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Fecal microbiota transplantation as a curative strategy during intestinal and neurological disorders

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ABSTRACT

The symbiotic gut microbiota is essential for optimal physiology. The gut microbiota composition and diversity may significantly alter leading to dysbiosis which may give rise to various pathologies in the host. Dysbiosis is correlated to metabolic syndrome, neurodegenerative disorders, gastrointestinal tract (GIT) complications, liver disease amongst many others. Fecal microbiota transplantation (FMT) is an effective strategy to introduce feces (containing normal microbial population) from a healthy donor into the gut of a compromised recipient. Originally, FMT was introduced to mainly treat gastrointestinal complications caused by pathogenic microbes. However, over the years FMT has gained popularity as a treatment regimen for relief during GIT and nervous system disorders. Initial findings from FMT studies are positive and promising in rodents and clinical trials. However, clinical applicability of FMT is currently limited due to its practical dissent in treating diseases. Moreover, FMT had little success in treating some conditions which could be due to variations in FMT strategy and patient factors. In this article, new reports resulting from FMT shall be reviewed with respect to a few intestinal and brain diseases.

KEYWORDS: Dysbiosis; Feces; Gut microbiota; Gastrointestinal; Inflammation; Metabolites

INTRODUCTION

The human gut is host to innumerable complexity and number of microorganisms called the microbiota that exhibit commensal and/or symbiotic relationship with the host. The gut microbiota is composed of bacteria, fungi, archaea, parasites, viruses and bacteriophages whose number for seeded the host total body cell counts (Thursby and Juge, 2017; Adak A and Khan, 2019). The gut microbiota constitutively interacts with the intestinal tract. Such interaction is essential to maintain optimal host physiological roles that provide immune sensing and antigenic tolerance including development of immunocytes (Shapiro et al., 2014; Rooks and Garrett, 2016; Illiano et al., 2020). The interplay also restrains pathogen colonization and propagation benefitting the host. Optimal gut microbial composition and diversity is essential in maintaining commensal/symbiotic [1]relationship (Kataoka, 2016). However, the gut microbial community may undergo shifts due to several factors that may alter its diversity (Karl et al., 2017; Mitrea et al., 2022). Exogenous factors include diet, therapeutic drugs, alcohol etc., whereas the endogenous factors comprise age, disease state, sex, genetics, host mucosa and immunity etc. (Karl et al., 2017; Bibbò et al., 2016; Gomaa, 2020; Nikolova et al., 2021). Gut dysbiosis in the host may trigger pathological outcomes ranging from metabolic disorders, inflammation to neuropsychiatric complications (Nikolova et al., 2021; Boulangé et al., 2016). Therapeutic interventions in treating such disorders are limited due to its inherent intractability. Gut microbial manipulations can be an approach especially in

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gut diseases such as inflammatory bowel disease (IBD) (Weingarden and Vaughn, 2017). Fecal microbiota transplantation (FMT) is a tried and tested strategy whereby normal fecal microbiota (from the feces) of a healthy donor is transferred to the diseased recipient's intestine. FMT has proven successful towards recurring Clostridium difficile infection and might be effective against IBD (Weingarden and Vaughn, 2017; Song and Kim, 2019). FMT has the potential to treat gastrointestinal diseases, cancer, autoimmune diseases, obesity, and metabolic syndrome.

Obesity has been demonstrated to have a link with dysbiosis. Obese mice show a relatively higher proportion of Firmicutes as against Bacteroidetes when compared to lean animals with similar findings in humans (Pedersen et al., 2013; Hansen et al., 2014; Walters et al., 2014). Interestingly, FMT from obese to the germ-free mice led to obesity onset (Turnbaugh et al., 2008; Backhed et al., 2007). Also, FMT from non-obese mice to obese led to dramatic improvement and shift to a lean status (Vrieze et al., 2020). The use of pro/prebiotics however has provided relief only in certain GIT complications. Currently, FMT is seen as a useful intervention in GIT and neurological disorders, especially those linked to the intestinal microbiome.

Fecal microbiota transplantation

The gut microbiome maintain communication with various tissues and organs exploiting the various microbial-derived metabolites and the vagus nerve (Pluznick et al., 2013). Many gut-microbial metabolites (tryptophan/indole metabolites, short-chain fatty acids, secondary bile acids, branched chain amino acids etc.) act by adjusting the hormonal and immune signaling pathways in the host (Table 1) (Maynard et al., 2012; El Aidy et al., 2015). Moreover, these metabolites stimulate the parasympathetic nervous system playing a crucial role in homeostasis through metabolic regulation (De Vadder et al., 2014). Furthermore, by modulating the gut-brain axis (GBA), microbial metabolites can profoundly influence the brain (Rhee et al., 2009). A dysbiotic gut may trigger metabolic disorders including neurological and neuropsychiatric complications. Gut dysbiosis may return to a normal state with interventions such as administration of specific antibiotic against pathogenic strains or probiotics or FMT (Deidda et al., 2021). However, use of antibiotics has limitations such as development of microbial resistance and off-target actions (Gorski et al., 2016). Probiotic supplements though beneficial in GIT complications, however it provides only a limited bacterial species and strains (Deidda et al., 2021; Dimidi et al., 1017). FMT is an alternative, physiologically relevant strategy tried to provide relief from certain human diseases linked to dysbiosis.

In FMT, feces containing active microbiota belonging to a healthy, disease-free donor is transferred to a subject with a disease linked to dysbiosis for therapeutic purpose (Ramai et al., 2013; Rossen et al., 2015; Vendrik et al., 2020). FMT has advantages as it transfers the entire pool of microbes safely and does not provoke immune response (Rossen et al., 2015). The donor fecal matter needs to be properly processed before transfer to the individuals (Kao et al., 2017; Haifer et al., 2022; Allegretti et al., 2020; Bajaj et al., 2019). Processed fecal matter can be administered to subjects by delivery methods broadly classified into upper (such as oral capsules) and lower gastrointestinal routes (by colonoscopy). However, clinical applicability of FMT is limited due to its practical dissent in treating diseases.

FMT and human diseases

FMT has been tried and tested in humans as a therapeutic regimen for several illnesses. However, the efficacy of FMT in treating certain human diseases is still in its infancy. Appropriate literature search and selection were carried out using various keywords in public databases such as PubMed (NLM, NIH, USA) and google scholar. The following diseases have been reviewed in detail owing to numerous data that underscores dysbiosis as a

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reason for their possible cause, progression and maintenance in the gut and the brain.

In irritable bowel syndrome

Irritable bowel syndrome (IBS) is a complex, less understood and chronic condition characterized by a group of symptoms that largely results from abnormal gut motility. Abdominal pain, diarrhoea and/or constipation, weight loss, anxiety and depression are typical symptoms, with 4-10% of people worldwide suffer from it (Ford et al., 2020). The etiology of IBS is unknown, and no single treatment is enough to provide relief. Physicians recommend changes in diet and lifestyle, medicines, probiotics supplement as interventions on an individual basis (Vasant et al., 2021; Masuy et al., 2020). IBS is generally characterized as IBS-D (diarrhoea-predominant), IBS-C (constipation-predominant) and IBS-M (mixed) subtypes. The gut microbiota composition and diversity of IBS subjects are different from the healthy ones indicating its role in IBS pathophysiology (Ohman and Simren, 2013). Modulation of gut microbiota may provide relief from IBS. FMT from healthy donors to IBS patients has been attempted in previous two studies (Johnsen et al.,2018; Halkjær et al., 2018).

Findings from one of those studies revealed decline of IBS symptoms, whilst the other found no effect. However, in a recent study of 165 patients with IBS to placebo (own faeces), 30 g FMT or 60 g FMT at a ratio of 1:1:1 was conducted (Holster et al., 2021). Results obtained showed significant decrease in IBS symptoms such as fatigue and improvement in physical health at three months after the introduction of FMT. Gut bacterial profiles in the group receiving FMT exhibited significant changes. Furthermore, the response to FMT increases with the fecal dose (Holster et al., 2021). FMT in the form of capsules were ineffective, rather colonoscopy and gastroscopy were successful in alleviating IBS symptom in patients. Allogenic FMT (faecal material from a healthy donor) is more potent compared to autologous FMT (own fecal matter).

Allogenic FMT altered the gut microbiota composition in patients and was detectable for 6 months, whereas no such observation was made by autologous FMT (El-Salhy et al., 2020). Allogenic FMT including autologous FMT however made no impact on the fecal metabolites measured. However, correlations between the gut microbial composition and secreted metabolites showed that the microbe-metabolite interactions get disrupted after allogenic FMT compared to autologous FMT. In another finding, however, FMT was ineffective in providing relief from IBS symptoms in randomized-controlled trials (RCTs) which could be due to variations in FMT strategy and patient factors (Myneedu et al., 2019). In one more RCTs, FMT relieved IBS symptoms as compared to placebo (autologous transplant). However, the positive effects declined after one year and a second FMT restored the effects to previous level in patients (Holvoet et al., 2021).

Additional RCT findings showed no differences in sex-related FMT response in IBS patients and in placebo (El-Salhy et al., 2021). However, the response rate (reduction in symptoms) was higher in female patients compared to males in IBS-D case at one month and three months after FMT.A 5-year effect was reported in a retrospective analysis (Hillestad et al., 2022). In the study, patients with all subtypes of IBS received FMT through nasojejunal administration, colonoscopy administration, or freeze-dried capsules from healthy donors. 50% of the patients showed improvement in symptoms after one month and 70% and 75% after one and two years, respectively. Also, after five years, 60% of patients showed improvement. These results point that repeated FMT might be required towards sustained relief from IBS symptoms and improve the quality of patient's life. In yet another investigation, double-blind, randomised, placebo-controlled patients aged 18-65 years with IBS-D were observed (Aroniadis et al., 2019). The subjects received either 75 FMT capsules or 75 placebo capsules over 3 days (25 capsules per day).

All the patients went to the alternate treatment at 12 weeks. Findings from the study indicate no significant relief from IBS symptoms because of FMT in patients compared to placebo. In a double-blind randomized placebo-controlled, parallel-group, single-centre study patients with non-constipated IBS were investigated (Johnsen et al., 2020). FMT was able to trigger significant relief from fatigue and improved the quality of life in the IBS patients compared to the placebo group. The administration route (colonoscopy or gastroscopy) of FMT is vital to get effective response and improve the quality of life in patients. Long-term effect of FMT during IBS is unknown and would be an interesting area for future research.

In inflammatory bowel disease

Inflammatory bowel disease (IBD) comprises of Crohn's disease, CD and ulcerative colitis, UC) that affects the gastrointestinal tract (GIT). It is associated with chronic inflammation of the GIT leading to persistent diarrhoea, weight loss, fatigue, abdominal pain and rectal bleeding in sufferers (Ananthakrishnan et al., 2018). The etiology of IBD is unknown but is attributed to attenuated host immune responses. Altered immune response to environmental triggers, such as microorganisms including viruses (Ananthakrishnan et al., 2018; Lee et al., 2018; Canakis and Qazi, 2020) may drive IBD. Secondly, individual genetic predisposition also might be the driver of IBD (Younis et al, 2020). In a recent study by Zhang et al, mice with experimental UC when subjected to FMT displayed higher body mass and gut weight and length (Zhang et al., 2020). There was a marked decrease in histopathological changes and cytokine expression in the gut accompanied with down-regulation of genes involved in pro-inflammatory mediator NF-κB signalling were observed compared to control animals. FMT increased the relative population of Firmicutes and reducing the abundances of Bacteroidetes and Proteobacteria. FMT treated mice showed higher counts of *Lactobacillus*, *Butyric coccus* along with a fall in the population of *Helicobacter*, *Bacteroides* and *Clostridium* (Zhang et al., 2020). Moreover, FMT increased the availability of anti-inflammatory SCFAs in the colon. These observations points to the beneficial role of FMT in alleviating UC in mice model. Gut bacteria have a prominent role in CD onset and progression and hence FMT might be useful as a therapeutic strategy. In fact, in a randomized, single-blind pilot trial of FMT, significant remission was reported from CD with decrease in CRP level (Sokol et al., 2020). Also, elevated dose of fecal matter led to beneficial effect and help sustain the remission state. The importance of FMT is further supported by antibiotic administration in murine models. Separate antibiotic pre-treatment with vancomycin, metronidazole and streptomycin of murine fecal donors before FMT to colitic animals gave interesting results. Gut microbiota and fecal metabolome analyses of the FMT-recipient animals showed reduced ability of FMT to control intestinal inflammation (Strati et al., 2021). Both vancomycin and streptomycin treatment, but not metronidazole led to the inability of FMT to attenuate recipient intestinal inflammation. Also, pathobiont growth with increased presence of oxidative microbial-secreted metabolites in the gut was akin to what was observed during IBD (Strati et al., 2021).

Characterization of the beneficial gut microbiota remains an enigma. In a double-blind clinical study by Paramsothy et al on UC, FMT increased the diversity and altered the composition of microbial population in the recipient fecal and colon samples (Paramsothy et al., 2019). Patients with UC remission after FMT exhibited increased counts of *Eubacterium. hallii* and *Roseburia inulivorans* as compared to control. Also, microbial generation of SCFAs and secondary bile acids were observed in the FMT recipient fecal and colon samples. Additionally, patients without remission from FMT were associated with enhanced *Fusobacterium gonidiaformans*, *Sutterellawads worthensis*, and *Escherichia* species counts with high levels of lipopolysaccharide (LPS) and heme synthesis

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(Paramsothy et al., 2019). Metagenomic investigations in the donor fecal sample and recipient FMT have identified *Odoribacter splanchnicus* as a key microbial mediator in providing relief from intestinal inflammation during UC (Lima et al., 2022). O. *splanchnicus* present in the recipient FMT sample enhanced the $Foxp3^+/ROR\gamma t^+$ regulatory T cells and induced the expression of interleukin IL-10, and generation of SCFAs thereby providing significant relief from colitis in mice. In an interesting finding, FMT from IBD sufferer plus depression (IBD/D^+) to recipient mice induces altered immune response with IBD-like colitis by increasing the myeloperoxidase activity and expression of pro-inflammatory mediators such as IL-1β and IL-6 in the colon (Jang et al., 2021). Also, FMTs from patients afflicted with IBD/D^+ led to anxiety-/depression-like behaviours and accompanied with decrease in hippocampal $BDNF^+/NeuN^+$ cell number. Moreover, FMT from healthy volunteer into mice previously transplanted with IBD/D^+-feces attenuated depression-/anxiety-like behaviours, hippocampal $NF-\kappa B^+/Iba1^+$ and $LPS^+/Iba1^+$ cell counts associated with low circulating and fecal levels of LPS and colonic IL-1β (Jang et al., 2021).CD is a chronic condition exhibiting sustained inflammation and irritation of the digestive tract accompanied with dysbiosis. Symptoms of CD include abdominal pain, diarrhoea, rectal-bleeding and weight loss. Although the etiology of CD is unknown, several factors increase the risk of getting the disease such as autoimmune disease, host genetics and smoking. In a randomized study, FMT was performed in a group of patients (n = 27) with CD by gastroscopy and colonoscopy (Yang et al., 2020). A second FMT was performed after one week. Most patients (n =18) with CD showed significant clinical remission (66%) from FMT. No difference was observed between gastroscopy and colonoscopy as the mode of fecal delivery. However, FMT recipients exhibited lower microbiota diversity in relation to the donors.

Moreover, recipients with second FMT after one week exhibited increased intestinal microbiota richness, with elevated populations of *Clostridium, Cronobacter, Fusobacterium*, and *Streptococcus* and reduced counts of *Bacteroides*, *Eubacterium*, *Faecalibacterium*, and Roseburia (Yang et al., 2020). Overall, the results from clinical studies on FMT towards IBD remission were favourable. However, there is an excessive degree of inconsistency in responses even amongst the recipients undergoing identical treatment protocol. FMT as a successful therapeutic strategy in IBD is however difficult to demonstrate. A major constraint of FMT during IBD is to maintain remission state in the recipients. Therefore, FMT at present is less of a treatment strategy and more of an intervention in IBD clinical trials and requires further refinements and normalization as a therapeutic strategy in the future.

In constipation

Constipation is a common condition characterized by a state whereby a person develops infrequent bowel motion with firm and dry feces. Constipation generally occurs due to slow gut contractions associated with increased reabsorption of water, resulting in dry and solid stool. Constipation can arise from medications, minimal dietary fibre, IBS, low water intake, intestinal complications and various other factors. Impaired gut motility is correlated with systemic inflammation and dysbiosis. FMT has been reported as effective in treating constipation by remodelling the gut microbiota and correcting dysbiosis. In a new clinical finding, individuals receiving FMT showed rise in bacterial populations of *Clostridiales, Fusicateni bacter* and *Paraprevotella* with significant relief from symptoms as compared to pre-FMT state (Zhang et al., 2021). On the other hand, however, gut bacteria including *Lachnoan aerobaculum* underwent reduction in counts, which might correlate with the severity of patients' constipation.

The levels of SCFAs were comparable between pre-and post-FMT. The pro-inflammatory

cytokine IL-8 level was however reduced in FMT recipients, but the others such as IL-4, IL-6, IL-10 and IL-12p70 remain unaltered (Zhang et al., 2021). Hence, FMT can regulate gut microbiota ecology and cytokine levels thereby influencing systemic inflammation in constipated individuals. In another study, eight patients underwent three exposures to FMT (Xie et al., 2021). Clinical remission in the recipients up to 75% was achieved after the third exposure. Additionally, 16S rRNA sequence show decreased population of *Bacteroidetes* and *Firmicutes* with associated increase in *Actinobacteria* (*Bifidobacterium*), Proteobacteria (Escherichia), and *Firmicute* (*Lactobacillus*) numbers after FMT (Xie et al., 2021). Metabolomic and biochemical analyses showed that the fecal samples of FMT-exposed patients exhibited relatively elevated levels of N-Acetyl-L-glutamate, gamma-L-glutamyl-L-glutamic acid and Glycero phosphocholine. Increased circulatory levels of L-Arginine, L-Threonine, Ser-Arg, Indole acrylic acid, Phe-Tyr, 5-L-Glutamyl-L-alanine was also detected in the FMT recipients.

These changes in the gut microbial metabolome of FMT recipients were accompanied with significant relief in STC symptoms. In another chronic functional constipation (CFC) study, thirty-four patients with FMT exhibited a clinical cure rate of 73.5% (25/34) followed by a remission rate of 14.7% (Tian et al., 2020). FMT induced intestinal peristalsis and motility and raised the levels of circulating nitric oxide and serotonin. The sequencing of 16S rRNA from the FMT gut microbiome showed increased counts of Bacteroides, *Klebsiella*, *Megamonas, Erysipelotrichaceae* and *Epulopiscium* which might be the cause of CFC (Tian et al., 2020). On the other hand, high prevalence of *Prevotella, Acidaminococcus* and *Butyricimonas* might be the reason for relief from CFC in FMT patients. Altogether, these findings suggest a role of FMT in improving gut microbiota ecology and metabolomic profile which could be beneficial in abating constipation.

In neurological disorders

The gut-brain axis (GBA) has been visualized as one of the primary routes whereby the brain communicates with the gut and vice-versa. The GBA involving the enteric nervous system (ENS) and CNS is critical for homeostatic regulation (Mayer, 2011). Over the years, the gut microbiome is seen as modulator of the GBA giving rise to the concept of microbiota-GBA (Rhee et al., 2009; Collins et al., 2012; Cryan et al., 2019; Cryan and Dinan, 2012). The gut microbiota-GBA is being considered as a vital modulator of the ENS and the CNS (Browning and Travagli, 2019). Gut dysbiosis may trigger alteration in microbiota-GBA inducing the onset and progression of some neurological disorders (Rao and Gershon, 2016; Margolis et al., 2021). Correction of dysbiosis by FMT might treat/cure dysbiosis-related neurological complications. Preliminary data indicate neurological disorders can be effectively managed by FMT, though few findings say the opposite. Parkinson's disease (PD) subjects typically show gut dysbiosis, however the link is unclear. FMT from PD mice model induced motor defects and striatal neurotransmitter decrease in healthy mice (Sun et al., 2018).

The population of *Firmicutes* and *Clostridiales* decreased, whilst an increase in Proteobacteria counts in fecal samples of PD mice with high fecal SCFAs level was observed. On the other hand, FMT reduced dysbiosis and fecal SCFAs level as well as physical impairment, and upregulated serotonin level in PD mice model. These findings show that dysbiosis might be participating in PD and that FMT can provide significant relief to PD mice by attenuating the TLR4/TNF-α signalling and neuroinflammation. In a similar finding, Zhao et al. reported gastrointestinal function impairment and altered behavioural performances in rotenone-induced PD mice model (Zhao et al., 2021).

However, FMT restored the gut bacteria composition and diversity and ameliorated gastrointestinal malfunctions and motor deficits in PD mice. FMT also decreased the levels of systemic inflammation in PD mice.

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Additionally, FMT treatment curtailed neuroinflammation in substantia nigra of the brain and lowered damage to dopaminergic neurons (Zhao et al., 2021). FMT treatment to PD mice also reduced gut and circulatory LPS levels, including the substantia nigra by attenuating the TLR4/MyD88/NF-κB signalling. Alzheimer's disease (AD) is the most prevalent form of dementia in senescent population exhibiting cognitive and memory decline. Clinically AD is an arduous to treat condition.

Although the etiology of AD is unknown, deposition of amyloid-β (Aβ) and tau-protein is a clinical feature in the patient's brain. The gut microbiota of AD patients and AD mice models differ in composition compared to healthy controls. FMT treatment improved the cognitive deficits and decreased the brain deposition of Aβ in APPswe/PS1dE9 transgenic (Tg) mice (Sun et al., 2019). Also, decreased phosphorylation of tau protein and the levels of Aβ40 and Aβ42 appeared in Tg mice. Further, synaptic plasticity was elevated in Tg mice with reduced expression of COX-2 gene and high fecal SCFAs level. These findings could be the effect of FMT which reversed the gut microbial composition (Sun et al., 2019). In another report, FMT from 5xFAD mice into normal C57BL/6 mice (5xFAD-FMT) reduced adult hippocampal neurogenesis and brain-derived neurotrophic factor (BDNF) expression with high p21 expression contributing to memory impairment (Kim et al., 2021). Also, elevated levels of gut TNF-α and interleukin-1β and in circulation were detected in 5xFAD-FMT mice. Composition of the 5xFAD-FMT mice gut microbiota showed differences compared control mice or wild type-FMT mice. Therefore, 5xFAD mice harboured a dysbiotic state that reduced neuronal generation by inducing colonic inflammation thereby aiding memory decline.

Depression is a complex, less-understood neuropsychiatric condition with extensive morbidity. Recent findings have reported gut microbiota compositional differences in humans and rodent models with depression. Altered brain chemistry was observed in patients and rodents with depression. Rat models exhibiting depression (CUMS), when subjected to FMT (CUMS+FMT) displayed improved symptoms of depression and colonic movements (Cai et al., 2022). Levels of serotonin, GABA and BDNF raised in the hippocampus of CUMS+FMT compared to the CUMS group. Also, GLP-1, IL-6 and LPS showed reduction in CUMS+FMT compared to CUMS. Also, CUMS group exhibited increase of glucagon-like peptide-1 (GLP-1), IL-6 and LPS and decrease of serotonin, GABA and BDNF when compared with the control (no depression) (Cai et al., 2022). The gut microbial richness of the CUMS+FMT group exhibit similarity to the control, whereas the richness decreased in the CUMS group. FMT was able to induce antidepressant effects on CUMS-induced depression involving a range of biochemical entities such as neurotransmitters, anti-inflammatory factors, neurotrophic factors and GLPs. Using an antibiotic cocktail, the gut microbiota of mice was depleted and then subjected to FMT colonization from normal mice or CUMS exposed mice (CUMS donors). Mice with CUMS and those with FMT from the CUMS donor exhibit higher anxiety- and depression-like behaviour when compared to the healthy controls (Li et al., 2019). Low abundance of gut Lactobacillus and increased richness of Akkermansia in the CUMS donor and recipient compared to control were observed. Hippocampal expression of Proinflammatory TNF-α and interferon-γ were upregulated in the CUMS mice group as against the control (Li et al., 2019). Dysbiosis impairs immunity and metabolic network which stimulate proinflammatory signalling pathways in the hippocampus via the GBA thereby inducing anxiety- and depression-like traits in mice.

Autism spectrum disorder (ASD) is a developmental complication due to discrepancies in the brain. ASD in some have a genetic component, whereas other causes are unknown. ASD subjects repeatedly exhibit anxiety, depression or attention-deficit

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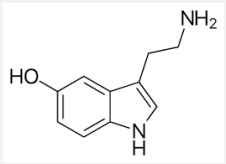
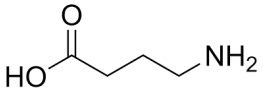
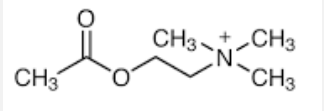
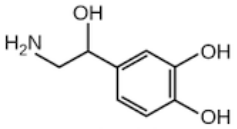
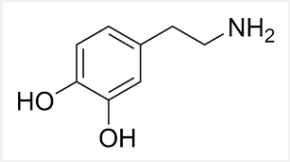
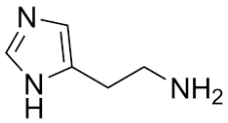
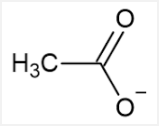
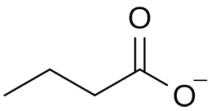
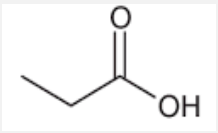
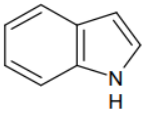
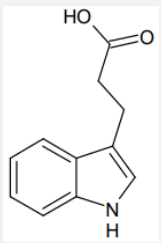
hyperactivity disorder (ADHD). In a clinical study by Kang et al, ASD children's fecal and plasma samples were analysed after undergoing FMT (Kang et al., 2020). ASD group metabolomic profiling revealed altered levels of plasma metabolites compared to the healthy children. FMT had a widespread effect in recipients, causing changes in circulating metabolites. Plasma metabolites (such as nicotinamide riboside, IMP, iminodiacetate, methyl succinate, galactonate, valyl glycine, sarcosine, and leucyl glycine) were decreased in the ASD group. (Kang et al., 2020). Metabolites like caprylate and heptanoate were however elevated in the ASD group. The plasma and fecal concentrations of these metabolites in the FMT recipients (with ASD) were modulated and attained levels typically observed in healthy children. Metabolic profiling study exhibited modulation of metabolites by the gut microbiome which is a key route to influence the GBA and could serve as biomarkers in treating ASD. By reestablishing gut microbial population through FMT from healthy subjects/animals to normal compositions ASD subjects can reverse various phenotypes associated with neurological impairments. It appears from several pre-clinical and clinical findings that FMT is a potent intervention for transmission and treatment of neurological complications.

Challenges and prospects

Over the years FMT has received encouraging support and popularity as a treatment intervention towards several GIT and nervous system disorders. Several pre-clinical and clinical findings show FMT as intervention for the treatment of intestinal and neurological disorders. At present, FMT is seen as a useful strategy in treating GIT and neurological disorders, especially those linked to the intestinal microbiome. However, clinical applicability of FMT is limited due to its practical dissent in treating diseases. A key hurdle is the isolation and characterization of key gut microbes responsible for the synthesis of host-modulating metabolites. Future studies on large randomized-controlled trials in a population are required to understand FMT in human diseases related to gut dysbiosis.

Table 1. Select gut microbial-derived metabolites secreted in the host intestine.

Metabolites	Chemical structure	Remarks	References
Serotonin		Regulates mood, memory, learning, cognition etc.	Boulangé et al, 2016; Collins et al, 2012
Gamma amino butyric acid (GABA)		Can reduce neuronal excitability by inhibiting nerve transmission. Can affect sleep.	Ananthakrishnan et a, 2018; Boulangé et al, 2016
Acetylcholine		Regulates attention, learning, arousal, involuntary muscle movement etc.	Canakis et al, 2020; De Vadder et al, 2014

Norepinephrine		A vasoconstrictor increases heart rate and cardiac output. Can function both as a hormone and a neurotransmitter.	Deidda & Biazzo, 2021; El Aidy et al, 2015
Dopamine		Regulates mood, attention, memory, movement etc.	El-Salhy et al, 2020; Gomaa, 2020
Histamine		Can modulate the gut immune function and intestinal inflammation.	Halkjær et al, 2018; Shapiro et al, 2014
Acetate		Directly interact with host nuclear histone deacetylase causing gene expression reprogramming (epigenetic modification). Stimulates ghrelin secretion and control sleep and appetite.	Vendrik et al, 2020; Sokol et al, 2020
Butyrate		Participates in epigenetic modification. Stimulates leptin secretion and control sleep and appetite.	Mayer et al, 2011; Lima et al, 2020
Propionate		Participates in epigenetic modification Stimulates leptin secretion and control sleep and appetite.	Lee et al, 2018; Kataoka, 2016
Indole		Weak aryl hydrocarbon receptor (AhR) agonist with anti-inflammatory and insulin sensitivity actions. Stimulate GLP-1 secretion.	Illiano et al, 2020; Holster et al, 2021
Indole 3-propionic acid (IPA)		Pregnane X receptor (PXR) agonist with anti-inflammatory effects and insulin sensitivity actions.	Haifer et al, 2022; El-Salhy et al, 2020

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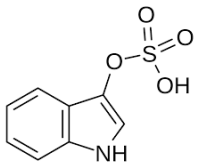
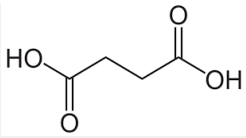
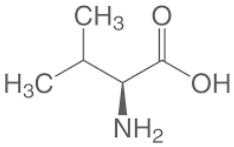
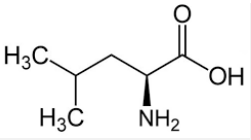
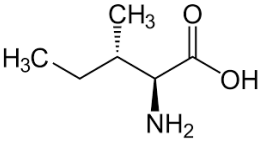
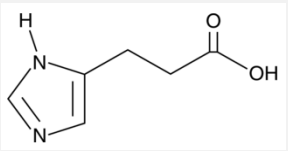
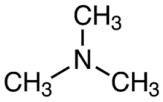
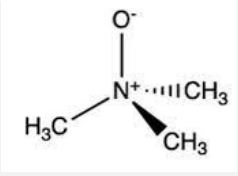
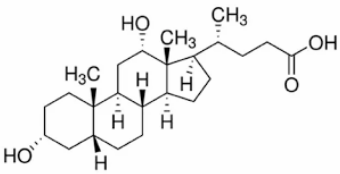
Indoxyl sulfate		Contribute towards anxiety, depression, kidney disease etc.	Dimidi et al, 2017; Collins et al, 2012
Succinate		Enhances insulin sensitivity and glucose tolerance	Browning et al, 2019; Myneedu et al, 2019
L-Valine		Roles in regulating protein, lipid and glucose metabolism, BAT thermogenesis & insulin resistance.	Nikolova et al, 2021; Pluznick et al, 2013
L-Leucine		Roles in regulating protein, lipid and glucose metabolism, BAT thermogenesis, insulin resistance.	Rhee et al, 2009; Strati et al, 2021
L-Isoleucine		Roles in regulating protein, lipid and glucose metabolism, BAT thermogenesis, insulin resistance.	Rhee et al, 2009; Strati et al, 2021
Imidazole propionate		Its level is significantly increased during T2D and is associated with insulin resistance.	Zhang et al, 2020; Zhao et al, 2021
Trimethylamine (TMA)		In hepatocytes it is metabolised by flavin monooxygenases 3 to yield TMA-N-oxide (TMAO).	Xie et al, 2021; Vendrik et al, 2020
TMA-N-oxide (TMAO)		Increased systemic TMAO level observed during diabetes and obesity.	Xie et al, 2021; Vendrik et al, 2020
Deoxycholic acid		Agonist for farnesoid X receptor (FXR) and Takeda G-protein coupled receptor (TGR5). Increases hepatic insulin sensitivity, insulin secretion from the pancreas.	Tian et al, 2020; Rossen et al, 2015

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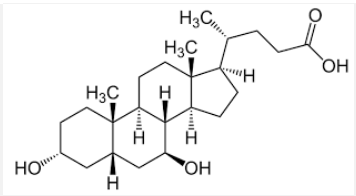
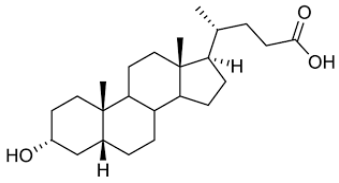
Ursodeoxycholic acid		Agonist of FXR and TGR5. Increases hepatic insulin sensitivity, insulin secretion from the pancreas.	Tian et al, 2020; Rossen et al, 2015
Lithocholic acid		Agonist of FXR and TGR5. Increases hepatic insulin sensitivity, insulin secretion from the pancreas.	Paramsothy et al, 2019; Mitrea et al, 2022

CONCLUSION

Research findings support GBA existence which can be modulated by the host signalling pathways, including the metabolites secreted by the gut microbiome. Gut dysbiosis can be overcome by the transfer of normal gut microbial population using FMT from healthy individuals to those who are dysbiotic. An altered gut microbiota can lead to various diseases of the GIT, including neuropsychiatric and neurological disorders. IBS, IBD and constipation are such GIT conditions that can be modulated by FMT from the healthy donors to the recipients. FMT increased the diversity and alters the composition of microbial population in the recipient fecal and colon samples. Differences across bacterial phyla are observed between the normal and those suffering from dysbiosis. Microbial metabolites are the major modulator of biological processes various tissues/organs, including the gut in the host. These metabolites have major effect in the host when their production is compromised due to dysbiosis and are key in IBS, IBD and constipation progression. Altered control of the microbiota-GBA due to gut dysbiosis could be the reasons for the onset and progression of some neurological disorders. Preliminary data obtained indicate that neurological disorders such as AD, PD, ASD and depression can be effectively treated by FMT, though some findings say the opposite. Neuroinflammation is a typical feature of most neurological disorders, and which gets elevated due to gut microflora compositional alteration. Dysbiotic gut microbiota impairs normal immune system and metabolic pathways which stimulate hippocampal proinflammatory signalling via the GBA thereby inducing depression and anxiety-like features in mice. Metabolic profiling study shows that metabolites level modulation by the gut microbiome can profoundly influence GBA and could serve as biomarkers in potential treatment strategy for ASD. In conclusion, several pre-clinical and clinical findings show FMT as potential therapeutic intervention for transmission and treatment of intestinal and neurological disorders. Future studies on large randomized-controlled trials in a population are required to understand FMT in human diseases related to gut dysbiosis.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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