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Microbiome and Inflammatory Bowel Disease: A Narrative Review

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ABSTRACT

The intestinal microbiome plays a central role in the pathogenesis of inflammatory bowel disease (IBD), which includes Crohn's disease and ulcerative colitis. Dysbiosis, characterized by reduced microbial diversity, expansion of pro-inflammatory taxa, and depletion of protective commensals such as Faecalibacterium prausnitzii, is a hallmark of IBD and contributes to chronic intestinal inflammation. Altered microbiota composition influences immune regulation, barrier function, and metabolic activity, perpetuating the cycle of inflammation and mucosal injury. Environmental factors, including diet, antibiotics, and lifestyle, further modulate microbial ecosystems, shaping disease onset and progression. Therapeutic strategies to restore microbial balance, such as dietary interventions, probiotics, prebiotics, and fecal microbiota transplantation (FMT), show promise as adjuncts to conventional pharmacological treatments. Advances in metagenomics, metabolomics, and multi-omics integration are driving personalized approaches to IBD care, yet challenges remain in standardization, reproducibility, and ethical considerations. This review summarizes current evidence on microbiome alterations in IBD, underlying mechanisms, therapeutic strategies, and the potential for microbiome-targeted precision medicine.

Keywords: Inflammatory bowel disease**,** Gut microbiome**,** Dysbiosis**,** Probiotics and FMT, and Precision medicine.

INTRODUCTION

The microbiome exerts a pivotal role in the pathogenesis of inflammatory bowel disease. Intestinal inflammation correlates with dysbiosis that entails decreased biodiversity and an imbalance between beneficial and harmful microbial species. Dietary components influence commensal microbiota; nutritional interventions are proposed to restore biodiversity and counteract dysbiosis. Strategies designed to selectively modulate microbiota composition include prebiotics, antibiotics, probiotics, and faecal microbiota transplantation [1]. This narrative review discusses microbiome composition, underlying mechanisms influencing IBD, current experimental approaches, and the potential for innovative and targeted strategies to modify IBD relapse and remission [1].

Understanding the Microbiome

The microbiome, comprising all organisms residing within the human body, outnumbers host cells by approximately tenfold [2]. The microbiota refers to the collective communities of these microbes across diverse body sites. Characterizing this community solely by composition obscures variations in gene content and functionality between individuals; therefore, the microbiome denotes the entire habitat, encompassing microorganisms, their genomes, and environmental conditions [2]. The human gut microbiome consists of approximately 150 times more genes than the human genome and is generally dominated by seven bacterial phyla: Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Fusobacteria, Cyanobacteria, and Verrucomicrobia. It inhabits various niches, including the mouth, skin, colon, and vagina. The microbiome's fundamental role in human physiology is increasingly recognized, with perturbations implicated in several diseases, notably inflammatory bowel diseases (IBD) such as Crohn's disease (CD) and ulcerative colitis (UC) [2].

Definition and Composition

The microbiome, a complex assembly of microbial communities residing on and within the human body, includes bacteria, viruses, fungi, and other microorganisms. Particularly dense in the gastrointestinal tract, with an estimated population of approximately 3.8 × 10^13 microbes, the microbiome has evolved together with its host, fulfilling essential physiological functions [3]. The gut, as the primary site of microbial colonization, harbors a diverse array of microorganisms that significantly influence the host's health [3]. The composition of the gastrointestinal microbiota is dynamic, subject to variations throughout the human lifespan, and sensitive to environmental factors such as dietary habits. Despite this variability, a state of virulence equilibrium is maintained, crucial for sustaining intestinal homeostasis and functionality [3].

Role in Human Health

The human microbiome comprises microorganisms, including bacteria, fungi, and viruses, that coexist with the human host within different anatomical sites. Commensal microorganisms interact with host tissues and influence local biology [2, 3, 4]. The microbiome influences cellular interactions and signalling, modulates immune responses, provides nutrition, facilitates energy production, metabolises toxins and drugs, and prevents the invasion of pathogens. Pathogenic alterations in the normal microbiome composition or location can have significant consequences and participate in aetiological pathways of disease [2, 3, 4].

Inflammatory Bowel Disease Overview

Inflammatory Bowel Disease (IBD) comprises two primary types: Crohn's Disease and Ulcerative Colitis [2]. Crohn's Disease can affect any part of the gastrointestinal tract, with a preference for the terminal ileum. Ulcerative Colitis, in contrast, is limited to the colon. When Crohn's Disease is confined to the colon, it is sometimes indistinguishable from Ulcerative Colitis and is referred to as indeterminate colitis [2, 5]. Both conditions have a significant impact on the normal structure and function of the gastrointestinal tract. IBD affects an estimated one million individuals in the United States and 2.5 million residents of the European Union. The incidence has increased sharply in the past two decades in developed countries. A variety of environmental and genetic risk factors have been identified as contributing to the disease [5].

Types of Inflammatory Bowel Disease

Inflammatory bowel disease (IBD), a term that encompasses two distinct conditions, Crohn's disease (CD) and ulcerative colitis (UC), is characterized by chronic intestinal inflammation. An estimated 1.4 million residents of the United States and 2.2 million inhabitants of Western Europe are affected by IBD [2]. Both diseases follow a relapsing and remitting course, with no known cure. Although the etiology is multifactorial and not yet fully elucidated, it is generally accepted that, in genetically predisposed individuals, one or several environmental factors trigger an abnormal immune response to the endogenous intestinal microbiota [4]. Despite similarities regarding clinical manifestations, genetic susceptibility, and inflammatory pathways, CD and UC represent two distinct pathological entities, displaying characteristic differences in macroscopic, histological, and immunological features. The types of intestinal inflammation that can be distinguished are described below, together with an account of other diseases that must be considered in the differential diagnosis [2, 4].

Epidemiology and Risk Factors

The incidence of IBD is increasing globally. The highest incidence rates are observed in Western countries, but rates are also rising in Asia and other regions that have previously been classified as low-incidence 4. The most common risk factors for IBD include genetic predisposition, smoking, ethnicity, and an extended period of oral contraceptive use [1]. Genetic factors have an important role in the onset of IBD. More than 230 gene loci have been identified as potentially linked to IBD susceptibility. Both UC and CD increase the risk of colorectal cancer, but for UC, the risk is much higher; 8% of UC patients develop colorectal cancer within 20 years of diagnosis. CD patients suffering from complications such as fistulas or strictures typically have a worse prognosis [1].

Microbiome Alterations in IBD

Interest in the human microbiome increased over the past decade, and the impact of microbes on global human health has become a focal point of scientific research [6]. The Human Microbiome Project (HMP) was launched in 2008 with the intent to characterize the microbial communities inhabiting the human body and advance understanding of the human microbiome in the maintenance of health and development of disease. Decades before, observations were made concerning the importance of the gut microbes to human health and the potential for bacteria to be at the center of disease [6]. Humans are a mix of both bacterial cells and human cells, with the gut microbiome thought to be composed of over 1,000 species and 7,000 distinct strains of bacteria [7]. The microbiome consists of variable groups of organisms, bacteria, fungi, protozoa, and viruses, where prevalence and abundance change throughout differentiated sections of the intestinal tract and are influenced by genetic and environmental factors. With regard to human health, the gut microbiome is particularly important because of its

widespread influence in fundamental functions such as energy metabolism, intestinal barrier integrity, angiogenesis, immunoregulation, and protection against pathogens [2]. In the United States, the prevalence of inflammatory bowel diseases (IBD) is estimated at approximately 1.3%, representing nearly 3 million individuals, and according to the Centers for Disease Control and Prevention (CDC), 2015 estimates, approximately 1.0% of adults in the United States have been diagnosed with Crohn's disease or ulcerative colitis. Both Crohn's disease and ulcerative colitis are characterized by chronic, waxing and waning inflammation of the gastrointestinal tract. While the pathogenesis of IBD remains unknown, environmental factors, including diet, cumulative exposure to antibiotics, infectious gastroenteritis, psychosocial stress, and genetic risk factors, are all believed to influence disease onset and progression [2, 7].

Dysbiosis and Its Implications

Dysbiosis is a microbial imbalance in the gut, associated with impaired barrier function, inflammation, and diseases like IBD, diabetes, and cardiovascular conditions [7]. It involves loss of beneficial microbes, expansion of pathobionts, and reduced diversity, though it remains uncertain whether these changes cause or result from IBD. The gut microbiome comprises bacteria, archaea, fungi, and viruses, all vital to host physiology; IBD alters this microbial consort and influences related immune pathways. Bacterial dysbiosis in IBD features diminished biodiversity and imbalanced composition, with declines in beneficial taxa varying between active and inactive disease states [7]. The pathogenesis of IBD stems from interactions among gut microbiota, the immune system, genetic factors, and the environment. An altered microbiota contributes critically to disease development [5]; although recent molecular advances link dysbiosis to IBD, precise roles and causal directions remain unclear. Dysbiosis and metabolic dysfunction potentiate inflammation; microbial influences on key metabolic pathways affect IBD progression, suggesting that microbiota-targeted interventions may help restore gut homeostasis [5, 7].

Specific Microbial Changes in IBD

In inflammatory bowel disease (IBD), alterations occur in the gut microbial composition, termed dysbiosis, which manifests as an imbalance between protective and harmful bacterial species [7]. Dysbiosis generally coincides with shifts in the abundance of three microbial groups, accompanied by a reduction in species diversity [8]. The gut ecosystem of patients with IBD contains an elevated population of pro-inflammatory organisms and a smaller group of protective genera. Several studies have demonstrated the association of clinical deterioration with an increase in bacteria from the Proteobacteria phylum and a reduction in representatives of the Firmicutes and Bacteroidetes families. The characteristic bacterial community diversity appears lower in subjects with intestinal inflammation. Samples from the inflamed area reveal a decrease in the microbial diversity at the phylum level. A multicentre investigation performed on stool samples from treatment-naive adult patients with Crohn's disease (CD) identified an augmentation of species within the Veillonellaceae, Pasteurellaceae, and Enterobacteriaceae families and a decrease of taxa belonging to the Bacteroidales, Erysipelotrichales, and Clostridioides, with the suggestion that microbial profiling may be useful as an early CD biomarker. IBD-associated dysbiosis correlates with an increase of pro-inflammatory microbes and a reduction of those with anti-inflammatory effects, including those belonging to the Faecalibacterium genus that contributes to gut homeostasis through the butyrate production [7]. The species F. prausnitzii is depleted in IBD, and the low abundance observed after surgery represents a risk factor for relapse. Decreased populations of Blautia faecis, Roseburia inulinivorans, Ruminococcus torques, and Clostridium lavalense represent additional features of the CD microbiome [7, 8].

Mechanisms Linking Microbiome to IBD

Deregulated immune response and altered intestinal barrier function, both impacted by the microbiome, have been implicated in IBD pathogenesis [2]. Gut bacteria influence disease development through direct interaction with the intestinal mucosa 9. IBDs are characterized by inappropriate immune upregulation in genetically susceptible patients; gut microbiota constitute the target of this response due to loss of tolerance or altered microbial diversity and function. Transgenic mice expressing aberrant T cell receptors spontaneously develop colitis in response to normal intestinal microbiota. Bacteria such as adherent-invasive Escherichia coli are proinflammatory, whereas Faecalibacterium prausnitzii exerts protective effects. Elevated abundance of Enterobacteriaceae and Fusobacteria is observed in clinical and experimental IBD [2]. Diversion of the fecal stream induces remission and prevents Crohn's disease recurrence, whereas infusion of intestinal contents reactivates mucosal lesions [7]. Culturing inflamed mucosal samples from active IBD patients with non-pathogenic Escherichia coli stimulates proinflammatory cytokine release, an effect attenuated by co-culture with Lactobacillus casei. A healthy bacterial community regulates immune responses, and its absence correlates with a lack of intestinal inflammation. Combining microbiome data with immune, genetic, psychological, and physiological factors is essential to fully understand microbiome impact on IBD. Gut microbiota stimulates the immune system, and altered composition in

early life can lead to an inadequately trained immune system that overreacts to commensal microbes, precipitating inflammatory diseases [7]. Specific microbial modifications affecting diversity and stability are identified in IBD; such alterations provoke dysregulated mucosal immune responses that initiate disease, as many altered taxa directly impact immune pathways and favor a proinflammatory environment. The functional significance and pathogenic role of these changes remain to be elucidated; the complex interplay among microbiota, intestinal mucosa, and immune system underscores the necessity of a comprehensive approach to unravel the mechanisms underlying intestinal dysbiosis. Revisit "Microbiome Alterations in IBD" for details on microbial changes [7].

Immune Response Modulation

The microbiome plays an important role in the modulation of the immune response. Numerous risk loci and observations suggest that deregulation of the immune system and microbiota interaction promotes chronic intestinal inflammation [10]. In sterile mice, when intestinal Th1 and Th17 decreased, the immune response was controlled by Th2, and this imbalance was improved through colonization of Bacteroides fragilis, indicating the microbiome's role in immune regulation and inflammatory bowel disease (IBD) mitigation. Polysaccharide A from Bacteroides fragilis exerted anti-inflammatory effects by suppressing IL-17 and increasing IL-10 secretion [11]. Gut commensal bacteria produced inflammatory products that enhanced T cell responsiveness, worsening inflammation. Certain Clostridia strains expanded Treg cells, attenuating colitis in mice. Interactions of Lactobacillus acidophilus surface proteins with immune receptors help maintain microbiota balance and protect the mucosal barrier [12]. Manipulating gut microbiota presents a potential approach for treating inflammatory diseases; however, further research is needed on microbes that induce stronger therapeutic responses, are host-compatible, and can precisely modulate the immune system [10]. The immune response in IBD is also modulated by therapies and microbiota. Minocycline has anti-inflammatory effects involving immunomodulatory and antimicrobial properties [12]. Antibiotics such as ciprofloxacin, metronidazole, and rifaximin influence microRNA/mRNA expression in immune cells and inhibit bacterial translocation, protecting against inflammation. Probiotics, including Lactobacillus fermentum, Lactobacillus salivarius, Bifidobacterium longum, and Bifidobacterium infantis, attenuate inflammation by modulating immune pathways and increasing regulatory T cell populations. Escherichia coli Nissle 1917 ameliorates colitis development, and probiotic mixtures like VSL#3 exert diverse effects on intestinal immune parameters, thereby contributing to immune response modulation in IBD [12].

Barrier Function and Intestinal Health

Barrier function represents the main site of interaction between luminal components and the mucosa of the gastrointestinal tract and is a core element of IBD pathogenesis [13]. The intestinal mucosa forms the largest interface for interaction between the host and the external environment; it is constantly exposed to the lumen of the intestine, which contains fluid, nutrients, microorganisms, and microbial products [13]. The intestinal cells must maintain a balance between preserving barrier function and facilitating the exchange of fluid and nutrients with their environment. This is achieved by two major components: the single layer of intestinal epithelial cells (IEC) and the overlying mucus layer. IECs of the intestinal epithelium are connected by several junctional complexes, which regulate the passage of lumen components to the underlying submucosa. The paracellular pathway is the major route for movement between the lumen of the intestine and host tissue. Several different molecules regulate the movement of ions and molecules across the IEC through the paracellular pathway, including tight junction-associated proteins such as claudin, junctional adhesion molecule (JAM), Zona occludens (ZO), and occludin, which all contribute to epithelial barrier function. Barrier maintenance is further challenged by the fact that intestinal epithelial cells are replaced every 4–5 days, which means that any damage to the barrier must be quickly repaired. Loss of barrier function would result in increased exposure of luminal antigens to immune cells populating the submucosa and subsequent uncontrolled immune activation [13].In homeostasis, the mucus layer of the intestinal lumen is dominated by a dense network of polymers called mucins. Mucins serve a number of functions, including maintaining lubrication and protection of the mucosal surface. In addition, mucins provide a matrix for the aqueous phase of the mucus layer and trap secreted antimicrobial molecules (such as immunoglobulin A and defensins, which can kill or immobilize pathogens). The majority of mucins secreted in the small intestine are relatively small and soluble, which allows them to permeate the mucus layer, providing lubrication [13, 14]. The large intestine secretes higher molecular weight mucins that have different physical properties 14. In addition, the glycosylation pattern of mucins varies across the length of the intestine, and this can influence bacterial adhesion to the mucosal surface. The composition of microbiota can influence the glycosylation pattern of mucins, and thus may affect the bacterial population and activity. Alteration of intestinal permeability can lead to disease, but increased permeability of the small intestine is a hallmark of both Crohn's disease and ulcerative colitis, with active disease characterized by a more significant increase in permeability than those with inactive disease [14].

Diet and the Microbiome in IBD

The impact of diet on the microbial environment in inflammatory bowel disease (IBD) is a subject of active investigation. Dietary intake is a critical determinant of skin and gut microbial diversity. In patients with IBD, specific dietary patterns are associated with an increased risk of developing the condition [15]. Human epidemiological data and animal models of colitis have shown that dietary modulation leads to several characteristic features of dysbiosis. Consequently, diet-based interventions have been proposed as an adjunct strategy to alter the intestinal microbial composition in IBD [15]. The incidence of IBD is increasing, affecting approximately 2.5 million people in the U.S., imposing a significant economic burden. The pathophysiology of IBD involves dysbiosis, epithelial barrier dysfunction, and immune system disturbances. Conventional treatment options have limited success in maintaining long-term remission and preventing flare-ups; moreover, many patients develop tolerance [15]. Microbial dysbiosis has been linked to epithelial barrier dysfunction and intestinal inflammation. Diet emerges as an attractive microbiome-targeted therapy due to its modifiable, noninvasive, and inexpensive nature. Dietary patterns are associated with an increased incidence of IBD and characteristic dysbiosis, while dietary modulation of the microbial milieu offers benefits as an adjunct therapy. Studies in animal models and preliminary clinical intervention trials have demonstrated that dietary changes, in combination with medication, can induce remission in IBD and are well tolerated [15]. IBD represents a group of idiopathic disorders characterized by chronic inflammation of the intestinal mucosa. Ulcerative colitis and Crohn's disease are the two main clinical manifestations, exhibiting wide geographical and seasonal heterogeneity. The prevalence of IBD has been increasing globally over the past 50 years. Alongside genetic predisposition, environmental factors, such as diet, smoking, hygiene, stress, sleep, and antibiotic usage, contribute to the onset of IBD [16].

Impact of Diet on Microbial Diversity

Diet represents a major exposure shaping gut microbial variation. Beyond the influence of geographical and cultural backgrounds, deep metagenomic sequencing has begun to reveal the extent to which habitual long-term dietary patterns are associated with the gut microbiome. Several dietary patterns, including legumes, breads, fish, and nuts, appear to be associated with a marked reduction in the abundance of opportunistic bacteria and in the inflammatory markers C-reactive protein (CRP) and leucocytes. Additionally, high-level consumption of nuts, oily fish, fruits, vegetables, cereals, and red wine correlates with higher levels of the core microbial genus Roseburia and other potentially health-promoting genera such as Faecalibacterium and Eubacterium [17]. Polyphenol-rich foods, including coffee, tea, red wine, and to some extent fruit, display a similar anti-inflammatory profile in the gut microbiome, showing a depletion in inflammation-linked markers and enrichment in potentially beneficial bacteria. Coffee consumption correlates with a higher relative abundance of Oscillibacter, while red wine intake is linked to increased abundance of acetate, propionate, and butyrate producers, as well as key SCFAs in the gut. Moderate red wine consumption is further associated with an increase in overall microbial diversity, both in terms of species richness and Shannon diversity, whereas total alcohol intake is significantly linked to pro-inflammatory pathways, with spirits appearing to be the main driver of this association [17]. Moreover, plant protein intake associates with an overall increase in indicators of anti-inflammatory fermentation, whereas animal-derived protein correlates with a lower abundance of the potentially probiotic genus Bifidobacterium, suggesting a potential mechanism by which high intake of animal protein might adversely affect the host. In human intervention studies, the composition of the gut microbiome can change rapidly following dietary shifts. Dietary modifications have become an increasingly attractive therapeutic option for IBD patients, aiming at restoring gut microbiota balance. One intervention leading to the remission of pediatric CD is the exclusive enteral nutrition, during which patients are provided with a liquid formula diet for 6–8 weeks and are progressively reintroduced to a normal diet. Clinical response to exclusive enteral nutrition treatment coincides with compositional changes in fecal bacteria, while a recurrent composition is observed following the reintroduction of a normal diet [17].

Specific Dietary Interventions

The microbiome comprises trillions of microorganisms whose composition varies between individuals and different anatomical sites, with the digestive tract hosting 70% of the total population (e.g., Bacteroidetes, Firmicutes, Proteobacteria) [18, 19]. These microbes contribute fundamentally to all aspects of human physiology. When present in physiologic amounts and proportions, the microbiome confers important benefits to the host [15]. Dietary interventions aim to modulate the intestinal microbiome and its metabolic environment to promote remission or prevent relapse in inflammatory bowel disease (IBD). Several diets have been developed specifically for the nutritional management of patients with IBD, including the specific carbohydrate diet (SCD), the low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diet and its modified version, the Crohn's disease exclusion diet (CDED), the IBD anti-inflammatory diet (IBD-AID), and the Mediterranean diet. However, despite strong anecdotal evidence, the lack of randomized controlled trials and the

difficulties associated with compliance and duration of the intervention have limited the potential of these dietary interventions [18]. Alternatively, exclusive enteral nutrition (EEN), which consists of a liquid formulation given orally or by nasogastric tube, emerged as an option for the induction of remission and treatment of the acute phase of active disease. EEN has been established as the preferred treatment over corticosteroids for the induction of remission in children with active luminal Crohn´s disease (CD) and is recommended as a first-line therapy according to the European Society of Pediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) and the European Crohn´s and Colitis Organisation (ECCO). Despite its short-term efficacy, the main drawbacks of EEN are a lack of palatability, poor compliance, and low tolerability [19].

Therapeutic Approaches Targeting the Microbiome

Apart from dietary interventions, other therapeutic strategies have been implemented to modify gut microbiota, targeting the stabilization of microbial communities at the mucosal and luminal interfaces [20]. Probiotics and prebiotics have been widely studied to treat microbial dysbiosis and exhibit anti-inflammatory effects [2]. Hence, these two have been implicated in improving remission in IBD as adjuvants to evidence-based treatment. A comprehensive overview of different therapeutic approaches addressing gut microbiota modulation for IBD management is available. Fecal microbiota transplantation (FMT) offers a direct method for introducing a corrected, stable microbial community through the transfer of fecal material from a healthy donor. Initially applied in treating Clostridium difficile and receiving FDA approval, its utility is now also explored or employed in inflammatory bowel diseases. FMT represents a promising therapeutic option in both Crohn's disease and ulcerative colitis; although further research is necessary to determine the optimal preparation mode and to better comprehend the mechanisms linking gut microbiota and IBD [2, 20]. The modulation of gut microbiota through diet, probiotics, and fecal microbiota transplantation could thus be regarded as a complementary approach to pharmacological therapies in managing IBD and associated extraintestinal manifestations, facilitating the re-establishment of intestinal immunological homeostasis.

Probiotics and Prebiotics

Conventional therapies targeting immune and inflammatory pathways in inflammatory bowel disease (IBD) remain unsatisfactory. Since the involvement of intestinal microbiota in IBD pathogenesis, particularly Crohn's disease (CD), was proposed, modulation of gut microbiota has attracted increasing attention as an alternative therapeutic strategy [21]. Probiotics are live microorganisms that confer health benefits on the host when administered in adequate amounts. Selected bacterial strains show effectiveness in the treatment and prevention of many diseases, including diarrheal diseases, lactose intolerance, ulcerative colitis (UC), and CD. Probiotics may prevent invasion of pathogenic bacteria, stimulate the synthesis of nutrients and vitamins, reduce cholesterol levels, regulate bowel transit time, and exert anti-carcinogenic effects. The gut microbiome of IBD patients differs significantly compared to that of healthy individuals, exhibiting reduced diversity, reduced relative abundance of Firmicutes, elevated Proteobacteria and Bacteroidetes, and an altered Firmicutes/Bacteroidetes ratio [21]. Loss of tolerance or increased immune response to resident intestinal bacteria is hypothesized to trigger inflammation during IBD pathogenesis. In the gastrointestinal tract, prebiotics, largely colon-indigestible dietary fibers, provide a substrate for fermentation by gut microbiota, leading to the production of short-chain fatty acids such as acetate, propionate, and butyrate [21]. Paraprobiotics, or non-viable microbial cells and cell fragments, may be beneficial in some clinical conditions; their use could be an alternative to live probiotics, which are not always feasible or advisable. Various approaches for restoring microbial imbalance include the use of probiotics, prebiotics, and faecal microbiota transplantation (FMT). Formulation of commercially available probiotics is typically based on overgrowth of Lactobacillus and Bifidobacterium species [21].

Fecal Microbiota Transplantation

Fecal microbiota transplantation (FMT) involves the infusion of fecal suspension from a healthy donor into the gastrointestinal tract of a recipient. This practice aims to restore a diverse, balanced intestinal microbiota in individuals with diseases caused by microbial imbalance [22]. Inflammatory bowel disease (IBD) has been a frequent indication for FMT, with literature reporting successes through various techniques. The evolution of FMT strategies and their application in IBD therapy continues to be a significant focus of clinical research [22].
Although FMT is well established in the treatment of recurrent Clostridioides difficile infection (CDI), its application across a wider spectrum of diseases, including non-gastrointestinal conditions, is still under investigation. Large clinical series and meta-analyses underscore the broad effectiveness of FMT in recurrent CDI [23]. FMT has also been explored for IBD treatment, with remission reported in patients with active ulcerative colitis and Crohn's disease, and broader use in ulcerative colitis, Crohn's disease, and pouchitis remains experimentally supported by case series and open-label trials. Systematic reviews consistently identify reduced microbial diversity and increased species of Enterobacteriaceae and Bacteroidetes in the context of IBD [24].

Current Research and Future Directions

Advances in microbiome analysis are expected to improve understanding and enable personalized management of IBD patients. The microbial community shapes a dynamic ecosystem that interacts with and influences host cells. Microbiome research should consider interpersonal variability, time-dependent ecosystem changes, and contextualize data within the microenvironment to increase signal-to-noise ratio [23]. Data-driven approaches and ecological theory provide clinically relevant models; insights into gut ecosystem assembly for IBD patients undergoing surgery and the concept of microbial invasion have led to an in silico invasion model for population-scale gut microbiome data. Establishing a framework involving billions of data points across multiple systems requires public engagement, patient involvement, and international commitment to open data sharing and research integration at the company level [24].

Emerging Technologies in Microbiome Research

Marketed as transformational, "multi-omics" technologies provide an important means of understanding the molecular and metabolic pathways that underpin the host–microbe cross-talk in a model agnostic way [25]. Given the rapid expansion of microbiome studies using new technologies, encouraging results have nonetheless been met with a degree of scepticism, stemming principally from a high level of interindividual microbiome variability, along with heterogeneity in study design and cohort selection. Still, ongoing metagenomics, metatranscriptomics, and metabolomics efforts are finishing, making it possible to move microbiome research beyond its current state of descriptive characterization towards identification of viable microbial-led treatment strategies [25]. In parallel, the rapid acceleration in diagnostic and surveillance tools informed by microbiome profiling is reaching sufficient sensitivity and specificity that it has become possible to envisage practical application in the clinic [25].

Potential for Personalized Medicine

Advances in genomic, metabolomics, and metagenomics technologies have broadened insights into the microbial composition of individuals and its impact on conditions such as inflammatory bowel diseases (IBDs). These research efforts, combined with epidemiological analyses, highlight opportunities to tailor therapeutic approaches to the specific characteristics of individual patients through a precision-medicine model [26]. Precision medicine implies neutralizing pathogenetic mechanisms, mechanistically linked to inflammation within the gut and the systemic compartment. Such approaches encompass pharmacological interventions with specific drugs (e.g., anti-TNF-α or anti-integrin agents) as well as lifestyle modifications (e.g., exercise, diet, or therapies influencing the brain–gut axis) in accordance with the patients' characteristics and variability [27, 28, 29]. IBDs exemplify how the available armamentarium for precision medicine also includes targeted interventions modulating the lifestyle and the environment. The ability to classify IBD patients about their dysbiosis profile is the current bottleneck in developing tailored interventions; however, the potential of this avenue remains remarkable [26].

Challenges in Microbiome Research

Microbiome research is subject to a variety of factors that limit the potential for clinical and translational application [4]. Specific challenges include variability in study designs, sample collection, confounders, and the sensitive and complex nature of microbiome ethical considerations, which are far from straightforward [4]. Microbiome science has therefore not yet been able to reach its full potential, remaining much more suited to describing phenomena than prescribing direct interventions [2, 30, 31].

Variability in Study Designs

The microbiome field is still relatively new but rapidly growing; therefore, there is currently little standardization across study design, including sample type, sampling methods, sequencing methods, and analysis techniques. One example of the need for standardization involves sample types: stool, biopsy, and colonic lavage [29]. After systematically searching PubMed and analyzing how these sample types affect results in inflammatory bowel disease gut microbiome studies, a meta-analysis finds that results from biopsy and stool samples clustered separately, but lavage samples clustered alongside both stool and biopsy samples, suggesting lavage would be an acceptable method with advantages for analyzing both luminal and mucosal bacterial communities [28]. Although many effects are reproducible across sequencing platforms, 16S rRNA versus metagenomics, there can be variable taxa estimates based on the platform, and therefore, batching by platform or cross-platform validation is recommended. Microbiome methods and analyses are a fast-moving field with new approaches that can separate environmental and genetic effects on the microbiome, identify possible causal directions in bacteria-host relationships, and incorporate microbiome information into genome-wide association studies. Complex approaches combining statistical and machine learning techniques may prove necessary, but increased cohort sizes and availability of quality metadata are also critical to aid in interpretation and the power of these studies [29]. The microbiome also opens up ethical questions of biobanking microbiome samples and the use and ownership of microbiome data that will require input from stakeholders, including patients and researchers, but more tools,

standardization, and discussion surrounding ethics and reproducibility are essential to further advance the field [29, 32, 33]. The study also illustrates critical confounding factors of cohort heterogeneity when searching for microbiome modifiers in inflammatory bowel disease. Stratification of cohorts by health status, inflammatory bowel disease condition, or disease location affects patterns of microbiome diversity, taxonomic relative abundance, and functional capacity, which in turn affect the interpretation of the relative strength of these factors in shaping the microbiome across inflammatory bowel disease cases [28, 29, 34, 35].

Ethical Considerations

The renewed interest in the microbiome has also led to new questions and challenges. Among them, ethical considerations are among the most important ones. Indeed, it seems essential to anticipate the potential societal consequences of microbiome analyses and modifications and to address public concerns [2]. These potential considerations are amplified if large-scale and longitudinal analyses with immediate clinical consequences are performed [4, 26, 36].

CONCLUSION

The gut microbiome is integral to the development and progression of IBD, mediating immune dysregulation, epithelial barrier dysfunction, and chronic intestinal inflammation. Dysbiosis in IBD is characterized by loss of protective microbes and overgrowth of pro-inflammatory taxa, with functional consequences for host immunity and metabolism. Interventions such as diet modification, probiotics, prebiotics, and FMT offer promising avenues to restore microbial balance and promote remission, though their clinical efficacy remains variable. Advances in sequencing technologies, metabolomics, and multi-omics platforms are enabling deeper insights into host–microbiome interactions and paving the way for microbiome-informed precision medicine. However, significant challenges persist, including methodological variability, cohort heterogeneity, and ethical considerations regarding microbiome data and interventions. Future progress will depend on standardized methodologies, well-designed longitudinal clinical trials, and integration of microbiome science into personalized treatment strategies. Harnessing the therapeutic potential of the microbiome offers a transformative opportunity for improving outcomes in patients with Crohn's disease and ulcerative colitis.

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