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Diet and Nutrition in Inflammatory Bowel Disease: Current Evidence and Therapeutic Strategies

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Abstract

Background. Inflammatory bowel diseases (IBD), including Crohn's disease and ulcerative colitis, are chronic immune-mediated disorders involving genetic susceptibility, environmental factors, the intestinal microbiota and mucosal immune responses. Diet may influence disease risk and inflammation, while IBD can adversely affect nutritional status.

Aim. This review aimed to summarize current evidence on the role of diet and nutrition in IBD, focusing on dietary patterns and disease risk, dietary interventions, enteral nutrition and nutritional deficiencies.

Materials and Methods. A narrative literature review was conducted using PubMed/MEDLINE. Clinical guidelines, systematic reviews, meta-analyses, randomized trials and relevant prospective studies were included, with preference given to recent evidence.

Results. Dietary patterns may influence IBD risk. Higher consumption of ultra-processed foods has been associated with increased risk, whereas fiber-rich, minimally processed patterns appear favorable. Exclusive enteral nutrition has an established role in pediatric Crohn's disease. The Crohn's Disease Exclusion Diet combined with partial enteral nutrition is supported by randomized trials, while a Mediterranean-style diet represents a less restrictive approach with potential benefits. A low-FODMAP diet may improve persistent functional gastrointestinal symptoms in patients with quiescent IBD, but should not be considered an anti-inflammatory treatment. Malnutrition, iron deficiency and other micronutrient deficiencies remain important clinical concerns requiring screening and correction.

Conclusions. Diet and nutrition should be considered an integral part of comprehensive IBD management. Dietary strategies should be individualized according to disease activity, phenotype, nutritional status and symptoms, while unnecessary restrictions should be avoided. Nutritional interventions should complement, rather than replace, established medical treatment.

Keywords: inflammatory bowel disease; Crohn's disease; ulcerative colitis; diet; nutrition; dietary therapy

1. Introduction

Inflammatory bowel diseases comprise a heterogeneous group of chronic inflammatory disorders of the gastrointestinal tract, primarily Crohn's disease and ulcerative colitis. Their pathogenesis is multifactorial and reflects interactions between genetic susceptibility, mucosal immune dysregulation, environmental exposures and the intestinal microbiome [1]. The growing epidemiological burden of IBD and observed associations between pre-diagnostic dietary patterns and disease risk have intensified interest in diet as a potentially modifiable environmental factor [2].

Food is a plausible environmental modifier because the intestinal mucosa is continuously exposed to nutrients, dietary constituents and microbial metabolites. Dietary composition can influence microbial communities and metabolites, epithelial barrier function and mucosal immune responses [1]. At the same time, active IBD can reduce intake, impair absorption and increase the risk of malnutrition and micronutrient deficiencies [3]. Consequently, the relationship between nutrition and IBD is bidirectional.

Patients frequently perceive diet as one of the most important determinants of their symptoms. Many modify food intake after diagnosis, often without professional supervision. This can provide symptomatic relief, but extensive self-imposed restriction may also reduce dietary diversity and increase the risk of inadequate energy, protein and micronutrient intake. Contemporary nutritional care therefore has two distinct goals: to evaluate whether a dietary strategy can influence inflammatory disease activity and to preserve adequate nutritional status throughout the course of illness [4].

The evidence base has expanded considerably. Prospective cohorts have examined dietary patterns before IBD onset; randomized trials have assessed specific therapeutic diets; and professional societies have issued increasingly detailed recommendations. Nevertheless, nutrition remains an area in which biologically plausible mechanisms often exceed the strength of clinical evidence. It is therefore important to distinguish diets that improve symptoms from interventions that have demonstrated an effect on objective inflammation or remission [5,6].

2. Aim of the review

The aim of this review was to summarize and critically discuss current evidence concerning diet and nutrition in inflammatory bowel disease. Particular attention was given to dietary exposures associated with the development of IBD, potential mechanisms linking food with intestinal inflammation, the role of the gut microbiota, therapeutic dietary strategies, enteral

nutrition, common nutritional deficiencies and current practical recommendations for patients with Crohn's disease and ulcerative colitis.

3. Materials and Methods

A narrative review of the literature was conducted using the PubMed/MEDLINE database. The search included combinations of terms such as "inflammatory bowel disease", "Crohn's disease", "ulcerative colitis", "diet", "nutrition", "dietary therapy", "dietary pattern", "Mediterranean diet", "Crohn's Disease Exclusion Diet", "specific carbohydrate diet", "low FODMAP", "exclusive enteral nutrition", "ultra-processed food", "fiber", "microbiome", "malnutrition", "iron deficiency" and "vitamin D". Priority was given to current clinical guidelines and consensus documents, systematic reviews, meta-analyses, randomized controlled trials and large prospective cohort studies. Recent publications were preferentially selected, while older studies were retained when they represented landmark evidence or remained relevant to present clinical practice. Studies concerning both adult and pediatric populations were considered. The review was designed to provide a clinically oriented synthesis rather than a formal systematic review, and no quantitative meta-analysis was performed.

4. Diet and the Development of Inflammatory Bowel Disease

4.1. Dietary patterns and the Western diet

The concept of a "Western diet" encompasses a dietary pattern characterized by a high intake of ultra-processed products, refined carbohydrates, saturated fat, added sugars and processed meat, with comparatively low consumption of fiber-rich plant foods. Such a pattern has been proposed as one factor contributing to the increasing incidence of IBD in populations undergoing rapid socioeconomic transition. Observational studies do not prove causality, but systematic reviews suggest that dietary patterns may be more informative than analysis of isolated nutrients because foods are consumed in combinations and may exert additive or interacting effects [2,5].

Prospective European data have supported the relevance of overall dietary patterns. In the EPIC cohort, a dietary pattern characterized by higher consumption of sugar and soft drinks and lower vegetable intake was associated with a higher risk of ulcerative colitis [7]. More recent systematic analyses emphasize substantial heterogeneity between studies and do not support identifying a single food as a universal cause of IBD [2,5]. These findings favor consideration of overall dietary patterns rather than isolated foods.

4.2. Ultra-processed foods

Ultra-processed foods (UPFs) have received particular attention because they combine several characteristics that may be relevant to intestinal health: high energy density, low fiber content, refined ingredients and frequent exposure to emulsifiers, stabilizers, artificial sweeteners and other additives. In a large prospective study involving participants from multiple countries, higher UPF consumption was associated with an increased risk of incident IBD [8]. Similar associations have subsequently been reported in other cohorts, although definitions of UPFs and patterns of consumption differ between populations [5,9].

The biological explanation is likely to be more complex than the NOVA category itself. Experimental work indicates that selected emulsifiers can alter microbial composition and pro-inflammatory potential, but effects are compound-specific and cannot automatically be generalized to all food additives [10]. Thus, reducing UPF intake is reasonable as part of a generally healthy dietary pattern, but current evidence does not support attributing IBD to a single additive in routine clinical counseling [11].

4.3. Dietary fiber, fruits and vegetables

Fiber is one of the most consistently discussed dietary components in IBD prevention. Fermentable fiber can be metabolized by intestinal bacteria to short-chain fatty acids, including butyrate, linking dietary substrates with epithelial and immune function [11]. A prospective study from the Nurses' Health Study cohorts found that higher long-term fiber intake, particularly fiber derived from fruit, was associated with a lower risk of Crohn's disease but not ulcerative colitis [12].

More recent prospective data also suggest an inverse association between higher fiber intake and IBD risk [13]. Meta-analytic evidence examining fiber, fruit and vegetable consumption is broadly compatible with a protective association, although results differ according to the source of fiber and IBD subtype [14]. These findings should not be interpreted as a recommendation for unrestricted intake of coarse fiber in every patient with established IBD. Fiber advice should be individualized according to disease activity, phenotype, obstructive symptoms and tolerance, and routine long-term restriction is not recommended in all patients [3,15].

4.4. Fat, meat and other dietary exposures

Dietary fat has been investigated because fatty acid composition may influence inflammatory mediators, bile acid metabolism and microbial ecology. Studies assessing total fat, saturated fat and polyunsaturated fatty acids have produced inconsistent results, and observational

associations are difficult to separate from the broader dietary pattern. Associations involving red and processed meat are likewise inconsistent across observational evidence [5].

This distinction is illustrated by a randomized trial in Crohn's disease in which a diet low in red and processed meat did not significantly reduce symptomatic relapse compared with a higher-meat diet [16]. Therefore, recommendations to limit processed meat are well aligned with general health guidance, but the evidence that this measure alone changes the natural history of Crohn's disease is weak. The current literature increasingly favors a whole-diet approach rather than isolated elimination of individual macronutrients [5,6].

5. Diet, Gut Microbiota and Intestinal Inflammation

The intestinal microbiota provides a mechanistic bridge between diet and mucosal immunity. Dietary substrates determine which microbial metabolic pathways are favored and influence the production of short-chain fatty acids, indoles, secondary bile acids and other molecules that interact with epithelial and immune cells. IBD is associated with alterations in microbial diversity and function, although it remains difficult to determine which changes are causal and which are consequences of inflammation, medication or altered eating behavior [11].

Fiber-rich foods provide fermentable substrates for many commensal organisms. By contrast, dietary patterns low in complex plant carbohydrates may reduce production of beneficial microbial metabolites. High-fat and highly processed diets can also influence bile acid pools and microbial composition. These mechanisms are biologically plausible, but translation into clinical dietary prescriptions remains challenging because the microbiome varies substantially between individuals [11,17].

Dietary intervention studies confirm that the microbiome is modifiable, yet a microbial change should not automatically be equated with clinical benefit. A systematic review of dietary interventions in IBD concluded that several diets alter microbiota composition, but available studies are heterogeneous and do not yet identify a reproducible microbial signature that can guide routine dietary treatment [17]. This is a useful caution against overinterpreting commercial microbiome tests or promising a personalized "microbiome diet" without validated clinical endpoints [18].

6. Dietary Interventions in Established IBD

6.1. Mediterranean diet

The Mediterranean diet emphasizes vegetables, fruits, legumes, whole grains, nuts, olive oil and other minimally processed foods, while limiting highly processed foods and excess added

sugars [18]. Its appeal in IBD is based on nutritional adequacy, dietary diversity and established benefits for general health. The American Gastroenterological Association advises patients with IBD, unless contraindicated, to follow a Mediterranean-style dietary pattern for overall health and well-being [19].

Clinical evidence is encouraging but should be interpreted cautiously. In the DINE-CD randomized trial, the Specific Carbohydrate Diet was not superior to a Mediterranean diet for symptomatic remission in adults with mild-to-moderate Crohn's disease [20]. This finding is clinically relevant because the Mediterranean diet is less restrictive and easier to reconcile with long-term nutritional adequacy.

In ulcerative colitis, a randomized controlled trial in patients with quiescent disease demonstrated that a Mediterranean dietary pattern was associated with favorable changes in the bacteriome and markers related to intestinal inflammation [21]. The study supports the concept that diet may influence biological pathways even in remission. However, the number of trials remains limited, and a Mediterranean diet should not be presented as a substitute for effective maintenance medication. Its strongest current position is as a nutritionally sound baseline pattern that may offer additional disease-related benefits.

6.2. Crohn's Disease Exclusion Diet

The Crohn's Disease Exclusion Diet (CDED) is a structured whole-food intervention designed to limit selected dietary components hypothesized to adversely affect the intestinal environment. It has been studied both with partial enteral nutrition (PEN) and, in adults, without obligatory PEN [22,23]. Unlike exclusive enteral nutrition, CDED permits selected ordinary foods.

A landmark randomized controlled trial in children with mild-to-moderate Crohn's disease compared CDED plus PEN with exclusive enteral nutrition followed by PEN. Both approaches were effective for induction, but CDED plus PEN was better tolerated and was associated with a higher proportion of sustained remission at week 12 [22]. The intervention also produced microbiome changes associated with remission. These findings provided important evidence that a structured food-based intervention can have effects beyond symptom control.

Evidence has also emerged in adults. In a pilot randomized trial, CDED with or without PEN was effective for induction and maintenance of remission in adults with mild-to-moderate biologic-naïve Crohn's disease, with some evidence of endoscopic improvement [23]. Nevertheless, CDED requires education and adherence, and its evidence base is stronger in

Crohn's disease than in ulcerative colitis. Implementation should ideally involve a dietitian familiar with IBD [20].

6.3. Specific Carbohydrate Diet

The Specific Carbohydrate Diet (SCD) restricts grains and a range of disallowed carbohydrates while permitting selected monosaccharides, fruits, vegetables, nuts, meats and other specified foods. It has attracted considerable patient interest, but its restrictive nature increases treatment burden and may reduce dietary variety.

The DINE-CD trial directly compared SCD with a Mediterranean diet in adults with Crohn's disease and mild-to-moderate symptoms. SCD did not produce superior symptomatic remission, fecal calprotectin response or C-reactive protein response [20]. The trial does not establish that SCD is ineffective for every individual, but it weakens the rationale for routinely recommending a more restrictive diet when a less restrictive Mediterranean pattern performs similarly. Current systematic reviews conclude that evidence for many solid-food therapeutic diets remains low or very low in certainty [24].

6.4. Low-FODMAP diet

A low-FODMAP diet restricts fermentable oligosaccharides, disaccharides, monosaccharides and polyols. In IBD it has primarily been studied as a treatment for persistent functional gastrointestinal symptoms in patients with quiescent disease rather than as a therapy for active mucosal inflammation [25,26].

In a randomized trial involving patients with quiescent IBD and persistent gastrointestinal symptoms, a four-week low-FODMAP diet improved symptom-related outcomes compared with a control diet [25]. Earlier randomized rechallenge data also showed that fermentable carbohydrates can exacerbate functional symptoms in IBD [26]. In the randomized trial, symptom improvement was not accompanied by evidence that the diet acted as an anti-inflammatory treatment [25]. Low-FODMAP treatment should therefore be considered a symptom-directed intervention after active inflammatory disease has been assessed.

Because low-FODMAP treatment is restrictive, it should be used with attention to nutritional adequacy and should not lead to unnecessary long-term food avoidance [25,27]. This is particularly relevant in IBD, where patients may already restrict multiple foods because of symptoms or fear of relapse [4].

6.5. Other exclusion and anti-inflammatory diets

A range of additional dietary approaches has been studied, including anti-inflammatory diets, low-fat high-fiber diets, gluten-free diets and various exclusion protocols. Some trials report improvements in symptoms, quality of life or biomarkers, but sample sizes are often small and interventions differ substantially. A systematic review and meta-analysis of controlled dietary trials concluded that evidence for solid-food diets is heterogeneous and that confidence in many estimates remains limited [24].

An anti-inflammatory dietary intervention in ulcerative colitis has shown potential to reduce subclinical inflammatory activity and improve selected outcomes, supporting further research [28]. Nevertheless, "anti-inflammatory diet" is not a single standardized intervention, and its components overlap substantially with general principles of minimally processed, plant-rich eating. Until larger replicated trials are available, such diets should be described as promising rather than established disease-modifying therapy.

7. Enteral Nutrition

7.1. Exclusive enteral nutrition in Crohn's disease

Exclusive enteral nutrition (EEN) involves the use of a nutritionally complete liquid formula as the sole source of nutrition for a defined period, usually several weeks. It occupies a unique position because it is both nutritional support and an anti-inflammatory treatment. In pediatric Crohn's disease, EEN is an established first-line option for induction of remission and avoids corticosteroid-related adverse effects on growth and bone health [29].

The mechanism of EEN is incompletely understood and may involve changes in the intestinal microbiota, epithelial barrier function and inflammatory pathways [29,30]. Available comparative evidence does not demonstrate a clear remission advantage of elemental over non-elemental formula preparations.

A Cochrane review comparing enteral nutrition with corticosteroids found particularly important support for enteral nutrition in pediatric Crohn's disease, while corticosteroids were more effective for induction in adults in the available trials [31]. In adult practice, EEN is used less consistently, in part because adherence is challenging, but it can be considered in selected patients and as part of nutritional optimization [30].

7.2. Partial enteral nutrition

Partial enteral nutrition provides only part of total energy requirements from formula while ordinary food supplies the remainder. PEN alone has not demonstrated the same induction

efficacy as EEN, but it can be useful as part of structured approaches such as CDED and may contribute to maintenance of nutritional status. The distinction between PEN as simple supplementation and PEN as a component of a defined therapeutic diet is important when interpreting studies [22,32].

For children and adolescents, nutritional therapy has additional relevance because active Crohn's disease can impair growth and nutritional development. Pediatric management therefore includes assessment of growth and nutritional status alongside control of intestinal inflammation [29].

8. Nutritional Status and Deficiencies in IBD

8.1. Malnutrition and body composition

Malnutrition in IBD can result from reduced food intake, abdominal pain, nausea, restrictive eating, increased inflammatory requirements, protein loss, malabsorption and previous intestinal resection. Importantly, malnutrition is not limited to underweight patients. Individuals with normal or elevated body mass index may have reduced lean mass or sarcopenia, which can remain unnoticed if assessment relies only on body weight [33].

Nutritional screening should therefore form part of routine IBD care, especially during active disease, hospitalization and before surgery. When risk is identified, assessment should include recent weight change, dietary intake, disease phenotype, gastrointestinal losses and relevant laboratory parameters. ESPEN guidance emphasizes that nutritional care is an integral component of IBD management rather than an optional adjunct [34].

8.2. Iron deficiency and anemia

Iron deficiency is among the most frequent nutritional problems in IBD. It may result from chronic gastrointestinal blood loss, reduced intake and impaired absorption. Inflammation also contributes to anemia through hepcidin-mediated iron sequestration and reduced availability for erythropoiesis. Consequently, hemoglobin alone is insufficient to characterize the problem, and interpretation of ferritin should take inflammatory activity into account..

Treatment of iron deficiency depends on disease activity, severity of anemia, tolerance and the likelihood of adequate intestinal absorption. Oral iron can be appropriate in selected patients, whereas intravenous iron is preferred in several clinical situations, including active disease and intolerance to oral preparations [35].

8.3. Vitamin D, calcium and bone health

Vitamin D deficiency is frequently reported in IBD and may reflect reduced intake, limited sunlight exposure, malabsorption or the consequences of active disease. Observational studies have linked lower vitamin D status with adverse disease outcomes, but these associations do not establish that supplementation beyond correction of deficiency modifies intestinal inflammation. Vitamin D should therefore be assessed and replaced according to nutritional and bone-health indications rather than presented as a stand-alone IBD therapy [3,36].

Bone health requires particular attention in patients with risk factors such as prolonged inflammation, corticosteroid exposure, low body weight or inadequate calcium and vitamin D intake [37]. Children and adolescents merit additional attention because chronic disease and nutritional compromise may affect growth and bone acquisition [3,29].

8.4. Vitamin B12, folate and other micronutrients

Vitamin B12 deficiency is most relevant in Crohn's disease involving the terminal ileum and after significant ileal resection. Folate status may be affected by reduced intake and selected medications. Other deficiencies described in IBD include zinc, magnesium and fat-soluble vitamins, particularly in patients with extensive small-bowel disease, high-output stomas or chronic diarrhea.

Micronutrient assessment and replacement should be individualized according to disease phenotype, previous surgery, dietary intake and documented or suspected deficiency rather than relying on indiscriminate high-dose supplementation [37]. Dietetic review is particularly valuable when intake is restricted or nutritional risk is increased [3,27].

9. Dietary Management During Remission and Active Disease

Dietary advice should change with clinical context. During remission, the main objectives are nutritional adequacy, dietary diversity and long-term health. A Mediterranean-style pattern rich in varied plant foods, whole grains when tolerated, legumes, nuts and olive oil is compatible with current expert recommendations [18]. Routine exclusion of dairy products, gluten, fruits or vegetables is not recommended for all patients and should instead be guided by specific clinical indications and individual tolerance [6,38].

During active disease, symptoms may temporarily limit tolerance of high-volume, high-fiber or high-fat meals. Patients with stricturing Crohn's disease may require modification of food texture and avoidance of poorly digestible large particles. This is different from prescribing a

permanently low-fiber diet to all patients with IBD. The degree of restriction should be proportional to the clinical problem and relaxed when it is safe to do so [39].

Hydration and electrolyte replacement deserve attention in patients with frequent diarrhea, ileostomy or short bowel. Small frequent meals may be easier to tolerate during periods of poor appetite. When oral intake is insufficient, oral nutritional supplements or enteral feeding should be introduced early rather than waiting for severe weight loss. Parenteral nutrition is reserved for situations in which the gastrointestinal tract cannot be used adequately; it is nutritional support, not primary anti-inflammatory therapy [3,6].

10. Food Avoidance, Patient Beliefs and the Risk of Over-Restriction

The gap between patient experience and clinical evidence is particularly visible in nutrition. Many patients can identify foods that worsen urgency, bloating or pain, and these observations are clinically meaningful. However, a food that triggers symptoms does not necessarily increase mucosal inflammation. Failure to distinguish these concepts can lead to increasingly restrictive diets and anxiety surrounding food.

Surveys and reviews consistently show that patients with IBD frequently modify their diet and often obtain advice outside specialist healthcare. Restriction can be extensive, especially during or after a severe flare. This creates a paradox: patients may pursue dietary strategies to improve health while inadvertently reducing energy intake, protein intake and dietary diversity [4].

Persistent gastrointestinal symptoms should not automatically be attributed to active IBD, and objective assessment of inflammatory activity is important before symptom-directed dietary restriction. A low-FODMAP diet may be reasonable for selected patients with quiescent IBD and persistent functional gastrointestinal symptoms [25].

11. Current Practical Recommendations

Current guidance increasingly moves away from universal lists of prohibited foods. The AGA recommends a Mediterranean-style dietary pattern for overall health in IBD and emphasizes low consumption of ultra-processed foods, added sugars and salt [19]. At the same time, it acknowledges that no single diet has consistently been shown to prevent flares in all adults with IBD. This balanced message is important: diet matters, but the evidence does not justify promising disease control through food alone.

Recent European consensus work has further formalized dietary management as a component of IBD care, reflecting the transition of nutrition from a largely empirical topic toward evidence-based clinical practice [38]. The strongest recommendations are those supported by

reproducible clinical data, such as EEN in pediatric Crohn's disease. For other approaches, recommendations must reflect the certainty of evidence and the burden imposed on the patient. In practice, dietary counseling should be individualized according to IBD subtype, disease activity, phenotype, history of surgery, nutritional status and patient preferences. Involvement of a dietitian with IBD expertise is recommended particularly when therapeutic or restrictive diets are considered, both to support adherence and to preserve nutritional adequacy [6,38].

12. Discussion

The relationship between diet and IBD is no longer limited to the question of which foods patients should avoid. Current research spans epidemiology, microbial ecology, metabolomics, clinical nutrition and therapeutic dietary trials. This broader perspective has produced a more coherent model in which diet can influence disease susceptibility and intestinal physiology, while established IBD simultaneously changes nutritional needs and food tolerance. However, the strength of evidence varies markedly between these domains [40].

One of the most consistent epidemiological observations is the association between lower dietary quality, particularly high UPF consumption, and IBD risk [8,9]. Prospective fiber studies point in the opposite direction, with higher fiber intake associated with lower risk in several analyses [12-14]. These findings are biologically plausible because minimally processed plant foods provide fermentable substrates and micronutrients, whereas UPFs often displace fiber-rich foods and introduce multiple additives. Nevertheless, observational studies cannot fully separate diet from socioeconomic status, smoking, physical activity, antibiotic exposure and other lifestyle factors. For this reason, risk associations should not be translated into claims that a particular food causes or prevents IBD in an individual patient [41].

The therapeutic literature provides a different level of evidence. EEN has the clearest established role, particularly in pediatric Crohn's disease [29,31]. CDED plus PEN represents an important development because it demonstrates that a structured whole-food intervention can induce and sustain remission in a proportion of patients while being more acceptable than complete formula feeding [22]. Adult CDED data are promising but less extensive [23]. Across solid-food dietary interventions, however, the certainty of evidence remains uneven [42]. These approaches also illustrate that dietary therapy in IBD is not synonymous with generic "healthy eating"; effective protocols may require defined composition, monitoring and adherence.

The Mediterranean diet occupies a somewhat different position because it is nutritionally balanced and less restrictive than many exclusion diets [18]. Randomized IBD studies have reported potential symptomatic, inflammatory or microbiome-related benefits [20,21], while

reviews focused specifically on this pattern still describe the evidence as insufficient to establish it as a stand-alone disease-modifying therapy. Its practical advantage is that it remains compatible with long-term general health recommendations even when the magnitude of its IBD-specific anti-inflammatory effect is uncertain [18].

The low-FODMAP diet demonstrates why outcomes must be defined carefully. It can improve persistent gastrointestinal symptoms in quiescent IBD [25,26], yet symptom improvement does not necessarily indicate suppression of intestinal inflammation. A patient may feel substantially better while fecal calprotectin and endoscopic activity remain unchanged. Conversely, a patient in endoscopic remission may continue to experience bloating and abdominal discomfort that respond to dietary manipulation. The same need for nuance applies to fiber: its physiological effects depend on fiber type, disease activity, phenotype and individual tolerance rather than a simple high-versus-low classification [15]. Separating inflammatory control from symptom control prevents both undertreatment of active disease and unnecessary escalation of medication in functional symptoms.

Another important issue is the nutritional cost of restriction. Patients with IBD frequently modify or restrict their diets, and these behaviors may be driven by symptoms, beliefs about food and fear of disease activity [4]. Because restrictive diets may compromise dietary adequacy and the evidence supporting many exclusion strategies is limited, the burden of an intervention should be weighed against the certainty of expected benefit [24,27].

The microbiome remains a major research focus but is not yet a routine tool for selecting diets. Different interventions can change microbial composition, and some changes correlate with clinical response, yet no validated microbial profile currently identifies which patient should receive CDED, Mediterranean diet or another intervention [11,17]. Future studies combining dietary assessment with metagenomics, metabolomics and objective inflammatory endpoints may allow more precise nutritional therapy. Until then, microbiome-based dietary recommendations should be presented cautiously.

There are several limitations to the current evidence. Dietary trials are difficult to blind, adherence is challenging to measure, and control diets may themselves change participant behavior. Definitions of remission vary between symptoms, biomarkers and endoscopy. Many studies are small and of short duration, whereas IBD is a lifelong disease. Furthermore, a diet that is feasible during a supervised trial with provided food may be much harder to maintain in routine practice. These limitations explain why systematic reviews frequently rate the certainty of evidence for specific solid-food diets as low [24].

Despite these uncertainties, several practical conclusions are robust. Nutritional screening should be routine and documented deficiencies should be corrected [34]. EEN remains a major therapeutic option in pediatric Crohn's disease [29,31], while CDED plus PEN is supported by randomized evidence [22]. Mediterranean-style eating is a reasonable general dietary pattern for many patients with IBD [18]. Low-FODMAP therapy can improve persistent functional symptoms in selected patients with quiescent disease [25,26]. Dietary strategies should complement rather than replace effective medical treatment [6,38].

The direction of future research should move from broad labels such as "Western diet" toward reproducible descriptions of food composition, processing and individual response. Trials should include objective endpoints such as fecal calprotectin and endoscopic healing, assess nutritional adequacy and report adherence. Long-term studies are needed to determine whether dietary interventions can reduce relapse, complications, surgery or treatment escalation. The recent development of formal dietary consensus recommendations indicates that nutrition is becoming a more mature field within IBD care, but substantial uncertainty remains [38].

13. Conclusions

Diet and nutrition are clinically important throughout the course of inflammatory bowel disease. Current evidence suggests that overall dietary quality is more informative than the elimination of individual foods. Higher consumption of ultra-processed foods has been associated with increased IBD risk, whereas fiber-rich and minimally processed dietary patterns appear more favorable, although observational associations do not prove causation.

Among therapeutic interventions, exclusive enteral nutrition has the strongest established role in pediatric Crohn's disease. CDED combined with partial enteral nutrition is supported by randomized clinical evidence, while Mediterranean-style eating is a practical and nutritionally balanced long-term approach with emerging IBD-specific data. A low-FODMAP diet may improve functional gastrointestinal symptoms in patients with quiescent disease but should not be considered treatment of active mucosal inflammation.

Nutritional assessment is as important as dietary prescription. Malnutrition, iron deficiency and other micronutrient deficiencies should be actively identified and corrected. Dietary recommendations should account for disease activity, phenotype, strictures, previous surgery, symptoms and individual preferences. The most appropriate nutritional strategy is therefore not a universal IBD diet, but an individualized approach that preserves dietary adequacy, minimizes unnecessary restriction and complements effective medical therapy.

Disclosure

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