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IRRITABLE BOWEL SYNDROME: THE ROLE OF GUT MICROBIOTA IN ETIOLOGY AND TREATMENT. A LITERATURE REVIEW

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ABSTRACT

Irritable bowel syndrome (IBS) is a common gastrointestinal disorder. Its etiology is thought to involve many factors. Recent reports suggest that gut microbiota disorders may play an important role in the pathophysiology of IBS. This opens the door to new treatment therapies.
Aim: The aim of this article is to review the latest research on the impact of changes in the gut microbiota on IBS, as well as methods of therapy that influence the modulation of the gut microbiota.
Material and methods: A literature review was conducted using the PubMed and Google Scholar databases. The focus was on studies published between 2015 and 2025.
Results: Changes in the gut microbiota play a significant role in IBS. However, there is still much uncertainty surrounding the exact pathomechanisms underlying this process. The low-FODMAP diet alleviates symptoms and causes changes in microbiota. Probiotics show moderate effectiveness, while the results of fecal microbiota transplantation are often inconclusive.
Conclusions: Further research is needed to determine the exact impact of gut microbiota on IBS. The low-FODMAP diet, probiotics, and fecal microbiota transplantation offer promising but inconclusive treatment results. Further standardized studies are needed to determine the best therapeutic strategies.

KEYWORDS

Gut Dysbiosis, Irritable Bowel Syndrome, Low FODMAP Diet, Gut Probiotics, Intestinal Microbiota Transplantation

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Introduction

Irritable bowel syndrome (IBS) is a chronic functional disorder of the small and large intestines that is associated with a reduced quality of life. It is estimated that approximately 11% of the world's population has IBS. (Dale et al., 2019) The main symptoms are recurrent abdominal pain and irregular bowel movements, which are associated with changes in stool form and frequency. (Rodiño et al., 2018) The pathophysiology of this syndrome is not fully understood, but it is currently believed to be multifactorial in nature. (Dale et al., 2019) Causes of IBS include disturbances in the brain-gut axis, intestinal motility disorders, visceral hypersensitivity, prior gastrointestinal infections, chronic microinflammation, and alterations in gut microbiota composition. (Hillestad et al., 2022) Because the pathophysiological cause of IBS is unclear, it is difficult to identify biomarkers and diagnostic tests that can confirm the diagnosis with certainty. The Rome IV criteria are currently used for diagnosis. There are four types of IBS based on predominant symptoms and stool appearance: constipation-predominant, diarrhea-predominant, mixed, and unsubtyped. (Rodiño et al., 2018) The unclear etiology of IBS is associated with an absence of effective treatment methods. (Dale et al., 2019) Therefore, a thorough understanding of the pathophysiological processes underlying the onset of IBS would enable the use of more effective treatments. (Rodiño et al., 2018)

Recently, more attention has been given to how intestinal microbiota disorders, known as dysbiosis, affect the development and continuation of IBS symptoms. (Raskov et al., 2016) It has been observed that some patients experience symptoms of IBS after suffering from a gastrointestinal infection. (Schwille-Kiuntke et al., 2015) Additionally, differences in gut microbiome composition were observed between IBS patients and healthy patients. (Sundin et al., 2020) It is assumed that this is related to the impact of dysbiosis and its metabolites on intestinal barrier dysfunction, inflammation, and disorders of the gut-brain axis (GBA). Changes in intestinal secretion and motility caused by intestinal dysbiosis and GBA disorders can lead to visceral hypersensitivity and cellular alterations in the enteroendocrine system. (Carabotti et al., 2015)

The link between dysbiosis and the development of IBS opens up a path for treatment targeting changes in the gut microbiota. The impact of probiotics, dietary interventions, and fecal microbiota transplantation on modulating the gut microbiota and improving the clinical condition of patients with IBS is currently under investigation. (Hillestad et al., 2022; Rodiño et al., 2018; Simon et al., 2021)

This article reviews the latest research on the impact of gut microbiota on IBS development and progression, as well as therapeutic methods that alter gut microbiota, including dietary interventions, probiotics, and fecal microbiota transplantation.

Material and Methods:

This article presents the results of a systematic review of articles from the PubMed and Google Scholar databases. The review focuses on the impact of intestinal dysbiosis on the development and symptoms of IBS, as well as methods of treating IBS that alter the microbiota. The review includes treatments such as the low FODMAP diet, probiotics, and fecal microbiota transplantation. We searched the PubMed and Google Scholar databases for articles published between 2015 and 2025. We also excluded articles that were not written in English. We used the following terms for our search: "gut dysbiosis," "irritable bowel syndrome," "microbiota-gut-brain axis," "low FODMAP diet," "gut probiotics," and "intestinal microbiota transplantation." This review was prepared using both original articles and review publications, as well as meta-analyses and case reports.

The impact of the microbiota on irritable bowel syndrome

The human digestive tract is home to a significant amount of bacteria, which is characterized by great diversity. It is estimated that there are approximately 10^{13} - 10^{14} microbes. The most numerous bacteria belong to the bacterial phylae Firmicutes, Actinobacteria, Bacteroides, and Proteobacteria. (Raskov et al., 2016) Under normal conditions, this microbiota is responsible for maintaining homeostasis in the body through numerous physiological processes. (Carabotti et al., 2015)

Patients with IBS show changes in their gut microbiome, consisting of a reduction in commensal bacteria and an increase in pathogenic bacteria. (Simon et al., 2021)

Dysbiosis may contribute to changes in intestinal barrier function, which can lead to the onset of IBS symptoms. (Yoo et al., 2020) Under normal conditions, the intestinal barrier performs defensive mechanisms, including protection against pathogenic bacteria, maintenance of the integrity of the epithelium, support of the transport of nutrients, and communication with commensal bacteria and substances found in the digestive tract. It is mainly composed of epithelial cells covered with a layer of mucus, lymphatic tissue, and the intestinal nervous system. (Chen et al., 2024) Intestinal dysbiosis can disrupt the function of the intestinal barrier, leading

to increased permeability, particularly to bacteria and their metabolites. This can lead to inflammation, visceral hypersensitivity, and pain. (Hillestad et al., 2022)

A number of physiological processes, including commensal bacteria, are responsible for maintaining the normal permeability of the intestinal barrier. People with IBS have been observed to have an increased intestinal barrier permeability. (Hanning et al., 2021) One of the factors that help maintain the proper structure of the intestinal barrier are short-chain fatty acids (SCFAs), which are produced by bacteria. Sodium butyrate, a bacterial metabolite, is an example of a SCFA. This compound has the ability to reduce intestinal permeability by increasing the expression of tight junction proteins. (Bhattarai et al., 2017) In a trial involving rats with an IBS model, enteral instillation of butyrate at physiological doses reduced visceral hypersensitivity and improved intestinal barrier function. (Nozu et al., 2019) Therefore, a reduction in butyrate levels may have an adverse effect on the intestinal barrier. This was confirmed by a trial involving reduced levels of butyrate-producing bacteria among people with IBS symptoms. (Pozuelo et al., 2015)

Other bacterial products that may influence the occurrence of IBS symptoms are proteases. People with IBS were found to have increased proteolytic activity (PA) compared to healthy patients. Furthermore, patients with elevated PA exhibited reduced microbial diversity and increased intestinal barrier permeability. (Edogawa et al., 2019) It is believed that proteases produced by dysbiotic bacteria cause this condition. They can affect the intestinal barrier and induce inflammation by activating protease-activated receptors (PARs). (Hillestad et al., 2022)

The etiopathogenesis of IBS increasingly points to the presence of inflammation of the intestinal mucosa. The exact role of immune system activation in IBS is not fully understood, but microinflammation involving mast cells, eosinophils, and intraepithelial lymphocytes has been found in people with the condition. Histopathological studies of individuals with IBS have found elevated levels of mast cells in particular. (Casado-Bedmar & Keita, 2020; Raskov et al., 2016) Activated mast cells were observed releasing inflammatory mediators, including histamine, serotonin, prostaglandins, and cytokines. It is known that mast cells interact with the gut microbiota and neurons through secreted mediators, which can lead to visceral hypersensitivity. (Bednarska et al., 2017; Casado-Bedmar & Keita, 2020) However, the exact mechanism of this process remains unclear.

Altered gut microbiota may also affect the expression of Toll-like receptors (TLRs). These receptors are present in immune system cells, intestinal epithelial cells, and intestinal neurons. TLRs recognize metabolites produced by pathogenic bacteria and induce an immune response. (Raskov et al., 2016) A study comparing TLR levels in people with IBS found increased TLR expression compared to the control group. An increase in TLR levels was correlated with higher levels of the pro-inflammatory cytokine IL-6 and lower levels of the anti-inflammatory cytokine IL-10 in patients with IBS. (Shukla et al., 2018)

It is assumed that communication between the gut microbiota and the GBA also plays an important role in the occurrence of IBS. The GBA is a complex system of bidirectional connections between the autonomic nervous system, central nervous system (CNS), enteric nervous system (ENS), and hypothalamic-pituitary-adrenal (HPA) axis. It is a complex network of neurohormonal connections that integrate and modulate the functioning of the CNS with the gastrointestinal tract, which is also influenced by the microbiota. (Carabotti et al., 2015) It is now known that the CNS influences the secretion of endocrine mediators in the intestinal tract. These mediators bind to bacterial receptors and interact with them directly, thereby regulating their quantity and activity. However, the CNS may also indirectly influence the microbiota by modulating the intestinal environment through the autonomic nervous system. (Osadchiy et al., 2018)

The GBA pathway is bidirectional. The autonomic nervous system also transmits afferent signals from the intestinal tract to the CNS. However, the main connection between the microbiota and the CNS is the vagus nerve, 80% of whose fibers are afferent fibers. (S. V. Singh et al., 2023)

The microbiota influences the GBA through various mechanisms. One of them is the modulation of afferent sensory fibers. A study on Lactobacillus reuteri showed that these bacteria increased sensory nerve excitability, thereby modulating intestinal motility. (Carabotti et al., 2015)

The ENS, often referred to as the "second brain," produces many neurotransmitters, including serotonin (5-HT), somatostatin, and dopamine. It also regulates the GBA. The gut microbiota can influence the ENS through the SCFAs it produces, particularly butyric acid, propionic acid, and acetic acid. This increases intestinal transit and affects serotonin secretion. (Raskov et al., 2016)

However, intestinal barrier dysfunction is considered to be particularly important in the impact of microbiota on GBA. In a study on mice, treatment with a mixture of Lactobacillus helveticus R0052 and

Bifidobacterium longum R0175 probiotics not only restored intestinal barrier function, but also reduced the activity of the HPA axis. (Ait-Belgnaoui et al., 2014)

It is known that altered gut microbiota affects intestinal permeability, induces inflammation, and causes changes in the GBA, thereby contributing to IBS. However, more research is needed on the pathomechanism of these processes because the exact mechanisms have not been fully elucidated.

The low-FODMAP diet and irritable bowel syndrome

An important therapeutic element for patients with irritable bowel syndrome is the low FODMAP (fermentable oligosaccharide, disaccharide, monosaccharide, and polyol) diet. (Bertin et al., 2024) These include substances such as lactose, fructose, fructo-oligosaccharides, galacto-oligosaccharides and polyols (sorbitol, mannitol, maltitol, isomalt, lactitol, and xylitol). (Jayasinghe et al., 2024)

This diet involves FODMAP restriction and then gradually reintroducing previously eliminated foods to assess tolerance. A modified FODMAP diet is then created based on individual tolerance. (Bertin et al., 2024; P. Singh et al., 2022)

There are several mechanisms by which FODMAPs may contribute to the symptoms of irritable bowel syndrome. FODMAPs are carbohydrates which, due to a slow transport mechanism and reduced enzymatic activity, are poorly absorbed in the small intestine and undergo rapid fermentation by the colon microbiota. (Barrett & Gibson, 2012; Reddel et al., 2019) This results in production of colonic gases, including hydrogen and methane, and consequently bloating, discomfort, and abdominal pain. (El-Salhy & Gundersen, 2015; Reddel et al., 2019)

In addition, these carbohydrates are osmotically active molecules, which increases the amount of water in the small intestine. These mechanisms can cause luminal distension and, as a result, cause IBS symptoms. (Reddel et al., 2019)

Another mechanism currently under research is the development of visceral hypersensitivity. Studies on rodents have shown a loss of colonic barrier function caused by a high-FODMAP diet. This mechanism was triggered by Toll-like receptor 4 (TLR4) – dependent mast cell activation (P. Singh et al., 2021) and an increase in the number of fecal Gram-negative bacteria and elevated lipopolysaccharides (LPS). (Zhou et al., 2017) It is suggested that FODMAPs cause an excessive number of Gram-negative bacteria, leading to increased luminal LPS. Then LPS activates mast cells via TLR4, releasing molecules like tryptase, histamine, and prostaglandin E2, increasing intestinal permeability and visceral sensitivity. (P. Singh et al., 2021) However, further research is needed to explain this process. (Bertin et al., 2024)

Other mechanisms through which FODMAPs exert their effects have been suggested. It is suspected that a low-FODMAPs diet causes changes in the gut microbiome (Halmos et al., 2015) and endocrine cells of the gastrointestinal tract. Endocrine cells respond to the contents of the intestinal lumen and release specific hormones into the lamina propria. In this way, they regulate motor function, absorption, secretion, local immune defense, and food consumption (appetite). It is considered that differences in endocrine cell density in patients with IBS influence the pathophysiology of this disease. However, it is suggested that the density of these cells normalizes after receiving dietary guidance. (Mazzawi et al., 2014, 2015) However, further research is needed. (El-Salhy & Gundersen, 2015)

The effect of a low-FODMAP diet on the human microbiota remains unclear. There are concerns because undesirable modulation of the gut microbiota may occur. The studies noted a decrease in the number of bifidobacteria and other changes similar to those observed in dysbiosis. (Vandeputte & Joossens, 2020) However, the mechanism is unclear and it is unknown whether the impact is long-term, so further research is needed. (Reddel et al., 2019)

There is evidence that a low-FODMAP diet reduces symptoms in people with irritable bowel syndrome and improves their quality of life. However, further investigation is needed to clarify the mechanisms of action and long-term effects on nutritional status and the gut microbiome. (Altobelli et al., 2017; van Lanen et al., 2021)

Probiotics, prebiotics, and synbiotics in irritable bowel syndrome

An approach focused on the gut microbiota to alleviate IBS symptoms involves the use of probiotics, prebiotics and synbiotics. Probiotics are live microorganisms that have a beneficial effect on the health of the host. Prebiotics are substrates that promote the growth of beneficial microorganisms in the patient's digestive tract. Prebiotics are substrates that promote the growth of beneficial microorganisms in the patient's gastrointestinal tract. Synbiotics combine both of these groups. There are two types of synbiotics. Complementary synbiotics affect the patient's microbiota, while in synergistic synbiotics, prebiotics serve as

a substrate for the administered probiotics. (Chlebicz-Wójcik & Śliżewska, 2021; Herndon et al., 2019; Swanson et al., 2020)

Most research on alleviating IBS symptoms concerns probiotics. They are considered beneficial in the treatment of IBS, but further research is needed. (Simon et al., 2021; Y. Zhang et al., 2016) Some studies suggest an improvement in quality of life and overall IBS symptoms, but not specific IBS symptoms such as abdominal pain and bloating. (Sun et al., 2018; Y. Zhang et al., 2016)

Probiotics may contain a single strain or a mixture of several strains. The most common species that are the subject of research in the context of IBS are Bifidobacterium, Lactobacillus, Streptococcus, and Enterococcus. (Horvat et al., 2021)

Probiotics exhibit several mechanisms of action that influence the pathophysiology of IBS. One of these mechanisms is their effect on the gut microbiome. Probiotic strains have the ability to disrupt the adhesion process of pathogenic bacteria. This process may occur through the induction of changes in intestinal mucins, steric hindrance at the level of the pathogen's intestinal receptors, or competitive exclusion of mucosal adhesion sites. (González-Rodríguez et al., 2012; Simon et al., 2021)

In addition, probiotics produce various substances that affect pathogenic strains. These include bacteriocins, which are cationic molecules that mainly act on the cytoplasmic membrane. (Umu et al., 2017) They create pores in it, damaging the bacteria. In addition, they inhibit cell wall synthesis. (Plaza-Diaz et al., 2019)

Other antimicrobial substances include short-chain fatty acids (SCFA) such as lactic acid, propionic acid, butyric acid, and acetic acid. They are produced as a result of carbohydrate metabolism by probiotics. They inhibit the growth of pathogenic strains by lowering the overall pH of the intestine. (Simon et al., 2021)

Another mechanism of action of probiotics is their effect on the intestinal barrier. An important element is the mucus layer and epithelial tight junctions. When this barrier is disrupted, bacteria can penetrate the submucosa. (Simon et al., 2021) Probiotics demonstrated a positive effect on mucus production. Previous studies have shown an increase in mucin expression. (Dudík et al., 2020) The intestinal barrier is also strengthened by reducing intestinal permeability and improving the integrity of the tight junctions between intestinal epithelial cells. (Simon et al., 2021)

In addition, it has been shown that some probiotics can stimulate the production of antimicrobial peptides, including defensins and cathelicidins. They have a broad spectrum of antimicrobial activity, but the mechanism is not fully understood. Some studies confirm an increase in beta-defensin-2 after the use of probiotics. However, further research is needed to better understand these mechanisms. (Simon et al., 2021)

Another mechanism of action of probiotics is the modulation of the immune response. The immune system of the gastrointestinal tract consists of various lymphoid and non-lymphoid cells, along with a humoral response. IgA plays an important role, whose secretion into the gastrointestinal tract depends on dendritic cells that present antigens, T cell activation, and ultimately in gut-associated lymphoid tissues (GALT) and mesenteric lymph nodes. (Yadav et al., 2022)

Probiotics interact with dendritic cells, epithelial cells, monocytes, and lymphocytes. These mechanisms are complex and require further study. (Plaza-Diaz et al., 2019)

Probiotics also stimulate the production of sIgA, which transports IgA dimers to the surface of epithelial cells. Probiotics also stimulate the production of sIgA, which transports IgA dimers to the luminal surfaces of epithelial cells (Plaza-Diaz et al., 2019; H. Wang et al., 2016) and induce the development of T-reg lymphocytes. (Giorgetti et al., 2015)

Probiotics also exhibit anti-inflammatory properties, suppressing pro-inflammatory responses by increasing concentrations of IL-10 and Th1 cytokines. Probiotics may also participate in the regeneration of the epithelial barrier by upregulating TNF-α. (Giorgetti et al., 2015; Plaza-Diaz et al., 2019)

In addition, probiotics have an effect on pattern recognition molecules or Toll-like receptors (TLRs), such as TLR-2 or TLR-4. This mechanism is based on the effect on epithelial cells and results in the production of cytokines that prevent apoptosis of epithelial cells. (Simon et al., 2021) They also promote the activation of dendritic cells, which influence the immune response. (Yadav et al., 2022)

Prebiotics are generally short-chain carbohydrates that are not digested but are used as substrates for the growth of microorganisms. These include various substances such as oligofructose, inulin, galactooligosaccharides, lactulose, and oligosaccharides from breast milk. (Yadav et al., 2022)

The importance of probiotics for intestinal microflora has been proven. However, there is not much research on the role of prebiotics and synbiotics. (Plaza-Diaz et al., 2019)

However, further research is needed to understand the exact mechanisms of action of probiotics, prebiotics, and synbiotics, as well as their appropriate use in IBS therapy. (Horvat et al., 2021)

Fecal microbiota transplantation and irritable bowel syndrome

Fecal microbiota transplantation (FMT) involves transfering stool samples from healthy donor and then transplanting them to a patient using endoscopy or capsules. This leads to a reconstruction in the imbalanced gut microbiota associated with IBS. This method can be used to treat other intestinal diseases, such as recurrent Clostridioides difficile infections (rCDI), inflammatory bowel disease (IBD), short bowel syndrome (SBS), constipation. However, further research into the use of FMT in these diseases is necessary, as the results are inconclusive and the effectiveness of this method has been demonstrated for rCDI. (Baunwall et al., 2020; Halkjær et al., 2023; Hou et al., 2025)

Various roles of FMT in the regulation of intestinal microflora have been demonstrated. A reduction in the number of pathogenic bacteria such as C. difficile and E. coli was observed, resulting in a decrease in inflammation. In addition, there is an increased growth of beneficial bacteria such as Lactobacillus and Bifidobacterium. Another effect of FMT is the destruction of pathogenic bacterial biofilms, which further improves the effectiveness of other treatments, including antibiotics. (Hou et al., 2025; Wen et al., 2021)

The exact effect of FMT on IBS remains unclear, but it is also suggested to have an impact on enteroendocrine cells and short-chain fatty acids (SCFA). Butyrate (SCFA) modulates the immune response, alleviates oxidative stress, reduces intestinal motility and intestinal cell permeability, and provides energy to colonic epithelial cells. (El-Salhy, Casen, et al., 2021; El-Salhy, Patcharatrakul, et al., 2021) In addition, it also regulates colon hypersensitivity, and its use has been associated with a reduction in abdominal pain in patients with IBS. It has been suggested that FMT increases butyrate levels in the stool of IBS patients, which may be related to the improvement observed after FMT. (Dai et al., 2024; J. Zhang et al., 2018)

In addition, an increase in fecal SCFAs increases the secretion of Peptide YY (PYY). This peptide binds to Y1 and Y2 receptors in intestinal epithelial cells and in neurons of the submucosal plexus and smooth muscle of the small intestine and colon, activating them. (El-Salhy et al., 2021; El-Salhy et al., 2020) However, in patients with irritable bowel syndrome, the density of PYY cells in the colon is reduced. It is suggested that the effect of SCFA on the increase in PYY secretion that occurs after fecal microbiota transplantation may contribute to the improvement of IBS symptoms. (El-Salhy et al., 2020)

Additionally, it is suggested that FMT and, as a result, changes in the intestinal bacterial composition may affect the serotonin regulatory system. The majority of serotonin is found in the gastrointestinal tract. Most of it is contained in enterochromaffin (EC) cells scattered among the epithelial cells of the gastrointestinal lumen and the rest in the enteric nervous system. It has a significant effect on gastrointestinal motility. (Dai et al., 2024) It has been discovered that people with IBS have impaired serotonin uptake and degradation, as well as lower EC cell density. However, some bacteria affect the serotonin regulatory system. Corynebacterium, Enterococcus spp, and Streptococcus are serotonin producers, while Clostridium ramosum regulates serotonin release from EC cells. Therefore, changes in bacterial composition after FMT may affect serotonin and thus IBS symptoms. (El-Salhy et al., 2021; Mishima & Ishihara, 2020)

Fecal microbiota transplantation appears to be a safe treatment for IBS. Possible side effects were reported as mild abdominal pain, cramps, tenderness, diarrhea, and constipation, which resolved spontaneously and occurred within the first few days after transplantation. (S. Wang et al., 2016)

However, the effectiveness of FMT in treating IBS has not been conclusively confirmed, and further research is needed. The procedure for the possible use of FMT, including the selection of donors and patients, as well as the administration route and dose of transplantation, also needs to be established. (Holster et al., 2019; Hou et al., 2025)

Discussion

There is no doubt that the therapy of irritable bowel syndrome is currently quite a challenge. Due to the prevalence of this disorder in the population and the reported reduction in quality of life among patients. (Dale et al., 2019) Therefore, it is extremely important to determine the exact cause of irritable bowel syndrome. An analysis of available studies indicates that IBS is influenced by many factors, including intestinal dysbiosis, which plays an important role.

With more research, our understanding of how much impact the microbiota has on our health is also growing. In recent years, intestinal microbiota disorders have been linked not only to diseases of the digestive tract, but also to neurodegenerative, cardiovascular, and metabolic diseases. (Chen et al., 2021) This shows how important the interactions between the human body and altered gut microbiota are.

Most studies confirm changes in the gut microbiota in people with IBS. A characteristic feature is the imbalance between commensal and pathogenic bacteria. A decrease in the amount of Lactobacilli and

Bifidobacteria was also observed. (Bhattarai et al., 2017; Raskov et al., 2016; Sciavilla et al., 2021) However, not all studies have yielded the same results in terms of changes in individual bacterial groups. (Jalanka et al., 2015; Tap et al., 2017) This may be related to the diversity of symptoms and types of IBS, as well as the methods used to conduct tests.

Many studies have also observed a link between intestinal dysbiosis and intestinal barrier dysfunction. (Bhattarai et al., 2017; Edogawa et al., 2019; Hanning et al., 2021) Various causes of these disorders have been identified, including reduced SCFA production and the influence of proteases. (Bhattarai et al., 2017; Hillestad et al., 2022) This may indicate the complexity of the processes affecting intestinal barrier permeability. However, the final conclusion in most studies was consistent that bacterial dysbiosis and its metabolites affect the occurrence of intestinal barrier disorders.

The available literature also indicates that intestinal dysbiosis is one of the key factors modulating the action of GBA. The influence of substances produced by bacteria, such as serotonin and SCFAs, is emphasized here, (Dai et al., 2024) as these elements directly affect the functioning of the GBA. The activity of the microbiota can also influence the modulation of afferent sensory fibers (Carabotti et al., 2015) and regulate the immune system through, among other things, TLR. (Raskov et al., 2016) This shows how complex the impact of microbiota is on GBA function and how many relationships have yet to be discovered.

A low-FODMAP diet is one of the most effective dietary interventions for treating IBS symptoms. In most cases, it reduces the severity of abdominal pain, bloating, and irregular bowel movements. (Bertin et al., 2024) Although following this diet alleviates the symptoms of IBS, it may potentially have an adverse effect on the gut microbiota. Studies have shown that following a low-FODMAP diet leads to a reduction in bifidobacteria and the occurrence of dysbiosis. (Halmos et al., 2015; Reddel et al., 2019) However, it is not known whether this diet affects the microbiota in the long term, as there are currently few long-term patient observations. (Reddel et al., 2019) Research results suggest that a low-FODMAP diet should be used on an individual basis and for as short a period as possible. However, further long-term studies are needed to determine the impact and changes of a low-FODMAP diet on the gut microbiota.

The results of probiotic therapy in patients with IBS are not always clear-cut. Some studies observe an improvement in patients' quality of life. (Sun et al., 2018; Y. Zhang et al., 2016) In other studies, improvement was not observed in all patients with IBS, only in those with constipation-related IBS. (Spiller et al., 2015) This raises the question of whether probiotics should be used in every patient with IBS. Should probiotics be selected individually, after adjustment to individual intestinal microbiota disorders?

FMT is a promising treatment for people with IBS. It has a beneficial effect on the diversity of the gut microbiota. (El-Salhy et al., 2021) However, despite improvements in the microbiota, research findings on the alleviation of IBS symptoms are not always consistent. (El-Salhy et al., 2019; Halkjær et al., 2018; Hou et al., 2025) These differences may result from the diverse characteristics of donors, research methodologies, and IBS subtypes. There is a need for broader randomized studies to determine not only the effectiveness of this method, but also the exact inclusion criteria, the dose used, and the donor profile.

Although knowledge about the impact of gut microbiota on the development and symptoms of IBS is constantly expanding, there are still many unknowns. A better understanding of these processes will certainly lead to more effective treatment. However, due to the multifactorial nature of IBS, the future of treatment seems to lie in individualized therapy tailored to each patient.

Conclusions

Intestinal dysbiosis has a significant impact on the development of irritable bowel syndrome, as evidenced by the results presented in this review. However, there is still much uncertainty in the exact pathomechanisms underlying this process. Research results are not always conclusive. (Aziz et al., 2021; Bhattarai et al., 2017)

Therefore, further research is needed in this area to better understand the connections between the microbiota and the host. At this stage, however, it can be concluded that the etiology of IBS is multifactorial and its course is influenced by many aspects, which is why single treatment methods are not effective. New research provides opportunities for the use of new therapies.

Personalized therapy can be beneficial in the treatment of IBS. Adapted to specific symptoms and disorders, including those related to the gut microbiota.

One of the important elements in the treatment of IBS is nutritional intervention in the form of a low-FODMAP diet. This diet has been shown to reduce symptoms such as abdominal pain and bloating. However, the exact mechanism of action remains unclear. The long-term impact on the gut microbiota is also unknown.

Research is also needed to develop safe use of the low-FODMAP diet and to assess the sustainability of its effects.

A rising number of studies confirm the effectiveness of probiotics in alleviating the symptoms of IBS. In the case of prebiotics and synbiotics, there are not many studies that describe this issue. However, it is necessary to develop standardized protocols and evaluate individual preparations and their impact on the microbiota. Possible side effects should also be assessed.

Another described method of treating irritable bowel syndrome is fecal microbiota transplantation (FMT). The method appears to be relatively safe and associated with mild side effects. However, the results regarding the effectiveness of IBS treatment are inconclusive, and further research is needed. The procedure for the possible use of FMT should also be clarified, including the criteria for selecting donors and recipients, as well as the route and dose of transplantation.

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