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# Major approaches and clinical studies on the relationship between inflammatory bowel diseases and nutrients, gut microbiota and exosomes/microRNAs: a systematic review

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## Abstract

**Introduction**: Inflammatory bowel diseases (IBDs) are multifactorial diseases characterized by chronic inflammation of the gastrointestinal tract. Nutrients, gut microbiota, exosomes, and microRNAs play crucial roles in the pathophysiology of IBD. **Objective:** It was to carry out a systematic review of the main approaches and clinical studies on the relationship between inflammatory bowel diseases and nutrients, intestinal microbiota, and exosomes/microRNAs. **Methods:** The PRISMA Platform systematic review rules were followed. The search was carried out from August to September 2025 in the Web of Science, Scopus, 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 177 articles were found, and 58 articles were evaluated in full, and 30 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 06 studies with a high risk of bias and 25 studies that did not meet GRADE and AMSTAR-2. Most studies showed homogeneity in their results, with $X^2$=77.7%>50%. It was concluded that inflammatory bowel diseases are associated with various gastrointestinal symptoms and, therefore, affect patients' quality of life. Although intestinal bacteria and the host's immune response are considered important factors in its pathogenesis, a sufficient explanation of their role in its pathophysiological mechanism has not been presented. Exosomes and microRNAs, together with nutrients and gut microbiota, participate in the molecular interactions of inflammatory bowel diseases. Recent studies have confirmed the important role of miRNAs in targeting certain molecules in signaling pathways that regulate intestinal barrier homeostasis, inflammatory reactions, and autophagy of the intestinal epithelium. Several studies have identified specific miRNAs associated with inflammatory bowel diseases in colon tissues. The correlation between the gut microbiota and cytokines suggests that exosomes and microRNAs can modulate intestinal immunity by influencing the gut microbiota.

**Keywords:** Inflammatory bowel diseases. Nutrients. Gut microbiota. Exosomes/microRNAs.

## Introduction

Inflammatory bowel diseases (IBDs) are multifactorial diseases characterized by chronic inflammation of the gastrointestinal tract, exemplified by Crohn's disease (CD) and ulcerative colitis (UC). CD and UC primarily affect young people, causing bloody diarrhea, abdominal pain, malabsorption, fatigue, and impaired quality of life. Long-term inflammation also increases the risk of colorectal cancer in patients with IBDs, which has a mortality rate of 10-15% [1,2].

In this context, microRNAs (miRNAs or miR-) play important roles in the pathophysiology of IBD [1]. miRNAs are non-coding, endogenous, single-stranded, evolutionarily conserved RNAs that bind to the 3' untranslated region (UTR), 5'UTR, or partially translated region of a target mRNA, inhibiting mRNA translation and blocking its expression. miRNAs are important regulators of cellular function and homeostasis, and their abnormal activity has been demonstrated in several diseases, including IBDs. Thus, new treatment options could be developed to alter imbalances in miRNA expression. miRNAs affect the intestinal barrier and inflammatory reactions through various pathological mechanisms [3].

In this respect, metabolism encompasses the interactions between diet, the microbiome, and cellular enzymatic processes that generate the chemical pathways necessary to maintain life, and regulate the balance of the intestinal microbiota, mainly in the treatment of IBDs. Endogenous metabolites as well as dietary nutrients can directly influence epigenetic enzymes [3-6]. Epigenetic modifications in DNA and histone proteins alter cell fate, controlling chromatin accessibility and downstream gene expression patterns [7-10].

In addition to the connection between metabolism and epigenetic pathways, nutrients can impact cellular status by modulating signaling pathway activity. A clear example is through the mechanistic target of rapamycin (mTOR) signaling pathway and, in particular, the mTOR 1 complex (mTORC1), which regulates cell growth only when nutrients and growth factors are present [3,10]. It is also noteworthy that nutrients impact cellular status through AMP-activated protein kinase (AMPK), which at low levels of cellular ATP phosphorylates substrates to restore the cell's energy balance [3,4].

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 IBDs, leading to some degree of nutritional impairment, and around 23.0% of outpatients and 85.0% of hospitalized patients with predominant malnutrition [11,12]. The Western diet is characterized by excessive consumption of refined sugars, salt, and saturated fats, as well as low consumption of dietary fiber and low overall dietary variability. New features of human nutrition in modern society include artificial sweeteners, gluten, and genetically modified foods [9].

Micronutrient and macronutrient deficiencies, as well as an overabundance of calories and macronutrients, trigger inflammatory processes and increase susceptibility to infections [13]. 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 [14-17].

Therefore, the present study aimed to conduct a systematic review of the main approaches and clinical studies on the relationship between inflammatory bowel diseases and nutrients, gut microbiota, and exosomes/microRNAs.

## Methods

### Study Design

This study followed an international model for systematic review, following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: 09/14/2025. The methodological quality standards of AMSTAR-2 (Assessing the methodological quality of systematic reviews) were also followed. Available at: https://amstar.ca/. Accessed on: 09/14/2025.

### Data Sources and Search Strategy

The literature search process was conducted from August to September 2025 and developed based on Web of Science, Scopus, PubMed, Lilacs, Ebsco, Scielo, and Google Scholar, covering scientific articles from various periods to the present day. The following descriptors (DeCS /MeSH Terms) were used: "Inflammatory bowel diseases. Nutrients. Gut microbiota. Exosomes/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 for systematic review articles or meta-analyses of randomized clinical trials,

followed by randomized clinical trials. Low quality of 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 177 articles were submitted to eligibility analysis, with 30 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-analyses, consensus, randomized clinical trials, and prospective and observational studies. Biases did not compromise the scientific basis of the studies. According to the GRADE instrument, most studies showed homogeneity in their results, with $X^2$=77.7%>50%. Considering the Cochrane tool for risk of bias, the overall evaluation resulted in 6 studies with a high risk of bias and 25 studies that did not meet the GRADE and AMSTAR-2 criteria.

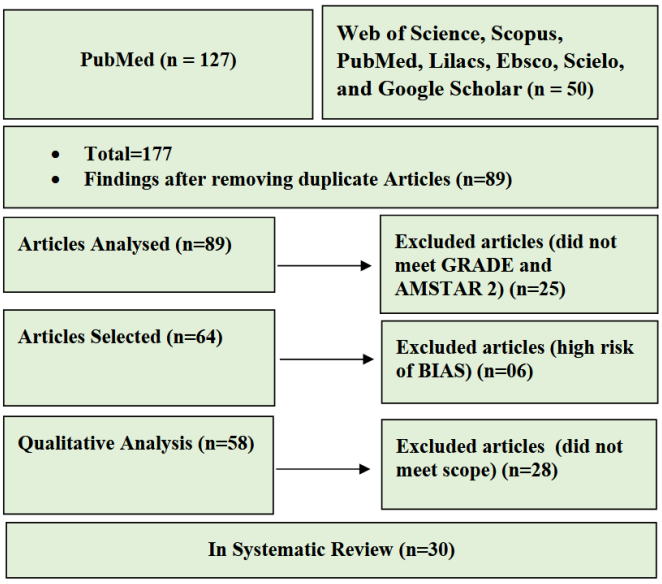


Figure 1. Flowchart showing the article selection process. Source: Own authorship.

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.

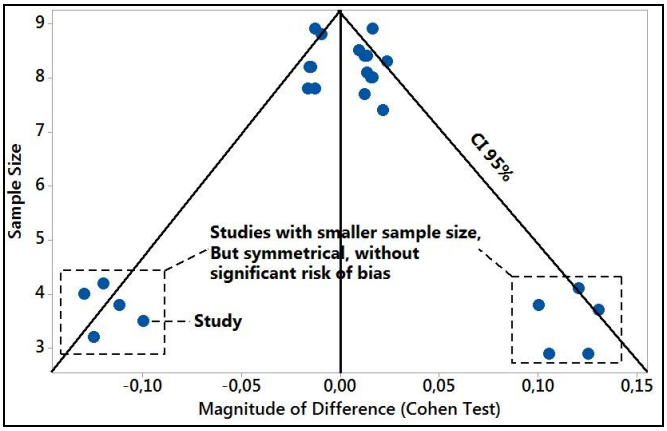


Figure 2. The symmetrical funnel plot does not suggest a risk of bias between the small sample size studies that are shown at the bottom of the graph. High confidence and high recommendation studies are shown above the graph (n=30 studies). Source: Own authorship.

### Main Clinical Outcomes

Disrupted intestinal membranes are one of the most significant factors in the pathogenesis of IBD. TNF-α is known to be an important pro-inflammatory cytokine in the pathogenesis of IBD [18]. miR-191a and miR-212 are known to damage intestinal barriers, while others strengthen the intestinal barrier. c-Jun and myosin light chain kinase (MLCK) have been shown to be targets of miR-200b [19]. Silencing protein tyrosine kinase 6 (PTK6) expression with miR-93 in the intestinal epithelium increases resistance to TNF-α-induced injury [20].

Moreover, miRNAs are known to contribute to the immunological reactions that lead to IBD. Protein 2 containing the nucleotide-binding oligomerization domain (NOD2) is one of the genes clearly associated with CD [21]. 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 [22]. Comparing the colon 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 [23].

Increased levels of miR-21, miR-155, miR-923, let-7a, let-7c, let-7d, and let-7g were found in colon biopsy samples from patients with active UC compared to healthy controls [24]. Frozen distal colectomy biopsy samples from patients with UC were found to show significant increases in miR-31, miR-146a, miR-206, and miR-424 levels [25], as well as increases in miR-20b,

miR-26b, miR-98, miR-99a, and miR-203 levels in colon biopsy samples from patients with active UC compared to healthy controls [26]. These and other findings are summarized in Table 1.

Table 1. Key considerations of microRNAs in IBDs.

| | | |
|---|---|---|
| **microRNAs that weaken the intestinal barrier** | | |
| **miR-874** | Aquaporin 3 | Decreases the expression of aquaporin 3 |
| **miR-675** | **C**adherin E, ZO-1 | Destabilizes the mRNA of cadherin E and ZO-1 |
| **miR-122a** | EGFR | Enhances the expression of zonulin and increases epithelial permeability |
| **miR-191a, -212** | ZO-1 | Reduce the expression of ZO-1 |
| **miR-21** | PTEN/PI3K/Akt | Increases the paracellular permeability pathway of the intestinal epithelium |
| **microRNAs - Strengthe n the intestinal barrier** | | |
| **miR-93 (downregulation)** | PTK6 | Reduces the expression of PTK6 and attenuates epithelial injury |
| **miR-200b** | c-Jun, MLCK | Decreases epithelial damage induced by TNF-α |
| **microRNAs - Reducing inflammation** | | |
| **miR-10a** | IL-12/23p40 | Downregulates the expression of IL-12/23p40 and Th1/Th17 cell responses |
| **miR-141** | CXCL12β | Inhibits CXCL12β-mediated leukocyte migration |
| **miR-320** | NOD2 | Decreases the expression of NOD2 |

Source: Own authorship.

A study carried out by Tong et al. (2021) [27] explored the therapeutic effects of exosomes and microRNAs (mEVs) from cow's milk in IBD. The microRNAs and protein content in mEVs were analyzed by RNA sequencing and proteomics, respectively, followed by functional annotation. The proteins and microRNAs abundant in mEVs were involved in the regulation of immunological and inflammatory pathways, and oral administration of mEVs prevented colon shortening, reduced intestinal epithelial disruption, inhibited inflammatory cell infiltration, and tissue fibrosis. The mEVs attenuated the inflammatory response by inhibiting the TLR4-NF-κB signaling pathway and activating the NLRP3 inflammasome. Furthermore, 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 in UC was also partially recovered after treatment with mEVs.

In this context of dietary manipulation of microRNAs, prebiotic and probiotic therapies can selectively manipulate the gut microbiota [3,4]. In this sense, prebiotics represent non-digestible carbohydrates that promote the growth of beneficial bacteria in the gut, increasing the production of short-chain fatty acids and modulating cytokine production in the intestinal mucosa [5]. Probiotics, on the other hand, contain live bacteria that appear to exert positive health effects in the human gut, modulating mucosal permeability and strengthening the immune system's ability to keep pathogens away from the intestinal mucosal surface [1].

The gut microbiota is fundamental for the activation of the immune system, with emphasis on *Lactobacillus acidophilus, Lactobacillus bulgaricus* and *Lactobacillus casei*, increasing IgA for the removal of antigens through a non-inflammatory pathway and increasing T and B lymphocytes, as well as *Faecalibacterium prausnitzii* is one of the most prevalent intestinal bacterial species in healthy adults, being beneficial and producing butyrate [7].

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 [1,3,6]. In this aspect, 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].

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 [3]. T cells can generate subpopulations whose immune response is pro-inflammatory or anti-inflammatory. Th1 and Th17 cells – helper T cells – are pro-inflammatory, while Treg cells (of CD4+ CD25+ phenotype) and Th2 cells are anti-inflammatory [10].

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].

Many studies have evaluated the ability of diet to modulate the gut microbiota and microRNAs to influence epithelial barrier function. Low-fiber diets have been associated with IBDs, 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 [28-30].

## Limitations

More robust randomized clinical trials with standardized methodologies are needed. Also, clinical studies with geographically diverse patients are also needed to better understand the different types of microRNAs involved in inflammatory bowel diseases.

## Conclusion

It was concluded that inflammatory bowel diseases are associated with various gastrointestinal symptoms and therefore affect the quality of life of patients. Although intestinal bacteria and the host immune response are considered important factors in their pathogenesis, a sufficient explanation of their role in their pathophysiological mechanism has not been presented. Exosomes and microRNAs, along with nutrients and intestinal microbiota, participate in the molecular interactions of inflammatory bowel diseases. Recent studies have confirmed the important role of miRNAs in directing certain molecules in signaling pathways that regulate intestinal barrier homeostasis, inflammatory reactions, and autophagy of the intestinal epithelium. Several studies have identified specific miRNAs associated with inflammatory bowel diseases in colon tissues. The correlation between the gut microbiota and cytokines suggests that exosomes and microRNAs may modulate gut immunity by influencing the gut microbiota.

## CRediT

Author contributions**: Conceptualization**- Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto, Rafael Tinoco Alves, Deangelo Cláudio Gomes de Lima, Jose Manuel Torres Garcia, Luciene Pereira de Oliveira, Renatto Souza Nunes Cabral; **Data curation-** Karlla Vieira Campos Ricatto, Rafael Tinoco Alves, Deangelo Cláudio Gomes de Lima, Jose Manuel Torres Garcia; **Formal Analysis -** Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto, Rafael Tinoco Alves, Deangelo Cláudio Gomes de Lima, Jose Manuel Torres Garcia, Luciene Pereira de Oliveira, Renatto Souza Nunes Cabral, Natashira Soares Torres, Jussara Santos Sousa; **Investigation-** Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto; **Methodology-** Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto; **Project administration-** Karlla Vieira Campos Ricatto; **Supervision-** Karlla Vieira Campos Ricatto; **Writing - original draft-** Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto, Rafael Tinoco Alves, Deangelo Cláudio Gomes de Lima, Jose Manuel Torres Garcia, Luciene Pereira de Oliveira, Renatto Souza Nunes Cabral, Natashira Soares Torres, Jussara Santos Sousa ; **Writing-review & editing-** Karlla Vieira Campos Ricatto, Celso Alexandre Alves, Iuri Sanzio Souto, Rafael Tinoco Alves, Deangelo Cláudio Gomes de Lima, Jose Manuel Torres Garcia, Luciene Pereira de Oliveira, Renatto Souza Nunes Cabral, Natashira Soares Torres, Jussara Santos Sousa.

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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.



## References

1. Alfaifi J, Germain A, Heba AC, Arnone D, Gailly L, Ndiaye NC, Viennois E, Caron B, Peyrin-Biroulet L, Dreumont N. Deep Dive Into MicroRNAs in Inflammatory Bowel Disease. Inflamm Bowel Dis. 2023 Jun 1;29(6):986-999. doi: 10.1093/ibd/izac250.
2. 1So D, Quigley EMM, Whelan K. Probiotics in irritable bowel syndrome and inflammatory bowel disease: review of mechanisms and effectiveness. Curr Opin Gastroenterol. 2023 Mar 1;39(2):103-109. doi: 10.1097/MOG.0000000000000902.

3. Jung H, Kim JS, Lee KH, Tizaoui K, Terrazzino S, Cargnin S, Smith L, Koyanagi A, Jacob L, Li H, Hong SH, Yon DK, Lee SW, Kim MS, Wasuwanich P, Karnsakul W, Shin JI, Kronbichler A. Roles of microRNAs in inflammatory bowel disease. Int J Biol Sci. 2021 May 17;17(8):2112-2123. doi: 10.7150/ijbs.59904.
4. Mah C, Jayawardana T, Leong G, Koentgen S, Lemberg D, Connor SJ, Rokkas T, Grimm MC, Leach ST, Hold GL. Assessing the Relationship between the Gut Microbiota and Inflammatory Bowel Disease Therapeutics: A Systematic Review. Pathogens. 2023 Feb 6;12(2):262. doi: 10.3390/pathogens12020262.
5. Zhang Y, Si X, Yang L, Wang H, Sun Y, Liu N. Association between intestinal microbiota and inflammatory bowel disease. Animal Model Exp Med. 2022 Dec;5(4):311-322. doi: 10.1002/ame2.12255.
6. Adolph TE, Zhang J. Diet fuelling inflammatory bowel diseases: preclinical and clinical concepts. Gut. 2022 Dec;71(12):2574-2586. doi: 10.1136/gutjnl-2021-326575.
7. Danilova NA, Abdulkhakov SR, Grigoryeva TV, Markelova MI, Vasilyev IY, Boulygina EA, Ardatskaya MD, Pavlenko AV, Tyakht AV, Odintsova AK, Abdulkhakov RA. Markers of dysbiosis in patients with ulcerative colitis and Crohn's disease. Ter Arkh. 2019 May 15;91(4):17-24. doi: 10.26442/00403660.2019.04.000211.
8. He Q, Gao Y, Jie Z, Yu X, Laursen JM, Xiao L, Li Y, Li L, Zhang F, Feng Q, Li X, Yu J, Liu C, Lan P, Yan T, Liu X, Xu X, Yang H, Wang J, Madsen L, Brix S, Wang J, Kristiansen K, Jia H. Two distinct metacommunities characterize the gut microbiota in Crohn's disease patients. Gigascience. 2017 Jul 1;6(7):1-11. doi: 10.1093/gigascience/gix050.
9. Green N, Miller T, Suskind D, Lee D. A Review of Dietary Therapy for IBD and a Vision for the Future. Nutrients. 2019 Apr 26;11(5). pii: E947. doi: 10.3390/nu11050947.
10. Shapira SN, Christofk HR. Metabolic Regulation of Tissue Stem Cells. Trends Cell Biol. 2020 Jul;30(7):566-576. doi: 10.1016/j.tcb.2020.04.004.
11. Basson A. Vitamin D. Crohn's disease in the adult patient: a review. J Parenter Enteral Nutr. 2014;38:438–58.
12. Roth MP, Petersen GM, McElree C, Vadheim CM, Panish JF, Rotter JI. Familial empiric risk estimates of inflammatory bowel disease in Ashkenazi Jews. Gastroenterology. 1989;96(4):1016-20.
13. Lee YK, Menezes JS, Umesaki Y, Mazmanian SK. Proinflammatory T-cell responses to gut microbiota promote experimental autoimmune encephalomyelitis. Proc Natl Acad Sci USA. 2011;108(suppl 1):4615-4622.
14. Teng F, Klinger CN, Felix KM, et al. Gut microbiota drive autoimmune arthritis by promoting differentiation and migration of Peyer's patch T follicular helper cells. Immunity. 2016;44(4):875-888.
15. Donohoe DR, Garge N, Zhang X, et al. The microbiome and butyrate regulate energy metabolism and autophagy in the mammalian colon. Cell Metab. 2011;13(5):517-526.
16. Smith PM, Howitt MR, Panikov N, et al. The microbial metabolites, short-chain fatty acids, regulate colonic Treg cell homeostasis. Science. 2013;341(6145):569-573.
17. Mowat C, Cole A, Windsor A, Ahmad T, Arnott I, Driscoll R, et al. Guidelines for the management of inflammatory bowel disease in adults. Gut 2011;60:571-607.
18. Ma TY, Boivin MA, Ye D, Pedram A, Said HM. Mechanism of TNF-{alpha} modulation of Caco-2 intestinal epithelial tight junction barrier: role of myosin light-chain kinase protein expression. Am J Physiol Gastrointest Liver Physiol. 2005;288:G422–30.
19. Shen Y, Zhou M, Yan J, Gong Z, Xiao Y, Zhang C. et al. miR-200b inhibits TNF-alpha-induced IL-8 secretion and tight junction disruption of intestinal epithelial cells in vitro. Am J Physiol Gastrointest Liver Physiol. 2017;312:G123–G32.
20. Haines RJ, Beard RS Jr, Eitner RA, Chen L, Wu MH. TNFalpha/IFNgamma Mediated Intestinal Epithelial Barrier Dysfunction Is Attenuated by MicroRNA-93 Downregulation of PTK6 in Mouse Colonic Epithelial Cells. PLoS One. 2016;11:e0154351.
21. Corridoni D, Arseneau KO, Cominelli F. Functional defects in NOD2 signaling in experimental and human Crohn disease. Gut Microbes. 2014;5:340–4.
22. Wu F, Zikusoka M, Trindade A, Dassopoulos T, Harris ML, Bayless TM. et al. MicroRNAs are differentially expressed in ulcerative colitis and alter expression of macrophage inflammatory peptide-2 alpha. Gastroenterology. 2008;135:1624– 35. e24.
23. Fasseu M, Treton X, Guichard C, Pedruzzi E, Cazals-Hatem D, Richard C, Identification of restricted subsets of mature microRNA abnormally expressed in inactive colonic mucosa

of patients with inflammatory bowel disease. PLoS One. 2010. 5.

24. Takagi T, Naito Y, Mizushima K, Hirata I, Yagi N, Tomatsuri N. et al. Increased expression of microRNA in the inflamed colonic mucosa of patients with active ulcerative colitis. J Gastroenterol Hepatol. 2010;25(Suppl 1):S129–33.
25. Lin J, Welker NC, Zhao Z, Li Y, Zhang J, Reuss SA. et al. Novel specific microRNA biomarkers in idiopathic inflammatory bowel disease unrelated to disease activity. Mod Pathol. 2014;27:602–8.
26. Coskun M, Bjerrum JT, Seidelin JB, Troelsen JT, Olsen J, Nielsen OH. miR-20b, miR-98, miR-125b-1*, and let-7e* as new potential diagnostic biomarkers in ulcerative colitis. World J Gastroenterol. 2013;19:4289–99.
27. Tong L, Hao H, Zhang Z, Lv Y, Liang X, Liu Q, Liu T, Gong P, Zhang L, Cao F, Pastorin G, Lee CN, Chen X, Wang JW, Yi H. Milk-derived extracellular vesicles alleviate ulcerative colitis by regulating the gut immunity and reshaping the gut microbiota. Theranostics. 2021 Jul 25;11(17):8570-8586. doi: 10.7150/thno.62046.
28. Dong Y, Xu T, Xiao G, Hu Z, Chen J. Opportunities and challenges for synthetic biology in the therapy of inflammatory bowel disease. Front Bioeng Biotechnol. 2022 Aug 10;10:909591. doi: 10.3389/fbioe.2022.909591.
29. Li G, Lin J, Zhang C, Gao H, Lu H, Gao X, Zhu R, Li Z, Li M, Liu Z. Microbiota metabolite butyrate constrains neutrophil functions and ameliorates mucosal inflammation in inflammatory bowel disease. Gut Microbes. 2021 Jan-Dec;13(1):1968257. doi: 10.1080/19490976.2021.1968257.
30. Parada Venegas D, De la Fuente MK, Landskron G, González MJ, Quera R, Dijkstra G, Harmsen HJM, Faber KN, Hermoso MA. Short Chain Fatty Acids (SCFAs)-Mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases. Front Immunol. 2019 Mar 11;10:277. doi: 10.3389/fimmu.2019.00277. Erratum in: Front Immunol. 2019 Jun 28;10:1486.

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