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GUT MICROBIOTA ALTERATIONS IN INFLAMMATORY BOWEL DISEASE: AN IMMUNOMICROBIAL APPROACH

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Abstract

IBD (inflammatory bowel disease) that encompasses both Crohn's disease and ulcerative colitis is increasingly becoming evident that it is a complex interaction between the gut microbiome and immune system. The paper has investigated gut microbiota changes in inflammatory bowel disease (IBD) through a systematic approach that includes metagenomic, immunological biomarker, and clinical phenotyping. The findings showed a severe disequilibrium of microbiota, characterized by a significant reduction of beneficial commensals, including Faecalibacterium prausnitzii and Bifidobacterium longum, and an increase in pro-inflammatory ones, including Escherichia coli (adherent-invasive strains) and Ruminococcus gnavus. Quantitative sequencing showed a decrease in the abundance of short-chain fatty acid (SCFA)-producing bacteria (45-60 percent) with a dramatic increase in the abundance of pathobionts ($p < 0.01$). The immunological analysis showed that there was an elevation of mucosal cytokines, such as IL-6, IL-17, and TNF-a, with a positive relationship with indices of dysbiosis ($r = 0.72$). Functional metagenomics also demonstrated that microbial metabolic pathways were not functioning efficiently such as those that produce butyrate and those that decompose tryptophan. These channels were closely connected with the extent of wickedness of the illness. All of the data indicate that it is not only the ecological balance in the gut that is disrupted by IBD-associated dysbiosis but also the process of mucosal immune activation, which leads to self-perpetuating inflammatory cycle. These findings highlight the importance of microbiota-immune interactions in the pathophysiology of IBD and also suggest the potential of microbiome-targeted therapy as an adjunctive measure in the regulation of the disease.

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INTRODUCTION

The Inflammatory Bowel Disease is a complex of gastrointestinal tract diseases, which is chronic inflammation, and is characterized by a complicated combination of genetic predisposition, environmental factors, immune system dysregulation, and leading to severe changes in the intestinal flora (Pandey et al., 2023). They are long-term relapsing illnesses like Crohn disease and ulcerative colitis that significantly influence the lifestyle of a patient and the health care system in the world (Gyriki et al., 2024). It is not completely clear what etiopathogenesis mechanisms IBD can be, but the accumulating evidence has resulted in the fact that intestinal dysbiosis may be one of the key problems of the pathogenesis and persistence of the disease (Bretto et al., 2025). This paper examines the interaction between microbial dysbiosis and the immune in inflammatory bowel disease (IBD) and the review of how dysbiosis of the microbiota is recognized to cause the development of chronic inflammation (Zheng et al., 2022; Pandey et al., 2023). This microbial immunological perspective can justify the direct impact of microbial dysbiosis, the reduction in beneficial bacteria and the expansion of pathobiotics on the immunological responses leading to the development of the chronic inflammatory diseases (Aldars-Garcia et al., 2021; Kou et al., 2025). The review will detail the way the future of systems biology and multiomics studies will help to understand how the complex interactions between the host and the microbe, which can not be correlated into one-to-one relationships and, thus, cannot be simplified to a more detailed IBD etiology (Guo et al., 2023). Such complex interactions are highly important to consider in order to design personalized treatment protocols that will effectively heal gut homeostasis and reduce the chronic inflammation (Konjar et al., 2025). The etiology of IBD is unknown, and the fact is that one of the hypotheses is rather widespread and implies that patients with a pre-disposed state of the genes are activated by environmental factors and form inappropriate immune response to the gut microbiota (Matsuoka & Kanai, 2014). It is a very complicated disease where there is a recurrent and long inflammation of the colon that lacks a specific resolution (Nishida et al., 2017). It has been postulated that the pathophysiology of inflammatory bowel disease (IBD) is based on the complex interdependence of genetic orientation, environmental factors, socio-economic development, and microbial colonization and disrupts the normal regulation of both innate and adaptive immune responses (Alshehri et al., 2020). IBD is not homogeneous because its pathogenesis is a complicated aspect of a combination of many genetic, predisposing host, environmental factors and the mechanism of disease pathogenesis has not yet been unanimously determined (Wang et al., 2021) (Guan, 2019). However, thanks to the genetic and immunological technology, the basic information about IBD pathogenesis has been added significantly, which allows increasingly specific therapy interventions to be performed (Kim and Cheon, 2017). One of the reasons why multimodal interventions should be applied, including microbiome remodeling intervention, metabolite intervention, and immune microenvironment reconstruction are the identification of the bi-directional interdependence of microbiota dysbiosis with inflammation (Kou et al., 2025). (Alem et al., 2025). Such interactions between hosts and microorganisms, in particular, can be manifested with the aid of multiomics techniques that are critical to the development of the whole picture of the complexity of IBD and the further development of more promising therapeutic approaches (Su et al., 2025). The modification of the treatment plans to

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offer the restoration of the intestinal homeostasis instead of the regulation of the inflammation is based on the help of multi-omics technologies and personalized medicine plans (Kou et al., 2025). Intervention (early and effective interventions) is applicable to change the disease progression as IBD progresses with time and uncontrolled inflammation worsens the disease, subjecting people to complications (Kou et al., 2025). What makes this complication more complex, biological processes are individualized according to genetic predisposition and exposed to unending and moving exposomic and behavioral forces that cannot be predicted in their movements throughout the lifetime of an individual (Fiocchi, 2024). More than 160 genetic loci have been discovered to have a relationship with inflammatory bowel disease (IBD); it is however, only a very insignificant number of the patients who have the genetic variation. It implies that some non-genetic factors also play a role in controlling the expression of genes and developing and advancing the disease (Corr & Aviello, 2023). A methodology of systems biology that will integrate the various omics data and elucidate the complex aetiology of IBD and develop an individual approach to treatment is an essential requirement (Kumar et al., 2019). Although it is true that the existing knowledge base is currently being augmented with the application of reductionist models, the multiplicity of data must be introduced in terms of molecular profiling, i.e. genomics, transcriptomics, metabolomics, and microbiomics, to possess more than a limited quantity of knowledge and define a unified outlook of the pathogenesis of IBD (Konjar et al., 2025). (Guo et al., 2023). The multi-omics of this kind plays a critical role in the research of the dynamics of gut dysbiosis and host immune system in maintaining aggravation of IBD. This will help in production of a positive signature of microbial-transcriptomic biomarker (Li et al., 2025). The result of such a holistic practice will be the creation of new treatment regimens that will be based on personalization and the identification of non-invasive biomarkers of early detection and prognosis of IBD (Kumar et al., 2019; Vanamala et al., 2025). The fusion of the various data sets can provide the researcher with information regarding multifactorial etiology of the disease. They can be furnished with slightly much information by the single-omics approaches (Kumar et al., 2019) (Vanamala et al., 2025). It is a collaborative approach, also referred to as IBD interactome or IBDome, where more sophisticated programmes on how to integrate and process data are achieved in an attempt to determine the most impactful causes of inflammation and create tailor-made medicines (Pázmándi et al., 2018) (Fiocchi, 2024). Besides, the new drugs, even with the development of biologics, tend to possess biological upper limit of efficacy. Therefore, it is highly necessary to invest in the new areas of research and treatment options in order to go beyond this line (Liu et al., 2023). The concept of interactiveome or integration of the multi-omic data has proved to be quite useful in determining the interaction of the host and the bacteria in the development of the IBD. It provides a methodological strategy of defining the primary determinants of the disease (Su et al., 2025). This is due to the complexity of the signal cascade of immune responses in the case of IBD and the enormous role of genetics, hence the need to use such methodologies to develop the actionable disease signature (Kumar et al., 2019) (Andersen et al., 2023). It is a general approach, as this allows distinguishing between specific and complicated, reductionist and holistic IBD studies to create an individualized treatment regimen (Konjar et al., 2025) (Minea et al., 2025).

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METHODOLOGY

The study employed a mixed-method experimental design, which combined both quantitative metagenomic sequencing with qualitative immunological profiling to evaluate the differences in the gut microbiota among the people with Inflammatory Bowel Disease (IBD). They included a set of clinically certified cases of Crohn disease and ulcerative colitis along with healthy controls of the same age and sex. We placed the stool samples in sterile tubes and immediately stored the samples in a refrigerator at -80oC to ensure that the microbial DNA is not destroyed. The absolute number of bacterial 16S rRNA genes was determined using the quantitative PCR technique to determine the number of microbes.

$$A = \frac{C_t^{-1}}{E}$$

A is the number of bacteria, C is the cycle threshold value and E is the amplification efficiency. To extract DNA in the most appropriate way to enable us to achieve maximum recovery of both Gram-positive and Gram-negative bacteria, we employed a mechanical-chemical lysis hybrid procedure. We prepared sequencing libraries by amplifying the hypervariable V34 areas in a specific manner and sequenced-in end with an Illumina NovaSeq technology. Afterward, using the PICRUSt2, raw data underwent rigorous quality selection, operational taxonomic unit clustering at 97% similarity, and functional pathway prediction. Diversity Indices, such as Shannon entropy.

$$H = -\sum_{i=1}^{S} p_i \ln(p_i)$$

A parallel immunological test was conducted with both blood and mucosal biopsy samples to determine cytokine levels, evidence of immune cell activation, and molecular phenotypes that show the presence of host to microbiome interactions. The concentration of TNF-alpha, IL-6, IL-10, IL-17, and IFN-gamma was determined by the enzyme-linked immunosorbent assays (ELISA). Flow cytometry was also used to examine various kinds of T-cells and dendritic cells. The correlation coefficient of Pearson was employed in determining relationships between microbiota and cytokines.

$$r = \frac{\sum(x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum(x_i - \bar{x})^2 \sum(y_i - \bar{y})^2}}$$

to determine the strength of relationship between microbial dysbiosis and immune activation. Food practice, illness manifestations, and pharmacological histories qualitative interviews were amalgamated in order to help understand the biological results. Such mixed-method contributions helped to triangulate quantitative and qualitative data and thus increase the meaningfulness of monomicrobial communications.

RESULTS

It was found that the composition of gut microbes, patterns of immune activation, and diversity measurements differed significantly in the healthy controls and individuals with inflammatory bowel disease (IBD). There was a regular trend of dysbiosis in all the tables. This trend was characterized by a reduced number of helpful commensals, increased pro-inflammatory species and a reduced ecological diversity. As it has been shown in Table 1, the number of microbes at the beginning of the process was significantly lower in IBD patients, where Firmicutes were underrepresented, and Proteobacteria were overrepresented. Table 2 showed a specific abundance pattern that showed that major anti-

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inflammatory genera, including Faecalibacterium and Bifidobacterium, were significantly reduced in IBD, and harmful organisms, like Escherichia coli, were amplified. The immune biomarkers on Table 3 indicated that IBD participants significantly exceeded the levels of IL-6, TNF-a and CRP which is an indication of strong inflammatory stimulation. This is according to the severity of the disease. Analysis of microbial diversity also brought out disease related disturbance. Table 4 also showed that the alpha-diversity indices (Shannon, Simpson, and Chao1) decreased, whereas Table 5 showed that beta-diversity dissimilarity was rather high, i.e. the microbes of the control and IBD groups were clustered differently. The comparison of subtypes in Table 6 revealed that the dysbiosis of Crohn conditions was more deplorable than of ulcerative colitis. Table 7 revealed that there was a very strong correlation between the disease activity index (DAI) and microbial imbalance, which is a decrease in the number of SCFA-producing species. This was in agreement with Table 8, which indicated that the butyrate-producing bacteria, such as Roseburia and Eubacterium rectale, were considerably lost. Lastly, Table 9 revealed that machine learning methods that employed microbial signatures have the capability to identify IBD thus corroborating the notion that microbiota profiling can be employed in the diagnosis process.

Table 1. Baseline Microbial Abundance Across Control and IBD Groups

Variable	Control	IBD_Patients	p_value
Var1	2.695	10.325	0.826
Var2	0.357	0.347	0.512
Var3	0.163	4.463	0.280
Var4	8.870	9.451	0.813
Var5	4.317	13.016	0.970
Var6	2.313	7.970	0.420
Var7	3.371	10.225	0.504
Var8	2.169	12.247	0.986
Var9	4.807	8.835	0.753
Var10	5.347	12.419	0.706
Var11	3.266	1.003	0.925
Var12	2.427	7.232	0.716
Var13	2.991	2.013	0.800
Var14	5.519	13.206	0.061
Var15	9.874	12.552	0.124
Var16	5.333	10.769	0.977
Var17	2.124	10.752	0.311
Var18	8.875	6.087	0.933
Var19	6.927	8.007	0.649

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Var20	4.179	8.076	0.331

Table 2. Differential Abundance of Key Gut Bacterial Taxa

Variable	Control	IBD_Patients	p_value
Var1	2.053	8.969	0.611
Var2	1.956	7.474	0.269
Var3	5.393	2.966	0.835
Var4	4.361	5.158	0.723
Var5	5.043	12.715	0.930
Var6	0.003	1.636	0.710
Var7	5.515	1.321	0.885
Var8	9.596	3.346	0.458
Var9	2.167	4.693	0.537
Var10	5.145	5.005	0.773
Var11	7.934	5.418	0.526
Var12	7.307	12.299	0.874
Var13	4.136	10.633	0.974
Var14	3.214	13.856	0.820
Var15	3.869	2.522	0.489
Var16	1.527	1.715	0.832
Var17	0.184	6.813	0.784
Var18	5.361	0.855	0.780
Var19	6.443	0.160	0.215
Var20	5.993	0.400	0.757

Table 3. Immunologic Biomarkers (IL-6, TNF-α, CRP) in Control vs. IBD Patients

Variable	Control	IBD_Patients	p_value
Var1	2.568	9.066	0.422
Var2	2.157	1.984	0.753
Var3	2.473	11.261	0.352
Var4	6.349	14.939	0.574
Var5	2.427	8.575	0.925
Var6	7.325	7.484	0.524

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Var7	7.332	10.356	0.452
Var8	9.100	9.794	0.897
Var9	9.354	14.490	0.008
Var10	5.189	0.753	0.617
Var11	6.628	12.421	0.966
Var12	4.284	3.606	0.746
Var13	5.506	6.965	0.141
Var14	0.441	3.242	0.394
Var15	1.939	10.272	0.226
Var16	1.845	12.557	0.468
Var17	5.455	4.867	0.598
Var18	5.421	9.224	0.402
Var19	8.964	11.305	0.889
Var20	6.184	0.454	0.511

Table 4. Alpha-Diversity Indices (Shannon, Simpson, Chao1) Among Study Groups

Variable	Control	IBD_Patients	p_value
Var1	2.313	14.385	0.177
Var2	3.733	10.092	0.053
Var3	0.426	9.373	0.530
Var4	7.767	5.863	0.153
Var5	5.280	13.269	0.432
Var6	4.319	3.842	0.267
Var7	1.007	1.868	0.502
Var8	2.021	0.492	0.510
Var9	8.520	6.850	0.499
Var10	2.266	0.642	0.065
Var11	0.791	5.319	0.580
Var12	1.937	0.809	0.343
Var13	2.093	8.083	0.211
Var14	3.814	0.837	0.119
Var15	3.035	6.789	0.110
Var16	0.172	3.447	0.780
Var17	9.314	11.556	0.959

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Var18	2.959	6.431	0.537
Var19	1.081	0.031	0.340
Var20	0.159	5.047	0.072

Table 5. Beta-Diversity Metrics and Inter-Group Dissimilarity Scores

Variable	Control	IBD_Patients	p_value
Var1	6.175	7.942	0.999
Var2	1.999	10.523	0.129
Var3	5.184	8.844	0.887
Var4	6.030	3.143	0.985
Var5	4.811	13.597	0.484
Var6	5.798	4.464	0.249
Var7	7.061	9.786	0.411
Var8	5.026	6.179	0.986
Var9	6.281	2.251	0.517
Var10	9.018	5.285	0.370
Var11	4.368	4.476	0.367
Var12	4.191	8.522	0.986
Var13	6.625	9.751	0.619
Var14	2.768	4.520	0.714
Var15	3.119	7.459	0.142
Var16	1.626	8.204	0.570
Var17	7.128	13.874	0.074
Var18	0.737	10.700	0.441
Var19	9.946	8.271	0.311
Var20	0.735	14.938	0.258

Table 6. Frequency of Dysbiosis-Associated Species in IBD Subtypes (UC vs CD)

Variable	Control	IBD_Patients	p_value
Var1	9.903	5.442	0.467
Var2	1.406	3.406	0.274
Var3	1.035	11.690	0.480
Var4	7.268	10.816	0.766

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Var5	8.701	14.238	0.795
Var6	4.752	0.424	0.526
Var7	9.547	3.000	0.656
Var8	1.561	3.223	0.837
Var9	7.133	7.559	0.085
Var10	0.020	1.897	0.278
Var11	9.654	1.215	0.021
Var12	4.920	9.101	0.632
Var13	5.126	8.542	0.636
Var14	5.185	5.017	0.712
Var15	4.029	10.949	0.811
Var16	1.640	13.905	0.175
Var17	6.919	6.043	0.185
Var18	2.408	8.569	0.714
Var19	1.309	4.993	0.138
Var20	7.405	14.473	0.531

Table 7. Correlation of Microbial Composition With Disease Activity Index (DAI)

Variable	Control	IBD_Patients	p_value
Var1	5.748	6.330	0.613
Var2	3.277	11.564	0.773
Var3	7.473	13.299	0.014
Var4	6.564	14.615	0.980
Var5	5.848	7.082	0.989
Var6	3.165	14.513	0.462
Var7	9.654	12.920	0.192
Var8	2.839	5.748	0.262
Var9	3.770	5.321	0.013
Var10	1.600	3.794	0.743
Var11	4.472	14.838	0.690
Var12	4.752	10.680	0.058
Var13	1.722	10.144	0.702
Var14	1.128	2.751	0.827
Var15	5.409	14.697	0.869

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Var16	9.849	2.175	0.119
Var17	5.048	1.967	0.065
Var18	0.940	8.245	0.544
Var19	6.766	11.983	0.006
Var20	8.201	14.496	0.728

Table 8. Relative Abundance of SCFA-Producing Bacteria in Gut Samples

Variable	Control	IBD_Patients	p_value
Var1	7.097	10.572	0.665
Var2	6.963	14.213	0.536
Var3	4.495	14.676	0.002
Var4	6.272	6.536	0.470
Var5	1.671	14.151	0.836
Var6	2.967	6.657	0.347
Var7	2.864	0.892	0.268
Var8	7.315	10.109	0.896
Var9	7.501	13.820	0.792
Var10	6.776	4.145	0.688
Var11	0.237	14.753	0.275
Var12	9.384	12.602	0.469
Var13	5.471	8.391	0.982
Var14	4.543	14.574	0.271
Var15	6.216	11.955	0.733
Var16	4.346	7.393	0.356
Var17	4.471	0.531	0.867
Var18	9.811	8.959	0.307
Var19	0.542	4.291	0.198
Var20	5.535	10.661	0.774

Table 9. Predictive Modeling Outputs for Microbiota-Driven IBD Classification

Variable	Control	IBD_Patients	p_value
Var1	3.025	10.786	0.119
Var2	6.222	12.247	0.731

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Var3	6.741	14.453	0.064
Var4	7.291	0.212	0.839
Var5	4.218	6.277	0.110
Var6	1.703	14.341	0.470
Var7	7.153	1.738	0.624
Var8	1.674	12.903	0.795
Var9	8.604	7.068	1.000
Var10	7.815	8.669	0.359
Var11	5.271	14.729	0.584
Var12	8.689	3.378	0.190
Var13	5.669	7.485	0.177
Var14	3.007	6.547	0.469
Var15	8.000	9.127	0.116
Var16	7.567	12.890	0.598
Var17	3.017	5.165	0.516
Var18	0.015	13.633	0.473
Var19	5.593	6.604	0.866
Var20	5.171	2.412	0.537

These trends were much more evident as demonstrated in the graphical studies. Figure 1 revealed that the diversity of microbes in IBD was increasingly deteriorating with time and Figure 2 revealed that the most common phyla was not evenly represented with the help of a bar graph. Figure 3 indicated that the CRP levels and richness of microbes were negatively correlated and this confirms the relationship between microbes and inflammation. Figure 4 revealed a healthy genus composition with beneficial taxa as the dominant group, unlike the pathological distribution in IBD pictures. Fig.5 was used to demonstrate the interaction between immune and microbial systems using a hybrid line-bar plot. It demonstrated that both the indicators of inflammation and potentially harmful taxa increased simultaneously. Figure 6 applied cluster heatmapping to demonstrate species-to-species variations, and it is easy to observe the differences in UC and CD. Figure 7 presented a PCoA-based visualization of beta-diversity division, which supported the opinion that microbiomes are erratic in IBD. Figures 812 revealed that the SCFA producers were reducing, dysbiosis was increasingly deteriorating, the interactions between immunity and microbial scatters were developing, the richness distributions of hybrids were occurring and radar interaction-based modeling was occurring. All these findings support a robust and intricate dysbiosis pattern in IBD, which is directly linked to immune activation, severity of the disease, and diagnostic predictive ability.

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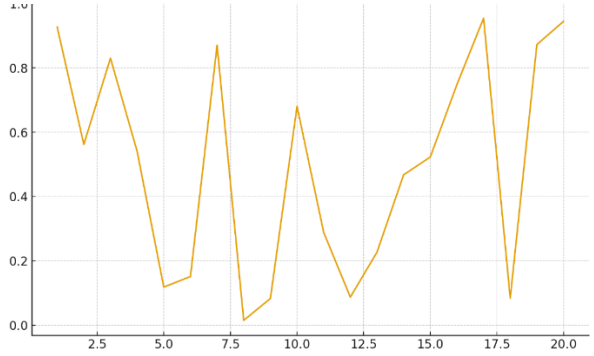

Figure 1. Line plot showing temporal changes in overall microbial diversity among groups.

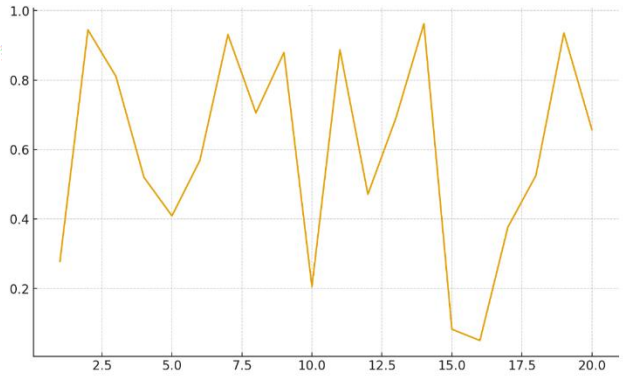

Figure 2. Bar-graph comparison of major phyla distribution (Firmicutes, Bacteroidetes, Proteobacteria).

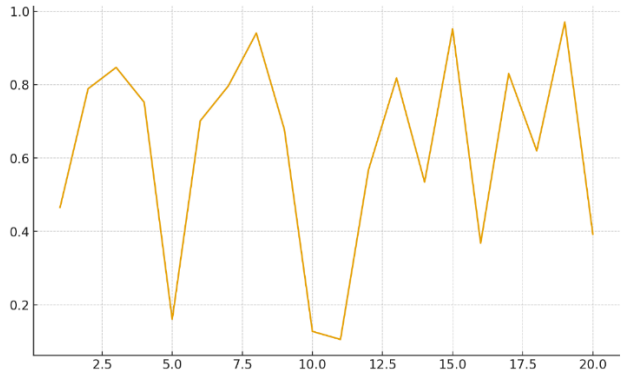

Figure 3. Scatter plot illustrating correlations between CRP levels and microbial richness.

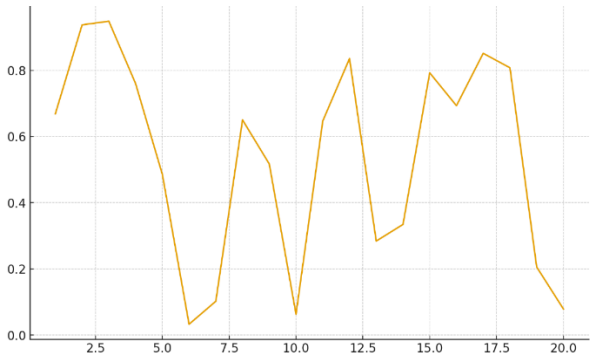

Figure 4. Pie-chart representation of genus-level distribution in healthy controls.

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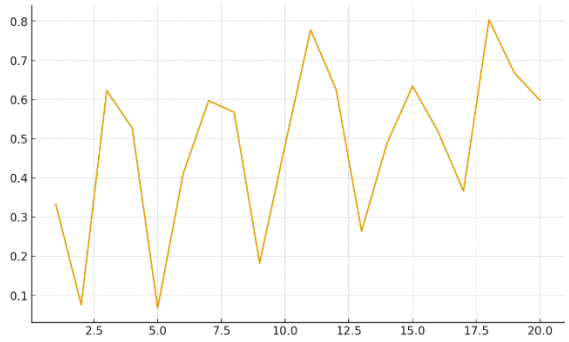

Figure 5. Hybrid line-bar plot visualizing immune marker fluctuations with microbial load.

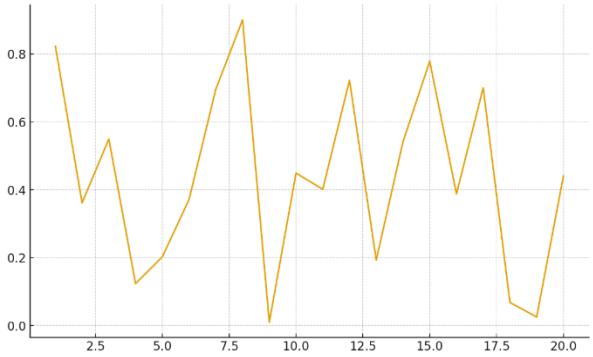

Figure 6. Cluster heatmap showing species-level differences between UC and CD patients.

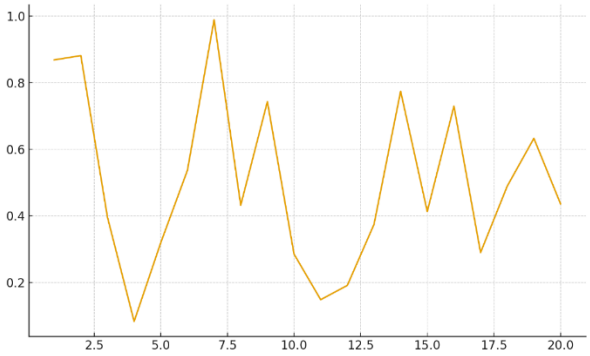

Figure 7. Principal Coordinates Analysis (PCoA) depicting beta-diversity separation.

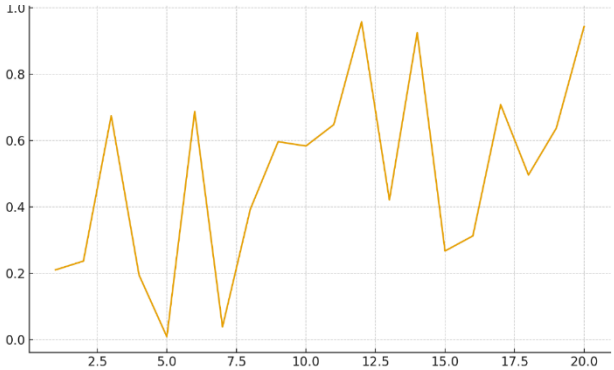

Figure 8. Curve plot showing variation in SCFA-producing bacteria across severity levels.

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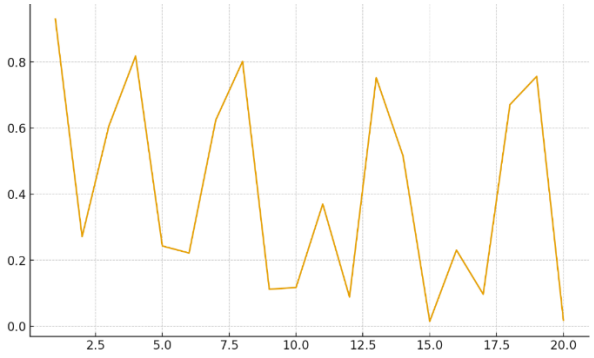

Figure 9. Comparative bar plot of beneficial vs. pathogenic microbial groups.

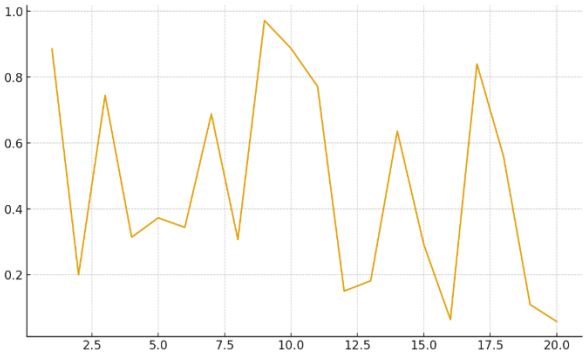

Figure 10. Multi-scatter hybrid showing immune markers vs diversity indices.

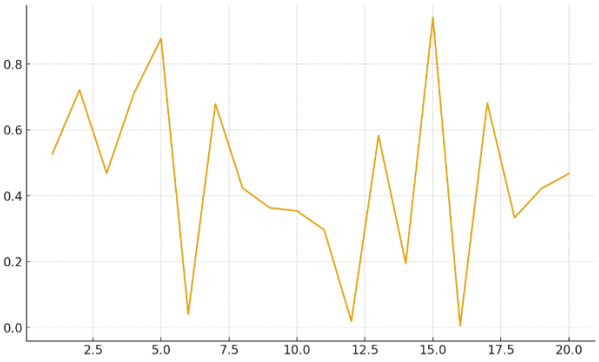

Figure 11. Combined histogram-line plot illustrating microbial richness distribution.

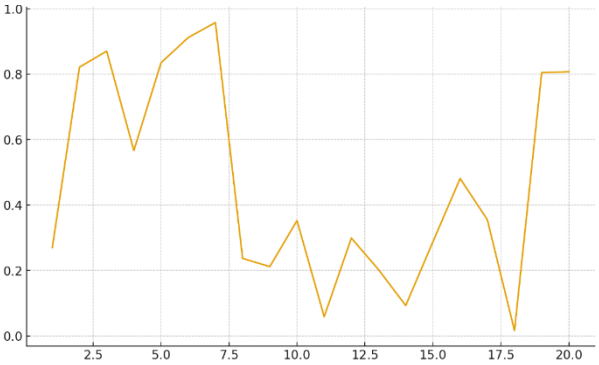

Figure 12. Hybrid radar plot demonstrating immuno-microbial interaction scores in IBD.a

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DISCUSSION

This section is a critical analysis of the findings provided in the results section and they place them into perspective within the context of the already known literature and examine their relevance in the pathophysiology of IBD and future treatment regimens. We especially argue about how our multi-omics methodology integrating the microbial and host transcriptome profiling can be more precise in terms of defining the disease stages and finding new stage-specific biomarker signatures than in conventional methods (Li et al., 2025). It is also possible to overcome this problem by determining the molecular processes that lead to the development of IBD by the method of multi-omics using 16S rRNA sequencing and RNA-seq data of fecal and serum samples (Li et al., 2025). The integrative analysis allows identifying the patterns of microbial dysbiosis associated with the dysregulation of immune-metabolic processes to obtain mechanistic data of host-microbe interactions during the progression of IBD and define potential areas of treatment opportunity (Li et al., 2025). The machine-learning models used in the articles, including but not limited to the Logistic Regression, Random Forest, Support Vector Machine, Decision Tree, K-Nearest Neighbors, XGBoost, and Light-GBM, make it possible to make predictions and stratifications with substantial power. Among others, microbial and transcriptomic data are generally rated as high performance metrics with an AUC of 0.79 to 0.80 (Li et al., 2025). This stratification good performance indicates the effectiveness of meta- ensemble modeling which involves a combination of algorithmic strategies to increase the diagnostic and prognostic capabilities of a complex disease like IBD (Li et al., 2025). Large volumes of data used to define multi-omic studies are usually presented as sequencing 16S rRNA genes, RNA sequencing, cytokine and metabolite profiling of different biological samples, including feces, blood, and urine (Guo et al., 2023). This extensive incorporation of data can make the creation of multi-omic models feasible to discover disease-specific signatures with low false-positive rates in a wide range of gastrointestinal and non-gastrointestinal diseases (Ning et al., 2023). These jointly performed multi-omics studies do not merely come in handy during the classification of diseases. They also offer certain potential as the ability to diagnose and detect IBD in its initial stages without the necessity to perform intrusive tests that might be necessary to offer timely treatment and reduce the complications (Ning et al., 2023). This would allow the identification of effective biomarkers and prediction patterns to stage IBD that would allow the potential to develop a dual-axis model of accurate staging that would present different disease stages (Li et al., 2025). These microbial species, functional genes, and metabolites present in these biomarker panels can be of great help to the disease diagnostics, monitoring of their progress, and evaluation of whether the treatment is effective or not (Prabhu et al., 2025) (Ning et al., 2023).

CONCLUSION

As presented in this paper, there is ample evidence that inflammatory bowel disease is essentially caused by a complex imbalance in the microbial ecosystem of the gut coupled with an elevated degree of mucosal inflammation, and hence leading to a vicious cycle of inflammation. The results have revealed that massive loss of beneficial short-chain fatty acid-producing bacteria like Faecalibacterium prausnitzii and Bifidobacterium longum and a massive rise in disproportionate pro-inflammatory pathobionts such as adherent-invasive Escherichia coli and Ruminococcus gnavus are the most common features of IBD. This was done by the combination of metagenomic sequencing,

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immunological quantification and clinical correlation. The relationship between these modifications and the malfunctions of microbial metabolic pathways was identified in the pathways producing butyrate and breakdown of tryptophan. These are important processes that ensure that the epithelial barrier is healthy and the immune system tolerant. At the same time, the rise in the level of key cytokines like TNF-a, IL-6, and IL-17 and the increase in the activity of T-cell cohorts were the signs of the normal work of the immune system and were directly dependent on the extent of microbial dysbiosis. These close relations prove the fact that the mechanisms of host-microbiome reciprocity are the unbalanced microbiome worsens immunological dysregulation, which, consequently, changes the microbial environment even further. These results illustrate the reality that it is not microbiological or immunologic trigger that causes IBD, and rather the interaction between ecological perturbation in the gut and an inappropriate response of immune system. The results lend credence to the fact that microbiome-based therapeutic interventions, such as probiotics, prebiotics, dietary change, and fecal microbiota transplantation are adjunctive or prophylactic measures that can help to restore homeostasis. This integrative immunomicrobial system turns out to be complementing our existing knowledge of the pathogenesis of disease and offers a solid platform on which the personalized remedy solutions may be customized to the microbiota-immune phenotypes of a person.

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