Section A-Research paper
ISSN 2063-5346

The Etiology of Inflammatory Bowel Disease Urges a Healthy Diet Rather Than a Wordy Prescription

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Abstract

Idiopathic gastrointestinal diseases include Crohn's disease (CD), ulcerative colitis (UC), and inflammatory bowel disease (IBD) (IBD). These cause severe morbidity and lower quality of life for patients and increase society's costs in both direct and indirect ways. The likelihood of getting IBD and the severity of the illness may be affected by dietary choices, as suggested by epidemiological studies. Symptoms include stomach discomfort, bloody diarrhea, and weight loss. These symptoms can lead to intestinal perforation, strictures, fistulising illness, and cancer. Inflammatory bowel disease has a complicated etiology, but current revelations in our comprehension of the pathophysiology of IBD have led to significant improvements in both diagnosis and treatment. This comprehensive study includes symptoms, diagnosis, clinical pathogenesis, genetic involvement, and mediators responsible for inflammatory bowel disease and related complications. Various animal models are also discussed in detail, along with their specific features. The role that certain nutrients in the diet have in the onset and treatment of inflammatory bowel disease is also highlighted.

Keywords: Ulcerative colitis, Crohn's disease, inflammatory bowel disease, IBD, and Dietary Supplements

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INTRODUCTION

"Inflammatory bowel disease," a kind of peptic ulcer, encompasses Crohn's disease and ulcerative colitis, two of the most common gastrointestinal disorders in both children and adults [1]. With annual incidence rates ranging from 4 to 10 per 100,000 people and prevalence rates ranging from 40 to 100 per 100,000 people, IBD affects people all over the world; however, it is more common in some regions (United States, United Kingdom, and Scandinavia) than others [2]. Approximately 20% of all IBD patients (97%) experience symptoms throughout childhood, with about 5% being treated before age ten [3]. ***Ulcerative Colitis:*** Inflammatory bowel disease is a type that includes UC. The inflammatory response and morphologic alterations in UC are limited to the colon [4]. Most of the inflammation is limited to the mucosa and includes long-term, variable-severity involvement as well as ulceration, emphysema, and bleeding along the colon's length.

Crohn's Disease: Unlike UC, a CD can impact any digestive tract area, including the oropharynx and perianal region [5]. The name "skip regions" comes from the fact that the normal intestine commonly separates diseased segments. Transmural inflammation can happen, which means that the inflammation spreads through the serosa and makes sinus tracts or fistulas form [6]. The clinical manifestations of the intestine are outlined in Crohn's disease and ulcerative colitis.

Clinical Manifestations of the Intestine

***Ulcerative colitis (UC)*:** The condition known as ulcerative colitis (UC) causes the intestines to become inflamed. Including blood and mucus in the stool is the most constant symptom of UC, accompanied by lower abdominal cramping, especially during bowel movements [7]. Clinically, UC and irritable bowel syndrome are distinguished by the presence of blood and mucus in the diarrhea rather than by the absence of blood. Most of the time, UC is found before CD because the blood in the stool is a sign of a disease in the digestive tract [8].

***Crohn's disease* (CD):** Unlike UC, Crohn's disease usually has mild symptoms, which makes it harder to spot complications and delay a diagnosis. A common sign of ileocolonic involvement, especially in young children, is abdominal pain, especially pain after eating that can spread to the periumbilical area. The Gastroduodenal CD is characterized by early satiety, nausea, emesis, epigastric pain, and dysphasia. Patients with Gastroduodenal CD generally restrict their calorie intake to alleviate postprandial pain and delayed stomach emptying.

Extraintestinal Features

Fevers: At the time of diagnosis, fevers are reported in 40% of individuals with IBD. On rare occasions, fevers can spike quickly, but they are usually sustained and prolonged or go unnoticed [9].

Loss of weight: It is one of the most prevalent symptoms seen in adults and children who have IBD, and they may suffer a weight loss.

***Delayed children's growth and sexual development*:** Delayed development is a higher prevalence in CD (60–88%) compared to UC (6–12%), with pubescent adolescents having the highest prevalence. Chronic corticosteroid use might potentially result in growth retardation [10].

***Arthralgias and arthritis*:** Arthralgias and arthritis are frequent in people with inflammatory

bowel disease and can sometimes come before the digestive symptoms. They are frequently involved in pathogenesis, and it is preferable if the inflammation in the digestive tract is treated medically from the inside out [11]. There are two types of involvement: peripheral and axial.

***Mucocutaneous lesions*:** Ulcers typically cause only minor discomfort but can occasionally be incapacitating. They frequently coincide with disease activity, and treatment is centered on the underlying issues.

***Ophthalmologic complications*:** IBD or chronic corticosteroid therapy may result in ocular complications. Scleral and conjunctival erythema, a burning sensation, and photophobia are characteristics of episcleritis, frequently associated with disease activity. They might have signs like eye pain, headaches, or blurry vision, or they might not have any symptoms at all and only be found with a slit-lamp exam.

***Hepatobiliary disease*:** Neurological symptoms, cholangitis is a fibrosing cholestatic liver disease characterized by total bile duct destruction and fibrosing inflammation which is frequently found in conjunction with UC [12].

***Bone anomalies*:** Low bone mass, or osteopenia, can develop at any time during the course of IBD or as a side effect of long-term corticosteroid therapy. Osteoporosis is a significant side effect of pediatric inflammatory bowel disease (IBD) because it happens when more than 90% of peak bone mass is reached in childhood and adolescence [13].

DIAGNOSIS OF IBD

It is impossible to overestimate the significance of avoiding enteric infections before or during an IBD flare. Some pathogens that can mimic IBD include, for example, *Salmonella, Shigella, Campylobacter, Aeromonas, Plesiomonas, Yersinia, Escherichia coli*, *Clostridium difficile, Giardia duodenalis, Histoplasma, Mycobacterium, and Schistosomiasis.* People with IBD may have infections at first, but once the infection is treated, the symptoms may not go away or come back. If intestinal infections are ruled out, additional testing is initiated.

***Symptoms of ulcerative colitis*:** UC causes the inner lining of the colon or rectum (large bowel) to swell and become ulcerated. The majority of those who get UC are between the ages of 30 and 40, and the disease has no gender preponderance. Diarrhea, stomach pain, exhaustion, loss of appetite and body weight, and anemia are common symptoms[14]. The severity of symptoms is determined by the extent to which the colon has been impacted or irritated **(Figure 1)**

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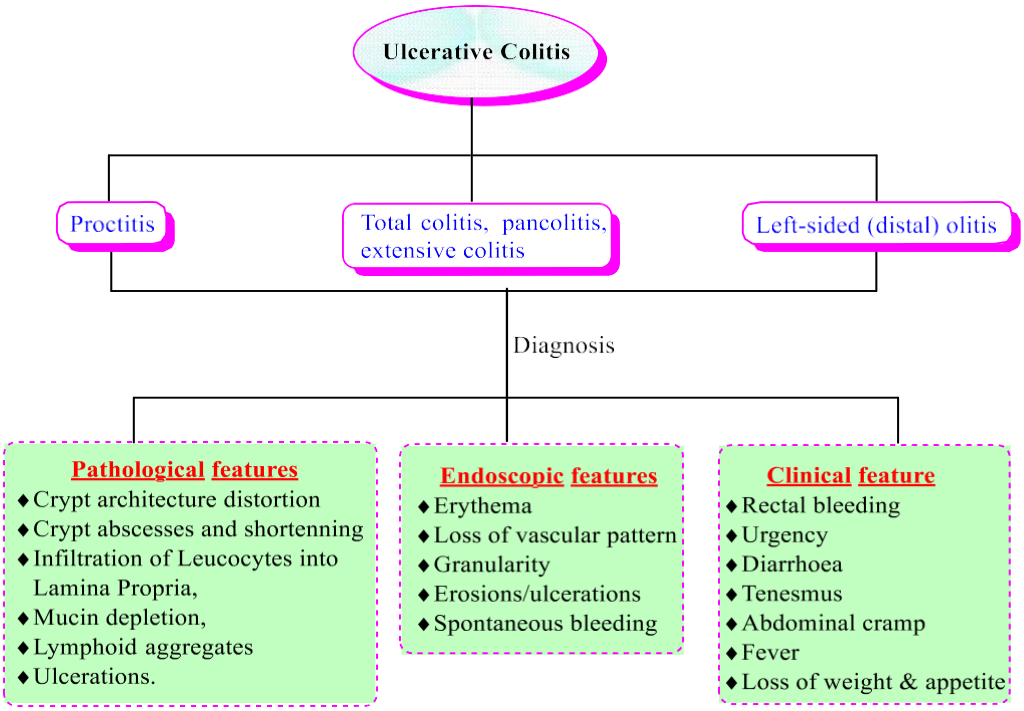

Figure 1. Schematic representation of various categories of ulcerative colitis

***Symptoms of crohn's disease*:** CD symptoms are frequently subtle, making diagnosis challenging **(Figure 2)**. In addition to other endoscopic or pathological findings, gastrointestinal pain, nocturnal or persistent diarrhea and discomfort, intestinal blockage, fever, nocturnal sweats, or weight loss are important factors in the initial diagnosis[15].

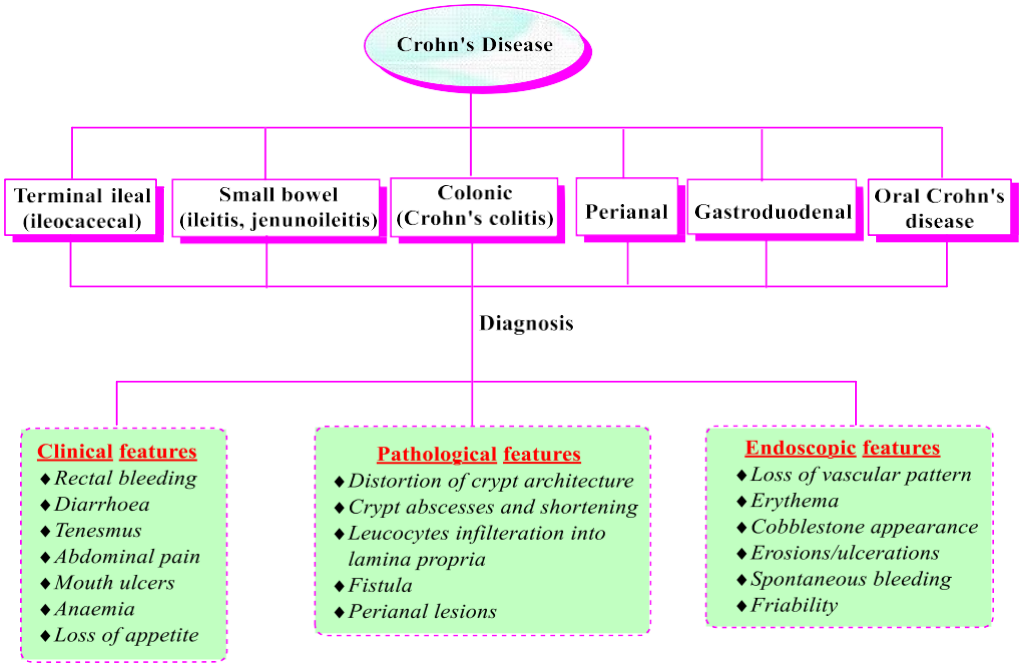

Figure 2: Graphical representation of various forms of crohn's diseases

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Hematologic tests

Inflammatory markers, and a metabolic profile, which includes liver enzymes and a complete blood count, are all included in IBD screening tests. IBD symptoms include thrombocytosis, microcytic anemia, and an elevated white blood cell count with increased band formations. In over 90% of juvenile CD patients but not in UC kids, acute phase reactants such as sedimentation rate, creative protein, and serum orosomucoid level are raised[16].

Evaluation by endoscope

An endoscopic examination with biopsies is advised when considering an IBD diagnosis to confirm the diagnosis. In some cases, the diagnosis of "ambiguous colitis" is made because the symptoms are unusual and histologic tests cannot reliably distinguish between UC and CD [17].

Radiologic studies

A small bowel follow-through X-ray examination is typically reserved for CD patients to ascertain the involvement of the terminal ileum and small bowel loops. On the other hand, barium enemas might help people with CD find stenosis, fistulae, or sinus tracts [18].

TREATMENT OF IBD

Medical management of Ulcerative colitis

In treating mild illness, oral sulfasalazine is used alone or in combination with topical medicines. Patients who cannot actually accept sulfasalazine due to side effects may benefit from Mesalamine, Olsalazine, and Balsalazide are more recent 5-aminosalicylic acid medicines [19]. ***Moderate to severe disease:*** Patients suffering from severe stomach cramps, bloody diarrhea, abdominal pain, anemia, or hypoalbuminemia should be admitted to the hospital for close supervision, intravenous medicines (corticosteroids), and food. Respiratory syncytial medications need to be avoided since they put patients at risk for poisonous products, which are common [20]. ***Immunosuppressive therapy:*** Because around 50% of patients experience deleterious effects from corticosteroids, azathioprine or 6-mercaptopurine are utilized for whose prednisone benefits. These medicines are not utilized to treat acute colitis because of their delayed onset of effect. When surgery seemed inevitable, cyclosporine and tacrolimus were utilised to treat acute steroid-refractory UC. Patients who attain remission notice clinical improvement within 7 to 10 days. When these drugs are stopped, however, the majority of patients revert. Prolonged remission is more likely when azathioprine or 6-mercaptopurine are started at least four weeks before stopping cyclosporine therapy [21]. ***Prognosis:*** About 25 to 40 percent of people with severe ulcerative colitis require a colonoscopy. An 8-year surveillance colonoscopy should be performed on patients with severe colitis to look for dysplasia [22]. Repeat colonoscopies should be done every 1 to 2 years after the surveillance colonoscopy is started. Swedish patients with colon cancer who were first diagnosed before the age of 15 had a one in 15, a 6.5% chance of 20, and a 15% chance after the age-of-20 chance of 20 of developing colon cancer, respectively [23].

Medical Treatment for Crohn's disease

The medical care for Crohn's Disease is tailored to the extremity of signs and the extent and location of intestinal involvement [24]. ***Corticosteroid:*** In most individuals, corticosteroids (1mg/kg/day) are beneficial in reducing disease activity and establishing remission. However, due to unfavorable side effects (including osteopenia, impacts on children's linear

development, and cosmetic side effects), prolonged use of corticosteroids is not advised [25]. ***Sulfasalazine and Mesalamine:*** Uses for mesalamine or sulfasalazine include treatment of moderate to severe illness and to keep corticosteroid-influenced remissions going. Ileocolonic and colonic illnesses benefit from sulfasalazine. About 30% of people can't take sulfasalazine because it gives them side effects, the most common of which are headaches [26]. ***Antibiotics:*** Treatment for individuals with mild to severe illness, especially those with perianal disease and systemic infection, is effective with metronidazole and ciprofloxacin. Once the antibiotic is stopped, sensory neuropathic pain, which can develop from sustained metronidazole use, usually goes away or gets better [27]. ***Immunosuppressive therapy:*** Patients with resistant fistulas, severe small bowel disease, a history of prior resections, gastric and duodenal disease, and steroid dependency should take 6-Mercaptopurine with azathioprine [28]. Patients with severe CD and active fistulae have been treated with cyclosporine [29]. ***Biological therapy:*** In CD mucosa, both histologically normal and inflamed, there has been an increase in proinflammatory production, particularly of tumor necrosis factor-alpha (TNF-alpha). An 81 percent clinical response and a 48 percent clinical remission were seen in CD patients after Infliximab, a single infusion of a chimeric monoclonal antibody against TNF that is 5 mg per kg. Fistulae in CD patients have now been successfully treated with infliximab infusions, and remission has been sustained [30].

PATHOGENESIS OF IBD

Pathogenesis of Crohn's Disease

Several studies have suggested that human CD is a Th1-mediated illness, with increased Th1- cell activity being a key component of the disease. For example, rising numbers of activated T cells in the intestinal mucosa are a feature of CD. These T cells release IFN-γ and have a Th1 phenotype [31]. IL-18, like IL-12, is a cytokine involved in the maturation of Th1 cells and the generation of interferon. In the intestinal mucosa of people with IBD, both IL-12 and IL-18 are detected at elevated levels. In addition, lamina propria lymphocytes from CD patients' intestines have been demonstrated to produce IL-12.

Extraintestinal manifestations' pathogenesis

Several extraintestinal symptoms of CD and UC have been noticed and well characterized. Although extraintestinal symptoms frequently arise in conjunction with intestinal disease activity, the pathophysiology of these IBD manifestations remains unknown [32]. Memory T cells may circulate again between the gut and the synovium in the case of arthritis, according to studies on HLA-B27 transgenic rats. This theory is supported by the fact that lamina propria lymphocyte adhesion to synovial high endothelial venules has been demonstrated.

Hypotheses in connection with pathogenesis

IBD has been linked to autoimmune reactions to luminal or mucosal antigens, compromised immune responses to commensal bacteria, and infections with pathogenic organisms that remain in the intestinal tissues and induce a persistent inflammatory response [33].

***Autoimmune disease*:** It has been hypothesized that a harmful inflammatory process directed against an identity like colonocytes, goblet cells, mucin, or other cells is the fundamental cause of IBD, particularly in the case of UC [18]. In conclusion, it is still unknown what

endogenous antigens in UC cause the inflammatory response and what mechanisms result in clinical disease [34].

***Infection with a pathogenic organism*:** IBD shares a number of symptomatic and histologic characteristics with gastrointestinal disorders caused by recognized pathogenic organisms. Acute CD, for example, can mimic gastrointestinal sickness caused by *Yersinia pestis* or *Mycobacterium tuberculosis* [35]. Other enteric bacteria, like Shigella or Campylobacter can also bring on UC-like symptoms. This observation, however, might result from the constrained mechanism the mucosal immune system of the intestine uses to respond to damage. Despite the fact that CD is named after the researcher who first separated it from intestinal tuberculosis in 1932, the idea that CD is caused by a mycobacterium tuberculosis infection has persisted.

GENETICS RELATED TO INFLAMMATORY DISEASES

Iceland now has one of the highest rates of UC in the world (12 per 100,000), which is also on the rise [36]. Although it has been noted that African Americans experience CD and UC less frequently than Caucasians do, a recent study of children in Georgia found that the incidence of both UC and CD was 7 to 12 per 100,000 and 5 to 7 per 100,000, respectively.

Genetic epidemiology

Both CD and UC have shown higher familial aggregation in family investigations with IBD. Referral-based research has found that 20–30% of people with IBD have a family history of the disease. On the other hand, population-based surveys have found that this number is between 5 and 10% [18]. Cohort studies or case-control studies can be used to figure out how common IBD is in the families of people with UC or CD and how likely they are to get it.

Genome-Wide genetic linkage studies

Finding disease genes that cause common, complex genetic disorders like IBD can be accomplished through investigation of potential genes or genetic code analyses utilizing genetic connection. Attempts to find illness genes are further complicated by genetic heterogeneity, which occurs when distinct sets of genes produce comparable phenotypic expression in different groups of patients [37]. For monogenic illnesses like cystic fibrosis, genome-wide studies to uncover disease genes have been successful in refining the localization of and eventually identifying disease genes.

Linkage analyses in the area of chromosome 6p

Significant evidence linking CD and UC to the chromosome 6p area has been discovered in addition to the chromosome 16 core region. The HLA genes and many other immune-related genes, such as the TNF gene, are located in a region on chromosome 6p called the major histocompatibility group [38]. It is unsurprising that there is a relationship with this area, given the chronic inflammatory nature of many illnesses. The genetic linkage evidence for IBD is one of many equally strong links, unlike the linkage evidence for type I diabetes mellitus, which is dominated by the chromosome 6p linkage. This suggests that Minor Histocompatibility Group (NMHC) genes have, I suppose, an equal impact on the etiology of disease as do MHC genes [18].

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Association studies of HLA class II genes and IBD

Given the significant genetic diversity found concerning the primary histocompatibility complex and the crucial role that HLA class II genes, which are involved in the immunological response, have the potential to be linked to IBD [39]. Numerous case-control studies have examined the associations of the HLA-DR gene with CD and UC. These researchers' interpretation is hindered by broad serologic subtypes in earlier research (which constitute a significant portion of the literature) that have since been demonstrated to relate to many DNA-based genotypes [40].

Genetics and environmental factors

Similar to how genetic factors are likely to include modifiers that affect the phenotype of an illness, environmental influences will change specific disease aspects (such as the expression of UC versus CD). Tobacco exposure is the most well-studied of these environmental influences [41]. Tobacco use has been linked to an increased risk of developing CD, despite being less frequently reported, and a lower risk of developing UC. Smokers who were actively smoking had a lower risk of developing UC than non-smokers (odds ratio, 0.53; 95 percent confidence interval, 0.24 to 1.14). The risk was also lower in passive smokers (odds ratio, 0.50; 95% confidence interval, 0.25 to 1.00) and in people whose parents smoked [42]. Like celiac disease, a chronic inflammatory condition of the proximal intestine, tobacco has a preventive effect against the emergence of UC. Contrarily, smoking seems to have a more long-lasting protective effect against celiac disease [43].

ANIMAL MODELS FOR IBD

Findings from animal model studies have shown the relevance of genetic and environmental variables in IBD pathogenesis. Furthermore, animal models have indicated that various immune system changes might result in intestinal inflammation.CD4 T cells, on the other hand, have been repeatedly demonstrated to play a role in inducing intestinal inflammation. Finally, animal studies imply that antigens produced from commensal bacteria may be involved in the development of IBD. Even though none of the current animal models completely mimics human IBD, studies of intestinal inflammation in animals have shed important light on the etiology of the intestinal inflammatory response. There are two types of animal models of IBD: spontaneous and induced [18].

Spontaneously occurring models

Cotton-top tamarins develop spontaneous colitis, which is similar to IBD. Additionally, Jackson Laboratories (Bar Harbor, Maine) developed the C3H/HeJBir strain of mice through selective breeding of C3H/HeJ mice. These animals naturally develop cecum and right colon inflammation, which peaks at 3 to 6 weeks of age and clears up by 10 to 12 weeks [44].

Induction of IBD with exogenous reagents

For instance, giving indomethacin, adding 5% dextran sulfate sodium to the water, or giving an ethanol-based trinitrobenzene sulfonic acid enema can all result in colitis. A standard model for investigating mucosal repair mechanisms is the regeneration of the colonic mucosa following repeated cycles of 5% dextran sulfate sodium injection, which takes several weeks [45].

IBD in genetically altered rodents

Several mouse strains developed by gene-targeting methods exhibit intestinal inflammation when kept in standard conditions. Several mouse models have been used to study mutations affecting cytokine production or the CD4 T-cell population. Small proteins known as cytokines are involved in several biological processes, including cell development, differentiation, and inflammatory and immune responses [46]. T-lymphocytes are stimulated to proliferate and expand, while other cells, including macrophages, are activated by the cytokine IL-2. IL-2 deletion mice experience symptomatic pancolitis, which includes gastrointestinal bleeding, diarrhea, and, in some cases, rectal prolapse [47].

Adoptive CD4 cell transfer into immune-deficient mice

When adoptively transferred into SCID mice, which lack B and T cells, CD4 T cells that express high levels of CD45RB cause intestinal inflammation, diarrhea, and weight loss. Concurrent transfer of CD45RB low-CD4 T cells reduces inflammation in this mouse by producing a transforming growth factor [48].

MEDIATORS: INVOLVEMENT AND RESPONSES IN INFLAMMATORY MECHANISM

***Role of T-cells*:** Animal models of IBD exhibit mucosal inflammation, which is significantly influenced by CD4 T cells. On the other hand, CD8 T cells seem to be of little importance.

***Role of cytokines*:** In most animal models, the T-cell subset responsible for mediating intestinal inflammation appears to be an IL-12-producing Th1 cell that secretes IFN- and TNF. Th2 cells might also be crucial in developing intestinal inflammation in some animal models of IBD.

***Other inflammatory mediators*:** Chemokines are a group of peptides that serve as chemoattractants, attracting and activating leukocytes. Chemokines like IL-8, MCP-1, and ENA-78 have high levels of expression in the intestinal mucosa in regions with active CD and UC, indicating that they may be important mediators of inflammation in IBD[49]. **Table 1** lists the secreted components linked to the inflammatory response in the intestine in IBD.

Table 1: Secreted factors implicated in the intestinal inflammatory response in IBD[50-51]

Class	**Factor**
Cytokines	IL-12, TNF, IFN-γ, IL-10, IL-18, IL-15, lymphotoxins alpha and beta, IL-1β
Chemokines	IL-8, MCP-1, ENA-78, RANTES
Growth factors	keratinocyte growth factor, vascular endothelial growth factor, epidermal growth factors, trefoil factors

Other	Matrix metallo-proteinases

HOW INFLAMMATORY BOWEL DISEASE IS AFFECTED BY DIET?

Environmental factors, especially food-related ones, may influence the development of inflammatory bowel disease (IBD). Nutrient intake influences host physiology and disease. There is a complicated interplay between dietary nutrients and gut immunity [52-53]. Diet and nutrition directly affect immune cells that live in the gut and play a crucial role in shaping the microbiota in the stomach. A balanced diet is essential for optimal health. In contrast, inflammatory bowel disease (IBD) may sever links between what we eat, our immune systems, and our gut microbes. Genetics and intestinal dysbiosis may influence nutrient absorption from the diet. Certain nutrients necessary for immunological and microbial balance may be more or less in demand depending on how much inflammation there is in the intestines [54]. Hence, dietary intervention in IBD causes and prolongs illness remission by reducing nutritional risks [55]. Certain dietary components can boost the host's immune system and intestinal barrier function, protecting the host against illness. Consequently, IBD dietary therapy must provide helpful nutrients while minimizing nutritional risks. Dietary nutrients affect host immunity, intestinal barrier function, and gut microbial composition and function. Host physiology and illness can be affected by changing gut flora. Inflammation also alters the metabolism of host immune and non-immune cells and gut flora [56]. Hence, IBD may alter host and microbial nutritional needs. A better understanding of the complicated relationship between dietary nutrients, host immunology, and gut microbiota is needed to improve IBD nutritional therapies [57]. Dietary fiber benefits individuals with IBD by nourishing the bacteria in the distal intestine, aiding disease management [58]. In animal models of colitis, preexposure administration of dietary fiber reduces the severity of the disease, which may be due to intestinal mucus layer protection during active illness. Increasing fiber intake has been shown to reduce UC disease activity in several research investigations [59-60]. Gluten exacerbates intestinal inflammation in TNF- deficient mice (a mouse model of genetic predisposition to IBD) [61]. Amylase trypsin inhibitors (ATIs), a class of non-gluten proteins present in gluten-rich cereals, may control the production of inflammatory cytokines, activate Toll-Like Receptors, and stimulate a T- cell response in both coeliac and non-coeliac individuals, including IBD patients [62-63]. A gluten-free diet has been shown to alleviate symptoms and reduce flare frequency in IBD patients without celiac disease, with up to 28% of IBD patients avoiding gluten in the past [64-65]. Red meat consumption may promote inflammation, which may clarify why eating

meat, processed meat, and alcohol increases the frequency of eruptions in UC patients. This may be related to the harmful effects of the cooking process and saturated fat [66]. Ammonia, amines, hydrogen sulfide, and nitrous compounds are produced by the fermentation of meat-derived protein by gut microbes [67]. Certain byproducts of fermentation have been linked to DNA damage and the development of genetic instability. This effect has been observed in healthy individuals after a red meat or

vegetarian diet. In a study, mice with colitis were fed red meat and developed a more severe form of the disease [68]. Zinc supplementation is recommended for IBD patients because it reduces colitis severity and myeloperoxidase enzyme activity, whether alone or with anti-TNF [69]. The postulated mechanism may be due to the change in TNF receptor expression and innate immune response. Dietary zinc from meat, fish, cereal, and dairy was shown to be inversely associated with CD but not UC [70-71]. Diets rich in saturated fat, omega-6 fatty acids, and polyunsaturated fatty acids are associated with an increased risk of CD and UC [72-73]. The body can produce most fatty acids on its own, except linoleic acid and alpha-linolenic acid, which are considered essential [74]. Fatty acids' biological activity as energy sources and membrane components influences the immune system and the gut flora [75]. The gut microbiota and barrier function are both affected by a high-fat diet, leading to intestinal and systemic inflammation. High-fat and sugar diets enhance the colonization of adherent- invasive *E. coli*, an IBD pathobiont [76]. There is a higher concentration of food additives in processed foods, and complex emulsifiers are commonly used in processed foods to enhance texture and prolong shelf life [77]. Several research studies have connected emulsifier use to inflammatory bowel disease (IBD) because emulsifiers disrupt the host-microbiota connection, leading to intestinal inflammation and colon cancer [78]. Mucolytic bacteria like *Ruminococcus gnavus* and *Akkermansia muciniphila* thrive in the presence of dietary emulsifiers, although the mechanism is unclear. Artificial sweeteners and other additives cause dysbiosis of the intestines [79-80]. Artificial sweeteners boost Proteobacteria growth and ileal lamina propria bacteria in CD-like ileitis model mice. Dietary phosphate, a common food additive, activates NF-kB in macrophages, increasing intestinal inflammation. These findings suggest that several dietary additives may increase the risk of IBD [81-82].

CONCLUSION

Inflammatory bowel disease (IBD) is prevalent in the United States and Europe; it is a widespread condition that causes significant morbidity. In the last few years, we've made considerable progress in understanding the immune mediators that cause intestinal inflammation. As a result of this insight, new IBD therapeutic techniques have been developed. Furthermore, a growing body of evidence suggests that genetic factors may play a role in the development of IBD by predisposing patients to a deregulated immune response. The commensal bacterial flora also appears to be necessary, especially in the case of CD. Environmental variables, particularly those connected to food, may have a role in the development of inflammatory bowel disease (IBD). Dietary phosphate, a frequent dietary ingredient, and emulsifiers have a deleterious influence on IBD, causing increased intestinal
inflammation and increasing the prevalence of the disease. A well-balanced diet is advised for IBD patients because specific dietary components can improve the host's immune system and intestinal mucosal function, hence protecting the host against disease. The current study has summarized the pathogenesis and route factor for IBD and emphasizes treatment options via a wide variety of therapeutic approaches.

Funding

This study does not receive any external funding.

Conflict of interest

Authors declare no conflict of interest

Acknowledgments

The Arpita Yadav would like to acknowledge the management of R.K. College of Pharmacy for providing the facility to complete this comprehensive study. Sudha Bhartiya are thankful to Dr. Anu Chaudhary, Principal & Director, Bhagwati College of Pharmacy, Baraut, Uttar Pradesh, for her motivation and support.

Ethics approval and consent to participate: Not applicable.

Human and animal rights: Not applicable.

Code availability: Not applicable.

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Figure Legends:

Figure 1. Schematic representation of various categories of ulcerative colitis

Figure 2: Graphical representation of various forms of crohn's diseases