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Fecal microbiota transplantation (FMT) and live biotherapeutic products (LBPs): from standard treatment of *Clostridioides difficile* to emerging perspectives in neuropsychiatry

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Abstract

Background: Fecal Microbiota Transplantation (FMT) has evolved from an experimental procedure into a recognized standard in the treatment of intestinal dysbiosis. Although this method is widely associated with the therapy of *Clostridioides difficile* infections, a growing understanding of the microbiota-gut-brain (MGB) axis suggests its potential in the treatment of neurodevelopmental and psychiatric disorders.

Aim: The aim of this review is to discuss the efficacy of Fecal Microbiota Transplantation (FMT) in the standard treatment of *Clostridioides difficile* infections, as well as to evaluate its potential in managing neurodevelopmental disorders (ASD, ADHD) and depression.

Material and methods: The analysis was based on a review of English-language literature from the years 2019–2025, along with selected historical references. The query was conducted in the PubMed, Google Scholar, and Web of Science databases using terms related to fecal microbiota transplantation, gut microbiota, donor screening, *C. difficile* infections and live biotherapeutic products.

Results: Treatment efficacy is the result of the interaction between donor and recipient characteristics. In cases of recurrent *C. difficile*, FMT remains an important therapeutic alternative, currently complemented by standardized live biotherapeutic products (LBPs) such as Rebyota and Vowst, which eliminate the issue of sample variability. In psychiatry, distinct mechanisms of action are observed. In patients with ASD and ADHD, neurotransmitter modulation and inflammation reduction occur, whereas in depression, the efficacy of microbiotic interventions strictly depends on the integrity of the vagus nerve.

Conclusions: The evolution of FMT toward ready-to-use pharmaceutical products confirms its efficacy in *C. difficile* infections, and growing evidence suggests its applicability in neuropsychiatric disorders. The future of the method depends on optimizing the donor-recipient-procedure triad and implementing precise bacterial consortia to minimize the risk of failure.

Keywords: fecal microbiota transplantation (FMT), gut microbiota, donor screening, *Clostridioides difficile* infection, live biotherapeutic products (LBPs)

Introduction

FMT (Fecal Microbiota Transplantation) is a procedure that has sparked controversy for years, despite the simplicity of the process, which involves introducing fecal material collected from a healthy donor into the recipient's intestine. The aim of the procedure is to treat intestinal dysbiosis associated with various disease entities [1]. It should be noted that FMT was originally implemented as a rescue therapy in patients with life-threatening conditions, despite the lack of clinical trial results and defined legal frameworks at the time. Regulatory bodies' reservations stemmed from the fact that the procedure involves the transfer of live microorganisms. Major concerns regarded the risk of co-infection, i.e., the unintended transfer of pathogenic factors or multidrug-resistant strains along with the desired microbiota [2].

Although the roots of this method date back to 4th-century China, the first documented use of FMT in modern medicine took place in the USA in 1958. However, this therapy did not gain broader recognition until the early 2000s [3,4]. A breakthrough occurred in 2011 when Bakken et al. proposed the term "Fecal Microbiota Transplantation" (FMT), and Brandt recommended this method as an inexpensive, safe, and effective first-line treatment for recurrent and severe *C. difficile* infection (CDI) [5,6].

Bearing in mind the historical background and the evolution of this procedure, the subsequent sections are dedicated to a detailed assessment of FMT efficacy in selected clinical indications. The analysis covers not only the therapeutic potential and benefits of the procedure but also the associated risks and existing limitations. A crucial element of this discussion will be identifying the factors that play a key role in determining the final therapeutic success.

Materials and Methods

This article constitutes a synthesis of the latest literature (covering the years 2019–2025), supplemented by selected key historical references. The aim of the review was to discuss the efficacy of FMT in the standard therapy of *Clostridioides difficile* infections, as well as to evaluate the potential of this method in treating neurodevelopmental disorders (ASD, ADHD) and depression. Particular attention was paid to the mechanisms affecting the microbiota-gut-brain axis, the role of the vagus nerve, and factors determining clinical success (the donor-recipient-procedure relationship). Data collection involved searching the PubMed, Google Scholar, and Web of Science databases using the keywords: *fecal microbiota transplantation*, *gut microbiota*, *donor screening*, *Clostridioides difficile infection*, *live biotherapeutic products*. The analysis primarily focused on publications in English.

Results

Clinical Success of FMT

The clinical success of fecal microbiota transplantation is the result of a complex, multifactorial interaction rather than solely a derivative of the quality of the material collected from the donor. The efficacy of the FMT process relies on a strict dependency between three pillars: the donor, the recipient, and the technical aspects of the procedure.

Regarding the donor, key factors for obtaining so-called "super-donor" status include high microbiota diversity, an abundance of strains with proven beneficial effects, and a low count of potentially pathogenic bacteria (pathobionts). To optimize this potential, modification of the donor's microbiome through targeted dietary interventions is increasingly considered prior to material collection [7].

On the recipient's side, conditions determining the permanent engraftment (colonization) of new strains are significant. These factors include the host's genetic profile, immune status, the specifics of the underlying disease, and the baseline composition of the microbiota, which may present so-called "colonization resistance." The final link is the procedure itself, where therapeutic success is significantly influenced by the route of administration, dosage, the

schedule of repeat procedures, and appropriate preparation of the recipient, such as antibiotic therapy or diet [7].

***Clostridioides difficile* – characteristics, pathomechanism, and new therapies**

Clostridioides difficile is an anaerobic, Gram-positive bacillus capable of producing spores and toxins. Due to their resistance to high temperatures, antibiotics, and bile acids, spores are considered the primary factor responsible for pathogen transmission, allowing it to survive in a latent (dormant) state. The process of spore germination in the intestines is strictly regulated by the composition of the bile acid pool, with the primary fraction exhibiting a stimulating effect and the secondary fraction an inhibitory one. Consequently, in patients undergoing FMT or therapy with live biotherapeutic products (LBPs), a restoration of physiological levels of secondary acids is observed, effectively preventing pathogen regrowth [8].

The essence of the infection lies in the activity of two potent exotoxins: enterotoxin A and cytotoxin B. Their action leads to the development of colitis and the occurrence of diarrhea. The disease process is initiated by the destabilization of tight junctions and the destruction of the actin cytoskeleton of colonic epithelial cells, subsequently potentiated by increased fluid secretion and enhanced neutrophil adhesion, which finally results in local acute inflammation, often manifesting as characteristic pseudomembranes [9].

Major risk factors for active *C. difficile* infection (CDI) include [10,11]:
contact with the hospital environment (hospitalization);
age (≥ 65 years);
antibiotics (particularly broad-spectrum penicillins, cephalosporins, clindamycin, and fluoroquinolones).

The literature also points to additional risk factors, such as [12]:

Caucasian race;
Cardiovascular diseases;
Chronic Kidney Disease (CKD);
Inflammatory Bowel Diseases (IBD).

The preventive strategy is based on two pillars. The first is antibiotic stewardship, and the second is the limitation of infection transmission through strict adherence to contact precautions. A key element of hygiene is hand washing with soap and water, which allows for the effective mechanical removal of *C. difficile* spores that are resistant to alcohol-based disinfectants [12]. Detailed therapeutic management schemas, considering dosage and recurrence, are presented in the table below [8]:

Clinical situation	Drug/ method p.o. (oral) i.v. (intravenous)	Dosage and management scheme
1st CDI Episode (mild, moderate, or severe)	Vancomycin (p.o.)	125 mg every 6 hrs for 10 days
	Fidaxomicin (p.o.)	200 mg every 12 hrs for 10 days
	Metronidazole (p.o.)	500 mg every 8 hrs for 10-14 days
1st Recurrence	Fidaxomicin (p.o.)	Standard: 200 mg every 12 hrs for 10 days; Extended regimen: 200 mg every 12 hrs for 5 days, followed by 200 mg every other day (days 7–25)
	Vancomycin (p.o.)	Standard: 125 mg every 6 hrs for 10 days;

		Pulsed-tapered regimen: 125 mg every 6 hrs for 10–14 days, then (at the same dose) every 12 hrs for 7 days, every 24 hrs for 7 days, and every 2–3 days for 2–8 weeks
Subsequent Recurrences	Fidaxomicin (p.o.)	Dosage as in 1st recurrence (standard or extended regimen)
	Vancomycin (p.o.) pulsed	As above;
	Vancomycin (p.o.) followed by Rifaximin (p.o.)	Vancomycin: 125 mg every 6 hrs for 10 days, followed by Rifaximin (p.o.): 400 mg every 8 hrs for 20 days
FMT - Fecal Microbiota Transplantation		
Fulminant Course (without ileus)	Vancomycin (p.o. or NG tube) + Metronidazole (i.v.)	Vancomycin: 500 mg every 6 hrs Metronidazole: 500 mg every 8 hrs
Fulminant Course (with ileus)	Metronidazole (i.v.) + Vancomycin (intracolonic)	Metronidazole: 500 mg every 8 hrs Vancomycin: 500 mg in 100 ml 0.9% NaCl every 6 hrs
FMT - Fecal Microbiota Transplantation		
NOTES:	Bezlotoxumab – monoclonal antibody against <i>Clostridioides difficile</i> toxin B. Recommended as adjunctive therapy to standard antibacterial treatment (especially with risk factors for recurrence). The drug is administered as a single intravenous infusion over 60 minutes at a dose of 10 mg/kg body weight.	

Table 1. Detailed dosing regimen and management of *Clostridioides difficile* infection (CDI) [8].

According to current knowledge, the FMT procedure is primarily considered in cases of recurrent *C. difficile* infection (rCDI), as well as in severe or fulminant courses resistant to standard pharmacotherapy. The essence of the procedure is the administration of fecal material (previously screened for pathogens and processed) via endoscopy, nasogastric tube, or retention enema to rebuild the protective microbiological barrier [13].

Despite high clinical efficacy, traditional FMT is associated with significant limitations that prevent its full approval as a standard drug by regulatory agencies such as the FDA (Food and Drug Administration). Obstacles such as heterogeneity and variability in the composition of biological material (a pharmacological profile difficult to standardize) and the risk of transmitting pathogens undetectable in routine screening have caused the FDA not to register classic FMT as a fully-fledged medicinal product, allowing its use only when other methods fail.

The response to these challenges is a new category of preparations—Live Biotherapeutic Products (LBPs). Unlike FMT, LBPs are biological drugs with a strictly defined or controlled composition (bacterial consortia), manufactured under good manufacturing practice (GMP). The introduction of the first standardized products of this type, which eliminate the problem of variability and infection risk associated with raw donor material, became a therapeutic breakthrough [25]:

REBYOTA (RBX2660) – A rectally administered preparation approved by the FDA in November 2022. The indication is the prevention of recurrence in adults following antibiotic therapy for rCDI. Clinical trials demonstrated a therapeutic success rate of 70.6% in the treated group compared to 57.5% in the placebo group [14].

VOWST (formerly SER-109) – The first oral live biotherapeutic product containing purified spores of Firmicutes bacteria in capsule form. The use of the preparation after standard antibiotic treatment was associated with a 68% lower risk of infection recurrence compared to the control group. The Number Needed to Treat (NNT) to prevent one recurrence was only 3.6. The safety profile was high - the incidence of adverse events was comparable between the study group (51%) and placebo (52%) [15].

FMT in the therapy of neurodevelopmental disorders: ASD and ADHD

The potential of FMT in treating neurodevelopmental diseases, such as Autism Spectrum Disorder (ASD) or ADHD, stems from the procedure's ability to reorganize the gut microbiota. The therapeutic mechanism relies here on complex interactions within the microbiota-gut-brain (MGB) axis [16].

Li et al. demonstrated that the use of FMT in children with ASD favorably influenced intestinal biodiversity, leading, among other things, to a significant reduction in the abundance of *Eubacterium coprostanoligenes*. The observed microbiological changes were accompanied by an improvement in the clinical picture regarding symptoms and behaviors characteristic of ASD. Importantly, a systemic impact of the therapy on the nervous system was also noted - the FMT procedure exhibited a restorative effect regarding serum concentrations of key neurotransmitters: serotonin, GABA, and dopamine. This suggests that microbiota transplantation effectively modulates neurotransmitter secretion, regulating central nervous system activity via the MGB axis [17].

In turn, preclinical studies on murine models conducted by Chen's team provided evidence for the efficacy of alternative therapeutic strategies. It was shown that the transplantation of microbiota cultivated *in vitro* (so-called artificial microbiota) effectively alleviates anxiety-like behaviors and stereotypies. At the metabolic level, this intervention correlated with an increase in the concentration of chemokines such as MCP-3, RANTES, and eotaxins, with a simultaneous reduction in GRO- α and MIP-1 α levels. Key bacterial taxa responsible for this effect were also identified, including the families S24-7 and *Clostridiaceae* and the genera *Prevotella* and *Candidatus Arthromitus*. These results indicate the potential of so-called donor-free FMT and the possibility of precise modulation of microbiota composition in laboratory conditions prior to implantation [18].

Regarding the etiology and treatment of Attention Deficit Hyperactivity Disorder (ADHD), targeted modulation of the microbiome, regulating the metabolism of short-chain fatty acids (SCFAs), tryptophan, and GABA, appears to be a significant therapeutic strategy [19]. Particular attention should be paid to specific species with anti-inflammatory and neuroprotective potential:

Faecalibacterium prausnitzii – may reduce neuroinflammation and alleviate symptoms by modulating the cytokine profile (increasing anti-inflammatory cytokine concentrations while reducing pro-inflammatory ones).

Lactobacillus ruminis – possesses genes for the pentose phosphate pathway, contributing to increased production of anti-inflammatory SCFAs, exerting a protective effect on the nervous system [20].

FMT in the treatment of depression and the role of the vagus nerve

The gut microbiota, serving as a key mediator in gut-brain axis communication, significantly influences mood regulation and cognitive processes. The link between dysbiosis (disruption of microbiological homeostasis) and the pathogenesis and progression of depression became the premise for seeking therapies targeted at reversing this state, directing researchers' attention to Fecal Microbiota Transplantation (FMT). This procedure, by restoring eubiosis (ecological balance of the microbiota), may optimize the functioning of the brain-gut axis and alleviate symptoms of low mood. The mechanism of FMT action involves modulating microbiome composition and activating beneficial signaling pathways, including regulating the immune

response, reducing inflammation, and sealing and maintaining the integrity of the intestinal barrier. Collectively, these processes may lead to the dampening of neuroinflammation associated with affective disorders [21, 22].

The vagus nerve, constituting the main channel of bidirectional communication between the brain and the gastrointestinal tract, is responsible for regulating intestinal activity and transmitting sensory signals to the central nervous system. The crucial role of this pathway in the pathomechanism of microbiota-dependent depression is confirmed by studies on animal models.

Wang's team demonstrated that transplanting fecal material from mice subjected to chronic social stress into previously antibiotic-sterilized *Ephx2* gene knockout mice (*Ephx2* KO) induced depressive behaviors in the recipients. This phenomenon correlated with the presence of *Faecalibaculum rodentium* bacteria, an increase in pro-inflammatory interleukin-6 (IL-6) concentration, and reduced expression of synaptic proteins in the prefrontal cortex. Significantly, performing a subdiaphragmatic vagotomy (severing the continuity of the vagus nerve) alleviated the observed behavioral anomalies, proving that pro-depressive signals are transmitted via the neural pathway [23].

Analogous conclusions are drawn from studies by Pu et al., where microbiota transfer from *Chrna7* KO mice to antibiotic-treated recipients also resulted in the occurrence of a depressive phenotype, accompanied by systemic inflammation and synaptic protein deficits in the prefrontal cortex. In this model as well, vagotomy effectively prevented symptom development, constituting further confirmation that the functioning of the microbiota-gut-brain axis in the pathogenesis of depression is strictly dependent on the integrity of the vagus nerve [24].

Identifying the vagus nerve as an essential element of signal transmission on the gut-brain axis gives the described observations a significant translational dimension. This constitutes a strong premise for intensifying research on the application of FMT in psychiatry. Confirmation of the biological mechanism of microbiota influence on the CNS justifies the necessity of devoting more attention to this procedure to definitively verify whether targeted microbiome modulation can become an effective and safe strategy supporting the treatment of depression in humans.

Conclusions

Fecal Microbiota Transplantation (FMT) is no longer merely an experimental last-resort method. It is currently a recognized standard in the treatment of recurrent *Clostridiooides difficile*. However, the most important change is happening before our eyes, as we move away from the transplant procedure toward precise pharmacotherapy. The FDA's decision to approve LBP preparations (such as Rebyota or Vowst) ends the era of reliance on variable donor material. Instead, physicians receive a standardized product. This resolves legal issues and guarantees treatment reproducibility, which has hitherto been the weakest link in the therapy.

Furthermore, the potential of the microbiota extends far beyond gastroenterology. Results of studies on disorders such as autism (ASD), ADHD, and depression clearly indicate that the gut-brain axis is not just a theoretical concept. Proving that the vagus nerve acts as a highway for signals between the intestines and the brain gives these discoveries a real clinical dimension. This suggests that by appropriately manipulating the microbiome, we may effectively support the therapy of psychiatric and neurodevelopmental diseases in the future.

However, it must be remembered that the transplant itself does not guarantee success. The outcome of the therapy depends on many variables: the specifics of the donor, the recipient's organism, and the technique of the procedure itself. Therefore, the future does not belong to the simple transfer of bacteria, but to personalized medicine. The key will be the search for so-called "super-donors" and the creation of synthetic bacterial sets, designed to target specific disease mechanisms.

Disclosure

Conceptualization: Michał Magiera, Piotr Czwałga

Methodology: Patrycja Koprowska, Miłosz Sikora

Software: not applicable.

Check: Michał Magiera, Piotr Czwałga, Miłosz Sikora, Patrycja Koprowska

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