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Investigation of the gut microbiota and nutrients in regenerative processes in inflammatory bowel diseases: a systematic review

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Abstract

Introduction: Metabolism encompasses the interactions between diet, the microbiome, and cellular enzymatic processes that generate the chemical pathways necessary to sustain life. Epigenetic and nutritional mechanisms are of paramount importance, as approximately 80.0% of patients lose weight during inflammatory bowel diseases (IBD). **Objective:** It was to develop a systematic review of the main clinical studies on the impact of nutritional treatment on inflammatory bowel diseases. **Methods:** The PRISMA Platform systematic review rules were followed. The research was carried out from March to April 2025 in the 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 132 articles were found, and 20 articles were evaluated in full and 17 were included and developed in the present systematic review study. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 14 studies with a high risk of bias and 20 studies that did not meet GRADE and AMSTAR-2. Most studies showed homogeneity in their results, with X^2=78.4%>50%. It was concluded that important randomized controlled clinical studies in recent years have highlighted the important role of diet modulation in the control and even remission of inflammatory bowel diseases. There was a reduction in persistent intestinal symptoms, balance of the gut microbiota, reduction of inflammatory markers, and improvement in quality of life.

Keywords: Nutrology. Diet therapy. Gut microbiota. Regenerative process. Inflammatory bowel diseases.

Introduction

Metabolism encompasses the interactions between diet, the microbiome, and cellular enzymatic processes that generate the chemical pathways necessary to sustain life and regulate the balance of the gut microbiota, particularly in the treatment of inflammatory

bowel disease (IBD). Endogenous metabolites and dietary nutrients can directly influence epigenetic enzymes **[1-4]**. Epigenetic modifications to DNA and histone proteins alter cell fate by controlling chromatin accessibility and downstream gene expression patterns **[5-7]**.

Most substrates and cofactors for chromatin-modifying enzymes are derived from metabolic pathways, including the tricarboxylic acid cycle, methionine cycle, folate cycle, glycolysis, β-oxidation, and the hexosamine pathway. These complex and interconnected networks generate intermediates that coactivate epigenetic enzymes and/or serve as direct substrates for modifications, including acetyl-CoA, alpha-ketoglutarate (α-KG), succinate, fumarate, S-adenosyl methionine (SAM), UDPGlcNAc, ketone bodies, lactate, NADH, FADH2 **[8]**.

In addition to the connection between metabolism and epigenetic pathways, nutrients can influence cellular state by modulating the activity of signaling pathways. A clear example is through the mechanistic target of rapamycin (mTOR) signaling pathway and, in particular, mTOR complex 1 (mTORC1), which regulates cell growth only when nutrients and growth factors are present. Depletion of specific nutrients, including arginine, leucine, and S-adenosyl methionine, prevents growth factor-induced mTORC1 activation by blocking Rag GTPase-mediated recruitment of mTORC1 to the lysosome where it can be activated by Rheb GTPase **[1,8]**.

Another way that nutrients are sensed to impact cellular state is through AMP-activated protein kinase (AMPK), which at low cellular ATP levels phosphorylates substrates to restore cellular energy balance, and in the process regulates cell growth and autophagy. Additionally, transcription factors can be directly regulated by metabolites. Tryptophan kynurenine is an endogenous agonist for the aryl hydrocarbon and alpha-ketoglutarate (α-KG) receptor that binds to and activates IKKβ and initiates NF-κβ signaling **[1,2]**. Furthermore, dietary and metabolite manipulations can affect tissue stem cells and direct cell fate decisions, as highlighted in the small intestine by intestinal stem cells (ISTCs). In this case, the enzyme 3-hydroxy-3-methylglutaryl-CoA synthase (Hmgcs2) is highly expressed. Also, ketogenic or glucose-rich diets regulate the balance of self-renewal by ITS **[8]**.

All these epigenetic and nutrological mechanisms are of utmost importance, since approximately 70.0 to 80.0% of patients lose weight during IBD, leading to some degree of nutritional impairment, and approximately 23.0% of outpatients and 85.0% of hospitalized patients with predominant malnutrition **[9,10]**. This nutritional deficit is associated with delayed clinical improvement and postoperative recovery, as well as mortality and increasing cases of surgical complications **[10]**. In this sense, it is worth noting that the Western diet is characterized by excessive consumption of refined sugars, salt, and saturated fat and low consumption of dietary fiber, as well as low overall dietary variability.

New features of human nutrition in modern society include artificial sweeteners, gluten, and genetically modified foods. Thus, micro and macronutrient deficiencies occur, and the overabundance of calories and macronutrients triggers inflammatory processes and susceptibility to infections **[11]**. Several micronutrients are especially important for immunonutrition, including vitamins such as vitamins A, C, D, and E, folic acid, betacarotene, and trace elements such as zinc, selenium, manganese, and iron. Deficiencies of zinc and vitamins A, C, and D can reduce the functions of natural killer cells **[12,13]**.

Also, vitamin D plays a role in intestinal defense, suppressing microbial invasion of the epithelium. In this regard, vitamin D deficiency has been identified in 82% of IBD patients, compared to a national average of 31%, and has been associated with defective epithelial processes **[14]**. The Mediterranean diet has been suggested to exert immunomodulatory effects and also to modulate epigenetic mechanisms in favor of IBD control. Likewise, the semi-vegetarian diet (SVD) has been shown to exert preventive effects against IBD relapse in patients who achieved remission in a two-year prospective clinical trial in a single center **[15]**.

Therefore, the present study aimed to develop a systematic review of the main clinical studies on the impact of nutritional treatment on inflammatory bowel diseases.

Methods

Study Design

This study followed the international systematic review model, following the PRISMA (preferred reporting items for systematic reviews and meta-analysis) rules. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: 12/04/2025. The AMSTAR-2 (Assessing the methodological quality of systematic reviews) methodological quality standards were also followed. Available at: https://amstar.ca/. Accessed on: 12/04/2025.

Data Sources and Search Strategy

The literature search process was carried out from March to April 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 day. The following descriptors (DeCS /MeSH Terms) were used *Nutrology. Diet therapy. Gut microbiota. Regenerative process. Inflammatory bowel diseases*, and using the Boolean "and" between MeSH terms and "or" between historical findings.

Study Quality and Risk of Bias

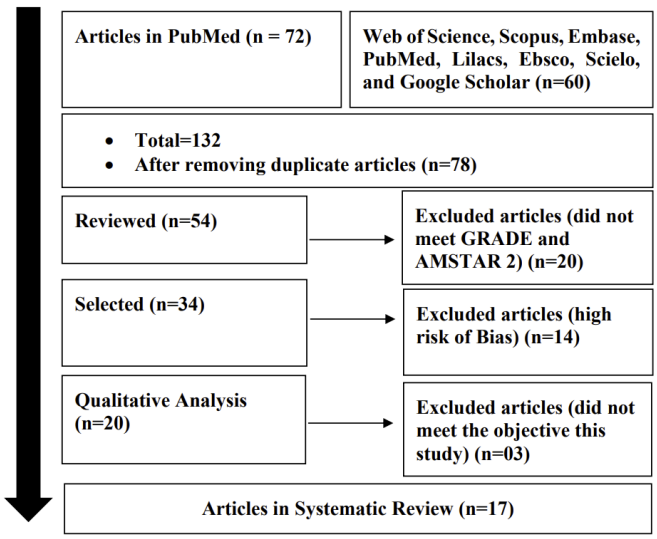
Quality was classified as high, moderate, low, or very low regarding the risk of bias, clarity of comparisons, precision, and consistency of analyses. The most evident emphasis was on systematic review articles or meta-analysis of randomized clinical trials, followed by randomized clinical trials. Low 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 by analyzing the Funnel Plot graph (Sample size versus Effect size), using Cohen's test (d).

Results and Discussion

Summary of Findings

A total of 132 articles were found that were subjected to eligibility analysis, and 17 final studies were selected to compose the results of this systematic review. The listed studies were of 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 presented homogeneity in their results, with X^2=78.4%>50%. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 14 studies with a high risk of bias and 20 studies that did not meet GRADE and AMSTAR-2.

Figure 1. Articles included in the systematic review.



Source: Own authorship.

Clinical Results - Nutrology, Gut Microbiota, and Inflammatory Bowel Diseases

Therapies with prebiotics and probiotics can selectively manipulate the intestinal microbiota **[1,2]**. In this sense, 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 **[3]**. Probiotics contain live bacteria that appear to have positive health effects on the human intestine, modulating mucosal permeability and strengthening the maintenance of the immune system by removing pathogens from the surface of the intestinal mucosa **[1]**.

In this sense, the intestinal microbiota is essential for the activation of the immune system, with emphasis on *Lactobacillus acidophilus*, *Lactobacillus bulgaricus*, and *Lactobacillus casei*, increasing IgA for the removal of antigens by a non-inflammatory pathway and increasing T and B lymphocytes. 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 the synthesis of vitamins **[1,4]**.

Also, *Faecalibacterium prausnitzii* is one of the most prevalent intestinal bacterial species in healthy adults, being beneficial and a producer of butyrate **[5]**. The reduction of this bacteria in the intestine can contribute to the onset or worsening of IBD. Therefore, to increase the numbers of these bacteria, it is necessary to eat foods rich in fiber, and increase the consumption of fruits, vegetables, legumes, whole grains and cereals, seeds, and nuts **[5,8]**.

Increasing dietary fiber increases butyrate, a short-chain fatty acid that is involved in colon health, with important anti-inflammatory properties **[8]**. Therefore, 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 **[13]**. The adaptive immune system of the intestine 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 **[1]**. T cells can generate subpopulations whose immune response is pro-inflammatory or anti-inflammatory. Th1 and Th17 cells – T helper cells are pro-inflammatory, while Treg cells (CD4+ CD25+ phenotype) and Th2 cells are anti-inflammatory **[8]**.

In this sense, the Gram-negative bacterium Bacteroides fragilis induces the differentiation of CD4+ T cells into Treg cells, leading to the production of antiinflammatory cytokines, such as interleukin-10 (IL-

10) and transforming growth factor beta (TGFβ), nullifying the pro-inflammatory response of Th17 [8]. The differentiation of Treg cells depends on the recognition by CD4+ T cells of the polysaccharide presented by CD. In turn, segmented filamentous bacteria, after contact with antigen-presenting cells, have been shown to induce pro-inflammatory cells, such as Th17 cells [8].

Most of the studies listed in this study followed a randomized, controlled model and were heterogeneous, suggesting the broad impact of dietary interventions on inflammation and clinical outcomes. Table 1 shows the studies that were selected to compose the systematic review and meta-analysis, revealing a high level of evidence for the main dietary results in the control and/or remission of IBD.

Table 1. Major clinical studies from the last ten years demonstrate the important nutritional role of diet in the control and/or remission of IBD. *RCT = randomized controlled trial.

AUTHORS	DIET	KEY RESULTS Showed improvement in.....
[16] Cox et al. 2020	FODMAP Reduction	• Persistent bowel symptoms • Gut microbiome • Circulating markers of inflammation
[17] Cox et al. 2017	FODMAP Reduction	• Persistent bowel symptoms • Gut microbiome • Circulating markers of inflammation
[18] Pedersen et al. 2017	FODMAP Reduction	• Persistent bowel symptoms • Gut microbiome • Circulating markers of inflammation
[19] Bodini et al. 2019	FODMAP Reduction	• Persistent bowel symptoms • Gut microbiome • Circulating markers of inflammation
[20] Papada et al. 2019	Mastiha (2.8g/day)	• Increased serum IL-6, fecal calprotectin and fecal lactoferrin in the placebo group • Attenuation in the increase in free AA levels in the Mastiha group
[21] Jian et al., 2018	Immunoglobulin G (IgG)-guided exclusion diet	• Extraintestinal manifestations decreased from 7 to 2 in the intervention group • Mean body mass index and albumin were higher in the intervention group
[22] Albenberg et al. 2019	Red meat reduction	• Moderate to severe relapse occurred in 62% of participants in the higher meat intake group and 42% of participants in the lower intake group.
[23] Svolos et al.,2018	EEN vs. CD-TREAT	• In children receiving CD-TREAT, 4 (80%) had a clinical response and 3 (60%) went into remission, with concurrent significant reductions in fecal calprotectin
[24] Levine et al. 2019	EEN vs. CDED+PEN	• In children receiving CDED plus PEN, corticosteroid-free remission was associated with sustained reductions in inflammation, based on serum C-reactive protein, fecal calprotectin, and fecal Proteobacteria
[25] Racine et al. 2016	High sugar and soft drinks	• Sugar and soft drink consumers were at higher risk of UC if they had low vegetable intake.
[26] Braly et al., 2017	vegetable reduction	• Six of 8 subjects gained weight, 1 subject lost weight, and 1 subject remained unchanged. Energy intake was significantly greater than 100% of the recommended daily intake (RDI)/adequate intake for 64% of daily doses completed.
[27] Machado et al., 2015	Specific carbohydrates	• Whey and soy protein supplementation alters body composition by reducing body fat and contributes to inflammation control.
[28] Brotherton et al., 2014	Whey and soy protein	• There were no adverse effects, and participants reported improvements in health-related quality of life ($p=0.028$) and gastrointestinal function ($p=0.008$) compared to the control group.
[29] Sökülmez et al., 2014	Cereal fiber (wheat)	• Macronutrient and water-soluble fiber intake levels improved persistent bowel symptoms with statistical significance.
[30] Kyaw et al., 2014	Regulated hospital diets (macronutrients and fiber)	• There was a mean increase in Inflammatory Bowel Disease Questionnaire score in the intervention group compared to a reduction in score in the control group. • A total of 69% of patients in the intervention group considered the dietary advice significantly helpful
[31] Hanai et al., 2012	Elental (≥900 kcal/day) VS. 6-mercaptopurine	• No significant differences were found between 6-mercaptopurine and Elental. - In the 6-mercaptopurine group, 2 patients had liver damage and one developed alopecia. • Elemental should be useful for long-term maintenance therapy in Crohn's disease
[32] Kang et al., 2015	Short-term PEN (n=17) (1 month)	• Nutritional status improved substantially after 1 year of treatment in the severe CD group. • The <13 years group demonstrated better improvement in nutritional status than the ≥13 years group

Note: **FODMAP:** Fermentable Oligo-, Di- and Monosaccharides, and Polyols; **EEN**= Exclusive Enteral Nutrition; **CDED**= Crohn`Disease Exclusion Diet; **PEN**= Partial Enteral Nutrition.

In this context, many studies have evaluated the ability of diet to modulate the gut microbiota and influence epithelial barrier function. Low-fiber diets 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 [33].

In this sense, short-chain fatty acids also promote immune tolerance by promoting the development of regulatory T cells [1]. Food additives are commonly consumed by IBD patients, and specific dietary emulsifiers (carboxymethyl cellulose and polysorbate 80) have been shown to induce low-grade inflammation and metabolic syndrome in wild-type mice and promote colitis in genetically predisposed IL-10 knockout mice [2]. Emulsifiers can alter the host microbiota, resulting in increased inflammatory potential with an increase in the number of mucolytic bacteria and erosion of the protective layer of the mucosa.

Conclusion

It was concluded that important randomized controlled clinical studies in recent years have shown the important role of dietary modulation in the control and even remission of inflammatory bowel diseases. A reduction in persistent intestinal symptoms, balance of the intestinal microbiota, reduction of inflammatory markers and improvement in quality of life were observed.

CRediT

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Conflict of Interest

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It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

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It was performed.

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