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REVIEW

The Role of Colonic Microbiota in Constipation Predominant Irritable Bowel Syndrome: A Literature Review

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Abstract

Irritable Bowel Syndrome with Constipation (IBS-C) is a functional gastrointestinal disorder characterised by abdominal pain, bloating, and altered bowel habits. Emerging research highlights the role of colonic microbiota in its pathophysiology, with IBS-C patients often exhibiting dysbiosis marked by reduced beneficial bacteria (*Bifidobacterium*, *Lactobacillus*) and increased inflammatory microbes (Enterobacteriaceae, *Escherichia coli*). Dysbiosis in IBS-C leads to reduced short-chain fatty acid production, impaired gut motility, and altered serotonin signalling, affecting peristalsis and sensitivity. It also increases intestinal permeability, inflammation, and gut-brain axis interactions, worsening pain and gastrointestinal dysfunction. These alterations impact gut motility, serotonin metabolism, and gut-brain axis signalling, contributing to IBS-C symptoms. Despite growing evidence, inconsistencies in study findings highlight the need for standardised research methods. Future studies should focus on long-term microbiota dynamics, targeted therapies, and personalised treatment strategies to improve symptom management and clinical outcomes. This review systematically summarises the changes in gut microbiota connected to IBS-C and provides a reference for future personalised treatment strategies.

Key words: irritable bowel syndrome; gastrointestinal microbiome; constipation; dysbiosis; brain-gut axis

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Introduction

Irritable Bowel Syndrome (IBS) is a gastrointestinal disorder with symptoms including abdominal pain, bloating, changes in stool frequency and consistency. It affects between 5%–20% of people worldwide and follows a pattern of recurrence and remission (NICE, 2023). The exact cause of Irritable Bowel Syndrome with Constipation (IBS-C) remains unclear, but one of the proposed causes is changes in colonic microbiota. Gut microbiota are microorganisms, which include bacteria, fungi, viruses and protozoa that exist in the gastrointestinal tract (Jandhyala et al, 2015). Alteration in the composition of colonic microbiota is known as dysbiosis and occurs in gastrointestinal conditions such as IBS (Carías Domínguez et al, 2025). Recent evidence suggests that patients with IBS-C exhibit different microbial profiles compared to healthy individuals. IBS can be categorised by the Rome

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criteria. This is a diagnostic guideline used to classify functional gastrointestinal disorders. IBS, as defined by the Rome IV criteria, is a functional bowel disorder characterised by recurrent abdominal pain that occurs in relation to defecation or is accompanied by changes in bowel habits. The Rome criteria further subclassify IBS with predominant constipation as IBS-C (Lacy and Patel, 2017).

This research aims to systematically assess the characteristics of gut microbiota in IBS-C patients and its impact on disease mechanisms. This review aims to enhance the understanding of IBS-C pathophysiology, aiding diagnosis and guiding targeted microbiota-based therapies.

Methods

To investigate the connection between colonic microbiota and constipation-predominant Irritable Bowel Syndrome, various information sources were utilised. Websites, research papers, review articles, and reports were identified through a combined approach using Google search and academic databases like the National Centre for Biotechnology Information (NCBI) (https://www.ncbi.nlm.nih.gov), PubMed (https://pubmed.ncbi.nlm.nih.gov) and Google Scholar (https://scholar.google.com). The search used keywords such as "Irritable Bowel Syndrome", "Gastrointestinal Microbiome", "constipation", "dysbiosis", and "Brain-Gut Axis" to ensure comprehensive coverage of relevant literature on colonic microbiota and constipation-predominant Irritable Bowel Syndrome, as well as the efficacy and safety considerations.

The literature was selected based on a set of criteria for inclusion, which required one or more of the specified keywords, relevance to the research question, published in English and a preference for meta-analysis and controlled trials. Many articles were excluded for not meeting these standards.

Each article was carefully reviewed with a focus on thoroughly reading and comprehending the main concepts. Key details—such as the author, year of publication, and significant findings—were systematically extracted and organised. The collected information was then analysed to uncover recurring themes, highlight gaps in the existing literature, and explore the implications, limitations, and possible avenues for future research.

Symptoms and Clinical Features of IBS-C

IBS is diagnosed using the Rome IV criteria, which require that patients experience recurrent abdominal pain at least one day per week over the past three months, along with two or more of the following: pain related to defecation, a change in stool frequency, or a change in stool form. The requirements must be fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis (Yang et al, 2022).

The Bristol Stool Scale can be used to classify IBS-C, which states that more than 25% of bowel movements must be 1 or 2, and less than 25% must be 6 or 7 on the Bristol Stool Scale (Chey et al, 2015).

The primary symptoms patients with IBS-C experience are abdominal pain with constipation, straining while trying to pass stools and incomplete bowel movements (Moshiree et al, 2024). Secondary symptoms include depression and anxiety (Zhang et al, 2018).

Pathophysiology: Gut Motility, Visceral Hypersensitivity

The cause of IBS is not fully known, but studies suggest it is multifactorial with etiological factors including increased epithelial permeability, dysbiosis, inflammation, visceral hypersensitivity, genetics and altered brain-gut interactions (Enck et al, 2016).

Gut Motility

Abnormal gut motility has been seen in a few patients presenting with IBS, patients with IBS-C may have an increased frequency and irregularity of luminal contractions along with prolonged transit time (Soares, 2014).

Visceral Hypersensitivity

Visceral hypersensitivity is defined as an altered sensation in response to physiological stimuli (Farzaei et al, 2016). It's regarded as one of the main factors in abdominal pain in patients with IBS (Farzaei et al, 2016). Visceral hypersensitivity is made up of two components, which are allodynia and hyperalgesia. The cause of visceral hypersensitivity is linked to multifactorial processes that occur both in the peripheral nervous system and the central nervous system (CNS) (Farzaei et al, 2016).

One of the factors contributing to visceral hypersensitivity is the dysfunction of the intestinal barrier (Awad et al, 2023). Barrier dysfunction and visceral hypersensitivity can occur due to the activation and movement of mast cells into the epithelial cells or sensory nerves in the intestinal mucosa, this leads to a reaction between the tissues and the immune cells releasing mediators such as prostaglandin E2, histamine, tryptase and cytokines (Hasler et al, 2022).

External Factors

Gut microbiota can be influenced by stress, diet and antibiotics (Bhattarai et al, 2017). Studies have shown psychological stress can impact intestinal sensitivity, motility, secretion and permeability. Stress-induced changes in the neuroendocrine immune pathways act on the gut-brain axis and may cause exaggeration of IBS symptoms (Qin et al, 2014). Diet can influence the composition of gut bacteria (Hillestad et al, 2022). Studies have shown diets low in fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) can alleviate symptoms of IBS (Zhang et al, 2018). FODMAP is a group of short-chain fermentable carbohydrates that are poorly absorbed (Cozma-Petruţ et al, 2017). They are unable to be absorbed by the colon, as they increase luminal water volume and lead to gas production, which can result in luminal distension and gastrointestinal (GI) symptoms in patients with IBS (Cozma-Petruţ et al, 2017). Antibiotics can also

play a role in the development of IBS, they can lead to a loss of diversity and a shift in the composition of the gut microbiome (Mamieva et al, 2022).

Healthy Gut Microbiota Composition and Function

Bacteroidota (formerly Bacteroidetes) and Bacillota (formerly Firmicutes) make up the majority in creating a "healthy" gut microbiota (Jandhyala et al, 2015). Other phyla, including Actinobacteriota, Pseudomonadota (formerly Proteobacteria), and Verrucomicrobiota, also contribute to gut health, although their distribution varies depending on location and the individual (Jandhyala et al, 2015). The functions of the gut microbiota include aiding digestion of proteins, carbohydrates and lipids (Macfarlane and Macfarlane, 2003; Rowland et al, 2018), synthesizing essential vitamins such as vitamin K and select B vitamins, and regulating the immune system (Wang et al, 2023).

Dysbiosis and Its Link to Gastrointestinal Disorders

Dysbiosis is a condition characterised by alterations in gut microbiota as a result of external and internal host factors (Carías Domínguez et al, 2025). It can be influenced by genetic factors, lifestyle choices, diet and usage of certain medications. It is a clinical condition associated with numerous gastrointestinal diseases (Chang and Lin, 2016).

Dysbiosis can be categorised into three types, which may occur simultaneously; these include loss of beneficial microbes, excessive growth of potentially harmful microorganisms and loss of microbial diversity (Petersen and Round, 2014). A reduction in diversity and an increase of facultative anaerobes, such as Enterobacteriaceae, are common alterations that contribute to uncontrolled local and systemic inflammation (Yoo et al, 2020).

Research indicates that individuals with IBS commonly exhibit alterations in their gut microbiota, including an increase in *Streptococcus spp.* (Firmicutes), a decrease in *Lactobacillus spp.*, and a reduction in *Bacteroidetes*. These changes reflect a decline in beneficial bacteria and a rise in pathogenic ones (Raskov et al, 2016).

Several studies have explored microbial differences across IBS subtypes. Su et al (2023) studied 942 IBS patients, including those with IBS-D (diarrhea), IBS-C (constipation), and IBS-U (unclassified), alongside 942 non-IBS controls, finding significantly lower bacterial diversity in individuals with IBS-D and IBS-U (Macfarlane and Macfarlane, 2003).

Mechanisms Linking Microbiota to IBS-C Symptoms

The microbiome-gut-brain axis is the bidirectional neurohumoral connection between the microbiota and the autonomic nervous system (Raskov et al, 2016).

By regulating intestinal transit, secretion, and regional gut motility, the brain can modify the composition and activity of the gut microbiota through the autonomic nervous system (Martin et al, 2018). However, the gut microbiota mostly uses neuroimmune and neuroendocrine pathways, including the vagus nerve, to communicate with the central nervous system. Furthermore, the microbiota has the ability to either manufacture or aid in the creation of certain neuroactive chemicals, including dopamine, serotonin, and noradrenaline (Martin et al, 2018).

The brain, through the hypothalamic pituitary axis and the autonomic nervous system, can influence the microbiota by mucosal immune function and epithelial permeability (Napolitano et al, 2023). Dysregulation of this communication is a critical part of the multifactorial pathogenesis of IBS-C. One factor in this dysregulation is alterations in tryptophan metabolism by increased activity of indoleamine-2,3-dioxygenase (IDO) (shifting synthesis toward the kynurenine pathway), which can lead to a potential serotonergic deficiency and possible enteric nervous system (ENS) and CNS dysfunction, as tryptophan is a precursor to Serotonin (5-HT), which is a key signalling molecule in the gut brain axis (Mayer et al, 2014).

Furthermore, impaired intestinal transit caused by compromised migrating motor complexes (MMC) is associated with bacterial overgrowth in the small intestine (Napolitano et al, 2023). Colonic levels of 5-HT are frequently reduced in IBS-C (Su et al, 2023). Although there is conflicting research on the specific changes in tryptophan metabolites, given the role of 5-HT in the healthy function of MMC, it can be speculated that this altered tryptophan metabolism may contribute to decreased stool frequency and increased Bacterial fermentation.

It's unclear how the dysbiosis of the microbiota in IBS-C directly causes the irregularities of enzyme activity. Some probiotic strains of Actinobacteria (*Bifidobacterium Longum*) are important for the mediation of tryptophan metabolism (Su et al, 2023). A research study analysed 62 patients with IBS-D, IBS-C and IBS-M (mixed) through qPCR fecal samples. In the constipation predominant patients, significantly reduced levels of *Actinobacteria* and *Bacteroidetes* were found ($p < 0.01$) (O'Mahony et al, 2005). Some probiotic strains of *Bifidobacteria* (*B. infantis*) have been shown to exert anti-inflammatory effects and inhibit pro-inflammatory cytokines in IBS patients (Chassard et al, 2012). A deficiency of these bacteria may contribute to visceral hypersensitivity (VH). However, their exact mechanism of alleviating pain in IBS-C has not been confirmed and requires further investigation.

Changes in Microbiota in IBS-C

Over the last 30 years, there have been many articles researching the correlation between IBS and constipation, a study which analysed bacterial DNA through stool samples and concluded that IBS-C patients carried increased amounts of *Veillonella spp.*, whereas there was no indication of *Helicobacter spp.* or *Clostridium difficile* in the stool samples (Malinen et al, 2005). Another study concluded that IBS-C patients had a marked increase in *Bacteroides spp.* and Enterobacteriaceae populations (Raskov et al, 2016). Similarly, Chassard et al (2012) found that the butyrate-producing *Roseburia-Eubacterium Rectal* group was less common ($p < 0.01$) in

IBS-C than in control ($p < 0.05$), and the number of lactate and H2-using sulphate-reducing populations was 10 to 100 times higher in IBS-C compared to healthy people. It has also been shown that levels of *Bifidobacteria*, *Clostridium leptum*, and *Faecalibacterium prausnitzii* are notably reduced in these patients. This further supports the correlation between colonic microbiota and IBS (Gobert et al, 2016).

Human microbiota-associated rats (HMAR) and models of experimental colitis produced by dextran sulfate sodium (DSS) have also been used to demonstrate persistent dysbiosis in IBS-C patients. Analysis of the microbiota's composition showed that *Bacteroides*, *Roseburia-Eubacterium Rectale*, and *Bifidobacterium* had decreased in relative abundance. In contrast to healthy controls, patients with IBS-C had higher levels of Enterobacteriaceae, *Desulfovibrio spp.*, and especially *Akkermansia muciniphila* (Pittayanon et al, 2019).

The studies reviewed highlight a significant correlation between colonic microbiota alterations and IBS-C. Consistent patterns of dysbiosis were observed, characterised by an increase in potentially pro-inflammatory bacteria, alongside a marked reduction in beneficial butyrate-producing and anti-inflammatory microbes. The elevated presence of lactate and H_2-utilising, sulfate-reducing populations further suggests a shift towards a more inflammatory gut environment in IBS-C patients. These microbial imbalances may contribute to the pathophysiology of IBS-C, influencing gut motility, inflammation, and symptom severity, thereby reinforcing the crucial role of the gut microbiota in the development and management of this condition.

The gut microbiota plays a crucial role in physiological functions such as immune regulation. This occurs by influencing neutrophil migration and the differentiation of T-helper cells and T-regulatory cells, which are essential for maintaining immune homeostasis. This process can be complicated by dysbiosis, or an imbalance in the microbiota, which could lead to autoimmune illnesses by reducing the immune system's capacity to discriminate between self and non-self, such as rheumatoid arthritis, multiple sclerosis, type 1 diabetes, and inflammatory bowel disease (IBD). Microbial metabolites, including short-chain fatty acids (SCFAs), lipopolysaccharides (LPS) and peptidoglycans, play crucial roles in modulating immune responses, either promoting inflammation or, in some cases, aiding tissue repair. SCFAs, particularly, help enhance Treg function and reduce pro-inflammatory cytokine production, maintaining immune tolerance.

However, when dysbiosis occurs, an excess of inflammatory molecules like LPS can lead to chronic inflammation and tissue damage, contributing to conditions like obesity, cardiovascular disease, and metabolic syndrome. Overall, the gut microbiota is integral to immune regulation, and its imbalance can lead to immune dysregulation and the development of chronic diseases, highlighting the importance of maintaining a balanced microbiome for optimal immune function (Hou et al, 2022).

Genetic and environmental factors are also said to affect gastrointestinal function and symptom development. Genetic variations in processes related to sensation, motility, and stress responses may contribute to IBS susceptibility. Different genetic factors might be responsible for diarrhea or constipation predominant IBS,

with additional variants linked to abdominal pain, which is not typically seen in non-painful disorders like functional constipation.

These genetic factors, combined with environmental influences such as diet, infections, and stress, help explain the varying symptoms and severity of IBS.

Limitations of Current Research

While numerous studies have shown significant changes in colonic microbiota in Constipation Predominant Irritable Bowel Syndrome (IBS-C) as mentioned above, some research has challenged this perspective. A number of studies have shown inconsistencies or minimal differences in the microbiota of individuals with constipation-predominant disorders.

For example, a research study conducted to examine the variations in colonic microbiota among patients with IBS-C, IBS-M, and healthy controls revealed that the microbiomes lacked significant diversity across all groups (Gryaznova et al, 2024).

Much of the data on gut microbiota in IBS comes from cross-sectional studies showing differences in the composition of microbiomes between IBS patients and healthy controls. In this respect, a systematic review by Pittayanon et al (2019) analysed 24 studies comparing gut microbiota in IBS and healthy individuals using various methods. While many studies indicate some range of dysbiosis in IBS, findings showed significant variability and a lack of consistent results (Ma et al, 2022; Pittayanon et al, 2019).

Moreover, studies conducted by Gobert et al (2016) showed that the microbiota of patients with IBS-C promoted protection from DSS-induced colitis. This highlighted its potential anti-inflammatory properties during experimental colitis. This finding is particularly interesting, since histological studies have previously identified microscopic inflammation in the colons of IBS patients, suggesting a complex and context-dependent role of the microbiota in modulating intestinal inflammation (Gobert et al, 2016).

These findings indicate the need for standardised methodologies for better explanation of the association of colonic microbiota changes with constipation-predominant disorders compared to healthy individuals. Such inconsistency may mean that the role of gut microbiota in IBS-C is less significant than previously estimated and, therefore, requires further research.

Although these findings do not refute the hypothesis entirely, they do point to the complexity of the gut-microbiota relationship and again highlight the need for extensive research with cautious interpretation of the current evidence.

Conclusion

This review highlights the role of colonic microbiota alterations in IBS-C, in particular the imbalance between beneficial and pro-inflammatory bacteria, bridging a gap in IBS research by focusing on microbial changes associated specifically with IBS-C.

Clinically, the findings in this review propose that microbiota profiling could assist in the diagnosis and treatment of this condition, especially with a personalised approach such as the use of probiotics or dietary interventions, alleviating symptoms.

Future research should incorporate clinical trials focusing on specific microbial populations, such as *Bifidobacterium longum* or *Faecalibacterium prausnitzii*. It is crucial that long-term studies are conducted to assess the changes in the gut microbiota in order to investigate the relationship between microbiotic dynamics and clinical trajectory. Integrating diverse analytical approaches could strengthen our understanding of how changes in colonic microbiota influence IBS-C, paving the way for more efficacious and personalised treatments in the future.

Key Points

- IBS-C is a gastrointestinal disorder characterised by abdominal discomfort and constipation.
- Recent studies suggest that this is caused by differences in the microbiota genome.
- The gut-brain axis, serotonin metabolism and intestinal motility are influenced by microbiota changes in IBS-C.
- The limitations of current research include inconsistencies in microbiota findings, small sample sizes, methodological variability, and a lack of standardised approaches, highlighting the need for larger, longitudinal studies to establish clearer associations between gut microbiota and IBS-C.

Availability of Data and Materials

All the data of this study are included in this article.

Author Contributions

YJR designed the research study. YJR, YM, JR, AHMA, AAG, AF and JSK analysed the data. JR, AHMA, AAG, AF drafted the manuscript. All authors contributed to editorial changes of the important content in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

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