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REVIEW ARTICLE

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Nutrigenomics and dietary control of inflammatory bowel diseases: a systematic review

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Abstract

Introduction: Inflammatory bowel diseases (IBD) have an increasing incidence and prevalence, such as Crohn's disease (CD) and ulcerative colitis (UC). Gut dysbiosis is affected by host genetics, nutrition, antibiotics, and inflammation, and is associated with the development of IBD, which can be attributed to impaired miRNA expression functions. Micro and macronutrient deficiencies and overabundance of calories and macronutrients trigger inflammatory processes and susceptibility to infections. **Objective:** To present the nutrological, microbiological, and extracellular vesicle (exosome-microRNAs) evidence in the modulation and control of symptoms of inflammatory bowel diseases. **Methods:** The systematic review rules of the PRISMA Platform were followed. The literary search process was carried out from September to October 2025 in the Web of Science, Scopus, Embase, PubMed, Science Direct, Scielo, and Google Scholar databases. The quality of the studies was based on the GRADE instrument and the risk of bias was analyzed according to the Cochrane instrument. **Results and Conclusion:** A total of 189 articles were found. A total of 27 articles were fully evaluated and 21 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 21 studies with a high risk of bias and 29 studies that did not meet GRADE and AMSTAR-2. Most studies showed homogeneity in their results, with X^2=75.7%>50%. Concluiu-se que os miRNAs são importantes reguladores da função celular e da homeostase, e sua atividade anormal foi demonstrada em diversas doenças, incluindo DII. O uso de miRNAs como biomarcadores e alvos terapêuticos pode ajudar a monitorar o tratamento da DII e apoiar o desenvolvimento de novas terapias mais individualizadas que minimizem os efeitos colaterais comuns. A manipulação dietética dos microRNAs por meio dos prebióticos e probióticos podem manipular seletivamente a microbiota intestinal, produzindo ácidos graxos de cadeia curta que fornecem energia para as células epiteliais intestinais e induzem respostas imunes regulatórias protetoras.

Keywords: Inflammatory bowel diseases. Modulation. Nutrology. Gut microbiota. MicroRNAs.

Introduction

Inflammatory bowel diseases (IBD) have an increasing incidence and prevalence, such as Crohn's disease (CD) and ulcerative colitis (UC) **[1]**. The etiology and pathogenesis of IBD remain uncertain and may be related to genetic susceptibility, gut microbiota, environmental factors, and immune dysfunction. In addition to therapeutic drugs such as aminosalicylic acid, corticosteroids, immunomodulators, and biological agents, the therapeutic effects of microRNAs and exosomes stand out **[2]**.

Among the genetic factors involved in the etiology of IBD, variants in genes related to autophagy have been identified. Three distinct forms of autophagy have been described, including microautophagy, chaperone-mediated autophagy, and macroautophagy. Autophagy plays a crucial role in maintaining intestinal homeostasis, regulating the interaction between the

gut microbiota and both innate and adaptive immunity, as well as host defense against intestinal pathogens. Autophagy dysfunction is associated with several human pathologies, including IBD **[3]**.

Furthermore, gut dysbiosis is influenced by host genetics, nutrition, antibiotics, and inflammation, and is linked to the development of IBD. In addition, intestinal epithelial dysfunction, altered autophagy, and immune hyperactivation are frequently detected in individuals with severe IBD, which may be attributed to impaired miRNA expression functions **[4]**. Added to this, the potential of microRNA-focused strategies in the diagnosis and treatment of IBD stands out, offering a scientific basis for the advancement of precision medicine in the management of IBD **[5]**.

All these epigenetic and nutritional mechanisms are of paramount importance, as approximately 70.0 to 80.0% of patients lose weight during the course of IBD, leading to some degree of nutritional impairment, and around 23.0% of outpatients and 85.0% of hospitalized patients with predominant malnutrition **[6-10]**. Thus, micronutrient and macronutrient deficiencies and an overabundance of calories and macronutrients trigger inflammatory processes and susceptibility to infections **[11-14]**. Several micronutrients are especially important for immunonutrition, including vitamins such as vitamins A, C, D, and E, folic acid, beta-carotene, and trace elements such as zinc, selenium, manganese, and iron. Deficiencies in zinc and vitamins A, C, and D can reduce the functions of natural killer cells **[15-18]**.

Therefore, this study described a systematic review in order to present the nutritional, microbiological, and extracellular vesicle (exosome-microRNA) evidence in the modulation and control of symptoms of inflammatory bowel diseases.

Methods

Study Design

This study followed an international systematic review model, following the PRISMA (preferred reporting items for systematic reviews and meta-analyses) guidelines. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: September 15, 2025. The methodological quality standards of AMSTAR-2 (Assessing the methodological quality of systematic reviews) were also followed. Available at: https://amstar.ca/. Accessed on: September 15, 2025.

Data Sources and Research Strategy

The literature search process was conducted from September to October 2025 and developed based on Web of Science, Scopus, Embase, PubMed, Lilacs, Ebsco, Scielo, and Google Scholar, covering scientific articles from various periods to the present. The following descriptors (DeCS/MeSH Terms) were used: *"Inflammatory bowel diseases. Modulation. Nutrology. Gut microbiota. MicroRNAs"*, and using the Boolean operator "and" between MeSH terms and "or" between historical findings.

Study Quality and Risk of Bias

Quality was classified as high, moderate, low, or very low regarding the risk of bias, clarity of comparisons, precision, and consistency of analyses. The most evident highlight was systematic review articles or meta-analyses of randomized clinical trials, followed by randomized clinical trials. Low-quality evidence was attributed to case reports, editorials, and brief communications, according to the GRADE instrument. The risk of bias was analyzed according to the Cochrane instrument through the analysis of the Funnel Plot (Sample size versus Effect size), using Cohen's d test.

Results and Discussion

Summary of Findings

A total of 189 articles were submitted to eligibility analysis, with 21 final studies selected to compose the results of this systematic review. The listed studies presented medium to high quality (Figure 1), considering the level of scientific evidence of studies such as meta-analysis, consensus, randomized clinical, prospective, and observational. Biases did not compromise the scientific basis of the studies. According to the GRADE instrument, most studies showed homogeneity in their results, with X^2=75.7%>50%. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 21 studies with a high risk of bias and 29 studies that did not meet the GRADE and AMSTAR-2 criteria.

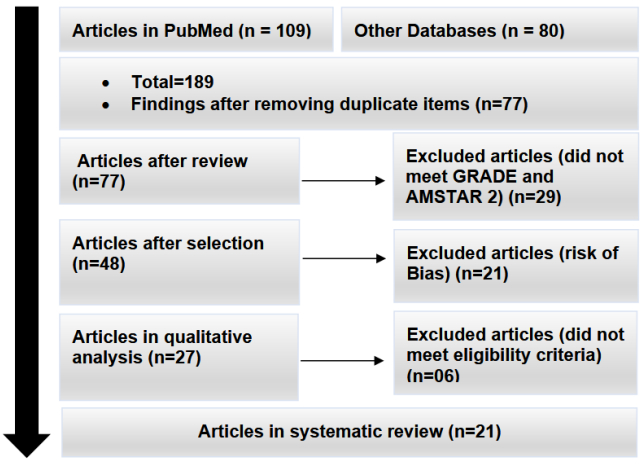

Figure 1. Selection and inclusion process of articles for the systematic review. Source: Own authorship.

Figure 2 presents the results of the risk of bias of the studies using the Funnel Plot, showing the calculation of the Effect Size (Magnitude of the difference) using Cohen's Test (d). The precision (sample size) was determined indirectly by the inverse of the standard error (1/Standard Error). This graph had a symmetrical behavior, not suggesting a significant risk of bias, both between studies with small sample sizes (lower precision) that are shown at the bottom of the graph and in studies with large sample sizes that are presented at the top.

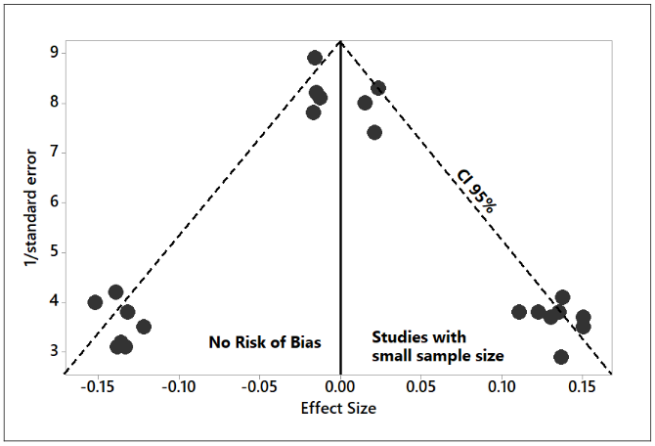

Figure 2. The symmetrical funnel plot does not suggest a risk of bias between the small sample size studies that are shown at the bottom of the graph (n=14 studies). High confidence and high recommendation studies are shown above the graph (n= 07 studies). Ntotal =21 studies. Figure 2. The symmetrical funnel plot does not suggest a risk of bias between the small sample size studies that are shown at the bottom of the graph (n=14 studies). High confidence and high recommendation studies are shown above the graph (n= 07 studies). Ntotal =21 studies.Source: Own authorship.

Main Clinical Findings

In the context of immunometabolic activities, microRNAs (miRNAs) play an important role as modulators of IBD, such as miR-31, miR-155, and miR-21. These miRNAs can modulate gene expression at the post-transcriptional level, being important biomarkers and also therapeutic interventions. Although miRNA-based therapies have significant potential, off-target effects, immune activation, and inefficiencies in administration methods may occur. Furthermore, the interaction between miRNAs and the gut microbiota can influence disease mechanisms and treatment responses **[19]**.

Studies have shown that miRNA dysregulation has been implicated in the development of IBD, as specific miRNAs are differentially expressed in patients with IBD compared to healthy individuals. The regulation of their expression can modulate the inflammatory response, the composition of the gut microbiota, and the function of the intestinal barrier. miRNAs can regulate the immune and inflammatory response through multiple mechanisms, from the regulation of Th1/Th17 and ferroptosis to the modulation of NLRP3 (NOD-like receptor family, pyrin domain 3) and the control of the NF-κB pathway (nuclear factor kappa-light chain-enhancer of activated B cells) **[20,21]**.

The authors Tong et al. (2021) **[22]** explored the therapeutic effects of extracellular vesicles (mEVs) in IBD. The microRNAs and protein content in mEVs were analyzed by RNA sequencing and proteomics, and the gut microbiota was analyzed by 16S rRNA sequencing. It was shown that proteins and microRNAs abundant in mEVs were involved in the regulation of immune and inflammatory pathways and that oral administration of mEVs prevented colon shortening, reduced intestinal epithelial disruption, inhibited inflammatory cell infiltration, and tissue fibrosis. mEVs attenuated the inflammatory response by inhibiting the TLR4-NF-κB signaling pathway and activating the NLRP3 inflammasome. In addition, mEVs were able to correct the cytokine production disorder and restore the balance between type 17 helper T cells (Th17) and interleukin-10+Foxp3+ regulatory T cells (Treg) in the inflamed colon. The disturbed gut microbiota was also partially recovered after treatment with mEVs.

The authors Lv et al. (2024) **[23]** investigated the relationship between the fecal gut microbiome (FGM) and intestinal tissue miRNAs at different stages of pediatric Crohn's disease (CD). Metagenomic analyses and miRNA sequencing were conducted to examine the intestinal FGM and miRNA profiles of CD patients before and after clinical induction therapy and controls. A total of 27 newly diagnosed and therapy-naive pediatric patients with active CD and 11 IBD controls were recruited. FGM and miRNA profiles were significantly altered between CD patients and controls. Seven key bacteria were identified at the species level, including *Defluviitalea raffinosedens*, *Thermotalea metallivorans*, *Roseburia intestinalis*, *Dorea sp.* AGR2135, *Escherichia coli*, *Shigella sonnei*, and *Salmonella enterica*, whose exact proportions were subsequently validated by quantitative qPCR analysis. A total of 8 key miRNAs were also identified, including hsa-miR-215-5p, hsa-miR-194-5p, hsa-miR-12135, hsa-miR-509-3-5p, hsa-miR-212-5p, hsa-miR-4448, hsa-miR-501-3p, and hsa-miR-503-5p. Functional enrichment analysis of differential miRNAs indicated significantly altered cyclin protein, cyclin-dependent protein, and cell cycle pathway. Both the gut microbiome structure and miRNA profiles were significantly altered at different stages of CD.

It is also observed that IBDs are characterized by chronic inflammation and damage to colonocytes, with etiology of genetic, epigenetic, and environmental factors. It has been observed that microRNA-223 (miR-223) is increased in patients with IBD **[24]**. In addition, the authors Li et al. (2024) **[25]** analyzed the mechanism of action of MIR2911 from honeysuckle, the main component of traditional Chinese medicine preparations for UC. The results demonstrated that MIR2911 can be absorbed from the diet and secreted into small extracellular vesicles of the host, acting directly on intestinal bacteria, reducing the abundance of *Escherichia shigella* and improving UC symptoms. It is known that miR-191a and miR-212 damage the intestinal barriers, and others strengthen the intestinal barrier. It has been shown that c-Jun and myosin light chain kinase (MLCK) are the targets of miR-200b **[26]**. Silencing protein tyrosine kinase 6 (PTK6) expression with miR-93 in the intestinal epithelium increases resistance to TNF-α-induced injury **[27]**.

It has also been found that miRNAs contribute to the immunological reactions that lead to IBDs. Protein 2 containing the nucleotide-binding oligomerization domain (NOD2) is one of the genes clearly associated with CD **[28]**. Some studies have found abnormally elevated miRNA levels in the mucosal tissues of patients with UC compared to healthy controls. Authors found that miR-16, miR-21, miR-23a, miR-24, miR-29a, miR-126, miR-195, and let-7f were upregulated in patients with active UC compared to healthy controls **[29]**. Comparing the colonic mucosa of patients with UC and healthy controls, the authors showed that miR-7, miR-26a, miR-29a, miR-29b, miR-31, miR-126, miR-127-3p, miR-135b, and miR-324-3p were increased in the inflamed mucosa of patients with UC **[30]**.

In this context of dietary manipulation of microRNAs, prebiotic and probiotic therapies can selectively manipulate the gut microbiota **[6,7]**. Prebiotics represent non-digestible carbohydrates that promote the growth of beneficial bacteria in the intestine, increasing the production of short-chain fatty acids and modulating the production of cytokines in the intestinal mucosa. Probiotics, on the other hand, contain live bacteria that appear to exert positive effects on human gut health, modulating mucosal permeability and strengthening the immune system's ability to keep pathogens away from the intestinal mucosal surface **[6]**.

The gut microbiota is fundamental for the activation of the immune system, with particular emphasis on *Lactobacillus acidophilus*, *Lactobacillus bulgaricus*, and *Lactobacillus casei*, increasing IgA for antigen removal via a non-inflammatory pathway and increasing T and B lymphocytes, as well as *Faecalibacterium prausnitzii*, one of the most prevalent intestinal bacterial species in healthy adults, being beneficial and a producer of butyrate **[15,16]**. Lactobacilli and Bifidobacteria inhibit the growth of exogenous and/or harmful bacteria, stimulate immune functions, aid in the digestion and/or absorption of food ingredients and minerals, and contribute to vitamin synthesis **[6,8]**.

In this respect, short-chain fatty acids, such as butyrate, propionate, and acetate, serve as an energy source for intestinal epithelial cells and induce protective regulatory immune responses **[15,16]**. The gut's adaptive immune system is also rapidly activated after exposure to commensal bacteria, with an increase in the expression of class II molecules of the major histocompatibility complex and an increase in T cells **[13]**. T cells can generate subpopulations whose immune response is pro-inflammatory or anti-inflammatory. Th1 and Th17 cells – helper T cells – are pro-inflammatory in nature, while Treg cells (of CD4+ CD25+ phenotype) and Th2 cells are anti-inflammatory. The Gram-negative bacterium Bacteroides fragilis induces the differentiation of CD4+ T cells into Treg cells, leading to the production of anti-inflammatory cytokines, such as interleukin-10 (IL-10) and transforming growth factor beta (TGFβ), abolishing the pro-inflammatory response of Th17. The differentiation of Treg cells depends on the recognition by CD4+ T cells of the polysaccharide presented by CD **[10,13]**.

Finally, many studies have evaluated the ability of diet to modulate the gut microbiota and microRNAs to influence epithelial barrier function. Diets with low fiber concentrations have been associated with IBD, with a postulated mechanism of reduced production of short-chain fatty acids by commensal bacteria whose preferred energy source is fiber. Butyrate, a short-chain fatty acid, is essential for colon health and the main energy source for colonocytes. In this sense, short-chain fatty acids also promote immune tolerance by promoting the development of regulatory T cells **[31-33]**.

Limitations

There is still a lack of randomized controlled clinical studies with large sample sizes on dietary manipulations, probiotics, and the control of gene expression by microRNAs, in order to better understand the main nutrients and probiotic strains for the control and treatment of inflammatory bowel diseases.

CONCLUSION

It was concluded that miRNAs are important

regulators of cellular function and homeostasis, and their abnormal activity has been demonstrated in several diseases, including IBD. The use of miRNAs as biomarkers and therapeutic targets can help monitor IBD treatment and support the development of new, more individualized therapies that minimize common side effects. Dietary manipulation of microRNAs through prebiotics and probiotics can selectively manipulate the gut microbiota, producing short-chain fatty acids that provide energy to intestinal epithelial cells and induce protective regulatory immune responses.

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No additional data are available.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate[@].

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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