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Interactions between diet, gut microbiota, and immune response in inflammatory bowel disease

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ABSTRACT

Inflammatory bowel disease (IBD) is a chronic disease associated with recurrent inflammation of the gastrointestinal tract. Recent studies indicate that diet and gut microbiota have an important influence on the development of the disease, its course, and treatment effectiveness. This review summarizes knowledge about diet function in a comprehensive review of current literature concerning the relationship between nutrition, gut microbiota composition, intestinal barrier integrity, and inflammatory activity in patients with IBD. The analyzed studies showed that disturbances in the composition of gut microbiota, known as dysbiosis, are one of the main factors involved in IBD development. Dysbiosis is characterized by lower microbial diversity, a reduced number of beneficial bacteria producing short-chain fatty acids (SCFAs), and an increased number of microorganisms that may promote inflammation. Special attention has been given to butyrate-producing bacteria because butyrate helps maintain the integrity of the intestinal barrier and supports proper immune system function. The review also describes the effects of pharmacological treatment, including biologic therapies and aminosalicylates, on reducing inflammation and restoring microbial balance. We have highlighted the diet as one of the most important nutritional principles for patients with inflammatory bowel disease (IBD). A diet rich in fruits, fiber, omega-3 fatty acids, legumes, vegetables, and fermented foods supports the growth of beneficial gut bacteria and supports the functioning of the intestinal barrier. However, Western diets and the consumption of processed foods can exacerbate dysbiosis, increase intestinal permeability, and promote inflammation.

Keywords: Inflammatory bowel disease, Crohn's disease, Microbiota, Ulcerative colitis, Mediterranean diet

1. INTRODUCTION

Inflammatory bowel disease is a group of digestive system diseases that include Crohn's disease and ulcerative colitis. There are moments of remission and exacerbation due to immune system malfunction and inflammation. Inflammation results from immunological, microbiological, genetic, and environmental factors (Hashash et al., 2025; Bischoff et al., 2022). An important mechanism is the abnormal

function of the intestinal epithelium due to an immune response. Antigen-presenting cells play an important role in the development of inflammation, including activated dendritic cells and macrophages that secrete pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin 1β (IL-1β), and interleukin 6 (IL-6) (Bischoff et al., 2022).

In recent years, the role of nutrition in the pathogenesis and treatment of inflammatory bowel disease (IBD) has become the subject of intensive clinical and experimental research. In the scientific literature, it is emphasized that the way of eating can affect the immune activity of the intestines, the mucosal barrier, and the microbial metabolism of the host.

Despite the increasing number of clinical studies and systematic reviews of the effects of diet on changes in the composition of microbiota in the course of IBD, there is still a shortage of data consolidation on the relationship between specific dietary patterns and clinical, microbiological, and immunological effects. Current publications indicate that certain dietary interventions may be associated with a reduction in the activity of intestinal symptoms, improvement of quality of life, modification of markers of inflammation, and correlation with changes in the composition of the intestinal microbiota, but the extent, consistency, and strength of these effects vary and require further analysis.

The main objective of this paper is a systematic review and synthesis of empirical and review studies available in scientific literature on the impact of various nutritional strategies on the state of the microbiota and the course of non-specific enteritis. The paper aims to identify and discuss the biological mechanisms through which diet affects the gut microbiota, the mucosal barrier, and the body's immune response in the context of IBD, and to analyze the available clinical evidence on the effects of the Mediterranean diet on symptom severity, frequency of remission, quality of life, and nutritional status of IBD patients.

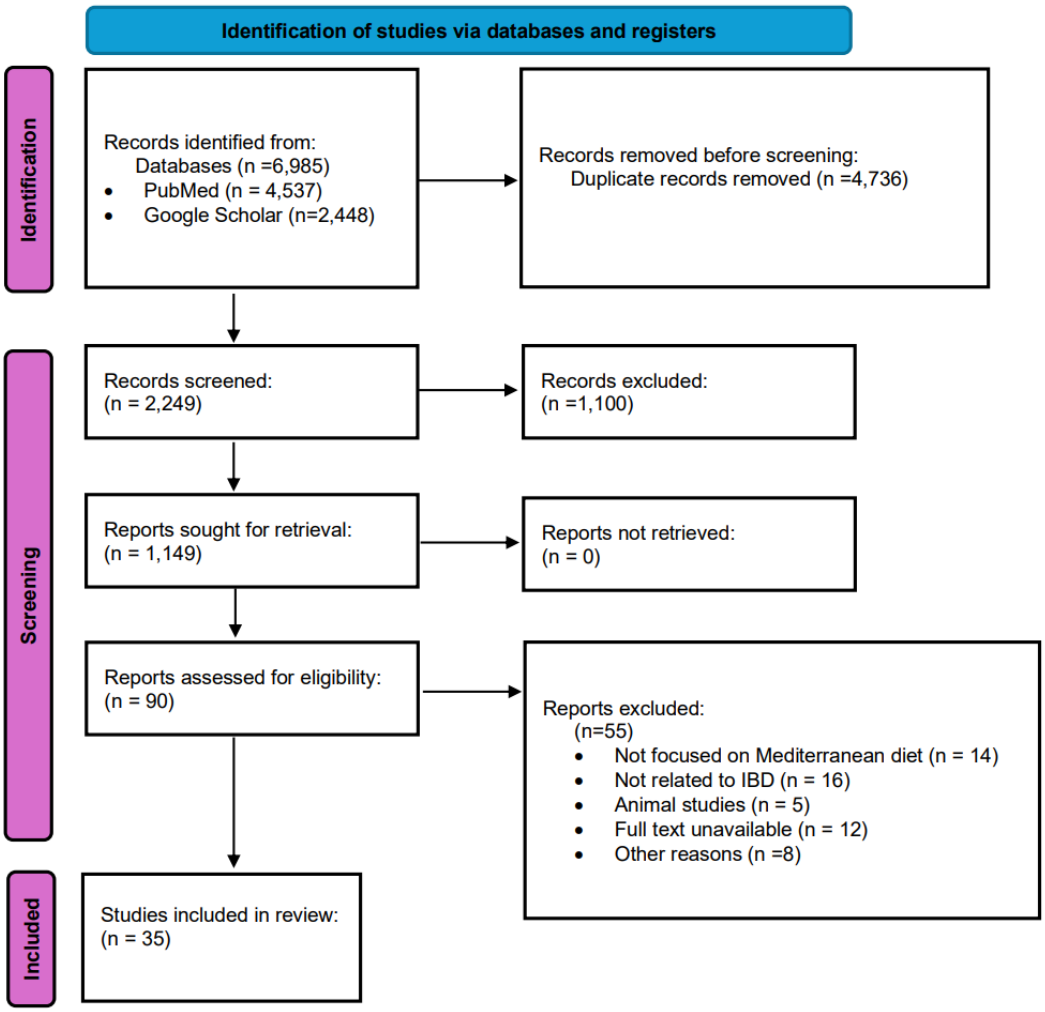

Figure 1. PRISMA flow diagram.

2. REVIEW METHODS

A literature review was conducted in PubMed and Google Scholar databases, covering English-language publications from 2015 to 2026. The following keywords were used: „Mediterranean diet", „IBD diet", „Crohn's disease diet", „ulcerative colitis diet", „microbiota in IBD", and „microbiota and diet". Original research papers, clinical trials, observational studies, systematic reviews, and meta-analyses were included in the analysis. We made the selection based on titles, abstracts, and full texts. The article screening process followed the PRISMA guidelines (Figure 1).

3. RESULTS

Definitions and Differences in Clinical Presentation

Ulcerative colitis is a form of chronic inflammatory bowel disease characterized by remissions and relapses. Superficial inflammation of the mucosa is characteristic and may affect only in a continuous, circular manner, in the proximal direction, occupying to varying degrees the large intestine. Diagnosis is based on a complex assessment of clinical data, inflammatory parameters, endoscopic image, and histopathological examination. There is no single gold standard for diagnosis, but colonoscopy with the collection of slices from different sections of the colon is the most sensitive and specific method of confirming the diagnosis (Le Berre et al., 2023).

Clinical picture

In the UC, the most common symptom is rectal bleeding. Other symptoms include frequent passing of loose stools and mucous discharge. There is also a painful need to have a bowel movement, abdominal cramps, and bowel movements at night (Le Berre et al., 2023). In about 15% of patients, the disease can manifest as an acute severe form. In most cases, the onset of the disease is insidious, and the symptoms persist for several weeks before the patient is reported to the doctor (Le Berre et al., 2023; Kucharzik et al., 2020). Parenteral symptoms occur in 20-35% of patients. They occur earlier than the gastrointestinal symptoms. They most often occur as inflammation of peripheral joints. If patients have severe disease, they are at risk of venous thromboembolism (Le Berre et al., 2023).

Endoscopic and histopathological image

Endoscopically, ulcerative colitis is characterized by continuous inflammation of the mucous membrane of the large intestine, with a clear boundary between the altered and unchanged tissue. In mild form, erythema, congestion, and partial loss of vascular drawing are observed. The moderate form is characterized by complete loss of vascular drawing, fragility of the mucous membrane, and the presence of erosions, while the severe form is characterized by spontaneous bleeding of ulcers (Le Berre et al., 2023). In histopathological examination, diffuse crypt architecture disturbances, mucosal atrophy, and inflammatory infiltration in the lamella with the presence of lymphocytes and plasmocytes are found (Kucharzik et al., 2020).

Pharmacological treatment of ulcerative colitis

Treatment requires individualization of the procedure and cooperation of a multidisciplinary team. Pharmacological therapy is the basis of treatment and allows for remission in most patients. Surgical treatment remains an effective and, in many cases, radical method, used in life-threatening situations, ineffectiveness of conservative therapy, or cancer transformation (Tian et al., 2025).

Pharmacological treatment

In acute severe cases, the treatment of choice is intravenous glucocorticoids in high doses, while treating water-electrolyte disorders, anemia, and malnutrition. In the event of ineffectiveness of steroid therapy, rescue treatment with cyclosporine A is considered, which can avoid urgent colectomy in about half of patients, although it is associated with a high rate of early relapses. In mild and moderate forms of the disease, the basis of treatment is aminosalicylates (5-ASA), used both in induction and maintenance of remission. In patients with recurrent course or steroid dependence, it is advisable to include immunomodulatory drugs (e.g., azathioprine). In the case of moderate and severe forms of the disease resistant to conventional treatment, biological therapies (including anti-TNF antibodies) and other targeted drugs are used (Tian et al., 2025).

Crohn's disease is a chronic, idiopathic, non-specific, relapsing-remitting inflammatory bowel disease, which may include any section of the gastrointestinal tract, which is the basis for the development of penetrating complications and fibrosis leading to mechanical obstruction. The most common location remains the final section of the ileum, in the corner. A characteristic feature is the

"skip lesions", consisting of alternating occurrence of fragments of inflamed mucosa and unchanged sections. The inflammatory process can lead to thickening of the intestinal wall, stenosis, formation of external and internal fistulas, and the formation of peri-intestinal abscesses. (Tian et al., 2025)

Clinical symptoms

Symptoms depend on the severity of inflammation and location. Typically, there is chronic diarrhea without blood, and abdominal pain in the lower right part of the abdomen. It is also accompanied by weight loss, fever, and anemia. When the small intestine is involved, absorption is destroyed. If the large intestine is involved, it is bloody diarrhea. There are also often fissures, fistulas, and abscesses (Tian et al., 2025).

Approximately 25–40% of patients also have other symptoms, including arthritis, papular rashes, gangrene, uveitis, and primary sclerosing cholangitis (Tian et al., 2025; Cushing & Higgins, 2021).

Fault finding

Diagnosis of Crohn's disease is based on the correlation of clinical, endoscopic, histopathological, laboratory, and imaging data. The characteristic results are laboratory tests taking into account the increased concentration of CRP and OB, anemia, hypoalbuminemia, and increased concentration of calprotectin in feces (Cushing & Higgins, 2021). Colonoscopy with intubation of the final section of the ileum is the basic diagnostic examination. Typical endoscopic features include: segmental ulcers, the image of “cobble-stoning", deep, oblong ulcers, and narrowing of the lumen of the intestine. In histopathological examination, transmural inflammation and the presence of non-serviant granulomas (although they do not occur in all patients) are found (Tian et al., 2025).

Pharmacological treatment in Crohn's disease

Pharmacotherapy

Pharmacological treatment of CD includes different classes of drugs, selected according to the severity of the disease, the location of inflammatory lesions, and the response to previous treatment. The therapy regimen is divided into a remission induction phase, which aims to rapidly reduce inflammation activity and reduce symptoms, and a maintenance therapy to maintain remission (Cushing & Higgins, 2021).

We use glucocorticosteroids at the beginning of the disease. They effectively calm acute inflammation. They are not used as maintenance therapy because they cause numerous side effects. Immunosuppressive drugs, for example, azathioprine, 66-mercaptopurine and methotrexate, are used to prevent relapses and maintain remission. We give them to patients with moderate to severe disease (Cushing and Higgins, 2021). Recently, biological therapies have become very important. Mainly monoclonal antibodies directed against TNF-α (e.g., infliximab, adalimumab), integrins, or interleukin (e.g., vedolizumab, ustekinumab). We are also increasingly using modern small-molecule inhibitors (e.g., YAK inhibitors) (Cushing and Higgins, 2021).

The effectiveness of pharmacocolagia depends on the action of drugs on the microflora (Deleu et al., 2024). This allows symptoms to be reduced by repairing the epithelium and restoring normal intestinal microflora (Haifer et al., 2022).

Treatment with infliximab or adalimumab leads to a significant increase in microbiota alpha diversity in responders (Haifer et al., 2022). It has been shown that the reduction of TNF-α in the intestinal environment promotes the expansion of butyrate-producing bacteria such as Faecalibacterium prausnitzii, which secondarily stabilizes tight junctions and reduces pathological permeability of the epithelium. Mesalazine, in addition to its anti-inflammatory effect, exhibits antioxidant properties that reduce aerobic stress in the lumen of the intestine (Deleu et al.,2024; Costello et al., 2019). This allows the reduction of aerobic pathobionts (e.g., Enterobacteriaceae) population in favor of anaerobic commensals, which is necessary to inhibit the chronic stimulation of TLR4 receptors by lipopolysaccharides (LPS) (Haifer et al., 2022; Costello et al., 2019). Corticoids are effective in inducing remission. When used for a long time, they can worsen dysbiosis. This makes it difficult to maintain a lasting therapeutic narrative if disease-modifying eks are not benefited (Costello et al., 2019; Van Der Vlist et al., 2022).

Conclusions from the analysis of drug-microbiota interactions suggest that the patient’s microbiological profile before the start of treatment may serve as a predictive biomarker of response to specific biological preparations (Deleu et al.,2024; Van Der Vlist et al., 2022). The integration of pharmacotherapy with targeted modulation of the microbiome, e.g., through diet or FMT, seems to be an essential step toward personalized medicine in IBD (Haifer et al., 2022).

The effect of classical pharmacotherapy on the gut microbiome and the host microbiota axis

Intestinal microflora is a large number of microorganisms that regulate immune homeostasis, body nutrition, and the continuity of the intestinal barrier. In inflammatory bowel diseases, there are disturbances in the composition and function of the microbiota, which are called dysbiosis (Akiyama et al., 2024). Current research shows that dysbiosis causes the development and progression of inflammatory bowel diseases. This is because it affects the immune response, increases the permeability of the intestinal barrier, and activates inflammatory processes. It is very important to reduce the amount of short-chain fatty acids (SCFA) and, at the same time, increase the number of pro-inflammatory aerobic and anaerobic bacteria (Akiyama et al., 2024).

According to the so-called "oxygen hypothesis", chronic inflammation leads to an increase in the concentration of oxygen in the intestinal lumen, which promotes colonization by aerobic bacteria and limits the development of beneficial, obligatorily anaerobic bacteria. This mechanism may be one of the main factors perpetuating chronic inflammation in IBD (Akiyama et al., 2024).

Studies show that patients with inflammatory bowel disease have a low diversity of bacterial flora. In healthy people, it should be very diverse. Reduced diversity is more pronounced in Crohn's disease than in ulcerative colitis. Microbiological analysis showed more bacteria causing inflammation. They are bacteria of the genera Escherichia, Shigella, and Citrobacter. There are more of these strains in Crohn's disease. There are quite a few strains of Escherichia coli because they can cross the intestinal barrier and multiply in macrophages (San-Martin et al., 2025).

In ulcerative colitis, there are more anti-inflammatory bacteria. These are Prevotella and Comamonas bacteria, which eliminate other pathogenic microorganisms. Studies have shown a reduction in the number of butyrate-producing bacteria. Such bacteria include Faecalibacterium prausnitzii and others in the family Lachnospiraceae. These changes can lead to a weakening of the integrity of the intestinal barrier, an increase in epithelial permeability, and an increase in chronic inflammation. There is evidence of a link between microflora and disease severity. Patients with severe disease have fewer Akkermansia muciniphila bacteria and more Pantoea carbekii bacteria. This allows us to use some microorganisms as biomarkers. These measures will allow for the assessment of disease activity (San-Martin et al., 2025).

Differences in intestinal microbiota in Crohn's disease and ulcerative colitis, and modern methods of microbiota diagnosis

Dysbiosis as a key component of IBD pathogenesis

Intestinal microflora is a large number of microorganisms that regulate immune homeostasis, body nutrition, and the continuity of the intestinal barrier. In inflammatory bowel diseases, there are disturbances in the composition and function of the microbiota, which are called dysbiosis. Current research shows that dysbiosis causes the development and progression of inflammatory bowel diseases. This is because it affects the immune response, increases the permeability of the intestinal barrier, and activates inflammatory processes. It is very important to reduce the amount of short-chain fatty acids (SCFA) and, at the same time, increase the number of pro-inflammatory aerobic and anaerobic bacteria (Akiyama et al., 2024).

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Differences in microbiota between Crohn's disease and ulcerative colitis

The immunological mechanisms are identical. Despite this, diseases have different microbiome compositions. Low diversity of gut microbiota is more typical in Crohn's disease. A reduction in the number of butyrate-producing bacteria is well described. These are Faecalibacterium prausnitzii, Roseburia spp., and other Firmicutes. Butyrate is a source of energy for colonocytes, strengthens the intestinal barrier, and is anti-inflammatory. In Crohn's disease, the number of bacteria from the Enterobacteriaceae family increases significantly. They can penetrate through the mucus layer. In addition, they can adhere to enterocytes and activate Toll-like receptors. In these bacteria, the expression of genes related to bacterial virulence and antibiotic resistance increases (Kang et al., 2023).

In UC, the number of relatively aerobic bacteria and bacteria of oral origin increases more frequently. These are the bacteria Veillonella dispar and Haemophilus parainfluenzae. Studies show that these bacteria cause more severe disease and increase the risk of colectomy (Akiyama et al., 2024).

Significant differences also apply to the mycobiome and the intestinal virus. In patients with Crohn's, an increased presence of Escherichia coli-associated bacteriophages and disorders in the composition of intestinal fungi, especially of the Saccharomyces genus, is observed. Interactions between bacteria, viruses, and fungi may play an important role in maintaining the chronic inflammatory process (Kang et al., 2023).

Meta-analytic studies involving over 1,700 patients have shown that a common feature of the microflora of both diseases is a decrease in the number of anaerobic bacteria and an increase in oxygen-tolerant bacteria. This is very noticeable in the mouth. The results show that microbiome changes are an important biomarker (Akiyama et al., 2024; Kang et al., 2023).

Functional significance of microbiota changes

Currently, microbiota analysis includes not only the assessment of taxonomic composition, but also the analysis of metabolic functions of microorganisms. In IBD, the pathways responsible for SCFA production, glycan metabolism, synthesis of secondary bile acids, and regulation of the immune response are observed. In Crohn's disease, the number of bacteria belonging to the Enterobacteriaceae family increases the most. They can penetrate through the mucus. In addition, they can adhere to enterocytes and activate Toll-like receptors. These bacteria also have increased expression of genes related to bacterial virulence and antibiotic resistance (Kang et al., 2023).

An abnormal microbiome reduces the fermentation of dietary fiber. This causes a decrease in butyrate production and a weakening of the intestinal barrier (Akiyama et al., 2024; Kang et al., 2023).

Modern diagnostics of non-specific enteritis is evolving toward the use of specific microbiological signatures as non-invasive differentiating biomarkers. The latest metagenomic analyses indicate that precise determination of the deficit of Faecalibacterium prausnitzii strain with simultaneous increase in the number of adhesive-invasive Escherichia coli (AIEC) allows not only to confirm the diagnosis of Crohn's disease, but also to predict the risk of early postoperative recurrence.

Intestinal barrier and immune response

The intestinal barrier is a complex protective system, the main function of which is to maintain intestinal homeostasis and protect the body from the penetration of harmful factors from the intestinal lumen into the host tissues. A properly functioning intestinal barrier promotes effective absorption of nutrients, preservation of intestinal homeostasis, and protection of the body by creating a barrier preventing the penetration of pro-inflammatory molecules, such as pathogens, toxins, and antigens, from the environment of light to the tissues of the mucous membrane and the circulatory system (An et al., 2022). The intestinal barrier itself consists primarily of a mechanical, chemical, immunological, and microbiological component. The intestinal barrier consists of the intestinal epithelium and the mucus that covers it.

The intestinal epithelium is made up of a single layer of epithelial cells (IEC), which acts as a selectively permeable barrier between the lumen of the intestine and the internal environment of the body. The single-layer intestinal epithelium is made up of several types of cells and forms specialized structures in the form of intestinal crypts, found in both the small and large intestine, and intestinal villi present only in the small intestine. At the base of the crypts are located intestinal stem cells, which undergo intensive proliferation and differentiation, giving rise to various types of epithelial cells, such as enterocytes, cup cells, Panetha cells, microfold cells, tubal cells, and enteroendocrine cells. Cup cells are responsible for the secretion of mucins – cross-linked proteins forming the layer of mucus covering the epithelium, while Paneth cells produce peptides with antimicrobial activity. Rare tubules have a regulatory function in the type 2 immune response, while M cells participate in the collection of antigens from the intestinal lumen, initiating a specific immune response to microbes. Mucus contains many protective factors, including peptides that act against microorganisms and

immunoglobulin A. These substances do not allow microorganisms to grow and come into contact with the intestinal epithelium (Wallaeys et al., 2023; Clevers, 2013).

A vital role in maintaining the integrity of the intestinal barrier is played by tight junctions (TJ), which are complexes of protein complexes located in the upper parts of the lateral membranes of intestinal epithelial cells. The interaction of TJ proteins with the actin cytoskeleton is crucial for maintaining their normal structure and enables cytoskeletal control of the integrity of the barrier formed by TJ. Disorders of intestinal barrier function are closely related to the development of inflammatory bowel diseases. Defects and disorders of intestinal barrier homeostasis have been directly linked to IBD. In addition, it is believed that even before the clinical manifestation of IBD, the function of the intestinal barrier is impaired and its permeability increases. As a consequence, the components of the intestinal content penetrate into the plaque, initiating the activation of the innate and acquired response, which ultimately leads to the development of intestinal inflammation.

Disruption of the TJ function, induced by cytokines leading to the activation of immune response and inflammation of tissues, is believed to play an important role in the initiation and/or progression of many intestinal and systemic diseases (An et al., 2022).

It is also assumed that the increased permeability of the intestinal epithelial barrier (IEB) remains closely related to dysbiosis. This relationship has a two-way character – an increase in permeability may promote the development of microbiota disorders, while changes in the composition of intestinal microflora may have a secondary effect on the modification of intestinal permeability.

Mechanisms of the effect of diet on IBD

IBD is associated with an altered gut microbiota profile, in which a decline in bacterial diversity is observed, constituting a typical manifestation of dysbiosis (Martin-Gallausiaux et al., 2021). In recent years, the growing interest in the scientific community has been raised by short-chain fatty acids (SCFA), which have clear anti-inflammatory properties and potential anticancer effects (Zhang et al., 2022). Therefore, they can play an important role both in the alleviation of IBD symptoms and in the prevention of inflammatory bowel diseases (Shin et al., 2023).

SCFAs are fatty acids containing fewer than six carbon atoms. They can be synthesized endogenously in the liver as a result of metabolic changes of the body, but their primary source remains the activity of the intestinal microbiota. Intestinal bacteria produce SCFA by fermentation of undigested dietary fiber fractions and resistant starch (Sankarganesh et al., 2025).

The most common SCFAs absorbed in the gastrointestinal tract are acetate (C2), propionate (C3), and butyrate (C4). Their main production takes place in the colon during the fermentation of the food fiber. This process involves various metabolic pathways: In the case of acetate, mainly the Wood-Ljungdahl pathway, for butyrate, the acetyl-CoA pathway, whereas propionate is formed in the acrylate, succinate, or propanediol pathways (Chambers et al., 2015). It should also be noted that acetate, propionate, and butyrate can also be synthesized from amino acids, but this mechanism is used by less than 1% of intestinal bacteria (Martin-Gallausiaux et al., 2021).

The production of individual SCFAs is related to the activity of specific groups of intestinal bacteria. Acetate is produced primarily by bacteria of the genera Akkermansia, Bifidobacterium, Bacteroides, Prevotella, Ruminococcus, Clostridium, and Streptococcus, as well as by the species Blautia Hydrogenotropica. Propionate is formed mainly with the participation of bacteria such as Bacteroides, Dialister, Veillonella, Salmonella, Megasphaera Elsdenii, Coprococcus Catus, Roseburia Inulinivorans, Phascolarctobacterium Succinattens, and Ruminococcus Obeum. In turn, the production of butyrate is attributed primarily to the bacteria Coprococcus Comes, Anaerostipes, Eubacterium Rectale, Coprococcus Catus, Eubacterium Hallii, Roseburia, and Faecalibacterium Prausnitzii (Martin-Gallausiaux et al., 2021).

SCFAs have different metabolic and physiological functions. Acetate participates in the lipogenesis process, initiating the synthesis of triglycerides and fatty acids. The resulting products store energy and form cell membranes. When there is too much acetate, it can turn into fat in adipose tissue. Then it can be an additional source of energy for the body. Acetate also regulates body weight, lipid metabolism, and prevents insulin resistance. Propionate participates in the regulation of appetite, contributing to the reduction of food intake. In addition, it affects the regulation of blood glucose levels and can modulate the process of gluconeogenesis occurring in the liver (Chambers et al., 2015). Butyrate has strong anti-inflammatory properties and other beneficial health-promoting effects. Its anti-inflammatory effect is associated, among others, with inhibition of the synthesis of pro-inflammatory cytokines (Zhang et al.,2022). In addition, it is the main source of energy for colonocytes, playing an important role in maintaining the proper function of the intestinal epithelium (Chambers et al., 2015).

SCFA acts by activating G protein-coupled receptors (GPCRs). These are the GPR41, GPR43, and GPR109 receptors (Wu et al., 2017). When GPR43 is activated, gA production increases, and inflammatory processes in the intestines decrease. Amphiregulin expression in

dendritic cells is also increased, which aids in tissue repair and reduces nuclear factor kappa-B activity (Chen et al., 2018). In turn, activation of GPR109a leads to a decrease in the activity of AKT and NF-κB P65 signaling pathways and a reduction in IL-23 production, resulting in suppression of ILC3 and reduction of colitis. The effect on GPR109a also allows correction of pathological angiogenesis and reduction of inflammatory changes (Hertati et al., 2020). SCFA can also regulate macrophage activity by affecting receptor.

The therapeutic use of microbiota metabolites, referred to as postbiotics, is currently one of the most promising directions of personalized medicine in IBD. Direct stimulation of the GPR109a receptor by butyrate leads to suppression of the NF-κB signaling pathway in mucosal macrophages, which results in decreased secretion of pro-inflammatory cytokines (TNF-α, IL-6) and accelerated intestinal epithelial restoration by stabilizing tight junctions (Vissers et al., 2022).

Effect of diet on clinical symptoms and inflammatory activity

In the course of IBD, diet can modulate both subjective symptoms and objective inflammatory indicators. The analyzed publications emphasize that dietary interventions, especially enteral nutrition, can lead to a reduction in the indicators of disease activity and lower levels of inflammatory markers such as CRP or calprotectin in feces. The elimination diet reduces abdominal pain, bloating, and diarrhea. We do not know its effect on the depth and extent of the inflammatory process. The importance of the model of nutrition characteristic of the so-called Western diet – rich in simple sugars, saturated fats, and highly processed foods – is also emphasized, which may increase the pro-inflammatory response and promote exacerbations of the disease (Jabłońska & Mrowiec, 2023).

The importance of diet for nutritional status

Patients with IBD are at high risk of malnutrition, which may result from chronic inflammation, reduced food intake, malabsorption, intestinal loss, and increased metabolic demand. Chronic inflammatory process promotes increased protein catabolism, weight loss, and micronutrient deficiencies (Balestrieri et al., 2020). Accordingly, a well-planned dietary intervention is crucial in the prevention and treatment of malnutrition and in improving the prognosis of patients. In addition to the potential anti-inflammatory effect, enteral nutrition allows a controlled supply of energy and nutrients, which promotes the improvement of anthropometric and biochemical parameters. Unnecessary dietary restrictions exacerbate nutritional deficiencies. This worsens the patient's condition (Jabłońska and Mrowiec, 2023).

Diet and intestinal microbiota

An important mechanism that links diet and IBD activity is changes in the bacterial microflora. Diet affects the balance between pro-inflammatory and anti-inflammatory bacteria. Low dietary fiber and fermentable carbohydrates result in reduced production of short-chain fatty acids (SCFA). Butyrate production is decreasing the most. Its role is to maintain the integrity of the intestinal barrier and regulate the immune response (Balestrieri et al., 2020). A diet rich in plants and prebiotics promotes the growth of protective bacteria and inhibits dysbiosis. This is important because dysbiosis causes IBD (Jabłońska & Mrowiec, 2023).

Nutritional disorders and deficiencies of vitamins and minerals are common and clinically significant complications in patients with inflammatory bowel disease, including Crohn's disease and ulcerative colitis. These deficiencies result from a combination of malabsorption, chronic inflammation, increased intestinal losses, limited food intake, and surgical complications such as small bowel resection (Balestrieri et al., 2020).

The most common deficiencies and their consequences

Micronutrient and vitamin deficiencies occur in a significant proportion of patients with IBD and are more common in Crohn's disease than in CML, especially in the active form of the disease and in the small intestine, where key nutrient absorption takes place (Table 1). Deficiencies of micronutrients most often concern, among others, iron, vitamin B12, vitamin D, folate, zinc, selenium, fat-soluble vitamins (a, E, K), and other factors (Balestrieri et al., 2020).

Lack of iron

It is the most common cause of anemia in patients with IBD and is associated with chronic blood loss, reduced absorption in the damaged intestine, and limited consumption of iron-rich foods. Iron deficiency anemia manifests itself in fatigue, weakness, shortness of breath, and a deteriorated quality of life, and its frequency is high in both diseases (Balestrieri et al., 2020).

Vitamin deficiency B12

In the case of seizure of the final section of the ileum in CD or after resection (e.g. ileo-colony), absorption of B12 is impaired, leading to its deficiency. Deficiency of B12 may result in megaloblastic anemia, neurological symptoms (neuropathy, memory disorders), and metabolic disorders.

Folic acid deficiency (B9)

It is common in IBD and may result from accelerated intestinal passage and active mucositis. Folate deficiency contributes to anemia, abnormalities in cell proliferation, and the potential risk of metabolic disorders (Balestrieri et al., 2020).

Vitamin D and calcium deficiency

These components play a key role in maintaining physiological calcium-phosphate homeostasis and bone health. Vitamin D deficiency is particularly common in patients with CD and UC, which may be due to both malabsorption and limited exposure to sunlight and changes in vitamin D metabolism associated with chronic inflammation. Vitamin D deficiency increases the risk of osteopenia and osteoporosis, which increases the risk of fractures and bone disorders in patients with IBD (Balestrieri et al., 2020).

Table 1. Most common vitamin and mineral deficiencies in Crohn's disease (CD) and ulcerative colitis (UC)

Component	Crohn's disease (CD)	UC	The main mechanisms of deficiency	Clinical consequences
Iron	Very common	Very common	Chronic blood loss, inflammation, impaired absorption	Iron deficiency anemia, fatigue, weakness, reduced quality of life
Vitamin B12	Frequent (especially when busy/resection of the ileum)	Rare	Impaired absorption in the final section of the ileum, surgical resections	Megaloblastic anemia, neuropathy, cognitive impairment
Folic acid (B9)	Common	Moderately frequent	Malabsorption, diarrhea, pharmacotherapy (e.g., sulfasalazine)	Megaloblastic anemia, disorders of cell proliferation
Vitamin D.	Very common	Common	Impaired absorption, chronic inflammation, and limited exposure to the sun	Osteopenia, osteoporosis, and increased risk of fractures
Calcium	Common	Common	Impaired absorption, vitamin D deficiency, and glucocorticoid therapy	Bone loss, osteoporosis
Zinc	Frequent (especially in the case of diarrhea)	Possible	Intestinal loss, reduced consumption	Impaired wound healing, decreased immunity
Selen	Possible	Possible	Insufficient supply, chronic inflammation	Disorders of antioxidant functions
Vitamins A, E, K	More frequent when dealing with the small intestine	Rare	Impaired absorption of fats	Impaired vision, coagulation, and antioxidant function

Deficiency of other micronutrients

In addition to iron, B12, folates, and vitamin D, the analyzed studies indicate that zinc, selenium, and vitamins A and E may also be deficient in patients with IBD. These deficiencies can be associated with wound healing disorders (e.g., zinc or vitamin A deficiency), reduced antioxidant immunity of the body, and potential impact on the intensification of inflammatory processes (Jabłońska & Mrowiec, 2023).

Mechanisms of deficiency

The deficiency of micronutrients and vitamins in IBD has many causes. They result from intestinal damage and malabsorption. In CD, damaged intestines absorb less iron, vitamin B12, folic acid, and fat-soluble vitamins (Jabłońska and Mrowiec, 2023). Chronic diarrhea and accelerated intestinal passage reduce the time of contact of nutrients with the mucous membrane, limiting their absorption. Loss of blood through the digestive tract, especially in the active phase of the disease, contributes to iron loss and to the deepening of anemia. Surgical resections of the small intestine increase the risk of deficiencies, especially B12 and vitamins soluble in fats, when the section responsible for their absorption is resealed. Inflammatory reactions and pharmacotherapy, including the use of sulfasalazine or methotrexate, may affect the metabolism of folic acid or other vitamins (Balestrieri et al., 2020).

Clinical relevance of monitoring and treatment of deficiencies

Micronutrient and vitamin deficiencies in patients with IBD are clinically relevant because they can worsen the symptoms of the disease (e.g., weakness, fatigue, muscle aches), increase the risk of complications (e.g., osteoporosis with D and calcium deficit), affect the quality of life of patients, and increase the risk of relapse and hospitalization (Balestrieri et al., 2020).

Nutrition assessment and monitoring of patients

Nutritional status assessment is a key element of comprehensive care for patients with non-specific inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis. Nutritional disorders and deficiencies are a big problem in inflammatory bowel diseases. This occurs due to chronic inflammation, malabsorption, bowel resection, and eating fewer meals (Hashash et al., 2025; Rimmer et al., 2026). It is important to note that abnormal nutritional status may occur in people with a normal body mass index.

Screening tools and assessment scales

In clinical practice, we use validated scales to identify patients requiring nutritional intervention quickly. The used ones are:

- NRS-2002 (Nutritional Risk Screening 2002): This tool is preferred in gastroenterological units because it takes into account not only the current nutritional status (BMI, weight loss), but also the degree of metabolic severity resulting from the severity of the disease relapse (Hashash et al., 2025; Rimmer et al., 2026).
- MUST (Malnourishment Universal Screening Tool): The scale recommended in outpatient care, based on BMI, the dynamics of unintentional weight loss, and the risk of food discontinuation for more than 5 days (Carvalho, 2021; Rimmer et al., 2026).
- SGA (subjective global assessment): Subjective global assessment combines nutritional history (dietary changes, gastrointestinal symptoms) and physical examination. It enables the detection of fat and muscle loss (Hashash et al., 2025; Carvalho, 2021).

Laboratory diagnosis and deficiency monitoring

Laboratory test results are the basis for assessing the nutrition of patients with IBD. There are a lot of shortcomings in the results. We specifically evaluate peripheral blood count, iron balance parameters, vitamin B12, folic acid, vitamin D, and serum proteins (Hashash et al., 2025; Rimmer et al., 2026).

Anemia, which is one of the most common complications of IBD, is diagnosed at hemoglobin levels <13 g/dl in men and <12 g/dl in women. Iron deficiency is a ferritin concentration <30 μg/l when there is inflammation. When there is no inflammation, it is <100 μg/l. When transferrin saturation is <20%, there is iron deficiency (Hashash et al., 2025; Rimmer et al., 2026). The American Guidelines state to pay attention to test results in patients with Crohn's disease (Carvalho, 2021).

An important non-invasive biomarker is fecal calprotectin. It corresponds to the infiltration of neutrophils in the intestinal mucosa. It can be used to assess disease activity in patients with IBD. Additionally, we use it in differential diagnosis and assessment of the course of the disease and treatment. Calprotectin values <50 μg/g are normal. Concentrations >250 μg/g indicate active inflammation. We refer values between (50–250 μg/g) to the clinical context and further diagnosis (Hashash et al., 2025; Rimmer et al., 2026).

Mineral disturbances are a common problem in patients with IBD and result from both impaired absorption and increased intestinal losses, especially in the course of diarrhea. Calcium plays a key role in maintaining normal bone mineralization, and its deficiency, often coexisting with vitamin D deficiency, increases the risk of osteopenia and osteoporosis. The reference values for total calcium are usually 2.1–2.6 mmol/l, but the interpretation should take into account albumin (Hashash et al., 2025; Carvalho, 2021).

Magnesium concentration <0.7 mmol/l increases gastrointestinal symptoms. Additionally, neuromuscular disorders may occur. Zinc is often lacking in IBD. This element plays an important role in immune function and intestinal mucosal repair. When albumin

<3.5 g/dl, the patient is malnourished. Such results show that the disease is very active and there is high intestinal permeability and protein loss to the gastrointestinal tract. Albumin is always checked against inflammatory markers such as CRP (Hashash et al., 2025; Carvalho, 2021).

Anthropometric and body composition monitoring

Traditional weight assessment is part of disease activity indicators, such as CDAI (Crohn's disease Activity Index), where below-due weight is scored. The guidelines point out, however, that patients with NCHJ may have so-called sarcopenic obesity, where a normal BMI masks a loss of muscle mass. Therefore, it is recommended to supplement the diagnosis with body composition analysis (e.g., BIA method) and functional tests, such as measuring the strength of the handshake (dynamometry) (Hashash et al., 2025; Rimmer et al., 2026).

Frequency of follow-up tests

Ultra-processed foods (UPF) are defined as ready-to-eat products resulting from a combination of food ingredients and food additives that undergo many industrial processes. These products are usually characterized by high content of sugar, saturated fat, and salt, while at the same time being low in protein, fiber, vitamins, and minerals. The UPF includes, among others, sweet and salty snacks, soft drinks, processed meat products, and packaged breads and confectionery. In recent decades, there has been a systematic increase in the share of these products in the daily diet.

Consumption of ultra-processed foods can influence the development of IBD through a variety of mechanisms. One concept assumes that high UPF consumption is associated with the displacement of unprocessed or minimally processed products (UMPs), which are usually an important source of dietary fiber. Reducing the supply of fiber may lead to a decrease in the production of short-chain fatty acids and adverse changes in the composition of the intestinal microbiota.

Another hypothesis is that ultra-processed foods, containing additives such as excess salt or artificial sweeteners, may increase the inflammatory process in the gut. It has been shown that high salt intake increases intestinal barrier permeability, increases the production of pro-inflammatory cytokines, among others, by lowering the level of short-chain fatty acids in the feces and reducing the number of Lactobacillus bacteria, and also intensifies chemically induced colitis in experimental models.

Artificial sweeteners present in ultra-processed foods can also promote the development of enteritis. In mouse models of spontaneous ileitis, sucralose and maltodextrin supplementation led to increased inflammatory changes. At that time, an increase in the number of pro-inflammatory bacteria, such as Salmonella, was observed, which, when developed under the influence of these additives, affects intestinal epithelial cells by reducing mucus production and increasing susceptibility to colitis (Gubatan et al., 2023).

Epidemiological studies show that high consumption of UPF increases the risk of developing Crohn's disease. High intake of UPF may increase the risk of developing ulcerative colitis, but it affects Crohn's more. A study was also conducted in which 245,112 people participated, and the total follow-up time was 5,468,444 person-years. During this period, 369 cases of Crohn’s disease (CD) and 488 cases of ulcerative colitis (UC) were reported. If there is a lot of amphiregulin in the dendritic cells after the procedure, the epithelium is repaired more quickly. The median age at diagnosis was 56 years (range 29–85 years). However, there was no consistent association between UPF consumption and UC risk (Gubatan et al., 2023).

Diet and intestinal microbiota in non-specific inflammatory bowel diseases

The importance of diet in the modulation of the intestinal microbiota

Diet is one of the most important environmental factors affecting the composition, diversity, and metabolic activity of the intestinal microbiota. In conditions of eubiosis, intestinal barrier integrity and host immune homeostasis are maintained. A well-balanced physiological diet supports maintenance in patients with non-specific inflammatory bowel diseases (IBD), including Crohn’s disease (CD) and ulcerative colitis (UC); however, numerous microbiological disorders, referred to as intestinal dysbiosis, are observed. The severity of which can be further modulated by the diet (Lavelle & Sokol, 2020).

A growing body of research indicates that nutritional interventions may affect not only the clinical symptoms of IBD, but also the composition and function of the gut microbiota. The diet affects the intestinal environment by changing the availability of substrates for commensal bacteria, modulating the production of bacterial metabolites, and influencing the activation of local immune mechanisms (Lavelle & Sokol, 2020). Therefore, nutrition is now considered to be one of the potential elements of supportive therapy aimed at restoring microbial homeostasis of the gastrointestinal tract.

Intestinal microbiota transplantation (FMT) as a specific intervention in the microbiota-host axis

Transplantation of intestinal microbiota (ang. Fecal Microbiota Transplantation (FMT) is currently a paradigm of modern compensatory therapy for inflammatory bowel disease, aimed at direct restoration of intestinal ecosystem homeostasis (Haifer et al., 2022; Le Berre et al., 2023). In contrast to conventional probiotic supplementation, based on the administration of isolated strains, FMT enables the horizontal transfer of complete and stable ecological niches, which induces a multidirectional recalibration of the recipient's immune response by restoring lost species biodiversity (Le Berre et al., 2023; Costello et al., 2019).

Molecular mechanisms and receptor signaling

Anti-inflammatory taxa were recreated in the intervention. Representatives of the families Lachnospiraceae, Ruminococcaceae, and Faecalibacterium prausnitzii are important. Bacterial population recovery enhances the synthesis of short-chain fatty acids (SCFA), especially butyrate (C4). Butyrate is an energy source for colonocytes. In addition, it is an important ligand for G protein-coupled receptors. These include receptors such as GPR109a and GPR43 (Le Berre et al., 2023; Costello et al., 2019).

Activation of these signaling pathways induces suppression of THE AKT kinase cascade and NF-κB complex, resulting in a drastic reduction in overexpression of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α), interleukin 1β (IL-1β), and interleukin 6 (IL-6) (Bischoff et al., 2022). In addition, the effect on the GPR109a receptor promotes the reduction of secretion of interleukin 23 (IL-23), which inhibits pathological activation of cells ILC3 and reduces transmural inflammation (Costello et al., 2019).

Physical and immune barrier restoration

FMT has a strong protective effect against the mechanical barrier of the intestine, which in the course of IBD is destroyed (the so-called leaky gut) (Costello et al., 2019). Under the influence of allogeneic microbiota from healthy donors, tight junctions such as occludens and zonula occludens (ZO-1, ZO-2) are stabilized (Haifer et al., 2022). This process is supported by stimulation of cup cells to regenerate glycokalliki and seal the mucous layer by secretion of mucin MUC2 (Hashash et al., 2025; Tian et al., 2025).

The epithelial-mesenchymal transition differentiates T cells towards a regulatory phenotype (Treg). Additionally, it stimulates the production of immunoglobulin A (IgA). Immunoglobulin restores immune tolerance to commensal antigens (Haifer et al., 2022). The large amount of amphiregulin in dendritic cells present after surgery accelerates the repair of damaged epithelium (Le Berre et al., 2023).

Clinical efficacy and the concept of super-donors

Clinical analyses confirm the high efficacy of FMT in inducing endoscopic and clinical remission, with the most spectacular results being reported in patients with ulcerative colitis (UC) [19, 20]. Therapeutic success is closely linked to the presence of specific species such as Akkermansia muciniphila and Roseburia spp., whose increase in numbers after surgery directly correlates with the extinction of disease activity (Le Berre et al., 2023; Van Der Vlist et al., 2022).

The variability of response to treatment is a major overestimation. As a result, "superdonors" were created. These are individuals characterized by a very high alpha diversity index and a stable metabolic profile rich in butyrate precursors (Hashash et al., 2025; Le Berre et al., 2023). Currently, various therapeutic protocols are emerging that replace invasive methods. These protocols utilize oral systems based on freeze-dried microbial capsules. These protocols therefore have an optimized safety profile and reduce the risk of periprocedural complications (Van Der Vlist et al., 2022).

Mediterranean diet and intestinal microbiota in Crohn's disease and ulcerative colitis

In recent years, the Mediterranean diet has been recognized as one of the most promising nutritional models supporting the treatment of inflammatory bowel diseases. Its ability to modulate intestinal microflora and influence the intestinal barrier is interesting. It leads to the regulation of the immune response of the gastrointestinal mucosa (Deleu et al., 2024; Lavelle & Sokol, 2020). Unlike the Western diet, which is characterized by high content of saturated fats, simple sugars, and ultraprocessed foods, the Mediterranean diet is based on a high intake of vegetables, fruits, whole grains, legumes, olive oil, and more. fish, nuts, and fermented dairy products (Lavelle & Sokol, 2020; Skoracka et al., 2025).

More and more studies indicate that the Mediterranean diet may counteract the phenomenon of intestinal dysbiosis observed in both Crohn's disease (CD) and ulcerative colitis (UC) (Deleu et al., 2024; Nishida et al., 2018). Patients with IBD have a reduction in microbial diversity and in the number of bacteria producing short-chain fatty acids (SCFA), especially butyrate. This applies to bacteria

such as *Faecalibacterium prausnitzii*, *Roseburia* spp., *Eubacterium rectale*, and the *Lachnospiraceae* family (Nishida et al., 2018). At the same time, there is an increase in the number of bacteria with pro-inflammatory potential, including *Escherichia coli* type AIEC (adherent-invasive *E. coli*), *Enterococcus fecalisczi* bacteria belonging to the *Enterobacteriaceae* family (Deleu et al., 2024).

The components of the Mediterranean diet affect the microbiota (Table 2) primarily by increasing the availability of fermentation substrates for commensal bacteria. Dietary fiber present in vegetables, fruits, and whole grains undergoes bacterial fermentation in the large intestine, leading to the production of SCFA – mainly butyrate, propionate, and acetate (Lavelle & Sokol, 2020; Nishida et al., 2018). Particular importance is attributed to butyrate, which is the primary source of energy for colonocytes and has a strong anti-inflammatory effect (Nishida et al., 2018; Bischoff et al., 2023).

The mechanism of action of butyrate includes, among other things, inhibition of the activation of NF-κB transcription factor responsible for the expression of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6. In addition, butyrate activates G-protein-coupled receptors (GPR41, GPR43, and GPR109a), leading to an increase in the number of Tregs, regulatory lymphocytes, and a reduction in the inflammatory response dependent on Th17 lymphocytes. Short-chain fatty acids also increase the expression of close-link proteins such as claudin and occludin, improving the integrity of the intestinal barrier and reducing the permeability of the epithelium (Nishida et al., 2018; Bischoff et al., 2023).

Table 2. Effects of Mediterranean diet elements on specific bacterial strains.

Part of the Mediterranean diet	**Related strains/groups of gut bacteria (most commonly described)**	**Potential significance in Crohn's disease**
Extra virgin olive oil (polyphenols + MUFA)	*Faecalibacterium prausnitzii*, *Roseburia spp.*, *Bifidobacterium spp.*	Growth of butyrate-producing bacteria (SCFA), reduction of inflammation of the mucous membrane; Low levels *of F. prausnitzii are often* observed in CD
Vegetables and fruits (soluble fiber, polyphenols)	*Bifidobacterium spp.*, *Lactobacillus spp.*, *Akkermansia muciniphila*	Better production of short-chain fatty acids, improvement of the intestinal barrier; caution with active tightening/narrowing of the intestine
Legume plants	*Bifidobacterium adulescentis*, *Prevotella spp.*, butyric bacteria	Prebiotic effect of fiber and resistant starch; beneficial mainly in remission, not always well tolerated in active disease
Whole grains (oats, barley, whole grain bread)	*Faecalibacterium*, *Ruminococcus*, *Bifidobacterium*	Increasing SCFA and microbiota diversity; in some patients in the active phase, a temporary reduction of fiber is necessary.
Nuts and seeds	*Lactobacillus spp.*, *Bifidobacterium spp.*, *Roseburia spp.*	Beneficial effect due to polyphenols and unsaturated fats; they can be problematic with bowel constrictions
Fatty marine fish (omega-3)	indirectly support anti-inflammatory bacteria; pro-inflammatory pathogens	Reduction of inflammatory mediators, support of remission; this is not a "probiotic" effect, more immunomodulatory
Fermented dairy products (natural yogurt, kefir – if tolerated)	*Lactobacillus spp.*, *Bifidobacterium spp.*	Support of microbiota and intestinal tolerance; it depends on individual lactose tolerance and disease activity
Reduce red meat and ultra-processed foods.	*Escherichia coli* (adherent-invasive strains), Proteobacteria	Reduced the severity of dysbiosis and expansion of pro-inflammatory bacteria associated with CD

Polyphenols are found in olive oil, vegetables, berries, and red grapes. They are important for modulating the microflora. These compounds have antioxidant properties and can stimulate the growth of protective bacteria. These are *Akkermansia muciniphila* and

Bifidobacterium spp. (Lavelle and Sokol, 2020; Skoracka et al., 2025). *Akkermansia muciniphila* participates in mucin metabolism and supports the reconstruction of the intestinal mucosa. This is especially important in people with UC who have a damaged mucosal barrier (Skoracka et al., 2025).

The Mediterranean diet also affects the metabolism of bile acids. Excessive consumption of saturated fats characteristic of the western diet leads to an increase in the concentration of secondary bile acids with pro-inflammatory effect and promotes the multiplication of bacteria tolerating an oxygen-rich environment (Deleu et al., 2024; Nishida et al., 2018). In turn, a diet rich in unsaturated fats and fiber supports the development of bacteria involved in the proper metabolism of bile acids, which may limit the activation of the inflammatory response within the intestinal mucosa (Lavelle & Sokol, 2020; Nishida et al., 2018).

The importance of the Mediterranean diet may vary depending on the activity of the disease. In the period of remission in both patients with CD and UC, it is recommended to fully implement the classic model of the Mediterranean diet with a high supply of soluble fiber, fermented products, and various sources of polyphenols (Lavelle & Sokol, 2020; Skoracka et al., 2025). During this period, the diet may promote the restoration of microbial diversity and an increase in the number of bacteria producing SCFA (Skoracka et al., 2025; Nishida et al., 2018). An increase in Faecalibacterium prausnitzii, whose low concentration is associated with an increased risk of recurrence of Crohn's disease, seems particularly beneficial.

Nutritional status is better in patients in remission. The Mediterranean diet provides plenty of folate, zinc, selenium, calcium, and antioxidants. Thanks to it, there are few nutritional deficiencies. (Lavelle and Sokol, 2020; Skoracka et al., 2025) Marine fish are rich in omega-3 fatty acids. A large number of fish in the diet reduces inflammation. This is due to the inhibition of the synthesis of pro-inflammatory mediators.

During the period of exacerbation of the disease, the use of the classic Mediterranean diet may be difficult due to the deterioration of the tolerance of insoluble fiber and the increased symptoms of the gastrointestinal tract. In patients with active CD, especially in the case of small intestine involvement or the presence of stenosis, an excessive supply of raw vegetables and whole grains may exacerbate abdominal pain, bloating, and diarrhea. In such cases, it is recommended to modify the Mediterranean diet by reducing heavy-digestible products and increasing the proportion of cooked products and soluble fiber (Lavelle & Sokol, 2020).

In the exacerbation of ulcerative colitis, the aim of the dietary procedure is primarily to reduce irritation of the intestinal mucosa and to maintain an adequate nutritional status. Preference is given to easily digestible products, cooked vegetables, rice, fermented dairy products, and sources of omega-3 acids. At the same time, maintaining the supply of anti-inflammatory ingredients may support the reconstruction of the microbiota and limit further damage to the intestinal barrier (Lavelle & Sokol, 2020; Skoracka et al., 2025).

It is increasingly emphasized that the Mediterranean diet should not be treated only as a dietary regimen, but as an element of therapy affecting the diet-microbiota-immune system axis (Deleu et al., 2024; Nishida et al., 2018). Thanks to the ability to increase the production of bacterial metabolites with anti-inflammatory effects and support the integrity of the intestinal barrier, this diet can be an important element supporting the treatment of both Crohn's disease and ulcerative colitis (Lavelle & Sokol, 2020; Nishida et al., 2018).

4. CONCLUSION

Analysis of the available literature indicates that intestinal dysbiosis is one of the most important factors in IBD pathogenesis and is associated with reduced microbial diversity, reduced abundance of bacteria that produce short-chain fatty acids, and the growth of microorganisms with pro-inflammatory potential. The study showed that diet can significantly affect both the composition of the microbiota and the inflammatory activity of the disease. Particular importance is attributed to a diet rich in fiber, prebiotic components, and the Mediterranean diet pattern, which promote the growth of butyrate-producing bacteria and support the maintenance of the integrity of the intestinal barrier. On the other hand, the Western diet, rich in highly processed foods, saturated fats, and simple sugars, may increase inflammation and deepen dysbiosis. The impact of pharmacotherapy on the intestinal microbiota also remains an important issue, as biological drugs and aminosalicylates may partially restore eubiosis and improve the function of the intestinal barrier. At the same time, it should be emphasized that the relationship between diet, intestinal microbiota, and the course of IBD is still a very wide and dynamically developing field of research. Despite the increasing number of publications, many biological mechanisms have not yet been fully elucidated, and the results of research often remain ambiguous. Microbiota, nutrition, and the immune system are needed, and more effective, personalized therapeutic strategies are needed. Further clinical and experimental studies will allow for a better understanding of the interactions between them.

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Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

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