Review Article

The current landscape of fecal microbiota transplantation in treating inflammatory bowel disease

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Abstract: Inflammatory bowel disease (IBD), which includes ulcerative colitis (UC) and Crohn's disease (CD), is a chronic, immune-mediated disorder that impacts the intestinal tract. The gut microbiota, a diverse community of microorganisms, plays a pivotal role in the initiation, development, and progression of IBD by modulating inflammation, and immune responses, and maintaining gut homeostasis. Dysbiosis, or an imbalance in the gut microbiota, is frequently observed in IBD patients and is believed to contribute to the pathogenesis of the disease by disrupting the mucosal immune system. Fecal microbiota transplantation (FMT) involves transferring feces from a healthy donor (HD) into a recipient and has emerged as a promising therapeutic approach for IBD. The primary goal of FMT is to restore microbial balance in the recipient's gut, improving both microbiota composition and immune function. Numerous preclinical and clinical studies have demonstrated varying degrees of success in alleviating IBD symptoms through FMT. The benefits of FMT include modulation of gut bacteria abundance, restoration of microbial diversity, and enhancement of immune system regulation, all of which contribute to reducing IBD-related inflammation. This review presents a comprehensive analysis of animal studies and clinical trials exploring using FMT as a treatment for IBD. Understanding the underlying mechanisms of FMT in IBD is crucial for designing effective therapeutic strategies and optimizing its clinical impact.

Keywords: Inflammatory bowel disease (IBD); ulcerative colitis (UC); Crohn's disease (CD); fecal microbiota transplantation (FMT); inflammation

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Introduction

Inflammatory bowel disease (IBD) is a chronic, immune-mediated gastrointestinal disorder that is composed of two clinically and morphologically distinct entities: ulcerative colitis (UC) and Crohn's disease (CD) (1,2). The incidence of IBD has progressively increased over the past decades in developed and newly industrialized countries and its chronic feature leads to the need for long-term and expensive monitoring and treatment (3,4). The pathogenesis of IBD is multifactorial involving genetic, immunological, infectious, environmental factors, and the gut microbiota (5,6).

Currently, the primary treatment approaches for IBD include pharmacological therapy and surgical intervention (7,8). Pharmacological approaches mainly include anti-inflammatory drugs and biological agents, such as tumor necrosis factor-α antagonists, 5-aminosalicylic acid, corticosteroids, anti-interleukins, and immunosuppressive and biological agents (9). Although these drugs can temporarily maintain and reduce inflammatory episodes, they potentially elicit some serious side effects, including an increased risk of infection and certain cancers, and do not cure the underlying cause of IBD (10,11). Surgery, on the other hand, is not considered a first-line option for all patients with IBD and is typically reserved for cases where medical treatment has proven ineffective or serious complications have arisen. Even after surgery, patients may experience complications that require ongoing medication to maintain disease control and prevent relapse (12). Accordingly, approaches targeting the modulation of host-microbes catabolites, including fecal microbiota transplantation (FMT), probiotics, synbiotics, antibiotics, and micro-nanocarriers, are expected to provide novel strategies for IBD treatment (13,14).

The fundamental role of the intestinal microbiota in the development and progression of IBD inflammation is gradually being explored and recognized (15,16). The intestinal microbiota is a complex multicellular community (17). About 3.3 million microbial genes, up to 10 bacterial phyla, and 1,000 species of bacteria (>90% of the species belong to the phylum *Bacteroidetes* and *Firmicutes*), as well as commensal fungi and viruses, have been identified in the human intestine (18). Compared with normal controls, the number, species, and diversity of intestinal microbiota are characteristically altered in IBD, with a decrease in the abundance of anti-inflammatory bacteria and an increase in the abundance of pro-inflammatory bacteria, with a highly unstable state environment (19). For example, the number of the dominant flora, *Firmicutes*, and *Bacteroides*, decreases significantly in the intestine of patients with IBD, while the number of non-dominant flora, such as Actinomycetes, tends to increase. In addition, *Roseburia spp*. is significantly reduced in the gut microbiota of healthy individuals with a high genetic risk of IBD (20).

FMT is also known as fecal bacterial therapy, human probiotic infusion, fecal transplantation, intestinal microbiome restoration, and fecal transfer. FMT is designed to modify the composition and function of the recipient's intestinal microbiota by collecting fecal matter from a pre-screened healthy donor (HD) and delivering prepared serums to the patient's gastrointestinal tract via nasogastric tube, colonoscopy, or enema (21). FMT has recently gained popularity for its successful treatment of *Clostridioides difficile* infection (CDI) and has also encouraged its application in patients with IBD to rebuild the microflora balance in patients with IBD (21). Thus, FMT has emerged as a promising effective method for treating IBD.

The mechanism of FMT in the treatment of IBD is still being explored. Several basic and clinical studies have been conducted to confirm the effectiveness of FMT to varying degrees. Overall, FMT can not only restore normal ecological and metabolic functions associated with beneficial commensal bacteria but also activate multiple anti-inflammatory mechanisms targeting mucosal immune cell types. Therefore, this review highlights the roles and mechanisms of FMT in improving IBD, hoping to fuel and foster the development of FMT strategies for IBD.

Preclinical studies of FMT in the treatment of IBD

Existing animal studies are mainly based on dextran sulfate sodium (DSS)-induced models of IBD in mice or rats. The gut microbiota reshapes the immune microenvironment in IBD by engaging in innate and adaptive immune responses (22). For instance, the immune-related Th17 function was impaired by bacterially produced bile acids, which is correlated to inflammatory pathophysiology in IBD (23). Immunomodulation of the intestine and improvement of the inflammatory state of IBD can be achieved through a rational microbiota-based therapeutic strategy with FMT, probiotic supplementation, and engineered microbiome (24).

In a DSS-induced IBD mouse model, Burrello *et al.* (25) found that FMT intervention resulted in downregulated proinflammatory genes, antimicrobial peptides genes, mucins genes, interferon-gamma (IFN-γ), interleukin-17 (IL)-17, IL-13, and increased IL-10 levels, with recovered microbial ecologies. In the DSS-induced IBD mouse model, FMT intervention with HD bacteria significantly decreased the inflammatory myeloperoxidase (MPO) and increased IL-10 expression (26). In the 2,4,6-trinitrobenzenesulfonic acid (TNBS)-induced IBD rat model, Qiu *et al.* (27) demonstrated that FMT inhibited transforming growth factor-beta 1 (TGF-β1) while upregulating anti-inflammatory Smad proteins, leading to improved disease activity and histology. Thus, it proved that FMT was a

feasible method to inhibit IBD inflammation via suppressing TGF-β1/Smad pathway. FMT intervention could alleviate colitis symptoms by remodeling cytokine homeostasis, anti-inflammatory bacteria, and metabolites, to defend the intestinal mucosal barrier for lowering the sensitivity of DSS-induced colitis. Therefore, FMT could suppress chronic intestinal inflammation by regulating immune function and the composition of the intestinal flora.

Additionally, Huang *et al.* (28) established a mouse model of DSS-induced colitis and administered FMT therapy. The study found that FMT significantly alleviated colitis symptoms in the mice, improved the disease activity index, body weight, and colon length, and effectively restored the pathological structure of the colon tissue. Besides, FMT therapy partially restored the expression of the tight junction protein ZO-1 and promoted the repair of goblet cells, thereby enhancing the intestinal mucus secretion function. DSS mice exhibited a high proportion of IgA/G-targeted bacteria, which returned to near-normal levels following FMT treatment. FMT also significantly reduced the proportion of IgA/IgG$^+$ B cells, suggesting that FMT might restore the intestinal immune response by regulating the function of IgA/IgG$^+$ B cells. Overall, FMT improved DSS-induced colitis and exerted a therapeutic effect by modulating the IgA/G-mediated immune response.

In the DSS-induced mouse IBD model, Ihekweazu *et al.* (29) found that triple-Bacteroides cocktail combination therapy was better than conventional FMT in terms of weight loss and mortality protection. *Bacteroides ovatus* ATCC 8483 was more effective than any single strain or combination of strains in avoiding weight loss, diminishing tissue damage, suppressing the inflammatory response, and promoting epithelial cell recovery. Moreover, Xu *et al.* (30) transplanted feces from donor mice with high diversity and abundance of *Bacteroides* and *Faecalibacterium* into a mouse model of colitis induced by DSS, significantly improving intestinal damage and immune imbalance. The key strains in the donor feces were identified as *Bacteroides thetaiotaomicron* and *Faecalibacterium prausnitzii*. Furthermore, compared to single-bacterium therapy, dual-bacterium therapy notably enhanced intestinal healing, increased M2/M1 macrophage polarization, corrected Th17/Treg imbalance, elevated IL-10 levels produced by Treg cells, and increased lecithin abundance in the glycerophospholipid metabolic pathway. In conclusion, *B. thetaiotaomicron* and *F. prausnitzii* may alleviate colitis in mice by promoting lecithin production and regulating IL-10 levels in intestinal Treg cells.

Clinical studies of FMT in the treatment of IBD

FMT in treating CD

CD is a prevalent chronic inflammatory gastrointestinal disorder associated with genetic susceptibility, environmental factors, and dysregulated immune responses (31). Personalized patient management will be crucial for improving therapeutic outcomes in CD (32). FMT is an emerging, safe, and effective treatment for CD, enhancing gut microbiome diversity and abundance (33).

About 65–75% of CD patients may develop malnutrition or emaciation status, which implicitly means that dietary or nutritional therapy has an important positive impact on the care of CD (34). Exclusive enteral nutrition (EEN) can to some extent, augment the beneficial microbiota, improve bile acid metabolism, and reduce the stimulation of unfavorable diets (35). EEN is a well-established therapy in inducing remission in pediatric CD, while its usefulness as a principal regimen for adult CD is yet to be determined. Hoelz *et al.* (36) evaluated whether FMT could promote EEN-induced clinical remission in pediatric CD patients. This result indicated that high pathogen load, low abundance microbiome, and practical deficiencies of EEN-conditioned fecal material may lead to poor efficacy of FMT in the treatment of CD (36). FMT may not be a good means of promoting EEN in some pediatric CD cases. Sokol *et al.* (37) performed a single FMT or false transplantation for CD patients who had achieved clinical remission on corticosteroids. The steroid-free clinical remission rate in the FMT group was higher than that in the sham group. Considering the small sample size and standardized detection method, this conclusion was not statistically significant and needed further confirmation.

In our previous retrospective study, we investigated the clinical and endoscopic remission rates and mucosal healing rates after FMT supplemented with partial enteral nutrition (PEN) for active CD in children as first-line treatment, by enrolling 25 pediatric patients from November 2016 to July 2019 (38). In a cohort of pediatric CD patients with a small sample size, repeated and multiple FMT plus PEN could facilitate not only the induction and maintenance of remission but also mucosal healing. Nevertheless, this study was not a randomized controlled trial and did not focus on alterations in the species and abundance of intestinal bacteria, and further prospective work is required to validate these findings.

FMT in treating recurrent C. difficile infection (rCDI) with underlying IBD

CDI is a leading cause of hospital-acquired infection with diarrhea symptoms, accounting for considerable morbidity, mortality, and healthcare threat (39). The high incidence and difficulty in treating CDI and the frequent development of rCDI after successful treatment of CDI is making it a challenging clinical concern (40). Strategies to improve current and future management of rCDI are under clinical investigation and include narrow-spectrum antibiotics, monoclonal antibodies, and FMT, which aims to re-establish normal microbiota. The ability of FMT to effectively treat rCDI has motivated the exploration of scenarios for its application in treating rCDI with underlying IBD.

The collection, preparation, and storage of feces are critical to the effectiveness of FMT in treating rCDI (41). Costello *et al.* (42) found that in the environment of –80 ℃, the fecal microorganisms in the fecal suspension of normal saline (NS) and 10% glycerol almost did not change at 2–6 months, and the number of fecal bacteria in NS decreased significantly after 6 months, while the number of fecal bacteria in 10% glycerol solution was almost unchanged. After 2–10 months of frozen stool storage in 10% glycerol, rCDI cure rates were 88% after a single FMT and 100% after repeated FMT. For rCDI therapy, fecal bacteria frozen in 10% glycerol was a safely stored method for sustaining their activity for at least 6 months.

FMT could act as a safe and valid protocol to remodel the indigenous component of the intestinal microbial population in rCDI patients for the final utilization in treating concurrent IBD (43). Fareed *et al.* (44) reported the *16S rRNA* gene sequencing analysis among children with recurrent rCDI via FMT treatment, showing an increased abundance of *Bacteroidetes* and decreased Proteobacteria in post-FMT signatures. This study confirmed the efficacy of FMT with limited rCDI recurrence or re-infection in treating pediatric rCDI, accompanied by specific microbiome alteration during the FMT course. Gholam-Mostafaei *et al.* (45) evaluated changes in the gut microbiota after FMT in Iranian rCDI patients with underlying IBD. They performed the microbiological analysis of 21 fecal samples, including 14 samples before and after FMT and 7 samples from HDs, and found that the fecal microbiota characteristics of the recipients were more similar to those of the donor samples after transplantation. The patients with pre-FMT had higher frequencies of *Enterococcus spp*. and *Streptococcus spp*., and the relative abundance of anaphylactic bacteria was significantly increased after FMT, and the thick-walled phylum was significantly decreased. By treating 8 children (5 with underlying IBD) with FMT, Chen *et al.* (46) found a higher proportion of primary bile acids and a lower proportion of secondary bile acids in pre-FMT recipients compared to donors. After FMT, secondary bile acids gradually increased and primary bile acids gradually decreased. Thus, changes in bile acid profiles after FMT, compared to intestinal bacterial diversity, suggested that bile acids might contribute to FMT efficacy in different ways, which requires further investigation to explore the mechanism of FMT effectiveness.

In 113 adult patients with IBD undergoing FMT for rCDI, van Lingen *et al.* (47) reported a 39% decrease in IBD activity, a 71% CDI cure rate, and a 5% flare rate. During up to 2 years of follow-up, it was found that 27% of patients developed infections, 39% were hospitalized, 5% underwent colectomy, and 10% died with a median age of 72 years old. Therefore, FMT was safe and effective for rCDI in patients with IBD, without a few IBD exacerbations. Kelly *et al.* (48) performed FMT in 80 immunocompromised (IC) patients with recurrent, intractable, or severe CDI, and found that 89% of IC patients had remission after FMT and the majority of these patients with CDI had a remission (78%) after a single FMT, while 12 patients had serious adverse events (15%). Thus, FMT was effective in the treatment of CDI in a small number of IC patients with SAEs or related AEs and there were no associated infectious complications. Cho *et al.* (49) performed a retrospective study of FMT for rCDI in pediatric IBD. This study substantiated that FMT was a valid treatment for rCDI in children with IBD, but there seemed to be a certain late relapse rate of rCDI at day 60 post-FMT. In consideration of the small sample size and standardized detection method, this conclusion still needs to be further confirmed.

FMT is well recognized by patients and medical institutions as an effective treatment for rCDI, but there are still many unknown problems that need further studies, such as optimal parameters, safety, adaptation disease, complications, and the result of FMT (50). Furthermore, due to the differences in the diversity of gut flora and inconsistent maturation of the immune system between pediatric and adult populations, it is warranted to note that more large cohort studies are needed to determine the long-term remission of rCDI with FMT in pediatric patients with IBD and to seek out biomarkers and factors predicting relapse.

FMT in treating UC

UC is a lifelong inflammatory disease primarily affecting the rectum and colon, and its global incidence is increasing. Not only does this disease significantly affect patients' quality of life, but it can also lead to long-term complications (51). The main manifestation of UC is bloody diarrhea, which manifests as abdominal pain, diarrhea, and mucopurulent and bloody feces. Mutual interaction between environmental factors, genetics, and the immune system are important factors contributing to the inflammation of the intestinal mucosa (52). The imbalance of intestinal bacteria is closely related to the development of UC and is involved in the whole process of UC occurrence and development. The decrease in the diversity of bacteria and the abundance of dominant flora in UC patients, and the increase in multiple bacteria with pro-inflammatory effects, which are directly or indirectly involved in the inflammation (53). FMT by repeated administration and multiple donors is effective in relieving or curing active UC (54). The strength, dose, and long-term effectiveness of FMT on clinical outcomes of UC need to be further ascertained by large-scale multicenter investigations.

In a randomized controlled trial, compared with the placebo group, FMT treatment in active UC patients observably promoted a higher remission rate and more diverse microbiota phenotype, with good biosafety (55). This study confirmed the efficacy of FMT in curing active UC and indicated the necessity of focusing on fecal donors and UC time for ultimate outcomes. In a randomized and controlled trial at three hospitals in Australia, compared with the placebo group, multi-donor intensive FMT treatment in active UC patients observably promoted higher remission and endoscopic response rates, with fewer adverse reactions (56). FMT treatment invoked a more heterogeneous microbiota phenotype, with some bacterial taxa associated with clinical outcomes. Especially, the existence of *Fusobacterium spp*. was implicated in the deficiency of remission. Additionally, Mańkowska-Wierzbicka *et al.* (57), UC patients with multiple FMT from HD fecal microbiota, showed a higher amount of *Lactobacillaceaea*, *Micrococcaceae*, *Prevotellaceae*, and TM7 *phylum spp*. oral clone EW055, and decreased *Staphylococcaceae* and *Bacillaceae*, by using 16S rRNA sequencing. Multi-session FMT was conducive to the rebuilding of microorganism ecology and exhibited excellent efficacy in alleviating active UC. In the trial of Crothers *et al.* (58), compared with sham treatment, UC patients with encapsulated oral FMT (cFMT) showed a higher clinical remission and more significant changes in gut microbiota, with increased C-reactive protein (CRP) and decreased fecal calprotectin, IL-17A, and IFN-γ + MAIT cells. Thus, oral FMT was conducive to prolonging the persistence of the structural change index of the intestinal bacterial community and exhibited excellent efficacy in UC. Porcari *et al.* (59) reported that UC patients with rCDI infection showed negative Clostridium difficile toxin by FMT after 8 weeks. Finally, 32 patients with rCDI (91%) were cured by FMT, and sustained cure of repeat FMT with CDI was significant compared with single FMT. The study confirmed that FMT effectively treated rCDI infection in patients with concomitant UC. The majority of patients achieved remission or amelioration of UC symptoms, which further emphasized the importance of sequential FMT in combating UC with rCDI.

Dietary intervention may play a crucial role in enhancing the effectiveness of FMT in the treatment of UC. Leibovitzh *et al.* (60) compared two approaches to treating UC using the UC exclusion diet (UCED) combined with FMT: one with and one without donor dietary regulation. The results showed that the group receiving FMT from diet-regulated donors along with UCED experienced significant beneficial microbial changes and reduced gut inflammation, indicated by lower levels of fecal calprotectin. These findings suggested that incorporating dietary interventions for both donors and patients may enhance the effectiveness of FMT in treating UC.

FMT in treating CD and UC

In some studies, investigators have included both CD and UC patients and investigated the curative benefit of FMT for these patients. Karolewska-Bochenek *et al.* (61) reported that pediatric patients with IBD including 8 patients with UC and 2 patients with CD colitis via FMT for 2 weeks, showing nine out of 10 patients (7 with UC and 2 with CD) had a clinical remission, and half had reduced calprotectin, with only a few had short-term, self-limited side effects. This study substantiated that short-term enhancement of FMT was a valid treatment for IBD children. Allegretti *et al.* (62) enrolled 15 patients with CD colitis and 24 patients with UC to be treated by FMT, confirming that 33 out of 49 patients (11 with CD and 22 with UC) had a clinical remission, and 15 patients had no change in condition, with only 1 patient had new symptoms and no adverse events occurred. After FMT, the alpha diversity

of feces increased significantly, the primary bile acids decreased, and the secondary bile acids increased. This study substantiated that FMT was a safe and effective treatment for IBD after rCDI and that FMT presented a more therapeutic role in UC patients.

What is coming for FMT and derived therapies

Before the advent of microbiome-based therapies, the clinical prognosis for rCDI was poor. Traditional treatment regimens primarily rely on antibiotics, which effectively eliminate toxin-producing bacteria but cannot eradicate bacterial spores (63). Upon spore germination, the emergence of new bacterial populations can lead to recurrent infections. In this context, FMT emerged as a pivotal therapeutic approach, offering one of the most effective and cost-efficient solutions for rCDI.

Rebyota, the first Food and Drug Administration (FDA)-approved FMT-derived therapy for CDI, represents a significant milestone in the clinical application of FMT (64). Composed of feces from qualified donors, Rebyota contains no antibiotics and is administered via a single rectal dose (65). The therapy is designed to help patients restore their gut microbiota balance, thereby preventing CDI recurrence. In a clinical trial involving 262 patients, 70.6% of those in the Rebyota group successfully avoided CDI recurrence over 8 weeks, compared to 57.5% in the placebo group (66). Furthermore, more than 90% of patients who experienced successful treatment remained free from CDI recurrence for up to 6 months (66). These findings validate the efficacy and safety of Rebyota in clinical practice.

Following Rebyota, Vowst has emerged as the second FDA-approved microbiome-based therapy for CDI (67). Unlike Rebyota, which is administered rectally, Vowst is taken orally in capsule form (68). The capsules contain firmicute spores that are screened and purified from the stool of HDs. Vowst has shown promise in reducing CDI recurrence by restoring the gut microbiome and promoting bile acid profiles that inhibit C. difficile spore germination. In a phase 3, double-blind, randomized, placebo-controlled trial, Vowst (SER-109) significantly reduced CDI recurrence, with a recurrence rate of only 12% in the treatment group compared to 40% in the placebo group (69). The safety profile of Vowst was comparable to that of the placebo, further supporting its potential for clinical use.

The successful application of Rebyota and Vowst highlights the immense potential of microbiome-based therapies. Both therapies are moving toward standardized, targeted approaches to microbiome treatment. By restoring gut microbiota balance, these therapies offer long-term health benefits, paving the way for the future development of microbiome-based interventions in clinical practice.

Limitations and perspectives

Here, we retrospectively analyzed the existing basic and clinical studies of FMT for IBD. However, the application of FMT for the treatment of IBD comes with a lot of confusion to be solved in terms of standardization of fecal preparation, therapeutic regimen, mechanism of action, and individual differences.

Firstly, in terms of safety assessment, there are genetic differences between the donor and the recipient, and there are more or less biological risks associated with transplanting the feces of other persons, such as viruses and pathogenic bacteria in the donor, host rejection of the transplanted microbiota, and differences in the recipient receptivity (70). The monitoring of adverse events requires a complete system to supervise the transplantation process from preparation to the administration of IBD (71). Obtaining fecal matter consists of two main types: one from a relative or friend recommended by the patient and the other from an undirected, unfamiliar donor (72). In addition, multi-donor FMT may increase the diversity of the gut microbiota even more (73). However, in either case, current studies have not been able to determine the optimal donor for FMT. Patients with different IBD genotypes and phenotypes and varying degrees of IBD severity will respond differently to FMT. Screening and selection to identify patients who respond to FMT have been a significant focus (74). Secondly, a standardized preparation of fecal bacteria for FMT still needs to be considered in terms of donor selection (relevant and irrelevant, whether it is the optimal donor), preparation (fresh, frozen or lyophilized, aerobic or anaerobic) or dosing (single versus multiple doses) (75) (*Figure 1*).

A standardized preparation process maximizes the biodiversity and overall viability of the microbiota, which can be of benefit to FMT efficacy (76). It is also worth noting that most studies on pediatric FMT used adult fecal donors (13,46). Further confirmation of the efficiency and safety of FMT in different age groups is needed. Rigorous screening of donor and donor feces remains particularly important (77).

Thirdly, a major problem with the use of FMT for IBD

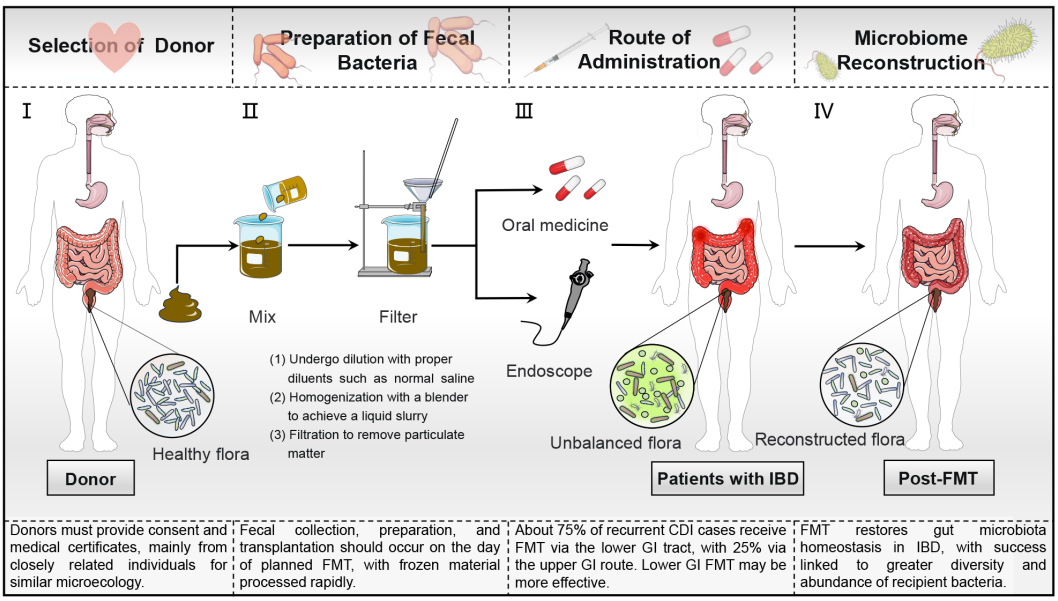

Figure 1 Flow chart of FMT. FMT is an effective technique to achieve intervention and treatment of IBD, which mainly includes donor selection, fecal bacteria preparation and preservation, route of administration, and observation of outcomes related to microbiome reconstruction. FMT involves extracting specific flora from the stool of selected healthy donors through mixing, stirring, and filtering steps, and then transplanting them into the intestine of recipient patients with IBD by oral administration or enema, to rebuild the intestinal micro-ecosystem of the recipient patients with IBD. CDI, *Clostridioides difficile* infection; FMT, fecal microbiota transplantation; GI, gastrointestinal; IBD, inflammatory bowel disease.

is the difficulty of achieving sustained remission (78). This condition requires long-term and repeated FMT treatment. The type of feces, the optimal dose, the best frequency, and the route of administration of FMT, including enemas, colonoscopic administration, or application of oral capsules, are all aspects that require extra attention regarding the treatment of IBD in treatment protocol development (79). Clinical remission rates for FMT for IBD are moderate, and patient response varies widely, even among patients receiving the same treatment regimen (80). These suggest that FMT treatment of IBD possesses significant individual differences in efficacy. The lack of adequate predictive targets or diagnostic models has caused the clinical long-term effectiveness of FMT in patients with IBD to be unmeasured (81). In particular, it is a worthwhile direction to explore the effects and long-term benefits that FMT may have on high-risk patients with IBD with poor prognoses (82).

Lastly, despite the wealth of studies included in this review, there are several important limitations of the current review that should be noted. The current research on FMT for IBD is sparse and the mechanisms are not yet fully understood, with existing studies primarily focusing on the microbiological and metabolomic changes in the recipient's gut and their relationship with clinical outcomes (83,84). However, many other key factors such as mucosal immunity, immune-mediated regulation of microbial communities, and their interactions with host responses have not been sufficiently explored in the literature (85). This limitation indicates a gap in our understanding of the full range of mechanisms that contribute to the therapeutic effects of FMT.

In addition, some studies included in this review were limited by small sample sizes, and there was variability in the follow-up duration and standardization of counseling and follow-up procedures. These methodological inconsistencies reduce the reliability of the results. Moreover, the data supporting the efficacy of FMT for IBD could have been strengthened by more robust and standardized trial designs (86,87). Inconsistent outcomes are also likely influenced by factors such as differences in donor selection, fecal administration methods, and preparation techniques (88,89), making it difficult to definitively determine the effectiveness of various FMT regimens.

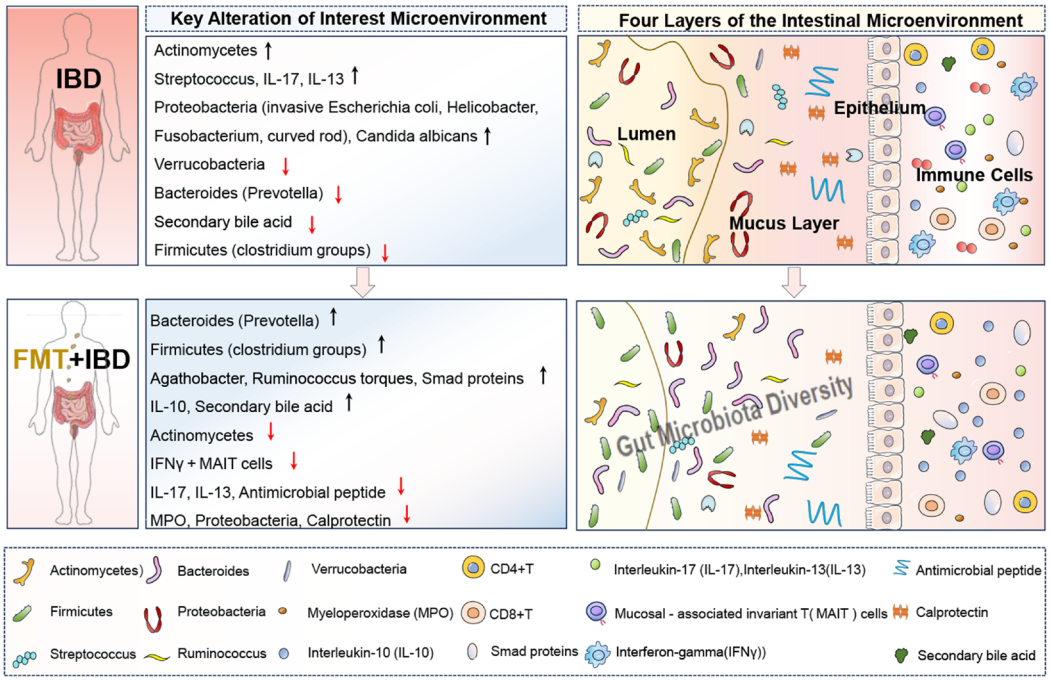

Figure 2 Changes in microflora, immune cells, and inflammatory factors in recipient patients with IBD by FMT. In patients with IBD, the abundance of *Bacteroides* and *Firmicutes* is reduced, leading to an immune imbalance characterized by a disrupted T cell composition. Additionally, there is a decrease in *Verrucobacteria* and secondary bile acids, while *Proteobacteria* and *Actinobacteria* are increased, contributing to the exacerbation of intestinal inflammation. Following FMT therapy, levels of antimicrobial peptides, calprotectin, IFN-γ, MAIT cells, IL-17, MPO and IL-13 are downregulated, while Smad proteins and IL-10 are upregulated. Furthermore, part of the *Bacteroides* and *Firmicutes* populations are restored. FMT, fecal microbiota transplantation; IBD, inflammatory bowel disease; IFN-γ, interferon-gamma; IL, interleukin; MAIT, mucosal-associated invariant T; MPO, myeloperoxidase.

Another issue is the heterogeneity of study designs (90). For example, differences in criteria for defining biological response, length of follow-up, sample size, ethnicity, disease subtype, and type of biological agent can all affect the generalizability of predictive models (91,92). The number of IBD studies is relatively small and more multicenter studies must be conducted to increase the number of samples and variables, such as IBD characteristics, phenotypic and genotypic characteristics of patients, and standardization of treatment regimens (41). Matijašić *et al.* (93) summarized present treatment options with gut microbiota composition and regulation of metabolic activity to treat or prevent the progression of IBD, such as dietary interventions, the use of probiotics, prebiotics, synbiotics, and antibiotics, and FMT. However, the underlying mechanisms of IBD were not fully interpreted, and knowledge of specific triggers and diagnostic markers for interventional approaches to IBD remained lacking (94,95). Thus, larger studies are needed to determine the role of microbiome in treating and preventing IBD.

Conclusions

In conclusion, we have systematically retrospectively analyzed animal studies and clinical studies of FMT in the treatment of rCDI, CD, and UC, showing that FMT has definite advantages in improving IBD by improving the abundance and composition of the intestinal bacteria (*Figure 2*).

The therapeutic effect of FMT can be combined with other nutritional and endoscopic treatments, to possibly achieve a synergistic effect. In addition, FMT has displayed certain differences in different cohorts due to differences in age groups, disease duration, and populations. Subsequent extensive basic and clinical studies focusing on the preservation of fecal bacteria, the selection of donor recipients, and the optimization of treatment protocols are still needed to comprehensively uncover and promote the cutting-edge progress of FMT for IBD.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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