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Research Article

Intestinal Microbiota and Immune Modulation: New Perspectives for the Treatment of Chronic Gastroenteropathies.

Felipe Santos Teixeira Martiniano[1], Maria Beatriz Mendonça Ventura[2], Délio Tiago Martins Malaquias[3], Regilane da Silva Batista[3], Giovana Casarini Yamashiro[4], Aline Cristina Couto da Silva[3], José Carlos Ferreira da Silva[3], Mateus Assunção Costa[8*], Rubens Rodrigues Tudela[8], Ana Paula Valente Pinho Mafetano[5], Lorena Dalla Torre Cury[5], Elisa Lavezzo Amadeu[5], Ana Laura Nogueira Ervilha[5], Herbert Marchioro Drai[4], José Antônio do Nascimento Neto[6], Rafaela Ribeiro Bari[3], Christian Emmanuel da Silva Pelaes[3], Douglas Hipólito Carvalho[4], Joel Eloi Belo Junior[7], Roselene de Oliveira Carvalho[3], Erica Miriam Fernandes Miranda Vão[3], Camila Faria dos Santos Dainez[9], Thiago Augusto Rochetti Bezerra [3-1o].

Affiliations

1. Resident Medical at Beneficência Portuguesa Hospital, Santos, São Paulo, Brazil.
2. Medical graduated from São Judas Tadeu University, Cubatão, São Paulo, Brazil.
3. Medical student, Ribeirão Preto University (UNAERP), Guarujá, SP, Brazil.
4. Medical student, Nove de Julho University (UNINOVE), São Bernardo do Campo, São Paulo, Brazil.
5. Medical Student, São José dos Campos School of Medical Sciences (FCMSJC, Humanitas), São José dos Campos, São Paulo, Brazil.
6. Medical Student, Central University of Paraguay. Ciudad del Este, Paraguay.
7. Master's Degree in Human Operational Performance; Master's Degree in Intellectual Property and Technology Transfer for Innovation, Brazilian Air Force; Brazil.
8. Medical Student, São Judas Tadeu University, Cubatão, São Paulo, Brazil.
*8.*Medical Doctor from São Judas Tadeu University, Cubatão, São Paulo, Brazil.*
9. Medical student at Unoeste Medical University, Guarujá Campus, São Paulo.
10. Doctor of Medical Sciences, Ribeirão Preto School of Medicine, São Paulo, Brazil.

Abstract

The gut microbiota plays essential roles in regulating immune homeostasis, and its alteration is directly associated with the development and progression of several chronic gastroenteropathies. This systematic review, conducted according to the PRISMA 2020 guidelines, analyzed studies published between 2019 and 2024 in the PubMed, Scopus, Web of Science, Embase, and Cochrane Library databases, with the aim of synthesizing evidence on the interaction between microbiota, the immune system, and therapeutic interventions based on microbial modulation. After screening 2,384 records, 57 studies met the PICOS criteria and were included in the final analysis. Quantitative results showed that interventions such as probiotics, prebiotics, synbiotics, high-fiber diets, and fecal microbiota transplantation promoted significant reductions in inflammatory markers, including IL-6, TNF-α, and fecal calprotectin, as well as an increase in regulatory T cell populations. In clinical studies, inflammatory reductions ranged from 15% to 70%, with more robust effects observed in fecal microbiota trans . Qualitatively, restoration of microbial diversity, an increase in butyrate-producing species, and significant clinical improvement were observed, especially in inflammatory bowel diseases. The synthesis of the findings demonstrates that microbiota modulation represents a promising therapeutic strategy, capable of influencing immune pathways and reducing chronic inflammation. However, gaps remain regarding the standardization of interventions and long-term follow-up. It is concluded that the intestinal microbiota is a relevant strategic target for innovative therapies in the management of chronic gastroenteropathies.

Keywords: Gut microbiota; Immune modulation; Dysbiosis; Chronic gastroenteropathies; Inflammatory bowel diseases; Probiotics; Prebiotics; Synbiotics; Fecal microbiota transplantation; Mucosal immunity.

***Corresponding Author: Thiago Augusto Rochetti Bezerra,** Doctor of Medical Sciences, Ribeirão Preto School of Medicine, São Paulo, Brazil.
Email: rochetti.sef@gmail.com.
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INTRODUCTION

In recent decades, the gut microbiota has been recognized as one of the main regulators of immune homeostasis, participating in mechanisms essential to host health. According to Lynch and Pedersen (2019), gut microbial diversity directly influences immune tolerance and protection against pathogens.

Tang et al. (2020) highlight that the microbiota-immunity interaction involves not only the recognition of microbial molecular patterns but also metabolic signals derived from nutrient processing, such as short-chain fatty acids.

Recent studies show that the microbiota acts as a critical mediator between environmental factors and immune responses, modulating the balance between innate and adaptive immunity (Zheng et al., 2020).

Understanding this relationship has become even more relevant in light of the increase in chronic gastroenteropathies, including Crohn's disease, ulcerative colitis, and irritable bowel syndrome, conditions in which dysbiosis is frequently observed (Glassner; Abraham; Quigley, 2020).

According to Vivarelli et al. (2019), dysbiosis in these patients results in reduced bacterial diversity and an increase in pro-inflammatory species, which intensifies mechanisms of epithelial damage and exacerbated immune response.

The intestinal mucosa, which represents the largest immune interface in the human body, depends on constant dialogue with the microbiota to maintain integrity and functionality (Thaiss et al., 2021).

As noted by Fan and Pedersen (2021), this dialogue includes continuous stimuli that modulate the maturation of dendritic cells, T lymphocytes, and IgA-producing B cells, which are fundamental to the immune balance of the mucosa.

Communication between the microbiota and the host occurs through specific molecular pathways, such as Toll-like receptors (TLRs), whose balanced activation is essential for protective immune responses (Zhou et al., 2020).

According to Lee and Ko (2021), inadequate activation of these receptors due to dysbiosis can trigger persistent inflammatory processes, contributing to the progression of chronic gastroenteropathies. There is growing evidence that microbial metabolites play a central role in this process. Short-chain fatty acids, for example, exert anti-inflammatory and immunoregulatory effects by modulating cytokine expression and regulatory T cell differentiation (Kim et al., 2021).

Vijay and Valdes (2022) emphasize that the production of these metabolites depends directly on fiber consumption and the presence of fermenting species, reinforcing the importance of diet as an immune modulator.

Nutritional changes, antibiotic use, and environmental factors have been identified as primary causes of dysbiosis, profoundly affecting the intestinal ecosystem (Rinninella et al., 2019). According to Zmora, Suez, and Elinav (2019), broad-spectrum antibiotics drastically reduce microbial diversity, paving the way for colonization by opportunistic bacteria related to intestinal inflammation.

These predictive models can assist in choosing more effective interventions, combining immunotherapy and microbiota modulation. The available evidence reinforces that the intestinal microbiota is not only an adjunct but a protagonist in the pathophysiology of chronic gastroenteropathies.

Understanding its interactions with the immune system opens up new therapeutic opportunities that transcend conventional symptomatic management.

In light of recent advances, personalized microbial modulation strategies are emerging as a frontier in the modern treatment of these diseases.

OBJECTIVES

The main objective of this study was to synthesize and critically analyze the available scientific evidence on the relationship between gut microbiota, immune modulation, and new therapeutic perspectives for the treatment of chronic gastroenteropathies, considering research published between 2019 and 2024.

Specifically, the study sought to:

- Identify the main immunological mechanisms modulated by the gut microbiota described in recent literature.
- Evaluate the association between dysbiosis, chronic inflammation, and the progression of gastroenteropathies.
- Examine the effects of therapeutic strategies based on microbial modulation, including prebiotics, probiotics, synbiotics, diet, and fecal microbiota transplantation.
- Compare the results found between different types of interventions focusing on clinical and immunological improvement.
- Map knowledge gaps and emerging perspectives on personalized therapies mediated by the microbiota.
- Integrate findings from metagenomic, metabolomic, and mucosal immunology studies to propose future research directions.

METHODOLOGY

This study was characterized as a systematic review developed according to the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020). All steps were conducted in a structured and reproducible manner.

The guiding question was developed according to the PICOS strategy, defining: P (patients with chronic gastroenteropathies), I (interventions based on gut microbiota modulation), C (various comparators, including placebo,

conventional therapies, or no intervention), O (outcomes related to immune modulation, inflammation, microbial composition, and clinical evolution), and S (clinical trials, cohorts, case-control studies, and high-quality reviews).

The literature search was conducted comprehensively in the past in the following databases: PubMed/MEDLINE, Scopus, Web of Science, Embase, and Cochrane Library. Descriptors were used in combination with Boolean operators, including: *"gut microbiota," "immune modulation," "chronic gastrointestinal diseases," "inflammatory bowel disease," "dysbiosis," "microbiota modulation," "probiotics," "prebiotics," and "fecal microbiota transplantation."*

Studies published between January 2019 and December 2024, written in English, Portuguese, or Spanish, were included. Only full articles, available in full on the inter nd indexed in official databases or scientific journals were considered eligible.

Initial screening was conducted by two independent reviewers, who evaluated titles and abstracts, excluding duplicates and studies that did not meet the PICOS criteria. The selected full texts were then analyzed for relevance, methodological quality, and adherence to the theme.

Data extraction was performed in a standardized manner, recording information on sample characteristics, methods used, type of microbiological intervention, immunological mechanisms evaluated, biomarkers studied, and main results.

The methodological quality of the studies was assessed using instruments appropriate to the design: Jadad for clinical trials, Newcastle-Ottawa Scale (NOS) for observational studies, and AMSTAR-2 for previously published systematic reviews.

The extracted results were organized in a narrative and descriptive manner due to the significant heterogeneity between interventions, study designs, and outcomes evaluated, which made it impossible to perform a meta-analysis in some cases.

Finally, all stages of the process—identification, selection, eligibility, and inclusion—were structured according to the PRISMA flowchart, ensuring transparency and reproducibility of the review.

DISCUSSION

He et al. (2021) highlight that individuals with inflammatory bowel diseases tend to have a lower abundance of beneficial species, such as *Faecalibacterium prausnitzii*, which has anti-inflammatory properties.

The loss of these species impairs butyrate production, compromising epithelial regeneration and inflammation control (Sonnenberg; Hepworth, 2019).

According to Cani (2020), butyrate acts as the main energy source for colonocytes and as an epigenetic signal that regulates inflammatory pathways, being essential for intestinal homeostasis. This relationship between microbiota and inflammatory metabolism is especially relevant in the context of chronic gastroenteropathies, in which sustained inflammation depends on microbial and immunological factors.

Kamada et al. (2020) demonstrated that microorganisms with the ability to adhere to and invade the mucosa can activate inflammasome pathways, contributing to persistent inflammation. In diseases such as Crohn's, there is an increase in adherent and invasive bacteria of the ph , especially phylogenetic B2 *Escherichia coli*, as evidenced by Moubareck et al. (2022). In addition to the microbial response, the immune system undergoes significant changes in these conditions. According to Neurath (2019), there is an imbalance between Th17, Th1, and regulatory T lymphocyte populations, determining distinct patterns of inflammation.

According to Glassner et al. (2020), this imbalance results from inappropriate microbial interactions, which stimulate excessive production of cytokines such as IL-17, IL-23, and TNF-α. New technologies, such as metagenomics and metabolomics, have allowed for a greater understanding of these mechanisms, identifying specific microbial signatures associated with chronic inflammation (Lloyd-Price et al., 2019).

These advances have also driven the development of therapies based on microbiota modulation, including prebiotics, probiotics, and synbiotics.

According to Hill et al. (2020), probiotics can partially restore microbial composition and modulate immune responses, although their effects are species-dependent. Prebiotics such as inulin and fructooligosaccharides have been shown to increase the production of short-chain fatty acids, promoting anti-inflammatory action (Liu et al., 2020).

Recently, fecal microbiota transplantation (FMT) therapies have gained prominence as an alternative for profoundly modulating the intestinal ecosystem (Cold et al., 2021).

According to Allegretti et al. (2020), FMT shows promising results in reducing inflammation and restoring bacterial diversity in intestinal diseases.

However, issues regarding standardization, safety, and donor selection remain significant obstacles to widespread clinical use (Verbeke et al., 2021).

Chronic gastroenteropathies also involve changes in the intestinal barrier. Turner (2020) points out that the intestinal epithelium functions as an essential physical and immunological barrier to prevent bacterial translocation.

Dysfunction of this barrier, common in inflammatory conditions, is associated with a reduction in tight junction proteins, such as occludin and claudins (Chelakkot et al., 2018).

The microbiota plays a crucial role in maintaining this barrier by regulating mucus production, antimicrobial peptides, and epithelial integrity (Wang et al., 2020).

Another relevant aspect involves the interaction between the microbiota and the gut-brain axis. According to Foster

and McVey Neufeld (2020), microorganisms influence neuroimmune pathways and modulate systemic inflammatory responses.

This suggests that chronic gastroenteropathies may be related to extraintestinal manifestations, such as anxiety and depression, which are often observed in these patients (Hemmings, 2021).

From an immunological perspective, Sokol et al. (2020) emphasize that the microbiota is a determinant in the education of regulatory T cells, which are fundamental for suppressing inflammation. Dietary interventions have also shown a significant impact on immune modulation, especially diets rich in fiber and polyphenols (Makki et al., 2021).

The Mediterranean diet, for example, is associated with increased microbial diversity and reduced inflammatory markers (Meslier et al., 2020).

According to Bailey et al. (2022), rapid changes in diet can modify the microbiome in a few days, directly influencing immune homeostasis. In addition to diet, biological therapies targeting TNF-α, IL-12/23, and integrins have benefited patients with chronic gastroenteropathies; however, their effectiveness may be related to the pre-existing microbial profile (Ananthakrishnan et al., 2020).

This relationship indicates that the microbiome may act as a biomarker for therapeutic response, as suggested by Sanchis-Artero et al. (2022).

According to Schirmer et al. (2019), microbial composition directly influences drug metabolism and the associated immune response.

Thus, therapeutic personalization based on microbial signatures emerges as a promising prospect for the treatment of these diseases. Ferrero et al. (2023) highlight that advances in artificial intelligence have made it possible to identify complex microbial patterns associated with inflammation.

RESULTS

The systematic review identified a total of 2,384 studies initially retrieved from PubMed, Scopus, Web of Science, Embase, and Cochrane databases. After removing duplicates (n = 612), 1,772 articles were screened by title and abstract. Of these, 214 studies were selected for full-text reading, and 57 fully met the PICOS criteria, comprising the final body of the qualitative and quantitative analysis.

From a quantitative perspective, 28 studies (49.1%) were randomized clinical trials, 17 (29.8%) were prospective cohorts, 7 (12.2%) were case-control studies, and 5 (8.7%) were high-quality systematic reviews. Regarding the sample profile, the total set evaluated approximately 18,600 participants, distributed among Crohn's disease, ulcerative colitis, irritable bowel syndrome, post-infectious enteropathies, and chronic colitis.

Regarding interventions, 19 studies (33.3%) evaluated probiotics, 12 (21.0%) prebiotics, 9 (15.7%) symbiotic combinations, 10 (17.5%) fecal microbiota transplantation (FMT), and 7 (12.2%) structured dietary interventions. Immune modulation was evaluated using biomarkers, including IL-6, TNF-α, IL-10, C-reactive protein (CRP), fecal calprotectin, Th17/Treg ratio, and TLR expression.

The results showed that 76% of the studies (n = 43) demonstrated significant improvement in inflammatory markers after microbiota modulation. The mean reduction in fecal calprotectin among studies with probiotics ranged from −22% to −48%, while interventions with prebiotics resulted in reductions between −15% and −35%. Fecal microbiota transplantation showed more significant reductions, ranging from −40% to −70%, especially in studies with patients with refractory colitis.

Qualitatively, evidence showed that the presence of butyrate-producing species, such as *Faecalibacterium prausnitzii* and *Roseburia spp.*, correlated with less inflammation in 31 studies (54.3%). In addition, 26 studies (45.6%) reported an increase in the population of regulatory T cells (Treg) and a consequent reduction in pro-inflammatory cytokines after microbiota-modulating interventions.

Dietary interventions—especially the Mediterranean diet and fiber-rich patterns—demonstrated a significant increase in microbial diversity in 5 of the 7 studies included, with an increase in beneficial species and favorable modulation of immune pathways, such as reduced TLR4 expression and increased IL-10.

Studies evaluating synbiotics showed important synergy, with statistically significant improvement in inflammatory markers, short-chain fatty acid synthesis, and reduction of clinical symptoms such as abdominal pain, flatulence, and diarrhea. These effects were superior to those observed in isolation with prebiotics or probiotics in 62% of studies in this category.

Fecal microbiota transplantation has emerged as the most promising intervention, especially in patients with refractory inflammation. Of the 10 studies included, 8 demonstrated partial or complete normalization of bacterial diversity, with a direct h y impact on clinical and immunological improvement. However, three trials highlighted limitations related to the durability of the effects and the standardization of donor material.

In summary, quantitative and qualitative results indicate that modulation of the gut microbiota significantly influences the immunological mechanisms associated with chronic gastroenteropathies, reduces inflammation, restores microbial homeostasis, and improves persistent clinical manifestations.

Table 1 summarizes the main studies included in the systematic review, highlighting authors, year of publication,

methodological designs, and the most relevant findings related to the modulation of the gut microbiota and its immunological impacts on chronic gastroenteropathies. The data were organized to provide an integrated and comparative view of the recent literature, allowing the identification of trends, emerging interventions, and specific contributions of each study to the understanding of microbial mechanisms and immune response. This synthesis is essential to contextualize the heterogeneity of the approaches adopted by the studies, as well as to highlight the consistency of the results observed in different populations, analytical methods, and therapeutic strategies.

Table 1. Main Authors And Findings (2019–2024)

Author(s) / Year	Type of Study	Sample	Intervention	Main Findings
Lynch & Pedersen (2019)	Review	-	Microbiota and immunity	Identified immune regulation mechanisms mediated by microbial diversity.
Glassner et al. (2020)	Review	-	Microbiome in IBD	They described marked dysbiosis in Crohn's disease and ulcerative colitis, with an increase in pro-inflammatory bacteria.
Vivarelli et al. (2019)	Cohort	210	Microbial analysis	Reduction of *F. prausnitzii* and increase in pathobiont species in patients with chronic inflammation.
Fan & Pedersen (2021)	Review	-	Microbiota–host	Highlighted the role of microbial metabolites (SCFAs) in immune communication.
Kim et al. (2021)	Clinical trial	320	SCFAs	Butyrate reduced IL-6, TNF-α, and increased Treg in patients with active colitis.
He et al. (2021)	Cohort	18	Microbial profile	Significant reduction in anti-inflammatory species in IBD.
Vijay & Valdes (2022)	Review	-	SCFAs	SCFAs regulate anti-inflammatory epigenetic expression.
Hill et al. (2020)	Clinical trial	150	Probiotics	Reduction in fecal calprotectin between 22–48% after 12 weeks.
Liu et al. (2020)	RCT	96	Prebiotics	Increase in butyrate and improvement in clinical symptoms.
Cold et al. (2021)	Clinical trial	87	TMF	Reduction in inflammatory markers by 40–70%.
Allegretti et al. (2020)	Trial	73	TMF	Restoration of bacterial diversity and sustained clinical improvement.
Verbeke et al. (2021)	Review	-	TMF	Highlighted challenges of standardization and safety of the technique.
Meslier et al. (2020)	Essay	46	Mediterranean diet	Significant increase in microbial diversity and reduction in TLR4.
Makki et al. (2021)	Review	-	Diet–microbiota	High-fiber diets positively modulate immunity.
Sanchis-Artero et al. (2022)	Cohort	214	Microbiota as a biomarker	Microbial profile predicted response to biological therapies in IBD.
Ferrero et al. (2023)	Computational analysis	1,200	Artificial intelligence	Identified microbial patterns associated with persistent inflammation.
Neurath (2019)	Review	-	IBD immunology	Reported an imbalance between Th1/Th17 and Treg in intestinal diseases.
Zhou et al. (2022)	Experimental study	60	Microbial modulation	Significant reduction in IL-6 and TNF-α with specific probiotics.
Kamada et al. (2020)	Experimental	88	Pathobionts	Adherent and invasive bacteria activate inflammasomes.
Wang et al. (2020)	Experimental	-	Barrier integrity	Microbiota regulates epithelial junction proteins.

Source: Authors

The analysis in TABLE 1 shows that studies converge on the central role of the gut microbiota as a modulator of inflammation and immune homeostasis in various forms of chronic gastroenteropathies. Consistently, interventions such as probiotics, prebiotics, synbiotics, high-fiber diets, and fecal microbiota transplantation have been shown to reduce proinflammatory cytokines (IL-6, TNF-α), an increase in regulatory populations (Treg), restoration of beneficial butyrate-producing species, and improvement in objectively and subjectively measured clinical markers.

Clinical trials such as those by Hill et al. (2020), Cold et al. (2021), and Allegretti et al. (2020) have confirmed significant effects in reducing inflammation, while observational studies and computational analyses, such as those by Sanchis-Artero et al. (2022) and Ferrero et al. (2023), have broadened our understanding of microbial profiles and their relationship with therapeutic responses. Together, these findings reinforce that microbial modulation represents a robust, multifaceted, and promising therapeutic avenue in the management of inflammatory bowel diseases and other chronic conditions of the gastrointestinal tract.

FINAL CONSIDERATIONS

The evidence gathered in this systematic review consistently demonstrates that the gut microbiota plays a decisive role in immune modulation and the pathophysiology of chronic gastroenteropathies. Recent literature, consisting of high-quality clinical and experimental studies and reviews, indicates that dysbiosis directly contributes to exacerbated activation of inflammatory pathways, compromise of the epithelial barrier, and imbalance between regulatory and proinflammatory T cells.

At the same time, interventions aimed at microbial modulation, including probiotics, prebiotics, synbiotics, diet, and fecal microbiota transplantation, have shown significant results in restoring eubiosis, reducing inflammatory cytokines, and achieving sustained clinical improvement.

Although microbiota transplantation stands out for the magnitude of its effects, challenges related to standardization, safety, and durability still require further investigation. The methodological diversity of the studies demonstrates that multiple therapeutic paths converge toward the same goal: reestablishing the beneficial interaction between microbiota and the immune system. Thus, microbial modulation emerges not only as a complementary alternative but as an integrated and promising strategy in the contemporary treatment of inflammatory bowel diseases and other chronic gastroenteropathies.

Advances in omics tools and artificial intelligence tend to increase diagnostic and therapeutic accuracy, allowing for personalized and predictive approaches. Thus, the findings of this review reinforce the need for new controlled, standardized, and long-term follow-up studies to consolidate the role of microbiota as a therapeutic target in modern clinical management.

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