse inflammatory infiltrate on biopsy. Specific antibodies. Anti-HMGCR or anti-SRP, may be present.
- **SUSAC syndrome:** It consists of the clinical triad of encephalopathy, branch retinal artery occlusion, and hearing loss.
- **Vogt-Koyanagi-Harada disease (Uveo-meningeal syndrome):** A multisystem autoimmune disorder that primarily affects the eyes, ears, and central nervous system.

Gut microbiome in autoimmune diseases

The gut microbiome (GM) describes the microbial ecosystem that coexists within an organism. It comprises a diverse community of microorganisms, including bacteria, fungi, viruses, archaea, and others, residing in the digestive tract. It has numerous beneficial functions for health and impacts immunological aspects, and is influenced by genetic factors [9]. The microbiome has been implicated in a long list of immune-mediated diseases. The mechanisms that account for this effect are multiple. The clinical implications of observations about the microbiome and disease are broad. Considering that the GM is defined as the sum of all microbial entities, as well as their genes, proteins, and metabolic products in a given space and time, its impact on the most common AD is analyzed below, with the aim of serving as a basis for future proposed therapies. Rheumatoid arthritis (RA) is a prevalent systemic autoimmune disease caused by a combination of genetic and environmental factors. Animal models suggest a role for gut bacteria in supporting the systemic immune response required for joint inflammation. 16S sequencing of stool samples from rheumatoid arthritis patients and controls, and shotgun sequencing, identified *Prevotella copri* as strongly correlated with disease in patients with untreated, new-onset rheumatoid arthritis. Increases in *Prevotella* abundance correlated with reductions in *Bacteroides* and loss of putatively beneficial microbes. Unique *Prevotella* genes have also been identified as correlating with disease. Furthermore, colonization of mice revealed the ability of *P. copri* to dominate GM, resulting in increased susceptibility to chemically induced colitis [10].

Inflammatory bowel disease (IBD) arises due to genetic risk, environmental factors, and, above all, the effects of celiac disease. Evidence, conceived in 1990, supports a bidirectional relationship between disease development and celiac disease composition and function. An increase in *Proteobacteria* and a decrease in *Firmicutes* are also reported. On the other hand, celiac disease composition is frequently the cause of spontaneous colitis severity [11]. The pathogenesis of autoimmunity in celiac disease has been considered to be due to modifications in the celiac disease environment. However, the mechanisms that regulate its balance and pathogenesis have not been identified. In a series of newborns at familial risk for CD, who had a lower proportion of *Bacteroidetes* and a higher abundance of *Firmicutes*, the decrease in *Lactobacillus* preceded the appearance of positive CD antibodies. Of interest is the role of GM in its proteolytic activity on gluten, generating degraded peptides as toxic and immunogenic elements, which may represent an important event in its metabolism [12]. The incidence of GM in psoriasis is significant, especially due to the dysbiosis that GM can create, as a determining factor in the disease. Patients with psoriasis are at increased risk of developing inflammatory bowel disease, which translates into a bidirectional

interaction between systemic inflammation. Therefore, GM restoration is of vital importance, both preventively and therapeutically. For four decades, it has been demonstrated that skin microorganisms are involved in the development of non-infectious skin conditions, such as psoriasis. NGS and bioinformatics have provided insight into the genetic functions of GM and its role in pathogenesis, manifestation, and prognosis [13].

MS and GM: The GM can be altered in multiple sclerosis, producing effector and regulatory phases of CNS demyelination. There is growing evidence from preliminary human studies suggesting that dysbiotic GM in MS could affect disease progression. Therefore, it has been proposed to consider the GM as a key organ in tolerance mechanisms. Intervention could result in new therapeutics for MS [14]. Ochoa-Repáraz J and his group [15] determined that there is link between MS and GM, and that recent experimental and clinical evidence suggests the presence of microbial imbalances in the gut of MS patients. Systemic lupus erythematosus and how it is impacted by GM. Both processes are linked, with GM influencing disease progression. Various health and disease parameters have been found to be associated with variations in the human gut microbiome. Although the disease mechanism involves genetic and environmental factors, lupus has been found to be affected by the composition of the microorganisms lining the intestines.

Elucidating the role of GM in SLE will shed light on how this autoimmune disorder develops and provide opportunities to improve disease biomarkers and the potential to investigate new therapies. Lupus patients present decreased GM in the intestinal lumen. They also frequently present dysbiosis, which translates to changes in intestinal permeability, with an increase in *Ruminococcus gnavus* [16].

Type 1 diabetes mellitus and GM: As in many AD, a strong link to GM is observed in type 1 diabetes mellitus, especially in the pathophysiological aspects. Dysbiosis often appears as the disease progresses. The disease destroys the insulin-producing pancreatic beta cells in the islets of Langerhans. Its incidence has tripled in recent decades, while its pathophysiology remains largely unknown. The only treatment consists of regulating blood glucose levels through insulin injections and intensive monitoring of blood glucose levels. Intestinal dysbiosis is likely a contributing factor to the disease. It has been noted that changes in the GM may occur before the onset of the disease, exacerbating the process with the use of antibiotics. The GM may be a treatment factor [17].

Incidence of GM in Addison's disease: Zhang YY and colleagues [18] point out that the causal effects of GM in Addison's disease are unclear. They point out that the Deltaproteobacteria class, Desulfovibrionaceae family, and Desulfovibrionales order have protective effects against adrenocortical insufficiency. Meanwhile, the Porphyromonadaceae family, *Lachnoclostridium* genus, and MollicutesRF9 order are associated with an increased risk of adrenocortical insufficiency. The Methanobacteria class, Lactobacillaceae family, *Lactobacillus* genus, and Methanobacteriales order protect against hyperaldosteronism. Finally, the genera *Parasutterella*, *Peptococcus*, and *Veillonella* are microorganisms at risk for hyperaldosteronism. Graves' disease and its relationship with the gut microbiome. An autoimmune disorder that primarily affects the thyroid gland and causes hyperthyroidism. Research has shown a significant correlation between GD and changes in the gut microbiome. Patients have altered gut microbiota, with increased numbers of certain bacteria and lower diversity compared to healthy individuals. This suggests a possible relationship between GD and the gut microbiome, which could influence the development and progression of the disease. The thyroid-gut axis, which involves the interaction between the thyroid gland and the gut, is gaining attention as a new area of research. It is speculated that its imbalance could contribute to autoimmune dysfunction in GD [19].

Correlation of Sjögren's syndrome with the gut microbiome: An autoimmune process characterized by chronic pathology. The gut microbiome and its metabolites play a significant role in immune regulation through interactions with the host. The microbiome in multiple locations plays a significant role in the onset and progression of the disease. Its pathogenesis remains unclear and likely includes hormonal, infectious, and genetic factors, with the microbiome being the most prominent. Oral, intestinal, and ocular dysbiosis play a crucial role [20].

Hashimoto's thyroiditis: Hashimoto's thyroiditis is an autoimmune disease that affects the thyroid gland and may be related to the gut microbiome. The microbiome plays a crucial role in thyroid health and function, and its balance can influence thyroid hormone production and the inflammatory response. Dysbiosis may contribute to the development of Hashimoto's thyroiditis, especially in individuals with a genetic predisposition. It can also affect iodine absorption, with autoimmune responses affecting thyroid function [21].

Myasthenia Gravis and its correlation with the intestinal microbiota: A chronic autoimmune neuromuscular disorder related to MG, evidenced by bacterial overgrowth. There is alteration of the intestinal microbiota in patients with higher levels of Gram-negative bacteria, such as *Bacteroidetes*, and lower levels of other microorganisms, such as *Bifidobacteria* [22].

Autoimmune vasculitis (Behçet's vasculitis): Correlated with MG. Autoimmune vasculitis characterized by inflammation of the blood vessels and may be related to MG. Behçet's disease is a chronic autoimmune disease. It is defined by its tendency toward remission and typical geographic distribution. From a clinical perspective, the association of venous thrombosis with arterial aneurysms, inflammatory cerebral parenchymal involvement, the classic pattern of posterior uveitis with retinal vasculitis, and the well-known triad of bipolar ulcers and erythema nodosum are distinctive features of this entity [23].

Pernicious anemia and its correlation with the gut microbiome: Pernicious anemia is the most common cause of megaloblastic anemia and is a consequence of vitamin B12 deficiency, which is due in turn to the decrease or absence of intrinsic factor due to atrophy of the gastric mucosa or autoimmune destruction of the parietal cells that produce it. In the presence of severe gastric atrophy, a decrease in acid and IF production and a subsequent impairment in vitamin B12 absorption occur [24]. There is significant competition for iron by both GM and the hosts; The anemia and GM are crucial. Deficiency not only causes anemia, but also various symptoms, such as dyspnea and general weakness. Furthermore, iron deficiency affects gut microbial health. As seen in the analysis of the correlation between AE and GM, all are linked, to a greater or lesser extent, so this relationship must be explored in depth in order to use a new tool in immunological processes.

Intestinal dysbiosis

AD are characterized by microbiome dysbiosis. Microbiome dysbiosis can be influenced by host genetics and environmental factors. Dysbiosis is also associated with changes in certain functional pathways. There is growing evidence that GM dysbiosis is associated with the pathogenesis of intestinal and extraintestinal disorders. In many of these conditions, the mechanisms leading to disease development involve a fundamental relationship between the colonic microbiota, its metabolic products, and the host immune system. Establishing a "healthy" relationship early in life appears to be critical for maintaining intestinal homeostasis. While we still do not have a clear understanding of what constitutes a "healthy" GM, recent studies are emerging that identify particular bacterial species associated with a healthy GM [25]. Bacterial species residing within the colonic mucosal layer, either through direct contact with host cells or through indirect communication via bacterial metabolites, can influence whether host cellular homeostasis is maintained or inflammatory mechanisms are activated. There is evidence that perturbations in the GM are involved in the development of colorectal cancer. In this case, dysbiosis may not be the most important factor, but rather the products of the interaction between diet and the microbiome. High-protein diets are thought to produce carcinogenic metabolites from the colonic microbiome that can lead to the induction of neoplasia in the epithelium [26]. The prevalence of many chronic diseases has increased in recent decades. It has been postulated that dysbiosis driven by environmental factors such as antibiotic use is changing the microbiome in ways that increase inflammation and the onset of chronic disease. Dysbiosis can be defined by the loss or gain of bacteria that promote health or disease. *Bacteroides, Prevotella*, and *Ruminococcus* were the most common dysbiotic taxa in terms of enrichment or depletion in disease populations, due in part to the diversity of operational taxonomic units within these genera [27]. Many of these diseases, including COVID-19 infection, are associated with alterations in GM composition and function, i.e. dysbiosis.

Short-chain fatty acids (SCFAs)

Are products of bacterial fermentation and play a vital role in intestinal homeostasis. Propionate and acetate are produced primarily by *Bacteroidetes*, while butyrate is predominantly produced by *Firmicutes*. They can be used as a carbon source by intestinal epithelial cells and bind to G protein-coupled receptors (GPCR). Therefore, they contribute to mucosal healing, histone deacetylation, and gene expression. In addition, they have immunomodulatory activity, including Treg development, cytokine production, and anti-inflammatory effects. They also regulate the rest of the microbial population by modifying the ecological environment to select specific members of the microbial community. Microbial butyrate promotes epithelial barrier function, independent of IL-10, by repressing Claudin-2, a channel-forming tight junction protein that normally disrupts intestinal barrier function. It induces macrophage differentiation and AMP production [28].

Secondary bile acids and autoimmune diseases

Secondary bile acids play a significant role in the pathogenesis of autoimmune diseases, particularly in autoimmune uveitis and autoimmune liver diseases. They are synthesized in the intestine and participate in maintaining immune homeostasis. GM dysbiosis can lead to decreased bile acid concentrations, which can exacerbate autoimmune diseases. Their metabolism is linked to immune cell differentiation and function, and their dysregulation can affect the development of inflammatory bowel diseases. Since secondary bile acids not only play an essential role in nutrient absorption but also in the regulation of metabolic functions and immune response, they are increasingly recognized as potential therapeutic targets in the context of chronic liver diseases. Secondary bile acid receptors, such as the G protein-1 bile acid-activated receptor and the farnesoid X receptor, are expressed on various immune cells involved in innate immunity. Recently, several studies have revealed distinct roles for secondary bile acids and their receptors within the adaptive immune system. Furthermore, several molecules targeting secondary bile acid receptors and transporters are currently in advanced stages of clinical development [29].

Intestinal barrier and autoimmune diseases

The intestinal barrier plays a primary role in autoimmune diseases, as it regulates the immune response and maintains homeostasis. Its deterioration often leads to abnormal immune responses, with the development of AD [30]. Autoimmune diseases are diseases that affect multiple tissues and organs, characterized by the overproduction of multiple abnormal antibodies. Most studies support that immune imbalance contributes to the development of autoimmune diseases, although their specific pathogenesis is not fully understood. Intestinal immunity, especially the intestinal mucosal barrier, has become a key research topic, considered a prior mechanism leading to immune imbalance. As an important defense barrier, the intestinal mucosal barrier regulates and maintains the homeostasis of the internal environment. Once the intestinal barrier function is compromised by multiple factors, it destroys immune homeostasis, triggers inflammatory responses, and ultimately contributes to the development of autoimmune diseases. Some studies suggest that it maintains the balance of immune homeostasis through the zonulin, GM, and Troll-like receptor signaling pathways.

Comments

Both quantitative and qualitative differences in GM composition have been observed in patients with autoimmune disorders. Furthermore, disease activity and response to diet may also play a significant role. Evidence points to the expansion of *Proteobacteria* and opportunistic pathogens such as *Neisseria* and *Escherichia coli*, as well as higher bacterial virulent genes, supporting the idea of a shift toward a pro-inflammatory microbial community. Currently, it is still unclear whether the observed changes in microbial composition are the cause or the result of the intestinal inflammatory process [31]. In RA, evidence demonstrates that GM directly and indirectly modulates the host immune system. *Lactobacillus rhamnosus*, *casei, reuteri, acidophilus, Bacillus coagulans*, and *Bifidobacterium bifidum* have been studied for their ability to treat RA in randomized controlled trials. They have been shown to be safe and effective for patients suffering from RA [32].

In their excellent editorial [33], Li Y and his group mention the following: The GM influences host health, especially immune homeostasis. It plays a pivotal role in the formation and development of the main components of the innate and adaptive immune systems and regulates the balance of the host-microorganism relationship. Understanding the symbiotic relationship between immunity and the GM is necessary for immune discipline and for influencing the pathogenesis of intestinal diseases. The evaluation of clinical models and biomarkers of the GM and the immune system allows for predicting the prognosis of intestinal disorders. Mendelian randomization provides a viable approach to investigate the causal relationships between microbial factors and intestinal diseases.

FECALM microbiot transplantation: FMT can restore the GM and reconstruct immune system function, producing a therapeutic effect. It is an effective method for treating recurrent Clostridioides difficile infections. Furthermore, it is emerging as a promising treatment for patients with inflammatory bowel disease and other AD. FMT has been shown to restore altered GM composition, rebuild the intestinal microecosystem, and mediate innate and adaptive immune responses [34]. FMT is of growing interest and has been proposed as a potential strategy to intervene in GM homeostasis in the treatment of various diseases.

Among all the approaches targeting GM, FMT has attracted increasing interest and has been proposed as a possible strategy to intervene in GM homeostasis in the treatment of various diseases. However, despite the reported positive curative effects and the clinical studies conducted on FMT, the detailed mechanisms for effective treatment of these diseases have yet to be elucidated. These mechanisms include the promotion, modulation, activation, or inhibition of the host immune system through interactions between microorganisms and the intestinal immune system, the gut-brain axis, gut-liver axis, gut-kidney axis, etc. [35].

Conclusion

- The interrelationship between autoimmune diseases and the gut microbiome is a fact.
- Although the symptoms of AD differ slightly, they have numerous similarities.
- The treatment is very similar, with the addition of Fecal Microbiota Transplantation.
- The TMF is successful, although it is important to elucidate the mechanisms that generate success.

Conflicts of interest

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

Ethical Approval

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

Informed Consent

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

Declaration on the Use of Artificial Intelligence

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

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