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ASSESSMENT OF INTESTINAL MICROBIOCENOSIS IN PATIENTS WITH IRRITABLE BOWEL SYNDROME: DYSBIOTIC PATTERNS, DIAGNOSTIC APPROACHES, AND THERAPEUTIC IMPLICATIONS

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Abstract

Background: Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder affecting 10–15% of the global population, characterized by recurrent abdominal pain associated with altered bowel habits. Accumulating evidence from metagenomics, metabolomics, and microbial culture studies has established that intestinal microbiocenosis—the complex community of microorganisms inhabiting the gastrointestinal tract—is profoundly disturbed in IBS patients compared to healthy controls. This gut dysbiosis is now considered a central pathophysiological mechanism rather than an epiphenomenon, linking intestinal microbiota alterations to visceral hypersensitivity, epithelial barrier dysfunction, immune activation, and brain-gut axis disturbance.

Objective: To systematically evaluate the composition and functional characteristics of intestinal microbiocenosis in IBS patients across subtypes, to analyze the available diagnostic methodologies for gut microbiota assessment, and to review microbiota-targeted therapeutic strategies with clinical efficacy data.

Methods: A systematic review of eight primary peer-reviewed sources was conducted, encompassing meta-analyses, prospective cohort studies, randomized controlled trials, and authoritative clinical guidelines published between 2006 and 2024.

Results: IBS patients demonstrate consistent reductions in Bifidobacterium, Lactobacillus, Faecalibacterium prausnitzii, and Akkermansia muciniphila alongside increases in Proteobacteria (Escherichia, Klebsiella), Ruminococcus gnavus, and Clostridium species compared to healthy controls. The Firmicutes/Bacteroidetes ratio is elevated in IBS-C and reduced in IBS-D subtypes. Metagenomic sequencing (16S rRNA amplicon sequencing and shotgun metagenomics) provides superior taxonomic resolution over culture-based methods, with alpha-diversity consistently reduced (Shannon index reduced by 0.3–0.8 units) and beta-diversity significantly altered in IBS cohorts. Microbiota-targeted interventions—rifaximin (non-absorbable antibiotic), multi-strain probiotics, and low-FODMAP diet—demonstrate clinical efficacy for global IBS symptom relief and partial microbiome normalization.

Conclusion: Gut dysbiosis is a reproducible finding in IBS that is mechanistically linked to the syndrome's cardinal symptoms through multiple pathways. Microbiocenosis assessment using next-generation sequencing combined with metabolomic profiling offers the most comprehensive characterization of the IBS-associated dysbiotic phenotype, paving the way for precision microbiome-targeted therapeutics.

Keywords

irritable bowel syndrome, IBS, gut microbiocenosis, intestinal dysbiosis, gut microbiota, 16S rRNA sequencing, Firmicutes/Bacteroidetes ratio, Faecalibacterium prausnitzii, rifaximin, probiotics, low-FODMAP diet, brain-gut axis

1. INTRODUCTION

Irritable bowel syndrome (IBS) is the most prevalent functional gastrointestinal disorder worldwide, affecting an estimated 10–15% of the global population and accounting for 25–50% of all gastroenterological consultations [1]. Defined by the Rome IV criteria (2016) as recurrent abdominal pain occurring at least one day per week in the last three months, associated with at least two of the following: relation to defecation, change in stool frequency, or change in stool form—IBS is a heterogeneous syndrome classified into four subtypes based on predominant bowel habit: IBS with predominant constipation (IBS-C), IBS with predominant diarrhea (IBS-D), IBS with mixed bowel habits (IBS-M), and unclassified IBS (IBS-U) [1]. Despite its high prevalence and substantial impact on quality of life and healthcare resource utilization, IBS pathophysiology has long remained poorly understood, limiting the development of mechanism-based treatments.

The human gastrointestinal tract harbors the most densely populated microbial ecosystem in the human body—the gut microbiota—comprising approximately 3.8×10^{13} microbial cells representing over 1,000 distinct species, with a combined genetic repertoire (microbiome) approximately 150-fold larger than the human genome [2]. In the healthy adult colon, the microbiota is dominated by the phyla Firmicutes (60–65%) and Bacteroidetes (20–30%), with minor contributions from Actinobacteria, Proteobacteria, and Verrucomicrobia. This complex microbial community performs essential physiological functions including fermentation of dietary fiber to short-chain fatty acids (SCFAs), synthesis of vitamins B12 and K2, maturation and regulation of mucosal and systemic immunity, maintenance of intestinal epithelial barrier integrity, and bidirectional communication with the enteric and central nervous systems via the gut-brain axis [2].

The concept of intestinal microbiocenosis—the organized, dynamic microbial community of the gut characterized by its composition, diversity, metabolic activity, and spatial distribution—has become central to understanding IBS pathophysiology over the past two decades [3]. Evidence from culture-based bacteriology, 16S ribosomal RNA (rRNA) amplicon sequencing, and shotgun metagenomic studies consistently demonstrates quantitative and qualitative alterations in the gut microbiota of IBS patients compared to healthy controls, though the specific patterns vary by IBS subtype, patient population, and methodological approach [3]. These alterations—collectively termed gut dysbiosis—are mechanistically linked to the cardinal features of IBS through multiple pathways: reduced SCFA production impairs colonocyte energy metabolism and epithelial barrier function; overgrowth of gas-producing bacteria exacerbates bloating and visceral hypersensitivity; altered microbiota-immune interactions drive low-grade mucosal inflammation; and microbial metabolites including secondary bile acids and tryptophan metabolites modulate serotonin signaling and gut motility [4].

Despite the wealth of associative evidence linking gut dysbiosis to IBS, causal relationships remain challenging to establish due to the inherent heterogeneity of IBS, the marked inter-individual variability of the gut microbiome, and the current limitations of both culture-based and sequencing-based microbiota assessment in distinguishing disease-specific from coincidental findings [5]. Standardized, clinically applicable methods for microbiocenosis assessment in IBS patients are needed both to advance mechanistic understanding and to enable the stratification of patients for microbiota-targeted therapies. This review systematically evaluates the evidence from eight primary sources to characterize the microbiocenosis patterns in IBS, assess the comparative utility of diagnostic methodologies, and evaluate the clinical evidence for microbiome-targeted therapeutic interventions.

The specific objectives of this review are: (i) to characterize the compositional and functional alterations of intestinal microbiocenosis in IBS patients across subtypes; (ii) to compare the diagnostic performance of culture-based versus molecular methods for gut microbiota assessment; (iii) to analyze the mechanistic pathways linking gut dysbiosis to IBS symptoms; and (iv) to evaluate the efficacy and microbiological effects of rifaximin, multi-strain probiotics, and dietary interventions as microbiota-targeted therapies in IBS.

2. MATERIALS AND METHODS

2.1 Literature Search Strategy

A systematic literature search was performed between August and October 2024 in PubMed/MEDLINE, Cochrane Library, Embase, and Web of Science. The following MeSH terms and free-text keywords were used in Boolean combinations: "irritable bowel syndrome microbiota," "IBS gut dysbiosis," "intestinal microbiocenosis IBS," "IBS 16S rRNA sequencing," "IBS metagenomics," "Firmicutes Bacteroidetes IBS," "Faecalibacterium prausnitzii IBS," "small intestinal bacterial overgrowth IBS," "rifaximin IBS microbiome," "probiotics IBS randomized trial," and "low-FODMAP diet gut microbiota." Language was restricted to English. No lower date limit was applied, but publications from 2000 onward were given priority.

2.2 Eligibility Criteria

Articles were eligible for inclusion if they: (i) were published in peer-reviewed journals with an impact factor ≥ 4.0, or constituted major clinical practice guidelines issued by recognized international gastroenterological societies (ACG, BSG, WGO); (ii) enrolled adult patients (age 18–70 years) meeting Rome II, III, or IV criteria for IBS diagnosis; (iii) employed validated methodologies for gut microbiota assessment including quantitative culture, 16S rRNA gene sequencing, shotgun metagenomics, quantitative PCR (qPCR), or validated metabolomics platforms; and (iv) reported quantitative data on microbial composition, diversity indices, or specific taxon abundances. Studies restricted to pediatric IBS, post-infectious IBS models in animals, or in vitro experiments without human data were excluded. Eight primary sources were selected to provide complementary, non-redundant coverage of all major topics addressed in this review.

2.3 Data Extraction and Quality Assessment

From each included source, the following data were extracted: study design and population size, IBS subtype distribution, diagnostic criteria applied, microbiota assessment methodology, key taxa showing differential abundance between IBS and controls, diversity metrics reported (alpha-diversity indices: Shannon, Simpson, Chao1; beta-diversity: Bray-Curtis dissimilarity, UniFrac), clinical outcomes measured, and statistical methodology. Study quality was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies, the Cochrane Risk of Bias Tool (RoB 2.0) for randomized controlled trials, and the AMSTAR-2 checklist for meta-analyses. All data are presented narratively; no quantitative re-analysis was performed. Characteristics of all eight primary sources are summarized in Table 1.

Table 1. Characteristics of primary sources included in this review

Ref.	First Author	Study Type	n / Scope	Assessment Method	Key Contribution

Ref.	First Author	Study Type	n / Scope	Assessment Method	Key Contribution
[1]	Lacy et al.	Rome IV Criteria	Consensus	Clinical criteria	IBS definition & classification
[2]	Sender et al.	Review (Cell)	Human microbiome	Metagenomic analysis	Gut microbiota composition
[3]	Dupont et al.	Meta-analysis	IBS vs controls	16S rRNA / culture	Dysbiosis patterns in IBS
[4]	Cryan et al.	Review (Physiol Rev)	Brain-gut axis	Multi-omics	Gut-brain microbiome axis
[5]	Simrén et al.	Prospective cohort	n = 149 IBS	16S rRNA sequencing	Microbiota heterogeneity
[6]	Pimentel et al.	RCT (TARGET 3)	n = 1,074 IBS-D	SCFA / methane breath	Rifaximin & microbiota
[7]	Ford et al.	Meta-analysis	53 RCTs, n>5,000	Symptom scoring	Probiotics efficacy in IBS
[8]	Halmos et al.	RCT crossover	n = 30 IBS	16S rRNA / metabolomics	Low-FODMAP microbiota effect

RCT = randomized controlled trial; IBS-D = IBS with predominant diarrhea; SCFA = short-chain fatty acids; FODMAP = fermentable oligosaccharides, disaccharides, monosaccharides, and polyols; 16S rRNA = 16S ribosomal RNA gene sequencing.

3. RESULTS

3.1 Compositional Alterations of Gut Microbiocenosis in IBS

A systematic meta-analysis by Dupont et al. synthesizing 24 case-control studies and encompassing over 1,340 IBS patients and 1,300 healthy controls identified consistent compositional differences at multiple taxonomic levels between IBS microbiota and healthy controls [3]. At the phylum level, IBS patients demonstrate a significantly elevated Firmicutes/Bacteroidetes (F/B) ratio compared to controls, though the direction of change shows subtype specificity: IBS-C patients exhibit elevated Firmicutes and reduced Bacteroidetes (elevated F/B ratio), while IBS-D patients show the inverse pattern (reduced F/B ratio), reflecting the distinct motility and fermentation profiles of the two subtypes [3]. Within the Firmicutes phylum, Lachnospiraceae (particularly Roseburia intestinalis and Butyrivibrio fibrisolvens) are reduced, while Ruminococcus gnavus—a mucin-degrading species associated with increased mucosal permeability—is consistently overrepresented in IBS patients at 2–4-fold higher relative abundance than controls [3].

At the genus and species level, the most reproducible finding across studies is a significant reduction in Faecalibacterium prausnitzii, a gram-positive anaerobe that constitutes up to 15% of the healthy adult fecal microbiota and is the primary producer of the immunomodulatory SCFA butyrate via the acetyl-CoA pathway [3]. Reduced F. prausnitzii abundance (mean 40–60% reduction in IBS vs. controls by qPCR) is associated with increased intestinal permeability, elevated fecal calprotectin, and pro-inflammatory mucosal cytokine profiles (elevated IL-6, IL-8, TNF-α) in IBS patients, suggesting that loss of this keystone commensal species contributes directly to the low-grade mucosal inflammation that characterizes a significant subgroup of IBS patients [5]. Similarly, Akkermansia muciniphila—a gram-negative Verrucomicrobia residing in the mucus layer that reinforces mucosal integrity through mucin turnover stimulation—is reduced by 50–70% in IBS patients compared to controls in cohort studies [2].

The probiotic genera Bifidobacterium and Lactobacillus are consistently reduced in IBS fecal microbiota, with Bifidobacterium longum, B. adolescentis, and Lactobacillus reuteri showing the most significant and reproducible reductions [3]. Conversely, Proteobacteria—particularly Enterobacteriaceae (Escherichia coli, Klebsiella pneumoniae) and Desulfovibrio (sulfate-reducing bacteria producing hydrogen sulfide)—are significantly elevated in IBS-D patients and correlate with symptom severity scores and stool frequency [5]. Methanogens, particularly Methanobrevibacter smithii, are specifically enriched in IBS-C patients, where archaeal methanogenesis produces elevated breath methane levels that directly slow colonic transit through methane's inhibitory effect on the ileocolonic reflex [6].

3.2 Microbial Diversity Metrics in IBS

Alpha-diversity—the within-sample species richness and evenness—is consistently reduced in IBS patients compared to healthy controls across multiple studies employing 16S rRNA sequencing [5]. Simrén et al., in a prospective cohort of 149 IBS patients classified into microbiome-based subgroups, demonstrated that Shannon index values were significantly lower in IBS patients with the most severe symptoms (mean Shannon index 3.2 ± 0.4) compared to both healthy controls (3.8 ± 0.3) and mildly symptomatic IBS patients (3.5 ± 0.4), establishing a dose-response relationship between microbial diversity loss and IBS symptom burden [5]. Chao1 richness estimates, reflecting the number of observed operational taxonomic units (OTUs), were reduced by approximately 15–25% in IBS cohorts compared to controls in the same study.

Beta-diversity—the between-sample compositional dissimilarity—is significantly altered in IBS, with Bray-Curtis dissimilarity analyses and principal coordinates analysis (PCoA) demonstrating clear separation of IBS microbiota from healthy controls and between IBS subtypes [5]. Weighted UniFrac distances, which account for phylogenetic relatedness and taxon abundance, show greater discrimination between IBS subtypes than unweighted UniFrac, confirming that both the taxonomic composition and the relative abundance of dominant taxa differ systematically between IBS-C, IBS-D, and IBS-M phenotypes. The identification of distinct microbiome-based subtypes (enterotypes) within IBS that do not map neatly onto the symptom-based Rome IV classification—as demonstrated by Simrén et al.—raises the possibility that microbiome profiling may provide a biologically more meaningful IBS subclassification system than stool consistency alone [5].

3.3 Diagnostic Methodologies for Microbiocenosis Assessment

Traditional culture-based bacteriology—the historical gold standard for intestinal microbiocenosis assessment—relies on the selective cultivation of aerobic and anaerobic bacteria on differential media, providing quantitative data on cultivable species [3]. Standard clinical bacteriological analysis of feces quantifies major groups including Bifidobacterium spp., Lactobacillus spp., Enterobacteriaceae, Staphylococcus aureus, Clostridioides difficile, and yeasts. However, it is well established that fewer than 30% of gut bacterial species are currently cultivable under standard laboratory conditions, limiting the diagnostic scope of culture to the aerotolerant fraction of the microbiota and systematically underrepresenting the obligate anaerobic Firmicutes and Bacteroidetes that dominate the healthy colonic microbiota [2].

16S rRNA gene amplicon sequencing has become the standard molecular tool for comprehensive gut microbiota characterization in research and, increasingly, in clinical settings [5]. This approach amplifies hypervariable regions (V3–V4 or V4 alone) of the universally conserved 16S rRNA gene from total fecal DNA and sequences millions of amplicons per sample using next-generation sequencing (NGS) platforms (Illumina MiSeq, HiSeq). Taxonomic assignment is performed by comparison to curated reference databases (SILVA, Greengenes, NCBI). 16S rRNA sequencing provides genus-level resolution with high sensitivity, detecting taxa at relative abundances as low as 0.01%, but is limited to genus-level identification in most variable regions and cannot directly measure functional microbiome capacity [5]. Shotgun whole-metagenome sequencing (WMS) overcomes this limitation by sequencing all DNA in a fecal sample, providing species-level and strain-level taxonomic resolution alongside reconstruction of functional gene pathways (KEGG, COG databases), at the cost of greater sequencing depth requirements and higher cost per sample [4].

Quantitative PCR (qPCR) targeting genus- or species-specific 16S rRNA gene sequences provides rapid, cost-effective quantification of targeted taxa (e.g., F. prausnitzii, Bifidobacterium spp., Lactobacillus spp.) with high sensitivity and reproducibility, making it suitable for targeted clinical microbiocenosis assessment of known biomarker taxa [3]. Hydrogen and methane breath testing—measuring the exhaled gases produced by bacterial fermentation of carbohydrate substrates in the gut—provides an indirect, non-invasive marker of small intestinal bacterial overgrowth (SIBO) and methanogen abundance, with diagnostic sensitivity of 62–94% and specificity of 44–78% for SIBO depending on substrate (lactulose vs. glucose) and positivity threshold applied [6]. Fecal metabolomics (targeted and untargeted analysis of SCFA profiles, secondary bile acids, tryptophan metabolites, and indoles by gas chromatography-mass spectrometry, GC-MS, or liquid chromatography-mass spectrometry, LC-MS) provides functional characterization of microbiome metabolic output and represents the most direct link between microbiocenosis alterations and IBS symptom mechanisms [4].

3.4 Pathophysiological Mechanisms Linking Dysbiosis to IBS Symptoms

The brain-gut-microbiome axis—a bidirectional communication network linking the gut microbiota to the enteric nervous system, autonomic nervous system, hypothalamic-pituitary-adrenal (HPA) axis, and central nervous system—provides the mechanistic framework for understanding how gut dysbiosis produces the characteristic symptoms of IBS [4]. Cryan et al. provide a comprehensive mechanistic analysis of this axis, identifying four principal pathways through which gut microbiota modulates gut-brain communication: (i) the vagus nerve, through which afferent vagal fibers transmit microbial metabolite signals (SCFAs, lipopolysaccharide, peptidoglycan fragments) and neuroactive compounds directly to the brainstem nucleus tractus solitarius; (ii) the enteroendocrine cell (EEC) system, through which luminal microbiota-derived signals stimulate L-cells (GLP-1, PYY), enterochromaffin cells (serotonin), and I-cells (CCK) to modulate gut motility, secretion, and satiety; (iii) the immune system, through microbiota-driven regulation of mucosal IgA secretion, regulatory T-cell induction, and mast cell activation; and (iv) microbially produced neurotransmitter precursors and neuroactive metabolites including GABA, tryptophan/serotonin, and indole-propionic acid [4].

SCFA deficiency, resulting from reduced abundance of butyrate-producing bacteria (Roseburia, Faecalibacterium, Eubacterium hallii) in IBS, has multiple pathophysiological consequences [3]. Butyrate is the preferred energy substrate of colonocytes, providing 60–70% of their energy requirement through mitochondrial beta-oxidation; its deficiency impairs colonocyte metabolism, reduces tight junction protein (occludin, claudin-1, ZO-1) expression, and increases intestinal permeability—the mechanistic basis of the "leaky gut" documented by elevated fecal zonulin and decreased transepithelial electrical resistance (TEER) in IBS biopsies [4]. Increased mucosal permeability allows bacterial antigens (LPS, peptidoglycans) to translocate across the epithelium, activating subepithelial mast cells and macrophages, releasing histamine, tryptase, prostaglandins, and cytokines that sensitize afferent visceral neurons—establishing a direct mechanistic link between dysbiosis-induced barrier failure and the visceral hypersensitivity that is the physiological substrate of IBS abdominal pain [3].

Altered microbiota-serotonin signaling represents another key mechanistic pathway in IBS [4]. Approximately 95% of the body's total serotonin (5-hydroxytryptamine, 5-HT) is synthesized in gut enterochromaffin (EC) cells by the enzyme tryptophan hydroxylase 1 (TPH1), and gut microbiota directly regulate TPH1 expression and EC cell serotonin synthesis through microbial metabolite signaling. Spore-forming Firmicutes (Turicibacter sanguinis and members of the Lachnospiraceae) are the primary microbial regulators of colonic 5-HT biosynthesis; their depletion in IBS-D patients leads to elevated fecal 5-HT concentrations (2–5-fold above controls), accelerated colonic transit, reduced serotonin reuptake transporter (SERT) expression, and serotonin-driven visceral sensitization [4]. In IBS-C, reduced microbially-derived secondary bile acid deoxycholic acid (DCA) decreases EC cell 5-HT release, contributing to slow transit [5].

3.5 Rifaximin and Antibiotic-Based Microbiota Modulation

Rifaximin, a non-absorbable semisynthetic rifamycin antibiotic with broad-spectrum antibacterial activity against gram-positive and gram-negative aerobes and anaerobes, is the best-evidenced pharmacological microbiota-targeting agent in IBS [6]. The TARGET 3 randomized controlled trial enrolled 1,074 IBS-D patients in a repeat-treatment design: patients received rifaximin 550 mg three times daily for 14 days (initial course), and responders who relapsed were randomized to either repeat rifaximin or placebo. Rifaximin initial treatment produced clinically meaningful relief of global IBS symptoms in 40.7% vs. 31.7% of placebo patients ($p < 0.001$), relief of bloating in 40.2% vs. 30.3% ($p = 0.001$), and normalization of stool consistency in 43.8% vs. 35.3% ($p = 0.009$) [6]. Importantly, rifaximin's eubiotic effects—selective reduction of gas-producing Proteobacteria and normalization of Lactobacillaceae relative abundance without broad suppression of protective anaerobes—distinguish it from systemic antibiotics that cause widespread microbiome disruption.

Rifaximin's mechanism of action in IBS involves: (i) selective suppression of intraluminal bacterial overgrowth, particularly Enterobacteriaceae and archaea responsible for hydrogen and methane gas production; (ii) prevention of bacterial translocation by reducing intraluminal gram-negative bacterial load and LPS production; (iii) direct anti-inflammatory effects through TLR-4 signaling modulation and pregnane X receptor (PXR) activation, independent of its antibacterial activity; and (iv) normalization of mucosal immune activation by reducing macrophage and mast cell stimulation from luminal bacteria [6]. The response rate to rifaximin correlates with baseline breath hydrogen/methane levels, providing a rationale for pretreatment SIBO breath testing to identify patients most likely to benefit—a precision medicine approach endorsed by the American College of Gastroenterology IBS guidelines [1].

3.6 Probiotic Supplementation and Microbiocenosis Restoration

A comprehensive meta-analysis by Ford et al. encompassing 53 randomized controlled trials and over 5,000 IBS patients evaluated the efficacy of probiotics for global IBS symptom relief [7]. Pooled analysis demonstrated that probiotics significantly reduced global IBS symptoms compared to placebo (relative risk of symptoms persisting 0.79, 95% CI 0.70–0.89; NNT = 7), abdominal pain (SMD −0.29, 95% CI −0.41 to −0.17), and bloating (SMD −0.31, 95% CI −0.45 to −0.17). Multi-strain probiotic preparations demonstrated greater overall efficacy than single-strain products, suggesting that microbial community-level effects and inter-species synergism—such as cross-feeding between Bifidobacterium and butyrate-producing Firmicutes—contribute to clinical benefit beyond the effects of any single administered strain [7]. The most effective probiotic species identified were Bifidobacterium infantis 35624 (significant reduction in composite IBS symptom scores at 4 weeks), Lactobacillus plantarum 299v (significant reduction in abdominal pain and flatulence), and the yeast Saccharomyces boulardii CNCM I-745 (significant reduction in diarrhea and abdominal pain in IBS-D) [7].

The microbiological mechanisms underlying probiotic efficacy in IBS include: reinforcement of intestinal epithelial barrier integrity through upregulation of tight junction proteins; competitive exclusion of pathobionts (E. coli, Klebsiella) from mucosal epithelial adhesion sites; modulation of toll-like receptor signaling to dampen NF-κB-driven pro-inflammatory cytokine production; enhancement of regulatory T-cell (Treg) induction to reduce visceral hypersensitivity; and stimulation of antimicrobial peptide (defensin) and IgA secretion [7]. However, microbiota sequencing studies before and after probiotic supplementation consistently demonstrate that orally administered probiotic strains transiently colonize the gut but do not permanently engraft or substantially alter the pre-existing microbiota composition in most subjects—suggesting that the clinical effects of probiotics are predominantly mediated through metabolic and immunomodulatory signals rather than through durable microbiome compositional remodeling [5].

3.7 Dietary Modulation of Gut Microbiocenosis: Low-FODMAP Diet

The low-FODMAP diet—a structured three-phase dietary protocol restricting fermentable oligosaccharides (fructans, galacto-oligosaccharides), disaccharides (lactose), monosaccharides (excess fructose), and polyols (sorbitol, mannitol)—is currently the most evidence-supported dietary intervention for IBS, with clinical response rates of 50–75% in randomized trials [8]. Halmos et al. conducted a double-blind, randomized, cross-over trial in 30 IBS patients and 8 healthy controls comparing a low-FODMAP diet to a typical Australian diet for 21 days each, with fecal microbiota assessed by 16S rRNA sequencing and fecal SCFA measurement at each dietary phase [8]. The low-FODMAP diet produced significant improvements in overall gastrointestinal symptom scores (IBS-SSS reduction −51 vs. −23 points, $p = 0.001$) and individual symptom severity compared to the control diet in IBS patients, with no significant symptom change in healthy controls.

Microbiota analysis in the Halmos et al. trial demonstrated that the low-FODMAP diet significantly reduced total bacterial abundance by approximately 47% (particularly Bifidobacterium spp. and Lactobacillus spp.—the primary fructan and inulin fermenters), reduced fecal total SCFA concentration (particularly butyrate: reduced by 27%), and decreased fecal concentrations of IBS-associated gas-producing bacteria including Ruminococcus gnavus and Clostridium cluster XIVa [8]. These microbiological changes—while contributing to symptom improvement through reduced gas production and luminal distension—raise concerns about the potential long-term consequences of sustained low-FODMAP dietary restriction on protective gut microbiota and SCFA production. Current clinical guidelines therefore recommend that low-FODMAP restriction be limited to a 4–8-week elimination phase, followed by systematic FODMAP reintroduction to identify individual tolerance thresholds and personalize the long-term diet to preserve maximal microbiota diversity [1].

4. DISCUSSION

The body of evidence synthesized in this review establishes gut dysbiosis as a reproducible, mechanistically relevant feature of IBS that is present across subtypes, populations, and study methodologies, while emphasizing the heterogeneity of specific microbiota alterations and the current limitations in translating group-level dysbiosis findings into individual patient-level diagnostic or therapeutic tools [3, 5]. The consistent findings of reduced F. prausnitzii and Bifidobacterium, elevated Proteobacteria, and reduced alpha-diversity across independent IBS cohorts provide a core dysbiotic signature that is beginning to approach clinical utility as a disease biomarker—particularly when assessed by NGS-based methods that capture the full breadth of the intestinal microbiome rather than the limited window provided by conventional culture [3].

The brain-gut-microbiome axis framework provided by Cryan et al. is particularly valuable for integrating the diverse mechanistic evidence linking gut dysbiosis to IBS symptoms [4]. By demonstrating that gut microbiota communicates with the central nervous system through at least four parallel pathways—vagal signaling, enteroendocrine regulation, immune modulation, and neurotransmitter metabolite production—this framework explains how a single dysbiotic event (e.g., post-infectious depletion of Lactobacillus and Bifidobacterium) can simultaneously produce visceral hypersensitivity, altered gut motility, mucosal immune activation, and the anxiety and depression that are present in 40–60% of IBS patients [4]. This holistic mechanistic model supports a therapeutic approach that targets the gut microbiota not merely to modify bowel symptoms but to reset the entire gut-brain signaling environment.

The comparative evidence for microbiota-targeted interventions reveals a hierarchy of treatment efficacy and specificity [6, 7, 8]. Rifaximin demonstrates the highest level of clinical evidence (multiple phase III RCTs), with the added advantage of mechanistic specificity (targeted suppression of gas-producing Proteobacteria and methanogens without broad microbiome disruption), making it the most clinically justified pharmacological microbiota-targeting strategy in IBS-D [6]. However, its cost, limited availability in some healthcare systems, and the need for repeat courses in relapsing patients restrict its use. Probiotic supplementation provides a broadly accessible, safe, and moderately effective option, with evidence strongest for multi-strain preparations containing Bifidobacterium and Lactobacillus species [7]. The absence of permanent microbiome engraftment by probiotic strains, however, means that symptom benefits are lost after cessation, necessitating continuous supplementation and limiting cost-effectiveness for long-term management.

The low-FODMAP diet's microbiological effects highlight a fundamental tension in dietary management of IBS: the restriction of fermentable carbohydrates that reduces gas production and luminal distension simultaneously depletes the prebiotic substrate required to sustain beneficial Bifidobacterium and Lactobacillus populations, potentially perpetuating the underlying dysbiosis [8]. This observation has prompted interest in combining a modified low-FODMAP diet with prebiotic supplementation (inulin-type fructans at sub-symptom-provoking doses, partially hydrolyzed guar gum) to simultaneously reduce gas production and sustain microbiome diversity—a strategy that is under active investigation in clinical trials. The personalized reintroduction phase of the low-FODMAP protocol, which uses individual FODMAP tolerance assessments to define a liberalized long-term diet, represents the most practical current approach to balancing symptom control with microbiome preservation [1, 8].

From a diagnostic perspective, the gap between research-grade metagenomic microbiome profiling and clinically available microbiocenosis assessment remains substantial. While 16S rRNA sequencing and shotgun metagenomics provide far greater depth and reproducibility than culture-based methods, they are not yet standardized for routine clinical use: there is no consensus on optimal fecal sample collection and storage protocols, bioinformatics pipelines, reference databases, or clinically validated thresholds for "normal" vs. "dysbiotic" microbiota composition [5]. The most clinically actionable microbiocenosis assessment currently available for IBS practice is the combination of quantitative culture for clinically relevant dysbiosis markers (C. difficile, SIBO by glucose hydrogen breath test, quantification of Bifidobacterium and Lactobacillus by qPCR) supplemented by fecal calprotectin and secretory IgA as functional markers of mucosal immune status—a panel that can guide probiotic selection and antibiotic treatment decisions without requiring full metagenomic sequencing [3].

Fecal microbiota transplantation (FMT), which transfers the entire microbiome of a healthy donor to an IBS patient, represents the most comprehensive microbiocenosis restoration strategy and has produced response rates of 65–89% for global IBS symptom improvement in small randomized trials, with sustained effects at 12-month follow-up in the subset of patients who achieve donor microbiome engraftment [2]. However, the optimal donor selection criteria, delivery route (colonoscopic vs. capsule), dosing frequency, and long-term safety profile remain incompletely characterized, and FMT for IBS is not yet endorsed by major clinical guidelines pending larger, controlled trials. The identification of donor microbiome characteristics that predict successful IBS treatment—particularly high F. prausnitzii abundance and alpha-diversity in the donor—points toward a future of precision donor matching that may substantially improve FMT success rates in IBS [5].

5. CONCLUSION

This systematic review has established that intestinal microbiocenosis is profoundly and reproducibly altered in IBS patients, with a consistent dysbiotic signature characterized by reduced Faecalibacterium prausnitzii, Bifidobacterium, Lactobacillus, and Akkermansia muciniphila, elevated Proteobacteria and Ruminococcus gnavus, reduced alpha-diversity, and subtype-specific Firmicutes/Bacteroidetes ratio changes. These dysbiotic alterations are mechanistically linked to IBS symptoms through SCFA deficiency-mediated barrier dysfunction, serotonin dysregulation, immune activation, and perturbation of the brain-gut-microbiome axis, providing a compelling pathophysiological foundation for microbiota-targeted therapeutic strategies.

Among available diagnostic tools, 16S rRNA gene sequencing and shotgun metagenomics provide superior sensitivity and comprehensiveness for microbiocenosis characterization compared to culture-based methods, but their integration into routine clinical practice requires standardization of pre-analytical and bioinformatic protocols. In current clinical practice, targeted qPCR for key dysbiosis biomarkers (F. prausnitzii, Bifidobacterium) combined with functional markers (fecal SCFA profile, calprotectin, secretory IgA) and hydrogen/methane breath testing for SIBO represents the most practical approach to actionable microbiocenosis assessment in IBS patients.

Evidence-based microbiota-targeted therapies—rifaximin for IBS-D, multi-strain probiotics (Bifidobacterium and Lactobacillus combinations) for global symptom relief, and low-FODMAP dietary intervention with structured reintroduction—each address distinct microbiological mechanisms and can be selected based on IBS subtype, symptom profile, and the results of microbiocenosis assessment. Future progress in IBS management will depend on the development of validated clinical-grade microbiome diagnostics, precision donor-matching protocols for FMT, and next-generation biotherapeutics (including engineered bacterial consortia and postbiotic SCFA formulations) that restore the specific functional deficits of the IBS-associated dysbiome. The ultimate goal—individualized microbiota-based profiling and precision treatment of IBS—is now scientifically feasible and clinically within reach.

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