RESEARCH PAPER

Comparative Metagenomics of the Gut Microbiome in IBD vs IBS Patients

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Received: 10th Mar, 2026 | Revised: 18th Mar, 2026 | Accepted: 22nd Mar, 2026 | Available Online: 28th Mar, 2026

Abstract

Background: Inflammatory bowel disease and irritable bowel syndrome commonly present with overlapping gastrointestinal symptoms but differ substantially in their underlying mechanisms.

Objective: To compare the gut microbiome composition and functional metagenomic profiles of patients with inflammatory bowel disease and irritable bowel syndrome.

Methodology: This comparative cross-sectional study was conducted at Allama Iqbal Teaching Hospital, Dera Ghazi Khan from June 2024 to June 2025 and included 170 patients equally divided into inflammatory bowel disease (n=85) and irritable bowel syndrome (n=85) groups.

Results: The inflammatory bowel disease group had significantly higher mean C-reactive protein (18.6 ± 9.5 vs. 4.2 ± 2.1 mg/L), erythrocyte sedimentation rate (32.4 ± 13.7 vs. 13.8 ± 6.5 mm/hour), and fecal calprotectin levels (416.8 ± 154.6 vs. 64.3 ± 32.8 µg/g) than the irritable bowel syndrome group; all $p<0.001$. Microbial diversity was significantly lower in inflammatory bowel disease, with reduced Shannon (3.28 ± 0.64 vs. 4.12 ± 0.58) and Simpson indices (0.73 ± 0.09 vs. 0.84 ± 0.07); both $p<0.001$. Patients with inflammatory bowel disease had lower Firmicutes abundance (36.8 ± 10.4% vs. 49.7 ± 11.3%) and higher Proteobacteria abundance (21.4 ± 7.8% vs. 9.3 ± 4.5%); both $p<0.001$.

Conclusion: It is concluded that inflammatory bowel disease is associated with markedly reduced microbial diversity, depletion of beneficial bacteria, enrichment of pro-inflammatory taxa, and altered microbial metabolic pathways compared with irritable bowel syndrome.

Keywords: Inflammatory bowel disease; irritable bowel syndrome; gut microbiome; metagenomics

How to cite this article: Rashid Karim1 Muhammad Usman Khan2 Nisar Ul Hassan3 Ajab Khan4 Sultan Zeb Khan5 Muhammad Muslim Khan6. Comparative Metagenomics of the Gut Microbiome in IBD vs IBS Patients. Int J Drug Deliv Technol. 2026;16(82s):768-773. DOI: 10.25258/ijddt.16.82s.86.

Introduction

IBD and IBS are chronic gastrointestinal conditions that share many clinical characteristics such as abdominal pain, a change in bowel habits, bloating, and a dramatic impact on quality of life, but differ in their pathophysiological mechanisms [1]. Irritable bowel syndrome (IBS) is a functional gastrointestinal disorder without any obvious structural or inflammatory changes, while inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, is associated with chronic immune-

mediated intestinal inflammation and progressive mucosal injury [2]. The importance of distinguishing these disorders lies in the fact that the approaches to treatment, prognosis, and outcomes vary greatly. Recent studies highlight the importance of gut microbes in regulating gut immune responses and homeostasis [3]. The human gut microbiome is a collection of trillions of bacteria, viruses, fungi, and archaea that all have a role in maintaining proper nutrient metabolism, epithelial barrier function, immunity against pathogenic organisms and disease resistance [4]. The term dysbiosis is used to describe changes in the composition and function of microbes and has been linked to the etiology and progression of various gastrointestinal diseases, especially those of inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS) [5]. Metagenomic sequencing has become a powerful new tool to characterise the gut microbiome in a more comprehensive way by studying the DNA of all the microorganisms in the gut without the need to do culture based analysis [6]. Although 16S ribosomal RNA sequencing is commonly used, shotgun metagenomic sequencing allows scientists to determine taxonomic resolution and to discover new genes, metabolic pathways, antimicrobial resistance proteins, and functional potential in addition to the microbes themselves [7]. This has greatly enhanced the understanding of the interactions between host and gut microbiome in gastrointestinal diseases.

Consistently, patients with inflammatory bowel disease have shown a decrease in microbial diversity, a decrease in beneficial commensal organisms such as Faecalibacterium prausnitzii and Roseburia spp., and enrichment of pro-inflammatory bacteria, such as adherent-invasive Escherichia coli and other Proteobacteria [8]. These microbial changes play a role in epithelial barrier dysfunction, abnormal immune activation, overproduction of cytokines and chronic intestinal inflammation [9]. On the contrary, microbial alterations, with decreased and specific bacterial diversity, fermentation, bile acid metabolism and visceral hypersensitivity have been linked to IBS [10]. Comparative metagenomic analysis can be used to discover microbial signatures for specific diseases that could enhance the accuracy of diagnosis and give clues to disease mechanisms [11]. In addition, understanding of microbial metabolism can help to devise specific strategies for therapeutic interventions such as probiotics, prebiotics, diet changes, fecal microbiota transplantation, and microbiome-based precision medicine [12]. Identifying the microbial differences between IBD and IBS could therefore help to tailor diagnosis and treatment. Inflammatory bowel disease (IBD) has been shown to have a significantly greater diversity of microbes, distinct bacterial composition, and metabolic pathways when compared to irritable bowel syndrome (IBS) patients in previous studies [13]. The results have been inconsistent, however, because of the differences in study populations, sequencing methods, diet and geographical location.

Objective

To compare the gut microbiome composition and functional metagenomic profiles of patients with inflammatory bowel disease and irritable bowel syndrome.

Methodology

This comparative cross-sectional study was conducted at Allama Iqbal Teaching Hospital, Dera Ghazi Khan from June 2024 to June 2025 and included 170 patients diagnosed with either inflammatory bowel disease (IBD) or irritable bowel syndrome (IBS). Patients with confirmed diagnosis of inflammatory bowel disease (Crohn's disease or ulcerative colitis based on clinical, endoscopic, histopathological and radiological findings) or irritable bowel syndrome (Rome IV criteria) aged 18-70 years were included. The participants were eligible to be included if they were not taking any antibiotics, probiotics, prebiotics or fecal microbiota transplantation in the last four weeks and gave written informed consent. Exclusion criteria included gastrointestinal malignancy, celiac disease, infectious colitis, recent gastrointestinal surgery, pregnancy, chronic liver disease, chronic kidney disease, HIV infection, immunodeficiency disease, and systemic autoimmune disease, and incomplete clinical or sequencing data within 4 weeks of recent hospitalization.

Data Collection

Eligible participants were enrolled after approval of Institutional Ethical Review Committee and written informed consent through a non-probability consecutive sampling technique. All baseline demographic and clinical data (age, gender, BMI, smoking status, disease duration, medication history, disease subtype, disease activity, dietary habits, and relevant laboratory parameters such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR) and fecal calprotectin) were entered into a standardized data collection form. Stool samples were obtained under standard conditions, before starting or changing medical treatment. Commercial validated microbial DNA extraction kits were used following manufacturer instructions. Libraries were prepared

and evaluated for quality before shotgun metagenomic sequencing on an Illumina sequencing platform. The raw sequencing reads were quality filtered, host DNA removed, taxonomically classified and pathway functional analyses performed on these sequences, following standard bioinformatics pipelines. The alpha diversity indices (Shannon and Simpson), beta diversity, microbial taxonomic composition, relative abundance of bacterial phyla and genera, antimicrobial resistance genes and microbial metabolic pathways were analysed. The main result was the comparison of microbial diversity and taxonomic structure of the gut in patients with IBS and IBD. Secondary outcomes ranged from microbial functional pathways to abundance of beneficial and pathogenic taxa, as well as associations with inflammatory biomarkers, and identification of microbial predictors distinguishing inflammatory bowel disease from irritable bowel syndrome.

Statistical Analysis

The data were analyzed with SPSS version 26.0. The continuous variables were presented as mean ± SD or median (25th–75th %iles) and the categorical variables were presented as frequencies and percentages. The difference between continuous variables was compared by the independent t-test while the difference of categorical variables was analyzed by Chi-square test. Three multivariable logistic regression models were built to determine microbial features that were independently associated with the presence of IBD. A p value of < 0.05 was deemed as statistically significant.

Results

The study included 170 patients equally divided between the IBD and IBS groups. The mean age was 39.8 ± 12.1 years in the IBD group and 41.2 ± 11.4 years in the IBS group (p=0.438). Males comprised 46 (54.1%) and 42 (49.4%) patients, respectively (p=0.542). Mean BMI was lower in patients with IBD than in those with IBS (24.8 ± 4.3 vs. 26.2 ± 4.6 kg/m²; p=0.041). Mean disease duration was 5.8 ± 3.2 versus 4.9 ± 2.8 years (p=0.053), while current smoking was reported by 18 (21.2%) and 14 (16.5%) patients (p=0.430).

Table 1. Baseline demographic and clinical characteristics of the study participants (n=170)

Characteristic	IBD (n=85)	IBS (n=85)	p-value
Age (years), Mean ± SD	39.8 ± 12.1	41.2 ± 11.4	0.438
Male gender	46 (54.1%)	42 (49.4%)	0.542
BMI (kg/m²), Mean ± SD	24.8 ± 4.3	26.2 ± 4.6	0.041
Disease duration (years), Mean ± SD	5.8 ± 3.2	4.9 ± 2.8	0.053
Current smokers	18 (21.2%)	14 (16.5%)	0.430
CRP (mg/L), Mean ± SD	18.6 ± 9.5	4.2 ± 2.1	<0.001
ESR (mm/hr), Mean ± SD	32.4 ± 13.7	13.8 ± 6.5	<0.001
Fecal calprotectin (µg/g), Mean ± SD	416.8 ± 154.6	64.3 ± 32.8	<0.001

The mean Shannon diversity index was 3.28 ± 0.64 in IBD and 4.12 ± 0.58 in IBS, while the Simpson index was 0.73 ± 0.09 and 0.84 ± 0.07, respectively; both p<0.001. Firmicutes abundance was lower in IBD (36.8 ± 10.4% vs. 49.7 ± 11.3%; p<0.001), whereas Proteobacteria were markedly higher (21.4 ± 7.8% vs. 9.3 ± 4.5%; p<0.001). Actinobacteria were also slightly higher in IBD (6.8 ± 2.9% vs. 5.6 ± 2.5%; p=0.004).

Table 2. Comparison of gut microbiome diversity and major bacterial taxa

Variable	IBD (n=85)	IBS (n=85)	p-value
Shannon diversity index	3.28 ± 0.64	4.12 ± 0.58	<0.001
Simpson diversity index	0.73 ± 0.09	0.84 ± 0.07	<0.001
Firmicutes (%)	36.8 ± 10.4	49.7 ± 11.3	<0.001
Bacteroidetes (%)	31.5 ± 8.9	34.2 ± 9.1	0.056
Proteobacteria (%)	21.4 ± 7.8	9.3 ± 4.5	<0.001
Actinobacteria (%)	6.8 ± 2.9	5.6 ± 2.5	0.004
Firmicutes/Bacteroidetes ratio	1.18 ± 0.46	1.47 ± 0.53	0.001

Faecalibacterium prausnitzii abundance was lower in IBD than IBS (4.8 ± 2.3% vs. 9.6 ± 3.1%), as were Roseburia species (2.9 ± 1.4% vs. 5.8 ± 2.0%) and

Bifidobacterium species (3.6 ± 1.8% vs. 5.2 ± 2.1%); all p<0.001. In contrast, Escherichia coli was more abundant in IBD (10.7 ± 4.2% vs. 4.3 ± 2.1%; p<0.001). Short-chain fatty acid biosynthesis activity was reduced in IBD (24.1 ± 6.5% vs. 34.8 ± 7.2%), whereas lipopolysaccharide biosynthesis activity was increased (17.8 ± 5.6% vs. 10.2 ± 4.3%); both p<0.001.

Table 3. Relative abundance of selected microbial genera and functional pathways

Variable	IBD (n=85)	IBS (n=85)	p-value
Faecalibacterium prausnitzii (%)	4.8 ± 2.3	9.6 ± 3.1	<0.001
Roseburia spp. (%)	2.9 ± 1.4	5.8 ± 2.0	<0.001
Escherichia coli (%)	10.7 ± 4.2	4.3 ± 2.1	<0.001
Bifidobacterium spp. (%)	3.6 ± 1.8	5.2 ± 2.1	<0.001
Short-chain fatty acid biosynthesis pathway (%)	24.1 ± 6.5	34.8 ± 7.2	<0.001
Lipopolysaccharide biosynthesis pathway (%)	17.8 ± 5.6	10.2 ± 4.3	<0.001

Increased Proteobacteria abundance increased the odds by 4.36 times (95% CI: 2.01–9.44; p<0.001), while reduced Shannon diversity increased the odds by 3.82 times (95% CI: 1.78–8.19; p<0.001). Reduced Faecalibacterium prausnitzii was associated with 3.11-fold higher odds (95% CI: 1.46–6.63; p=0.003), elevated CRP with 2.64-fold higher odds (95% CI: 1.21–5.76; p=0.015), and reduced short-chain fatty acid pathway activity with 2.53-fold higher odds of IBD (95% CI: 1.17–5.46; p=0.018).

Table 4. Multivariable logistic regression analysis identifying predictors of inflammatory bowel disease

Variable	Adjusted Odds Ratio (95% CI)	p-value
Reduced Shannon diversity index	3.82 (1.78–8.19)	<0.001
Increased Proteobacteria abundance	4.36 (2.01–9.44)	<0.001
Reduced Faecalibacterium prausnitzii	3.11 (1.46–6.63)	0.003
Elevated fecal calprotectin	5.28 (2.42–11.53)	<0.001
Elevated CRP	2.64 (1.21–5.76)	0.015
Reduced short-chain fatty acid pathway activity	2.53 (1.17–5.46)	0.018

Discussion

In the present study, the gut microbiome composition of the patients with IBS and IBD was compared using shotgun metagenomic sequencing of the microbiome. Demographic data between the two groups were similar in age, gender, disease duration and smoking status. Patients with IBD, however, had a significantly lower mean BMI (24.8 ± 4.3 vs. 26.2 ± 4.6 kg/m²) and had significantly higher inflammatory markers such as CRP, ESR and fecal calprotectin levels. These results reflect the chronic inflammatory state seen in the presence of IBD and not in that of IBS. Inflammatory biomarkers were also significantly higher, and BMI was lower in patients with inflammatory bowel disease than in patients with IBS in previous studies [14]. The most significant result was the huge decrease in diversity of microbes found in people with inflammatory bowel disease, that was seen in this study. Both the Shannon diversity index (3.28 ± 0.64 vs. 4.12 ± 0.58) and Simpson diversity index (0.73 ± 0.09 vs. 0.84 ± 0.07) were significantly lower in IBD than IBS. A decrease in microbial diversity is believed to be a hallmark of intestinal dysbiosis and reflects the depletion of beneficial commensal microorganisms that help to maintain intestinal homeostasis. Earlier studies have consistently identified a significantly reduced diversity of microbes in inflammatory bowel disease patients than in healthy people and IBS patients [15]. Bacterial phyla analysis showed significant differences in microbial communities between patients with inflammatory bowel disease. The abundance of Firmicutes decreased significantly, while Proteobacteria increased significantly for the IBS group compared with the other groups. Firmicute / Bateroidetes ratio was also significantly decreased in the IBD group and there was no significant difference in abundance of Bacteroidetes between the groups. Proteobacteria expansion is thought to be a reflection of microbial imbalance and linked to intestinal inflammation and epithelial barrier disruption. Inflammation-related bowel disease has also been shown to be characterized by a decrease in Firmicutes and an enrichment of Proteobacteria in previous studies [16]. Both beneficial and pathogenic microorganisms were depleted at the genus level in the IBD patients, with Faecalibacterium prausnitzii, Roseburia species and Bifidobacterium species

significantly decreased, while there were significant increases in the abundance of Escherichia coli. The findings are biologically plausible since Faecalibacterium prausnitzii and Rosburia species are key producers of short-chain fatty acids (SCFAs) such as butyrate, which help maintain intestinal barrier function and have anti-inflammatory activity. In contrast, high levels of Escherichia coli could be linked to the maintenance of mucosal inflammation by triggering innate immune pathways. The same deletion of butyrate-producers and proliferation of Escherichia coli has also been shown in IBD patients in previous studies [17].

Two disorders had distinct metabolic differences according to functional metagenomic analysis. Patients with inflammatory bowel disease had a decrease in short-chain fatty acid biosynthesis pathways and an increase in lipopolysaccharide biosynthesis. These functional changes indicate that there is a reduction of the production of beneficial microbial metabolites and an increase in inflammatory signaling in the intestinal microbiome. The same study revealed that the decrease in the amount of short-chain fatty acids and the increase in the amount of endotoxin-related metabolic pathways are linked to intestinal inflammation and disease in inflammatory bowel disease [18]. Elevated abundance of the Proteobacteria, reduced microbial diversity, decreased abundance of Faecalibacterium prausnitzii, increased CRP, and decreased short-chain fatty acid biosynthesis were also highly associated with inflammatory bowel disease independently of elevated fecal calprotectin when analyzed by multivariable logistic regression. This study suggests inflammatory biomarkers and microbiomic signatures are independent, and both distinguish inflammatory bowel disease from irritable bowel syndrome. Inflammatory bowel disease has also been associated with several other biomarkers, such as faecal calprotectin, microbial dysbiosis, decreased bacterial diversity and loss of bacteria that produce anti-inflammatory compounds, which are also reported to be amongst the most reliable [19]. The noted changes in the microbial community could contribute to the pathogenesis of chronic intestinal inflammation in inflammatory bowel diseases in several ways. A decrease in beneficial commensal bacteria may lead to defects in epithelial barrier integrity and loss of production of anti-inflammatory metabolites, while the increase of Proteobacteria and Escherichia coli can trigger activation of pro-inflammatory immune pathways due to increased production of lipopolysaccharide and translocation of bacteria. Similarly, previous studies have suggested that imbalances between beneficial and harmful microbes play an important role in chronic inflammation and disease activity in inflammatory bowel disease [20]. This cross-sectional study was limited in its ability to analyze the causal links between the alterations in gut microbiome and the development of the disease. The results might not be generalizable due to the single-center design and small sample size. What's more, the diet consumed and other environmental factors that could potentially affect the microbiome of the gut were not fully controlled.

Conclusion

It is concluded that patients with inflammatory bowel disease exhibit significantly reduced gut microbial diversity, depletion of beneficial bacterial taxa, enrichment of pro-inflammatory microorganisms, and altered microbial metabolic pathways compared with patients with irritable bowel syndrome. Elevated fecal calprotectin, increased Proteobacteria abundance, reduced microbial diversity, and impaired short-chain fatty acid biosynthesis were independently associated with inflammatory bowel disease. Comparative metagenomic profiling may serve as a valuable tool for differentiating inflammatory bowel disease from irritable bowel syndrome and may facilitate the development of microbiome-targeted diagnostic and therapeutic strategies.

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