QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

GRABOWSKA, Natalia, GRACZYK, Alicja, HAJT, Michał, MISIASZEK, Dastin, WOLSZCZAK, Anna, MOTOR, Jakub, MARTYNOWSKA, Weronika, CIECIERSKI, Piotr and TURZYŃSKA, Paulina. Nutritional Deficiencies in Crohn's Disease: Pathogenesis, Clinical Significance, and Therapeutic Management. Quality in Sport. 2026.65.73070. https://doi.org/10.12775/QS.2026.65.73070

ARTICLE TIMELINE
Received: 04.06.2026 Revised: 20.07.2026
Accepted: 20.07.2026 Published: 27.07.2026

INDEXING & EVALUATION
MEiN points: 20 Unique ID: 201398
Disciplines: Economics & Finance; Management & Quality Sciences

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024 No. 32553. Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).

Punkty Ministerialne z 2019 — aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Nutritional Deficiencies in Crohn's Disease: Pathogenesis, Clinical Significance, and Therapeutic Management

Natalia Grabowska[1,*], Alicja Graczyk[2], Michał Hajt[3], Dastin Misiaszek[3], Anna Wolszczak[4], Jakub Motor[5], Weronika Martynowska[4], Piotr Ciecierski[6], Paulina Turzyńska[7]

[1] Specialized Provincial Hospital in Ciechanów, Poland, e-mail: natalka.grabowska007@gmail.com (corresponding author), ORCID: https://orcid.org/0009-0007-9797-4416;
[2] Ks. J. Popieluszko Bielanski Hospital in Warsaw, Poland, e-mail: graczyk.ala@wp.pl, ORCID: https://orcid.org/0009-0002-8684-0846;
[3] University Clinical Center of the Medical University of Warsaw, Poland, e-mail: michalhajt@icloud.com, ORCID: https://orcid.org/0009-0001-6558-0902; e-mail: dastinmisiaszek@outlook.com, ORCID: https://orcid.org/0009-0000-6932-9515;
[4] Prof. W. Orłowski Independent Public Clinical Hospital of the Medical Centre of Postgraduate Education, Poland, e-mail: aa.wolszczak@gmail.com, ORCID: https://orcid.org/0009-0006-7348-1185; e-mail: weronikamartynowska@gmail.com, ORCID: https://orcid.org/0009-0003-6403-6926;
[5] Medical University of Lodz, Poland, e-mail: oliwiermotor@gmail.com, ORCID: https://orcid.org/0009-0008-3769-0101;
[6] District Medical Center in Grójec, Poland, e-mail: piotr.ciecierski06@gmail.com, ORCID: https://orcid.org/0009-0005-7225-7918;
[7] University Clinical Centre in Gdansk, Poland, e-mail: turzynska10@gmail.com, ORCID: https://orcid.org/0009-0000-4072-8213.

Abstract

Background. Crohn's disease (CD) is a chronic, relapsing-remitting inflammatory disorder of the gastrointestinal tract in which impaired nutritional status and nutritional deficiencies are common. This issue is of major clinical relevance, as deficiencies in both macro- and micronutrients not only reflect disease activity but may also influence disease course, treatment efficacy, and patients' quality of life.

Aim. The aim of this review was to present the mechanisms leading to nutritional deficiencies in Crohn's disease, discuss their clinical significance, and summarize current diagnostic and therapeutic strategies.

Material and methods. This study is a narrative review. A literature review was conducted using the PubMed database, focusing on publications addressing nutritional deficiencies in Crohn's disease, with particular emphasis on their pathogenesis, clinical significance, and contemporary diagnostic and therapeutic approaches. Articles published between 2013 and 2025 were included.

Results. The pathogenesis of nutritional deficiencies in Crohn's disease is multifactorial and includes malabsorption, reduced dietary intake, increased metabolic demand, nutrient loss, and the effects of pharmacotherapy. The most frequently observed deficiencies involve protein and energy, iron, vitamin B12, vitamin D, folate, and selected minerals such as zinc and magnesium. Their clinical consequences include anemia, impaired bone mineralization, immune dysfunction, delayed tissue repair, and overall deterioration in health status. The problem is particularly important in the pediatric population, in whom deficiencies may result in impaired growth and development.

Conclusions. The available evidence indicates that systematic nutritional assessment and appropriately selected nutritional interventions should constitute an integral part of comprehensive patient care. Early identification of deficiencies and implementation of individualized therapeutic management are of key importance.

Key words: Crohn's disease, nutritional deficiencies, malnutrition, micronutrients, clinical nutrition, diet, inflammatory bowel disease

HIGHLIGHTS

1. Malnutrition affects 38.9% of CD patients in remission and 82.8% with active disease.
2. Multifactorial pathogenesis encompasses mucosal malabsorption, anorexia, and drug-induced losses.
3. Iron deficiency anemia and mixed anemia of chronic disease are the most prevalent hematologic complications.
4. Vitamin D and B12 deficiencies drive bone demineralization and neurological risk, respectively.
5. Systematic screening with MUST/NRS-2002 and targeted supplementation improve disease outcomes.

1. Introduction

Crohn's disease (CD) is a form of inflammatory bowel disease of unknown etiology characterized by a relapsing-remitting course [1,2]. Inflammatory lesions may involve any segment of the gastrointestinal tract, from the oral cavity to the anus; however, they are most commonly located in the terminal ileum and colon [2,4]. The transmural nature of inflammation predisposes patients to complications such as strictures, fistulas, and abscesses [1,4]. Its heterogeneous clinical presentation and variable course make CD a substantial diagnostic and therapeutic challenge [5].

The etiopathogenesis of CD is multifactorial. Genetic, environmental, and immunological factors all contribute to disease development [4,6]. Among the best-established modifiable risk factors is cigarette smoking, which is associated with a more severe disease course and poorer response to treatment [6,7]. Other environmental factors, including diet, antibiotic exposure, nonsteroidal anti-inflammatory drug use, degree of urbanization, and lifestyle patterns typical of industrialized countries, are also considered relevant [6–9]. These factors may alter gut microbiota composition and intestinal barrier function, thereby promoting both the development and persistence of intestinal inflammation. CD is most commonly diagnosed in young adults, typically during the second and third decades of life, although it can occur at any age. Despite extensive research, the precise mechanisms initiating and sustaining inflammation remain incompletely understood [4,10].

In recent years, growing attention has been paid to the role of nutritional status in patients with Crohn's disease. Nutritional deficiencies and malnutrition are common clinical problems and may affect both macro- and micronutrients. Malnutrition has been reported in 38.9% of patients with CD in remission and in 82.8% of those with active disease, adversely affecting disease course, quality of life, and treatment outcomes [12].

The mechanisms underlying nutritional deficiencies are complex and result from the interaction of multiple processes. The most important include malabsorption due to mucosal injury, reduced food intake, increased metabolic demand associated with chronic inflammation, nutrient loss resulting from diarrhea and the effects of treatment on nutrient metabolism and absorption [11,14,15].

The clinical consequences of nutritional deficiencies in CD are broad and may contribute to anemia, impaired bone mineralization, impaired immune responses and deterioration in overall health status. In addition, poor nutritional status is associated with a higher risk of complications and poorer response to treatment [11,13,16,18].

Despite increasing awareness of the importance of nutrition in the course of the disease, the diagnosis and management of nutritional deficiencies remain challenging. In many cases, nutritional disorders remain unrecognized or undertreated, highlighting the need for systematic nutritional assessment and timely therapeutic interventions. Increasing importance is also being placed on individualized management and comprehensive care that incorporates nutritional aspects [11,12,17].

The aim of this paper was to present the pathogenesis of nutritional deficiencies in Crohn's disease, discuss their consequences and review current therapeutic strategies.

2. Pathogenesis of Nutritional Deficiencies

2.1. Malabsorption

One of the principal mechanisms leading to nutritional deficiencies in Crohn's disease is malabsorption resulting from damage to the intestinal mucosa. Chronic inflammation leads to structural alterations, such as villous blunting and disruption of intestinal barrier integrity, which

directly impairs the absorptive capacity of the small intestine. Particular importance is attributed to involvement of the terminal ileum, which is responsible for the absorption of vitamin B12 and bile acids [11,12,15].

2.2. Reduced Dietary Intake

Reduced nutrient intake represents another important component in the pathogenesis of deficiencies. Symptoms such as abdominal pain, diarrhea and postprandial fullness often result in reduced food consumption. During periods of active disease, loss of appetite and chronic fatigue are also common, further decreasing caloric intake.

In clinical practice, self-imposed dietary restrictions are also relevant. Elimination of specific foods, often undertaken without consultation with a physician or dietitian, may lead to inadequate intake of essential nutrients, particularly vitamins and micronutrients. Persistent deficiencies of this kind contribute to worsening nutritional status [11–13].

2.3. Nutrient Loss

Increased nutrient loss is another major mechanism contributing to deficiency states. Chronic diarrhea, a hallmark of Crohn's disease, leads to losses of water, electrolytes and minerals, potentially resulting in disturbances of fluid and electrolyte balance. In addition, chronic gastrointestinal bleeding is a significant cause of iron deficiency and anemia. In patients undergoing surgical treatment, including intestinal resection, the absorptive surface area is reduced, which further increases the risk of nutritional deficiencies [13,16].

2.4. Impact of Treatment

Pharmacotherapy used in the treatment of Crohn's disease, although essential for controlling the inflammatory process, may also adversely affect nutritional status. Glucocorticoids disrupt calcium-phosphate homeostasis and vitamin D metabolism, thereby increasing the risk of bone demineralization. They also promote catabolic processes and disturbances in protein metabolism [11,17,18].

Methotrexate affects folate metabolism through antagonism, thereby contributing to folate deficiency and anemia. Sulfasalazine may impair intestinal folate absorption. In the case of biologic therapies, their effects on nutritional status are mainly indirect and result from reduction of inflammatory activity [11,17,19].

In summary, the pathogenesis of nutritional deficiencies in CD involves several interrelated mechanisms, including malabsorption, reduced dietary intake, increased nutrient loss, the effects of chronic inflammation and the impact of treatment. Their combined action leads to worsening malnutrition, which in turn affects disease course by increasing the risk of complications and worsening prognosis [11–13].

Table 1. Effects of selected drugs used in Crohn's disease on patients' nutritional status

DRUG	POTENTIAL DEFICIENCY	MECHANISM
Glucocorticoids	Calcium, vitamin D	Disturbances in bone metabolism, increased bone resorption
Methotrexate	Folate	Inhibition of folate metabolism
Sulfasalazine	Folate	Reduced intestinal absorption
Biologic agents	Indirect effect	Modulation of inflammation and microbiota

Source: Authors' own elaboration based on [11,17–19].

3. Nutritional Deficiencies and Their Clinical Significance

3.1. Macronutrient Deficiencies

Protein-energy malnutrition is one of the most common metabolic disturbances in patients with CD, particularly during periods of active disease [11,13]. It develops as a result of an imbalance between energy intake and increased metabolic demand associated with chronic inflammation [11,14].

Proinflammatory cytokines such as TNF-α and IL-6 intensify catabolic processes, leading to proteolysis and reduced protein synthesis. As a consequence, patients may develop loss of muscle mass, sarcopenia and negative nitrogen balance [11,15]. At the same time, increased resting energy expenditure contributes to progression of the energy deficit.

Ileal involvement may impair fat absorption due to bile acid loss, resulting in diarrhea and secondary deficiencies of fat-soluble vitamins (A, D, E, and K) [12,15].

The clinical consequences of protein-energy malnutrition include worsening overall condition, reduced physical capacity and increased risk of complications, including infections and postoperative complications [11–13,19].

3.2. Micronutrient Deficiencies

Iron Deficiency and Anemia

Iron deficiency is the best-documented deficiency and the leading cause of anemia in patients with inflammatory bowel disease [13,16]. Its pathogenesis includes both chronic gastrointestinal blood loss and disturbances in iron metabolism associated with inflammation.

A central component of this process is hepcidin, a peptide hormone produced in the liver that serves as the principal regulator of systemic iron homeostasis. Under conditions of chronic inflammation, especially in response to proinflammatory cytokines such as IL-6, its synthesis increases. Hepcidin inhibits the activity of ferroportin, the protein responsible for iron export

from enterocytes and macrophages into the bloodstream, leading to intracellular iron sequestration and reduced availability of iron for erythropoiesis [20].

This mechanism contributes to the development of anemia of chronic disease (ACD), which frequently coexists with classic iron deficiency anemia. In clinical practice, a mixed form of anemia is most commonly observed, which significantly complicates diagnosis and treatment [16,20].

Anemia in CD may affect as many as 20–70% of patients, particularly during active disease [16,19]. Its presence is associated with marked impairment in quality of life, manifested by chronic fatigue, weakness, decreased physical performance, and impaired concentration. Anemia has also been shown to correlate with higher hospitalization rates and poorer treatment outcomes [11,16,19].

Vitamin B12 Deficiency

Vitamin B12 deficiency occurs primarily in patients with terminal ileal involvement or after ileal resection [11,14,15]. This vitamin plays a key role in hematopoiesis and nervous system function. Its deficiency leads to megaloblastic anemia and neurological symptoms such as paresthesias, sensory disturbances, and impaired balance. When diagnosis is delayed, neurological damage may be only partially reversible [14,15].

Vitamin D Deficiency and Bone Disorders

Vitamin D deficiency is common among patients with CD and results from both impaired absorption and the effects of chronic inflammation and pharmacotherapy [11,18,19]. Vitamin D plays a crucial role in bone metabolism and modulation of the immune response. Its deficiency leads to reduced bone mineral density and an increased risk of osteopenia and osteoporosis. In addition, proinflammatory cytokines enhance bone resorption and suppress osteoblast activity, thereby exacerbating skeletal complications [18].

Folate, Zinc, and Magnesium Deficiencies

Folate deficiency may result from both malabsorption and the effects of medications such as methotrexate and sulfasalazine [11,13,19]. Its consequences include megaloblastic anemia and impaired hematopoietic function.

Zinc plays an important role in tissue repair and immune function, whereas magnesium is involved in numerous enzymatic processes. Deficiencies of these elements, often associated with chronic diarrhea, may lead to immune dysfunction, impaired wound healing, and neuromuscular disturbances [11,12,15].

3.3. Special Importance of Nutritional Deficiencies in the Pediatric Population

Nutritional deficiencies are of particular importance in pediatric patients, in whom they may result in growth failure, delayed puberty, and abnormal somatic development [21]. These mechanisms involve both insufficient nutrient intake and the effect of chronic inflammation on the endocrine system. A fundamental role is played by disturbances in the growth hormone (GH)–insulin-like growth factor 1 (IGF-1) axis. Proinflammatory cytokines such as TNF-α and IL-6 inhibit IGF-1 synthesis and promote tissue resistance to GH, ultimately leading to impaired linear growth [21].

Growth impairment may be one of the first manifestations of the disease and often precedes the onset of gastrointestinal symptoms. It is estimated to affect 15–40% of pediatric patients with inflammatory bowel disease [11,12,21].

If left untreated, nutritional deficiencies may lead to permanent consequences, including reduced final adult height, developmental disturbances, and diminished quality of life. Therefore, early diagnosis and intensive nutritional therapy, including the use of exclusive enteral nutrition, are of particular importance in this group [19,25].

Table 2. The most common nutritional deficiencies in Crohn's disease: mechanisms, clinical manifestations, and clinical significance

NUTRIENT	MECHANISM OF DEFICIENCY	CLINICAL MANIFESTATIONS AND CONSEQUENCES	CLINICAL SIGNIFICANCE
Protein/energy	Inflammation, increased catabolism, reduced intake	Loss of muscle mass, sarcopenia, weakness	Worse prognosis, increased risk of complications and hospitalization
Iron	Bleeding, increased hepcidin, reduced ferroportin activity	Anemia, fatigue, reduced exercise tolerance	Impaired quality of life, more frequent hospitalizations
Vitamin B12	Ileal involvement/resection, impaired absorption	Megaloblastic anemia, neurological symptoms	Risk of permanent neurological damage
Vitamin D	Malabsorption, glucocorticoids, inflammation	Osteopenia, osteoporosis, bone pain	Increased fracture risk, impaired quality of life
Folate	Medications (methotrexate, sulfasalazine), decreased absorption	Macrocytic anemia	Hematologic disturbances, deterioration of general condition
Zinc	Diarrhea, intestinal losses	Immune dysfunction, impaired wound healing	Higher risk of infection
Magnesium	Diarrhea, malabsorption	Weakness, neuromuscular disturbances	Deterioration of overall functioning

Source: Authors' own elaboration based on [11–19].

4. Therapeutic Management

Therapeutic management in patients with CD should involve not only control of inflammatory activity but also systematic evaluation and treatment of nutritional deficiencies. Contemporary approaches emphasize the need for comprehensive and individualized management that takes into account both the patient's clinical status and nutritional needs. Early identification and appropriate correction of deficiencies may substantially influence disease course and improve prognosis [11,17,19,23].

4.1. Diagnosis of Nutritional Deficiencies

Assessment of nutritional deficiencies should constitute an integral component of care in patients with CD. Regular evaluation of nutritional status is recommended and should include laboratory testing, clinical assessment, and body composition analysis [11,12,17,19]. Importantly, such assessment should be performed both during active disease and in remission. Nutritional deficiencies may persist despite clinical improvement and do not always correlate directly with inflammatory activity [11,12].

Basic laboratory tests include measurements of hemoglobin, ferritin, transferrin, vitamin B12, folate, and vitamin D. In clinical practice, correct interpretation of iron metabolism parameters is particularly important. Ferritin is an acute-phase protein, and its concentration may be elevated in the presence of inflammation, potentially masking coexisting iron deficiency [16,19]. Therefore, interpretation of results should include simultaneous assessment of inflammatory markers.

Another important aspect of diagnosis is nutritional assessment based on anthropometric parameters such as body weight and body mass index (BMI). However, normal BMI values do not exclude abnormalities in body composition, particularly sarcopenia, which may develop in patients with chronic inflammation [12,19].

Standardized screening and diagnostic tools are used in clinical practice to enable early detection of malnutrition risk and its severity. One of the most commonly used tools is the Malnutrition Universal Screening Tool (MUST), which is based on BMI, weight loss, and the impact of acute disease on oral intake. Although widely used in the general population, its sensitivity in inflammatory bowel disease may be limited because body composition abnormalities often occur despite normal BMI [12,19,23].

Among hospitalized patients, the Nutritional Risk Screening 2002 (NRS-2002), recommended by the European Society for Clinical Nutrition and Metabolism, is also frequently used. This tool takes into account both the degree of malnutrition and the severity of the underlying disease, and has been shown to correlate well with complication risk and length of hospital stay [19,23].

4.2. Supplementation

Nutrient supplementation is a fundamental component of the treatment of nutritional deficiencies in Crohn's disease. The route of administration depends on the severity of deficiency, disease activity, and the capacity of the gastrointestinal tract to absorb nutrients [13,17,19,23].

In cases of iron deficiency, oral treatment may be effective in patients with mild disease and preserved intestinal function. However, in patients with active inflammation or intolerance to oral iron preparations, intravenous administration is preferred because it allows faster replenishment of iron stores and better tolerability [16,17,19].

Vitamin B12 supplementation is particularly important in patients after ileal resection, in whom parenteral administration is often required. For vitamin D, regular monitoring of serum levels and appropriate supplementation are recommended, especially in patients with reduced bone mineral density [14,15,18,19].

The choice of supplementation should be individualized, and its effectiveness should be monitored through follow-up laboratory testing. Potential interactions between medications and nutrients should also be taken into account [17,19,23].

4.3. Clinical Nutrition

Enteral nutrition is the preferred form of nutritional support because it helps preserve gastrointestinal function and has beneficial effects on the intestinal microbiota. In pediatric patients, exclusive enteral nutrition (EEN) is an established method for inducing remission. In adults, its role is more limited, but it may still serve as an effective adjunct to treatment [17,19,25].

Parenteral nutrition is used when enteral feeding is insufficient or impossible, for example in severe complications such as bowel obstruction or extensive fistulizing disease. Although effective in correcting nutritional deficits, it is associated with a higher risk of complications and therefore should be used only for clearly defined indications [19,23].

4.4. Diet in Crohn's Disease

The role of diet in CD treatment is the subject of intensive investigation, and its importance in clinical practice continues to grow. Although no single universal diet exists for all patients, appropriately selected nutritional strategies may support treatment and improve quality of life [17,19,23].

One dietary approach used in patients with Crohn's disease is the low-FODMAP diet, which restricts fermentable oligosaccharides, disaccharides, monosaccharides, and polyols. This diet was originally developed for irritable bowel syndrome but is also used in patients with inflammatory bowel disease, particularly to reduce functional symptoms such as bloating, abdominal pain, and diarrhea. Its mechanism of action is based on reducing fermentable carbohydrates, which decreases intestinal gas production and osmotic load within the bowel lumen [26].

It should be emphasized, however, that the low-FODMAP diet does not directly control intestinal inflammation, and its prolonged use may reduce microbial diversity. For this reason, it should be used under specialist supervision and only for a limited period, followed by gradual reintroduction of foods [26].

In recent years, increasing attention has also been paid to exclusion diets such as the Crohn's Disease Exclusion Diet (CDED), which aims to reduce exposure to dietary components that may exacerbate inflammation and to modulate the intestinal microbiota. Such diets have been shown to contribute to symptom improvement and better inflammatory parameters [17,22,23].

5. Discussion

Nutritional deficiencies in Crohn's disease represent an important yet often underestimated clinical problem. Their pathogenesis is multifactorial and includes malabsorption, reduced dietary intake, increased metabolic requirements, and excessive nutrient loss [11–15]. As a consequence, disturbances in nutritional status are not merely secondary to active disease but may also influence disease course, treatment response, and prognosis.

Iron, vitamin B12, and vitamin D deficiencies are of particular clinical relevance. Anemia, most commonly of mixed etiology, is among the most frequent metabolic complications in patients with CD and is associated with marked impairment in quality of life, increased hospitalization rates, and worse overall functioning [16,19]. Vitamin B12 deficiency is especially important in patients with terminal ileal involvement or prior ileal resection, as it may lead not only to hematological abnormalities but also to neurological complications [14,15]. Vitamin D deficiency and concomitant disturbances in bone metabolism increase the risk of osteopenia and osteoporosis, particularly in patients treated with glucocorticoids [18,19].

Appropriate diagnosis of nutritional deficiencies remains a challenge in routine clinical practice. Commonly used parameters such as body weight and BMI do not always reflect true nutritional status, especially in patients with concomitant sarcopenia [12,19]. Furthermore, interpretation of laboratory markers, particularly those related to iron metabolism, requires consideration of the inflammatory state. Ferritin, as an acute-phase protein, may be elevated despite coexisting iron deficiency, which further complicates clinical assessment [16,19].

Therefore, nutritional assessment should be multidimensional and include clinical evaluation, appropriately selected laboratory tests, and, whenever possible, body composition analysis.

In recent years, increasing attention has been given to nutritional interventions as a component of comprehensive CD management. Supplementation of selected nutrients, enteral nutrition, and selected dietary strategies may improve nutritional status and support treatment of the underlying disease [17,19,23,25]. However, it should be emphasized that the effectiveness of specific dietary interventions, particularly in adult patients, is variable and in some cases remains insufficiently established. This applies particularly to exclusion diets, which require careful implementation to avoid secondary deficiencies [19,25].

However, the available literature is heterogeneous with regard to definitions of nutritional deficiencies, study populations, and assessed clinical outcomes, which limits direct comparison between studies. This is particularly relevant for dietary interventions in adult patients, where the quality of evidence remains limited.

6. Conclusions

Nutritional deficiencies should be regarded as an integral component of the clinical picture of Crohn's disease rather than merely a secondary consequence. Their presence has important implications for disease course and treatment efficacy, which justifies the need for systematic assessment in routine clinical practice.

Early identification of deficiencies and implementation of individualized therapeutic management, including supplementation and nutritional support, are of key importance. Incorporating nutritional aspects into comprehensive patient care may contribute to improved treatment outcomes and better quality of life in patients with Crohn's disease.

Abbreviations

- **CD:** Crohn's disease
- **IBD:** Inflammatory bowel disease
- **TNF-α:** Tumor necrosis factor-alpha
- **IL-6:** Interleukin-6
- **BMI:** Body mass index
- **MUST:** Malnutrition Universal Screening Tool
- **NRS-2002:** Nutritional Risk Screening 2002
- **EEN:** Exclusive enteral nutrition
- **CDED:** Crohn's Disease Exclusion Diet

- **FODMAP:** Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols
- **GH:** Growth hormone
- **IGF-1:** Insulin-like growth factor 1
- **ACD:** Anemia of chronic disease

DECLARATIONS

Authors' contributions

Conceptualization: Natalia Grabowska;
Methodology: Weronika Martynowska, Michał Hajt, Paulina Turzyńska;
Formal analysis: Dastin Misiaszek, Alicja Graczyk;
Investigation: Anna Wolszczak, Jakub Motor, Paulina Turzyńska;
Writing — original draft preparation: Weronika Martynowska, Piotr Ciecierski;
Writing — review and editing: Natalia Grabowska, Alicja Graczyk;
Supervision: Natalia Grabowska, Michał Hajt.
All authors have read and agreed to the published version of the manuscript.

Funding

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Acknowledgments

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

References

1. Feuerstein JD, Cheifetz AS. Crohn Disease: Epidemiology, Diagnosis, and Management. Mayo Clin Proc. 2017;92(7):1088-1103. https://doi.org/10.1016/j.mayocp.2017.04.010

2. Gajendran M, Loganathan P, Catinella AP, Hashash JG. A comprehensive review and update on Crohn's disease. Dis Mon. 2018;64(2):20-57. https://doi.org/10.1016/j.disamonth.2017.07.001

3. Lusetti F, Bezzio C, De Bernardi A, Puricelli M, Manes G, Saibeni S. Upper Gastrointestinal Crohn's Disease: Shedding Light on the Obscure L4 Classification Meaning. J Clin Med. 2025;14(22):8260. https://doi.org/10.3390/jcm14228260

4. Petagna L, Antonelli A, Ganini C, Bellato V, Campanelli M, Divizia A, Efrati C, Franceschilli M, Guida AM, Ingallinella S, Montagnese F, Sensi B, Siragusa L, Sica GS. Pathophysiology of Crohn's disease inflammation and recurrence. Biol Direct. 2020;15(1):23. https://doi.org/10.1186/s13062-020-00280-5

5. Cushing K, Higgins PDR. Management of Crohn Disease: A Review. JAMA. 2021;325(1):69-80. https://doi.org/10.1001/jama.2020.18936

6. Ananthakrishnan AN. Environmental triggers for inflammatory bowel disease. Curr Gastroenterol Rep. 2013;15(1):302. https://doi.org/10.1007/s11894-012-0302-4

7. Ananthakrishnan AN. Environmental risk factors for inflammatory bowel diseases: a review. Dig Dis Sci. 2015;60(2):290-298. https://doi.org/10.1007/s10620-014-3350-9

8. Mark-Christensen A, Lange A, Erichsen R, Frøslev T, Esen BÖ, Sørensen HT, Kappelman MD. Early-Life Exposure to Antibiotics and Risk for Crohn's Disease: A Nationwide Danish Birth Cohort Study. Inflamm Bowel Dis. 2022;28(3):415-422. https://doi.org/10.1093/ibd/izab085

9. Klein A, Eliakim R. Non Steroidal Anti-Inflammatory Drugs and Inflammatory Bowel Disease. Pharmaceuticals (Basel). 2010;3(4):1084-1092. https://doi.org/10.3390/ph3041084

10. Calvez V, Puca P, Di Vincenzo F, Del Gaudio A, Bartocci B, Murgiano M, Iaccarino J, Parand E, Napolitano D, Pugliese D, Gasbarrini A, Scaldaferri F. Novel Insights into the Pathogenesis of Inflammatory Bowel Diseases. Biomedicines. 2025;13(2):305. https://doi.org/10.3390/biomedicines13020305

11. Valvano M, Capannolo A, Cesaro N, Stefanelli G, Fabiani S, Frassino S, Monaco S, Magistroni M, Viscido A, Latella G. Nutrition, Nutritional Status, Micronutrients Deficiency, and Disease Course of Inflammatory Bowel Disease. Nutrients. 2023;15(17):3824. https://doi.org/10.3390/nu15173824

12. Jabłońska B, Mrowiec S. Nutritional Status and Its Detection in Patients with Inflammatory Bowel Diseases. Nutrients. 2023;15(8):1991. https://doi.org/10.3390/nu15081991

13. Massironi S, Rossi RE, Cavalcoli FA, Della Valle S, Fraquelli M, Conte D. Nutritional deficiencies in inflammatory bowel disease: therapeutic approaches. Clin Nutr. 2013;32(6):904-910. https://doi.org/10.1016/j.clnu.2013.03.020

14. Weisshof R, Chermesh I. Micronutrient deficiencies in inflammatory bowel disease. Curr Opin Clin Nutr Metab Care. 2015;18(6):576-581. https://doi.org/10.1097/MCO.0000000000000226

15. Kilby K, Mathias H, Boisvenue L, Heisler C, Jones JL. Micronutrient Absorption and Related Outcomes in People with Inflammatory Bowel Disease: A Review. Nutrients. 2019;11(6):1388. https://doi.org/10.3390/nu11061388

16. Maas LA, Krishna M, Parian AM. Ironing It All Out: A Comprehensive Review of Iron Deficiency Anemia in Inflammatory Bowel Disease Patients. Dig Dis Sci. 2023;68(2):357-369. https://doi.org/10.1007/s10620-022-07599-1

17. Hashash JG, Elkins J, Lewis JD, Binion DG. AGA Clinical Practice Update on Diet and Nutritional Therapies in Patients With Inflammatory Bowel Disease: Expert Review. Gastroenterology. 2024;166(3):521-532. https://doi.org/10.1053/j.gastro.2023.11.303

18. Kreienbuehl AS, Rogler G, Emanuel B, Biedermann L, Meier C, Juillerat P, Restellini S, Hruz P, Vavricka SR, Aeberli D, Seibold F. Bone health in patients with inflammatory bowel disease. Swiss Med Wkly. 2024;154:3407. https://doi.org/10.57187/s.3407

19. Forbes A, Escher J, Hébuterne X, Kłęk S, Krznaric Z, Schneider S, Shamir R, Stardelova K, Wierdsma N, Wiskin AE, Bischoff SC. ESPEN guideline: Clinical nutrition in inflammatory bowel disease. Clin Nutr. 2017;36(2):321-347. https://doi.org/10.1016/j.clnu.2016.12.027

20. Ganz T, Nemeth E. Iron homeostasis in host defence and inflammation. Nat Rev Immunol. 2015;15(8):500-510. https://doi.org/10.1038/nri3863

21. Walters TD, Griffiths AM. Mechanisms of growth impairment in pediatric Crohn's disease. Nat Rev Gastroenterol Hepatol. 2009;6(9):513-523. https://doi.org/10.1038/nrgastro.2009.124

22. Levine A, Wine E, Assa A, Sigall Boneh R, Shaoul R, Kori M, Cohen S, Peleg S, Shamaly H, On A, Millman P, Abramas L, Ziv-Baran T, Grant S, Abitbol G, Dunn KA, Bielawski JP, Van Limbergen J. Crohn's Disease Exclusion Diet Plus Partial Enteral Nutrition Induces Sustained Remission in a Randomized Controlled Trial. Gastroenterology. 2019;157(2):440-450.e8. https://doi.org/10.1053/j.gastro.2019.04.021

23. Bueno-Hernández N, Yamamoto-Furusho JK, Mendoza-Martínez VM. Nutrition in Inflammatory Bowel Disease: Strategies to Improve Prognosis and New Therapeutic Approaches. Diseases. 2025;13(5):139. https://doi.org/10.3390/diseases13050139

24. Turner D, Ricciuto A, Lewis A, D'Amico F, Dhaliwal J, Griffiths AM, Bettenworth D, Sandborn WJ, Sands BE, Reinisch W, Schölmerich J, Bemelman W, Danese S, Mary JY, Rubin D, Colombel JF, Peyrin-Biroulet L, Dotan I, Abreu MT, Dignass A; International Organization for the Study of IBD. STRIDE-II: An Update on the Selecting Therapeutic Targets in Inflammatory Bowel Disease (STRIDE) Initiative of the International Organization for the Study of IBD (IOIBD): Determining Therapeutic Goals for Treat-to-Target strategies in IBD. Gastroenterology. 2021;160(5):1570-1583. https://doi.org/10.1053/j.gastro.2020.12.031

25. Narula N, Dhillon A, Zhang D, Sherlock ME, Tondeur M, Zachos M. Enteral nutritional therapy for induction of remission in Crohn's disease. Cochrane Database Syst Rev. 2018;4(4):CD000542. https://doi.org/10.1002/14651858.CD000542.pub3

26. Halmos EP, Gibson PR. Controversies and reality of the FODMAP diet for patients with irritable bowel syndrome. J Gastroenterol Hepatol. 2019;34(7):1134-1142. https://doi.org/10.1111/jgh.14650