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REVIEW

Role of Gut Microbiota in the Development of Some Autoimmune Diseases

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Abstract: The gut microbiota is crucial for maintaining the homeostasis and function of the immune system. It interacts with the host's immune system through various mechanisms, including promoting immune tolerance, affecting the differentiation and function of immune cells, and participating in the metabolism of immune regulatory substances. The disruption of the gut microbiome may lead to impaired mucosal barrier function, allowing bacteria and their metabolites to invade into the host, activate or interfere with the immune system, and potentially trigger or exacerbate autoimmune responses. Understanding the relationship between the microbiome and autoimmune diseases may help develop new treatment strategies. This article reviewed the recent progresses of microbiome involved in the occurrence and development of some autoimmune diseases and the treatment methods based on regulation of the microbiome, highlighted the key role of microbiome in autoimmune diseases.

Keywords: gut microbiota, autoimmune disease, systemic lupus erythematosus, type 1 diabetes, rheumatoid arthritis

Introduction

More than 10^{14} cells of bacteria, fungi, and viruses reside in the human gastrointestinal tract.[1] The gut microbiota comprises approximately 100 trillion bacterial cells, which is 10 times the total number of human cells. The most predominant bacterial species in the human gut include *Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Verrucomicrobia* and *Fusobacteria*, of which *Firmicutes* and *Bacteroidetes* were the dominant groups of bacteria.[2,3] The gut microbiota plays a crucial role in regulating many aspects of host physiology, including glucose and lipid metabolism, systemic immunity and the function of central nervous system.[4] Known as the "human second genome",[5] the gut microbiota co-evolves with the host in a symbiotic manner, and promotes the development of the immune system. The immune system has evolved not only to defend against pathogens but also to tolerate beneficial microbiota, which have evolved to coexist symbiotically with the host. Under immune homeostasis, the immune system tolerates commensal bacteria, but during homeostatic perturbations, it can respond to the microbiota and leads to pathological process. The interaction of the gut microbiota with the innate immune system is significant for maintaining homeostasis. Changes in the gut microbiota alter the metabolic environment, activate specific pattern recognition receptors in epithelial cells, and induce a pro-inflammatory state. The resulted epithelial barrier leakage leads to tissue micro-inflammation by triggering bacterial translocation to the interepithelial and subepithelial regions (Figure 1).[6,7] Dysbiosis of gut microbiota can be categorized into three types: the loss of beneficial organisms, the overgrowth of potentially harmful organisms, and the loss of overall microbial diversity. Moreover, these three types are not mutually exclusive and can occur simultaneously.[8] Dysbiosis of the gut microbiota has been proven to be the cause of various intestinal lesions and can also lead to systemic autoimmune diseases. However, the exact mechanisms by which the disturbed microbiota causes the diseases still need to be elucidated.

Autoimmune diseases occur when there is a breakdown of immune tolerance to self-components, resulting in the immune system attacking its own organs, tissues, or cells.[10] These diseases are divided into organ-specific and non-organ

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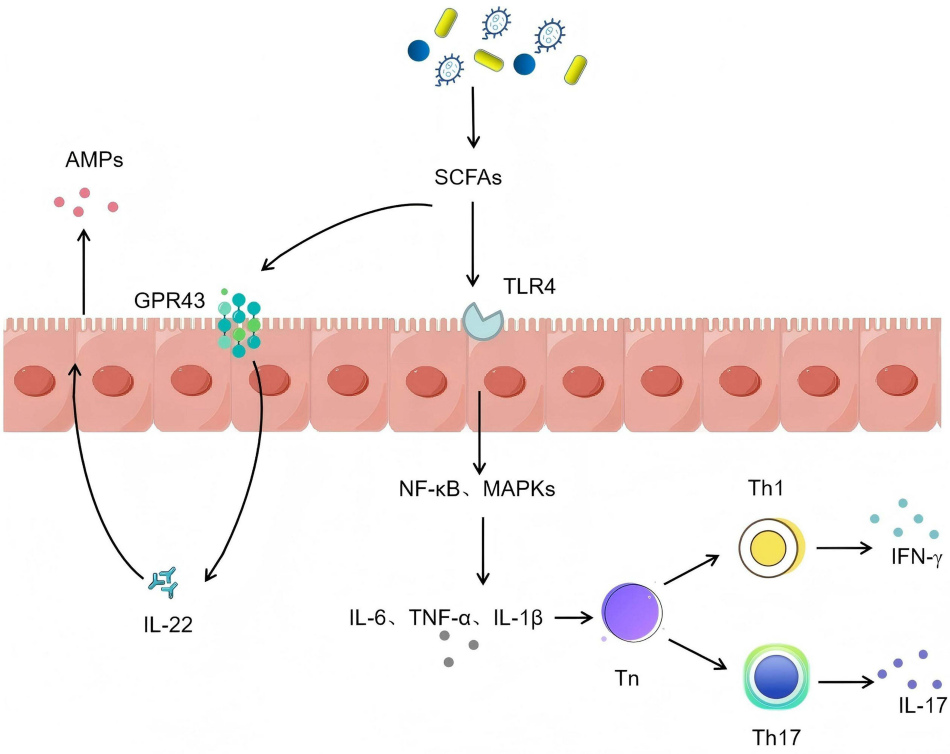

Figure 1 The gut microbiota plays a crucial role in modulating the immune response. When the gut flora is dysfunctional, short-chain fatty acids (SCFAs) produced by the microbes can enter the intestinal lumen. On the one hand, SCFAs can bind to G-protein-coupled receptor 43 (GPR43), leading to the production of interleukin-22 (IL-22) and stimulating the formation of antimicrobial peptides (AMPs). AMPs are a vital component of the innate immune defense system, serving to restrict pathogen-epithelial cell interactions.[9] On the other hand, SCFAs can enter the intestinal lumen via Toll-like receptor 4 (TLR4), activating the p38 mitogen-activated protein kinase/nuclear factor κB (p38 MAPK/NF-κB) pathway,[7] which results in the production of pro-inflammatory cytokines such as IL-6 and TNF-α, thereby eliciting an immune response.

-specific types based on the range of affected tissues and organs.[11] Organ-specific autoimmune diseases, such as type 1 diabetes (T1D), Hashimoto`s thyroiditis (HT), involve the pathological damage and dysfunction of tissues and organs limited to a certain organ targeted by antibodies or sensitized lymphocytes.[12] Non-organ-specific autoimmune disorders result from the extensive deposition of antigen-antibody complexes on the vascular walls, causing multi-organ damage throughout the body, such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA),Multiple sclerosis (MS).[13] About 5–8% of the global population suffer from autoimmune diseases, which are recognized as a global public health issue.[14] Recent reports indicated that microbial dysbiosis can disrupt immune function, leading to inflammation and sensitization of the immune system and the development of autoimmune diseases (Figure 2).[15] For example, multiple studies have reported a decreased *Firmicutes*/*Bacteroidetes* ratio in patients with SLE and T1D.[16,17] In patients with psoriatic arthritis, the relative abundance of *Clostridium, Akkermansia*, and *Ruminococcus* in the gut microbiota is reduced.[18] Compared with healthy age-matched controls, patients with celiac disease have higher abundances of *Bacteroides, Clostridium*, and *Staphylococcus*.[19] The interactive "host-gut microbiota" axis is crucial for maintaining local homeostasis and may also participate in the pathogenesis of certain autoimmune diseases by reshaping the intestinal immune system.[20]

Bell et al suggest that reshaping the gut microbiota can significantly affect the immune system of patients with autoimmune diseases,[21] and adjusting the gut microbiota may become a new therapy for treating autoimmune diseases. Probiotics are live microorganisms that, by colonizing the intestinal tract, produce substances beneficial to human health. Prebiotics can serve as an alternative to probiotics and, after fermentation, influence the activity and growth of probiotics in the colon.[22] Prebiotic and probiotic therapies modulate gut ecology by competing with harmful microorganisms for nutrients and colonization niches in combination with specific dietary patterns or prebiotics that support their colonization.[1] The functions of probiotics include maintaining the function of the gastrointestinal epithelial barrier, promoting the production of antimicrobial peptides, ensuring adequate interaction between the gut microbiota and

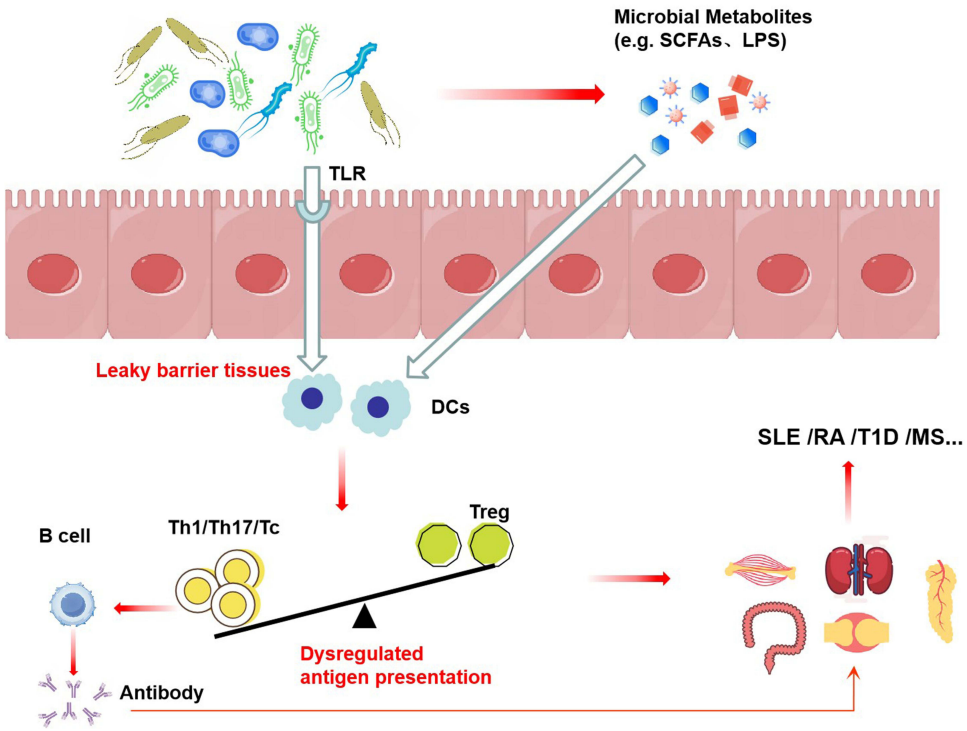

Figure 2 Dysbiosis causes autoimmune diseases.

mucosal immune cells, and ultimately helping to activate the host immune system in response to pathogenic bacteria.[23] Fecal microbiota transplant (FMT) involves transferring feces from a healthy donor to a patient by infusing bacterial liquid to provide colonization resistance, produce beneficial metabolic products, and restore interaction with the mucosa.[24,25] FMT can treat diseases by correcting the disordered gut microbiota of patients, reshaping it to promote the repair of intestinal barrier damage and alleviate metabolic inflammation.[26] It has been proven effective and relatively safe for various diseases including autoimmune diseases.[27] Deciphering the relationship between the gut microbiota and the innate immune system may elucidate various unknown causes of diseases.

In this paper, we reviewed the recent progresses of microbiome involved in the occurrence and development of the autoimmune diseases and the treatment methods based on regulation of the microbiome, highlighted the key role that the microbiome plays in some important autoimmune diseases, especially SLE, T1D and RA.

Systemic Lupus Erythematosus (SLE)

SLE is an autoimmune disease, which is mainly a chronic diffuse connective tissue disease caused by abnormal activation of the immune system to attack its own tissues,[28] characterized by the presence of hyperactive immune cells and aberrant antibody responses to nuclear and cytoplasmic antigens. The exact pathogenesis of SLE has not been fully elucidated, but dysregulation of the gut microbiota is closely related to the development of SLE.[29] Modulating the gut microbiota appears to be a potential approach for treating SLE. A recent metagenomic study demonstrated that transferring the probiotic flora of lupus-prone mice to non-autoimmune germ-free wild-type mice induced autoimmunity, indicating that the dysregulated gut flora can disrupt immune function, induce inflammation and immune system sensitization, resulting in autoimmune diseases.[30] Researches aim to establish effective experimental schemes to explore the microbiological basis of SLE between the gut microbiota and autoimmune diseases, which could lead to the development of effective treatment methods.

Bacterial infections are common in SLE patients, accounting for almost 80% of infections, with the most common bacteria being *Staphylococcus aureus, Streptococcus pyogenes, Streptococcus pneumoniae*, and *Escherichia coli*.[31] Studies have reported dysbiosis of the gut microbiota in patients with SLE, which is characterized by reduced bacterial

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diversity and biased community structure, with an increase in some genera such as *Campylobacter, Streptococcus*, and Micrococcus, and a decrease in other genera, such as *Bifidobacterium*,[32] resulting in a lower *Firmicutes*/*Bacteroidetes* ratio.[33] Many studies have also revealed gut microbiota dysregulation in lupus mouse models. Zhang et al found that the gut flora of MRL/lpr mice showed a significant decrease in *Lactobacillaceae* and an increase in *Lachnospiraceae*, which was particularly severe in female lupus mice.[34] Azzouz et al[35] reported that the average amount of *Ruminococcus gnavus* in lupus patients was five times higher than in healthy controls, with the greatest expansion in patients with high disease activity, especially those with lupus nephritis. Chen et al demonstrated that the abundance of *Candida albicans, Sulfurovum, Odoribacter*, and *Rothia* in mice treated with HCMVpp65 polypeptide was significantly associated with lupus-like effects.[36] These studies suggest that microbial dysbiosis in the gut of patients with SLE results in reduced bacterial diversity and a decreased proportion of protective commensal species, and that immune disorders triggered by the gut microbiota, together with genetic factors, mediate the occurrence and development of SLE.

Li et al showed that *Roseburia* and *Faecalibacterium prausnitzii* were depleted in SLE patients, especially *Bifidobacterium*, which was negatively correlated with SLE activity. [32] Some beneficial commensal microbes are part of the core community of the gut microbiota and can produce short-chain fatty acids (SCFAs), which play key roles in maintaining human health, such as reducing the severity of inflammation, maintaining intestinal barrier function, and enhancing colonic motility. [37–39] In addition, viruses have also been implicated in the development of SLE. An in vitro study demonstrated that virus-like particles extracted from the fecal samples of SLE patients increased the production of interferon-α in immune cells, suggesting a potential role of these virus-like particles in the pathogenesis of SLE, [40] but this conclusion requires further validation through comprehensive studies. Furthermore, Epstein-Barr virus (EBV) infection has been implicated in the development of SLE, with EB antigens having molecular similarity to SLE antigens, leading to an autoimmune reaction during EB activation. [41,42]

The gut microbiota is not only a potential therapeutic target for SLE, but also a potential biomarker for the diagnosis and prognosis of SLE, and it has been found that some biomarkers in the gut microbiome of SLE patients are related to disease activity indicators.[43] Traditionally, SLE is difficult to treat due to its heterogeneity and complex pathogenesis, while targeting intestinal bacteria may offer a breakthrough. Probiotic therapy has been extensively examined for the treatment of autoimmune disorders, effectively alleviating lupus-related syndromes and enhancing intestinal integrity.[44] For example, the presence of *Lactobacillus* in the gut attenuates renal inflammation in lupus mice in a sex hormone-dependent manner, and oral administration of five *Lactobacillus* strains to mice largely alleviated lupus-like symptoms.[45,46] In lupus-like animal models, administration of retinoic acid restored the genus *Lactobacillus* and ameliorated lupus symptoms, indicating that these strains can be used as probiotics to reduce inflammation in SLE patients.[34] The combined use of probiotics *Lactobacillus delbrueckii* and *Lactobacillus rhamnosus* can lessen the expression of chemokine receptors on the surface of dendritic cells in SLE patients, thereby prevent the migration of dendritic cells and reduce inflammation.[47] Furthermore, FMT can also be used to treat SLE, Huang et al[48] conducted the first FMT clinical trial in patients with active SLE and found that FMT treatment resulted in a significant increase in SCFA-producing bacterial taxa and a decrease in inflammation-associated bacterial taxa. FMT has shown potential therapeutic effects in SLE by reducing the predominantly FMT-responsive lymphocyte populations.[49] These data indicated that FMT may act through gut microbiota remodeling, reducing intestinal permeability and immunomodulation.[50] However, there are currently few studies on the interventional treatment of SLE patients by regulating the gut microbiota, such as fecal microbiota transplantation, and more studies are needed to verify the feasibility of this approach.

Type 1 Diabetes (T1D)

Type 1 diabetes is an autoimmune disease caused by T-cell-mediated destruction of insulin-secreting pancreatic β-cells.[5] The onset of T1D results from a complex interplay between genetic predisposition and environmental factors, which include dysbiosis of the gut microbiota, a compromised intestinal mucosal barrier, and altered immune responses within the intestine.[51] The gut microbiota plays a pivotal role in maintaining the integrity and function of the intestinal barrier. Various viral factors, such as rotavirus, cytomegalovirus, and enterovirus (EV), are linked to T1D.[52–54] Additionally, fungal dysbiosis in T1D relates to the richness of fungal species and the diversity of yeast-like species.[55] The increase in fecal yeast in T1D patients may be attributed to the impaired cellular immunity in a hyperglycemic environment, though the causal

relationship is unclear.[56] Disruption of this microbial balance can compromise the integrity of the gut barrier, facilitating the translocation of bacteria, fatty acids, and lipopolysaccharides into systemic circulation.[25] These substances can activate the immune system, leading to antibody production that may cross-react with antigens present on islet cells' surfaces. This process ultimately contributes to cellular destruction and facilitates the development of T1D.[57] Following the emergence of autoantibodies associated with T1D, there is frequently an observed deficiency in butyric acid-producing bacteria within the gut microbiome. Additionally, low bacterial diversity and community stability are common features during this phase.[58] Consequently, it becomes evident that gut microbiota is integral to immune regulation and plays a significant role in T1D pathogenesis.[59] Furthermore, it also serves as a crucial component in sustaining appropriate gut immune responses. Studies have shown that mice with MyD88 gene defects have an increased risk of T1D under germ-free conditions, while exposure to specific microbial mixtures reduces the incidence of diabetes.[60] This indicates a disorder in the gut microbiota of T1D patients and underscores the connection between improved blood sugar levels and changes in the gut microbiota.

Significant alteration occurred in gut microbial diversity and taxonomic composition of gut microbial communities between healthy controls and patients with T1D. T1D patients are enriched in Proteobacteria, Actinobacteria, and Bacteroidetes, and lack butyric acid-producing bacteria,[61,62] especially *bacteroidetes* dominate in areas with a high incidence of T1D.[63] Butyrate has anti-inflammatory properties, promotes the production of granzyme B in IL-10 tolerant Th1 cells to modulate intestinal inflammation, and enhances the intestinal barrier through tight junctions.[64,65] In addition, T1D is characterized by increased abundance of *Akkermansia muciniphila* and *Barnesiella*, both of which are lipopolysaccharides (LPS) producing bacteria,[66] LPS released by these bacteria may mediate inflammation, obesity, and insulin resistance.[67] Kostic et al assessed fecal samples from 33 children genetically predisposed to T1D. They observed a decrease in the number of Gram-negative bacteria in the children as well as a negative correlation between *Lachnospiraceae* and Gram-negative aerobic *Enterobacteriaceae*.[68]

Evidence from germ-free Non Obese Diabetes (NOD) mice suggests that specific pathogen-free (SPF) NOD mice are more susceptible to T1D,[69,70] with a significant increase in *Firmicutes, Bacteroidetes, Ruminococcaceae, Proteobacteria, A. muciniphila*, and *Enterococcus* prior to the T1D autoimmune response in these mice.[71] This further supports the important role of the gut microbiota in the occurrence and progression of T1D. In addition to bacteria, enteroviral infections in pregnant women have also been reported to increase the risk of T1D in their offspring,[72] and certain viral exposures during childhood are also associated with T1D.[73,74]

During the progression of T1D, the immune system targets three major autoantigens: insulin, glutamic acid decarboxylase (GAD) and islet-associated antigen (IA-2),[75] with autoantibodies against these antigens serving as markers for predicting the disease. The unique characteristics of gut microbiota in T1D patients can also serve as biomarkers for T1D.[16] The gut microbiota is a potential target for T1D prevention and treatment, with certain antibiotics or probiotics potentially influencing the development of disease by altering the gut microbiota balance.[76]

Commensal bacteria are active participants in shaping the host immune network. *Lactobacillus* can enhance the integrity of the intestinal epithelial barrier and prevent intestinal leakage by promoting the expression of tight junction proteins.[77] Doxycycline can reduce the incidence of T1D in SPF NOD mice by decreasing *Bacteroides, Clostridium*, and *Lactobacillus*.[78] A murine study found that when NOD mice consumed dietary fiber inulin-type fructans, the occurrence of T1D could be prevented.[79] In newly diagnosed T1D patients, FMT may preserve residual pancreatic β-cell function,[80] and reduce islet β-cell apoptosis.[81] Metformin, a preferred drug for blood sugar control in diabetic patients, interacts with the gut microbiota by modulating inflammation, intestinal permeability and short-chain fatty acid-producing bacteria.[82] Bryrup et al found that following six consecutive weeks of oral metformin administration, patients experienced significant changes in gut microbiota richness, with a decrease in *Enterobacter* and *Clostridium* and an increase in *Salmonella*.[83] The clear association between gut microbiota dysbiosis and T1D has significant clinical implications, and microbiota-based interventions, such as probiotics, could reduce or even prevent the heavy demand for insulin injections.

Rheumatoid Arthritis (RA)

RA is a systemic autoimmune disease characterized by synovial joint inflammation. The defining feature of RA is persistent synovial inflammation with extensive neutrophils infiltrating into bone tissue, mediated by cytokines such as

TNF-α and IL-17, eventually leading to damage to articular cartilage and bone.[84] Researchers employing the spontaneous arthritis model, K/BxN mice, have discovered the connection between the gut microbiota and autoimmune arthritis.[85] An enrichment of fragmented filamentous bacteria within the gut microbiota induces the activation of Th17 cells in the lamina propria of the small intestine, thereby triggering an immune response. Subsequently, Th17 cells migrated to peripheral lymphoid tissues and secreted IL-17.[86] In turn, IL-17 directly acted on B cells, facilitating their differentiation and production of autoantibodies, ultimately leading to disease progression. Growing evidence suggests that the gut microbiota induces systemic inflammation and polyarthritis through multiple mechanisms including impaired gut barrier function, immune regulation mediated by gut microbiota-derived metabolites, modulation of the impact of gut microbiota on immune cells, autophagy in intestinal epithelial cells, interactions between the microbiome and alleles of human leukocyte antigen, as well as microRNAs.[87]

The gut microbiota plays a key role in the progression of RA.[88] An abundance of *Prevotella copri* has been observed in both RA patients and mouse models,[89] which stimulates the production of Th17-related cytokines, particularly IL-6 and IL-23.[90] Furthermore, a study comparing the gut microbiota of anti-citrullinated protein positive individuals and healthy controls, *Prevotella* was positively correlated with rheumatoid factor (RF) titer.[91] Earlier observations in a type II collagen-immunised RA mouse models revealed a higher relative abundance of *Trichosporonaceae, Ruminococcaceae* and *Bacteroidaceae* as a distinctive feature of changes in the gut microbiota of RA.[92,93] Some studies have also shown that specific bacterial families such as *Muribaculaceae, Proteobacteria*, and *Firmicutes* are negatively correlated with RA progression. Additionally, a decrease in the relative abundance of Firmicutes/Bacteroidetes has been observed.[94,95] *Faecalibacterium prausnitzii* is one of the important commensal bacteria in the human gut flora, accounting for 3–5% of the total number of bacteria detected in fecal samples from healthy individuals.[96] A decrease in the abundance of *Faecalibacterium perfringens* in the intestine may be a sign of dysbiosis, such as a decreased self-defense ability against inflammatory responses.[97] Interestingly, *Clostridium difficile* infection can promote mesenteric Tregs and Th2 polarization, thereby prevent the development of RA.[98] Furthermore, Zhu et al found that *Candida* and *Debaryomyces* were more enriched in patients with RA and may serve as potential biomarkers for predicting early RA.[99] However, the relationship between fungi and RA, as well as the biological functions of fungi, require further exploration.

Current approaches for treating RA include probiotics, prebiotics, antibiotic therapy and FMT.[100,101] Antibiotic therapy can alleviate arthritis severity by inhibiting Th17 cell differentiation and serum amyloid A (SAA) production and eliminating part of the gut microbiota.[94] However, these treatments reduce the levels of *Ruminococcus, Faecalibacterium* and *Bacteroides*, which lead to the reduction in intestinal metabolites (uracil, primary bile acids and essential amino acids).[102] While gut bacterial intervention is a promising therapeutic approach. Dietary supplements and prebiotics have been used in the clinical management of RA by rebalancing the composition of the gut flora.[103,104] Oral probiotics can competitively inhibit the colonization of pathogenic *Prevotella* and suppress the production of autoantibodies.[105] In a recent study, the oral administration of *Bacillus coagulans* with anti-inflammatory properties significantly reduced serum levels of SAA and tumor necrosis factor in a rat model of RA.[106] Mechanistic studies of high-fiber supplements have shown that short-term high-fiber supplementation improved immune dysregulation by increasing Treg counts and the ratio of Th1/Th17 and altering the ratio of *Firmicutes* to *Bacteroidetes*.[107,108] In an in vivo study of RA, human histobacterium alleviated disease severity without damaging intestinal tissue, suggesting its therapeutic potential.[109] *Streptococcus lactis* induces immune tolerance through the recombination of Hsp65. Hsp65 reduces the synthesis of autoantibodies and the production of inflammatory cytokines via the TLR2 signaling pathway, thereby inhibiting the progression of type II collagen-induced arthritis.[110] FMT has been applied to various parenteral diseases. The first trial of FMT for the refractory RA has achieved a favorable therapeutic outcome with reducing arthritis scores and RF levels.[101] Therefore, combination therapy of probiotics, dietary interventions, and FMT therapy in regulating the microbiota to prevent RA progression would be a fruitful area for further work.

Future Perspectives

In summary, this article has reviewed the recent progresses of the relationship between gut microbiota imbalance and autoimmune diseases from the perspectives of gut microbiota and SLE, T1D, and RA. By unraveling the complex relationship between the gut microbiota and the immune system, the efficacy of autoimmune diseases treatment can be

greatly enhanced. Factors such as impaired intestinal barrier function, intestinal metabolites, specific pathogens and sex hormones can disrupt the gut microbiota homeostasis and exacerbate the autoimmune diseases. We have reviewed the disturbances in the gut microbiota associated with SLE, T1D and RA, highlighting the composition of gut-related microbial communities and their impact on disease development. Additionally, we have summarized the latest therapeutic modalities for the treatment of SLE, T1D and RA with the strategy of microbiota regulation, including probiotics and FMT.

FMT is an active and effective therapeutic approach to modify the composition of the microbiota in a limited clinical setting. To date, FMT has been extensively used to treat a variety of diseases including *C. difficile* infection,[111,112] autism, multiple sclerosis, and Parkinson's disease.[113,114] The application of FMT in the treatment of autoimmune diseases is effective and relatively safe, and it is expected to become an effective treatment for autoimmune diseases.

Microorganisms have exerted an influence on the evolution of multicellular host organisms from the dawn of time, making their role in host biology particularly notable.[115] Even though the gut microbiota plays a vital role in molding the immune system and has a strong connection with innate immunity and adaptive immunity, the association between the gut microbiota and autoimmune diseases still calls for further research to clarify their inevitable correlation. Probiotics, as advantageous microorganisms, not merely play a significant role in regulating intestinal immune function but also hold potential as novel adjuncts in cancer treatment.[116] However, individual variances, safety, efficacy and cost-effectiveness of the probiotic treatment remain matters that need to be tackled. Future research ought to focus on identifying the ideal combinations of probiotic species for multispecies therapy to alleviate immune diseases through modulation of immune response, metabolism and gut microbiota. While these findings are preliminary, they are highly auspicious as they showcase the potential of microbial communities as future therapeutic targets.

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Disclosure

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