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Probiotics and diet quality: modulation of gut microbiota on intestinal infection and inflammation

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Abstract

Inflammatory bowel disease (IBD) is a chronic inflammatory condition affecting the digestive tract. The precise etiology of IBD remains unclear; however, there is a growing body of evidence suggesting that dietary factors may play a significant role in the pathogenesis of this disease. Dietary habits exert a significant influence on the structure of the gut microbiota, which in turn plays a pivotal role in maintaining intestinal balance. Conversely, intestinal inflammation has the potential to trigger gut dysbiosis, which affects the absorption of nutrients by host cells and gut microbiota. The interaction between diet and gut microbiota is impaired in individuals with IBD. A deeper understanding of the complex links between diet and the gut microbiome offers vital insights into the pathogenesis of IBD and encourages the creation of novel therapeutic approaches. This article reviews the current literature on the interaction between diet, probiotics, and gut microbiota in maintaining gut homeostasis.

Keywords: Diet, Microbiota, Probiotics, Inflammatory bowel disease, Crohn's disease, Ulcerative colitis

Introduction

The human neonatal gut flora is known to be colonized by vaginal or cutaneous bacteria, depending on the mode of delivery, and specific bacterial species, especially Helicobacter pylori, are highly predominant in neonates. Colonization of the intestine is known to begin *in utero* and the composition changes continuously during the first few years of life, depending on both environmental and nutritional factors. By around 3 years of age, the microbial composition stabilizes.[1] The intestine is exposed to luminal contents containing numerous microorganisms and food antigens throughout life. To maintain normal homeostasis, the system must have the ability to determine whether a stimulus is pathogenic or not and to mount an appropriate response that may lead to inflammation or tolerance. In the gut context, defence mechanisms and tolerance must be compatible to enable the organism to control the inflammatory response and tissue damage that can occur after exposure to a particular pathogen. Immunodeficiency results in recurrent infections, whereas defective tolerance can result in uncontrolled inflammation and immunopathology. Indeed, an abnormal relationship between host and microbiota is thought to cause an immune imbalance in the gut, leading to the emergence of disorders such as inflammatory bowel disease (IBD). IBD includes two main forms, Crohn's disease (CD) and ulcerative colitis (UC).[2]

The global burden of IBD has been steadily increasing, emerging as a significant public health concern. IBD has emerged as a global disease in the 21st century, with an estimated worldwide prevalence of 7 million cases.[3] Prevalence exceeds 0.3% in high-burden regions such as North America, Oceania, and Europe, and is rising in newly industrialized countries across Asia, Africa, and South America, driven by urbanization and the adoption of Westernized lifestyles. In the United States, over 0.7% of the population has been diagnosed with IBD, with the highest incidence occurring in early adulthood. In Canada, approximately 322,600 individuals (0.82% of the population) were living with IBD in 2023, a figure projected to rise to 1.1% by 2035. European data indicate around 1.3 million affected individuals, corresponding to 0.2% of the regional population. Although IBD predominantly affects young adults, about 25% of cases occur before the age of 20, and it can manifest at any age. These trends highlight the critical need for strategic planning, resource allocation, and the development of preventive health policies to manage the growing global burden of IBD.[4–6] Various hereditary and environmental factors are known to play a role in the development and course of IBD.

Research method

This review examines the fundamental mechanisms and potential interactions between diet, gut microbiota, and the development of IBD. Relevant literature was systematically retrieved from databases such as PubMed, Web of Science, and Google Scholar using specific keywords ("diet," "microbiota,"

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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"probiotics," "inflammatory bowel disease," "Crohn's disease," and "ulcerative colitis"). A range of studies, including epidemiological, experimental, and review articles, were considered based on their relevance to the topic.

Gut microbiota: composition and functions

Human intestinal microbiota is a microecosystem that has been the subject of numerous studies in recent years and produces trillions of microorganisms, especially bacteria, yeasts, viruses, and parasites. It is one of the main components of the intestine and plays important roles in many critical events for human health, including protection of the intestinal barrier, shaping and maturation of the immune system, regulation of nutrient intake, and drug absorption in human metabolism.[7,8] The microbiota, which constitutes the largest bacterial reservoir in the human body, can shape both local and systemic immune responses.[9]

There is a reciprocal relationship between the host and the microbiota population. These microorganisms perform various actions such as metabolite production, which turns out to be suitable for the human host under physiological conditions, reducing the harmful effects of potentially toxic substances, interacting with the host's immune system and increasing its functionality. In this way, they can affect both the physiological homeostasis of the gastrointestinal tract and the development of the disease. In addition, microorganisms in the microbiota of the host have developed some features such as evading bacteriophages and the host immune system to survive.[10]

The human intestine is actually sterile at birth; the microbiota is a structure that forms later. During birth and after respiration, the microbiota begins to colonize the intestinal tract in contact with the external environment. With nutrient intake, the species and diversity of gut microorganisms are continuously enriched and eventually the stable structure of the gut microbiota is established.[11] Our immune system has developed various strategies to tolerate commensal bacteria that maintain homeostasis, so it does not cause a pro-inflammatory response. However, when this delicate balance is disturbed due to damage to the interface between the host and the microbiome or disruption of the immune system, consequences such as systemic spread of commensal microorganism, susceptibility to pathogen infiltration, and abnormal immune response are observed.[12]

The gut microbiota includes majorly Verrucomicrobia, Proteobacteria, Actinobacteria, Firmicutes, and Bacteroidetes, among which Firmicutes and Bacteroidetes usually account for more than 90% of the total gut microbiota. The composition of the gut microbiota and its metabolites, short-chain fatty acids (SCFA), play an important role in the regulation of the immune system.[11] Imbalances in the gastrointestinal microbiota affect many pathways such as host health, energy absorption, choline, SCFA, gut-brain axis, and bile acids.[10,13]

Factors affecting intestinal microbiota

Gut microbiota is a dynamic ecosystem influenced by age, genetics, environment, lifestyle, and mainly diet.[8] As a result of microbiome-host genetic effect studies in monozygotic and dizygotic twins to determine the heritability of microbiota, it was determined that many genetic factors affect the intestinal microbiota. Mutations in host genes such as *MUC2, MyD88, IgA, NOD2, NLRP6, TLR5,* and *FUT2* are associated with significant alterations in gut microbial composition. Variants in the *NOD2* and *MUC* genes disrupt mucosal barrier integrity, leading to gut dysbiosis; notably, NOD2-related dysbiosis is linked to colitis, whereas MUC variants are more closely associated with UC.[14] The host immune system can be modulated by microbiota-derived molecules *via* inflammasome, Toll-like receptor (TLR), and NOD-like receptor (NLR) signaling (Hrncir 2022).[15] Pathogen recognition receptors (PRRs) are essential components of host innate immune systems that detect specific conserved pathogen-associated molecular patterns (PAMPs) presented by microorganisms. Members of two families of PRRs, transmembrane TLRs (1, 2, 4, 5, and 6) and cytosolic NOD receptors (NOD1 and NOD2), are stimulated upon recognition of various bacterial PAMPs. Such stimulation leads to the induction of a number of immune defense reactions, mainly triggered *via* activation of the transcription factor nuclear factor kappa B (NF-κB).[16] Among these, TLR5 plays a critical role by recognizing bacterial flagellin and driving NF-κB activation together with innate cytokine production (eg, interleukin [IL]-6, tumor necrosis factor [TNF]-α), thereby controlling microbial dysbiosis and preventing inflammation. Importantly, TLR5 polymorphisms impair flagellin-specific IgG/IgA responses and are negatively associated with CD.[17] In addition, HLA gene polymorphisms influence antigen presentation and immune responses to various gut-derived proteins, ultimately shaping microbial colonization patterns. Some microorganisms in the microbiota are also associated with histone acetylation. Although the microbiota–host relationship starts early in life, it plays a critical role in establishing and maintaining homeostasis and affects health in the long or short term.[1] The type of birth affects the type of bacteria that will colonize the neonatal microbiota; on the other hand, the feeding status of the newborn with breast milk or infant formula affects the composition of the gut microbiota.[10] Diet is an important factor determining the diversity of microbiota.[18] Supporting this, many studies have demonstrated that diet is the main factor contributing to both long-term and short-term changes in the diversity of the gut microbiome. Recent studies have suggested that altered bacterial composition as a result of diet may be linked to certain diseases, particularly those caused by chronic low-grade inflammation, such as type 2 diabetes.[19] Various environmental factors play a role in shaping the microbiota, including geographical location and thus factors such as dietary culture, climatic conditions, surgical operations, smoking, depression, and life changes. Antibiotic use also significantly disrupts both short- and long-term microbial balance, including reductions in the richness and diversity of the microbiota.[20]

Environmental factors

Dysbiosis of the gut microbiota due to environmental factors such as diet, antibiotic exposure, sleep disturbance, physical activity, and psychological stresses can lead to changes in gut microbiota composition and bacterial metabolite production, disruption of host intestinal barrier integrity and development of immune system, intestinal inflammation, and IBD.[21] The type of delivery, cesarean or vaginal, affects the type of bacteria that will colonize the neonatal microbiota; in those born by cesarean section, *Escherichia coli* and Clostridium difficile dominate, while *Bifidobacteria* are present to a lesser extent. On the other hand, the microbiota of babies born vaginally is dominated by Lactobacilli and Prevotella spp. Breastfed infants are mostly colonized by *E. coli*, Streptococci, and Bifidobacteria, whereas formula-fed infants are primarily colonized by Enterobacteria, Clostridia, and Bacteroidetes.[10] There are also differences in microbiota according to the geographical areas where individuals grow up and live. These differences are related to both dietary changes and other environmental factors.[1] In a study conducted by Kim *et al*[22] in South Korea, it was observed that the genus Firmicutes was abundant in rural elderly, while Bacteroidetes tended to prevail in urban elderly.[10] One of the environmental factors affecting microbiota composition is heat exposure. In humans and rats, heat exposure has been found to increase pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α, which are known to decrease tight junction protein expression. It can also cause inflammation and sepsis by increasing intestinal barrier dysfunction and permeability.[21]

Dietary influences on gut microbiota

Dietary macronutrients, minerals, fat, vitamins, trace elements, dietary fiber, alcohol, non-nutritive bioactive elements such as probiotics and prebiotics, as well as phytochemicals influence immunomodulatory and inflammatory processes. In addition, the gut microbiota can bioconvert food elements by producing metabolites that can modulate host immune and metabolic responses when food components are used alone or combined.[12] Changes in intestinal microbiota and related metabolite levels and disruptions in the intestinal barrier are evidence of the relationship between intestinal microbiota and intestinal inflammation.[1] Since dietary habits tend to vary according to geographical factors, it can be seen that this variability is reflected in the microbiota of different populations.[8]

Modulation of the microbiota by diet is evident from the beginning of life, when human milk oligosaccharides (HMOs) participate in the maturation of the microbiota from early infancy, followed by an increase in bacterial richness, which begins to change with the consumption of solid foods and results in a decrease in the richness of microbiota diversity observed in aging populations, possibly due to reduced food diversity. Foods with higher anti-inflammatory potential may reduce the formation of pro-inflammatory factors such as IL-6, IL-1β, IL-8, TNF-α, reactive oxygen species (ROS), nitric oxide (NO), and prostaglandins (PGs).[12]

In general, studies on the Mediterranean Diet (MD) have been linked to beneficial metabolomic profiles associated with microbiota, such as increased levels of SCFA and decreased levels of long-chain fatty acids, bile acids, and trimethylamine N-oxide (TMAO). Various studies on the MD, which is a dietary pattern characterized by high amounts and frequent consumption of grains, preferably whole grains, legumes, nuts, vegetables, and fruits, have shown that consumption of foods included in the typical MD has been improving obesity, lipid profile, and inflammation *via* changing the composition of the intestinal microbiota by leading to an increase in beneficial bacteria such as Lactobacillus, Bifidobacterium, and Prevotella and a decrease in pathogenic bacteria such as Clostridium.[23]

Gut microbiota and intestinal inflammation in IBD

Immune dysfunction drives the pathogenesis of IBD, including UC and CD.[24] IBD describes a heterogeneous group of chronic diseases of the gastrointestinal tract characterized by a recurrent inflammatory process and changes in the immune system. It is characterized by intestinal dysbiosis, although the cause-and-effect relationship is not clear. Both the incidence and prevalence of the disease have been rapidly increasing globally in recent years.[24]

IBD is thought to result from a combination of hereditary and environmental factors, leading to an imbalance of pro- and anti-inflammatory mediators. Environmental factors are being investigated in depth to understand their role in both disease onset and risk of disease recurrence.[12]

Among environmental factors, diet is known to play an important role in IBD, influencing the developing risk and modulating disease activity. Specific dietary compounds have been shown to have favorable effects on gut microbiota homeostasis, intestinal mucosal permeability, and modulation of the inflammatory response.[12] The role of the microbiome in IBD is also increasingly recognized. Animal models have described how dysbiosis can lead to the development of IBD in a genetically susceptible host and similar relationships have been found in human studies.[25] Furthermore, the influence of gut microbiota on the pathogenesis and progression of IBD has been extensively studied. Mainly, decreased abundance of Firmicutes, Bifidobacterium genus, and *Faecalibacterium prausnitzii* species was observed and a reduction in overall diversity was also found.[12] Likewise, a dysbiotic microbiota with low diversity, depletion of beneficial species, and an excess of Proteobacteria are among the most consistent microbial features of IBD.[26]

IBD represents the archetypal disease in which the intestinal microbiota and the intestinal immune system lose their balanced interaction. An important contributor to the establishment of this balance is microbial competition for food nutrient components. However, since microbiota architecture also depends on nutrient components, the gut microbiota is often considered as a mediator through which foods exert their pro- and anti-inflammatory effects.[12] The complex symbiotic interaction between microbiota and host is mediated by a balance in tolerance and inflammatory response to microbial products in the gut. However, in addition to the composition of the microbiota, certain common bacteria, such as *Clostridium perfringens* and *Bacteroides fragilis*, can express a wide range of toxins and metabolic compounds to induce inflammation. *C. perfringens* is usually a gram-positive anaerobic bacterium in the environment and is part of the resident microbiota. However, it can produce more than 20 toxins with the expression of toxin genes; through the toxins it produces, it can disrupt the epithelial barrier, cause a pro-inflammatory and pro-pathogenic environment and ultimately become lethal.[9]

It has been demonstrated that the IBD-associated virome is mainly represented by members of the Caudoviridae family; a possible pathogenic effect of viruses such as Caudovirales bacteriophages may be explained by their ability to cause intestinal dysbiosis. Polydnaviridae, Tymoviridae, and Virgaviridae; less Herpesviridae are found in IBD patients. Fungal dysbiosis also plays a role in the development of IBD. One study reported that the overall fungal diversity was higher in IBD patients than in healthy individuals, and some species were detected only in IBD samples.

Another bacterial metabolic pathway involved in the pathogenesis of IBD is the production of SCFA by specific strains of Clostridia spp. It has been shown that SCFA enhances regulatory T cell (Treg) function in the intestinal mucosa through activation of G protein-coupled receptors and subsequently causes epigenetic effects through inhibition of histone deacetylase (HDAC).[10] IBD often develops a dysbiosis characterized by the relative depletion of *Faecalbacterium praustnitzii* and *Roseburia hominis* and the enrichment of *E. coli*, Ruminococcus torques, and *R. gnavus*. Dysbiosis may have functional significance in disease manifestation. This is exemplified by a decrease in *F. praustnitzii* and *R. hominis* producing butyrate, a useful metabolite.[27]

An increase in Escherichia and Shigella was detected in UC patients compared to healthy individuals. Shiga toxin-producing Shigella species and *E. coli* are considered pathogenic associated with diarrhea and colitis. These toxins can induce activation of the NLR protein 3 inflammasome, triggering IL-1β and IL-18 production and cellular death *via* pyroptosis. The virulence of Shiga toxin-producing *E. coli* may lead to diarrheal diseases and death.[9] While Hepadnaviridae and Pneumoviridae abundance is observed in UC patients, the opposite is observed for anelloviruses. In CD patients, Fernandes *et al*[28] found an increase in Caudovirales and a reduced proportion of strains in the Microviridae family compared to healthy controls.[10]

Characteristics of gut microbiota dysbiosis in IBD patients

Recent scientific studies have demonstrated that the composition and metabolic functions of the gut microbiota play a critical role in the development of IBD. A significant reduction in SCFA-producing bacteria has been observed in IBD patients, which offers an important insight into the disease's pathophysiology. SCFAs—namely acetate, propionate, and butyrate—contribute to gut health maintenance, support epithelial barrier integrity, and regulate anti-inflammatory responses. Therefore, understanding the disruption in SCFA production is essential for developing microbiome-based therapeutic strategies.[5] SCFA depletion in IBD patients correlates with increased intestinal permeability and chronic inflammation, highlighting the functional significance of microbiota metabolites in disease pathogenesis.[29]

Research has revealed significant structural changes in the microbiota of individuals with IBD. In particular, a marked decrease in butyrate-producing Firmicutes (such as *F. prausnitzii*, Roseburia, and Ruminococcus) has been reported, along with an increase in potential pathogens like Proteobacteria. IBD patients often show a reduction in butyrate-producing Firmicutes such as *F. prausnitzii* and an increase in Proteobacteria, contributing to dysregulated immune responses and impaired barrier function. This microbial imbalance is not merely a consequence of inflammation but also plays a direct role in the pathogenesis by promoting immune dysregulation, increasing intestinal permeability, and sustaining chronic inflammation.[5]

Different subtypes of IBD, such as CD and UC, exhibit distinct patterns of microbial dysbiosis. In CD, there is a decrease in taxa typically associated with a healthy microbiome and an increase in species such as *E. coli* and Fusobacteriaceae. Conversely, UC is characterized by increased Proteobacteria and Patescibacteria, along with decreased Desulfobacterota and Verrucomicrobiota. CD is frequently associated with decreased Firmicutes and increased Enterobacteriaceae, whereas UC shows distinct shifts in Bacteroidetes and mucosal-associated microbes. These shifts in gut microbiota are not only important for understanding disease development but may also serve as potential therapeutic targets in the future.[5]

Therapeutic approaches

Dietary patterns and nutritional interventions in IBD

Dietary patterns and dietary components can have a positive or negative effect on the gut microbiome, and various anti-inflammatory diets have been used with clinical success in patients with CD. However, evidence on dietary manipulation strategies in patients with UC is limited and therefore recommendations on diet as primary or adjunctive therapy cannot be made.[24]

Dietary intake is associated with enrichment and reduction of multiple pro-inflammatory and anti-inflammatory taxa of the gut microbiome. In contrast, Western dietary intake has been associated with an increased risk of colitis and flare occurrence in IBD. The typical Western diet is characterized by exposure to higher amounts of processed foods, red meat, fat, sugar, emulsifiers, and additives, as well as reduced amounts of fiber, fruits, and vegetables.[25] In the meta-analysis conducted by Black *et al*,[30] the low FODMAP diet was found to be effective particularly in alleviating symptoms such as bloating and overall discomfort in patients with IBS. However, long-term restriction of FODMAPs is not recommended. A low FODMAP diet reduces the fermentable substrate in the colon and changes the microbiome composition; a consistent reduction in the abundance of Bifidobacteria has been observed.[31] Bifidobacteria is beneficial to the host as it produces acetic and lactic acid, thereby preventing the growth of pathogenic bacteria and maintaining the immune system.[32] Given the well-established bifidogenic effects of fructans and galacto-oligosaccharides (GOS), their restriction during a low FODMAP diet has been associated with reductions in Bifidobacteria abundance.[33]

Amid speculation on its safety, microbiome changes induced by 3 to 4 weeks of FODMAP restriction are specific for Bifidobacteria and do not involve broad changes to microbial composition and function. This should allay concerns about the safety of a short-term low FODMAP diet with regard to the colonic microenvironment.[34] Although the long-term consequences of these changes are unknown, the reintroduction of high FODMAP foods that are tolerated is a critical phase in the clinical application of the low FODMAP diet and may help mitigate its impact on both dietary intake and the gut microbiota.[30] To address these concerns, strategies such as the reintroduction phase, dietary personalization, and the incorporation of probiotics have been proposed to help restore microbial diversity and promote gut health while maintaining symptom relief. These approaches underscore the importance of individualized dietary management in low FODMAP diets, recognizing the unique responses of each person's microbiome to dietary interventions.[35]

CD causes a decrease in bifidobacteria and an increase in *F. prausnitzii*, which is positively associated with IL-100, which is known to express dendritic cells.[31] Highly restricted diets requiring avoidance of more than one food should be followed under close supervision of a multidisciplinary IBD team, including a dietitian. Patients should be regularly checked for calorie, macronutrient, and micronutrient adequacy, and deficiencies should be corrected immediately when identified.[25]

Probiotics in the modulation of gut microbiota and IBD therapy

In recent studies, two theories have been proposed regarding the role of bacteria in the pathogenesis of IBD.[36] The first of these theories is excessive bacterial translocation and dysbiosis as a result of changes in the intestinal microbiota or disruption of the mucosal barrier. It has been observed that the microbiota of IBD patients has fewer protective bacteria (Bifidobacterium and Lactobacillus) and more bacteria with pathogenic potential (*E. coli* and Clostridium species) compared to the microbiota of healthy individuals. In addition, there are changes in the metabolic activity of microorganisms in the gut microbiota; the synthesis of SCFA decreases and the synthesis of toxins increases.[37] SCFAs, primarily produced by core gut microbiota such as Faecalibacterium, Roseburia, and Lachnospira, contribute to the maintenance of intestinal barrier integrity by enhancing tight junction protein expression and regulating mucosal immune responses. Among them, butyrate, a key SCFA, acts as a HDAC inhibitor, promoting anti-inflammatory Treg differentiation and suppressing pro-inflammatory cytokine production in the gut. Furthermore, propionate and acetate, produced by Bacteroidetes and other core genera, modulate dendritic cell function and support epithelial defense mechanisms against pathogenic invasion.[29]

The second theory is abnormal immune responses involving changes in gut microbiota and epithelial cell function.[36,37] This dysregulated mucosal immune response predisposes to chronic inflammation.[38] IBD is characterized by exacerbation and remission states, and one of the therapeutic goals is to sustain the remission phase, which is likely to be prolonged.[38] Modulation of gut microbiota can be achieved by antibiotics but it is not a good treatment option for chronic disease due to antibiotic resistance, potential side effects, and ecological concerns. Therefore, there has been a search for a potential mechanism of assistance to existing conventional therapies in IBD.[38]

The term probiotic refers to "live microorganisms that provide health benefits to the host when they reach the gut in sufficient quantities in an active state."[37,39] It provides positive modulation of the natural microbiota composition and causes improvement in the immune response.[37] Given the current evidence that the gut microbiota is involved in the pathogenesis of IBD, manipulating the gut microbial composition, especially probiotics that modulate the microbial composition, may be beneficial for the treatment of the disease.[40] They mostly include lactic acid-producing bacteria such as Bifidobacteria and Lactobacilli, but organisms such as *E. coli* and yeast *Saccharomyces boulardii* have also been reported to have some beneficial effects in maintaining remission in patients with IBD. Although their mechanism of action has not been determined, some studies suggest that these probiotics modulate membrane permeability and mucosal immune system.[39] Mechanisms by which probiotic microorganisms alter the gut microbiota include lowering luminal pH, competition for nutrients, secretion and stimulation of the production of antimicrobial compounds (bacteriocins and defensins) by the host, and changes in nitrogen metabolism.[37] They also play an immunomodulatory role with important immune effects such as their ability to promote the formation of SCFA (acetate, propionate, and butyrate), reduction of NF-κB activation, maturation of dendritic cells, increase in the activity of natural killer cells, stimulation of cytokine synthesis such as IL-10, activation of antigen-presenting cells in Peyer plaques. In this way, it can reduce the expression of pro-inflammatory factors and inflammatory response and offer an alternative approach to traditional treatment.[37,41]

The European Society for Parenteral and Enteral Nutrition (ESPEN), in its latest guidelines on clinical nutrition in IBD, recommends probiotic supplementation as an adjuvant therapy for the induction and maintenance of remission period in UC patients. Probiotic supplementation, especially with Lactobacillus and Bifidobacterium species, offers several therapeutic effects in UC, such as induction or maintenance of the remission period and attenuation of symptoms and clinical signs. These effects are due to decreased synthesis of pro-inflammatory mediators and increased synthesis of anti-inflammatory factors. Regarding CD, the results of clinical trials are controversial and do not support the use of probiotics in this disease. Although evidence suggests an immunomodulatory role of probiotics in CD patients, most studies do not demonstrate therapeutic effects of probiotic supplementation in CD. In this context, probiotic administration is recommended as an adjuvant in the treatment of UC, not in the treatment of CD.[37] The differences in probiotic benefits between CD and UC may reflect the complexity of probiotic–host interactions and can be attributed to differences in lesion distribution and immune-mediated pathophysiology. While lesions in UC are limited to the mucosal and submucosal layers, CD typically affects the entire intestinal wall, making mucosal healing more difficult in CD than in UC. Moreover, the fact that inflammation in CD and UC is driven by different T helper cell pathways may explain why probiotics show limited therapeutic effects and no significant improvement in CD, whereas they are more effective in UC.[42]

The aim of the study conducted by Shadnoush *et al*[36] with a total of 305 participants was to investigate the effects of probiotic yoghurt consumption on intestinal microbiota in patients with IBD. In the study, participants were divided into three groups: group A (IBD patients receiving probiotic yoghurt), group B (IBD patients receiving placebo), and control group (healthy individuals receiving probiotic yoghurt). Lactobacillus, Bifidobacterium, and Bacteroides populations were measured in stool samples collected 8 weeks before and after the intervention. At the end of the intervention, the mean Lactobacillus, Bifidobacterium, and Bacteroides counts in group A were significantly higher than in group B. It was concluded that consumption of probiotic yoghurt by patients with IBD may help improve bowel function by increasing the number of probiotic bacteria in the intestine and colon.[36]

Currently, sulfasalazine (SASP) is a commonly used drug to treat UC. The study by Wu *et al*[41] aimed to compare the therapeutic effects of *Lactobacillus plantarum* HNU082 (Lp082), one of the main probiotics used in the treatment of IBD, and SASP on UC induced by dextran sulphate sodium (DSS). Both animal and clinical experiments reported that *L. plantarum* can reduce chronic mucosal inflammation in patients with UC and alleviate the occurrence of experimental colitis induced by DSS. The results showed that Lp082 had a better therapeutic effect on DSS-induced UC than SASP, and the treatment significantly improved DSS-induced UC in mice. Increases in body weight, water intake, food intake, and colon length; decreases in disease activity index, immune organ index, inflammatory factors, and histopathological scores were observed after Lp082 ingestion. It was concluded that the mechanisms of action include protecting the mucosal barrier, actively modulating the intestinal microbiome, attenuating the inflammatory response by modulating inflammatory pathways, reducing neutrophil infiltration, and treating UC in this way.[41]

Bjarnason *et al*[43] conducted a study evaluating the efficacy of a multi-strain probiotic (*Lactobacillus rhamnosus* NCIMB 30174, *Lactobacillus plantarum* NCIMB 30173, *Lactobacillus acidophilus* NCIMB 30175, and *Enterococcus faecium* NCIMB 30176) in improving quality of life issues and intestinal inflammation in asymptomatic UC and CD patients. In a single-center, randomized, double-blind, placebo-controlled trial involving 81 UC and 61 CD patients, participants were treated with either the probiotic or placebo for 4 weeks. No serious side effects were observed in the probiotic group. There were no significant differences observed in IBD Quality of Life Questionnaire (IBDQL) scores or laboratory data between the placebo and probiotic groups. However, *post hoc* analyses indicated a significant reduction in fecal calprotectin (FCAL) levels in UC patients receiving probiotics compared to placebo, with no significant change observed in CD. Elevated FCAL levels, exceeding five times the normal upper limit, are indicative of ongoing intestinal inflammation and pose a significant risk for clinical relapse in individuals with clinically inactive IBD within the next 6 to 12 months. Consequently, while this multi-strain probiotic did not show efficacy in CD, it was associated with reduced intestinal inflammation in UC patients and was well-tolerated. Further research is needed to determine whether probiotics reduce clinical relapse incidence in asymptomatic IBD patients.[43]

Tamaki *et al*[39] investigated the efficacy of Bifidobacterium longum 536 (BB536) supplementation for remission induction in 50 Japanese patients with active UC in a randomized, double-blind, placebo-controlled trial. Patients were treated with BB536 or placebo for 8 weeks. Results showed that 63% of patients in the BB536 group achieved clinical remission by week 8, compared to 52% in the placebo group. Significant decreases in UC Disease Activity Index scores were observed in the BB536 group, whereas no significant change was noted in the placebo group. Similarly, significant reductions in Rachmilewitz endoscopic index and the Modified Mayo Disease Activity Index subscore were observed at week 8 in the BB536 group, but not in the placebo group. Only one patient in the BB536 group reported mild adverse effects, with no other side effects observed.[39]

E. coli has garnered increased interest recently and has been associated with both CD and UC, particularly during active phases of UC. Interestingly, probiotic *E. coli* Nissle (EcN) has been found to have equivalent efficacy to mesalazine (5-ASA) in preventing disease flares in UC patients.[44] Due to its beneficial effects on intestinal homeostasis, EcN is one of the most studied probiotics in UC patients.[40] Although findings regarding its efficacy in maintaining remission have shown considerable variability across clinical trials. For instance, Petersen *et al*[44] aimed to assess the effects of EcN, in addition to traditional therapies, in patients with active UC. In a single-center, double-blind, randomized, placebo-controlled trial involving 100 individuals with active UC, patients were randomized to receive 1 week of ciprofloxacin (Cipro) or placebo followed by 7 weeks of EcN or placebo. Surprisingly, fewer patients (54%) in the placebo/EcN group achieved remission compared to those in the placebo/placebo group (89%). Patients treated with Cipro/EcN and Cipro/placebo achieved remission rates of 78% and 66%, respectively. Additionally, the placebo/EcN group had the highest dropout rate (44%) compared to any other group (20%). Moreover, only 29% of patients in the placebo/EcN group reported no blood in stool at week 12, indicating a lack of mucosal healing in this group. These findings suggest that EcN as an adjunctive therapy to traditional treatments does not confer any benefit for active UC. Furthermore, treatment with EcN without a prior antibiotic course resulted in fewer patients achieving clinical remission.[44]

In another study involving EcN, Park *et al*[40] conducted a multicenter, double-blind, randomized, placebo-controlled trial with a total of 133 UC patients to investigate the efficacy of EcN as adjunctive therapy for UC. Patients were randomly assigned to receive EcN or placebo once daily for 8 weeks. Subsequently, IBDQL scores (primary endpoint) and response rates for clinical remission (secondary endpoints) were compared. While the number of patients reaching the primary endpoint did not differ between the EcN and placebo groups, significantly fewer patients in the EcN group exhibited low IBDQL scores. Additionally, significantly more patients in the EcN group achieved clinical response at week 4 and endoscopic remission at week 8 compared to the placebo group.[40] The discrepancies observed between the studies may stem from various methodological and population-related factors. First, while the study by Park *et al*[40] was conducted in Korea, the study by Petersen *et al*[44] was carried out in Denmark. It is well established that ethnic and geographic differences can influence baseline gut microbiota composition,[1] which in turn may affect EcN colonization and its biological activity. Additionally, in the Petersen *et al*[44] study, patients were randomized to receive a 1-week course of ciprofloxacin (or placebo) prior to EcN or placebo treatment—a factor that could significantly alter gut microbiota. In contrast, the Park *et al*[40] study excluded any prior antibiotic use, administering only 5-ASA alongside EcN or placebo. These differences in pre-treatment protocols may have contributed to the divergent outcomes regarding EcN efficacy. Furthermore, it is not only the population and treatment design that differ; the primary endpoints of the studies are also distinct. While Park *et al*[40] focused on health-related quality of life (IBDQ score), Petersen *et al*[44] evaluated clinical remission based on CAI score. This variation in outcome measurement could also contribute to the inconsistent findings.

Palumbo *et al*[38] evaluated the long-term effects of combination therapy (5-ASA) plus probiotic mixture: *Lactobacillus salivarius*, *Lactobacillus acidophilus*, and Bifidobacterium bifidus strain (BGN4) on UC activity in 60 patients with moderate to severe UC. In this 2-year study, patients were divided into two groups: Group A received once-daily 5-ASA, while group B received once-daily 5-ASA plus twice-daily probiotic mixture. Overall, all patients treated with combination therapy showed better improvement in disease activity measured by Modified Mayo Disease Activity Index compared to controls. Group B also demonstrated a significant improvement in stool frequency compared to group A. These results confirm the beneficial effects of probiotics in enhancing the response to anti-inflammatory treatments and suggest that the combined use of anti-inflammatory drugs and probiotics could serve as an alternative therapy to corticosteroids in mild to moderate UC.[38]

S. boulardii, a probiotic yeast shown to have beneficial effects on intestinal epithelial barrier and digestive immune system, has shown preliminary evidence for its use in treating CD. Bourreille *et al*[45] conducted a randomized, placebo-controlled trial involving 165 patients who achieved remission during treatment with steroids or aminosalicylates to evaluate the effects of *S. boulardii* in preventing relapse in CD patients. Patients were randomly assigned to receive *S. boulardii* or placebo for 52 weeks. The primary endpoint was the percentage of patients in remission at week 52, with secondary endpoints including time to relapse, CD activity index scores, and changes in inflammation parameters. There was no significant difference observed in relapse rates and time between the *S. boulardii* and placebo groups. Similarly, no significant difference was observed between groups in secondary endpoints. A *post hoc* analysis suggested that non-smokers who received *S. boulardii* had a lower likelihood of CD relapse compared to non-smokers who received placebo, but this finding requires confirmation. Thus, while *S. boulardii* was safe and well-tolerated, it did not demonstrate any beneficial effect for maintaining remission in CD patients following steroid or salicylate.[45]

Yılmaz *et al*[46] investigated the effects of kefir consumption on gut microbiota and symptoms in patients with IBD in a single-center, prospective, open-label, randomized, controlled trial involving 45 patients. Patients were divided into two groups and administered 400 mL/day of kefir for 4 weeks. Real-time quantitative polymerase chain reaction was used to determine Lactobacillus content in stool samples before and after consumption. Daily records by patients included abdominal pain, bloating, stool frequency, stool consistency, and well-being scores. Results showed a significant decrease in erythrocyte sedimentation rate and C-reactive protein, as well as an increase in hemoglobin in CD patients. Significant reductions in bloating scores and increases in well-being scores were observed in the last 2 weeks. Abdominal pain scores were significantly lower in CD patients compared to UC patients in the last 2 weeks, with significantly higher well-being scores in CD. There was no deterioration in disease symptoms in either group and no adverse effects were observed. The initial and final measurements of Lactobacillus bacterial load in the stool of all subjects in the treatment group were statistically significant. According to the data, kefir consumption can modulate gut microbiota and may improve both symptoms and quality of life in the short term for individuals with CD.[46]

Scott *et al*[47] developed Saccharomyces-based probiotics designed with enhanced extracellular ATP (eATP) sensitivity through a modified human P2Y2 purinergic receptor, which activates purinergic signaling to promote intestinal inflammation and pathology. This engineered P2Y2 receptor activation is linked to the secretion of the enzyme apyrase, which breaks down ATP, thus aiming to detect and neutralize pro-inflammatory molecules, providing a proportionate, self-regulating response. These self-regulating modified probiotic yeasts suppressed intestinal inflammation in murine models of IBD, reducing intestinal fibrosis and dysbiosis with efficacy similar to or higher than standard care treatments, which can often lead to serious side effects. These self-regulating modified probiotic yeasts could provide a new therapeutic platform for IBD and other inflammatory disorders.[47]

Lactic acid, a metabolic byproduct of the host and intestinal microbiota, has been identified as an active signaling molecule in the immune system. Lactic acid binds to receptors on the surface of intestinal macrophages, reducing cyclic AMP (cAMP) and protein kinase A (PKA) signaling, inhibiting the expression of pro-inflammatory factors, and potentially protecting against organ damage following inflammation and is of. In a study by Sun *et al*,[48] a lactic acid biosynthesis pathway producing high levels of lactic acid directly from glucose was reconstituted in probiotic *Saccharomyces cerevisiae* bacteria, instead of ethanol. In this study, modified *S. cerevisiae* regulated macrophage polarization status in DSS-induced mice, with improved histological damage, increased mucosal barrier, and decreased intestinal immune response observed. Furthermore, *in vivo* and *in vitro*, it exhibited anti-inflammatory activity by inhibiting the expression of pro-inflammatory cytokines. Additionally, it changed the abundance of metabolic products in mice with colitis by changing the diversity and composition of the intestinal microbiota. As a result, the application of modified *S. cerevisiae* suppressed macrophage piroptosis and modulated the intestinal microbiota, reducing DSS-induced colitis in mice, demonstrating an effective and safe treatment strategy for IBD.[48] Although engineered probiotics offer advantages such as stability, specificity, preference, low cost, and relative safety, their practical application still faces several challenges. For instance, genetic tools available for manipulation are limited for many microorganisms, and the strains currently used in research represent only a small portion of the vast microbial diversity. This restricts probiotic modification to just a few strains. In addition, the prolonged colonization of some engineered probiotics in the host body has been highlighted as a potential concern.[49]

The studies are summarized in Table 1.

Conclusions and recommendations

Intestinal microbiota is related to various hereditary and environmental factors and has a specific composition. The importance of the Firmicutes/Bacterioides ratio is particularly emphasized. Exposure to various environmental factors can lead to changes in the diversity and number of dominant species and this is called dysbiosis. As the microbiota collectively produce a wide range of compounds (such as SCFA, vitamins, and signaling molecules), some of these may cause an inflammatory response from the gut, which may promote the development and/or progression of IBD. Specifically, an alteration in the proportion of Firmicutes and Bacteroides species has been reported in patients with IBD. Ruminococcaceae and Lachnospiraceae and *F. prausnitzii* are significantly reduced and these bacteria produce butyrate, which maintains intestinal integrity and promotes mucosal immune tolerance. Attenuating dysbiosis and inflammatory cytokine increase and increasing alpha diversity are among the goals of treatment. Engineered probiotics present multiple advantages—such as enhanced stability, targeted specificity, favorable host preference, affordability, and relative safety—yet their widespread application continues to be constrained by practical obstacles. As research in this field is still in its early stages, further well-designed studies are required to establish their efficacy, safety, and translational potential.

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Table 1

Summaries of studies.

Number of individuals	Age (years)	Type of IBD	Type of probiotic	Duration of treatment	Results	Reference
305	27–50	UC + CD	Probiotic yoghurt (*Lactobacillus acidophilus* La-5 and *Bifidobacterium* BB-12)	8 weeks	It was concluded that consumption of probiotic yogurt by patients with IBD may help improve intestinal function by increasing the number of probiotic bacteria in the intestine and colon	Shadnoush *et al*[36]
142	26–62	UC + CD	*Lactobacillus rhamnosus* NCIMB 30174, *Lactobacillus plantarum* NCIMB 30173, *Lactobacillus acidophilus* NCIMB 30175, and *Enterococcus faecium* NCIMB 30176	4 weeks	While multi-strain probiotic showed no effect in CD, it was associated with reduced intestinal inflammation in patients with UC	Bjarnason *et al*[43]
45	19–68	UC + CD	Kefir (*Lactobacillus, Lactobacillus kefir)*	4 weeks	Kefir consumption can modulate the intestinal microbiota. Regular consumption of kefir can improve both symptoms and quality of life in the short term in people with CD.	Yılmaz *et al*[46]
100	<18	UC	EcN	7 weeks	Data suggest no benefit from using EcN as an adjunctive therapy to conventional therapies for active UC. Treatment with EcN without a prior course of antibiotics resulted in fewer patients achieving clinical remission.	Petersen *et al*[44]
50	30–60	UC	*Bifidobacterium longum*536 (BB536)	8 weeks	The rate of clinical remission at week 8 was higher in patients receiving BB536. A significant decrease in UC disease activity index, Rachmilewitz endoscopic index, and UCDAI scores was observed in the BB536 group.	Tamaki *et al*[39]
60	35–69	UC	Strains *of Lactobacillus salivarius, Lactobacillus acidophilus* ve *Bifidobacterium bifidu s*BGN4)	2 years	The combined use of anti-inflammatory drugs and probiotics may be an alternative to corticosteroids in mild to moderate UC as a long-term treatment method.	Palumbo *et al*[38]
133	≤19	UC	EcN	8 weeks	Although the number of patients achieving the primary endpoint did not differ between the EcN and placebo groups, significantly fewer patients in the EcN group exhibited low IBDQ scores. However, a significantly higher number of patients in the EcN group showed clinical response at week 4 and endoscopic remission at week 8.	Park *et al*[40]
165	23–52	CD	*Saccharomyces boulardii*	52 weeks	Although the probiotic yeast *S. boulardii* is safe and well-tolerated, it did not appear to have any beneficial effects for patients with CD in remission after steroid or salicylate treatments.	Bourreille *et al*[45]
-	8–10 weeks old mice	UC + CD	*Saccharomyces maya* (genetically modified)	1 week	These self-adjustable engineered probiotic yeasts suppress experimental intestinal inflammation in mice and may provide a novel therapeutic platform for IBD and other inflammatory disorders.	Scott *et al*[47]
26 C57BL/6 (mice)	6–8 weeks old	UC	*Saccharomyces cerevisiae* (genetically modified)	1 week	Engineered *S. cerevisiae* administration reduced DSS-induced colitis in mice by suppressing macrophage pyroptosis and modulating the intestinal microbiota; it shows that it is an effective and safe treatment strategy for UC.	Sun *et al*[48]
32 (mice)	7 weeks old	UC	*Lactobacillus plantarum* HNU082 (Lp082)	15 days	The study showed that Lp082 had a better therapeutic effect than SASP on DSS-induced UC, and the treatment significantly ameliorated DSS-induced UC in mice.	Wu *et al*[41]

CD, Crohn's disease; DSS, dextran sulphate sodium; EcN, Escherichia coli Nissle; IBDQ, IBD Quality of Life Questionnaire; SASP, sulfasalazine; UC, ulcerative colitis; UCDAI, Ulcerative Colitis Disease Activity Index.

Author Contributions

Z.Z.B. contributed to the conceptualization, literature review, draft of the first version of the review and editing. B.B.A. contributed to literature review, review writing and editing. E.C. contributed to review writing and editing. All authors provided comments, guidance, and advice in updating manuscript drafts to the final version.

Conflicts of Interest

The authors declare no conflicts of interest.

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