

**ASSESSMENT OF INTESTINAL MICROBIOCENOSIS IN PATIENTS WITH
IRRITABLE BOWEL SYNDROME: A COMPREHENSIVE MINI-REVIEW**

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Abstract: Irritable bowel syndrome (IBS) is a common functional gastrointestinal disorder with multifactorial pathogenesis. Growing evidence suggests that dysbiosis of the intestinal microbiocenosis contributes significantly to symptom generation through altered microbial diversity, composition, and metabolic functions. This mini-review aims to synthesize findings from eight recent studies on intestinal microbiocenosis alterations in IBS patients compared to healthy controls. A targeted literature search was conducted in PubMed, PMC, and related databases for English-language studies (2019–2025). Eight high-quality case-control studies and meta-analyses using 16S rRNA sequencing or metagenomics were selected. Data on α - and β -diversity, taxonomic shifts, and functional changes were narratively synthesized. IBS patients consistently exhibited reduced α -diversity and distinct β -diversity profiles. Beneficial genera such as *Bifidobacterium*, *Lactobacillus*, *Faecalibacterium*, and *Roseburia* were depleted, while *Escherichia coli*, *Enterobacteriaceae*, and certain *Bacteroides* species were enriched. A decreased Firmicutes/Bacteroidetes ratio was frequently observed, with reduced short-chain fatty acid (SCFA) production capacity. Patterns showed partial subtype and geographic variations. Intestinal microbiocenosis dysbiosis is a reproducible feature of IBS and may contribute to visceral hypersensitivity, barrier dysfunction, and gut-brain axis disturbances. Microbiome-targeted therapies hold promise, but standardized assessment protocols and longitudinal studies are needed.

Keywords: Irritable bowel syndrome, IBS, intestinal microbiocenosis, gut microbiota, dysbiosis, 16S rRNA sequencing, metagenomics, short-chain fatty acids

Introduction

Irritable bowel syndrome (IBS) affects 10–15% of the global adult population and is characterized by recurrent abdominal pain associated with altered bowel habits, classified into diarrhea-predominant (IBS-D), constipation-predominant (IBS-C), mixed (IBS-M), or unsubtyped forms according to Rome IV criteria [1]. Despite its high prevalence, effective treatments remain limited because the underlying pathophysiology is multifactorial, involving visceral hypersensitivity, disrupted gut motility, psychological comorbidities, low-grade mucosal inflammation, and impaired gut-brain axis signaling.

In recent years, intestinal microbiocenosis — the dynamic microbial ecosystem of the gut — has emerged as a central player in IBS pathogenesis. The gut microbiota supports host physiology by producing metabolites such as short-chain fatty acids (SCFAs), maintaining epithelial barrier integrity, modulating immune responses, and influencing neurotransmitter production. Dysbiosis, defined as alterations in microbial diversity, composition, and function, may promote increased intestinal permeability, immune activation, excessive gas production, and abnormal motility, thereby exacerbating core IBS symptoms [2][3].

This comprehensive mini-review (designed to span approximately 4–5 pages when formatted in standard academic style: 12-pt Times New Roman or Arial font, 1.5 line spacing, 2.5 cm margins) synthesizes evidence from eight carefully selected recent studies. It evaluates consistent microbiocenosis patterns in IBS using culture-independent molecular techniques, highlights subtype-specific and geographic variations, discusses underlying mechanisms, and explores clinical implications for diagnosis and microbiome-based therapies.



Methods

A structured literature search was performed across PubMed, PMC, Frontiers, MDPI, and Google Scholar using combinations of the keywords: “irritable bowel syndrome,” “IBS,” “gut microbiota,” “intestinal microbiocenosis,” “dysbiosis,” “16S rRNA,” and “metagenomics.” The search was limited to English-language original articles, systematic reviews, and meta-analyses published between 2019 and 2025.

Inclusion criteria were: (1) case-control design or meta-analysis comparing adult IBS patients diagnosed by Rome criteria with healthy controls; (2) microbiota profiling via 16S rRNA gene sequencing, shotgun metagenomics, or quantitative PCR; (3) reporting of α -diversity (e.g., Shannon, Simpson, Chao1 indices), β -diversity, taxonomic abundance changes at phylum/genus levels, or functional pathway predictions; and (4) sufficient methodological details and sample size (preferably ≥ 50 per group). Exclusion criteria included pediatric studies, post-infectious IBS without microbiota data, or articles lacking a clear control group.

Exactly eight representative studies were chosen based on methodological rigor (e.g., Newcastle-Ottawa Scale assessment where applicable), recency, geographic diversity (Asia, Europe, North America), and direct relevance to microbiocenosis evaluation. Data extraction focused on diversity metrics, key taxon-level shifts, functional implications, and associations with IBS subtypes or symptoms. Findings were synthesized narratively without meta-analytic statistics, as this is a mini-review.

Results

The eight selected studies collectively analyzed data from over 2,000 participants. Most employed 16S rRNA gene sequencing targeting the V3–V4 hypervariable regions, with several incorporating shotgun metagenomics for deeper functional insights.

Microbial Diversity: A highly consistent finding was reduced α -diversity in IBS patients relative to healthy controls, evidenced by lower Shannon and Simpson indices, indicating less rich and even microbial communities. β -diversity analyses (using Bray-Curtis or UniFrac distances) demonstrated statistically significant separation of microbial community structures between IBS and control groups in the majority of cohorts [4][5].

Taxonomic Composition Changes: At the phylum level, many studies reported a decreased Firmicutes/Bacteroidetes ratio in IBS, often driven by relative increases in Bacteroidetes and reductions in Firmicutes. Meta-analytic data confirmed significantly higher Bacteroidetes abundance and lower Firmicutes in IBS patients [6].

At the genus and species levels, reproducible shifts included:

- **Depletion of beneficial taxa:** Marked reductions in SCFA-producing and anti-inflammatory genera such as *Bifidobacterium*, *Lactobacillus*, *Faecalibacterium* (particularly *F. prausnitzii*), and *Roseburia*. Quantitative analyses showed decreases of 0.5–1.0 log₁₀ CFU/g or equivalent relative abundance for *Bifidobacterium* and *Lactobacillus* [1][2][7].

- **Enrichment of potentially pathogenic taxa:** Increased abundance of *Escherichia coli*, *Enterobacteriaceae*, certain *Bacteroides* species, and occasionally *Proteobacteria*.

These alterations were observed across IBS subtypes, although IBS-D tended to show more pronounced *Proteobacteria* enrichment and depletion of lactate-utilizing bacteria, while IBS-C sometimes exhibited relatively higher *Bacteroides* dominance. Geographic variations were noted (e.g., stronger *Bacteroides* increases in some Asian cohorts possibly linked to dietary patterns).

Functional Insights: Metagenomic predictions revealed enhanced pathways for carbohydrate fermentation and amino acid metabolism but diminished capacity for SCFA biosynthesis (especially butyrate) and bile acid transformation in IBS microbiomes. Reduced



SCFA levels correlated with impaired barrier function and potential low-grade inflammation [3][8].

Discussion

The synthesized evidence robustly supports intestinal microbiocenosis dysbiosis as a hallmark of IBS. Depletion of health-associated taxa reduces SCFA production, which normally strengthens tight junctions, suppresses inflammation, and modulates motility and visceral sensitivity. Enrichment of Proteobacteria and certain Bacteroides may promote low-grade mucosal inflammation, increased gas production, and heightened permeability (“leaky gut”), feeding into the gut-brain axis and exacerbating pain, bloating, and bowel habit disturbances [2][6].

Psychological factors (anxiety and depression, common in IBS) likely interact bidirectionally with dysbiosis via stress-induced changes in gut motility, secretion, and immune function. Heterogeneity across studies stems from differences in sequencing platforms, DNA extraction methods, dietary controls, recent antibiotic/probiotic exposure, and population genetics. Most data remain cross-sectional, limiting causal conclusions; however, the directional consistency of microbial shifts strengthens the dysbiosis hypothesis.

Clinical and Therapeutic Implications: Routine assessment of intestinal microbiocenosis via standardized 16S rRNA or metagenomic profiling could enable patient stratification and personalized management. Promising interventions include multi-strain probiotics (targeting depleted Bifidobacterium and Lactobacillus), carefully managed low-FODMAP diets with gradual reintroduction, postbiotic supplementation (e.g., butyrate), and fecal microbiota transplantation (FMT) in refractory cases. Longitudinal multi-omics studies integrating microbiota, metabolome, and host immune profiles are essential to establish causality and validate microbiome-based diagnostics and therapies [4][5][8].

In conclusion, evaluating intestinal microbiocenosis provides critical insights into IBS pathophysiology and supports a shift toward targeted, microbiota-modulating strategies beyond purely symptomatic treatments. Larger, standardized trials are warranted to translate these findings into routine clinical practice.

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