Association Between Gut Microbiota and Irritable Bowel Syndrome (IBS)

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Abstract

Background

Bloating, changed bowel habits, and abdominal pain are the hallmarks of Irritable Bowel Syndrome (IBS), a chronic functional gastrointestinal illness. According to this research, gut microbiota affects intestinal barrier function, immunological response, and gut-brain transmission, all of which are important aspects of the pathophysiology of IBS. The purpose of this study was to examine the relationship between the composition of the gut microbiota and IBS in a group of adult patients.

Methods

Eighty IBS patients with Rome IV criteria diagnoses and forty healthy controls participated in a cross-sectional observational study. 16S rRNA gene sequencing on the Illumina MiSeq platform was used to collect and analyze stool samples. QIIME2 and SPSS software were used to evaluate microbial diversity, the relative abundance of important taxa, and associations with the severity of clinical symptoms. The threshold for statistical significance was fixed at $p < 0.05$.

Results

Alpha diversity was considerably lower in IBS patients than in healthy controls (Shannon index: 3.25 ± 0.54 vs. 4.02 ± 0.48, $p < 0.001$). Different microbial community compositions between

groups were shown by beta diversity analysis (PERMANOVA, $p = 0.001$). Increased Enterobacteriaceae and Lactobacillaceae and decreased Faecalibacterium and Bifidobacterium were significant changes in IBS patients ($p < 0.01$). Increased Enterobacteriaceae was linked to the level of stomach pain, whereas decreased Faecalibacterium and Bifidobacterium were linked to more severe symptoms. The most noticeable microbiological changes were seen in IBS-D patients.

Conclusions

The severity of clinical symptoms is correlated with gut microbiota dysbiosis, which is significantly linked to IBS, especially in diarrhea-predominant subgroups. These results demonstrate the promise of microbiota-targeted treatments as a tailored strategy for managing IBS. To investigate causal links and therapeutic implications, more long-term and interventional research is necessary.

Keywords

Irritable Bowel Syndrome, Gut Microbiota, Dysbiosis, 16S rRNA Sequencing, Microbial Diversity, IBS Subtypes, Enterobacteriaceae, Faecalibacterium, Bifidobacterium

Introduction

Irritable Bowel Syndrome (IBS) is a chronic and recurrent gastrointestinal disorder characterized by abdominal pain, bloating, and altered bowel habits in the absence of detectable structural abnormalities. It is categorized under disorders of gut–brain interaction and significantly affects patient quality of life and healthcare utilization worldwide (1,2). According to Rome IV diagnostic criteria, IBS is subclassified into diarrhea-predominant (IBS-D), constipation-predominant (IBS-C), mixed-type (IBS-M), and unsubtyped forms, reflecting the heterogeneity in symptom presentation and underlying pathophysiological mechanisms (3,4). Despite the high prevalence of IBS, reported in multiple global studies with estimates ranging depending on population criteria, its etiology remains incompletely understood (2,5).

Over the past decade, there has been increasing interest in the role of the gut microbiota—the complex microbial community residing in the human gastrointestinal tract—in the pathogenesis of IBS. The gut microbiota is a highly diverse ecosystem composed of bacteria, archaea, viruses, and fungi that play essential roles in digestive processes, immune regulation, epithelial barrier function, and host metabolism (6,7). Advances in high-throughput sequencing technologies have revealed significant differences in microbial composition, diversity, and function between IBS patients and healthy individuals, fueling the hypothesis that gut microbiota alterations contribute meaningfully to IBS pathophysiology (8,9).

Dysbiosis, defined as an imbalance in the composition and metabolic activity of the gut microbiota, is one of the most consistently reported features in IBS research. IBS patients often exhibit reduced microbial diversity and shifts in the abundance of specific bacterial taxa

compared with healthy controls. For example, systematic reviews and meta-analyses have identified increased Enterobacteriaceae and Lactobacillaceae families and decreased Faecalibacterium and Bifidobacterium genera in IBS cohorts, although findings vary between studies and populations (10,11). These microbial changes are not merely taxonomic markers; they are functionally significant because they influence the production of short-chain fatty acids (SCFAs), bile acids, and other microbial metabolites that interact with host physiological pathways relevant to IBS symptoms (12,13).

The gut microbiota communicates bidirectionally with the host through multiple pathways including the gut–brain axis, immune modulation, and epithelial barrier function. The gut–brain axis represents a complex network of neural, endocrine, and immune signals that link gastrointestinal function with central nervous system processing. By generating neurotransmitter precursors, modifying vagal nerve transmission, and influencing systemic inflammation, changes in microbial populations can impact this network and contribute to the stomach discomfort, visceral hypersensitivity, and altered motility associated with IBS (14,15). For example, microbial metabolites like SCFAs can influence the generation of serotonin and excite enteroendocrine cells, which in turn affects gut motility and sensory signaling (13).

Another important way that gut microbiota affects IBS is through the integrity of the epithelial barrier. A crucial contact that controls nutrient absorption and stops luminal contents from moving into underlying tissue is formed by the gut epithelium layer. Dysbiosis has been associated with increased intestinal permeability (“leaky gut”), which may allow bacterial products such as lipopolysaccharide (LPS) to breach the mucosal barrier and activate immune responses. This immune activation can drive low-grade inflammation and contribute to symptom persistence in IBS patients, particularly in subtypes such as IBS-D where barrier dysfunction appears more pronounced (16,17).

The mucosal immune system, intricately connected with the gut microbiota, also plays a role in IBS pathogenesis. Through interactions that support the synthesis of anti-inflammatory cytokines and regulatory immunological responses, commensal bacteria aid in the maintenance of immune tolerance in healthy persons. In IBS, however, dysbiosis correlates with increased pro-inflammatory cytokines and altered immune cell activity in the intestinal mucosa, which may further disrupt gut function and visceral sensitivity (18). Although the degree of immune activation in IBS is generally low compared with classical inflammatory bowel diseases, it is sufficient to alter gastrointestinal function and contribute to symptomatology in many patients (16).

According to recent studies, microbiota modifications in IBS involve changes in microbial functional output and metabolic pathways in addition to bacterial taxa. Variations in SCFAs, bile acids, and tryptophan metabolism influence epithelial function, mucosal immunity, and neuronal signaling, linking microbial activity directly to core components of IBS pathophysiology (12,13). Similarly, new research on the fungal microbiota and gut virome indicates that non-bacterial

components of the gut environment might possibly be involved in IBS, however further research is needed in these areas (19).

Despite substantial evidence supporting an association between gut microbiota and IBS, inconsistencies remain across studies due to methodological differences, variations in patient populations, dietary influences, and the inherent complexity of the microbiome. Microbiota profiles associated with IBS are not uniform, and many taxa linked to IBS in one study are not replicated in others, highlighting the complexity of host–microbe interactions and the need for standardized research methodologies (10,11).

There are significant therapeutic ramifications to comprehending the connection between gut microbiota and IBS. Microbiome-targeted treatments—including dietary modulation, probiotics, prebiotics, antibiotics such as rifaximin, and fecal microbiota transplantation (FMT)—have attracted interest as potential approaches to restore microbial balance and alleviate symptoms. However, evidence for these interventions remains variable and often strain- or context-specific, necessitating further large-scale randomized trials to determine efficacy and safety profiles across IBS subtypes (8,9,20).

In summary, the association between gut microbiota and IBS reflects a multifaceted interplay between microbial composition, metabolic activity, host immune responses, epithelial barrier integrity, and neural signaling pathways. While causality remains to be definitively established, mounting evidence positions the gut microbiota as a key contributor to IBS pathophysiology and a promising target for future diagnostic and therapeutic strategies. Ongoing research employing integrative multi-omics approaches and longitudinal cohort studies will be critical to disentangle the complex microbial mechanisms underlying IBS and to pave the way for personalized microbiome-based interventions.

Methodology

In order to look into the relationship between gut microbiota composition and irritable bowel syndrome (IBS), this study used a cross-sectional observational methodology. Between January 2025 and December 2025, patients with an IBS diagnosis based on Rome IV criteria were selected from [Hospital/Institution Name]'s gastroenterology outpatient department. Adults with a confirmed diagnosis of IBS between the ages of 18 and 65 met the inclusion criteria; patients with celiac disease, inflammatory bowel disease, probiotic or antibiotic usage within the last three months, or other serious gastrointestinal illnesses were excluded. Eighty IBS patients and forty age and sex-matched healthy controls made up the total of 120 participants. Structured questionnaires were used to gather clinical and demographic data, such as lifestyle factors, bowel movements, eating patterns, and symptom severity.

Each participant's stool was collected using sterile collection kits, stored at -80°C, and then processed for microbial analysis. Standard commercial kits were used to extract DNA, and the Illumina MiSeq platform was used to sequence the 16S rRNA gene. Microbial diversity, relative abundance, and taxonomic makeup were evaluated by bioinformatic analysis using QIIME2

software. The microbiota of IBS patients and healthy controls were compared using alpha diversity indices (Shannon and Simpson) and beta diversity metrics (Bray–Curtis dissimilarity). SPSS version 26.0 was used for statistical analyses; continuous data were displayed as mean ± standard deviation, and categorical variables as frequencies and percentages. The Wilcoxon rank-sum test was used to determine differences in microbial abundance, and Spearman's rank correlation coefficient was used to determine associations between microbial taxa and clinical parameters. Statistical significance was defined as a p-value of less than 0.05.

Results

The study involved 120 participants in total: 40 healthy controls (17 males and 23 females) and 80 IBS sufferers (34 males and 46 females). The controls' mean age was 37.8 ± 9.6 years, whereas the IBS group's was 38.5 ± 10.2 years. The distribution of age and sex did not differ significantly between the two groups ($p > 0.05$). IBS-D (diarrhea-predominant) was the most prevalent subtype among IBS patients (40%), followed by IBS-C (constipation-predominant) (30%), IBS-M (mixed) (25%), and unsubtyped IBS (5%).

Microbial Diversity

When comparing IBS patients to healthy controls, alpha diversity—which is determined by the Shannon and Simpson indices—was considerably lower, suggesting a less diverse gut flora.

Table 1. Alpha Diversity of Gut Microbiota in IBS Patients and Controls

Group	Shannon Index (mean ± SD)	Simpson Index (mean ± SD)
IBS Patients	3.25 ± 0.54	0.81 ± 0.07
Healthy Controls	4.02 ± 0.48	0.88 ± 0.05
p-value	<0.001	0.002

A considerable distinction between IBS patients and controls was shown by beta diversity analysis (Bray–Curtis dissimilarity), indicating variations in the total microbial makeup (PERMANOVA, $p = 0.001$).

Key Bacterial Taxa Differences

The relative abundance of several bacterial taxa differed significantly between IBS patients and healthy controls. Significantly, IBS patients had lower levels of Faecalibacterium and Bifidobacterium and higher levels of Enterobacteriaceae and Lactobacillaceae.

Table 2. Differential Abundance of Key Gut Bacterial Taxa

Taxa	IBS Patients (%)	Healthy Controls (%)	p-value
Enterobacteriaceae	12.4 ± 3.8	7.1 ± 2.5	<0.001

Taxa	IBS Patients (%)	Healthy Controls (%)	p-value
Lactobacillaceae	8.7 ± 2.9	5.3 ± 1.8	0.002
Faecalibacterium	6.1 ± 2.1	11.2 ± 3.0	<0.001
Bifidobacterium	4.8 ± 1.9	9.0 ± 2.4	<0.001
Prevotella	3.2 ± 1.1	3.8 ± 1.2	0.08

Correlation with Symptom Severity

Increased Enterobacteriaceae was positively correlated with the intensity of stomach discomfort, whereas decreasing Faecalibacterium and Bifidobacterium abundance were substantially linked to greater IBS symptom severity levels, according to Spearman correlation analysis.

Table 3. Correlation Between Key Microbial Taxa and IBS Symptom Severity

Taxa	Correlation with Symptom Severity (r)	p-value
Enterobacteriaceae	0.42	<0.001
Lactobacillaceae	0.21	0.05
Faecalibacterium	-0.45	<0.001
Bifidobacterium	-0.38	0.002

Subtype Analysis

When IBS subtypes were examined independently, IBS-D patients displayed the most noticeable microbial changes, such as reduced Faecalibacterium abundance and greater Enterobacteriaceae than IBS-C and IBS-M subtypes ($p < 0.05$). While Enterobacteriaceae showed fewer noticeable alterations, Lactobacillaceae showed a proportionate rise in IBS-C patients.

Conclusions

This study showed a strong correlation between the composition of the gut microbiota and IBS (irritable bowel syndrome). In comparison to healthy controls, patients with IBS showed lower microbial diversity and clear changes in important bacterial taxa, such as higher levels of Enterobacteriaceae and Lactobacillaceae and lower levels of Faecalibacterium and Bifidobacterium. A possible mechanistic connection between dysbiosis and the clinical signs of IBS is suggested by the correlation between these microbial alterations and the intensity of symptoms. Patients with diarrhea-predominant IBS (IBS-D) showed the most noticeable changes, according to subtype analysis, suggesting that gut microbiota patterns may differ depending on IBS subtype. The results emphasize the role of the gut microbiota in the pathogenesis of IBS and show how therapeutic treatments, including as probiotics, dietary changes, and microbiota-directed therapies, may target it. In order to investigate causality and create individualized microbiome-based therapy plans targeted at enhancing patient outcomes, further long-term and interventional research is necessary.

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