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Review

Role of Nutrition in the Management of Inflammatory Bowel Disease

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Abstract

The persistent inflammation of the intestinal mucosa is the main characteristic of inflammatory bowel disease (IBD), a lifelong illness that affects people of both genders. The exact etiology is still undefined while previously it was attributed to genetic components. Later, it was investigated that the environment has an equal effect on the development of IBD encompassing both Crohn's disease (CD) and Ulcerative colitis (UC). Among environmental risk factors linked to IBD, diet, notably the transition toward the Western diet, is the most noticeable element that contributes to IBD. This comprehensive overview summarizes the

present literature on the role of food and food groups in IBD nutrition and management. Increased consumption of sugar, fats, and/or proteins, decreased consumption of fruits and vegetables, and increased use of emulsifiers or other binding compounds are among the most significant candidates that lead to inflammation in the intestines of healthy individuals. Furthermore, low dietary habits, including selective malnutrition, obesity, and sarcopenia, are linked with poor clinical results, treatment responsiveness, and, as a result, quality of life. Therefore, the trend in the management of IBD has shifted toward dietary therapies and strategies like Specific Carbohydrate Diet (SCD), the Mediterranean diet (MD), Enteral Nutrition (EN), Crohn's disease exclusion diet (CDED), Inflammatory bowel disease-Anti-inflammatory diet (IBD-AID), and others are reported to have had a positive association with reducing the underlying IBD symptoms and remission of intestinal homeostasis.

Keywords
Inflammatory bowel disease; Crohn's disease; ulcerative colitis; nutrition; nutritional management; gut microbiome; diet therapies

1. Introduction

Crohn's disease (CD) and Ulcerative colitis (UC) are collectively known as inflammatory bowel disease (IBD), a lifelong condition that affects both males and females from a young age. It is characterized by chronic inflammation of the intestinal mucosa with an unknown etiology, leading to significant psychosocial and physical challenges for patients [1, 2]. Since the latter half of the 20th century, the prevalence and incidence of IBD have increased substantially, establishing it as one of the most common gastrointestinal disorders of the 21st century, with rising incidence rates in newly industrialized countries. More than 1.5 million individuals in North America and 2 million Europeans are affected with IBD, with medication costs comprising a large share of healthcare expenses. The epidemiological evolution of IBD emergence in developing countries over the past 25 years suggests a link between industrialization and the Western lifestyle [2-4]. Although the precise cause of IBD remains unidentified, its pathogenesis is considered multifactorial, involving factors such as mucosal barrier dysfunction, genetic and epigenetics predisposition, immune dysregulation, microbial disturbances in the gastrointestinal tract (GIT), and environmental as well as lifestyle influences as depicted in Figure 1 [5, 6].

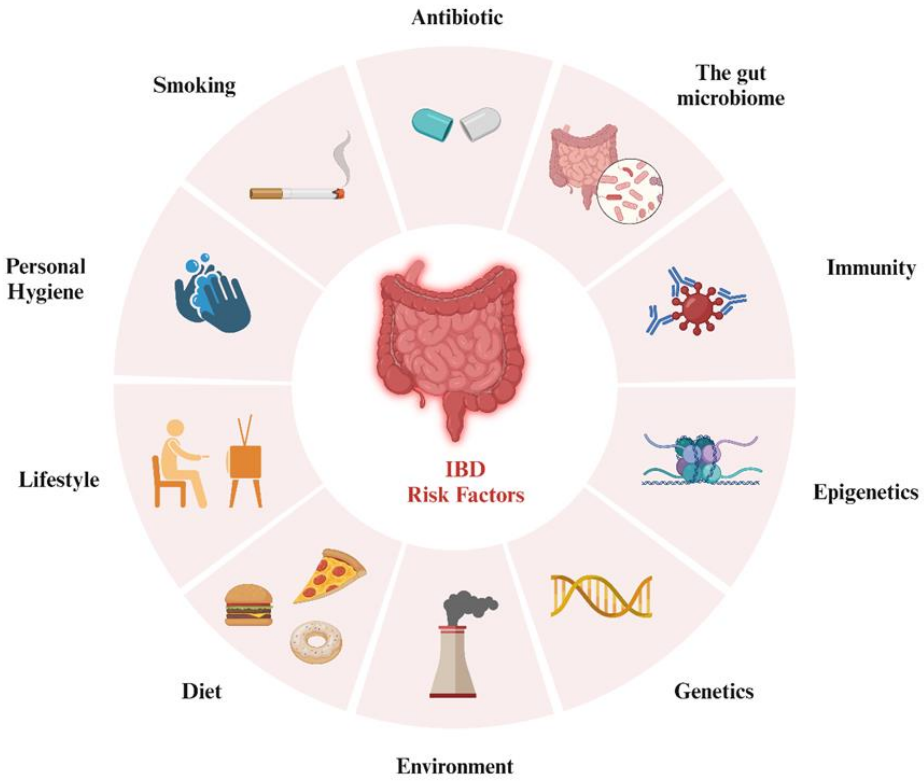

Figure 1 illustrates the risk factors reported in the literature associated with worsening symptoms of IBD, including CD and UC.

Early research on IBD primarily focused on genetic factors, leading to the discovery of the NOD2/CARD15 susceptibility gene on chromosome 16 in CD. NOD2, a cytosolic protein, encodes a pattern recognition receptor critical to the host's immune response to microbes by binding to muramyl dipeptide (MDP), a fragment of bacterial cell walls. Upon NOD2 interaction, oligomerization triggers the assembly and activation of nuclear factor-kappa B (NF-κB) and Mitogen-activated protein kinase (MAPK) pathways, resulting in the transcription of inflammatory cytokines that drive pathological inflammation and mucosal injury [7, 8] as depicted in Figure 2. However, this enhanced acquired immune response accounts for only 25% of CD cases in Caucasian populations and is not implicated in the pathogenesis of UC [9]. In the last decade, genome-wide association studies (GWAS) have identified over 200 genetic loci linked to IBD, with some overlapping across other chronic autoimmune diseases [10]. Most of these loci are commonly shared across different ancestral populations, although two notable risk variants —IL23R and NOD2 — are absent in East Asians and predominantly found in European populations. NOD2, the first gene identified to influence the immune response to the gut microbiota, exemplifies these population-specific differences [11]. Despite this significant investment in genetic research, these findings have yet to translate into improved clinical outcomes, either predicting therapy responses or establishing risk assessment tools for disease progression. Consequently, this has led to a renewed focus on exploring the role of environmental exposures [3, 10].

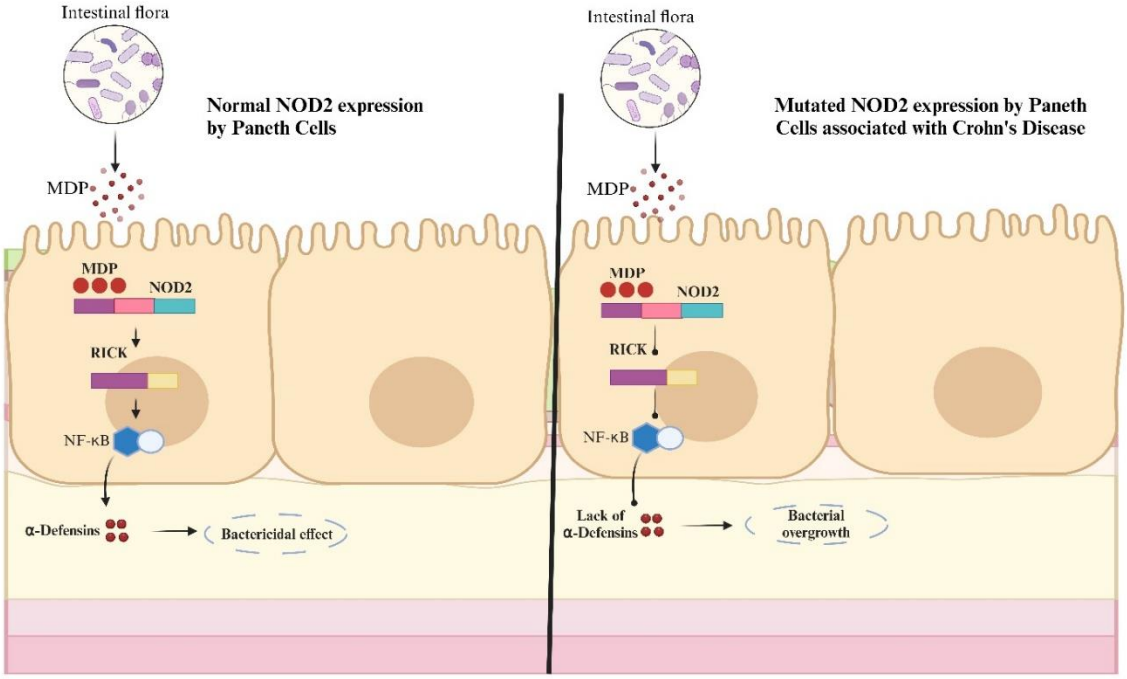

Figure 2 Paneth cells' reduced ability to produce α-defensin when NOD2 mutations associated with Crohn's disease are present.

The limited understanding of the genetic role in IBD, along with the rapid increase in the incidence of IBD in many geographic locations, recommended that both biological and environmental factors contribute to IBD [12]. Environmental risk factors associated with IBD include medication (e.g., antibiotics), smoking, viral infections, air pollutants, psychological stress, diet, and exposure to chemicals; however, evidence remains inconclusive [13, 14]. The human GIT hosts millions of microorganisms inhabiting a unique ecological niche. Gut microbiota are closely linked to many diseases, including IBD, and their impact on human health is progressively recognized, particularly regarding their role in energy metabolism and immune system regulation [15, 16]. Among the environmental factors implicated in IBD, dietary constituents emerge as key influencers of the gut microbiome's composition and function composition of the gut microbiome, wielded by the transition towards Westernized eating habits, characterized by the higher intake of proteins and unsaturated fats and diminished consumption of vegetables, fruits, and dietary fibers. This dietary "Westernization" appears to foster a proinflammatory environment, which could have a therapeutic implication for managing the disease [17, 18].

Based on the above discussion, this review aims to enhance our understanding of how various foods and food groups interact with IBD. Drawing on the available literature, our focus centers on exploring the diverse nutritional components and their mechanisms that may influence IBD progression. We also explore current nutritional strategies reported in the literature that promise to develop personalized diets for individuals affected by UC or CD.

2. Nutritional Management Strategies for IBD

2.1 Specific Carbohydrate Diet

The Specific Carbohydrate Diet (SCD) was introduced by Dr. Sydney Haas in the 1920s as a treatment for celiac disease [19]. Later, it gained popularity as a treatment for various diseases, including UC and CD [20]. This diet excludes grains, sugars (except honey), milk (excluding hard cheeses and fermented yogurt), and processed foods, because the fundamental theory of SCD states that insufficient absorption of disaccharides and polysaccharides leads to bacterial and yeast overgrowth. Mucosal injury and autoimmune responses lead to persistent carbohydrate malabsorption and chronic intestinal inflammation [21]. By addressing these factors, SCD aims to reduce intestinal inflammation, rebalance gut microbiota, and resolve dysbiosis commonly observed in IBD [22].

While small studies evaluating the role of SCD in IBD have shown clinical improvements and reductions in inflammation markers [23, 24], these findings are not universally applicable to all patients. For instance, a case report by Savini Lanka Britto and Richard Kellermayer [25] indicated that SCD monotherapy in male pediatric patients with CD supported long-term biochemical and clinical remission but did not achieve complete mucosal healing. Similarly, an online survey of IBD on SCD reported symptom reduction, with clinical improvement observed over time [26]. However, these results should be interpreted cautiously, as the diet's effectiveness can vary among individuals. Thus, while SCD may be integrated as dietary therapy for some patients, which may improve clinical and laboratory parameters, further research is needed to determine its broader applicability and efficacy across diverse patient's populations.

2.2 Mediterranean Diet

The Mediterranean Diet (MD) emphasizes plant-based foods like fruits and vegetables, as well as antioxidant-rich sources such as omega-3 polyunsaturated fats from fish and nuts and unsaturated fats like olive oil [27], while limiting red meat consumption [28]. This dietary pattern is associated with reduced inflammatory markers and improved outcomes in IBD [29], which can be attributed to its nutritional components that help control inflammation, such as antioxidants, fiber, omega-3, SCFA, and polyphenols [30]. Soluble fibers from fruits, legumes, and certain vegetables act as prebiotics, promoting the growth of beneficial gut microbiota and SCFA production, which enhances intestinal barrier function and reduces inflammatory cytokine secretion [31].

Adherence to the MD, characterized by the consumption of fruits, vegetables, and legumes has been linked to increased fecal SCFA levels [32]. This effect is facilitated by bacteria from the *Firmicutes* and *Bacteroidetes* phyla, which degrade non-digestible carbohydrates. Apart from alterations in gut microbiota, patients with CD adhering to the MD for six weeks experienced changes in the expression of over 3500 genes [33]. Transcriptomic analysis highlights the involvement of key immune regulatory pathways. For instance, IRF2 regulates NF-κB activity—a critical factor in immune control—and indirectly interacts with the signal transducer and activator of transcription 3 (STAT3) within the regulatory network [34]. STAT3 is an essential transcription factor in the JAK/STAT pathway, a notably impacted pathway in IBD [35].

While studies indicate that proper adherence to the MD positively influences gut microbiota in healthy individuals and shows favorable effects on disease activity and nutritional status in patients

with IBD [33, 36], individual responses to dietary interventions can vary [37]. The MD's high fiber content may not be suitable during disease flares. Therefore, it is recommended that the MD be modified significantly during active disease states, and individual tolerance levels should be considered when implementing this dietary model [17, 38, 39].

2.3 Enteral Nutrition

Enteral nutrition (EN) involves the administration of nutritionally complete formulas providing essential nutrients such as vitamins, minerals, proteins, carbohydrates, and fats, typically through various methods [40]. EN is widely used as a therapeutic strategy for inducing remission in CD, particularly in the pediatric population, where remission rates can reach up to 84% with exclusive enteral nutrition (ENN) over 6-12 weeks [41]. However, its role in adults as a first-line treatment for CD remains unclear. Nevertheless, clinicians often interpret the review negatively, as it shows that corticosteroids are more effective than EN when evaluated on an intention-to-treat basis. Furthermore, significant evidence suggests that providing 50% of calorie intake is an effective maintenance treatment with EN [42].

Evidence exclusively obtained from pediatric studies provides even more substantial support for the role of EN, indicating equivalent effectiveness to corticosteroids and superior progress in growth and mucosal healing [43, 44]. A recent global survey of 35 pediatric IBD centers revealed significant variation in the utilization of EN in CD [45]. The duration of EN was typically 6-8 weeks, with 90% utilizing polymeric formulas and varied strategies for food reintroduction, including low-fiber diets or gradual reintroduction of regular food. Orally administered EN, typically flavored with additives such as Nes- quick [46], is as effective as continuous feeding via a nasogastric tube [47]. While EN is particularly effective for CD involving the small bowel than the colon [48], positive results have also been observed in colonic disease. However, nearly 50% of patients experience relapse within six months of returning to a regular diet, presenting a significant challenge [49].

The mechanism through which EN benefits CD remains unclear. Potential mechanisms include reduced gut microbiota particularly, in the distal small intestine due to its low residue nature, avoidance of long-chain fats that may impair macrophage function if absorbed, and the exclusion of other harmful components such as food additives (e.g., emulsifiers or nanoparticles) [50]. Additionally, EN formulas often include anti-inflammatory substances like transforming growth factor beta (TGF-β), found in milk-derived feeds where casein is the primary protein source [51]. Subgroup analyses comparing various elemental (amino-acid-based) and non-elemental diets (semi-elemental, peptide-based, and polymeric whole protein-based, (usually with casein as the protein source) [46] have shown no significant differences in efficacy. However, Amino-acid-based feeds are characterized by lower energy density and increased hyperosmolarity, possibly contributing to lower adherence rates [49]. As a result, polymeric feeds are typically now preferred [46]. Figure 3 shows how nutritional management in IBD, whether MD, EN, or SCD, can help restore the intestinal epithelial barrier and immune hemostasis.

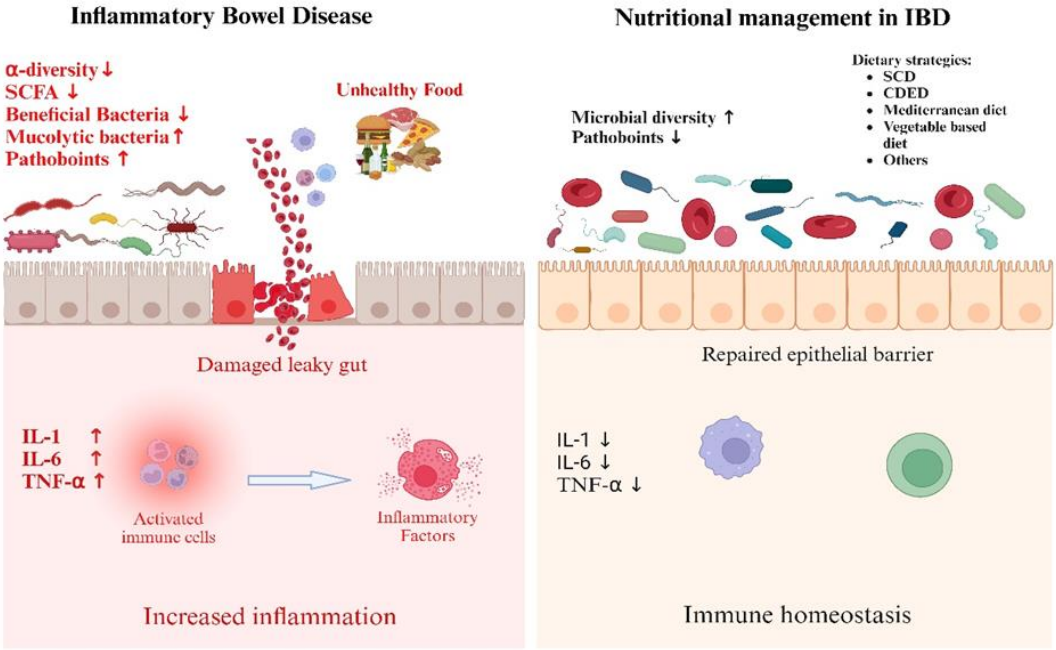

Figure 3 Depicts that literature have proven that adopting recommended diet strategies in IBD can ameliorate the symptoms and help in remission of intestinal epithelial.

2.4 Other Dietary Strategies

Several other diet-based approaches have also been proposed for the management of IBD, focusing on particular dietary inclusions and exclusions to reduce inflammation and improve gut health. However, no particular nutritional approach has been demonstrated to induce remission in adults with active IBD [52]. The efficacy of EEN, which appears comparable to steroids in inducing remission in children with CD [53], additionally, EN support utilizing a polymeric liquid formula high in TGF-β (Modulen IBD) has shown effectiveness in pediatric IBD [54]. Several diets, such as SCD, Crohn's Disease Exclusion Diet (CDED), Groningen Anti-inflammatory Diet (GrAID), Inflammatory Bowel Disease Anti-inflammatory Diet (IBD-AID), Low FODMAP Diet, CD-treat, Paleolithic diet, IgG4 exclusion diet, and semi-vegetarian diet, have been proposed as effective “anti-IBD” therapies. However, they are not widely endorsed by gastroenterologists due to limited evidence for their effectiveness [55]. Table 1 presents some IBD-recommended diets with different foods and groups being allowed or restricted in IBD (CD, UC) [56-62].

Table 1 Some of the recommended diet-based models in IBD with selected inclusion and exclusion food categories.

Food Category	SCD	MD	CDED	GrAID	IBD-AID	Low FODMAP	CD-Treat	Legend	
Added Sugar									Mandatory
Refined Sugar									Allowed
Honey									Limited
Sweetened beverages									Not allowed

SCD: Specific carbohydrate, **MD:** Mediterranean diet, **CDED:** Crohn's disease exclusion diet, **GrAID:** Groningen anti-inflammatory diet, **IBD-AID:** Inflammatory bowel disease anti-inflammatory diet, **FODMAP:** Fermentable oligosaccharides, disaccharides, and polyols, **CD:** Crohn's disease

3. Role and Effects of Specific Nutrients and Food Groups in IBD Management

Numerous studies have investigated the impact of nutrition on the susceptibility of IBD. In 2011, Hou et al [63] published the initial review paper titled "Dietary Intake and Risk of Developing IBD" employing a methodology endorsed by guidelines, they evaluated the relationship between pre-illness nutrient intake (carbohydrates, protein, fats) and food categories (fruits, vegetables, meat) and the subsequent risk of an IBD diagnosis. Similarly, the European Society for Clinical Nutrition and Metabolism (ESPEN) released updated guidelines 2011 regarding nutrition for people with IBD. However, in 2015, new standard operating procedures replaced the ESPEN guidelines and consensus papers [64].

These new and stringent ESPEN recommendation approaches emphasize disease management more than the previous technique-based approach (enteral vs parenteral) [65]. The interdisciplinary, international approach persists, but the recommendations are more organized and based on a systematic review, with the opinion of experts used only when the systematic method is not possible or provides inconsistent results [66, 67]. Figure 4 provides a general representation of specific nutrients and food groups recommended for managing IBD in patients suffering from or at high risk of developing the condition.

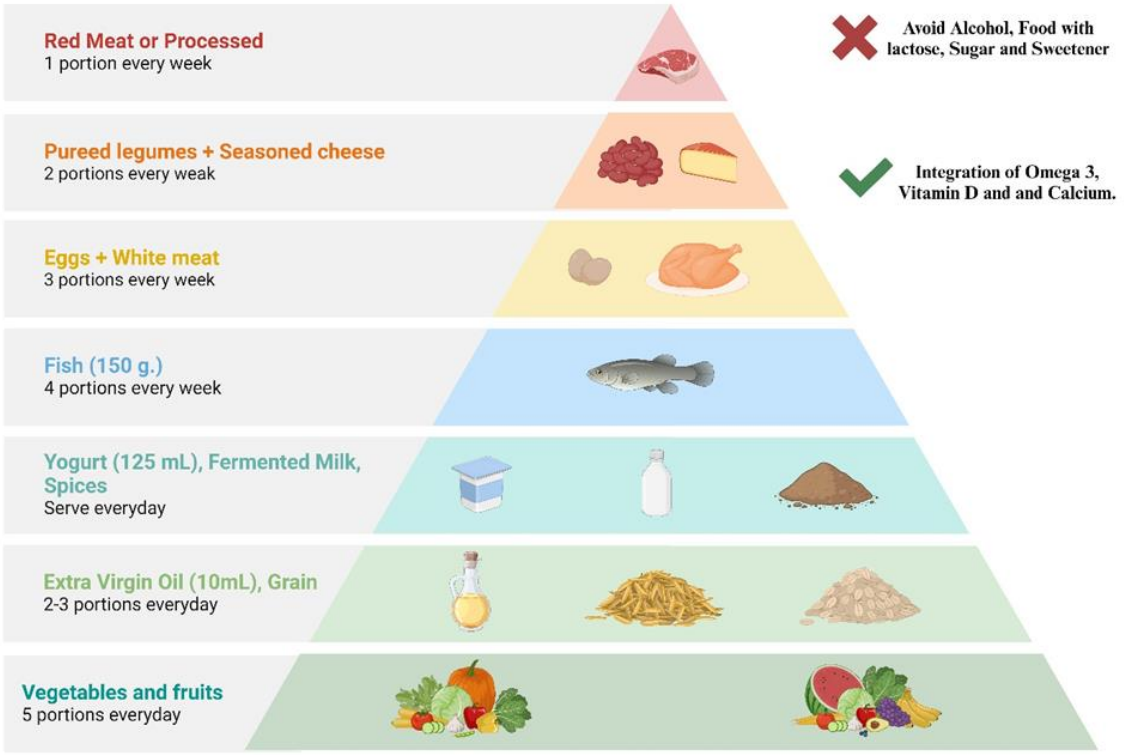

Figure 4 The recommended food pyramid for individuals who are suffering from or are at high risk of developing IBD.

3.1 Carbohydrates

Dietary carbohydrates are a group of diverse compounds with distinct physiological and physical properties, each contributing differently to health. Based on these properties carbohydrates are classified into three major groups; sugars, oligosaccharides, and polysaccharides [68]. Several studies have reported inconsistent findings regarding the link between carbohydrate consumption and IBD. While some suggest a positive association between carbohydrate intake and CD, the relationship with UC appears weaker compared to CD [69]. A larger study [54] utilizing data from the EPIC-IBD cohort across eight European countries, found no significant association between total carbohydrate intake and IBD risk in either univariate or multivariate analysis, after adjusting for total energy intake, BMI and smocking. However, when comparing the highest and lowest sugar consumers, subgroup analysis revealed a positive association between a "high sugar and soft drinks" pattern and UC, particularly among those with low vegetable intake.

In 2021, Zainab et al [70] conducted a systematic review of nine studies (1992-2019), primarily from Europe, the US, and the Asia-Pacific region, focusing on the link between total carbohydrates, sugar-sweetened beverages (SSBs), and IBD risk. No significant association was found between total carbohydrate intake and CD, UC, or IBD incidence. Similarly, another review indicated that five studies found no significant correlation between carbohydrate consumption and CD risk, while two studies reported an elevated risk with high monosaccharides and disaccharides intake, and one study found a considerable CD incidence with polysaccharides. Regarding UC, six investigations found no relationship between carbohydrate consumption and risk. Still, three studies reported a

higher UC risk with a high intake of monosaccharides and disaccharides, given the challenges in assessing dietary data, including recall bias [71]. Many other studies corroborate the finding that excessive dietary total carbohydrate intake, compared to low levels, showed no significant connection with an elevated risk of IBD as Zhong-Qin Jin et al [72] including 15 such studies in their meta-analysis.

3.2 Proteins

Dietary proteins have a significant impact on the origin or causation of IBD, resulting from increased hydrogen sulfide (H_2S) production, a potential toxin generated as a byproduct of microbial fermentation in the colon by sulfidogenic sulfate-reducing bacteria (SRB) [73-75]. Numerous research studies have explored the link between protein consumption and IBD, including UC and CD. The high intake of animal protein has been reported in incident cases of IBD [76, 77], and CD [78], surprisingly in these two investigations, this relationship was discovered only for animal proteins, not plant proteins. Particularly, among the various types of animal proteins, excessive intake of meat or fish but not eggs or dairy items has been related to IBD risk [76, 78]. In patients with UC, consumption of protein and alcohol was discovered to be related to an elevated risk of relapse [79, 80]. In contrast, poultry, fish, egg, and dairy goods were not related to the UC incidence [81] which may be due to the presence of sulfur and sulfate contents in these foods [79]. These findings indicate that, in addition to quantity, the dietary proteins may influence the likelihood of developing an IBD [82]. Concerning the amount of protein and calorie consumption in the diet, some research has indicated that patients with IBD may consume more of these nutrients than controls. However, these findings have not been consistently observed, and it is unclear whether these factors contribute to or result from the illness [73].

3.2.1 Meat

Meat is an excellent source of protein, iron, and vitamin B12, along with saturated fat [74, 83, 84]. Red meat encompasses all meat derived from livestock (lamb, mutton, beef, pork, veal, goat, horse). Processed meats, whether red or white meat undergo preparation methods such as smoking, salting (adding salt enhanced with nitrates and nitrites), curing or adding preservatives [85]. Intestinal bacteria metabolize red meat to produce branched-chain amino acids and toxic components such as hydrogen sulfide, nitrous compounds, amines, and ammonia that cause DNA damage to eukaryotic cells and induce inflammation in the colon [86-88]. Its consumption has been related to an increased risk of colon cancer and inflammation particularly IBD, because of its high saturated fat content and cooking process [89], confirmed by extensive epidemiological IBD data, particularly UC [76, 88], where it was also discovered to impact the likelihood of relapse [90].

UC patients who were on aminosalicylate therapy noted that dietary consumption of myristic acid, a saturated fatty acid common in red meat, was related to an elevated risk of a flare [91]. Similarly, in CDED, red meat is prohibited in patients who experience flares [58, 92] because relapse and flares are also reported in CD patients with high meat consumption [93]. In a prospective 2-year study, patients with IBD who consumed 15/16 of a semi-vegetarian diet maintained remission compared with 2/6 on a typical diet ($p = 0.0003$) [94]. This lacto-ovo-vegetarian is a semi-vegetarian diet includes meat once every two weeks and fish once a week [84]. Moreover, the fecal microbiome

of vegans and vegetarians revealed a decrease in the abundance of *Bifidobacteria* and *Bacteroides* species compared to omnivores residing in the same region [95].

3.2.2 Eggs

Eggs have potential dietary components that are beneficial for health. Preserved egg white, rich in high-quality protein, undergoes alkaline pickling, producing peptides and amino acids with anti-inflammatory properties [96]. Egg white protein is composed of ovalbumin, ovotransferrin (OVT), and lysozyme [97]. In comparison to other protein sources, it claims good digestibility and a remarkable protein-digestibility-adjusted amino acid score (PDCAAS) [98]. Enzymatic hydrolyses of egg white protein produce bioactive peptides with diverse biological activities, including anti-inflammatory and antioxidant, suggesting their potential in functional foods [99]. In the context of IBD, dietary components such as egg lysozyme and egg yolk peptides have demonstrated disease-alleviating effects [100]. An experimental study on DSS-induced colitis in mice revealed that OVT reduced inflammatory cytokine expression, mitigated histological and morphological damage, and improved the pathophysiology of IBD [101].

3.3 Fats

Meat and dairy products are the primary sources of saturated fats in animal-based foods. In in-vivo investigations, diets high in saturated fats have been shown to promote chronic inflammation in animal models [102], but the mechanisms remain fully elucidated. One explanation is that the amino acid taurine, found in saturated fats, and connected to bile acids, appears to improve substrate availability for sulfur-reducing bacteria such as *Bilophila Wadsworthia*, which is particularly abundant in the dysbiotic microbiota of patients with IBD. Furthermore, Muhomah et al [103] found that saturated fats can lower the secretory immunoglobulin A (sIgA) levels, altering immune responses to gut microbiota [88]. In addition to the initial risk of IBD, a diet rich in saturated fatty acids appears to enhance the likelihood of relapse, especially in UC [91]. In contrast, Olive oil contains Omega-3 fats which are effective antioxidants and are associated with a lower risk of IBD in both UC [104, 105], and CD; for instance, children who consume a diet high in omega-3 fats are at lesser risk of developing CD [106].

3.4 Oils

3.4.1 Fish Oil

Fish oil, rich in omega-3 fatty acids, holds promise for ameliorating IBD, particularly UC, by reducing inflammation, oxidative stress, and corticosteroid dependency [107, 108]. Studies highlight its ability to improve histological outcomes and anti-inflammatory properties, with a notable reduction in baseline levels of CRP (C-reactive protein), a sign of inflammation [109, 110]. In a 12-month randomized control trial, it was demonstrated that supplementation with fish oil resulted in a modest reduction in corticosteroid usage in patients with UC, yet failed to be beneficial for remission patients [111]. However, contrary to anticipated outcomes, Woodworth, Hillary L et al [112] reported that dietary fish oil intake exacerbated colitis in a murine IBD model, highlighting its effects' context-dependent nature and underscoring the need for cautious interpretation. Compared to CD, tissues obtained from UC patients after an administration of a fish oil-enriched

diet had led to a more pronounced increase in the ratio of anti-inflammatory cytokines, indicating a differential effect of fish oil on various forms of IBD [113]. Additionally, the diet's higher omega-3 to omega-6 ratio is linked to protective effects against UC, underscoring the dietary balance's role in IBD pathogenesis [114].

Fish oil also contains docosahexaenoic acid (DHA), eicosatetraenoic acid (EPA), and long-chain n-3 polyunsaturated fatty acids (PUFAs) all of which have anti-inflammatory effects in several chronic inflammatory illnesses including IBD [115]. These compounds regulate inflammatory and immune responses by altering cell membrane phospholipid composition, disrupting lipid rafts, inhibiting the pro-inflammatory transcription factor NF-κB, and activating the anti-inflammatory transcription factor peroxisome proliferator-activated receptor γ (PPAR-γ) [96]. Animal studies have shown that DHA and EPA provide benefits across various models of inflammatory conditions [116]. Although therapeutic trials suggest the biological plausibility of fish oil benefits in IBD, systematic reviews reveal conflicting evidence about the relationship between fish and IBD risk [63]. Three of the four investigations link higher fish consumption with an increased risk of UC, but no clear association has been found with decreased clinical relapse [117]. Similarly, no significant correlations have been observed between fish consumption and IBD relapse [118]. However, increased intake of animal proteins, including fish, has been associated with IBD incidence in some studies (Ptrend = 0.05) [76]. IBD-AID also allows fish consumption [119].

3.4.2 Olive Oil

Olive oil, particularly its extra virgin variant, is a valuable component in IBD management due to its high content of mono-saturated fats and potent antioxidants [120]. Its anti-inflammatory properties, attributed to compounds like oleic acid and polyphenols, provide relief from the persistent inflammation inherent in IBD such as EVOO-PE (a concentrated form of polyphenols extracted from extra virgin olive oil) is considered a potential beneficial food for managing UC due to its anti-inflammatory protective effects [121]. In one study, an extra virgin olive oil-based dietary regimen exhibited protective and prophylactic effects against colorectal cancer associated with UC and reduced the incidence and multiplicity of tumors [122]. Similarly, its consumption serves both as a primary and secondary preventive measure against gut microbiota equilibrium, and its role in mitigating weight loss, rectal bleeding, and inflammatory markers in murine models of IBD [123, 124]. Biophenol derived from olive trees also offers therapeutic applications in IBD due to its antioxidative and anti-inflammatory properties [125].

3.4.3 Canola Oil

Canola oil, derived from crude sources, offerings a range of characteristics suggesting potential benefits for individuals struggling with the complexities of IBD, due to the presence of canola, a compound that effectively alleviates IBD symptoms and lowers the risk of colon cancer by suppressing oxidative stress and inflammatory cytokines [126]. Its ingestion improves serum lipids, liver enzymes, and basal inflammation in subcutaneous adipose tissue, however, it elicits acute pro-inflammatory response following meal intake, unlike olive oil, particularly affecting obese individuals [127]. Despite initial expectations, canola oil did not demonstrate significant efficacy in ameliorating inflammatory indicators and gastrointestinal manifestations in patients with UC, as evidenced by a study comparing its effects with extra virgin olive oil [128]. Compared to corn oil,

canola oil exhibits anti-inflammatory properties, suggesting its potential therapeutic value in conditions exacerbated by inflammation, as indicated by its effects on Jurkat cells [129]. Similarly, in another study [130] on assessing the influence of canola oil on adiponectin concentration, among polyunsaturated fatty acids sourced from plants, Docosahexaenoic acid (DHA)-enriched canola oil distinguished itself for anti-inflammatory properties.

3.4.4 Palm Oil

The role of palm oil in IBD remains complex yet not fully established. Studies reveal that high dietary intake of palm oil negatively impacts growth and anti-oxidant capacity while promoting inflammation in juvenile large yellow croaker through activation of the TLR-NF signaling pathway [131]. Also, it has been reported that consumption of a palm oil-based diet enhances the delivery of lipopolysaccharides (LPS) to tissues by upregulating lipopolysaccharides-binding protein (LBP) levels and downregulating soluble CD14 (sCD14) levels, thereby intensifying inflammatory responses [132]. Moreover, brief exposure to palm oil or its constituent palmitic acid disrupts intestinal homeostasis by compromising barrier integrity, and provoking inflammation, which may contribute to gastrointestinal complications observed in metabolic disorders [133]. Conversely, the tocotrienol-rich fraction of palm oil exhibits significant anti-inflammatory properties, suggesting potential benefits for individuals with IBD [134].

3.4.5 Soybean Oil

Soybean-derived compounds may have some anti-inflammatory properties and potentially benefit individuals with IBD by attenuating colonic inflammation, as isoflavones found in soybeans, play a role in regulating innate immunity and may offer protection against tissue damage in IBD by inhibiting experimental colitis [135]. Both soybean meal and soybean oil have the capacity to impact gut microbiota and innate immune responses [136], though soy oil may exacerbate IBD-related joint pain in some cases [137]. Bioactive constituents of soybean, such as phytosterols, protease inhibitors, and bioactive peptides, improve the prognosis of IBD [138]. Additionally, germinated soybean extract suppresses matrix metalloproteinases and inflammatory mediators, while Soybean Bowman-Birk inhibitors (BBI) support gastrointestinal health by targeting the serine proteases [139].

3.4.6 Coconut Oil

Coconut oils present a potential therapeutic intervention in IBD owing to their anti-inflammatory properties. Incorporating virgin coconut oil into the diet of mice subjected to a highly high-refined carbohydrate diet yielded favorable outcomes by reducing inflammation [140]. Still, in healthy mice, even small amounts of coconut oil supplementation triggered inflammation and activated pro-inflammatory pathways [141]. Virgin coconut oil possesses properties that can reduce inflammation, relieve pain, and lower fever [142]. Additionally, the saturated fat content in coconut oil, predominantly myristic and lauric fatty acids, may exert anti-inflammatory effects and shield gut health through pathways not conventionally anticipated [143].

3.5 Beverages

3.5.1 Sweet Beverages and Soft Drinks

Sugars are common components in food and drinks. In Western countries, many Sugar-sweetened products, such as chocolate, cookies, cakes, and beverages including energy drinks, soft drinks, fruit punches, lemonade, and iced tea are widely consumed [144]. A trend now spreading globally [145]. Most dietary sugars contain glucose and fructose, naturally present in fruits and vegetables [146, 147]. High Fructose Corn Syrup (HFCS) is the predominant form of sweetened used in baked products and beverages, typically comprising 42% fructose in baked goods and 55% fructose in beverages, with the glucose making up the remaining syrup [148].

The function of excessive sugar in the development of IBD is still debated. In the aforementioned study [70], a substantial positive correlation was observed between dietary sugar consumption and the risk of UC, CD, and total IBD. However, no important associations were found between SSB consumption and the chance of UC, CD, or total IBD. Michael Laffin et al [149] in their investigation on mice concluded that short-term contact to a high-sugar diet increases vulnerability to colitis. Furthermore, population studies indicate that approximately 10% of individuals with IBD perceive sweet foods as triggers for symptoms, potentially due to promoting the growth of pathogenic bacteria while suppressing beneficial microbes, leading to dysbiosis that may exacerbate IBD in susceptible patients [150]. In several prospective investigations, the intake of HFCS and sugar-sweetened beverages (SSB) has been observed to be positively correlated with an increased risk of developing IBD [151-153]. Overall, the researchers believe that sugar is strongly associated with the composition of the gut microbiota and the onset and progression of IBD [150].

Eight studies have examined the association of soft drinks with CD risks, with one specifically focusing on subtypes of cola drinks. The findings indicate that the high consumption of soft drinks may elevate the incidence of CD [154]. Similarly, another meta-analysis of five investigations evaluated the correlation between soft drink consumption and UC incidence [155]. The association of soft drinks with IBD is also controversial, with findings ranging from no associations [156] to possible associations [154, 155] and even inconclusive associations [151]. Carbonated beverages contain carbon dioxide, which can lead to stomach distension and may irritate the GIT, potentially worsening symptoms like bloating and discomfort [157]. For patients with IBD, this irritation may further aggravate gastrointestinal distress. One study found an association between the consumption of SSB, rather than artificially sweetened beverages or natural juices, and IBD risk [158]. Taken together, soft drinks and SSB are reported to have many detrimental effects on the human body; therefore, avoiding them is a better option [51].

3.5.2 Alcohol

With a growing prevalence of alcohol consumption [159] and a rising incidence of IBD worldwide [160]. The relationship between alcohol and IBD has received considerable attention [161]. Alcohol consumption may influence IBD pathogenesis and disease progression through several mechanisms, such as alterations in microbiome composition and dysbiosis [162]. Alcohol disrupts the intestinal barrier in both the colon and small intestine by depleting anti-inflammatory bacteria, leading to intestinal damage and increased permeability [163]. This increased permeability exposes the submucosal immune system to luminal bacteria, often pro-inflammatory strains, and bacterial products like endotoxins. This exposure can lead to transient endotoxemia, which activates proinflammatory mediators, including tumor necrosis factors-α (TNF-α) and interleukin-1 and 6 [164], however the connection between alcohol and IBD is still debated [165], but the consensus

studies believe that alcohol intake is a probable cause for flare in IBD and worsening symptoms [161, 165, 166] including abdominal pain, reflux and diarrhea [167].

3.5.3 Coffee

Coffee intake influences the gut microbiota (composition, diversity, and growth), potentially through caffeine and other bioactive compounds like micronutrients and phenolic compounds [168]. For instance, caffeine has been shown to partially reverse amoxicillin-induced dysbiosis in the murine gut, an effect not observed with decaffeinated coffee, suggesting a potential role for caffeine [169]. However, other studies report no significant differences between caffeinated and decaffeinated coffee on gut microbiota, indicating that additional components in coffee may also play an essential role in modulating gut microbial composition [170, 171].

Although patients with IBD commonly consume coffee, its safety remains debated. Several studies have reported that CD and UC patients typically drink less coffee than controls [172], with some suggesting coffee exacerbates symptoms [173] or contributes to IBD onset [174]. Conversely, a Turkish study found no link between Coffee and IBD [175]. While a study focused on UC indicated that coffee consumption might be beneficial (with adjusted odds ratio = 0.51; 95% CI = 0.51–0.98) [176]. A Mendelian randomization analysis showed no evidence that genetically determined coffee consumption is causally related to the development of IBD [177]. Similarly, a meta-analysis of 16 epidemiological observational studies, including five focused on coffee consumption, found no significant correlation between coffee intake and CD risk (RR = 0.82; 95% IC = 0.46–1.46).

While coffee may offer protective effects during mucosal inflammation, its impact on the intestinal tract before disease onset may be influenced by many factors, leading to variable effects [154].

3.5.4 Tea

Tea, the second most consumed beverage globally, has received attention for its potential therapeutic benefits in IBD due to its anti-inflammatory and antioxidant properties. The key ingredients in tea such as polysaccharides, polyphenols, and pigments, are believed to alleviate inflammation, reduce oxidative stress and improve gut microbiome balance, making tea a potential nutritional intervention for IBD [178]. For instance, Polysaccharides extracted from tea flowers have been demonstrated to modulate the composition of the gut microbiome and increase the formation of short-chain fatty acid (SCFA), conferring a probiotic effect on IBD intestinal environments [179]. Catechins present in tea can also reduce excessive oxidative stress by activating antioxidative compounds like glutathione peroxidase (GPO) and glutathione (GSH), helping mitigate oxidative damage in the colon [180].

Green tea extracts have been shown to significantly alleviate diarrhea and weight loss in experimental colitis, improving colonic integrity and reducing inflammation [181]. Polyphenols in green tea have demonstrated anti-inflammatory effects in IL-2 (-/-) mice, indicating potential therapeutic benefits for chronic inflammatory diseases like IBD [182]. Another study found that green tea polyphenols mitigate intestinal inflammation and improve IBD symptoms by decreasing inflammatory cytokines and oxidative stress [183]. Additionally, dietary intake of green tea polyphenols modulates multiple anti-inflammatory molecular pathways in animal models of IBD

[184]. To summarize, supplementation with curcumin and green tea has been considered effective in reducing IBD symptoms and inflammatory indices [185].

The role of other types of tea in IBD, such as the primary polyphenol present in black tea, thearubigin, demonstrates advantageous properties in experimental colitis, suggesting its potential efficacy in managing IBD [186]. Likewise, Fuzhuan brick tea (FBT) has exhibited protective properties against colitis in mice, effectively reducing symptoms and expression of inflammatory cytokines [187]. Ripened Pu-erh tea (RPT) is shown to be beneficial in easing Dextran sulfate sodium (DSS)-induced colitis by inhibiting the HIF-1β/NF-κB signaling pathways, thereby decreasing inflammation [188].

3.6 Dairy and Dairy Products

Dairy products are significant sources of protein, riboflavin, and calcium, essential for maintaining bone health and especially crucial for individuals with IBD to prevent metabolic bone diseases. Despite their nutritional value, lactose in dairy can cause gastrointestinal discomfort in individuals with lactase deficiency. However, the prevalence of lactase deficiency is not higher in patients with IBD compared to healthy individuals [189]. Consequently, unless dairy exacerbates symptoms, patients with IBD are typically not advised to restrict dairy intake during flares. They may choose low-lactose options like yogurt or lactose-free alternatives [79]. Research suggests that dairy consumption is not significantly linked to the development of IBD (ptrend = 0.93) [76] or to experiencing disease relapse [117]. Some dietary protocols, such as a systematic review investigating dairy's role in UC development, reported odds ratios ranging from 0.79 to 2.67 (DI & RD in IBD 41). CDED excludes dairy products [92], while others, like the IBD-AID and Low FODMAP diet, allow controlled quantities [59]. While dairy provides essential nutrients; it could also contribute to the development of IBD by influencing gut microbiota and immune responses [190, 191]. Limited epidemiological studies have explored the relationship between dairy intake and CD or UC, yielding mixed findings [116, 191]. Clinical and molecular studies suggest that dairy nutrients may have anti-inflammatory effects and influence cytokines such as TNF-α involved in IBD pathogenesis [192].

3.6.1 Milk

Milk-derived saturated fats can elevate inflammatory markers by modulating cytokine gene expression [193]. Conversely, milk products may alter gut microbiome composition by increasing beneficial lactic acid bacteria, which may help reduce intestinal inflammation [194]. Studies have found that sheep and goat milk are less likely to exacerbate symptoms in patients with CD compared to cow's milk [195]. Soy milk, although not a dairy product, generally demonstrates greater tolerance than milk-based items [196].

A study on nutritional strategies involving 16 patients with CD in remission identified cow's milk as the most common trigger for nutritional symptoms [197]. Furthermore, an ecological study in Japan revealed a notable positive correlation between the incidence of CD and milk protein consumption [198]. Some studies suggest a link between cow’s milk consumption and IBD onset, with patients exhibiting greater serum antibody levels against cow's milk protein than healthy controls [199]. Additionally, breastfeeding has been explored as a protective factor against IBD, due to its protective mechanisms such as gastrointestinal infection prevention and immunological

stimulation [200]. Clinical improvement was observed in some patients after milk was eliminated from their diet, with symptoms recurring upon reintroduction, leading researchers to hypothesize that a milk protein allergy might be involved in UC etiology [201].

3.6.2 Yogurt and Kefir

Research in animal and human models has examined the impact of fermented milk (e.g. yogurt, kefir) on cytokine production, T-cell function, phagocytic activity, antibody production, and natural killer (NK) cell activity [202]. Fermentation enhances microbial viability and productivity while preserving probiotic properties. Some microorganisms like *Bifidobacterium longum* integrate into the human intestinal microbiota, while others, like *Lactobacillus casei*, exert transient effects as they pass through the gut, modulating the microbial community [203]. Lorea Baroja et al [204] found that probiotic yogurt had anti-inflammatory effects in a study involving 20 healthy individuals and 20 with IBD (15 with CD and 5 with UC) increasing CD4 + CD25 high T-cells in patients with IBD. Fermented milk products like yogurt and cheese showed anti-inflammatory properties in a mouse model of colitis, suggesting potential benefits for patients with IBD [205].

Milk kefir, a fermented form of dairy similar to yogurt, showed promising results in patients with IBD. In a study of 25 patients (10 with CD and 15 with UC), consuming 400 mL of kefir daily for 4 weeks significantly reduced inflammatory markers such as C-reactive protein and bloating scores while improving hemoglobin levels (Hb) in patients with CD [206], and increasing *Lactobacillus* levels in UC patients [207]. A double-blind randomized controlled trial involving 82 individuals with *Helicobacter pylori* (H Pylori) receiving antibiotics treatment revealed that 78% of patients treated with kefir had *H pylori* eliminated, with lower occurrences of nausea, diarrhea, and abdominal pain [208].

3.6.3 Emmental and Mozzarella Cheese

The fermentation of Emmental cheese by *Propionibacterium freudenreichii*, and another cheese made from a single strain of this bacteria increased the histological scores and DSS-induced colitis while decreasing the disease activity index and weight loss, decreasing the IgA secretion in the small bowel and enhanced the expression of occludin genes, which prevented the activation of interferon-gamma (IFN-γ), TNF-α, and IL-17 [209]. Mozzarella cheese is made from buffalo milk, which contains a variety of bioactive peptides that are largely produced during milk digestion. A peptide from buffalo milk, found in Buffalo Mozzarella Cheese, has been shown to reduce H_2O_2-induced oxidative stress in intestinal epithelial and erythrocyte cells [210]. In studies conducted on human intestinal Caco2 cell line (human colon carcinoma) that were inflamed as well as in vivo on colitis-DNBS mice, non-toxic concentrations of this peptide (MBCP) improved adherent epithelial junction organization, controlled the NF-κB pathway, and decreased intestinal permeability [211]. In the DNBS-induced colitis model, oral treatment of this peptide reduced intestinal inflammation. Therefore, eating buffalo mozzarella cheese may have advantages [212].

3.6.4 Butter

Milk fat is used to make butter. Although no specific human studies have examined butter in relation to IBD, dairy and milk consumption are often reported to aggravate symptoms in patients

with IBD. However, epidemiological studies show inconsistent findings on this relationship [213]. However, butter consists of butyrate, a fatty acid generated by gut bacteria, is found in butter and has anti-inflammatory properties because it feeds colonocytes and increases NF-κB [214]. According to some, butter may help patients with IBD. However, a recent systematic analysis showed that in individuals with IBD, sodium butyrate enemas have a minimal impact on inflammatory and histological parameters [215]. Studies on animals show that diets high in saturated milk fat in IL10-/-mice increased *Bacteroidetes* and decreased *Firmicutes* [102].

3.7 Vegetables

Vegetables contain dietary fiber, minerals, phenolic compounds, trace elements, and antioxidant vitamins like A, C, and E [216]. The anti-inflammatory attributes of these phenolic constituents offer a natural approach to preventing IBD. Polymers of fructose known as oligofructose and inulin, present in some vegetables including onions, artichokes, and asparagus, are documented to support the growth of beneficial gut bacteria like *Bifidobacteria* and *Faecalibacterium prausnitzii* in individuals with healthy intestinal flora [31]. One key characteristic of inulin is its resistance to breakdown by digestive enzymes in the small intestine, which are specific to β-glycosidic bonds, classifying it as a "non-digestible" oligosaccharide, which instead is fermented by gut microbiota in the colon, producing SCFAs such as acetate, butyrate, and propionate. These SCFAs provide colonocyte energy sources and confer diverse health benefits to the host [217]. Patients who consumed more vegetables showed a reduced likelihood of developing a risk of CD and UC, respectively [218]. A number of studies support these facts for both CD and UC. Also, no disease relapse was found in patients with IBD associated with high vegetable intake [219-221]. In the following sections, we will review some vegetables used daily and highlight their role in IBD.

3.7.1 Specific Vegetables

Potatoes and their derivatives and other nutrient-rich vegetables, play a significant yet nuanced role in managing inflammatory bowel diseases (IBD). Glycoalkaloids such as solanine and chaconine, found in potato skins, have been shown to destabilize the epithelial lining by permeabilizing cholesterol-containing membranes and enhance intestinal permeability dose-dependently in mouse models [222]. A study on adult patients with IBD found that a diet rich in potatoes and legumes was associated with a reduced risk of disease relapse, with patients in the highest consumption quartile having a 79% lower likelihood of active disease [117]. Additionally, potatoes contain higher concentrations of resistant starch, exhibiting prebiotic effects after cooking and cooling [223]. Therefore, the CDED recommends consuming two fresh potatoes daily, which must be peeled, cooked, and cooled before eating [57]. In contrast, potatoes are not allowed on the SCD diet [224]. Ipomoea batatas L. (Sweet potato) a member of the Convolvulaceae family, is ranked as the 7th most significant food crop globally [225]. It is rich in starch, fiber, minerals, vitamins, and other nutrients. It is prized for its high anthocyanin content, which contributes to its growing popularity among consumers due to its health-promoting properties [226], including anti-inflammatory properties, among others [227].

Similarly, tomatoes also play a complex role, as patients with CD often exhibit heightened immune responses [228]. Nonetheless, the CDED incorporates two tomatoes daily, alongside other vegetables like cucumbers, spinach, carrots, lettuce, and zucchini in its early phases, while

restricting certain vegetables like kale, leeks, and asparagus [57]. Brassicaceous vegetables such as broccoli, Brussels sprouts, and cauliflower, although known to cause abdominal distension in some individuals, are rich in indole-3-carbinol. This compound activates the aryl hydrocarbon receptor (AHR) to reduce inflammation and restore gut integrity [229]. Studies in model organisms have shown that consuming broccoli increases intestines AHR activity, which reduces the abundance of *Erysipelotrichaceae* microbes and mitigates colon inflammation [230]. Additionally, human-cultured Caco-2 cell transepithelial electrical resistance increases significantly with 3,3-diindolylmethane (DIM), which is also derived from brassicaceous crops, thus restoring gut permeability [231].

3.7.2 Legumes

In an aforementioned cross-sectional study [117], the consumption of legumes showed protective effects in 103 adult patients with IBD (53 in remission and 50 with active disease), indicating an inverse relationship between the consumption of potatoes and legumes and disease relapse. Compared to patients in the lowest quartile, those in the highest quartile consumed more potatoes and legumes and had a 79% lower risk of developing an active illness. Although Legumes may worsen symptoms, they are allowed in the second phase of the CDED diet, an exclusion diet for patients with CD who have flare-ups [57]. On the SCD, legumes such as split peas and lentils are allowed, but others like chickpeas and soybeans are not allowed [223]. In a study, the intake of red kidney beans, which are high in resistant starch and dietary fiber, significantly influenced the intestinal microbiota and fermentation in the cecum of rats [232].

3.7.3 Corn

Despite being tolerated by only a minority, consumption of corn as a vegetable was linked to adverse impact in 45 percent of CD patients [200]. Similar negative outcomes were observed for maize-based products, such as cornflakes, popcorn, and corn crackers [200]. These findings align with earlier studies, and corn has been identified as a significant dietary item that certain people with CD may avoid [233]. This may be attributed to the makeup of fructans or the physical characteristics of the testa, the outer layer of the seed. Additionally, the unique polyphenol content of maize may contribute to the negative effects observed in these investigations [234].

3.7.4 Mushrooms

For centuries, people have utilized mushrooms as a form of medicine to reduce inflammation, and in individuals with IBD, a combined extract of basidiomycetes mushrooms has been shown to reduce inflammatory symptoms [235]. By regulating cytokine profiles and enhancing phagocyte activity, mushroom glucan improves the condition of IBD and protects against infections, and sepsis inflammation [236].

3.8 Fruits

Fresh fruits, rich in vitamin C, fiber, and other inhibitors such as phenolic acids, are absorbed by the gut wall and may exhibit anti-inflammatory effects [237]. Among phenolic acids, fruits such as apple berries, blueberries, citrus fruits, mangos, kiwis, and plump blueberries, citrus fruits, mangos, kiwis, and plums have the highest concentration of chlorogenic acid [238]. Phenolic acid containing

raspberry seed flour, also lessens inflammation of adipose tissue and hepatic stress induced by a high-sucrose food [239]. Citrus fruit consumption exhibited a negative association with CD development [84], while grapefruit consumption often worsens symptoms, leading to significant distress among patients with IBD [240]. Moreover, in a preliminary trial involving patients with IBD, eating 200-400 g of pulp from mangos (Magnifera indica L) over eight weeks showed reduced levels of pro-inflammatory cytokines such as IL-8 and enhancements in the Simple Clinical Colitis Activity Index (SCCAI) score [84]. Mango, which contains gall tannin, improves the patient's fecal microbial composition. Mango improved symptoms in mice with colitis caused by DSS by decreasing NF-kB and MAPK signaling [241]. Bananas are rich in inulin, a well-known prebiotic, while green bananas have considerable levels of type 2 granular-resistant starch [242]. Resistant starch, like high-amylose starch, serves as a prebiotic component [243], as it resists digestion and can be digested by colonic bacteria into SCFA [244]. One apple and two bananas per day are required for the CDED diet, while strawberries, melons, avocado, peach, pear, kiwi, and blueberries are permitted. All fruits are allowed after ten weeks, except for dried fruits [84].

3.9 Cereals

3.9.1 Wheat

Whole-grain wheat and wheat bran may confer anti-inflammatory benefits and promote gut health in specific contexts, while other wheat components such as amylase/trypsin inhibitors (ATIs) and gliadin can exacerbate inflammation by promoting intestinal dysbiosis and triggering the innate immune response via toll-like receptor 4 (TLR4), which may have implications for human IBD [245]. Dietary fiber from wheat, such as wheat bran, may enhance bowel function and improve quality of life in patients with CD without adverse effects [246]. Anti-inflammatory effects of whole-grain wheat consumption are evidenced by decreased plasma TNF-α and elevated levels of interleukin-10 (IL-10), suggesting potential benefits for individuals with inflammatory conditions [247, 248]. Wheat germ supplementation supports an anti-inflammatory gut environment, increases beneficial *Lactobacillaceae*, and reduces circulating pro-inflammatory cytokines in mice [249]. Furthermore, the particle size of wheat bran can influence its anti-inflammatory effects, with smaller particle sizes potentially offering more significant benefits in reducing inflammation associated with fructose over-consumption [250].

3.9.2 Rice

Rice and its fermented products, particularly rice bran and protein peptides, have demonstrated potential therapeutic effects in IBD. These effects include alleviating colitis symptoms, modification of the intestinal microbiome, reduction of inflammation, and protection against extra intestinal manifestations [251]. Fermented rice bran (FRB) supplementation has been shown to attenuate colitis and inflammation in muscle and lower proinflammatory cytokines. It may prevent liver disorders and neuroinflammation in IBD, suggesting a role in extra-intestinal manifestation prevention [252]. Rice-based meals are well tolerated by patients with functional gastrointestinal disorders (FGID), producing little intestinal gas and having low allergenicity, which may improve gastrointestinal symptoms [253]. Proanthocyanidin-rich red rice extract (PRRE) has been shown to protect against colitis, reducing the severity of symptoms and inflammatory cytokine production,

and suggesting potential as a natural treatment for IBD [254]. Enzyme-treated rice fiber (ERF) and other rice bran-derived prebiotics have demonstrated anti-inflammatory effects in murine colitis models by modulating intestinal homeostasis and the mucosal immune system [255]. Moreover, fermented rice bran by specific strains of bacteria and yeast shows antioxidant and anti-IBD properties, with protective effects in DSS-induced IBD model mice [256].

3.9.3 Maize

Maize plays a multifaceted role in both agriculture and human health due to its genetic variability and the presence of beneficial compounds such as flavan-4-ols contribute to its potential to alleviate symptoms of IBD and promote gut health as evidenced by flavan-4-ols maize-enriched diet alleviating colonic inflammation by reinstating gut barrier function and modulating intestinal microbiota in mouse models of IBD [257]. Maize, a staple crop rich in starch, protein, and fat, has been extensively studied for nutritional enhancement through biofortification. Such improvements could have potential health benefits, including implications for managing IBD [258].

3.9.4 Barley

Barley has been studied for its potential health benefits, particularly in the context of IBD and related gastrointestinal disorders. Barley and its components, including fermented products, dietary fibers, and functional ingredients, might improve intestinal health and the management of IBD [259]. Dietary barley leaf supplementation can attenuate colitis by activating Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) signaling and modulating gut microbiota-derived metabolites, suggesting a protective role against IBD [260]. Whole grain barley and its soluble dietary fibers, such as β-glucans, have been associated with modulating gut microbiota and improving intestinal functions, which could be beneficial in managing IBD [261]. Barley grass contains functional ingredients with preventive and therapeutic roles for chronic diseases, including IBD, by enhancing immunity and improving gastrointestinal function [262]. Barley intake has been shown to induce bile acid excretion and reduce the expression of intestinal Apical Sodium-dependent Bile Acid Transporter (ASBT) and Niemann-Pick C1-Like 1 (NPC1L1) in mice, which may contribute to its hypocholesterolemic effects and potentially benefit patients with IBD [263]. A fermented mixture of barley and soybean enhances intestinal barrier function [264].

3.9.5 Oats

Potential benefits of oats for IBD management include anti-inflammatory effects, modulation of gut microbiota, and improvement of gut health, with animal studies showing promising results that oat β-glucan demonstrates protective effects against colitis in mice by reducing clinical symptoms, inflammation, and pro-inflammatory markers [265]. Still, studies on IBD are limited, and no firm conclusions can be drawn. Some studies suggest that eating oats or oat bran regularly may help to treat IBD, but more evidence is needed to confirm this [266]. Non-starch polysaccharides, including those from oats, may have potential as adjuvant therapy for IBD due to anti-inflammatory and gut microbiota-modulating activities [237]. Oat β-glucan can modulate the gut microbiota, influencing bile acid metabolism and potentially affecting cholesterol metabolism, which may have implications for IBD [267]. Whole grain oats and barley, rich in β-glucans, may reduce the risk of chronic

conditions like IBD by modulating gut microbiota and reducing postprandial blood glucose [261]. Oat consumption is also associated with immunomodulation, which could benefit IBD management [268].

3.10 Nuts

Nuts contain essential nutrients, unsaturated fatty acids, protein, and fiber, as well as vitamins (folic acids, niacin, pyridoxine, tocopherol), minerals (copper, magnesium, and potassium), and phytochemicals [269]. Pistachios, pecans, and walnuts are incredibly high in phenolic compounds, such as anthocyanins, flavanones, flavonoids, flavonols, isoflavones, proanthocyanidins, stilbenes, phenolic acids, and hydrolyzable tannins, all of which act as antioxidants [270]. Nut consumption has advantages in IBD, as well as reducing mortality rates. Interestingly, in patients with IBD, nuts and seed intake was inversely associated with BMI, indicating a potential role in weight management for these patients [271].

3.10.1 Pistachio

Pistachios have a lower fat content than other nuts, primarily composed of PUFA and monounsaturated fatty acids (MUFA), and a higher fiber content, including both soluble and insoluble forms. They also have more significant quantities of vitamin K, phytosterols, gamma-tocopherol, xanthophyll carotenoids, and certain minerals [84]. The potent antioxidant and anti-inflammatory properties of pistachio are likely attributed to their abundant gamma-tocopherol content and the antioxidant effects of zinc (Zn) and selenium (Se) [272]. Moreover, unprocessed pistachios contain approximately thirteen times higher levels of lutein and zeaxanthin than hazelnuts, the next highest nut variety [273]. Lutein and zeaxanthin are two xanthophyll carotenoids that impart color to pistachio nuts and possess antioxidant properties. In a prospective study, 32 healthy young men followed MD for 4 weeks, which was subsequently enriched with pistachios for 4 weeks, replacing approximately 20% of the daily energy intake from monounsaturated fats [274]. The pistachio-enriched diet notably enhanced endothelium-dependent vasodilation and reduced serum levels of IL-6, total oxidant status, lipid hydroperoxide, and malondialdehyde while enhancing superoxide dismutase activity [275]. However, no significant alterations were observed in C-reactive protein and TNF-a levels. Overall, these findings support the anti-inflammatory effects of pistachios [84].

3.10.2 Cashew

Cashew nuts are among the top four recognized nuts in the world, valued for their unique taste and richness of nutrients [27]. Cashews can help lower the incidence of metabolic and cardiovascular diseases when eaten as part of a balanced diet [276]. Previous studies, along with diet impact, also looked at the effects of industrial by-products, such as cashew (Anacardium occidentale L.) fruit, on rats' intestinal health and cholesterol metabolism with diet-induced dyslipidemia [277]. Rats with dyslipidemia had improved conditions when fed cashew nuts [278], similarly, giving diabetic mice an oral ethanolic extract of cashew flowers regulated the inflammatory response system in a model of cecal ligation and puncture (CLP) [279]. Despite these findings, research on the biological impact of cashews in unhealthy individuals is limited. Based on

these findings, Impellizzeri et al [280] worked on a mouse model of colon inflammation induced by intrarectal injection of DNBS to examine the anti-inflammatory and antioxidant properties of orally administered cashew nuts. This model supports human CD-like features, including NF-kB dependent Th1 activation.

3.10.3 Walnuts

In a randomized crossover study involving 18 healthy participants, consuming 42 g of walnuts increased the relative abundance of *Faecalibacterium*, *Clostridium, Dialister*, and *Roseburia* by 49-160% but decreased the relative abundance of *Ruminococcus, Dorea, Oscillospira,* and *Bifidobacterium* by 16-38% ($p < 0.05$) when compared to the control period [281]. Additionally, it reduced LDL cholesterol levels and proinflammatory secondary bile acids produced by microbes. In another randomized crossover trial involving 194 healthy individuals, consuming 43 g of walnuts for eight weeks significantly increased the abundance of *Bifidobacteria* and *Ruminococcaceae*, while species from the *Clostridium sp. cluster XIVa* (*Blautia; Anaerostipes*) showed a significant decrease ($p < 0.05$) [282]. Therefore, eating walnuts greatly enhanced the amount of prebiotic and butyric acid-producing species in healthy individuals' gut microbiota. In a study with rats, feeding them a diet containing 6 or 9% walnuts notably inhibited the activation of P38-MAPK and NF-kB in brain tissues, which are proinflammatory molecules [283].

3.10.4 Almonds

Participants who consumed 42 g of almonds daily had a higher relative abundance of *Clostridium clusters IV and XIVa*, which include *Clostridium, Lachnospira,* and *Roseburia* in a randomized crossover study in comparison to the control diet period without almonds [284]. Furthermore, comparisons between various almond forms (whole, whole roasted, roasted chopped, and almond butter) and controls showed that ingestion of roasted chopped almonds raised the relative abundance of *Lachnospira*, *Oscillospira*, and *Roseburia*. The relative abundance of *Lachnospira* was similarly raised by whole roasted almonds. However, compared to the control group, consumption of almond butter did not alter the microbiome [84].

3.11 Probiotics and Prebiotics

Probiotics consist of live organisms that reach the intestine in adequate amounts, remain active and offer health benefits [285]. They comprise lactic acid-producing yeasts and bacteria that may enter the intestines without harming the host. The combined action of probiotics and prebiotics is called "symbiotic" [286, 287]. The mechanism of action remains unclear; however, they likely regulate the permeability of the membrane and immune system of the mucosal tract, thereby preventing infectious agents from adhering to the intestinal lining. Probiotics influence cell signaling pathways that regulate TNF-α expression and may act at various points within the MAPK pathway, including NF-κB, like toll-like receptors, protease activity, and their regulators and stimuli [288]. Angélica Vincenzi et al [289] concluded in their review paper that probiotics operate through multiple mechanisms, mainly by inhibiting lκB phosphorylation and degradation, which prevents the translocation of NF-κB. Beyond their immune-modulating effects, probiotics offer additional health benefits to the host. The indigenous microbiota performs various biological activities, from anabolic

to catabolic processes, yielding advantages for both the host and the microbiota [290]. For instance, intestinal microflora can ferment endogenous mucus and indigestible dietary residues and produce essential vitamins like B12 and K [291]. Additionally, *Lactobacillus* and *Bifidobacteria* form substances damaging to both Gram-positive as well as Gram-negative bacteria, competing with harmful microorganisms like *Clostridium*, *Staphylococcus*, *Bacteroidetes*, and *Enterobacter* for cell adherence [292, 293].

Alternatively, prebiotics are ingredients that undergo selective fermentation, leading to particular changes in the composition and/or activity of gut bacteria, thereby promoting the host's well-being and health [285]. These are nondigestible oligosaccharides, including galacto-oligosaccharides (GOS), fructo-oligosaccharides (FOS), insulin, and lactulose; they can promote the growth of beneficial and selective intestinal flora [294]. Due to their makeup, these substances are not absorbed until they enter the intestinal tract, where certain bacteria can break them down into lactate and SCFA [295]. SCFAs possess immunomodulatory properties, influencing Toll-like receptor-4 signaling and the production of pro-inflammatory cytokines [296]. Fructose, lactulose, and GOS are particularly effective in promoting the growth of *Lactobacilli* and *Bifidobacteria*, with GOS and lactulose showing greater efficacy than inulin [297]. Kanner et al. [298] demonstrated that gastric acid secretion could promote the oxidation of lipids and other food components. Their research suggests that dietary antioxidants, including inulin, may help prevent lipid peroxidation in the stomach. Overall, dietary supplementation with inulin or oligofructose appears to protect against oxidative stress, potentially reducing inflammatory reactions linked to oxidative stress [299].

After pouch surgery, the multi-strain probiotic (VSL No. 3) appears to help maintain remission [300] and may aid in preventing pouchitis in individuals with this condition [301]. The single-strain probiotic *Escherichia coli Nissle 1917* is just as effective for maintaining UC as amino-salicylates [77]. While prebiotics are commonly studied alongside probiotics, this makes it difficult to isolate their individual effects [302]. Insufficient data supports using prebiotics or probiotics to induce or sustain illness remission in UC and CD [303, 304].

4. Nutritional Suggestions in Outpatient Management

The occurrence of malnutrition in IBD has been reported between 20% and 85%, with hospitalized CD patients being at greater prevalence [18, 53] for both micro and macronutrient deficiencies. The nutritional investigation is a critical step in the clinical examination [305]. Even in patients with normal BMI, lean mass depletion or sarcopenia should be treated as separate conditions and seen as a different feature of malnutrition. Several screening tools are developed for malnutrition diagnosis based on phenotypic (age, weight loss, BMI) and etiologic (disease activity, food intake) criteria [306]. For these patients to be properly managed, mainly those suffering from short bowel syndrome and intestinal failure, a multidisciplinary approach and close coordination with expert dieticians are important [307]. About 15-40% of adult patients with IBD are obese, and the other 20-40% are overweight, equally occurring in CD and UC patients. Evidence indicates that obesity might lower the therapeutic effect of drugs by accelerating the elimination of drugs and increasing perioperative complications [308]. Obesity and sarcopenia in patients with IBD can lead to sarcopenic obesity which adversely affects the patient's condition raising disability, and mobility [309].

4.1 Micronutrients

Recent studies, such as Massironi et al [310], emphasize the detrimental effects of chronic inflammation on nutrient absorption in patients with IBD. Inflammation compromises the efficiency of the intestinal mucosa, reducing the absorption of essential nutrients, including vitamin B12, iron, and vitamin D. These deficiencies contribute to malnutrition and worsen the disease's clinical symptoms and progression [311]. To address this further, the following subsections explore the role of various micronutrients in IBD. Table 2 summarizes the key micronutrients discussed in this paper, highlighting their deficiencies, dietary sources, and supplementation strategies relevant to IBD management.

Table 2 Summary of micronutrient deficiency impacts, dietary sources, and treatment recommendations in IBD.

Micronutrient	Dietary Sources	Role in IBD	Causes of Deficiency	Deficiency Symptoms	Treatment Recommendations	Ref
Iron	Fish, liver, beef, eggs, legumes, poultry	Essential for oxygen transport and immune function	Malabsorption in CD, active UC (bloody diarrhea), GIT loss	IDA, fatigue	Prefer IV administration; but new oral formulations are also effective	[312-319]
Folate	Leafy greens, fortified cereals, beans	DNA synthesis, immune support	Blood loss, malabsorption, medications (salazopyrine, methotrexate)	Megaloblastic anemia, hyperhomocysteinemia (risk of thrombosis)	Check for anti-tissue transglutaminase antibodies to rule out celiac diseases	[320-323]
Zinc	Seafood, meat (liver), eggs	Maintains gut permeability, immune modulation, wound healing	Diarrhea, fluctuations in dietary intake	Immune dysfunction, poor wound healing	40-110 mg three times daily for 8 weeks, followed by reassessment	[324-329]
Vitamin B12	Liver, beef, fish, shellfish, dairy, poultry	RBC production, nerve health	Mucosal inflammation, terminal ileum involvement in CD, inadequate intake	Megaloblastic anemia, peripheral neuropathy	5000 µg IM cyanocobalamin or sublingual formulations	[330-333]
Vitamin D	Sunlight exposure, Salmon, Dairy, eggs, liver,	Bone health, immune modulation	Malabsorption in severe cases of IBD, limited sub-exposure	Bone pain, Muscle weakness, Fatigue	cholecalciferol supplement (25000 to 50000 IU per month)	[334-337]
Vitamin E	Safflower oil, Sunflower oil,	Antioxidant immune support	CD-specific malabsorption, fat malabsorption	Neuromuscular issues in	No standard recommendations; consider in CD with	[338-341]

corn oil, margarine	severe deficiency	substantial fat malabsorption

CD: Crohn's disease, UC: Ulcerative colitis, IDA: Iron deficiency anemia, GIT: Gastrointestinal tract, IV: Intravenous, IM: Intramuscular, RBC: Red blood cell

4.1.1 Iron

Iron deficiency is a widespread problem in patients with IBD, affecting about 20% of them [312]. In CD, iron malabsorption is primarily attributed to impaired absorption in the GIT and chronic blood loss, while in active cases of UC (marked by bloody diarrhea), are the leading causes of iron deficiency. Severe but less clinically apparent anemia can also result from ulcers associated with colonic or ileal involvement in CD [313]. A thorough assessment of iron metabolism parameters, such as iron serum levels, transferrin, and ferritin, enables the differentiation of iron deficiency from anemia associated with chronic diseases or combined anemia, which are common occurrences [314]. Iron deficiency anemia is characterized by reduced serum levels of iron, transferrin saturation, and ferritin. In contrast, inflammatory anemia is characterized by ferroportin-1 inhibition due to elevated hepcidin levels, which causes iron sequestration in its deposits and leads to high ferritin and reduced transferrin serum levels [315]. Iron malabsorption in CD patients with extensive minor bowel inflammation or resection can contribute to anemia. Ferritin levels can misleadingly appear normal in combined anemia, showing intermediate values influenced by iron deficiency and inflammation [316]. However, other conditions such as hemolysis and renal failure might make anemia worse in patients with IBD. Iron is present in a variety of foods — fish, beef, liver, eggs, poultry, and legumes — but there is an ongoing discussion about the best way to administer medication for treating iron deficiency anemia, with most authors preferring intravenous administration due to a theoretical risk of IBD recurrence with oral iron supplementation [317]. However, recent studies have shown the safety and effectiveness of new oral iron formulations [318, 319].

4.1.2 Folate

Folate serves as a cofactor in DNA synthesis. It is the second most prevalent micronutrient deficiency in patients with IBD, affecting approximately 30% of those with CD and 10% of those with UC [320]. The main contributors are either malabsorption (particularly in cases of extensive CD with involvement of proximal small bowel malabsorption or short bowel syndrome) or inadequate consumption, which is insufficient to compensate for blood loss from the GIT [321]. Additional factors contributing to folate deficiency include medications prescribed for IBD, such as methotrexate or salazopyrine, which prevent folate absorption [322]. In individuals with IBD with folate deficiency, celiac disease should be eliminated by assessing serum anti-tissue transglutaminase antibodies [323].

4.1.3 Zinc

Zinc is an essential trace element and is vital in wound healing, immune system modulation, and the maintenance of optimal intestinal permeability [324]. It is estimated that 15% of patients with IBD experience zinc deficiency even though gastroenterologists do not typically assess its serum

levels, except for individuals with intestinal failure [325]. However, the accuracy of zinc measurement is uncertain due to fluctuations in intake [326, 327]. Given the association between low zinc levels and poor clinical outcomes in IBD, continuous monitoring and zinc replacement should be recommended in patients with IBD, particularly in those with diarrhea [328]. Zinc-rich foods include meat (especially liver), seafood, and eggs. Zinc supplementation requires 40-110 mg three times daily for 8 weeks orally and followed by reassessment [329].

4.1.4 Vitamin B12

Approximately 20% of patients suffer from vitamin B12 deficiency [330]; since vitamin B12 absorption primarily occurs in the terminal ilium, it is the most afflicted part in CD and hence is removed sometimes [331]. While the main causes of deficiency in these patients are mucosal inflammation and/or inadequate oral intake, in cases of severe or unexplained deficiency autoimmune gastritis should be ruled out by assessing anti-parietal cell antibodies. Vitamin B12 deficiency may also present clinically as peripheral neuropathy and megaloblastic anemia [332]. Animal goods like fish, tuna, shellfish, beef, liver, poultry, eggs, and dairy products are high in vitamin B12. To address this deficiency, 5000 µg of intramuscular cyanocobalamin injection is usually used, and more recently sublingual formulations have been made available [333].

4.1.5 Vitamin D

Dietary vitamin D intake from sources like dairy products (milk, yogurt), eggs, liver, cod liver oil, and salmon meets only a small portion of daily requirement. Consequently, vitamin D deficiency is common among patients with IBD, affecting 90% of malnourished individuals and 80% of those who appear well-nourished. A negative correlation has also been found between serum vitamin D levels and BMI [303, 304]. The primary source of vitamin D is skin synthesis triggered by ultraviolet B (UVB) radiation from sunlight. Hence, in young adults, 15 minutes of sun exposure, two to three times per week during summer, covering approximately 25% of the body surface (face and arms), can produce vitamin D levels comparable to an oral intake of 25 µg (1000 IU) [88]. Patients on steroids must receive an oral cholecalciferol supplement of at least 25,000 IU per month, while patients whose serum 25-OH vitamin D levels are below 20 ng/mL must take an oral cholecalciferol supplement of at least 25,000 to 50,000 IU per month [334]. If levels are below 10 ng/mL, a weekly dosage of 50,000 IU for 6-8 weeks, followed by a daily intake of 800 lU is recommended [335]. In case of severe malabsorption, injectable formulations may be considered, with a suggested dosage of 300,000 IU administered intramuscularly every six months [336, 337].

4.1.6 Vitamin E

Vitamin E encompasses a group of fat-soluble vitamins known for their antioxidant role [338]. Various forms of vitamin E exist, but the most frequent one in the North American diet is gamma-tocopherol, which can be found in corn, soybean, margarine, and dressings. The second most common and physiologically active form of vitamin E overall is Alpha-tocopherol which is abundant in sunflower and safflower oils [339]. Since vitamin E is absorbed similarly to other fat-soluble vitamins, it is possible that therapy with cholestyramine and/or fat malabsorption [340]. To our knowledge, three studies have examined vitamin E status in IBD across different cohorts: one

exclusively focusing on CD, another on UC [306], and one including both UC and CD patients [341]. Among these, only the study involving patients with CD notably identified lower serum vitamin E levels compared to controls, irrespective of disease activity. Due to the limited data on vitamin E deficiency in IBD, there are currently no recommendations for monitoring and supplementation. However, it might be considered in CD patients with substantial fat malabsorption [306].

4.2 Macronutrients

Macronutrients include carbohydrates, proteins, and fats. As discussed, the Western diet, rich in sugar, red meat, and saturated fats, yet low in fiber is widely considered pro-inflammatory [341] and might elevate the risk of IBD. Conversely, macronutrient insufficiency might manifest in individuals with active IBD who limit oral food consumption due to nausea/vomiting, abdominal pain, or fear of worsening symptoms, especially during flares, and in instances of structuring CD [342]. In severe cases of micronutrient deficiency, such as those seen as short bowel syndrome complicated by intestinal failure, collaboration with a skilled dietician is essential, and parenteral nutrition (PN) might be necessary [343].

4.2.1 Sarcopenia

The evaluation of sarcopenia in patients with active IBD was recently incorporated into clinical practice [344]. Sarcopenia, typically associated with aging is characterized by a decline in lean (skeletal) muscle mass resulting in reduced muscular strength, and it stems from decreased mobility, chronic inflammation, and malnutrition [345]. In IBD, chronic inflammation not only exacerbates malnutrition but also triggers profound metabolic alterations, such as increased energy expenditure and muscle wasting, further contributing to the development of sarcopenia [310]. Patients experiencing diminished oral intake, severe inflammation, or short bowel syndrome in CD, particularly in the elderly, are susceptible to muscle tissue catabolism, which can be exacerbated by IBD medications like systemic steroids, which contribute to muscle mass reduction by stimulating myostatin [346]. Sarcopenia serves as a predictive factor for surgery and heightens the rate of postoperative complications [347]. Merely examining BMI alone in patients with IBD can be misleading: body composition may change, with a reduced lean mass offset by increased fat mass [348]. Therefore, tests like hand-grip strength are essential for disease detection [349]. It has recently been shown that in older patients with IBD, the ratio of thyroid hormones, which indicates a loss in lean muscle mass, could serve as an indirect therapeutic biomarker of response to biological therapy [350]. The effective way to increase lean muscle mass is still through physical activity [351, 352].

4.2.2 Obesity

The obesity "pandemic" in Western countries does not spare patients affected by IBD approximately 25% of these patients are obese and roughly 30% are overweight [353]. Obesity complicates surgical resection and raises the risk of anal and perianal fistulas, along with perioperative complications [354]. Obesity and sarcopenia synergistically impact physical function metabolism, mobility, and mortality [307]. Additionally, obesity appears to diminish the response to biological therapy, particularly the fixed-dosing drugs, by accelerating their clearance, making

weight loss an adjunctive therapy in obese patients with IBD [355, 356]. Dietary and lifestyle changes often yield only short-term outcomes. In selected cases of extremely obese patients, bariatric surgery is suggested as a safe and effective option [357].

5. Conclusion and Recommendations

The nutritional aspects of IBD are of significant relevance as they have the potential to influence disease activity and consequently its morbidity. This comprehensive review presents the current evidence on the role of various foods and food groups in the nutrition and nutritional management of IBD. Existing literature suggests that a Western lifestyle, particularly a Western diet, plays a significant role in the increased prevalence of IBD in both industrialized and emerging nations. High consumption of sugar, fats, and/or proteins, along with the reduced intake of fruits and vegetables, as well as the increased use of emulsifiers or other binding substances, are among the important candidate factors that are believed to promote intestinal inflammation in healthy individuals. Furthermore, poor nutrition, as well as selective nutrition, obesity, or sarcopenia, has been associated with poor clinical outcomes, response to therapy, and therefore, quality of life. Therefore, the trend in the management of IBD has shifted toward dietary interventions. Strategies such as SCD, MD, CDED, IBD-AID, and Low FODMAP, among others, have had a positive association with reducing the underlying IBD symptoms and remission of intestinal homeostasis. Given these findings, it is strongly recommended to conduct further research and implement existing dietary strategies on a larger scale to come up with specialized diet therapies and vigorous research of emerging nutrition approaches such as low FODMAP, GrAID, CD-Treat, semi-vegetarian diet, and others to expand the treatment options for patients with IBD.

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Competing Interests

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