International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	EXPLORING THE THERAPEUTIC POTENTIAL OF FECAL MICROBIOTA TRANSPLANTATION IN INFLAMMATORY BOWEL DISEASE
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4160
RECEIVED	02 October 2025
ACCEPTED	03 November 2025
PUBLISHED	07 November 2025
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EXPLORING THE THERAPEUTIC POTENTIAL OF FECAL MICROBIOTA TRANSPLANTATION IN INFLAMMATORY BOWEL DISEASE

Agata Mytych *(Corresponding Author, Email: agata.mytych@student.umw.edu.pl)*
Wroclaw Medical University, Poland
ORCID ID: 0009-0004-4575-6327

Julia Groszewska
Medical University of Lodz, Poland
ORCID ID: 0009-0002-4637-1264

Michał Romaniuk
Medical University of Lodz, Poland
ORCID ID: 0009-0008-6002-000X

Agata Rapior
Medical University of Lodz, Poland
ORCID ID: 0009-0002-0300-2303

Daria Julia Makowska-Woszczyk
Medical University of Lodz, Poland
ORCID ID: 0009-0004-9897-0618

Kinga Lubomska
Medical University of Lodz, Poland
ORCID ID: 0009-0002-8777-5273

Patrycja Jagura
Medical University of Lodz, Poland
ORCID ID: 0009-0008-2394-3673

Jan Romaniuk
Medical University of Lublin, Poland
ORCID ID: 0009-0000-3017-3330

Marta Dziedziak
Wroclaw Medical University, Poland
ORCID ID: 0009-0004-3463-2804

Łukasz Nosek
Wroclaw Medical University, Poland
ORCID ID: 0009-0006-8294-5842

ABSTRACT

Background: Inflammatory bowel disease (IBD), including ulcerative colitis (UC) and Crohn's disease (CD), is a chronic, relapsing inflammatory disorder of the gastrointestinal tract associated with dysbiosis of the gut microbiota. Fecal microbiota transplantation (FMT) has emerged as a microbiome-based therapeutic approach aimed at restoring a healthy microbial ecosystem.
Methods: A narrative review was performed, incorporating recent randomized controlled trials, cohort studies, and meta-analyses published in peer-reviewed journals. The review focused on studies investigating FMT as a therapeutic approach for inducing or maintaining IBD.
Results: Originally validated for recurrent Clostridioides difficile infection, FMT has shown promising results in IBD, particularly in inducing clinical and endoscopic remission in patients with active UC. Evidence suggests that treatment response is influenced by factors including donor microbiota composition, disease severity, baseline microbiome of recipients, and administration protocols. Despite encouraging outcomes, heterogeneity in study design, stool preparation, delivery methods, and treatment schedules limits definitive conclusions. Moreover, the efficacy of FMT for maintenance of remission in UC or induction and maintenance of remission in CD remains uncertain. Safety data are generally favorable in the short term, though long-term risks and standardized procedural protocols require further investigation.
Conclusions: Overall, FMT offers a unique strategy to modulate gut microbial composition and investigate causal relationships in IBD pathogenesis, but well-designed, large-scale studies are needed to establish optimized protocols, long-term efficacy, and safety across diverse patient populations.

KEYWORDS

Crohn's Disease, Inflammatory Bowel Disease, Fecal Microbiota Transplantation, Ulcerative Colitis, Gut Microbiota

CITATION

Agata Mytych, Julia Groszewska, Michał Romaniuk, Agata Rapior, Daria Julia Makowska-Woszczyk, Kinga Lubomska, Patrycja Jagura, Jan Romaniuk, Marta Dziedziak, Łukasz Nosek. (2025) Exploring the Therapeutic Potential of Fecal Microbiota Transplantation in Inflammatory Bowel Disease. *International Journal of Innovative Technologies in Social Science.* 4(48). doi: 10.31435/ijitss.4(48).2025.4160

Introduction

IBD, encompassing UC and CD, is a chronic, relapsing inflammatory disorder of the gastrointestinal tract. Its global prevalence has been steadily rising, currently affecting approximately 3 million individuals in the United States and 2.5 million in Europe [49]. Although the precise etiology remains unclear, IBD is widely recognized as a multifactorial disease involving complex interactions among genetic susceptibility, environmental influences, and the intestinal microbiota [47-49].

The gut microbiome is a complex and dynamic ecosystem that exists in close symbiosis with the host, playing a critical role in maintaining intestinal homeostasis. Disruption of this balance, known as dysbiosis, alters both the composition and function of the microbial community, impairing host–microbe interactions and contributing to disease development. Increasing evidence indicates that such microbial disturbances are central to the initiation and progression of IBD [47-49].

Patients with IBD commonly exhibit reduced microbial diversity, lower levels of anti-inflammatory bacteria, and an increase in pro-inflammatory bacterial species. This dysbiotic state contributes to mucosal inflammation and disease persistence [1,3,5,7,13].

FMT, also known as microbiome restoration therapy, fecal transplantation, human intestinal microbiota transfer, or fecal bacteriotherapy, has emerged as a promising microbiome-based therapeutic approach for IBD, given the strong association between gut microbial composition and intestinal inflammation [5]. The procedure involves administering processed fecal material from healthy donors into the gastrointestinal tract of affected individuals through various delivery routes, including colonoscopy, enema, or oral capsules. Initially recognized as a highly effective therapy for recurrent Clostridioides difficile infection (rCDI), FMT has shown encouraging results in clinical studies, with improvements in clinical remission and mucosal healing among

patients with IBD. However, variability in donor selection, preparation methods, administration routes, and treatment schedules has led to inconsistent outcomes, highlighting the need for standardized protocols and further research [1-7].

Gut microbiota

The human microbiota constitutes a highly diverse and dynamic community of microorganisms—including bacteria, archaea, fungi, and viruses—that inhabit multiple body sites such as the skin, oral cavity, respiratory tract, urogenital tract, and particularly the gastrointestinal tract. Among these, the gut microbiota is the most complex and metabolically active, comprising hundreds of bacterial species dominated by the phyla Firmicutes and Bacteroidetes, along with Proteobacteria and Actinobacteria [14,15,16].

Microbial colonization begins at birth and is strongly shaped by the mode of delivery. Vaginally delivered infants acquire microbial communities resembling their mother's vaginal and intestinal flora, rich in Lactobacillus species, whereas cesarean-delivered infants are colonized predominantly by skin-associated taxa such as Staphylococcus, Corynebacterium, and Propionibacterium. Early nutrition further influences microbial development: breastfed infants exhibit an abundance of Bifidobacterium species that diversify after weaning, while formula-fed infants tend to have lower levels of Bifidobacteria and higher proportions of Bacteroides, Clostridium difficile, and coliform bacteria [10,17].

In adulthood, the composition of the gut microbiome remains susceptible to modification by factors such as diet, antibiotic exposure, infections, pollutants, and psychosocial stress. Diet is particularly influential—plant-based, fiber-rich diets rich in micronutrients like magnesium are associated with reduced levels of pro-inflammatory taxa (e.g., Escherichia coli, Clostridium innocuum) and enhanced growth of beneficial anaerobes such as Faecalibacterium prausnitzii and Agathobaculum butyriciproducens. In contrast, diets high in animal-derived fats and proteins promote the expansion of bile-tolerant microorganisms, including Alistipes, Bilophila, and Bacteroides [8-12,15,17,18].

Microbiota and gut immunity

Consistent evidence from both human and animal studies demonstrates that the establishment of the intestinal microbiota is critical for proper immune system development and may influence susceptibility to IBD. In the absence of microbial exposure, germ-free (GF) animals exhibit profound defects in gut-associated lymphoid tissue (GALT) development, including disrupted formation of crypt patches and isolated lymphoid follicles, as well as markedly reduced Peyer's patches and germinal centers [2,49-52,60].

GF mice also show significant reductions in key components of mucosal immunity, such as immunoglobulin A (IgA), Th17 cells, and B cells. These immune elements, however, are rapidly restored following microbial colonization. IgA plays a central role in maintaining intestinal homeostasis by forming a protective mucosal barrier, neutralizing pathogens and toxins, modulating the composition of the gut microbiota, and supporting the stable engraftment of commensal species. Certain commensals, including Segmented Filamentous Bacteria (SFB), are particularly important in stimulating mucosal T cells to produce IL-17, forming Th17 cells. While Th17 cells are essential for defense against pathogens, under inflammatory conditions they can exacerbate immune responses and contribute to intestinal inflammation [2,49-52,55-56].

Early-life exposure to a diverse and balanced microbiota is also crucial for long-term resistance to chemically induced colitis. In germ-free mice, the absence of microbial colonization leads to abnormal accumulation of invariant natural killer T (iNKT) cells in the colonic lamina propria, resulting in exaggerated inflammatory responses and increased severity of oxazolone-induced colitis compared with conventionally raised animals [53-54].

Beyond these direct immune interactions, microbial metabolites play a critical role in regulating host immunity. Short-chain fatty acids (SCFAs)—including acetate (C2), propionate (C3), and butyrate (C4)—are particularly important for maintaining intestinal barrier integrity and modulating inflammatory processes. Butyrate supports epithelial stability by promoting tight junction protein expression, likely through activation of the AMP-activated protein kinase pathway or suppression of claudin-2. Both acetate and butyrate further strengthen the mucosal barrier by stimulating mucin secretion. SCFAs also influence immune signaling through toll-like receptors (TLRs), free fatty acid receptors, G protein–coupled receptors, and histone deacetylases, modulating pathways such as mitogen-activated protein kinase (MAPK), c-Jun N-terminal kinase (JNK), and nuclear factor kappa B (NF-κB). Through these mechanisms, SCFAs regulate the production of inflammatory and oxidative mediators, including IL-8, IL-6, tumor necrosis factor (TNF), monocyte chemoattractant protein-1 (MCP-1), and inducible nitric oxide synthase (iNOS) [2, 12,57-59].

Microbial signals not only shape host immune cells but also coordinate microbial gene expression, collectively directing the production and secretion of cytokines, chemokines, and immune receptors. Although introducing specific pathogen-free microbiota later in life can partially restore immune function in germ-free animals, transcriptional profiles in the jejunum and colon remain distinct from those of conventionally raised counterparts. This persistent divergence highlights a critical developmental window during which microbial exposure is essential for the proper maturation of mucosal and immune structures [2,53,60].

Gut dysbiosis in IBD

IBD, comprising CD and UC, are chronic, relapsing inflammatory disorders of the gastrointestinal tract that arise from a disruption in the balance between the intestinal immune system and the gut microbiota in genetically susceptible individuals. Aberrant mucosal immune activation, resulting from impaired tolerance to commensal microorganisms or dysfunction of the epithelial barrier, drives persistent inflammation and contributes to disease progression. Increasing evidence indicates that intestinal microbial dysbiosis—characterized by reduced diversity and an imbalance between beneficial and pathogenic species—plays a pivotal role in the initiation and perpetuation of these disorders [21–22,24].

Most studies report a marked reduction in microbial diversity in both CD and UC patients compared to healthy individuals. Another defining feature of intestinal dysbiosis in IBD is the marked reduction of butyrate-producing bacteria, accompanied by an expansion of sulfate-reducing microorganisms. The overrepresentation of lipopolysaccharide (LPS)-producing taxa further contributes to mucosal inflammation by activating Toll-like receptor 4 (TLR4)–mediated signaling, leading to NF-κB activation and sustained pro-inflammatory responses [48]. Moreover, the increased abundance of sulfate-reducing bacteria exacerbates intestinal injury through the production of hydrogen sulfide, which interferes with butyrate oxidation, disrupts immune homeostasis, and promotes bacterial persistence within the gut mucosa [47].

Among the Firmicutes, the Clostridium leptum group (cluster IV), particularly Faecalibacterium prausnitzii, is notably reduced in IBD. A decrease in F. prausnitzii—a key SCFA-producing bacterium—compromises the integrity of the intestinal epithelial barrier, leading to increased gut permeability and enhanced bacterial translocation into the lamina propria. This disruption also hinders the differentiation of regulatory T cells (Tregs), which play a crucial role in maintaining immune tolerance [47]. Concurrently, Proteobacteria are typically increased, reflecting a shift toward a pro-inflammatory microbial environment [2,19–22,24]. Beneficial genera such as Roseburia, Eubacterium, and Bifidobacterium are diminished, while pathogenic bacteria including Escherichia coli, Ruminococcus gnavus, and Clostridium spp. are often enriched [21–22,24].

Certain bacterial species have also been strongly associated with the occurrence of IBD. For example, Mycobacterium avium subspecies paratuberculosis has been implicated in the development of Crohn's disease, whereas Fusobacterium varium has been linked to ulcerative colitis [46].

The gut microbiota of IBD patients also displays temporal instability. In contrast to the relatively stable microbial profiles of healthy individuals, IBD microbiota composition fluctuates between active and quiescent disease states and remains unstable even during remission. Before relapse, decreases in normal anaerobes such as Bacteroides, Eubacterium, Lactobacillus, and Ruminococcus have been observed, alongside an overall reduction in microbial richness [19].

Beyond bacteria, dysbiosis in IBD extends to the fungal and viral communities. Increased levels of Candida albicans and Malassezia restricta have been reported, accompanied by reductions in beneficial fungi such as Saccharomyces cerevisiae. Moreover, expansion of Caudovirales bacteriophages correlates with reduced bacterial diversity and heightened inflammation, particularly in UC. Patients with ileal CD often exhibit fungal overgrowth at the expense of bacterial populations, whereas UC and non-ileal CD cases show decreased fungal diversity [2,3,19–23].

Environmental and therapeutic factors further modulate dysbiosis. Drugs such as mesalazine can significantly reduce total bacterial load, while antibiotics and bowel rest exacerbate compositional imbalances [2]. Inflammation itself alters gut conditions—inducing oxidative stress, nutrient depletion, and changes in oxygen levels—that favor the proliferation of pro-inflammatory microbes, including adherent-invasive E. coli, Proteobacteria, Veillonellaceae, Ruminococcus gnavus, Fusobacterium, and Pasteurellaceae [19–22].

FMT

FMT is a therapeutic approach designed to restore a healthy gut microbial balance by transferring fecal microbiota from a healthy donor into the gastrointestinal tract of a patient [19,25].

The therapeutic use of fecal material dates back over 1,600 years. The earliest known record originates from 4th-century China, where the physician Ge Hong described administering a fecal suspension, referred to as "yellow soup," to treat patients suffering from severe diarrhea and food poisoning. By the 16th century, Li Shizhen further documented the use of fecal preparations for managing gastrointestinal disorders such as constipation, abdominal pain, vomiting, and fever [5,18,27]. Similar concepts appeared in 17th-century Europe, when Fabricius Acquapendente noted that transferring rumen contents between animals could restore digestive function—an early example of what would later be termed "transfaunation" in veterinary medicine. Such procedures were eventually used to treat diarrhea and other gastrointestinal illnesses in livestock, including horses, cows, and alpacas. During World War II, anecdotal reports described Bedouins in North Africa recommending the ingestion of fresh camel feces to German soldiers with bacterial dysentery, reflecting a continued empirical understanding of microbial therapy. The modern scientific foundation for fecal transplantation, however, emerged in the early 20th century, following Élie Metchnikoff's work on the beneficial roles of microbes in human health. The first documented medical application of FMT occurred in 1958, when Dr. Ben Eiseman and colleagues successfully treated patients with pseudomembranous colitis using fecal enemas after antibiotic failure. This marked the formal beginning of FMT in contemporary medicine [5,18,26].

Nowadays, FMT has emerged as a validated and highly successful therapeutic strategy for rCDI. Numerous randomized controlled trials (RCTs) and meta-analyses have consistently confirmed its effectiveness, leading to its inclusion in international guidelines as a treatment for patients with multiple recurrences [28-33,36]. For instance, a meta-analysis conducted by Porcari et al. evaluated 15 studies encompassing 777 patients and demonstrated that FMT achieved high cure rates in recurrent Clostridioides difficile infection, with an overall success rate of 81% following a single treatment and 92% when multiple FMT procedures were considered across nine studies involving 354 patients [35].

FMT in IBD

Over the past decade, multiple randomized controlled trials (RCTs) have evaluated the therapeutic efficacy of FMT in IBD. The majority of these investigations have focused on ulcerative colitis (UC) [2,18].

The earliest documented use of FMT for UC was reported in 1989, when one of the study's authors self-administered the treatment for refractory disease, achieving complete, drug-free remission [38].

In a 2014 study by Ianiro et al., 133 patients received FMT, including 77 with UC, 53 with Crohn's disease CD, and 3 with indeterminate IBD. Most participants were refractory to standard therapy or dependent on medication. Among them, 57 patients (43%)—25 with UC, 31 with CD, and 1 with unclassified IBD—had recurrent or rCDI. Overall, FMT led to a 71% reduction in clinical symptoms, which remained consistent (69%) after excluding CDI cases. However, interpretation of these findings is limited by methodological heterogeneity, incomplete procedural documentation, and poorly defined endpoints. Most patients underwent bowel preparation with polyethylene glycol or unspecified antibiotics prior to FMT. Delivery routes included upper gastrointestinal administration (n = 42), enema (n = 20), colonoscopy (n = 23), and combined upper and lower approaches (n = 11) [39].

A separate 2014 analysis evaluated 122 patients with inflammatory bowel disease (IBD), excluding three individuals who were unable to tolerate FMT administered via enema. The remaining 119 patients were stratified by disease severity into mild/mild–moderate (n = 27, 23%), moderate/severe (n = 16, 13%), and severe (n = 19, 16%) categories. Among these, 10 patients (8%) had therapy-refractory disease, 44 (37%) presented with active disease, and 5 (4%) had refractory pouchitis. Following fecal microbiota transplantation, clinical remission was achieved in 54 of 119 patients (45%), while mucosal healing was documented in 12 of the 16 patients (75%) [40].

In 2017, Paramsothy et al. reviewed 555 patients with UC across 42 studies examining FMT, including 9 case reports, 4 randomized controlled trials, 5 case series, and 24 prospective cohort studies (20 uncontrolled and 4 controlled). Across all studies, clinical remission was observed in 36% of patients (201/555) . In a meta-analysis of 24 cohort studies involving 307 patients, the pooled clinical remission rate was 54%, with moderate heterogeneity among studies [41].

Further insights into microbial determinants of response were reported in 2019. Patients achieving remission exhibited gut microbiota enriched in Eubacterium and Roseburia, higher levels of short-chain fatty

acids, and enhanced secondary bile acid biosynthesis, whereas non-responders had elevated abundances of Fusobacterium, Sutterella, and Escherichia species. Donor stool composition also influenced outcomes: Bacteroides species were associated with clinical remission, whereas the presence of Streptococcus correlated with lack of response [42].

More recently, a 2023 Cochrane review by Imdad et al. evaluated 12 studies with 550 participants to assess FMT for both UC and CD. FMT increased rates of clinical remission in active UC compared to controls (risk ratio [RR] 1.79, 95% CI 1.13–2.84), although the evidence was of low certainty. Endoscopic remission may also be improved (RR 1.45, 95% CI 0.64–3.29). FMT appeared to have little effect on adverse events (RR 0.99, 95% CI 0.85–1.16), while evidence regarding serious adverse events, quality of life, and maintenance of remission in UC or CD was very uncertain. Overall, this recent analysis supports a potential benefit of FMT for inducing remission in active UC, but highlights the need for further well-designed trials to clarify long-term efficacy, safety, and its role in CD [43].

In addition, a systematic review and meta-analysis of 14 studies (10 randomized, 4 non-randomized) demonstrated that multi-donor FMT (MDN) was more effective than single-donor FMT (SDN) for inducing remission in IBD. Both MDN and SDN were superior to placebo (RRs 4.41 and 1.57, $P \leq 0.001$), with MDN outperforming SDN (RR 2.81, $P = 0.005$). Analysis of the 10 high-quality studies confirmed MDN's superiority (RR 2.31, $P = 0.042$) [44].

On the other hand, a review and meta-analysis of six high-quality RCTs involving 324 patients found no significant differences in outcomes based on donor type (single vs. multiple), FMT preparation (fresh vs. frozen), or delivery route. Overall, FMT was associated with a significant benefit in inducing combined clinical and endoscopic remission compared with placebo (odds ratio 4.11; 95% CI 2.19–7.72; $P < 0.0001$). Subgroup analyses indicated that pre-FMT antibiotics, bowel lavage, concomitant biologic therapy, and topical rectal therapy did not influence remission rates. Clinical remission, response, and endoscopic outcomes were all significantly improved with FMT versus placebo, without increased risk of serious or specific adverse events [45].

Limitations

Despite the proven efficacy of FMT in certain gastrointestinal conditions, several barriers continue to limit its routine clinical use. One of the key challenges is the absence of standardized treatment protocols. Variations in disease type, patient physiology, and methodological approaches contribute to inconsistent therapeutic outcomes. The success of FMT depends on numerous factors, including patient selection, donor eligibility, pre-procedure preparation, stool processing, route of administration, and the number or volume of infusions administered. Differences across these parameters make it difficult to compare study results and establish universal guidelines [2,18,34].

While FMT has shown encouraging results, particularly in Clostridioides difficile infection, evidence supporting its use in IBD, including UC, remains less robust. Many UC studies are limited by small sample sizes, heterogeneous study designs, and inconsistent endpoints, which may overestimate therapeutic efficacy. Larger, rigorously controlled trials are therefore necessary to validate these findings and define standardized clinical endpoints [2,18,34].

Donor variability is also a major determinant of treatment response. Whereas diverse donor material is often sufficient for treating recurrent C. difficile infection, differences in host–microbe interactions and the complex pathophysiology of IBD likely account for the inconsistent efficacy observed across UC trials. Additional factors—such as the recipient's baseline microbiome composition, disease severity, and concurrent medications—may further influence outcomes. Post-hoc analyses have suggested that patients with milder disease activity, left-sided colitis, lower fecal calprotectin levels, and no prior exposure to biologic therapy tend to respond more favorably to FMT [34,37].

Procedural heterogeneity adds another layer of complexity. Variations in stool preparation (aerobic, anaerobic, or washed microbiota), delivery routes (colonoscopy, enema, oral capsules), frequency of administration, and antibiotic pre-conditioning all contribute to inconsistent efficacy. Although colonoscopic delivery often yields higher response rates, similar remission outcomes have been reported with repeated or even single intensive administrations. The use of antibiotics before FMT remains debated, as pre-conditioning may alter both microbial engraftment and treatment safety [18,34,37].

Furthermore, the long-term safety of FMT remains insufficiently defined. Although short-term outcomes are generally favorable, data on long-term risks are limited. Concerns persist regarding the potential transmission of infectious agents or unforeseen microbiome-related complications, particularly in cases where donor screening or testing protocols are inadequate. Continued research and standardization are therefore essential to ensure both the efficacy and safety of FMT in clinical practice [17,18].

Conclusions

With the growing body of research on FMT, clinical evidence increasingly supports its potential as a therapeutic strategy for various gastrointestinal disorders. Current trials suggest that FMT may improve clinical and endoscopic outcomes in patients with active UC. Unlike prebiotics or probiotics, FMT introduces a complete, healthy microbial ecosystem, offering unique advantages by restoring overall microbial balance rather than targeting individual strains. This approach also provides a valuable tool for investigating causal relationships between the microbiota and disease progression. Despite these promising observations, evidence regarding the efficacy of FMT for maintenance of remission in UC, as well as induction and maintenance of remission in CD, remains highly uncertain. Similarly, the impact of FMT on quality of life and risk of serious adverse events has not been conclusively established. One of the major challenges in implementing FMT is the absence of standardized treatment protocols, with variations in disease subtype, individual patient characteristics, and methodological approaches contributing to inconsistent therapeutic outcomes. Further well-designed studies are required to clarify the therapeutic benefits, safety profile, and long-term potential of FMT in both adult and pediatric patients with IBD [1,2,5,18,34,47].

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