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Gut microbiota and inflammatory diseases: current evidence and future perspectives

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ABSTRACT

Introduction: The gut microbiota is a complicated and dynamic community of microorganisms exerting critical regulatory functions in immune homeostasis, nutrient metabolism, and defense against pathogens. Growing evidence demonstrates that disruption of microbial balance — referred to as dysbiosis — recreates a pivotal role in the pathogenesis of a wide range of inflammatory diseases, including inflammatory bowel disorder (IBD), rheumatoid arthritis, systemic lupus erythematosus, neurodegenerative conditions, and allergic disorders. *Methods*: The present narrative review was conducted using electronic databases and searches were performed between January 2018 – April 2026. Some of the important words were: microbes; community of microbes; imbalance of microbes; swelling; long-chain fatty acids; moving microbes from one place to another; and ways to treat illness. The study included peer-reviewed articles published in indexed journals, in English and other languages with available summaries. *Results*: Research consistently shows that disease-associated dysbiosis is characterized by reduced microbial diversity and altered ratios of certain bacteria, and decreased production of SCFAs. Specific bacteria, for example *Faecalibacterium prausnitzii* and *Akkermansia muciniphila*, have been identified as protective microorganisms, and their depletion has been linked to increased inflammatory activity. Therapeutic strategies, including probiotics, prebiotics, dietary interventions and faecal microbiota transplantation (FMT), offer promising but variable results across disease states. *Conclusion*: The gut microbiota constitutes a modifiable therapeutic target for multiple inflammatory conditions. The future of precision medicine, multi-omics profiling and personalized nutrition holds great prospects for microbiota-targeted interventions.

Keywords: gut microbiota; dysbiosis; inflammatory bowel disease; immune modulation; therapeutic targets

1. INTRODUCTION

The human gastrointestinal tract harbors approximately 100 trillion microorganisms, collectively known as the gut microbiota—a diverse community

encompassing bacteria, fungi, viruses, archaea, and protozoa. The genetic and functional profile of this microbial assembly is referred to as the gut microbiome. The bacteria inhabiting the gut collectively encode an estimated 3.3 million genes, which is substantially more than the approximately 23,000 genes that comprise the human genome. This fact serves to emphasize the remarkable metabolic and immunological potential of this ecosystem (Al Bander et al., 2020).

The gut microbiota fulfills multiple homeostatic functions, including the fermentation of dietary fibers into SCFAs, synthesis of vitamins, protection against enteric pathogens, regulation of intestinal barrier integrity, and training of the immune system (Benameur et al., 2023). Early colonization of the gastrointestinal tract in neonatal life — influenced by mode of delivery, feeding practices, and antibiotic exposure — is critical for the proper maturation of both innate and adaptive immunity (Maciel-Fiuza et al., 2023). Experimental data in germ-free animal models confirm that, in the lack of gut microbiota, the intestinal mucosal immune system is profoundly underdeveloped, with reduced numbers of functional regulatory T cells and impaired capacity to resist pathogenic invasion.

The term dysbiosis describes a qualitative and/or quantitative alteration in the composition of microbiota that impairs its normal functions. Dysbiosis can arise from antibiotic use, dietary patterns, life habits, age, host genetic polymorphisms and environmental exposures. When dysbiotic states persist, they can trigger aberrant immune activation, intestinal barrier dysfunction, and systemic low-grade inflammation — mechanisms linked to the pathogenesis of conditions as diverse as IBD, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), allergic diseases, chronic obstructive pulmonary disease (COPD), and neurodegenerative disorders (Zeng et al., 2026; Aziz et al., 2023).

The objective of this review is to give a comprehensive synthesis of current evidence on the relationship between the gut microbiota and inflammatory diseases, to explain the molecular and immunological mechanisms by which microbial imbalance contributes to inflammation, and to explore emerging therapeutic methods targeting the microbiome.

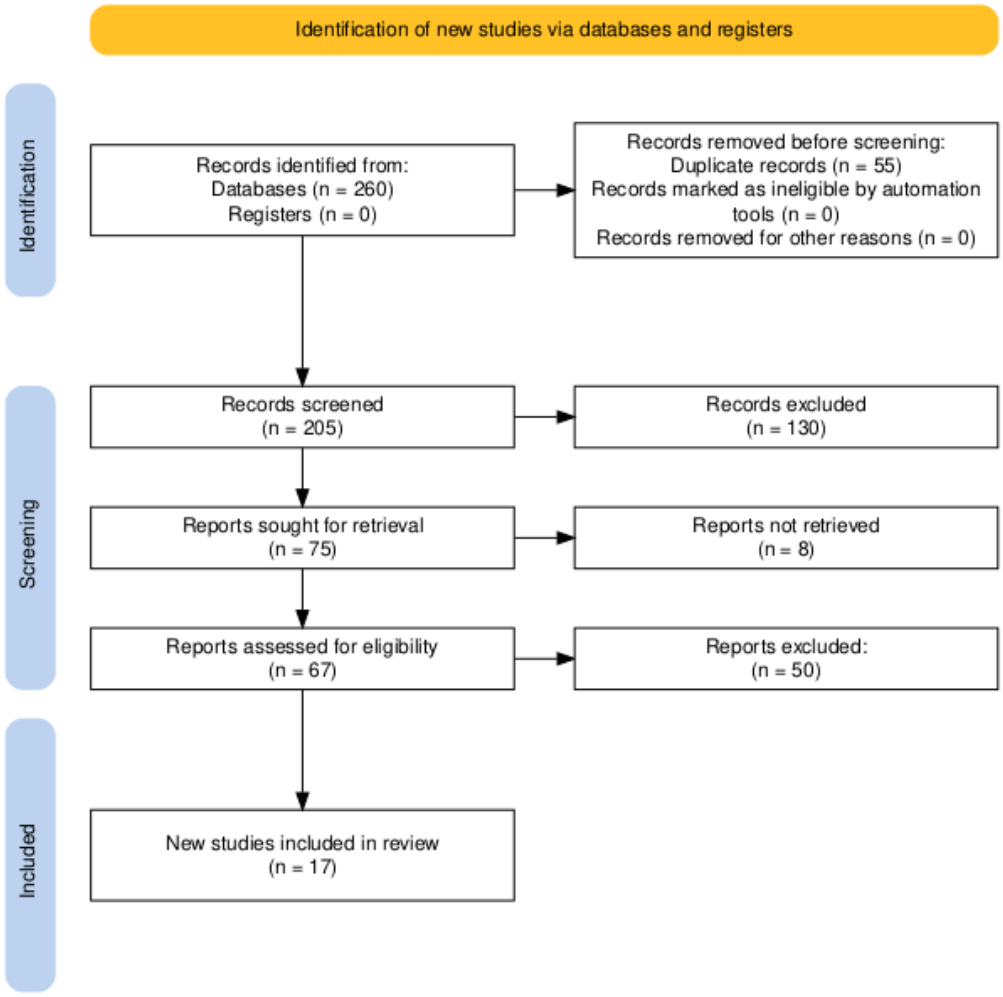

Figure 1. PRISMA flow diagram of study selection

2. REVIEW METHODS

Databases: PubMed and Google Scholar.
Time range: Articles published in the period January 2018 – April 2026, with earlier seminal studies included where relevant.

The search keywords used were: gut microbiota, microbiome, dysbiosis, inflammation, inflammatory bowel disease, autoimmune disease, short-chain fatty acids, faecal microbiota transplantation and treatment targets.

Inclusion criteria: Peer-reviewed articles from indexed journals in English or with available summaries/abstracts in other languages.
Exclusion criteria: non-peer-reviewed studies, articles published outside the January 2018 – April 2026 date range, articles without an abstract, and duplicate publications.

PRISMA flow diagram illustrating the method of selecting studies (Figure 1). A total of 260 records were identified through database searches of PubMed, Google Scholar. After removing 55 duplicates, the remaining 205 records were studied based on their titles and abstracts. Of these, 130 were excluded because they were irrelevant to the topic or outside the January 2018 – April 2026 date range. Of the 75 reports sought for retrieval, eight were not retrieved due to an unavailable full text, leaving 67 reports to be assessed for eligibility. Following the exclusion of 50 reports (due to them being non-peer-reviewed sources, being outside the specified date range, or being of low relevance), 17 analyses were included in the final analysis.

3. RESULTS AND DISCUSSION

Composition and Function of the Gut Microbiota

Microbial Diversity and Influencing Factors

The gut microbiota is defined as a dynamic ecosystem that is subject to continuous shaping by a range of intrinsic and extrinsic factors. Mode of delivery at birth has been demonstrated to significantly influence the composition of the infant's microbiota. Neonates delivered vaginally have been shown to be primarily colonized by maternal vaginal bacteria, including *Lactobacillus*, *Prevotella*, and *Sneathia* species, while caesarean-born infants have been observed to harbour more cutaneous flora such as *Staphylococcus* and *Corynebacterium* (Al Bander et al., 2020). As demonstrated in the extant literature, breastfeeding has been shown to promote the predominance of Bifidobacterium species, which have been linked to immune tolerance and protection against infection. Diet is considered to be one of the most significant modifiable factors influencing microbiota composition. A plant-based diet, abundant in fibre, has been observed to promote higher abundances of genera that are competent of producing short-chain fatty acids (SCFAs). In contrast, diets characterized by elevated levels of saturated fats and processed foodstuffs have been demonstrated to stimulate the proliferation of taxa associated with pro-inflammatory metabolic outputs (Al Bander et al., 2020; Aziz et al., 2023). Antibiotic exposure is among the most disruptive extrinsic factors, capable of inducing rapid and often permanent alterations in microbiota structure. As demonstrated in extensive research (Al Bander et al., 2020), there is a correlation - the utilization of antibiotics during childhood and the subsequent manifestation of metabolic dysregulation, as well as an elevated body mass index.

Metabolites and Immunological Functions

The gut microbiota exerts its systemic effects largely through the production of metabolically active compounds. SCFAs — primarily butyrate, propionate, and acetate — are generated by the anaerobic fermentation the dietary fiber by commensal bacteria. Butyrate, produced by certain types of gut bacteria, has been shown to have health benefits by blocking cell signals and increasing receptors that can be stimulated by touch. Butyrate promotes regulatory T (Treg) cell differentiation and suppresses NF-κB-mediated inflammation (Zheng and Wen, 2021; Liu et al., 2022; Al Bander et al., 2020). Butyrate alleviates inflammation in Crohn's disease (CD) when taken orally. This finding serves to illustrate the therapeutic potential of SCFA modulation. Furthermore, tryptophan metabolites, including indole derivatives produced by gut bacteria, have been shown to maintain epithelial cell integrity (Zheng & Wen, 2021; Maciel-Fiuza et al., 2023), stimulate mucin secretion (Zheng & Wen, 2021) and promote the differentiation of anti-inflammatory innate lymphoid cells (ILC3s) and Treg cells (Maciel-Fiuza et al., 2023). Secondary bile acids, which are formed by means of microbial processes, have regulatory functions in relation to bile acid-related processes. The binding of bacterial components to immune cell receptors serves as a catalyst for the initiation of inflammatory cascades. The presence of LPS in the bloodstream can result in the induction of endotoxaemia and systemic inflammation. Trimethylamine N-oxide (TMAO), a by-product of microbial metabolism of dietary choline and carnitine,

has been demonstrated to further link dietary patterns to endothelial dysfunction, vascular inflammation, and elevated TNF-α and IL-6 levels (Al Bander et al., 2020).

Microbial Alterations in IBD

Inflammatory bowel disease (IBD) is the most studied condition involving the gut microbiota. It includes Crohn's disease (CD) and ulcerative colitis (UC). The number of cases of IBD around the world has increased a lot in the 21st century, which has had a big impact on people's lives. IBD is understood to be caused by a mix of genetics, environmental factors, and an immune response that goes wrong when it comes to the bacteria in the gut (Zheng and Wen, 2021; Stange, 2022). Researchers have found over 163 susceptibility loci, many of which are involved in how the body responds to microbes. These loci include variants in NOD2, ATG16L1, and CLEC7A (Zheng and Wen, 2021; Yan et al., 2022).

Patients with IBD consistently demonstrate reduced microbial diversity and stability compared to healthy individuals, characterized by an increase in Proteobacteria — notably adherent-invasive Escherichia coli (AIEC) in CD and diffusely adherent E. coli in UC — alongside decreases in beneficial species such as *Faecalibacterium prausnitzii, Bifidobacterium, Lactobacillus*, and *Akkermansia muciniphila* (Zheng and Wen, 2021; Maciel-Fiuza et al., 2023). The abundance of *F. prausnitzii* — a major butyrate-producing bacterium with demonstrated anti-inflammatory properties — is significantly reduced in CD patients and has been proposed as a biomarker for disease relapse (Zheng and Wen, 2021; Laing et al., 2018). The microbial anti-inflammatory molecule (MAM) protein produced by F. prausnitzii stops the NF-κB pathway in epithelial cell lines, showing a clear connection that microbial depletion leads to increased intestinal inflammation (Duru, 2022).

Immune cells in Crohn's disease the ulcerative colitis are different. CD is associated with Th1 and Th17 cytokine profiles (IL-12, IL-18, IL-23), whereas UC is influenced by Th2 immunity and IL-5 secretion (Maciel-Fiuza et al., 2023). Gut microbiota (the microbes living in the gut) in IBD (inflammatory bowel disease) work with other cells in the gut to change the environment and the levels of cytokines (chemicals that regulate the immune system) in the gut. This changes the gut environment and makes it harder for the gut to repair itself. When these findings are considered as a whole, it seems that a imbalance in the bacteria in the gut may be a contributing aspect to the onset and ongoing of IBD. However, the direction of this relationship is still being debated (Zheng and Wen, 2021; Oliveira et al., 2023).

Metabolomic Disturbances and miRNA–Microbiota Interactions

The metabolomic profile of IBD patients is significantly different to that of healthy controls, with a big decrease in concentrations of short-chain fatty acids (SCFAs), which are created by bacteria in the gut (Zheng and Wen, 2021). Intestinal tryptophan metabolism is also impaired in IBD, with decreased production of indole derivatives contributing to epithelial barrier dysfunction and reduced IL-22-mediated mucosal repair. Secondly, there are changes to the secondary bile acid profiles, and vitamins B3 (nicotinate) and B5 (pantothenate) – which can reduce inflammation and prevent cell death – are specifically lower in the intestinal microenvironment of IBD patients. This suggests that microbial metabolic failure can lead to nutritional deficiency at the mucosal level (Zheng and Wen, 2021; Primke et al., 2025). MicroRNAs regulate microbiota, which then modulates host miRNAs, perpetuating intestinal inflammation in IBD (Oliveira et al., 2023).

Rheumatoid Arthritis

Studies have shown that there is a link between gut dysbiosis and rheumatoid arthritis (RA). People with RA have less diversity in their intestinal microbes, which is linked to how long they've had the disease and the levels of autoantibodies in their body (Maciel-Fiuza et al., 2023; Zeng et al., 2026). In patients with rheumatoid arthritis, who have not yet been treated with drugs, there are more of a type of bacteria called Prevotella copri and fewer of a type called Bacteroides. Collinsella aerofaciens has been identified as a possible cause of arthritis, and lower levels of Faecalibacterium, it is linked to a loss of anti-inflammatory effects, further connect intestinal imbalances to joint inflammation (Maciel-Fiuza et al., 2023). Recent studies have shown that there are fewer different types of microbes in autoimmune diseases, and these microbes can make the disease worse by causing a reaction that is like an allergic reaction and by not letting the immune system work properly (Zeng et al., 2026).

Systemic Lupus Erythematosus

In another study, researchers found that people with systemic lupus erythematosus (SLE) had fewer bacteria in their guts. These bacteria were called Firmicutes and Bacteroidetes. The researchers also found that these bacteria were not as diverse as they should have been. Some types of bacteria were more common in people with SLE than in people without it. These included *Rhodococcus, Eggerthella, Klebsiella*, and *Prevotella*. Dysbiosis (a problem with the bacteria in the gut) in SLE is linked to how active the disease is. The bacteria Streptococcus and Veillonella are linked to disease flares because they cause inflammation, and the bacteria Bifidobacterium is linked to a lower risk of disease. This shows the protective effect of certain types of bacteria (Maciel-Fiuza et al., 2023; Zeng et al., 2026). Molecular mimicry is another way in which the imbalance of microbes in the body (dysbiosis) can cause or increase autoimmune responses in SLE. During molecular mimicry, microbial antigens (substances that cause an immune reaction) share a structure with host autoantigens (substances that cause an immune reaction to the body's own cells).

The table 1 compares characteristic microbial shifts and underlying mechanisms across four major inflammatory conditions reviewed in this study, highlighting depletion of beneficial taxa (e.g., *Faecalibacterium prausnitzii*, *Akkermansia muciniphila*) and expansion of pro-inflammatory bacteria as recurring features linked to disease pathogenesis.

Table 1. Summary of gut microbiota alterations across inflammatory diseases

Disease	Decreased microbiota	Increased microbiota	Key mechanism
Inflammatory bowel disease (IBD)	*Faecalibacterium prausnitzii, Bifidobacterium, Lactobacillus, Akkermansia muciniphila*	Proteobacteria, adherent-invasive E. coli (AIEC)	Reduced SCFA production, NF-κB pathway activation, impaired mucosal repair
Rheumatoid arthritis (RA)	*Bacteroides, Faecalibacterium*	*Prevotella copri, Collinsella aerofaciens*	Loss of anti-inflammatory effects, joint inflammation via immune activation
Systemic lupus erythematosus (SLE)	*Firmicutes, Bacteroidetes, Bifidobacterium*	*Rhodococcus, Eggerthella, Klebsiella, Prevotella, Streptococcus, Veillonella*	Molecular mimicry between microbial and host autoantigens, disease flares
Allergic diseases (asthma, eczema, food allergies)	Overall bacterial diversity, SCFA-producing taxa	Not specified	Weakened skin barrier, gut-skin axis dysregulation, hygiene hypothesis

Allergic Diseases

The microbes in our guts are very important for our immune system to accept other microbes. If these microbes are not there in the first few years of life, it can lead to allergic diseases like asthma, eczema, food allergies and hay fever. The hygiene hypothesis suggests that vulnerability to germs can shift the immune system. In those with atopic dermatitis, lower bacterial diversity in the gut is linked to reduced SCFA production. This reduced production can weaken the skin's barrier, intensify inflammation and promote communication between the gut and skin. Probiotics, especially Lactobacillus rhamnosus GG (LGG), can help stop babies from getting eczema and make the symptoms of peanut allergies better when used with a treatment called oral immunotherapy (Samberger et al., 2026).

Barrier Dysfunction and Endotoxaemia

The intestinal epithelial barrier is an essential determinant of the systemic inflammatory tone. Under homeostatic conditions, tight junction proteins limit the passage of microbial products from the luminal to systemic compartment. Dysbiotic states compromise tight junction integrity, allowing LPS and other pathogens to enter the blood. This triggers an immune reaction, producing pro-inflammatory cytokines. Some scientists think that a build-up of bacteria in the gut can induce inflammation throughout the body, which can lead to conditions like metabolic syndrome, heart disease, and autoimmune diseases (Al Bander et al., 2020).

Immune Cell Polarization and Cytokine Networks

The gut microbiota shapes immune responses by interacting with antigen-presenting cells, T lymphocytes and innate immune cells (Maciel-Fiuza et al., 2023). A type of bacteria called Clostridium and a type of bacteria called Bacteroides fragilis help to control the immune system. Dysbiosis decreases helpful bacteria in the gut, which support bodily functions, and increases pro-inflammatory bacteria. In fact, this change is seen in all three of these diseases. Butyrate is made by SCFA and it strengthens the effects of regulatory T-cells by blocking HDAC and activating glucocorticoid-receptor (GCPR) activity. At the same time, tryptophan metabolites help IL22-producing ILC3 cells and tissue repair (see references below). Some bacteria in the gut can make the immune system attack other parts of the body (Al Bander et al., 2020).

Probiotics and Prebiotics

Probiotics are live microorganisms that, when administered in sufficient amounts, confer health benefits to the host. Clinical evidence for probiotic efficacy is strongest in ulcerative colitis (UC), where preparations such as VSL#3 — combining multiple Lactobacillus, Bifidobacterium, and Streptococcus salivarius strains — have demonstrated benefits in inducing and maintaining remission and preventing pouchitis (Zheng and Wen, 2021). Evidence for CD remains limited. Non-digestible dietary fibres may complement probiotic strategies by supporting beneficial bacteria (Laing et al., 2018). In diseases where the body is allergic to something, using a mixture of prebiotics and probiotics at the same time has had good results. This includes getting used to cow's milk and preventing eczema (Samberger et al., 2026). The idea of synbiotics — combining probiotics with their preferred prebiotic substances — is used more and more to maximise the process of colonisation and long-term microbiome change.

Dietary Interventions and Personalized Nutrition

The things we eat have the biggest effect on the types of bacteria in our stomachs. Eating a Mediterranean-style diet is good for your health. These diets help to grow helpful bacteria in your body. They also help to reduce inflammation. But eating a lot of ultra-processed foods, saturated fat, sugar, and meat can make you more likely to have inflammation. In IBD, a special type of feeding called exclusive enteral nutrition (EEN) is very good at getting children's CD under control. It does this by changing the bacteria in their gut, which is called microbiome modulation (Zheng and Wen, 2021). Emerging microbiome-based personalized nutrition approaches show short-term benefits (Duquesnoy et al., 2025). Adding vitamins and other nutrients can help the microbes in the gut work better in diseases like PSC and IBD (Primke et al., 2025).

Future Perspectives

The rapid advancement of sequencing technologies — including 16S rRNA amplicon sequencing, shotgun metagenomics, metatranscriptomics, and metabolomics — is enabling increasingly granular understanding of the gut microbiome's functional contributions to inflammatory disease. Research in the future should deal with the main problems of the current studies. These include the fact that people vary a lot from each other, differences in how the samples are collected and how they are studied, and the difficulty of showing that one thing causes another in the data (Stange, 2022; Oliveira et al., 2023). Combining information about microbes in the body with information about genes, proteins and metabolites could help us to find new ways to target treatments (Duquesnoy et al., 2025).

The gut–brain and gut–lung axes show the microbiome influences intestinal inflammation and beyond. For diseases affecting the nervous system, breathing, and the body's processes, strategies that target microbes can change the body's inflammatory processes (Roudenský, 2025; Zhu et al., 2025). To turn the potential of microbiome science into real improvements in patient care, we need to overcome some existing challenges. For example, we need to agree on ways to assess the microbiome. We also need to make sure that everyone can access precision medicine tools and that there is strong evidence for new treatments.

4. CONCLUSION

Substantial and converging evidence supports the gut microbiota as a major regulator of immune homeostasis and a driver of inflammatory pathology when dysbiotic. There are some common features that occur in a number of health problems, including autoimmune diseases, allergic conditions and neurodegenerative disorders. Decreased microbial diversity, impaired SCFAs, altered immune cell polarization, and compromised epithelial barrier function are features to watch. Current treatment strategies — including probiotics, prebiotics, changes to people's diet, and FMT — can be very helpful in some cases, but they don't always work the same

way. The field is advancing rapidly toward precision, individualized interventions guided by multi-omics profiling, and as understanding of the molecular language of the microbiome deepens, opportunities for targeted prophylactic and therapeutic modulation of the gut microbiota will become increasingly concrete, with the capacity to transform the management of inflammatory diseases across multiple medical specialties.

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Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

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