

Gut Microbiota, Irritable Intestinal Syndrome and Dysbiosis: Pathophysiology, Medications and Perspectives on Mechanisms

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Abstract: The human gut harbours a complex and diverse community of microorganisms that plays an important role in digestion, immune regulation, metabolism, and maintenance of intestinal health. A healthy gut microbiota is dominated by beneficial microorganisms, particularly anaerobic bacteria such as *Faecalibacterium prausnitzii*, *Bacteroides*, *Lactobacillus*, and *Bifidobacterium*, which contribute to the production of short-chain fatty acids and maintenance of the intestinal barrier. In inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, this microbial balance is disturbed, resulting in a condition known as gut microbiota dysbiosis. Dysbiosis is commonly associated with a reduction in beneficial anaerobic bacteria and an increase in potentially harmful or oxygen-tolerant microorganisms, including certain strains of *Escherichia coli* and *Enterococcus*. These changes may alter intestinal oxygen availability, microbial diversity, metabolite production, epithelial barrier function, and immune responses, thereby contributing to intestinal inflammation and disease progression. Increasing evidence suggests that microbial alterations may occur early in IBD and may contribute to both disease initiation and progression. This review summarizes the major changes in gut microbiota associated with IBD, discusses the mechanisms through which dysbiosis may promote intestinal inflammation, and highlights its role in disease pathogenesis. The review also discusses emerging therapeutic approaches aimed at restoring microbial balance, including probiotics, prebiotics, dietary interventions, faecal microbiota transplantation, and other microbiota-targeted strategies. Understanding the relationship between gut microbiota and IBD may provide opportunities for improved diagnosis, prevention, and development of personalised therapeutic approaches.

Keywords: Gut Microbiota; Dysbiosis; Inflammatory Bowel Disease; Crohn's Disease; Ulcerative Colitis; Anaerobic Bacteria; Intestinal Inflammation; Gut Barrier; Short-Chain Fatty Acids; Probiotics; Faecal Microbiota Transplantation.

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I. INTRODUCTION

The human gastrointestinal (GI) tract is a complex environment and an important interface between the host, food, environmental factors and microorganisms. The gut microbiota is the community of microorganisms that colonise the gastrointestinal tract, and the gut microbiome is the sum of their genes. They consist of bacteria, archaea, fungi, viruses and other microbial populations that constantly interact with the host and are involved in intestinal health and physiological functions (Manichanh et al., 2012; Kostic et al., 2014).

The distribution of microorganisms varies along the gastrointestinal tract. The stomach and small intestine generally have lower numbers of microbes due to acidity, bile and rapid transit through the intestine. The large intestine has the greatest density and diversity of microorganisms, especially anaerobic bacteria. Microorganisms have been

found in the intestinal lumen and associated with the mucus layer and epithelial surface where they interact with dietary components and host tissues (Manichanh et al., 2012).

A healthy gut microbiota plays a role in digestion, nutrient metabolism, intestinal barrier integrity, and immune regulation. Microorganisms ferment dietary fibres and complex carbohydrates into short-chain fatty acids (SCFAs), mainly acetate, propionate and butyrate. These metabolites support intestinal epithelial cells and contribute to metabolic and immune homeostasis (Koh et al., 2016; Parada Venegas et al., 2019). The microbiota also helps to protect against potentially harmful microorganisms and contributes to appropriate development and regulation of intestinal immune responses (Round & Mazmanian, 2009; Dalal & Chang, 2014).

In general, the host-gut microorganism interactions are relatively stable. Gut microbiota dysbiosis refers to the

alteration of microbial composition, diversity, or function in the gut. Dysbiosis can include a depletion of beneficial microorganisms, an increase in potentially pathogenic microorganisms, and changes in the microbial metabolic activity (Kostic et al., 2014; Schirmer et al., 2019). Changes have been linked to several gastrointestinal disorders, in particular inflammatory bowel disease (IBD).

Inflammatory bowel disease (IBD) is a chronic relapsing inflammatory disease of the gastrointestinal tract, mainly including Crohn's disease (CD) and ulcerative colitis (UC). Despite differences in their anatomical distribution and pathological features, both CD and UC are characterised by complex interactions between genetic susceptibility, environmental factors, intestinal microorganisms, epithelial barrier function, and immune responses (Matsuoka & Kanai, 2015; Sartor & Wu, 2017). Crohn's disease can affect any part of the gastrointestinal tract and is typically associated with discontinuous inflammation, while ulcerative colitis is characterised by continuous inflammation of the mucosa and mainly involves the colon.

Alterations in the gut microbiota are common in patients with IBD. The changes can involve a reduction in microbial diversity, a loss of beneficial bacteria and an increase in the number of microorganisms associated with inflammation (Halfvarson et al., 2017; Schirmer et al., 2019). Reduced levels of the butyrate-producing bacterium *Faecalibacterium prausnitzii* have been associated with Crohn's disease, while some members of Proteobacteria, including some strains of *Escherichia coli*, may increase during intestinal inflammation (Sokol et al., 2008; Sartor & Wu, 2017).

Microbial dysbiosis may affect IBD through multiple interconnected mechanisms. Changes in microbial composition (Lavelle & Sokol, 2020) can affect SCFA production and other metabolites, intestinal epithelial barrier function and mucosal immune responses. Reduced production of beneficial metabolites such as butyrate could lead to disruption of intestinal homeostasis. Increased exposure to microbial products could lead to aberrant immune activation.

The relationship between dysbiosis and IBD is complex because intestinal inflammation can also alter the microbial environment. Thus, microbiota imbalance could induce inflammation and inflammation could induce microbiota changes, which is a vicious cycle (Matsuoka & Kanai, 2015; Ni et al., 2017). Dysbiosis is therefore increasingly seen as an important component of the pathogenesis of IBD, rather than just a consequence of the disease.

However, with the advances of 16S rRNA sequencing, metagenomics, metabolomics and multi-omics technologies, we now have more insight into the composition and functional characters of the IBD-associated microbiota (Lloyd-Price et al., 2019). Furthermore, these developments have increased interest in microbiota-based approaches such as dietary modification, probiotics, prebiotics, faecal

microbiota transplantation and other microbiota-targeted treatments (Lavelle & Sokol, 2020; Sartor & Wu, 2017).

Despite major progress, important questions persist regarding the precise relationship of dysbiosis with IBD. It is still difficult to know if specific microbial changes cause inflammation, are induced by inflammation, or are the result of the interaction between genetic and environmental factors (Ni et al., 2017; Ananthakrishnan et al., 2018). Therefore, understanding the interactions between gut microorganisms, microbial metabolites, intestinal barrier function and host immunity is important for the discovery of new diagnostic and therapeutic opportunities.

Therefore, this review focuses on the dysbiosis of gut microbiota in inflammatory bowel disease and the microbial alterations, mechanisms involved in disease pathogenesis, the role of microbial metabolites and intestinal barrier dysfunction, and novel microbiota-targeted treatments. Also discussed are current challenges and future perspectives in microbiota-based management of IBD.

II. IMPORTANT BENEFICIAL MICROBES OF THE GUT

In the healthy human intestine, there is a rich diversity of microorganisms that play a role in digestion, metabolism, intestinal barrier function, immune regulation, and microbial homeostasis. Some of the important beneficial or potentially protective bacteria include *Faecali* bacterium, *Bacteroides*, *Lactobacillus* and *Bifidobacterium*. These microorganisms interact with dietary components and other members of the gut microbial community to produce metabolites that influence intestinal health. They can change in abundance and functions in dysbiosis and inflammatory bowel disease (IBD).

➤ *Faecal Bacterium*

Faecal bacterium, and particularly *Faecalibacterium prausnitzii*, are important anaerobic bacterium in the human colon. It is considered one of the important beneficial microorganisms regarding intestinal health. *F. prausnitzii* is involved in the fermentation of dietary substrates and produces butyrate, an important short-chain fatty acid (SCFA). Butyrate is a major energy source for colonic epithelial cells and improves intestinal barrier function. *F. prausnitzii* is also associated with anti-inflammatory effects and modulation of the host immune responses (Sokol et al., 2008; Miquel et al., 2013).

F. prausnitzii has frequently been reported to be decreased in patients with Crohn's disease and other intestinal inflammatory conditions. Its low abundance may lead to a reduction in the production of beneficial metabolites and a disruption of intestinal homeostasis. Therefore, *F. prausnitzii* is considered as an important marker of a healthy microbial environment and has been gaining interest as a potential medicinal microorganism based on microbiota (Sokol et al., 2008; He et al., 2021).

➤ *Bacteroides*

Bacteroides is one of the major bacterial genera in the human colon and has an important role in the metabolism of dietary carbohydrates. These bacteria have a wide range of enzymes which allow them to use complex polysaccharides and dietary fibres that human enzymes can not fully break down. *Bacteroides* produce metabolites like acetate and propionate during fermentation, which might play roles in host metabolic and intestinal processes (Wexler, 2007; Koh et al., 2016).

Bacteroides species also engage in interactions with other gut microorganisms and contribute to the overall microbial food web. Metabolism can influence pH, nutrient availability and the structure of the microbial community in the intestine. Many *Bacteroides* species are normal and beneficial members of the gut microbiota, but changes in their abundance or location can be associated with disease. Thus, the effects depend on species, strain, abundance and the intestinal environment (Wexler, 2007).

➤ *Lactobacillus*

Lactobacillus species are well-characterised lactic acid bacteria, and they have been conventionally linked to beneficial effects in the gastrointestinal tract. They ferment carbohydrates and produce lactic acid, which helps to create an acidic intestinal environment which can limit the growth of some potentially harmful microorganisms. Some lactobacilli are also able to produce other metabolites and antimicrobial substances that contribute to microbial balance (Lebeer et al., 2008).

Lactobacillus species can interact with intestinal epithelial cells and the mucosal immune system. These interactions may contribute to maintenance of epithelial barrier function and modulation of inflammatory responses. These properties have prompted the investigation of selected strains of *Lactobacillus* as probiotics for the support of intestinal health and the management of some gastrointestinal disorders. However, probiotic effects are generally strain-specific, and not all *Lactobacillus* species or strains exert the same biological effects (Lebeer et al., 2008).

➤ *Bifidobacteria*

Bifidobacterium is a group of helpful bacteria that are common in the human gut. These bacteria are particularly important in carbohydrate metabolism, and they ferment dietary carbohydrates to produce organic acids, mainly acetate and lactate. *Bifidobacteria* have metabolic activities that contribute to the intestinal microbial environment and may be involved in cross-feeding interactions with other bacteria (O'Callaghan & van Sinderen, 2016).

Bifidobacterium species also have interactions with the intestinal epithelial cells and the immune system. They might promote the integrity of the intestinal barrier and modulate mucosal immune responses. A healthy abundance of bifidobacteria is generally related to the balance of intestinal microbes, and changes in their abundance have been reported in several gastrointestinal disorders. Therefore, *Bifidobacterium* is extensively studied as a probiotic

candidate and a microbiota-supporting microorganism (O'Callaghan & van Sinderen, 2016).

III. MAJOR FUNCTIONS OF BENEFICIAL GUT BACTERIA

➤ *Complex Carbohydrates Digestion*

Many complex carbohydrates and dietary fibres cannot be fully digested by human digestive enzymes. Thus, because of microbial fermentation, gut micro-organisms are important in their digestion. Other anaerobic bacteria such as *Bacteroides*, *Bifidobacterium* and *Faecalibacterium* possess enzymes that help to utilise different dietary carbohydrates. This microbial fermentation transforms normally indigestible substrates into metabolites that can be consumed by the host or other microorganisms (Wexler, 2007; O'Callaghan & van Sinderen, 2016). Thus, the gut microbiota enhances the capacity of the human gastrointestinal system to digest and recover energy and nutrients from dietary components.

➤ *Short-Chain Fatty Acid Formation*

One of the most important functions of gut microorganisms is the production of short-chain fatty acids, principally acetate, propionate and butyrate, via fermentation of dietary fibre and other carbohydrates. Different bacterial groups produce different SCFA and microorganisms can interact through cross-feeding processes (Koh et al., 2016; Mukhopadhyaya & Louis, 2025).

Butyrate is particularly important because it is a major energy source for colonocytes and contributes to the maintenance of the epithelial barrier. SCFAs also act as signalling molecules that influence metabolism, immune responses and intestinal homeostasis (Koh et al., 2016; Mann et al., 2024).

➤ *Intestinal Barrier Protection*

The gut microbiota and its metabolites are beneficial for maintaining the intestinal epithelial barrier. The barrier prevents excessive contact of luminal microorganisms with host tissues. SCFAs, particularly butyrate, can support epithelial cell metabolism and affect tight-junction proteins and other barrier function components (Ma et al., 2022; Mann et al., 2024). In this regard, *Faecalibacterium prausnitzii* is of particular importance as its metabolites and other microbial products have been associated with maintenance of epithelial integrity and reduction of inflammatory responses (Sokol et al., 2008; Miquel et al., 2013).

➤ *Immune Response Regulation*

The gut microbiota is in constant contact with the intestinal immune system. The beneficial microorganisms along with their metabolites may help regulate immune responses and promote proper immune tolerance. SCFAs can modulate immune cells via receptor-mediated signalling and other molecular pathways and may contribute to the balance between inflammatory and regulatory immune responses (Round & Mazmanian, 2009; Mann et al., 2024). This interaction is particularly important in IBD because disruption of the normal relationship between the

microorganisms and the immune system may contribute to ongoing intestinal inflammation.

➤ *Maintaining Intestinal Microecological Balance*

Desired microorganisms improve microbial homeostasis by competing for nutrients and ecological niches with potentially detrimental microorganisms. They also excrete metabolites and other products that affect the growth and activity of nearby microorganisms. These interactions help to promote microbial diversity and stability of the intestine (Round & Mazmanian, 2009; Koh et al., 2016).

Dysbiosis is a disruption of microbial balance that results from a decrease in beneficial microorganisms and an increase in potentially harmful microorganisms. In IBD, reductions in specific beneficial bacteria, such as *Faecalibacterium prausnitzii*, and alterations in other microbial populations have been linked to changes in intestinal metabolism, barrier dysfunction, and inflammation (Sokol et al., 2008; Schirmer et al., 2019).

Table 1 Maintaining Intestinal Microecological Balance

Beneficial microorganism	Major function
<i>Faecalibacterium</i>	Butyrate production, barrier support, anti-inflammatory effects
<i>Bacteroides</i>	Complex carbohydrate degradation and SCFA production
<i>Lactobacillus</i>	Lactic acid production, microbial balance, epithelial and immune interactions
<i>Bifidobacterium</i>	Carbohydrate fermentation, acetate/lactate production, immune and barrier support

IV. DYSBIOSIS OF THE GUT MICROBIOTA

Gut microbiota dysbiosis is defined as an imbalance in the composition, diversity or function of the microorganisms that live in the gastrointestinal tract. These alterations may be represented by a reduction of beneficial microorganisms, an increase of potentially harmful or inflammation-associated microorganisms, or changes in microbial metabolic activities. Dysbiosis may influence the interaction between the microorganisms and the intestinal epithelium and immune system. It is increasingly being recognised as a key feature of several gastrointestinal diseases, including inflammatory bowel disease (IBD) (Kostic et al., 2014; Schirmer et al., 2019).

including some strains of *Escherichia coli*, have been reported to increase in intestinal inflammation. Some strains such as adherent-invasive *E. coli* have been studied in particular with reference to Crohn's disease. However, the presence of a microorganism does not always imply that it is harmful, its effects depend on the species, strain, abundance and intestinal environment (Sartor & Wu, 2017; Schirmer et al., 2019).

➤ *Microbial Diversity Loss*

In a healthy gut, there is a diverse microbial community and different microbes have complementary roles. In IBD, microbial diversity is often reduced and the relative abundance of certain bacterial groups may change. Such alterations may impact microbial stability and the generation of beneficial metabolites. Alterations in microbial diversity and community structure have been associated with Crohn's disease and ulcerative colitis (Halfvarson et al., 2017; Schirmer et al., 2019).

➤ *Modification of Microbial Metabolites*

Dysbiosis may also affect the production of metabolites produced by gut bacteria. A reduction in the number of fibre-fermenting and SCFA-producing bacteria could lead to a decreased production of acetate, propionate and butyrate. These metabolites are involved in the function of intestinal epithelium, energy metabolism and immune regulation. Other microbial metabolites such as those derived from bile acids and tryptophan may also contribute to intestinal homeostasis and inflammation (Koh et al., 2016; Lavelle & Sokol, 2020).

➤ *Loss of beneficial microorganisms*

Dysbiosis is often associated with a decreased number of microorganisms involved in intestinal homeostasis. Important examples are *Faecalibacterium*, *Bifidobacterium*, *Lactobacillus* and other butyrate-producing bacteria. Reduced levels of *Faecalibacterium prausnitzii* have been described in particular in patients with Crohn's disease. These microorganisms are involved in SCFA production, epithelial barrier function, and immune regulation, and their reduction may have a negative impact on intestinal health (Sokol et al., 2008; Miquel et al., 2013).

➤ *Impaired Intestinal Barrier*

The intestinal barrier prevents excessive exposure of underlying tissues to the microorganisms and substances present in the gut lumen. Dysbiosis and impaired production of beneficial microbial metabolites can affect epithelial integrity and tight-junction function. Increased intestinal permeability may allow for increased interactions between microbial products and host immune cells and potentially contribute to inflammatory responses (Dalal & Chang, 2014; Lavelle & Sokol, 2020).

➤ *Rise in Harmful Microorganisms*

Dysbiosis can also be characterised by an increase in potentially harmful or inflammation-associated microorganisms. Some members of the Proteobacteria,

➤ *Atypical Immune Response*

The gut microbiota is important for the regulation of intestinal immune system. Changes in the composition of the microbiota and in microbial products can influence immune signalling and the balance between inflammatory and regulatory responses. In susceptible individuals, these changes may contribute to sustained mucosal inflammation which is a key characteristic of IBD (Round & Mazmanian, 2009; Matsuoka & Kanai, 2015).

➤ *Reasons for Gut Microbiota Dysbiosis*

Diet, medications, antibiotic exposure, lifestyle, environmental factors, age and host genetic background are some of the factors that can impact the gut microbial composition. A low dietary fibre and a high highly processed food diet could affect the microbial composition and metabolic activity. Antibiotics can also cause major changes in the microbial community. These factors may interact with genetic susceptibility and immune responses to impact the intestinal microbiota in IBD (Ananthakrishnan et al., 2018; Kostic et al., 2014).

➤ *Dysbiosis and IBD: A Self-Perpetuating Cycle*

The relationship between IBD and dysbiosis is complex. Microbial imbalance may lead to epithelial barrier dysfunction, altered metabolite production and abnormal immune activation. Meanwhile, inflammation in the intestine can change the intestinal environment and further change the microbial community. Thus, dysbiosis and inflammation can reinforce each other and form a self-perpetuating cycle (Ni et al., 2017; Sartor & Wu, 2017).

V. CHANGES IN MICROBIOME IN INFLAMMATORY BOWEL DISEASE

Inflammatory bowel disease (IBD) is associated with marked changes in the composition and function of the gut microbiota. Collectively, these changes are termed dysbiosis and include decreased microbial diversity, loss of beneficial microorganisms, expansion of potentially harmful bacteria, and changes in microbial metabolism. These changes are often observed in both Crohn's disease and ulcerative colitis, although the exact pattern varies between patients, and may be involved in intestinal inflammation and disease progression (Manichanh et al., 2012; Halfvarson et al., 2017; Schirmer et al., 2019).

➤ *Decrease in Microbial Diversity:*

A reduction in microbial diversity is an important characteristic of IBD-associated dysbiosis. In IBD patients, decreased bacterial richness and alterations in the relative abundance of different microbial groups can be observed. Reduced diversity can impact the stability and functional capacity of the gut microbial community and may decrease its capacity to maintain intestinal homeostasis (Halfvarson et al., 2017; Schirmer et al., 2019).

➤ *Loss of beneficial bacteria:*

In IBD, beneficial anaerobic bacteria are depleted. Important examples include *Faecalibacterium prausnitzii* and *Ruminococcaceae*. These microbes are involved in dietary fibre fermentation and short-chain fatty acid (SCFAs) production, especially butyrate. These bacteria may produce beneficial metabolites, and their reduced abundance may compromise intestinal barrier integrity and immune regulation (Sokol et al., 2008; Lavelle & Sokol, 2020).

➤ *Development of Potentially Pathogenic Bacteria:*

IBD may also be associated with an increase in potentially harmful or inflammation-associated microorganisms. Inflammation of the intestines can lead to an

increase in the number of Enterobacteriaceae, which includes some strains of *Escherichia coli* and *Klebsiella*. Certain strains of *E. coli*, particularly adherent-invasive *E. coli*, have been linked to Crohn's disease and are capable of interacting with intestinal epithelial cells and eliciting inflammatory responses (Sartor & Wu, 2017; Schirmer et al., 2019).

➤ *Translocation of Oral Microbes to the Gut:*

Another interesting feature of IBD-associated dysbiosis is the increased detection of microorganisms that are normally more prevalent in the oral cavity. Some bacteria such as *Fusobacterium nucleatum* and *Veillonella* are increased in the gut microbiota of some individuals with IBD. The intestinal environment, such as inflammation and alteration of the mucosal barrier, might favour the survival and colonisation of these microorganisms in the gut (Sokol et al., 2017; Schirmer et al., 2019).

➤ *Short-Chain Fatty Acid Production Decrease:*

Microbial community composition changes can also have important functional and metabolic consequences. Reduced fibre-fermenting and SCFA-producing bacteria can reduce the production of acetate, propionate and butyrate. Butyrate is especially important as it is an energy source for colonic epithelial cells and contributes to the maintenance of the intestinal barrier and regulation of immune responses (Koh et al., 2016; Lavelle & Sokol, 2020).

➤ *Modifications of the Intestinal Oxygen Environment:*

The normal colon is a relatively low oxygen environment which favours growth of obligate anaerobic bacteria. Changes in epithelial metabolism and tissue oxygenation induced by intestinal inflammation may increase oxygen availability in the gut lumen. This new environment may be harmful to some beneficial anaerobes and beneficial to facultative anaerobic and oxygen tolerant bacteria including members of the Enterobacteriaceae family. Inflammation and dysbiosis may be mutually reinforcing and contribute to a continuing cycle of intestinal inflammation (Rigottier-Gois, 2013; Sartor & Wu, 2017).

VI. ROLE OF ANAEROBIC AND AEROBIC BACTERIA

The healthy colon is an environment of low oxygen tension, favouring the proliferation of many beneficial obligate anaerobic bacteria. Important anaerobic microorganisms ferment dietary fibre, producing short-chain fatty acids, maintaining the intestinal barrier, and balancing the microbiota (Rigottier-Gois, 2013; Koh et al., 2016).

During the inflammation associated with IBD, the metabolic state of the intestinal tissue and the availability of oxygen can increase the oxygen levels in the gut environment. This may inhibit the growth of some oxygen-sensitive anaerobes and favour the growth of oxygen-tolerant and facultative anaerobes, including Enterobacteriaceae. These changes might result in microbial dysbiosis and may also promote intestinal inflammation (Rigottier-Gois, 2013; Sartor & Wu, 2017).

Low oxygen levels in a healthy intestine promote the growth of beneficial anaerobic bacteria. These bacteria help to keep the microbial balance and intestinal health.

In IBD, inflammation increases oxygen availability to the intestinal environment. This may lead to a reduction of the beneficial anaerobic bacteria and to an increase in the oxygen-tolerant bacteria. This dysbiosis can lead to further inflammation, creating a vicious cycle of disease.

➤ *In Simple Terms:*

Healthy gut → Low oxygen → Good anaerobes → Balance

IBD → Inflammation → More oxygen → Oxygen tolerant bacteria → Dysbiosis → More inflammation

VII. MECHANISMS LINKING DYSBIOSIS TO INFLAMMATION

Gut microbiota dysbiosis could promote intestinal inflammation through several interconnected mechanisms, including disruption of the intestinal barrier, microbial translocation, metabolite imbalance, immune activation, and expansion of inflammation-associated bacteria. These processes can interact with each other, creating a cycle in which dysbiosis causes inflammation, which, in turn, further modifies the gut microbiota (Kostic et al., 2014; Lavelle & Sokol, 2020).

➤ *Intestinal Barrier Disruption*

Dysbiosis can affect the intestinal epithelial barrier by altering the integrity of tight junctions and the protective mucus layer. Fewer beneficial microorganisms and changes in mucus-associated bacteria can increase intestinal permeability and lead to more direct contact of microbial components with intestinal tissues and immune cells (Dalal & Chang, 2014; Lavelle & Sokol, 2020).

➤ *Microbial Translocation & Immune Activation*

When the intestinal barrier is compromised, microbial products such as lipopolysaccharide (LPS) can more easily reach the host's immune receptors. LPS can activate Toll-like receptor 4 (TLR4) and downstream inflammatory signalling pathways such as NF- κ B, which leads to the production of inflammatory mediators such as TNF- α , IL-6, and IL-1 β . This can lead to chronic inflammation of the intestines (Sartor & Wu, 2017).

➤ *Imbalance of Metabolites*

Dysbiosis may alter the production of important microbial metabolites. Reduced production of short-chain fatty acids (SCFAs), especially acetate, propionate and butyrate, can result from a reduction in fibre-fermenting bacteria. Butyrate is particularly important for colonic epithelial cells and participates in the maintenance of the intestinal barrier and immune regulation (Koh et al., 2016; Lavelle & Sokol, 2020).

➤ *Dysregulated Immune Activation*

Changes in the microbial composition and microbial products may disrupt normal communication between the microbiota and the intestinal immune system. Increased exposure to microbial molecules can activate immune cells and amplify inflammatory signalling. In susceptible individuals, dysregulation of these responses may promote chronic intestinal inflammation (Round & Mazmanian, 2009; Matsuoka & Kanai, 2015).

➤ *Bacterial Proliferation Related to Inflammation*

The intestinal environment can be itself altered by inflammation. The availability of oxygen and nitrate may change and give a growth advantage for facultative anaerobic bacteria, especially for members of the Enterobacteriaceae family. So, bacteria such as *Escherichia coli* and other Enterobacteriaceae may increase while oxygen-sensitive beneficial anaerobes decrease (Rigottier-Gois, 2013; Hughes et al., 2017).

➤ *Intestinal Inflammation (Chronic)*

Reduced beneficial metabolites, impaired barrier function, increased microbial stimulation and abnormal immune responses can sustain chronic intestinal inflammation in combination. Inflammation can in turn alter the intestinal environment and worsen microbial dysbiosis, thereby creating a self-perpetuating cycle (Ni et al., 2017; Sartor & Wu, 2017).

VIII. DYSBIOSIS AND IBD PATHOGENESIS

Gut microbial dysbiosis is believed to be an important factor in the pathogenesis of inflammatory bowel disease (IBD). These include changes in microbial diversity, composition and function, for example, reduction of beneficial microorganisms and expansion of potentially harmful or inflammation-associated microorganisms. These changes may affect the integrity of the intestinal barrier, microbial metabolism as well as host immune responses, thereby contributing to intestinal inflammation (Kostic et al., 2014; Schirmer et al., 2019).

➤ *Diminution of Microbial Diversity*

Common features in IBD patients include a decrease in microbial diversity and an altered microbial community structure. A decrease in microbial richness and evenness may lead to a decrease in functional stability of the intestinal ecosystem. These changes can impact the metabolism of nutrients, the production of microbial metabolites that are beneficial and the resistance to micro-organisms that may be harmful (Halfvarson et al., 2017; Schirmer et al., 2019).

➤ *Microbial Compositional Changes*

The dysbiosis of IBD is characterised by a reduction of beneficial bacteria and, in particular, of SCFA-producing bacteria such as *Faecalibacterium prausnitzii*. Meanwhile, inflammation-associated bacteria may proliferate, including some strains of *Escherichia coli* and other Enterobacteriaceae. These kinds of changes can disturb normal microbial functions and contribute to intestinal inflammation (Sokol et al., 2008; Sartor & Wu, 2017).

It's not only bacteria that are evolving. Changes in the gut mycobiome and virome have also been associated with IBD, suggesting that interactions between bacteria, fungi, viruses and the host may also be implicated in intestinal health and disease (Iliev & Cadwell, 2021).

➤ *Dysfunction of the Intestinal Barrier*

The intestinal epithelial barrier forms a barrier between the intestinal lumen and the underlying tissues. Dysbiosis can cause damage to the mucus layer, epithelial cells, and tight-junction integrity, leading to increased intestinal permeability. A damaged barrier allows more interaction between microbial products and host immune cells, and may lead to inflammatory responses (Dalal & Chang, 2014; Lavelle & Sokol, 2020).

➤ *Dysregulation of the Immune System*

Microbial products can bind to pattern-recognition receptors (including Toll-like receptors) on intestinal epithelial and immune cells. This interaction can activate inflammatory signalling pathways, such as NF- κ B, and increase the production of inflammatory cytokines. The balance of pro-inflammatory and regulatory immune responses may be disturbed, leading to sustained inflammation in vulnerable individuals (Round & Mazmanian, 2009; Matsuoka & Kanai, 2015).

➤ *Metabolic Changes*

Dysbiosis may lead to decreased microbial fermentation of dietary fibre, and consequently decreased production of beneficial SCFAs, particularly butyrate. Butyrate maintains the integrity of the intestinal barrier and immune regulation and supports colonic epithelial cells. Therefore, decreased SCFA production might contribute to impaired intestinal homeostasis and inflammation (Koh et al., 2016; Lavelle & Sokol, 2020).

➤ *Dysbiosis and Inflammation: A Vicious Cycle*

The relationship between dysbiosis and IBD is not unidirectional. Microbial dysbiosis may be a factor in barrier dysfunction and immune activation, and active intestinal inflammation can change the gut environment, including oxygen availability and nutrient status. These changes may promote the growth of facultative anaerobic and inflammation-associated bacteria, which can further disrupt the microbial community (Rigottier-Gois, 2013; Ni et al., 2017).

IX. THERAPEUTIC PERSPECTIVES

Rebalancing the gut microbes is a new frontier in the management of inflammatory bowel disease (IBD). Dysbiosis is associated with changes in the microbial composition, a decrease in beneficial metabolites, impaired intestinal barrier function and abnormal immune responses. Thus, several approaches have been investigated to modify the gut microbiota and improve intestinal health (Sartor & Wu, 2017; Lavelle & Sokol, 2020).

➤ *Probiotics*

Probiotics are live micro-organisms which, when administered in adequate amounts confer a health benefit on the host. Some strains of probiotics, such as some *Lactobacillus* and *Bifidobacterium* species, may help to support microbial balance, intestinal barrier function and immune regulation. However, their effects can differ depending on the bacterial strain, disease type, and individual patient, and thus probiotics cannot be considered equally effective for all forms of IBD (Sanders et al., 2019; Sartor & Wu, 2017).

➤ *Prebiotics*

Prebiotics are substrates that are selectively utilised by host microorganisms. Dietary fibres and other prebiotic compounds can stimulate the growth and activity of beneficial bacteria and may enhance production of SCFAs such as butyrate, acetate and propionate. These effects may contribute to the intestinal barrier function and microbial homeostasis through prebiotics (Gibson et al., 2017; Koh et al., 2016).

➤ *Dietetic Intervention*

Diet is a major driver shaping the composition and function of the gut microbiota. A diet high in dietary fibre from vegetables, fruits, whole grains and other plant foods can provide substrates for microbial fermentation and SCFA production. In contrast, diets high in highly processed foods and low in fibre may have negative effects on microbial diversity and metabolic activity. Thus, dietary patterns may play a role in a healthier intestinal microbial environment, but dietary recommendations should be individualised in IBD patients (Ananthakrishnan et al., 2018; Lavelle & Sokol, 2020).

➤ *Faecal Microbiota Transplantation*

Faecal microbiota transplantation (FMT) is the transfer of processed faecal material from a healthy donor to the gastrointestinal tract of a patient. The aim is to introduce a broader microbial community and to change the intestinal microbiota of the patient. FMT has shown promising results especially in ulcerative colitis but the treatment protocols, donor selection, safety and long-term efficacy need further exploration (Paramsothy et al., 2017; Costello et al., 2019).

➤ *Therapies Targeting the Microbiota*

New therapeutic approaches targeting specific microorganisms, microbial metabolites or host-microbe interactions are being developed. These include methods to increase beneficial bacteria, alter the metabolic pathways of the microbes, restore SCFA production, and target microbial signals that control intestinal immunity. These approaches could ultimately enable a more accurate and personalised treatment of IBD (Lavelle & Sokol, 2020; Sartor & Wu, 2017).

X. CONCLUSION

The gut microbiota is fundamental to maintaining the integrity of the intestinal barrier, microbial composition, metabolism and immune homeostasis. A healthy microbial

population ensures normal intestinal function and protects the host from excessive inflammation. Conversely, gut dysbiosis may interfere with the intestinal ecology, with an increase in bacteria associated with inflammation and a decrease in beneficial microorganisms and metabolic products. These changes may cause chronic intestinal inflammation, damage to the intestinal barrier, and abnormal immune activation. Understanding these interrelationships may help to develop novel microbiota-based therapies for the management and prevention of IBD.

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