Diet and Inflammatory Bowel Disease

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Abstract: Patients with inflammatory bowel disease (IBD) are increasingly becoming interested in nonpharmacologic approaches to managing their disease. One of the most frequently asked questions of IBD patients is what they should eat. The role of diet has become very important in the prevention and treatment of IBD. Although there is a general lack of rigorous scientific evidence that demonstrates which diet is best for certain patients, several diets—such as the low–fermentable oligosaccharide, disaccharide, monosaccharide, and polyol diet; the specific carbohydrate diet; the anti-inflammatory diet; and the Paleolithic diet—have become popular. This article discusses the diets commonly recommended to IBD patients and reviews the supporting data.

Inflammatory bowel disease (IBD) is a chronic immune disorder of unclear etiology. Multiple factors play a role in the pathogenesis of IBD. These may include diet, environmental factors, immunologic factors, infectious agents, genetic susceptibility, and the microbiome. The emergence of rapid increases in the incidence of IBD over the past several decades in low-incidence parts of the world, such as China, South Korea, and Puerto Rico, clearly points to the important role that environment plays in disease development.[1-3] Specifically, the introduction of the Western diet (which is high in fat and protein and low in fruits and vegetables) has been proposed as an explanation for the increase in IBD incidence.[4]

Therefore, clinicians, and especially patients, have questioned whether diet influences the onset or course of IBD (Figure 1). The question of what to eat is the most commonly encountered question as well as the most challenging one posed to gastroenterologists managing IBD patients. At present, there is no specific IBD diet that is supported by robust data, leaving patients to seek nonmedical resources for dietary guidance. Dietary intervention trials have been limited by their lack of a placebo control group and the difficulty in meticulously capturing dietary intake conjointly with the potential for complex interactions between foods. Furthermore, dietary trials may not detect significant differences for patients undergoing withdrawal of specific drug therapies.[5]

The lay literature has promoted several popular diets that have been touted to alleviate intestinal inflammation and have thereby been advocated for patients with IBD. These include the specific carbohydrate diet (SCD); the low–fermentable oligosaccharide,

Figure 1. The impact of diet on inflammatory bowel disease.

disaccharide, monosaccharide, and polyol (FODMAP) diet; the Paleolithic diet (Paleo); and the anti-inflammatory diet (IBD-AID). These diets are promoted in the lay literature through anecdotal success stories but to date lack rigorous scientific assessment. As patients look to complementary therapies for management of their disease, it is important that clinicians understand the effectiveness and/or lack thereof of these dietary interventions in order to better advise and direct patients.

The Specific Carbohydrate Diet

The SCD was first described by Dr Sidney Haas in 1924 as a means to treat celiac disease. The SCD was popularized for the treatment of IBD by biochemist Elaine Gottschall through her lay book *Breaking the Vicious Cycle* after her daughter was cured of ulcerative colitis using the diet.[6] This diet is based on the theory that disaccharides and polysaccharides pass undigested into the colon, resulting in bacterial and yeast overgrowth, which causes overproduction of mucus. It is further hypothesized that this malabsorption may cause intestinal injury. Therefore, strict adherence is recommended to prevent additional mucosal damage. The SCD permits consumption of only monosaccharides and restricts intake of simple carbohydrates (Table 1). The diet generally results in weight loss and is quite difficult for patients to follow strictly.

The Paleolithic Diet

The Paleo diet was introduced by Dr Walter L. Voegtlin, a gastroenterologist, who published a lay book entitled *Stone Age Diet*.[7] The theory behind this diet is that the human digestive tract has not evolved to handle the modern diet, which is laden with agriculturally derived foods. The Paleo diet emphasizes the intake of lean, nondomesticated meats and noncereal, plant-based foods (Table 2). The diet does not focus on eliminating certain foods, as does the SCD, but rather focuses on the source and balance of caloric intake. The Paleo diet advocates that lean protein be the source of 30% to 35% of daily caloric intake in addition to a very high-fiber diet from noncereal, plant-based sources, up to 45 to 100 g daily.[8]

Table 1. Characteristics of the Specific Carbohydrate Diet

Definition	Disaccharide and polysaccharide carbohydrates are poorly absorbed in the gastrointestinal tract, causing bacterial and yeast overgrowth resulting in overproduction of mucus. This diet limits monosaccharides (glucose, fructose, and galactose).	
Limitations	References consist of only case studies on systemic D-lactic acidosis, not mucosal concentrations of organic acids or mucosal injury. The diet has the potential to contribute to vitamin D deficiency.	
	Include	*Avoid*
Grains	None	All cereal grains
Fruits	All but canned or frozen fruits	None
Vegetables	All but canned or frozen vegetables	Potato, yam, corn
Proteins	All others	Processed, canned, or smoked meats
Nuts, seeds, legumes	Lentil, split pea	Most legumes (eg, chickpea, soybean)
Dairy	Lactose-free	All others
Beverages	Wine	Milk, instant tea, instant coffee, soybean milk, beer
Other	Saccharin, honey, butter	Chocolate, margarine, corn syrup
Reference	Gottschall E. *Breaking the Vicious Cycle: Intestinal Health Through Diet*. Baltimore, Canada: Kirkton Press; 2012.	

The Low–Fermentable Oligosaccharide, Disaccharide, Monosaccharide, and Polyol Diet

FODMAPs, which are highly fermentable but poorly absorbed carbohydrates and polyols, were first described in 2005 when researchers hypothesized that the rapid fermentation and passing of these substances led to

increased intestinal permeability. This permeability has been identified as a predisposing factor to IBD in a genetically susceptible host.[9] Evidence followed in 2006 when gastrointestinal complaints in patients with irritable bowel syndrome (IBS) were found to be related to fructose malabsorption. Three out of 4 patients reported symptomatic improvement with restrictions of FODMAP intake.[9] The impact of FODMAPs has been attributed to their diminutive molecular size and high osmotic effect, which is associated with increased colonic bacterial fermentation. Symptoms associated with a high-FODMAP diet include increased gas, bloating, distention, cramping, and diarrhea. Luminal distention caused by the fermentation of FODMAPs by bacteria in the small and proximal large intestines could result in IBD patients who have superimposed IBS.[10]

Patients with gut motility disorders and visceral hypersensitivity appear to be more afflicted by these side effects. Compared with healthy individuals, patients with IBS have significantly worse symptoms when consuming a high-FODMAP diet (50 g daily) due to higher hydrogen production.[10] Similar results have been observed in patients with nonceliac gluten sensitivity.[11] The low-FODMAP diet initially consists of eliminating foods high in FODMAPs for 6 to 8 weeks; this diet is not intended to be a long-term therapy. After symptom resolution, patients are guided by a dietitian on how to gradually reintroduce foods high in fermentable carbohydrates to determine individual tolerance to specific FODMAPs.[12-14] Although the low-FODMAP diet is very restrictive, Gearry and colleagues reported successful implementation and diet adherence for the majority of their patients with IBD (Table 3).[15] The efficacy of the diet in IBD patients was associated with dietary adherence.[15] However, most of the evidence has been based on a few retrospective pilot studies and has been limited to symptomatic responses. Further studies are needed to determine how rigorous the diet needs to be to provide favorable long-term outcomes. Long-term, well-controlled studies are needed to assess possible nutritional inadequacies, evaluate mucosal healing, and examine changes in gut microflora in IBD patients on a low-FODMAP diet.

Table 2. Characteristics of the Paleolithic Diet

Definition	The human gastrointestinal tract is poorly evolved to handle the modern diet that resulted from the development of modern agricultural methods. Exposure to foods that were not present at the time of human evolution may result in modern diseases.	
Limitations	No research studies have been conducted to test this diet in the IBD population. The diet has the potential to contribute to vitamin D deficiency.	
	Include	*Avoid*
Grains	Cereal grains	All others
Fruits	All	None
Vegetables	All others	Potato, corn, yucca, butternut squash, yam, beet
Proteins	Lean-game meats, fish, shellfish	Domesticated meats
Nuts, seeds, legumes	All nuts and seeds	All legumes, peanut
Dairy	None	All
Beverages	All others	Soft drinks, alcoholic beverages, fruit juice
Other	Honey	Refined sugar, artificial sweeteners
Reference	Eaton SB, Konner M. Paleolithic nutrition. A consideration of its nature and current implications. *N Engl J Med*. 1985;312(5):283-289.	

IBD, inflammatory bowel disease.

Oligosaccharides

Fructans are composed of a long chain of fructose that ends in a glucose molecule. Their classification is based on fructose-fructose bonds, either inulins (β1-2 bond) or levans (β2-6 bond). Within the inulin terminology, those with a degree of polymerization (chain length) of less than 10 are referred to as fructooligosaccharides and those with at least 10 are called inulins. Inulin-type fructans are classified as nondigestible in the small intestine because they have a β configuration in their fructose monomers, whereas α-glucosidase, maltase-isomaltase, and sucrose enzymes are α-osidic linkages. Most dietary fructans are found as fructooligosaccharides, and major sources are wheat, garlic, rye, barley, pistachio, peach, watermelon, artichoke, beetroot, leek, pea, and onion.[16]

The dietary forms of galactooligosaccharides are raffinose (which consists of a fructose, glucose, and galactose molecule) and stachyose (which is a raffinose with an additional galactose molecule). After ingestion, galactooligosaccharides are poorly absorbed due to the lack of the digestive enzyme α-galactosidase, which is required for the hydrolysis of these oligosaccharides to their simple sugar constituent. After 100% of ingested stachyose and raffinose arrive in the colon, they are metabolized by resident microbiota into lactate; short-chain fatty acids (SCFAs) such as butyrate, acetate, and propionate; and other volatile compounds. Significant dietary sources include legumes, such as lentil, bean, chickpea, cabbage, Brussels sprout, chicory, and onion.[17,18]

Table 3. Characteristics of the Low-FODMAP Diet

Definition	FODMAP is an acronym that stands for fermentable oligo-, di-, and monosaccharide and polyol carbohydrates, grouped together based on the length of their carbohydrate chains. Foods containing these forms of carbohydrates worsen the symptoms of IBS and IBD. These foods are poorly absorbed in the small intestine, highly osmotic, and rapidly fermented by bacteria in the gut, which can lead to increased IBS and IBD symptoms.	
Limitations	Based on only a few retrospective pilot studies and limited to symptomatic responses.	
	Include	*Avoid*
Grains	Gluten-free foods, oat, rice, quinoa	Wheat, barley, rye
Fruits	Banana, blueberry, cantaloupe, clementine, grape, kiwi, lemon, lime, mandarin, melons (variety), orange, passion fruit, pineapple, raspberry, strawberry	Apple, applesauce, apricot, blackberry, canned fruit, date, dried fruit, grapefruit, mango, nectarine, pear, peach, plum, prune, watermelon
Vegetables	Alfalfa, bean sprout, bell pepper, bok choy, broccoli (≤1/2 C), Brussels sprout (≤2 sprouts), carrot, corn, cucumber, eggplant, green bean, kale, lettuce, potato, spinach, spring onion (only green top), squash, tomato, turnip, zucchini	Artichoke, asparagus, avocado, beetroot, cauliflower, cabbage, garlic, leek, mushroom, onion, pea, shallot, snow pea, sweet corn, sweet potato
Proteins	All others	Breaded meat or meat made with HFCS
Nuts, seeds, legumes	Almond (≤10 nuts), chia seed, nut butter, macadamia, peanut, pecan, pumpkin seed, walnut	Bean, cashew, chickpea, lentil, pistachio, soybean
Dairy	Lactose-free yogurt and milk; almond, coconut, rice, or soy milk (from soy protein); hard and low-lactose cheese	Cow, goat, sheep, condensed, and evaporated milk; buttermilk; soy milk (from soybean); soft cheese and cream
Beverages	Fruit and vegetable juice made with allowed foods (limit to 1/2 C at a time), wine (5 fl oz), vodka, gin (1.5 fl oz)	Coconut water, green tea, rum, soft drinks, sports drinks, white tea
Other	Brown sugar, dark chocolate, maple syrup, golden syrup, stevia	Milk chocolate, sweeteners ending in "-ol," honey, HFCS
Reference	Gibson PR, Shepherd SJ. Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J Gastroenterol Hepatol*. 2010;25(2):252-258.	

HFCS, high fructose corn syrup; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome.

Disaccharides

Lactose is a disaccharide composed of 2 sugar molecules (glucose and galactose) hydrolyzed by lactase at the intestinal brush border to be readily absorbed. Inadequate lactase activity or an insufficient level of lactase allows undigested lactose to reach the large intestine, where it is converted to SCFAs and hydrogen gas.[19] The SCFAs are absorbed by the colonic mucosa, where malabsorbed lactose is used for energy utilization. The estimated prevalence of lactose intolerance in the United States and Europe varies among different ethnic and racial groups. The prevalence is highest among Native Americans (80%-95%), followed by Africans and African Americans (65%-75%), Hispanics (50%), and whites (7%-20%). The prevalence exceeds 90% in some populations in eastern Asia.[20] Cohen and colleagues recently assessed the dietary patterns and beliefs of a broad cohort of patients with IBD, 51% of whom avoided milk and milk products because of the belief that dairy worsened their symptoms,[21] which could reflect a component of lactose intolerance. Common foods rich in dairy include milk, custard, ice cream, soft cheese, and yogurt.[14]

Monosaccharides

Dietary fructose is usually ingested as monosaccharides, enzymatically extracted from the disaccharide sucrose or polymerized as fructans. Common dietary sources include apple, pear, watermelon, asparagus, sugar snap pea, honey, and products sweetened with high fructose corn syrup.[22] Fructose is thought to be transported via the facilitative transporters GLUT5 or GLUT2 across the intestinal epithelium. Fructose absorption in humans appears to be limited at high fructose concentrations, consistent with the limited absorptive capacity of a facilitative transport system. Additionally, some adults may not adequately upregulate GLUT5 expression, and their capability to absorb fructose may be inadequate.[23] Fructose absorptive

capacity is compromised when the dietary concentration exceeds that of glucose, and malabsorption occurs. Fructose malabsorption can occur in 80% of individuals with a dietary load of 50 g of fructose.[24] Approximately 50% of the general population is unable to completely absorb 25 g of free fructose load in the small intestine.[12] Ingestion of foods and beverages containing more than 0.5 g of excess fructose per 100 g of glucose, and/or an excess of 3 g of fructose regardless of the amount of glucose, is desirable to minimize symptoms related to fructose malabsorption.[14]

Polyols

Sorbitol, mannitol, maltitol, and xylitol are sugar alcohols. They are poorly absorbed in the small intestine, consequently entering the colon, where they are subject to anaerobic fermentation.[25] The diffusion rate depends on molecular size, intestinal permeability, and, if pore size is affected, by mucosal disease. Gaseous end products, such as carbon dioxide, hydrogen, and methane, exert osmotic effects leading to abdominal pain, flatulence, and osmotic diarrhea.[26] Compared with healthy individuals, patients with IBS report an increased and discordant absorption of mannitol and sorbitol.[27] Natural food sources include apple, pear, apricot, cherry, nectarine, peach, plum, watermelon, mushroom, and cauliflower. However, the artificial sweeteners mentioned above have been popularized by the food industry to produce low-calorie food products or to fulfill technological functions, acting as emulsifiers, bulking agents, stabilizers, thickeners, texturizers, and so on. These artificial sweeteners can be used as a sugar substitute in a 1:1 ratio while maintaining low-calorie content.[28]

The Anti-Inflammatory Diet

Olendzki and colleagues have reported the results of a promising retrospective case series using a novel diet.[29] Their dietary intervention was based on addressing nutrient adequacy, IBS symptoms, and malabsorption for IBD patients who had failed pharmacologic therapy. The development of the IBD-AID is based on the theory that dysbiosis is caused by certain carbohydrates acting as substrates to pathogenic bacteria in the lumen of the gut. Therefore, this diet limits certain carbohydrates, such as refined sugar, gluten-based grains, and particular starches that are thought to stimulate the growth of inflammatory bacteria in the digestive tract (Table 4). The IBD-AID has 5 basic components: (1) the modification of specific carbohydrates (eg, refined or processed complex carbohydrates and lactose); (2) emphasis on restoring the intestinal flora balance through ingestion of prebiotics and probiotics in the form of soluble fiber such as leek, onion, and fermented foods; (3) focus on decreasing total and saturated fats, eliminating hydrogenated oils, and encouraging the increase in food sources rich in omega-3 fatty acids; (4) review of the overall dietary pattern, identification of food triggers and intolerances, and detection of missing nutrients; and (5) food texture modification to enhance absorption and reduce intact fiber. The initial foods recommended (soft, well-cooked foods without seeds) were based on the severity of the patient's symptoms. As symptoms improved, patients were further advanced with more whole foods (Figure 2). Among patients who followed the diet for 4 or more weeks, 100% reported reduced symptoms.[29] However, due to the small number of patients and the study design, researchers were unable to confirm the hypothesized mechanisms of action in the intestinal microbiome.

Table 4. Characteristics of the Anti-Inflammatory Diet

Definition	Loosely based on the Specific Carbohydrate Diet, this diet limits some carbohydrates, such as refined sugar, gluten-based grains, and certain starches that are thought to stimulate the growth of inflammatory bacteria in the digestive tract, and adds pre- and probiotics to help restore an anti-inflammatory environment.	
Limitations	Based on a small, retrospective case study. Further research is needed to determine changes in bacterial microbiota composition.	
	Include	*Avoid*
Grains	Oat	Gluten-based grains
Fruits	Most allowed if pureed and seeds are strained out	Fruits with seeds
Vegetables	Most vegetables with soft texture that are well cooked	Cruciferous vegetables during phase I and II
Proteins	All fish, lean meats, omega-3 egg	High-fat meats
Nuts, seeds, legumes	Flax meal and chia seed as tolerated, pureed nut and bean	Whole seeds and nuts
Dairy	Lactose-free, limited aged cheeses (made with active cultures and enzymes), fresh cultured yogurt, kefir	All others
Beverages	Not specified	Not specified
Other	Honey, stevia	Not specified
Reference	Olendzki BC, Silverstein TD, Persuitte GM, Ma Y, Baldwin KR, Cave D. An anti-inflammatory diet as treatment for inflammatory bowel disease: a case series report. *Nutr J.* 2014;13:5.	

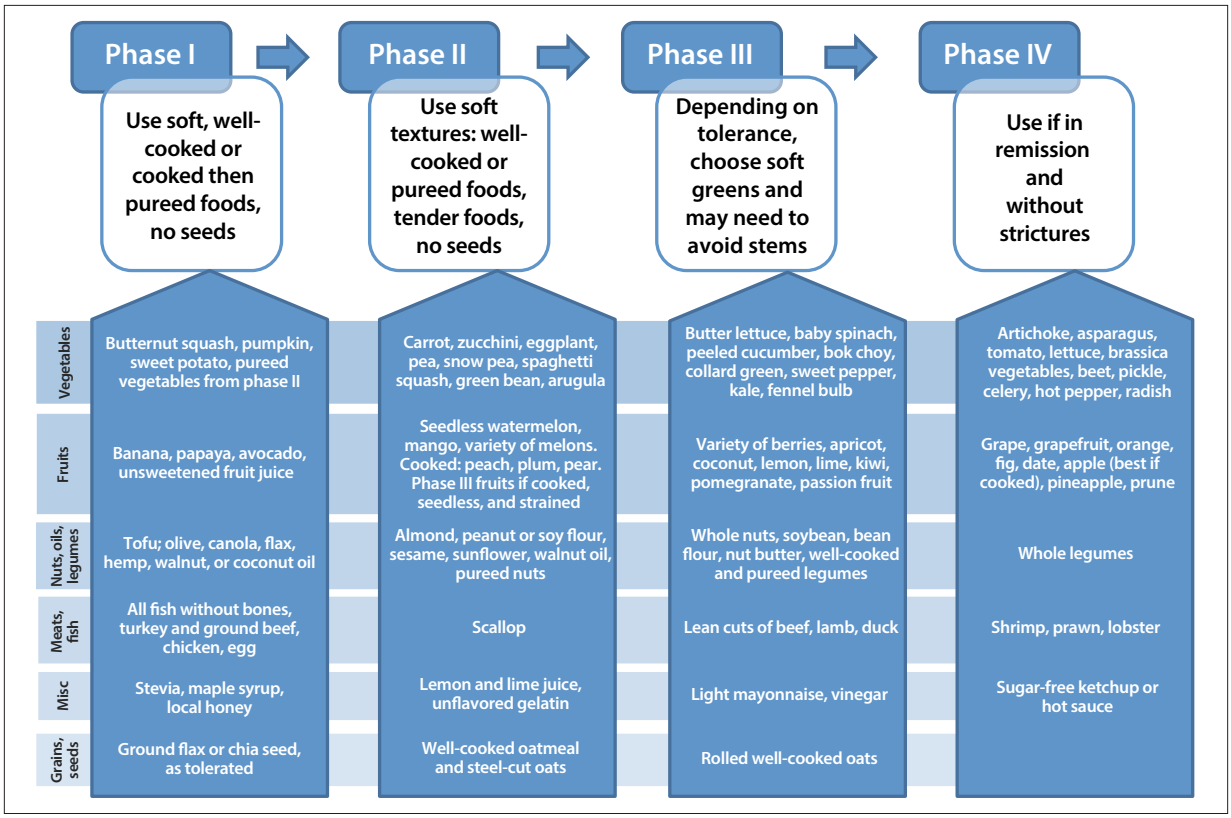

Figure 2. Food phase charts for the anti-inflammatory diet. Adapted from Olendzki et al.[29]

Animal Models to Study Diet

To further the quest for dietary recommendations to give to IBD patients, there has been an increase in studies with animal models to test different diets and specific nutrients. In humans and rodents, one of the largest impacts of diet is on the gut microbiome. In recent years, a growing body of evidence has shown that diet can alter the ecology of the intestinal microbiota. Analyses of fecal 16S rRNA gene sequences from humans and 59 other mammalian species living in zoos and in the wild indicate that the host diet influences intestinal bacterial diversity, increasing from carnivores to omnivores to herbivores.[3,30] Shotgun metagenomic sequencing has also established a functional evolution of the gut microbiome in relation to diet, with similarities among herbivores vs carnivores in microbial genes involved in metabolism.[31]

The High-Fat Diet

Consuming a Western diet, high in fat (particularly saturated fat), is enough to induce endotoxemia in healthy subjects.[32] These results suggest that even in a healthy state, this diet may be causing a leaky gut with increased permeability and changes in the microbiota, resulting in systemic low-level inflammation. Adherent-invasive *Escherichia coli* (AIEC) has been shown to be more abundant in the intestinal microbiota of patients with Crohn's disease compared with healthy controls.[33] In murine models, infection with AIEC results in a Crohn's disease–like phenotype, with transmural chronic inflammation and fibrosis.[34] Interestingly, patients with Crohn's disease have an abnormal expression of carcinoembryonic antigen–related cell adhesion molecule (CEACAM) 6, a receptor for AIEC, in the ileal epithelium.[35,36] On the other hand, ingesting a diet high in fat and sugar, mimicking the current Western diet, alters the composition of the intestinal microbiota. Wild-type mice and CEABAC10 transgenic mice that express human CEACAMs have greater dysbiosis after a diet high in fat and sugar.[37] In particular, CEABAC10 transgenic mice are more susceptible to infection by AIEC and have increased intestinal permeability, a decreased mucus layer, and an increase in tumor necrosis factor (TNF)-α secretion[37] after a diet high in fat and sugar. Another study showed that, upon switching to a high-fat diet, changes occur in the gut microbiota composition, including a decrease in *Bacteroidetes* and an increase in *Firmicutes* and *Proteobacteria*.[38] In humans, a long-term diet characterized by content high in animal protein and fats and low in carbohydrates, similar to a Western

diet, was associated with an increase in *Bacteroides* and a decrease in *Prevotella*.[39] However, a long-term agrarian diet (high in simple sugars) was associated with an enterotype dominated by *Prevotella*, a genus that is frequently observed in people from rural Africa.[39]

There are numerous studies indicating that the alteration in the microbial composition induced by changes in diet occurs rapidly.[40,41] Diet-induced alterations in the microbiota lead to demonstrable changes in bacteria-derived metabolites found throughout the host,[42] including changes in SCFA levels.[40] Bacterial-derived SCFAs modulate the immune system by promoting regulatory T cells[43] and energy abstraction.[44] GPR43, a receptor for SCFAs that has been identified on colonic neutrophils, directly connects host sensing with aspects of the microbiota that are altered in different dietary conditions.[45]

A high omega-6 to omega-3 ratio, as is found in today's Western diet, promotes the pathogenesis of many chronic diseases, including cardiovascular disease, rheumatoid arthritis, and IBD. The past century has been witness to large increases in dietary omega-6 fatty acids because of an overreliance on vegetable oils, such as corn, safflower seed, and cottonseed. The modern standard American diet (SAD) typically contains a ratio of 20-30:1 of omega-6 to omega-3 fatty acids, as opposed to the traditional ratio of 1-2:1.[46] Omega-6 fatty acids tend to be proinflammatory,[47] whereas omega-3 fatty acids have strong anti-inflammatory effects by suppressing interleukin (IL)-1βa, TNF-α, and IL-6.[47,48] A study using IL-10 knockout mice (mice that spontaneously develop colitis) demonstrated significantly reduced colonic inflammation when mice were fed omega-3 polyunsaturated fatty acid (PUFA)-rich fish oil, as compared with mice that were fed omega-6 PUFA-rich corn oil.[49] Because inflammation is the basis of many chronic diseases, dietary intake of omega-3 fatty acids is desirable over omega-6.

In another study using IL-10 knockout mice, a high-fat diet containing 37% of calories from saturated milk fat increases the rate of colitis and alters the gut microbiota, promoting the expansion of sulfite-reducing bacteria, which are found in low amounts in homeostatic conditions, and Th1 proinflammatory cytokines.[50] Moreover, a high-fat diet decreased gut microbiota richness and diversity in C57BL/6 mice.[50]

Animal meat and the SAD contain a high amount of saturated fat. Saturated fats have previously been shown to induce NF-κB activation in macrophage and adipocyte cell lines.[51-53] Immune activation has been shown to be mediated through toll-like receptor 4 (TLR4) and is abolished in TLR4-/- cells.[53] Diets high in saturated fat have also been shown to worsen colitis induced by dextran sodium sulfate in animal models.[54-56] The administration of a high-fat diet in mice has been shown to cause a change in the gut microbiome.[57] Some studies have shown that intestinal TNF-α was increased in mice fed a high-fat diet, which preceded weight gain and increases in fat mass.[58,59] In mice with a genetic susceptibility to colitis, feeding a high-fat diet (60% of calories from fat) was associated with more severe clinical and histopathologic inflammation and increased expression of inflammatory markers.[51] Other studies have found increased NF-κB expression in epithelial cells and an increase in certain lymphocyte populations in the intestinal tissue after a high-fat diet.[54] These inflammatory events occurred in the intestine before the appearance of increased serum cytokines. This provides evidence that diet alone can induce local intestinal inflammation in the absence of obesity and prior to the elevation of systemic markers of inflammation. Additionally, a high-fat diet has been shown to decrease the expression of intestinal tight junction proteins, including zonulin-1,[60] occludin, and claudin-1.[61] Increased intestinal permeability through the loss of tight epithelial junctions may contribute to increases in systemic lipopolysaccharides observed in obese patients. Furthermore, giving a high-fat diet to $TNF^{\Delta ARE/WT}$ mice, a Crohn's disease ileitis model characterized by a gene deletion that results in high levels of circulating TNF-α, accelerates disease onset and worsens inflammation compared with wild-type counterparts.

Dietary Carbohydrates

The effect of dietary carbohydrates has been extensively studied, especially given that the current Western diet is also high in sugars. Hou and colleagues recently examined the literature and found that diets high in monosaccharides and disaccharides increase the risk of Crohn's disease.[3]

Dietary fibers, or polysaccharides, have been the object of dispute among groups advocating for a low-fiber diet in IBD patients, specifically those with Crohn's disease. However, recent studies have demonstrated that a diet high in fiber is beneficial to both patients with ulcerative colitis and Crohn's disease[62,63] and decreases the incidence of the disease.[3] Dietary fibers can be divided in 2 categories: fermentable and nonfermentable fibers (eg, cellulose, lignins, waxes, resistant starch). Fermentable fibers, such as pectins, beta-glucans, beta-fructans, gums, inulins, oligosaccharides, and dextrins, are fermented by the gut microbiota, producing lactate, SCFAs (ie, acetate and butyrate), and gas. Fermentable and nonfermentable fibers have anti-inflammatory effects in chemically induced colitis in rats.[64,65] Cellulose, an insoluble and nonfermentable polysaccharide present in most vegetables and fruits, has been found to serve as a trophic factor for colonocytes.[66] Interestingly, despite not normally being fermented by the gut microbiota, cellulose has been shown to be able to modify the

microbiota composition. In addition, giving cellulose early in life decreases mucosal inflammation in chemically induced and spontaneous colitis in adult mice and increases richness in gut microbiota.[67]

Currently, prebiotics are described as nondigestive food ingredients (mostly oligosaccharides) that have a specific stimulatory effect upon select populations of gut bacteria.[68] Some studies performed in rodents have previously shown that supplementation with prebiotics such as inulin or lactulose helps to restore balance in the gut microflora, resulting in decreased mucosal inflammation in chemically induced colitis.[69-72]

Supplements and Additives

The use of dietary supplements is very common in modern society, thus making them a target of constant screening. Several studies have shown the benefits of vitamin D supplementation. The administration of vitamin D ameliorates dextran sodium sulfate–induced colitis,[73] reducing bacterial translocation to mesenteric lymph nodes and colonic lamina propria, decreasing permeability, and increasing the expression of tight junction proteins. These results were corroborated in vitro using the intestinal epithelial cell line caco-2.

Oral administration of iron sulfate has been shown to cause intestinal inflammation and gut dysbiosis in mice. The mechanism behind this observation stems from the fact that iron catabolism increases oxidative stress, potentially causing cellular injury and ultimately promoting inflammation through NF-κB activation.[5] In addition, heme dietary iron, which is present in meat, worsens chemically induced colitis in mice[74] and rats.[75]

In a recent study, Chassaing and colleagues reported that administration of the emulsifiers carboxymethylcellulose and polysorbate-80, which are very common food additives, results in low-grade inflammation and metabolic syndrome in C57BL/6 wild-type mice, as well as severe colitis in genetically susceptible IL-10 knockout mice.[76] This study also showed that exposure to these emulsifiers disrupts intestinal biofilm, decreasing the thickness of the mucus layer and the distance between the intestinal epithelium and the microbiota. In addition, treatment with emulsifiers increased intestinal permeability in IL-10 knockout mice.

Conclusion

The roles of diet and nutrition are major concerns of patients with IBD. Patients often have avid beliefs about the role of diet in their underlying disease as well as the source for either exacerbating or relieving their symptoms. There is scientific evidence from animal models and epidemiologic studies that dietary factors may influence both the risk of developing IBD and intestinal mucosal inflammation. However, the role of dietary interventions in the management of IBD still needs to be tested vigorously in patients. There is a need for large prospective, controlled trials to provide the appropriate dietary recommendations that patients desire instead of leaving them to seek nonmedical resources for dietary guidance. In the meantime, several of the diets that are popular, especially the low-FODMAP diet and the IBD-AID, seem to be reasonably balanced and sound without causing weight loss in ill IBD patients.

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