REVIEW ARTICLE

Fecal Microbiota Transplantation

Saurabh Padole, Siddhant Ingle, Jitendra Ingole

Department of Medicine, Smt. Kashibai Navale Medical College and General Hospital, Pune, Maharashtra, India

ABSTRACT

Fecal microbiota transplantation (FMT) has been used to treat C. difficile infection and other conditions. FMT can be used to create a unique gut microbiota profile for patients with Ulcerative colitis and Inflammatory Bowel Disease.It should be emphasized, nonetheless, that the faecal microbiota is a complicated starting point, and those planning to reverse engineer it will likely have to determine how the microbial communities in the samples interact with one another and its mode of operation. Future study should concentrate on determining the long-term effects of FMT. Unquestionably, FMT is effective against C. difficile infection. Before wide adoption of FMT as a therapeutic benefit beyond recurrent CDI, further randomized controlled trials and pieces of evidence are required.Among other conditions, it is being investigated as a therapy for IBD, IBS, and metabolic syndrome/insulin resistance.

Key words: Fecal microbiota transplantation, human gastrointestinal, inflammatory bowel disease

INTRODUCTION

Numerous bacterial species are found in the human gastrointestinal (GI) tract, where they play important roles in digesting, nutrient provision, colonic epithelial maturation, and pathogen defense.[1-3] Although the composition of the human gut microbiome varies from person to person and is generally stable and resilient over time, external variables, such as nutrition, probiotics, prebiotics, infections, and medications, notably antibiotics, can change it.[4-7] Infectious conditions (infectious gastroenteritis and Clostridium difficile infection [CDI]), autoimmune conditions (allergic disease, diabetes, and inflammatory bowel disease [IBD]), some general conditions (overweight and functional GI disorders), and behavioral conditions are just a few of the disease groups linked to the gut microbiota.[8] The term "gut microbiota" refers to the more than 98% of the human microbiota that is found in the GI tract. These microbes make up a dynamic community of microbes that work together to produce a symbiotic superorganism with roughly the same mass as the human brain and 100 times as many genes as the human genome. It is important to note that the gut microbiota is not a solitary community that just exists within the host and in the gut; rather, it engages in complex communication with other organs, even those that are distant, using microbial signals that are carried through the intestinal epithelium and through various pathways, including (i) the trimethylamine (TMA)/TMA N-oxide (TMAO), (ii) the short-chain fatty acids (SCFAs), (iii) the primary and secondary bile acid (BAs) pathways,[9] and (iv) the vagus nerve.

Access this article online	
Quick Response code	**Website:** www.mjbar.in
	Recevied on: 26/12/2022 **Accepted on:** 20/02/2023

DONOR SELECTION

In the past, a spouse or close family was thought to as the perfect fecal microbiota transplantation (FMT) donor. Due to common environmental risk factors, faces from the spouse may reduce the chance of illness transmission. Since the receiver and a close relative should share similar microbial species, the mucosal immune system's adaptive immunity may show greater tolerance for the donor's microbiota.[10]

Nevertheless, more clinical data show no connection between donor and FMT results.[11,12] In circumstances where genetics play a role in the disease, such IBD, unrelated FMT volunteer donors may be more advantageous.[13] Undoubtedly another key component is the time between screening and donation.

Address for correspondence:
Dr. Saurabh Padole, Junior Resident, Department of Medicine, Smt. Kashibai Navale Medical College and General Hospital, Pune, Maharashtra, India.
E-mail: padolesaurabhv@gmail.com

Table 1: Donor inclusion and exclusion criteria

Inclusion criteria	Aged 18–65 year
	No prior history of persistent gastrointestinal illness symptoms
	No other significant medical conditions exist
	Little routine medication with no drugs that could affect tool viability, including no antimicrobials (antibiotics, antifungals, and antivirals) or probiotics in the previous 3 months
Exclusion criteria	*Risk of infectious agent*
	Known HIV, hepatitis B, or hepatitis C infection
	Known exposure to HIV or viral hepatitis with in the previous 12 months
	High-risk sexual behavior, such as having intercourse with someone who has HIV/AIDS or viral hepatitis, having sex with other men, or having sex for drugs or money, is prohibited
	Use of illicit drugs
	Tattoo or body piercing within the preceding 6 months
	Current communicable disease that is well-known (such an upper respiratory tract infection)
	Risk elements for variation The disease Creutzfeldt-Jakob
	Travel within the past 6 months to regions of the world where travellers' diarrhea is at high risk or where the disease is endemic
	Gastrointestinal comorbidities
	History of or current IBD
	Irritable bowel syndrome, persistent constipation, chronic diarrhea, or any other intrinsic gastro-intestinal disease or ailment in the past
	Known polyposis, a history of gastrointestinal malignancy that has returned, or a significant family history of colorectal cancer
	History of significant gastrointestinal surgery, such as partial colectomy or a gastric bypass Factors that may have an impact on the gut microbiota's makeup
	Antimicrobials (antibiotics, antifungals, and antivirals) or probiotics with in the preceding 3 months
	Major immunosuppressive drugs include biological medicines, exogenous glucocorticoids, and calcineurin inhibitors. systemic anti-cancer medications
	household members that have a stomach virus that is still active
	Other conditions
	Systemic auto immunity (e.g., multiple sclerosis, connective tissue disease) A topic disease (e.g., moderate severe asthma, eczema, and eosinophilic disorders of the gastro intestinal tract)
	Metabolic syndrome , obesity (BMI >30), or moderate-to-severe undernutrition/malnutrition
	Chronic pain syndromes (e.g., chronic fatigue syndrome, and fibromyalgia) or neurologic/neuro developmental disorders
	History of malignant illness or ongoing oncologict herapy
	Incarceration for long-term care facility residence
	Body piercing or tattoo on prior 6 months

To lessen the possibility of contamination, the FMT French group advises that the term be as brief as feasible and not to exceed 21 days [Table 1].[14]

SCREENING INVESTIGATION FOR DONOR

Donor screening investigations noted in Table 2.

SAMPLE PREPARATION

Fecal stools derived from selected donors need to be processed and prepared before being transplanted into the recipient. How is FMT prepared? The detailed method varies across the different studies. In general donor stools (~100–150 g) are collected and a sterile saline solution (NaCl, 0.9%) is added for a preliminary homogenization to get a feces slurry employing a speed blender [Figure 1].[15-17] Then, larger particles, fibers,

and undigested food are removed by filtration using a metal sieve, and the homogeneous liquid fresh fecal sample can be transferred in sterile syringes and ready for FMT within <6 h after the emission. The fresh fecal preparation was the first to be used for C. difficile infections.[18] Alternatively, the preparation can be further processed with multiple steps of filtration where the diameters of the filters keep decreasing (from 2 to 0.1 mm), cryprotected in glycerol (10%), frozen, and kept at −80°C for later FMTs; prior FMT, the frozen slurry has to be thawed at 4°C overnight and reconstituted with normal saline [Figure 1].[19]

Table 2: Donor screening investigations

Blood testing	Cytomegalovirus
	Epstein-Barr virus
	Hepatitis A, B, C, E
	Syphilis
	HIV-1 and HIV-2
	Entamoeba histolytica
	Complete blood cell count with differential
	C-reactive protein and erythrocyte sedimentation rate
	Albumin Urea, creatinine, and electrolytes
	Aminotransferases, bilirubin, gamma-glutamyl transferase, alkaline phosphatase
	HumanT-lymphotropic virus types I and II antibodies
Stool testing	Clostridium difficile toxin polymerase chain reaction
	Fecal microscopy/culture/sensitivity with routine bacterial culture forenteric pathogens
	Fecal Giardia antigen
	Fecal Cryptosporidium antigen
	Fecalova/cysts/parasites (including B. hominis and D. fragilis)
	Norovirus/Rota virus enzyme immunoassay
	Calprotectin
	Fecal occult blood testing

Preparation of stool samples for faecal microbiota transplantation. Larger particles are eliminated by filtration after the faces (100–150 g) are homogenized in saline solution (NaCl, 0.9%). Within fewer than 6 h, the fresh faecal sample can be used for FMT. In contrast, the fresh faecal preparation is subjected to additional filtration procedures, cryoprotected in 10% glycerol, frozen, and stored at 80°C for later use. The process of making FMT capsules includes adding the freeze-drying protectant glycerol (20%), centrifuging the mixture at a high speed for 10,000 g, discarding the supernatant, and combining the sediment with an enteric-soluble capsule to be stored at −80°C.,[19] the substance can be lyophilized (vacuum dried) to create faecal powder that can be put inside of capsules and kept at a temperature of −80°C for later usage. It is known as washed microbiota preparation (WMP) when numerous microfiltration, centrifugation, and suspension processes are completed automatically. Washed microbiota transplantation, often known as WMT, is the transfer of WMP through colonic transendoscopic enteral tubing (TET).

DELIVERY METHOD

Techniques of FMT delivery: Lower gastrointestinal routes, such as enema colonoscopy and colonic TET, and upper GI routes, such as nasogastric/nasoduodenal/nasojejunal tubes and capsules, are divided among the delivery techniques.

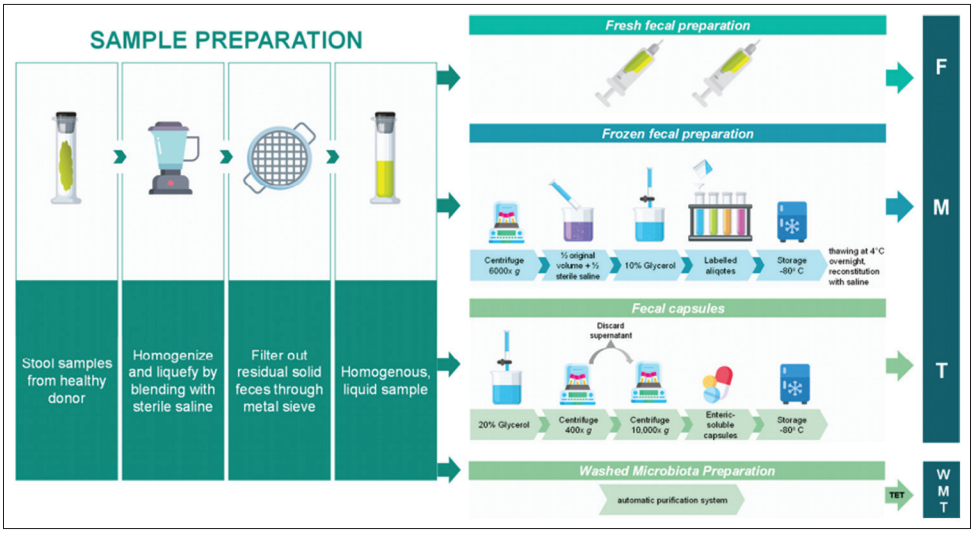

Figure 1: Method for fecal sample preparation for feacal microbiota transplantation

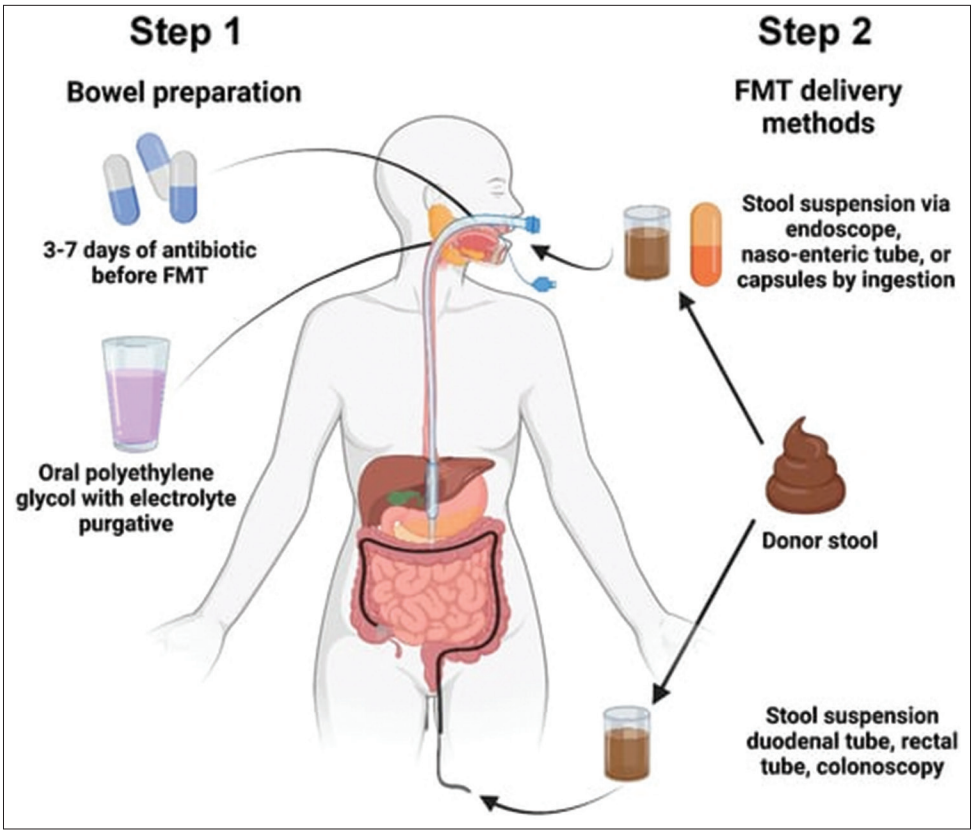

Figure 2: Bowel preparation and different methods for fecal microbiota transplantation

When using one of the upper GI routes, FMT can be administered using a nasal tube that is put through the nose and reaches the stomach, duodenum (nasoduodenal tube, or jejunum (nasojejunal tubes) to deliver the faecal transplant [Figure 2].[20] The most recent delivery approach is oral administration of encapsulated FMT, which has been demonstrated to be safe and efficacious[21,22] and is better accepted by patients [Figure 2]. The term "autologous FMT" is used to describe faecal stool that is taken from the patient who will receive it; otherwise, the term "allogenic or heterologous FMT" is used.

FMT IN HUMAN DISEASES

FMT: First Use for C. Difficile Infections

FMT frequently only needs one administration, eliminates C. difficile without the need for full microbiota engraftment,[23] and alters the patient's gut microbiota in a major and lasting way.[24,25] Even regular fidaxomicin and vancomycin, which are preferred medications to treat CDI, were compared to FMT and shown to be inferior.[26] These days, FMT for C. difficile clinical trials are also carried out at home.[27] Kumar *et al.* used human faecal transplantation in gnotobiotic mice to examine the colonization capacity of the donor, recipient, and recipient post-FMT in order to give light on how FMT reconstitutes the gut microbiota. In the recipient's pre-FMT, which contained Enterobacteriaceae, Lactobacillaceae, Enterococcaceae, and an abnormally higher proportion of Clostridiales (including C. difficile), members of the families Bacteroidaceae and Lachnospiraceae were not highly represented. The relative abundance of Bacteroidaceae and Lachnospiraceae increased to levels similar to the donor in gnotobiotic mice transplanted with faecal stools from patients who received FMT after 3 days, but it decreased to 7% of the donor in gnotobiotic mice transplanted with stools from patients who received FMT in the last 2–4 weeks. These findings suggested that commensal Bacteroidaceae and Lachnospiraceae begin colonizing the recipient's gut soon after FMT and compete with non-commensal clostridiales for niche space.[28]

FMT: Inflammatory Bowel Disease

The unique microbiota profile attained after FMT and linked to a clinical response, together with the degree of engraftment, are essential aspects of FMT in persons with ulcerative colitis that need to be researched, along with the variability in the remission rate. To address this problem, a first small experiment including five patients discovered that clinical remission was connected with a donor similarity index of 40–50% following a single FMT by colonoscopy in 60% of the patients.[29] FMT did increase microbial diversity, but this increase was higher in patients who achieved remission and

Table 3: Adverse events of FMT

Minor	Abdominal discomfort
	Bloating flatulence
	Diarrhea/constipation
	Borborygmus
	Nausea/vomiting (particularly withoral FMT route)
	Transient fever
Serious	Complications of endoscopy (perforation, bleeding)
	Adverse effects related to sedation (aspiration)
	Transmission of enteric pathogens
	Peritonitis in a patient undergoing peritoneal dialysis
	Pneumonia
	IBD flares Infection and/orsepsis (infection may be along-term sequelae)
	Post-infectious irritable bowel syndrome
Potential	Transmission of unrecognized infectious agents that cause illness years later (e.g., hepatitis C and HIV)
	Induction of chronic diseases based on alterations in the gut microbiota (e.g., obesity, diabetes, atherosclerosis, IBD, colon cancer, nonalcoholic fatty liver disease, IBS, asthma, and autism)

IBD: Inflammatory bowel disease, HIV: Human immunodeficiency virus, FMT: Fecal microbiota transplantation

was associated with an enrichment of Eubacterium hallii and Roseburia inulivorans and increased levels of SCFAs compared to patients who did not achieve remission.[30] Paramsothy *et al.* analyzed the faecal samples collected before and after intensive FMT treatment (5 days/week, for 8 weeks). Instead, individuals who did not exhibit remission exhibited elevated levels of lipopolysaccharide, Sutterella wadsworthensis, and Fusobacterium gonidi-aformans. Interestingly, the presence of Bacteroides in the donor stool was associated with FMT response, whereas Streptococcus species were associated with a lack of response, when donor microbiota profiles and remission were correlated.[30] The long-term sustained remission is associated with overall increased butyrate production and levels of butyrate producers.[31] These pieces of evidence again point out the pivotal importance of the donor microbiota profile and the number of transplants choice when designing an FMT intervention for ulcerative colitis.

CROHN'S DISEASES

In a study published in 2017, He *et al.* assessed the effectiveness and safety of numerous fresh FMTs in 25 patients with Crohn's disease who also had an intra-abdominal inflammatory mass. Even though FMT temporarily relieves clinical symptoms, it does not result in a long-lasting therapeutic effect, as evidenced by the fact that less than half of the patients demonstrated clinical response and remission 3 months after the first FMT and that this percentage dropped at 12 and 18 months.[32] By examining mucosal T-cell phenotypes and inflammatory markers, Vaughn *et al.* examined in a small cohort (nineteen individuals) the impact of a single FMT not only on clinical remission but also on mucosal inflammation. FMT led to a rise in gut microbial diversity, a number of regulatory T-cells, and remission in about half of the patients.[33] FMT single treatment, however first seeming promising, is unable to sustain clinical remission over the long run.

ADVERSE EVENTS OF FMT

The majority of clinical trials and systemic reviews showed that minor adverse events like abdominal pain, diarrhea, constipation, and low-grade fever were briefly experienced after FMT, and rare severe side effects were frequently linked to potential endoscopy and sedation complications.[24,34-37] Table 3 lists the most frequent adverse incidents.[38,39]

FUTURE OF FMT

Our knowledge of the GI microbiota (GiMb) has advanced significantly in recent years thanks to the Human Microbiome Project[40] and the European-based Metagenomics of the Human Intestinal Tract (MetaHIT) consortia, which were founded to investigate the human GI microbiome. GiMbs are now thought to actively contribute to human health and immune-mediated disorders rather than only being thought of as benign intestinal colonizers. It is interesting to note that most studies on the gut microbiome to date have concentrated on the structure and operation of bacterial communities, despite the fact that it also includes archaea, viruses, and eukarya.[41] However, there has not been much research done on how viruses, such as bacteriophages, fungi, and protozoa, affect the gut microbiota,[42] which, if done, might completely change how FMT is used and how its potential therapeutic approaches are developed.

For instance, when the gut microbiota is still developing in children, asthma usually develops.[43] Recent research has shown a connection between asthma and gut microbial dysbiosis.[44] Even though it is still a relatively new field of study, data to date suggests that the gut microbiota may be a valuable target for the prevention or management of allergic asthma. To prevent and cure microbiome dysbiosis and restore a healthy microbiome, probiotics, faecal microbiota transplants, and bacterial lysates have not yet reached clinical use.[45] As a result, more mechanistic study is required to clarify the function of microbial composition in the etiology of asthma, and FMT may be a future asthma therapeutic option.

TAKE HOME POINT

1. Over the past few years, FMT has gained recognition as a valid therapeutic alternative by mainstream therapists due to its clever simplicity and cost-effectiveness.
2. It should be emphasized, nonetheless, that the faecal microbiota is a complicated starting point, and those planning to reverse engineer it will likely have to determine how the microbial communities in the samples interact with one another and its mode of operation.
3. Future study should concentrate on determining the long-term effects of FMT. Unquestionably, FMT is effective against C. difficile infection. Before wide adoption of FMT as a therapeutic benefit beyond recurrent CDI, further randomized controlled trials' pieces of evidence are required. Among other conditions, it is being investigated as a therapy for IBD, IBS, and metabolic syndrome/insulin resistance.

REFERENCES

1. Duncan SH, Louis P, Flint HJ. Cultivable bacterial diversity from the human colon. Lett Appl Microbiol 2007;44:343-50.
2. Rashid MU, Zaura E, Buijs MJ, Keijser BJ, Crielaard W, Nord CE, *et al.* Determining the Long-term effect of antibiotic administration on the human normal intestinal microbiota using culture and pyrosequencing methods. Clin Infect Dis 2015;60 Suppl 2:S77-84.
3. Chang JY, Antonopoulos DA, Kalra A, Tonelli A, Khalife WT, Schmidt TM, *et al.* Decreased diversity of the fecal microbiome in recurrent *Clostridium difficile*-associated diarrhea. J Infect Dis 2008;197:435-8.
4. Tamburini S, Shen N, Wu HC, Clemente JC. The microbiome in early life: Implications for health outcomes. Nat Med 2016;22:713-22.
5. Kernbauer E, Ding Y, Cadwell K. An enteric virus can replace the beneficial function of commensal bacteria. Nature 2014;516:94-8.
6. David LA, Maurice CF, Carmody RN, Gootenberg DB, Button JE, Wolfe BE, *et al.* Diet rapidly and reproducibly alters the human gut microbiome. Nature 2014;505:559-63.
7. Abdollahi-Roodsaz S, Abramson SB, Scher JU. The metabolic role of the gut microbiota in health and rheumatic disease: Mechanisms and interventions. Nat Rev Rheumatol 2016;12:446-55.
8. Collado MC, Rautava S, Isolauri E, Salminen S. Gut microbiota: A source of novel tools to reduce the risk of human disease? Pediatr Res 2015;77:182-8.
9. Pluznick JL, Protzko RJ, Gevorgyan H, Peterlin Z, Sipos A, Han J, *et al.* Olfactory receptor responding to gut microbiota-derived signals plays a role in renin secretion and blood pressure regulation. Proc Natl Acad Sci U S A 2013;110:4410-5.
10. Bakken JS, Borody T, Brandt LJ, Brill JV, Demarco DC, Franzos MA, *et al.* Treating *Clostridium difficile* infection with fecal microbiota transplantation. Clin Gastroenterol Hepatol 2011;9:1044-9.
11. Weingarden AR, Chen C, Bobr A, Yao D, Lu Y, Nelson VM, *et al.* Microbiota transplantation restores normal fecal bile acid composition in recurrent *Clostridium difficile* infection. Am J Physiol Gastrointest Liver Physiol 2014;306:G310-9.
12. Shankar V, Hamilton MJ, Khoruts A, Kilburn A, Unno T, Paliy O, *et al.* Species and genus level resolution analysis of gut microbiota in *Clostridium difficile* patients following fecal microbiota transplantation. Microbiome 2014;2:13.
13. Kelly CR, Kahn S, Kashyap P, Laine L, Rubin D, Atreja A, *et al.* Update on fecal microbiota transplantation 2015: Indications, methodologies, mechanisms, and outlook. Gastroenterology 2015;149:223-37.
14. Sokol H, Galperine T, Kapel N, Bourlioux P, Seksik P, Barbut F, *et al.* Faecal microbiota transplantation in recurrent *Clostridium difficile* infection: Recommendations from the French Group of Faecal microbiota Transplantation. Dig Liver Dis 2016;48:242-7.
15. Ramai D, Zakhia K, Ofosu A, Ofori E, Reddy M. Fecal microbiota transplantation: Donor relation, fresh or frozen, delivery methods, cost-effectiveness. Ann Gastroenterol 2019;32:30-8.
16. Marotz CA, Zarrinpar A. Treating obesity and metabolic syndrome with fecal microbiota transplantation. Yale J Biol Med 2016;89:383-8.
17. Nicco C, Paule A, Konturek P, Edeas M. From donor to patient: Collection, preparation and cryopreservation of fecal samples for fecal microbiota transplantation. Diseases 2020;8:9.
18. Allegretti JR, Korzenik JR, Hamilton MJ. Fecal microbiota transplantation via colonoscopy for recurrent *C. difficile* Infection. J Vis Exp 2014;94:52154.
19. Kao D, Roach B, Silva M, Beck P, Rioux K, Kaplan GG, *et al.* Effect of oral capsule-vs colonoscopy-delivered fecal microbiota transplantation on recurrent *Clostridium difficile* infection: A Randomized Clinical Trial. JAMA 2017;318:1985-93.
20. Holvoet T, Joossens M, Vázquez-Castellanos JF, Christiaens E, Heyerick L, Boelens J, *et al.* Fecal microbiota transplantation reduces symptoms in some patients with irritable bowel syndrome with predominant abdominal bloating: Short-and long-term results from a placebo-controlled randomized trial. Gastroenterology 2021;160:145-57.e8.
21. Crothers JW, Chu ND, Nguyen LT, Phillips M, Collins C, Fortner K, *et al.* Daily, oral FMT for long-term maintenance therapy in ulcerative colitis: Results of a single-center, prospective, randomized pilot study. BMC Gastroenterol 2021;21:281.
22. Mocanu V, Zhang Z, Deehan EC, Kao DH, Hotte N, Karmali S,

et al. Fecal microbial transplantation and fiber supplementation in patients with severe obesity and metabolic syndrome: A randomized double-blind, placebo-controlled phase 2 trial. Nat Med 2021;27:1272-9.
23. Staley C, Kelly CR, Brandt LJ, Khoruts A, Sadowsky MJ. Complete microbiota engraftment is not essential for recovery from recurrent *Clostridium difficile* infection following fecal microbiota transplantation. mBio 2016;7:e01965-16.
24. Van Nood E, Vrieze A, Nieuwdorp M, Fuentes S, Zoetendal EG, de Vos WM, *et al.* Duodenal infusion of donor feces for recurrent *Clostridium difficile*. N Engl J Med 2013;368:407-15.
25. Jalanka J, Hillamaa A, Satokari R, Mattila E, Anttila VJ, Arkkila P. The long-term effects of faecal microbiota transplantation for gastrointestinal symptoms and general health in patients with recurrent *Clostridium difficile* infection. Aliment Pharmacol Ther 2018;47:371-9.
26. Hvas CL, Dahl Jørgensen SM, Jørgensen SP, Storgaard M, Lemming L, Hansen MM, *et al.* Fecal microbiota transplantation is superior to fidaxomicin for treatment of recurrent *Clostridium difficile* Infection. Gastroenterology 2019;156:1324-32.e3.
27. Drekonja DM, Shaukat A, Zhang JH, Reinink AR, Nugent S, Dominitz JA, *et al.* Microbiota or placebo after antimicrobial therapy for recurrent *Clostridioides difficile* at home: A clinical trial with novel home-based enrollment. Clin Trials 2021;18:622-9.
28. Kumar R, Maynard CL, Eipers P, Goldsmith KT, Ptacek T, Grubbs JA, *et al.* Colonization potential to reconstitute a microbe community in patients detected early after fecal microbe transplant for recurrent *C. difficile*. BMC Microbiol 2016;16:5.
29. Damman CJ, Brittnacher MJ, Westerhoff M, Hayden HS, Radey M, Hager KR, *et al.* Low level engraftment and improvement following a single colonoscopic administration of fecal microbiota to patients with ulcerative colitis. PLoS One 2015;10:e0133925.
30. Paramsothy S, Nielsen S, Kamm MA, Deshpande NP, Faith JJ, Clemente JC, *et al.* Specific bacteria and metabolites associated with response to fecal microbiota transplantation in patients with ulcerative colitis. Gastroenterology 2019;156:1440-54.e2
31. Fuentes S, Rossen NG, van der Spek MJ, Hartman JH, Huuskonen L, Korpela K, *et al.* Microbial shifts and signatures of long-term remission in ulcerative colitis after faecal microbiota transplantation. ISME J 2017;11:1877-89.
32. He Z, Li P, Zhu J, Cui B, Xu L, Xiang J, *et al.* Multiple fresh fecal microbiota transplants induces and maintains clinical remission in Crohn's disease complicated with inflammatory mass. Sci Rep 2017;7:4753.
33. Vaughn BP, Vatanen T, Allegretti JR, Bai A, Xavier RJ, Korzenik J, *et al.* Increased intestinal microbial diversity following fecal microbiota transplant for active Crohn's disease. Inflamm Bowel Dis 2016;22:2182-9046.
34. Brandt LJ, Aroniadis OC, Mellow M, Kanatzar A, Kelly C, Park T, *et al.* Long-term follow-up of colonoscopic fecal microbiota transplant for recurrent *Clostridium difficile* infection. Am J Gastroenterol 2012;107:1079-87.
35. Cammarota G, Ianiro G, Gasbarrini A. Fecal microbiota transplantation for the treatment of *Clostridium difficile* infection: A systematic review. J Clin Gastroenterol 2014;48:693-702.
36. Mattila E, Uusitalo-Seppälä R, Wuorela M, Lehtola L, Nurmi H, Ristikankare M, *et al.* Fecal transplantation, through colonoscopy, is effective therapy for recurrent *Clostridium difficile* infection. Gastroenterology 2012;142:490-6.
37. Rossen NG, MacDonald JK, de Vries EM, D'Haens GR, de Vos WM, Zoetendal EG, *et al.* Fecal microbiota transplantation as novel therapy in gastroenterology: A systematic review. World J Gastroenterol 2015;21:5359-71.
38. Choi HH, Cho YS. Fecal microbiota transplantation: Current applications, effectiveness, and future perspectives. Clin Endosc 2016;49:257-65.
39. Vindigni SM, Surawicz CM. Fecal microbiota transplantation. Gastroenterol Clin North Am 2017;46:171-85.
40. Turnbaugh PJ, Ley RE, Hamady M, Fraser-Liggett CM, Knight R, Gordon JI. The human microbiome project. Nature 2007;449:804-10.
41. Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. Nature 2012;486:207-14.
42. Mcilroy J, Segal J, Mullish B, Quraishi MN, Gasbarrini A, Cammarota G, *et al.* Current and future targets for faecal microbiota transplantation. Hum Microbiome J 2019;11:100045.
43. Ly NP, Litonjua A, Gold DR, Celedón JC. Gut microbiota, probiotics, and Vitamin D: Interrelated exposures influencing allergy, asthma, and obesity? J Allergy Clin Immunol 2011;127:1087-94.
44. Johnson C, Ownby D. The infant gut bacterial microbiota and risk of pediatric asthma and allergic diseases. Transl Res 2017;179:60-70.
45. Hufnagl K, Pali-Schöll I, Roth-Walter F, Jensen-Jarolim E. Dysbiosis of the gut and lung microbiome has a role in asthma. Semin Immunopathol 2020;42:75-93.

How to cite this article: Padole S, Ingle S, Ingole J. Fecal Microbiota Transplantation. Med J Basic Appl Res 2023; 4(1):22-28.

Conflicts of Interest: None. **Source of support:** None.