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Fecal microbiota transplantation: an update on the advances in the treatment of complex diseases

Carlotta Magrì[1], Giuliana Calabrese[1], Letizia Masi[2], Sara Troisi[1], Federica di Vincenzo[1], Pierluigi Puca[1], Cristina Graziani[2], Loris R. Lopetuso[2,3-4], Valentina Petito[2]

[1]Dipartimento Universitario di Medicina e Chirurgia Traslazionale, Università Cattolica del Sacro Cuore, Rome, Italy.

[2]Centro per le Malattie dell'Apparato Digerente (CeMAD), UOS di Malattie Infiammatorie Croniche Intestinali, Dipartimento di Scienze Mediche e Chirurgiche, Fondazione Policlinico Universitario "A. Gemelli" IRCCS, Rome, Italy.

[3]Dipartimento di Medicina e Scienze dell'Invecchiamento, Università degli studi "G. D'Annunzio", Chieti-Pescara, Chieti, Italy.

[4]Center for Advanced Studies and Technology (CAST), Università degli studi "G. D'Annunzio", Chieti-Pescara, Chieti, Italy.

Corresponding Author: Valentina Petito, Ph.D; e-mail: valentina.petito@unicatt.it

ABSTRACT

In addition to its proven effectiveness in the treatment of recurrent *Clostridium difficile* infections (rCDI), fecal microbiota transplantation (FMT) is on the rise to become one of the most studied innovative therapies for the treatment of several complex diseases. This review aims to discuss the many promising applications of FMT in intestinal diseases such as inflammatory bowel diseases (IBD) and extraintestinal diseases such as neuropsychiatric disorders, metabolic syndrome, and autoimmune diseases. For each disease group, preclinical data will be presented in addition to clinical trial evidence.

KEYWORDS

FMT
MICROBIOTA
IBD
NEUROPSYCHIATRIC DISORDERS
METABOLIC SYNDROME
AUTOIMMUNE DISEASES

INTRODUCTION

Gut microbiota is a complex microbial consortium composed of more than 1500 species, distributed in more than 50 different phyla[1], whose most prevalent are *Bacteroidetes* and *Firmicutes* followed by *Proteobacteria*, *Fusobacteria*, *Tenericutes*, *Actinobacteria,* and *Verrucomicrobia* (90% of the total)[2]. Its composition is however variable and depends on the health/disease state of the host, diet, and environment, alterations in the relative abundance of "good bacteria" is related to several human conditions, such as inflammatory bowel diseases (IBD)[3-11], obesity and diabetes[12-20], allergy[21-27], autoimmune diseases[28-31], and even neuropsychiatric disorders[32-40].

Fecal microbiota transplantation (FMT) is a therapeutic approach that aims to normalize the composition of a dysbiotic gut through colonization with stool from a healthy donor[41,42]. So far, FMT has proven effective in treating *Clostridium difficile* infections (CDI) with meta-analysis reporting a 92% clinical resolution rate compared to prolonged anti-microbial therapy with vancomycin[42] with a success rate between 20% and 30%[43-45]. Depending on the source of the material for transplantation, FMT can be divided into heterologous (h-FMT) if it is from a different individual and autologous (a-FMT) if it is the patient's own.

Implementing a-FMT has been hypothesized to outflank the risk of disease transmission and host-graft reaction[46]. Current guidelines for randomized controlled trials recommend using a-FMT as a placebo because of the assumption that it represents the most inert option compared to h-FMT[47]; however, it is important to highlight how the time of collection is fundamental to the outcome of the intervention, this is because stool samples can have a pro or anti-inflammatory effect[48]. Studies have shown that frozen stool samples are non-inferior to fresh samples in terms of efficacy of the treatment[49]; because of this, stool banking is becoming a valid option to collect healthy samples from the patient during the remission phase and to standardize the process[50].

To execute the procedure, stool donors must be tested for viral and non-viral transmittable diseases (e.g., HIV, HBV, *Helicobacter pylori*, *Salmonella*); in addition, a questionnaire is asked to be completed as a screening procedure to exclude unsuitable candidates (e.g., drug use, fat-rich diet, and alcohol use). It was believed that the best stool donor was a close relative, this belief has been disproven[51] since no evidence of any difference in terms of efficacy has been observed; age and gender however must be considered when selecting the donors[52]. Once the quality of the specimen has been evaluated, the fecal material is resuspended in 3 to 5 times the volume of sterile saline solution; in addition, if the sample must be stored for long periods, glycerol should be added up to a final concentration of 10%[53].

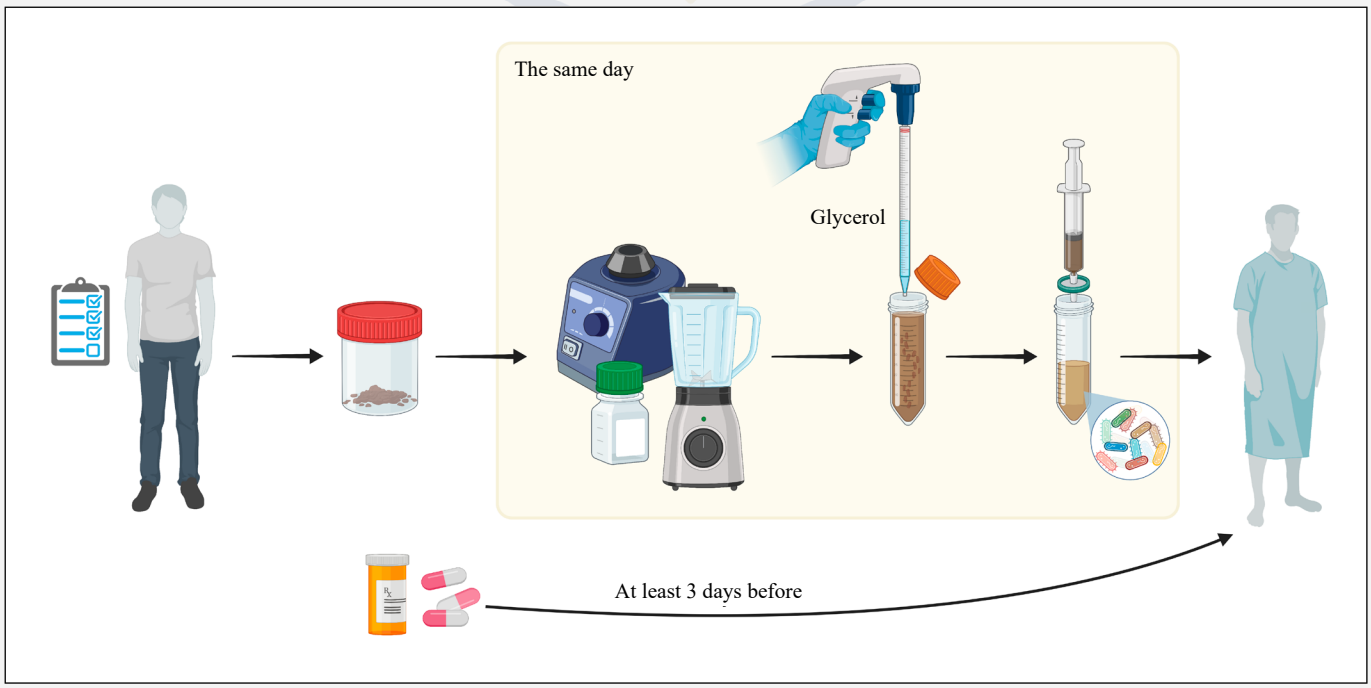
The resuspension in a solvent is fundamental for the homogenization process, which is generally carried out with a mechanical homogenizer or with non-aggressive lysis solutions. Once the sample is dissolved, it must be strained using gauze[49], tea strainer, or similar device[54] to eliminate bigger pieces that could clog infusion syringes and tubes. The patient treated with FMT must go through a preparation process that consists of a vancomycin treatment at least three days before the infusion, it is important to note that the antibiotic treatment must cease at least 24 to 48 hours before the infusion[49,55-60]. In addition, bowel lavage with polyethylene glycol before the procedure when FMT is performed reduces the abundance of *C. difficile* in the GI tract[49,55,58-62] (Figure 1).

Currently, there is not a defined period of observation after the procedure; however, the European consensus advised conducting at least an 8-week follow-up period after FMT in CDI patients[53,63]. During the follow-up, clinical response is indicated by a reduction of stool frequency and improvement of stool consistency, as well as amelioration of other parameters of disease severity as laboratory parameters, radiological, and/or endoscopic findings[53].

Although FMT has been utilized more frequently in recent years, many concerns regarding its application, safety, efficacy in the long-term, and the criteria to apply in the selection of the donors are still pending[64].

A case report has observed the transmission of *ESBL Escherichia coli* in two immunocompromised patients

Figure 1. Phases of the FMT procedure. The therapy starts with the evaluation of the stool donor. Once it has been thoroughly tested, the sample is blended, enriched with glycerol, and strained. The patient is treated with broad-spectrum antibiotics for at least 3 days prior to the infusion. The figure has been made with biorender.

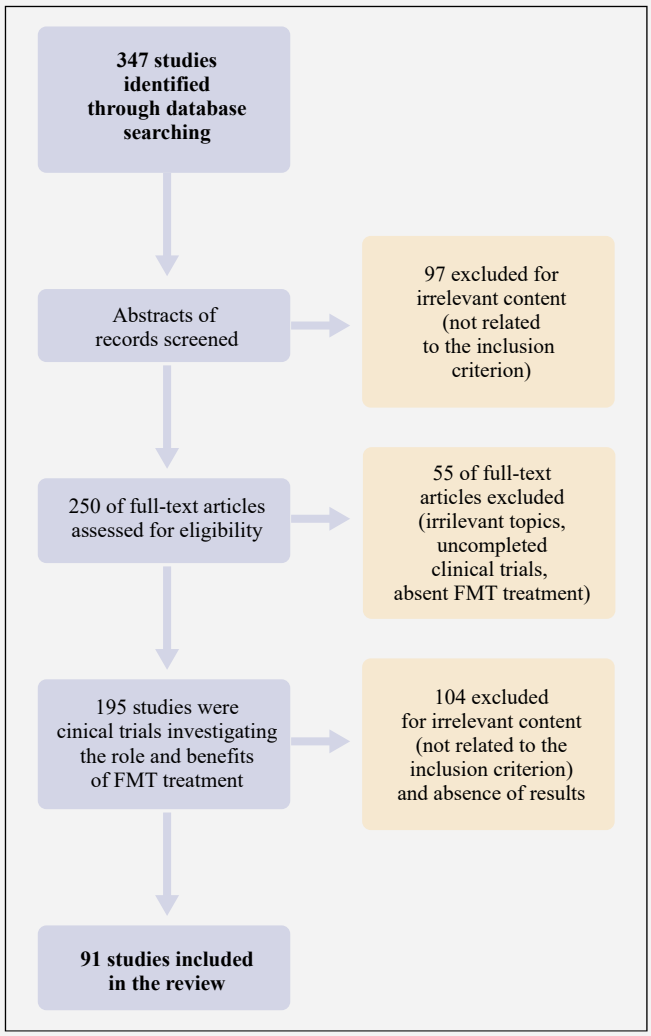


after FMT from a common stool donor[65]; therefore, a thorough analysis of potential pathogens in the donor's microbiota must be carried out before transplantation. There is also a great need for standardization of the procedure since, so far, every medical center follows its own rules; because of this, the United States and European consensus conference both suggest using a donor questionnaire to meet the exclusion and inclusion criteria[53,63,66-68].

Since the encouraging results obtained in the treatment of CDI, the range of FMT applications has been constantly expanding[69]. As expected, the first conditions treated with FMT were linked to the GI tract due to the clinical similarities between infectious and non-infectious types of colitis[6,70,71]. Past the gastrointestinal clutter, the applications of FMT rapidly expanded to different areas of extra-gastrointestinal infections in later years, such as obesity and metabolic syndrome[72-77]. Several case reports and animal models have too uncovered the plausible helpful impacts of FMT in patients with severe multiple sclerosis[78,79], autism[80,81], multidrug-resistant organisms (MDRO) infections[82-84], and multiple organ dysfunction in critical patients[85]. Moreover, recent studies illustrated the positive impacts of immunotherapy on melanoma[86-88] with FMT in an animal model and clinical trial.

This review aims to update on the latest applications of FMT in the treatment of inflammatory bowel diseases, neuropsychiatric disorders, metabolic syndrome, and autoimmune diseases through *in vivo* experiments and clinical trials.

Figure 2. Flowchart for review of the studies.



MATERIALS AND METHODS

Search strategy

The literature search was conducted in PubMed (https://pubmed.ncbi.nlm.nih.gov) and Scopus (https://www.scopus.com) databases considering articles published between 2019 and 2022, using the following search string: ("IBD", "ulcerative colitis", "Crohn's disease") AND ("FMT", "innovative therapy"). The "AND" operator was used to create all possible combinations of selected terms. ClinicalTrials.gov (https://clinicaltrials.gov) contains information about medical studies in human volunteers and was used to search for ongoing clinical trials (Figure 2).

Study selection

The initial screening of documents, using abstracts and titles, was carried out, including only English-language research articles, while articles without full text and abstract, duplicate studies, conferences, review articles, and editorial reports were excluded.

The selected clinical trials included in this review considered new therapies in the experimental phase II, II, or III used in IBD (UC and CD) patients. In addition, the efficacy and safety of FMT and how it could be a supplement in the treatment of IBD were evaluated.

RESULTS

FMT in the treatment of inflammatory bowel diseases

Inflammatory bowel diseases (IBDs), primarily ulcerative colitis (UC), Crohn's disease (CD), and irritable bowel disease (IBS) are multifactorial immune-mediated conditions that affect the lower gastrointestinal tract and have been linked to the onset

of colorectal cancer[89]. IBDs have become, especially in newly industrialized countries that are now exposed to a western diet, the growing diseases of the 21st century[90,91]. The exact etiopathology of IBDs is unclear, what is known is that it embroils alterations in the immune response with dysbiosis of the gut microbiota; in addition to that, environmental factors and genetic predisposition can contribute to an early onset of IBD[92]. The main focus of the investigation of the pathogenesis of IBD has always been on imbalances of T cell response on a mucosal level. New evidence suggests that the aberrant inflammatory response in IBD patients is also correlated to adaptive and innate immunity[92]. Previous studies have led to believe that CD was driven by a Th1 response and UC by a Th2 response[93,94]; however, this is not necessarily the case since it has been recently observed how Th17 cells are also involved in the inflammatory response in IBDs[95]. On a microbial level, IBD patients are characterized by a lower abundance of Firmicutes, in particular *Clostridium* clusters IV and XIVa (e.g., genera from the *Ruminococcaceae* and *Lachnospiraceae* family), Lactobacillus and Bifidobacterium[3,8,96], and an increase of the abundance of members of the Proteobacteria phylum, such as Enterobacteriaceae[97,98], including *Escherichia coli*[3,99,100].

FMT was first used to treat UC in 1988[101], since then, many clinical and preclinical studies have been carried out to verify its success rate and safety. Scientific literature is full of examples of murine models of colitis, such as chemically induced (DSS, TNBS, and oxazolone), genetically induced (Rag1$^{-/-}$), and spontaneous models (SAMP1/YitFc); each one differs in terms of reliability and complexity. For example, oral administration of the sulfated polysaccharide DSS to mice via drinking water induces severe colitis characterized by weight loss, bloody diarrhea, ulcer formation, loss of epithelial cells, and infiltrations with neutrophils, resembling some features of flares in human UC[102]. Another example is the SAMP1/YitFc mouse strain, which represents a model of CD-like ileitis that is ideal for investigating the pathogenesis of chronic intestinal inflammation[103]; this is because it develops the ileitis spontaneously due to a single susceptibility locus on chromosome 9 (D9Mit123). Because of the positive results obtained on mice, the was a huge call for clinical trials. A single-center, prospective cohort study conducted by Ianiro et al[104], which consisted of 8 weeks of follow-up, after the FMT treatment, of 18 IBD patients with a *C. difficile* infection, whose disease activity was assessed through Harvey- Bradshaw Index (HBI ≥4)[105] for Crohn's disease and partial Mayo score for ulcerative colitis (Mayo ≥2)[106]. The primary outcome of this study was to establish if the patient was negative for *C. difficile* toxin at 8 weeks after the infusion. At the end of the study, the *C. difficile* toxin was negative in 17 patients (94%), and most of them experienced an improvement in the clinical picture, 10 patients were in clinical remission (59%), and in four patients (24%) the disease was ameliorated, while it did not improve in three patients despite CDI decolonization[104]. Another study conducted by Madsen et al[107] had a primary outcome to evaluate the effect of FMT on abdominal pain, stool frequency, and stool form in IBS patients. The randomized, double-blind, placebo-controlled study included 52 adult patients with moderate-to-severe IBS assigned randomly to treatment with FMT capsules or placebo capsules for 12 days; after that, the patients were followed for a total of 6 months. A statistically significant improvement in stool frequency was found in the FMT group; however, statistically significant differences were found between groups at any time during the study.

FMT in the treatment of neuropsychiatric disorders

The intestinal microbiome plays major roles in immune, neuroendocrine, and neural pathways[108]. It has been demonstrated that the gut-brain axis can recruit, through microbiota, a bidirectional communication network to regulate brain function, development, and even behavior[109,110]. Intriguingly, several environmental risk factors for schizophrenia (SCZ), including cesarean section[111,112], early-life stress[113,114], maternal malnutrition[115,116], and maternal immune activation[117,118] have been shown to have a profound impact on the gut microbiome of the newborn.

A preclinical study by Zhu et al[119] used FMT to transfer the microbiota of 11 SCZ patients into C57BL/6J mice. At the end of 3 weeks of infusion, the SCZ mice displayed hyper-kinetic behavior, and an altered tryptophan-kynurenine metabolism was observed. Schizophrenia isn't the only neurologic disorder that has been linked to microbiota alterations, Valeri et al[120] worked with 5xFAD mice treated with antibiotics and FMT of the cecal content of young or old mice to demonstrate how the microbiota could induce Alzheimer's disease (AD) in the transplanted mice. An increase in plaque deposition in PFC and hippocampus DG has been observed in addition to a higher level of LPS binding proteins; these observations have been observed in the very early stages of AD in humans[121]. In conclusion, the authors suggest thata correlation between AD and FMT from an old mice microbiota may exist.

The impact of microbiota transplantation on the progression of neuropsychiatric disorders, such as depression and autism, has been assessed in clinical studies. Lahtinen et al[122] (NCT03561519) revealed in a

randomized controlled trial that IBS patients that have received heterologous FMT had a decrease in both IBS symptoms and depression, based on the IBS-Quality of life questionnaire (IBS-QoL) that was asked to fill in. Autism spectrum disorders (ASD) have long been linked to gastrointestinal distress such as constipation and diarrhea[123-125]; these symptoms seem to be partially due to a dysbiotic microbiota[126] which is unable to process metabolites like 4-ethylphenylsulfate, indolepyruvate, and corticosterone[127,128]. An open-label study published in 2017 (NCT02504554)[129] involved 18 ASD-diagnosed children, between the age of 7 and 16, treated with FMT using a high initial dose followed by daily lower maintenance doses for 7-8 weeks. At the end of the study, significant changes in the gut microbiota composition, GI manifestations, and ASD symptoms were observed:

- the average GSRS score (Gastrointestinal Symptom Rating Scale) dropped 82% from the beginning to the end of the treatment and remained improved at the end of the maintenance phase;
- the ASD-linked behaviors evaluated with PGI-II (Parent Global Impressions) assessment score were significantly improved during treatment without relapse 8 weeks after;
- the high-throughput analysis confirmed the success of the engraftment on a microbial level, illustrating that the transferred microbiota changes the gut environment in a way that is more hospitable to recruiting new commensal bacteria.

FMT in the treatment of metabolic syndrome

Metabolic syndrome (MetS) is a cluster of conditions such as increased blood pressure, high blood sugar, excess body fat in the waist area, and abnormal cholesterol and/or triglyceride levels; this altogether increases the risk of heart disease, stroke, and type 2 diabetes[130]. Depending on which aspect of metabolic syndrome is studied, many strains of mouse models (also genetically induced) can be used (Table 1). A metabolic syndrome-like phenotype can be reproduced in C57BL/6 mice with the introduction of a high-fat diet[155-157]. In a 2020 preclinical study by Yu et al[158], db/db mice were used as stool donors to demonstrate that their altered microbiota composition could induce alterations in Hippo signaling in pseudo-germ-free mice. This pathway is recognized as a key regulator of organ size and tissue homeostasis[159]. In another study, the lupus-prone NZB/WF1 strain was given a low-fiber diet to demonstrate how this ultimately leads to accelerated lupus pathology and the associated immune dysregulation. This hypothesis is linked to bacterial metabolism, and especially the generation of SCFA, induced by the consumption of a fiber-rich diet. Their data showed that the altered diet induced an increase in inflamed white adipose tissue in the treated mice, in addition, to intestinal leakage and accelerated disease development have been recorded. These two studies demonstrate how the composition of the gut microbiota can lead to metabolic disorders in prone to the disease mice.

Table 1. Mouse models implemented for studying MetS. The table shows some of the most frequently used mouse models, each divided based on the disease in which they are used.

Mouse model	Characteristics	Ref.
Models of obesity and diabetes		
Lep$^{ob/ob}$	Early onset of pronounced obese phenotype, hyperphagia and hyperleptinemia followed by hyperinsulinemia, insulin resistance, severe obesity, mild diabetes and fatty liver.	131-138
db/db	More severe hyperglycemia and hyperinsulinemia manifest early (10th day from birth) and progressively aggravate till 3 months. Later on, insulin levels fall accompanied by β-cells atrophy.	139,140
s/s	Knock-in mutation which blocks STAT3 pathway inducing leptin resistance, obesity and diabetes.	141,142
Models of hyperlipidemia		
Ldlr$^{-/-}$	Knock-out mutation for LDL receptor protein which leads to two-fold increase in cholesterol, seven- to ninefold increase in intermediate density lipoproteins (IDL) in the bloodstream.	143
ApoE$^{-/-}$	Hypercholesterolemia, develop severe atherosclerosis.	144,145
High-fat diet-fed A(y)/a LDLR$^{-/-}$	Severe hyperlipidemia, high levels of VLDL and LDL and insulin resistance.	146,147
Models of hypertension		
NZB/WF1	Autoimmune disease model, especially for systemic lupus erythematosus (SLE), develop insulin resistance and hypertension.	148-150
KKA^{y}/a	Develop hyperglycemia, hyperinsulinemia, glucose intolerance and obesity by eight weeks of age	151-154

The implementation of FMT for clinical use has gained much attention in recent years. A 2022 randomized, triple-blind study (NCT03477916) aimed to treat obese patients with metabolic syndrome through microbiota transplantation, in addition to a fiber-rich diet; their results showed that show that a low-fermentable fiber supplementation following oral FMT improved insulin sensitivity from baseline to 6 weeks in the patients. Mounting preclinical and clinical evidence has proved the causal role of gut microbiota on the pathogenesis of primary hypertension. In a randomized, double-blind trial started in 2021 (NCT04406129), 120 patients aged between 18 and 60 with an established diagnosis of Grade 1 hypertension will be divided into two groups and treated either with FMT capsules or placebo; the assigned treatment will be given three times in the outpatient clinics on day 1, day 7, and day 14 after the trial initiation. The patients will be visited once a month for a follow-up period of 3 months. In each visit, the clinician will perform a comprehensive examination to evaluate the efficacy of FMT for hypertension treatment, including physical examination, blood pressure, heart rate, 24-h ABPM, routine blood tests, routine urine tests, biochemical blood tests, urine micro-albuminuria protein, creatinine, electrocardiography, and arterial stiffness assessments (ABIs, PWVs)[160]. Unfortunately, there are no results available because the research group is still recruiting.

FMT in the treatment of autoimmune diseases

Autoimmune diseases are a group of over one hundred disorders characterized by the dysfunction of the immune system that attacks the body's tissues, causing inflammation and tissue damage. The inflammation is due to the presence of autoreactive lymphocytes or antibodies that causes the death of a specific subpopulation of cells; the consequence is the release of intracellular molecules that, as a vicious cycle, contribute to the stimulation of the immune system[161,162]. The etiopathogenesis of these disorders isn't fully comprehended, but both genetic and environmental factors contribute to the onset[163]. In the last decades, several studies have been conducted on the microbiome's effects on autoimmune patients hypnotizing its role in the loss of immune tolerance and the activation of autoreactive T cells in the intestine. Dysbiosis was associated with the onset or the severity of several autoimmune diseases such as rheumatoid arthritis, spondyloarthritis, Type 1 diabetes, systemic lupus erythematosus, systemic sclerosis, multiple sclerosis, Celiac disease, and Sjögren Syndrome[164-172]. Furthermore, in most of these cases, patients affected by autoimmune diseases showed, also before the diagnosis, the presence of anti-Saccharomyces cerevisiae autoantibodies, which may modify the microbiome balance[173].

Consequently, several preclinical studies have been conducted to evaluate the possibility that an FMT can improve the onset and severity of these pathologies. In a multiple sclerosis mice model with experimental autoimmune encephalomyelitis (C57BL/6 mice) was tested the potential efficacy of FMT treatment: mice achieved an amelioration in the dysbiotic gut microbiota, a minor activation of microglia and astrocytes, and protection of the blood-brain barrier, myelin, and axons[174]. In another model of autoimmune disease, the systemic lupus erythematous mice model (MRL/lpr mice), FMT alleviated the disease severity and progression[175].

For these reasons, different clinical studies were conducted to test the hypothesis that modifying the microbiome through FMT can improve autoimmune diseases' onset, progress, severity, and treatment also in humans. The randomized controlled trial NTR3697 of FMT on new-onset type 1 diabetes patients indicates that treatment is capable of slowing down the decline in insulin production and preserving beta cell activity[176]. Currently, the following clinical trial is ongoing, exploring the effects of FMT in these patients: NCT04124211, NCT05323162, and NCT04749030 (all in the recruiting phase). In 2022, Huang et al published the results of a 12-week, single-arm pilot clinical trial in which 20 patients with active systemic lupus erythematosus were treated for three weeks with the oral encapsulated fecal microbiome and monitored for 12 weeks: the response rate was 42.12%, there were significant reductions in the severity score and a modification in the bacteria population (a decrease in the inflammation-related bacterial taxa and an increase in SCFAs-producing bacterial taxa)[177]. A case report on FMT performed in a rheumatoid arthritis patient indicates a good potential therapeutical effect[178]. Two clinical trials are ongoing trying to demonstrate this thesis: NCT03944096 (recruiting) and NCT04924270 (not yet recruiting). An exploratory randomized clinical trial on gastroscopic-guided FMT in active peripheral psoriatic arthritis patients (NCT03058900) demonstrated the safety of the technique, but the treatment had no efficacy superior to the placebo[179]. The early phase 1 clinical trial NCT03726645, which started in 2018, will provide new information. The NCT03444220 clinical trial on systemic sclerosis patients demonstrated an amelioration in the lower GI symptoms but demonstrated the presence of adverse events correlated to the gastroduodenoscopy procedure[180]. Is currently ongoing the NCT04300426

clinical trial that will increase the available data. Regarding multiple sclerosis, there are not yet publications available but were started different clinical trials: NCT04150549 (not yet recruiting), NCT04203017 and NCT04096443 (recruiting), NCT03594487 (active, not recruiting), and NCT03975413 (completed). Only one clinical trial (NCT03926286, completed) was performed on Sjogren's Syndrome patients, but the results aren't available. Were also performed clinical trials that included patients with different autoimmune diseases: NCT04924270 (phase 2, not yet recruiting), NCT04014413 (started in 2019, actively recruiting). As these clinical studies will be completed and their results will be published, we will understand better if, as it seems, transplantation can be good therapy for autoimmune diseases. Comparative studies will then be needed to understand which is the best FMT by evaluating both the efficacy and the related adverse effects.

CONCLUSIONS

It is undeniable that FMT is becoming a well-established therapy with the potential to be used routinely for the treatment of complex diseases. Many preclinical and clinical studies are demonstrating its efficacy in the treatment of extraintestinal conditions such as autism spectrum disorders, type 2 diabetes, and rheumatoid arthritis. The call for standardization of the procedure and the implementation of high-quality randomized controlled trials will give further insights into its long-term effects and benefits. Hence, with the development of personalized medicine, this procedure can be adapted to meet the personal needs of each patient and achieve stable and effective results.

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CONFLICTS OF INTEREST. The authors declare that they have no conflicts of interest or competing financial interests.

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