International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

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Poland 00-773
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ARTICLE TITLE	DIETETARY MODULATION IN THE MANAGEMENT OF ULCERATIVE COLITIS: AN EVIDENCE-BASED REVIEW
ARTICLE INFO	Patryk Kowalczyk, Bartłomiej Trzciński, Wiktoria Socha, Igor Winogrodzki, Alicja Stryczek-Schlusche, Aleksandra Magdalena Furczyńska, Filip Bracichowicz, Aleksandra Gęsińska, Hanna Paszkiewicz, Kamil Nowak, Oliwia Gugała. (2025) Dietetary Modulation in The Management of Ulcerative Colitis: An Evidence-Based Review. *International Journal of Innovative Technologies in Social Science*. 3(47). doi: 10.31435/ijitss.3(47).2025.3584
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3584
RECEIVED	05 July 2025
ACCEPTED	21 August 2025
PUBLISHED	28 August 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

DIETETARY MODULATION IN THE MANAGEMENT OF ULCERATIVE COLITIS: AN EVIDENCE-BASED REVIEW

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ABSTRACT

Ulcerative colitis is a chronic, idiopathic inflammatory disease of the large intestine, limited to the mucous membrane. The manifestation of the condition is characterised by periods of exacerbation and remission. The onset of Ulcerative Colitis (UC) is most often due to a combination of genetic susceptibility, immune system hyperactivity, microbiota disorders, epithelial barrier defects and environmental factors, including diet. In addition to conventional pharmacological interventions, there is an increasing focus on the importance of a nutritionally balanced diet as a supportive component of the treatment process. A review of the extant preclinical and clinical studies indicates the beneficial effects of fibre, especially the soluble fraction, vitamin D3, curcumin and zinc. These substances have been demonstrated to alleviate the symptoms of the disease and promote remission by supporting the gut microbiota and inhibiting inflammatory processes. The effectiveness of omega-3 fatty acids remains to be fully confirmed, necessitating further research, but the preliminary results are encouraging. A balanced diet is an integral component of the therapeutic management of patients diagnosed with UC. However, further research is required to standardise clinical recommendations.
Materials and Methods: A detailed analysis of 36 peer- reviwed scientific articles published between 2002 and 2025 from sources such as PubMed and Google Scholar focused on recent reports on diet in UC.

KEYWORDS

Ulcerative Colitis, Fibre, Curcumin, Zinc, Omega-3 Fatty Acids, Vitamin D3

CITATION

Patryk Kowalczyk, Bartłomiej Trzciński, Wiktoria Socha, Igor Winogrodzki, Alicja Stryczek-Schlusche, Aleksandra Magdalena Furczyńska, Filip Bracichowicz, Aleksandra Gęsińska, Hanna Paszkiewicz, Kamil Nowak, Oliwia Gugała. (2025) Dietetary Modulation in The Management of Ulcerative Colitis: An Evidence-Based Review. *International Journal of Innovative Technologies in Social Science*. 3(47). doi: 10.31435/ijitss.3(47).2025.3584

1. Introduction

Ulcerative colitis is a chronic disease that is classified as an inflammatory bowel disease. The condition affects the mucous membrane of the large intestine, spreading from the rectum to the commencement of the large intestine. The inflammation is characterised by its continuity, with no healthy mucous membrane present between the inflamed areas. The disease progresses with periods of exacerbation and remission. The active phase of the disease is characterised by abdominal pain, diarrhoea, blood in the stool, an urge to defecate, and fever. During periods of remission, patients may not exhibit any symptoms, whether clinical, laboratory or endoscopic. The incidence of the condition is increasing, and varies depending on the geographical region, with the highest reported incidence in Europe and North America. The incidence is also increasing in newly industrialised countries and those adopting Western trends, especially dietary trends [19][23][28][33].

Materials and Methods

A detailed analysis of 36 peer- reviwed scientific articles published between 2002 and 2025 from sources such as PubMed and Google Scholar focused on recent reports on diet in UC.

2. Etiology and pathogenesis

The precise pathogenesis of ulcerative colitis remains elusive. The aetiology of the condition is hypothesised to be multifactorial, with genetic, immunological and environmental factors, including diet and lifestyle, being recognised as potential contributing elements. This phenomenon is most often the result of an interaction between genetic predisposition and environmental factors that can modulate the expression of genes involved in the immune response [19][23][28][33].

2.1 Genetic factors

Variants of certain genes have been demonstrated to increase susceptibility to the disease. To date, over 200 loci associated with IBD have been identified, including approximately 30 that are specific to UC[34][35].

Key genes and genetic variants:

CARD9 has been identified as a gene that is associated with both CD and UC [35].

The present study investigates the association between JAK/STAT pathway genes and the development of UC. It is hypothesised that polymorphisms in JAK2, TYK2, STAT1, STAT3 and STAT4 genes increase the risk of developing UC [35][36].

RIPK3 has been demonstrated to be associated with epithelial cell necrosis in murine models, and there is a concomitant increase in susceptibility to colitis[35].

2.2 Environmental factors

Western diet: A dietary regime of a Western nature, with a low intake of fibre and high intake of saturated and trans fatty acids, as well as sugar, has been shown to result in intestinal dysbiosis. This results in increased intestinal barrier permeability, leading to the penetration of antigens and microorganisms into the submucosal layer. This, in turn, stimulates the immune system, causing inflammation. In the UC model, a decline in the population of beneficial bacteria responsible for the production of anti-inflammatory substances, such as butyric acid, has been observed [19][23][28][33].

Air and water pollution: The presence of elevated levels of pollutants in the atmosphere can result in an immune response that is either abnormal or inadequate.

Antibiotics: The overuse of antibiotics has been demonstrated to alter the intestinal microflora, thus inducing immune system disorders that can result in the onset of UC [36].

2.3 Immune factors

A diet from the Western dietetic tradition has been demonstrated to result in the production of excessive amounts of free radicals, which in turn initiate a cascade of oxidative stress and inflammatory signalling pathways, including cytokines such as TNF-α, IL-1β, IL-6 and IL-13. This results in the activation and hyperactivity of the immune system, involving, among others, Th2 lymphocytes. The recruitment of immune cells has been demonstrated to result in a further increase in reactive oxygen species, thereby maintaining inflammation [19][23][28][33]. Individuals afflicted with UC also experience immune system dysregulation, which results in increased activation of pro-inflammatory cytokine secretion pathways and reduced activation or abnormal regulation of inflammatory inhibition pathways [35].

3. Fibre

A pivotal aspect of dietary fibre pertains to its capacity to enhance the composition of the gut microbiota, a process that has been demonstrated to play a pivotal role in the prevention of numerous diseases. The composition of the material can be categorised into two distinct types: water-soluble fibre (SDF) and water-insoluble fibre (IDF). It is evident that each fraction possesses a distinct function within the broader context of the sequence [1]. Insoluble fibre, which includes cellulose, hemicellulose and lignin, has been shown to reduce intestinal transit time and increase water binding, thereby increasing stool volume. This has been demonstrated to enhance intestinal peristalsis, thereby alleviating constipation [2]. Soluble fibre, including fructooligosaccharides, galactosaccharides, pectins, β-glucans and inulin, is readily fermented by intestinal bacteria into short-chain fatty acids (SCFA), including butyric, propionic and acetic acids. The function of these substances is to regulate the pH of the intestinal environment, nourish colonocytes and inhibit the expression of inflammatory mediators [3][4][5].

3.1 β-glucan

It is evident that β-glucan constitutes a significant fraction of SDF, and it has been demonstrated that it possesses immunomodulatory and anti-inflammatory properties. Research has demonstrated that the isolation of β-glucan from mountain barley has the capacity to reduce C-reactive protein (CRP) levels in patients diagnosed with UC. Concurrently, an enhancement in clinical parameters was observed within the study groups. It is important to note that no adverse effects were observed after the consumption of β-glucan [6]. The anti-inflammatory effect of β-glucan is most likely attributable to its fermentation by intestinal bacteria to SCFA,

particularly butyrate, which has been demonstrated to possess anti-inflammatory properties [7]. A further study was conducted with patients suffering from symptoms of IBD and IBS. A mixture of β-glucan, inositol and digestive enzymes was administered to patients taking mesalazine. The control group consisted of individuals who were administered mesalazine in isolation. The study group reported a greater reduction in abdominal pain and bloating, and an improvement in general well-being, in comparison to the control group. The anti-inflammatory effect of β-glucan has been posited as a potential explanation for this phenomenon, given its ability to reduce the production of pro-inflammatory cytokines such as IL-10, IL-12 and TNF-α [8].

3.2 Pectin

The substance has been found to be present in fruit, and has been shown to have a clear protective and anti-inflammatory effect in UC. The most recent preclinical studies on animal models demonstrate that pectin supplementation exerts a substantial inhibitory effect on inflammation and promotes colonic epithelial regeneration. The mechanism of action of pectin is twofold. The initial process entails the fermentation of SCFAs, predominantly butyrate, which serves to curtail the expression of pro-inflammatory cytokines, namely TNF-α, IL-6 and IL-1β. The second is modulation of the immune system. Pectin has been demonstrated to inhibit the production of pro-inflammatory interleukins IL-6 and IL-1β, and to lead to increased expression of NLRC4 and IL-1 receptor antagonists (IL-1Ra). This, in turn, limits the inflammatory cascade and protects the mucous membrane from further damage. Research has indicated that the most beneficial immunomodulatory effect is obtained after the use of pectins with a high content of neutral sugar chains [9][10].

3.3 Resistant starch

Resistant starch is categorised as part of the insoluble fibre fraction, and is distinguished by its high resistance to hydrolysis in the small intestine, as well as its capacity to undergo fermentation in the distal sections of the digestive tract. Resistant starch has been demonstrated to promote the growth of beneficial bacteria, which in turn leads to increased production of SCFAs, with butyrate being the main constituent.[11] This increase in SCFAs has been shown to inhibit inflammation. A further significant function is the reduction of the pH level in the colon, which has been demonstrated to inhibit the growth and proliferation of pathogenic microbes, whilst simultaneously creating a suitable environment for normal microbiota [12].In both animal models and clinical studies, RS supplementation has been shown to significantly reduce colonic mucosal damage, increase the clinical remission rate in patients with IBD, and reduce the risk of flare-ups [12]. When adequate dietary intake is ensured, resistant starch has been demonstrated to reduce colonocyte apoptosis, thereby increasing the integrity of the intestinal wall. It is hypothesised that this phenomenon is attributable to elevated levels of SCFA production [13].

3.4 Inulin

Inulin, a prebiotic, is found in chicory, artichokes, garlic and asparagus. It undergoes bacterial fermentation in the large intestine, resulting in the production of SCFA [18]. Inulin has a beneficial effect on the microbial balance in the intestines by promoting the growth of beneficial bacteria and reducing the number of potentially pathogenic bacteria. It also reduces the expression of TNF-α, IL-1β and IL-6, while increasing the level of anti-inflammatory cytokines, particularly IL-10 [14, 15]. Clinical studies have shown that, after seven days of supplementation with inulin and oligofructose, patients with active ulcerative colitis experienced a decrease in faecal calprotectin levels and a reduction in gastrointestinal symptoms. Notably, there were no adverse effects associated with the use of the supplement [16]. Another clinical study evaluated the effects of inulin supplementation at doses of 7.5 g and 15 g per day for nine weeks (n = 12 and 13, respectively). Significant clinical improvement and a decrease in disease activity were reported in 77% of patients receiving the higher dose. A marked increase in butyrate production was also observed in the group receiving the higher dose of inulin [17].

3.5 Hemicellulose

Hemicellulose occurs naturally in plant seeds, barley bran and rye. It has anti-inflammatory and probiotic properties [19]. It modulates the immune response by reducing the production of pro-inflammatory cytokines, including TNF-α, IL-6 and IL-1β, while stimulating the production of the anti-inflammatory cytokine IL-10. Preclinical studies have demonstrated that hemicellulose decreases the abundance of M1-phenotype macrophages and Th1 and Th17 lymphocyte subpopulations within the intestinal mucosa. This reduction in cell populations results in reduced pro-inflammatory cytokine production and suppression of local

inflammation [20, 21]. In a clinical study, patients with mild to moderate UC were given 30 g of barley fibre three times a day for four weeks. This resulted in reduced intestinal inflammation and restoration of the colonic epithelium [22].

4. Micro- and macroelements

4.1 Vitamin D

Vitamin D is a fat-soluble vitamin. The most biologically active form in the human body is 1, 25-dihydroxyvitamin D3 (1, 25$(OH)_2D_3$, or calcitriol), an important regulator of the immune system. Some immune cells have a nuclear receptor for 1, 25D (VDR), which is found in T and B lymphocytes, as well as in monocytes and macrophages [23]. Activation of the VDR reduces the activation of the NLRP6 inflammasome, thereby lowering the production of pro-inflammatory cytokines and apoptosis markers in enterocytes [24]. Observations have shown that vitamin D deficiency is associated with a higher risk of exacerbations, a more aggressive form of UC, a poorer response to biological therapy, and a higher correlation with the incidence of colorectal cancer in the patient population [23, 25]. In light of these reports, it is recommended that serum vitamin D3 levels are maintained at a minimum of 30 ng/ml in all patients diagnosed with UC [23]. Due to its high bioavailability, low cost, and immunoregulatory properties relevant to UC, pharmacological supplementation is recommended for patients with UC. In the context of cancer prevention, supplementation with vitamin B9 is also recommended for patients with UC, as its levels are reduced due to inflammation of the gastrointestinal tract, increased utilisation by cells, and drug interactions, particularly with sulfasalazine [26].

4.2 Curcumin

It is a polyphenol obtained from the rhizome of Curcuma longa through a process of cooking and drying the plant. Due to its antioxidant, anti-cancer and anti-inflammatory properties, it has been the subject of research for years. It works by inhibiting IFN-γ signalling, thereby inhibiting epithelial cell migration, sealing the intestinal barrier and accelerating mucosal healing. It can also inhibit the expression and release of pro-inflammatory cytokines, such as IL-1, IL-6 and TNF-α [27, 28]. In a study involving 50 patients with mild to moderate UC, curcumin at a dose of 3 g/day was combined with oral and rectal mesalazine therapy. In the study group (n = 14), there was an increase in the percentage of patients in clinical (54% vs. 0%, p = 0.01) and endoscopic (38% vs. 0%, p = 0.04) remission, as well as a higher clinical response rate (65% vs. 13%, p = 0.01). Another study compared the recurrence rate in 89 patients receiving curcumin at a dose of 2 g/day or a placebo, both in combination with mesalazine and sulfasalazine, all of whom were in remission for UC. After six months of follow-up, the recurrence rate was lower in the study group (5% vs 21%, p = 0.04) [28]. According to some reports, consuming as little as 1.5 g of curcumin per day can be effective in achieving remission in patients with mild to moderate UC [27].

4.3 Zinc

Zinc is a micronutrient that modulates the activity of both the innate and acquired immune systems. It does this by limiting the expression and release of pro-inflammatory cytokines such as TNF-α, IL-6 and IL-1β, as well as the enzyme myeloperoxidase. This results in reduced local inflammation in the large intestine. At the same time, zinc increases the synthesis of the anti-inflammatory cytokine IL-10. An important mechanism of zinc's action is modulation of the NF-κB signalling pathway, which activates the expression of numerous proteins that are responsible for developing and maintaining the inflammatory response, as well as producing pro-inflammatory enzymes such as COX-2. Zinc also inhibits the PI3K/Akt pathway, thereby reducing NF-κB activation. These modulatory effects lead to the inhibition of an excessive immune response in UC [30]. Zinc deficiency occurs in approximately 30% of patients with IBD. This is due to chronic inflammation and mucosal damage in UC patients, as well as an absence of zinc storage mechanisms. Zinc deficiency has been shown to correlate with poor clinical outcomes, an increased risk of exacerbations and hospitalisation, and a greater number of disease-related complications. Studies have shown that normalising zinc levels improves treatment outcomes in patients with IBD [29] and that omega-3 fatty acid supplementation in addition to zinc supplementation increases the clinical remission rate [31].

4.4 Omega-3 acids

Omega-3 fatty acids, which primarily comprise EPA and DHA, are polyunsaturated fatty acids that have been proven to have immunomodulatory and anti-inflammatory properties. They regulate the cyclooxygenase and lipoxygenase pathways involved in the synthesis of leukotrienes and prostaglandins, competing with omega-6 fatty acids, which have pro-inflammatory effects. Omega-3 fatty acids inhibit the synthesis of IL-6 and TNF-α, as well as reducing the production of H_2O_2, one of the activators of NF-κB which is responsible for the expression of numerous inflammatory mediators [32]. The International Organisation for the Investigation of Inflammatory Bowel Disease (IOIBD) recommends increasing omega-3 intake from sources such as fish oils, fresh fish and olive oil, noting that excessive red meat consumption has a significant pro-inflammatory effect [33]. Clinical studies indicate that anti-inflammatory effects can only be observed with a minimum daily intake of 2 g of omega-3 fatty acids [23]. A study in rats showed that a combination of omega-3 fatty acids and 5-aminosalicylic acid (5-ASA) is more effective than a higher dose of 5-ASA alone in reducing NF-κB activation and producing better results in UC therapy [32].

5. Conclusions

The number of people affected by inflammatory bowel diseases is increasing every year. These diseases mainly affect people in North America, Europe and Asia. Those affected are predisposed by their diet, particularly the Western diet (high in sugar, saturated fatty acids and trans fats, and low in fibre), as well as by a fast-paced and stressful lifestyle and genetic and immunological factors. Despite the significant impact of diet on the onset and progression of UC, few substances have been proven to prevent, inhibit or reduce the activity of UC. Available clinical and preclinical data confirm that targeted dietary interventions, such as increasing soluble fibre intake and taking supplements of curcumin, zinc, vitamin D3 and vitamin B9, may alleviate symptoms and prolong remission in patients with IBD by improving the gut microbiome and inhibiting inflammatory pathways. While results regarding the effectiveness of omega-3 fatty acids in maintaining remission remain inconclusive, new studies suggest that they are effective when used alongside other substances or drugs discussed here. However, multicentre randomised studies with uniform standardisation of doses, forms of administration, and analysis of interactions between dietary components and pharmacotherapy are required to develop clinical recommendations.

Acknowledgments.

Author's Contributions.

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Data collection and/or compilation - Patryk Kowalczyk, Kamil Nowak, Alicja Stryczek-Schlusche, Aleksandra Gęsińska, Filip Bracichowicz.

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All authors have read and agreed to the published version of the manuscript.

Funding statement: The study did not receive any external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Conflict of Interest Statement: The authors declare that they have no conflict of interest.

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