TOURO COLLEGE & UNIVERSITY SYSTEM

The Science Journal of the Lander College of Arts and Sciences

Volume 13
Number 1 *Fall 2019*

2019

The Role of Diet in Inflammatory Bowel Diseases

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Abstract

Inflammatory Bowel Disease encompasses two diseases, Crohn's Disease and Ulcerative Colitis. During the last 70 years the evolving of diet in the industrialized countries has led to the utilization of foods that have been processed rather than those in their more natural original state. This increase in the processing of foods has been correlated with the more recent occurrence of metabolic diseases such as Inflammatory Bowel Disease. Patients suffering from this problem exhibit lesser amounts of anti-inflammatory bacteria such as Roseburia and Faecalibacterium and increased amounts of pro-inflammatory bacteria Escheria and Fusobacterium. Since diet directly alters the composition of the gut biome, this review aims to define the role of diet in the pathogenesis and management of Inflammatory Bowel Disease.

Abbreviations:

IBD – Inflammatory Bowel Disease
CD – Crohn's Disease
UC - Ulcerative Colitis

Introduction

What is Inflammatory Bowel Disease?

Inflammatory Bowel Disease (IBD) encompasses two diseases, Crohn's Disease (CD) and Ulcerative Colitis (UC). CD is characterized by inflammation of the digestive tract anywhere from mouth to anus. In CD inflammation may reach through many layers of the walls of the gastrointestinal tract causing severe tissue damage. However, UC occurs when there is an inflammation of the colon and the rectum. In UC, the inflammation only affects the innermost layer of the lining of the colon. There are many symptoms for CD and UC. Some of the more common ones include diarrhea, abdominal pain, cramping, blood in stool, reduced appetite, unintended weight loss and severe bowel constriction (Conners, et al., 2017). Many times, inflammation is so severe that the intestinal tissue is damaged beyond repair. In the event of such inflammation the damaged tissue is removed via colectomy. IBD tends to affect individuals of all ages. However, IBD is most commonly found in individuals at a young age, interfering with education, work ability, social interaction and basic quality of life. Therefore, researchers feel compelled to find resolutions for this disease (Lovasz, et.al., 2013).

How the Diet has Changed

Over the centuries the western diet has changed incredibly. In the traditional diet, food was produced and consumed shortly after harvest. In contrast, much of today's food supply is processed, modified, and transported over great distances before being ingested. In recent years, the western diet has been dominated by an increased consumption of refined sugars, omega-6 polyunsaturated fats and fast food, in addition to a deficiency in fruits, vegetables and fiber. The western diet is prevalent in industrial countries such as the United States, Canada, Western Europe, Australia, and New Zealand, and has been spreading and reaching newly developing countries as well (Lovasz, et.al., 2013). This change in diet has led to the worldwide development of many metabolic diseases, such as IBD. (Ng, 2013) (Moschen, 2012). Recent studies indicate that the incidence rate of IBD in developing industrial countries has been increasing in the past 50 years. This increase suggests that IBD may be triggered by an environmental factor (Martinez & Chang, 2013). Although many studies have been done, there is still no definite pathogenic pathway nor cure for this disease.

Recent research suggests that diet is the key environmental risk factor in IBD pathogenesis. IBD is an intestinal disease and is associated with a dysbiosis in the gut microbiota, the microbe population found in the intestine (Reddavide, et al., 2018). In general, IBD patients have been found to have lower amounts of anti-inflammatory bacteria such as *Roseburia* and *Faecalibacterium* and have increased amounts of pro-inflammatory bacteria *Escheria* and *Fusobacterium* (Aleksandrova, et.al., 2017). Many studies have shown that one's diet directly alters microbe composition, making diet a pivotal risk factor of IBD. Moreover, changes to the traditional diet of newly industrialized countries can be linked to the increasing incidence of IBD. The purpose of this review is to define the role of diet in the pathogenesis and management of IBD.

Methods

Critical analysis of peer reviewed articles and clinical research papers were used to write this review paper. Articles were obtained from the Touro College online library in order to determine the relationship between diet and IBD.

Discussion:

IBD and Gut Microbes

The gut microbiome is an ecosystem, often referred to as a Super Organ (Dolan, 2017). The microbes live in a symbiont relationship with the host, performing beneficial tasks such as nutrient production, development and maturation, regulation of immune system and preventing the growth of harmful microorganisms (Aleksandrova, et.al. 2017). Dysbiosis, which is an alteration to the gut flora, can cause disruption to these vital tasks. Several studies show that IBD is correlated with altered composition of gut microbes in the intestines.

Interestingly, Studies have shown, that there is an age-related variation of IBD onset with three distinct stages. Early onset is less than 10 years of age, with a peak onset at 15-30 years old, and late onset cases occur at 60 years old. These changes correspond to the change in the microbiome's stability and diversity. Early life is associated with a microbiome of minimal complexity and stability and can be affected by diet change, illness and puberty.

As individuals reach adulthood, the microbiome stabilizes and shows improved resilience, but the composition may be altered. Correspondingly, decreased stability is often observed in the elderly (Kostic, et.al., 2013). Considering this, the microbe composition and stability in the gut is a key factor in IBD progression.

One role of the microbiota is to maintain a healthy mucosa by the production of anti-inflammatory interleukins. This is the production of SCFA- short chain fatty acids such as butyrate, succinate, lactate, phenols, thiols, and indoles (Reddavide, et al., 2018). These SCFA act as energy for colonocytes which protect against inflammatory responses (Frank, 2007). Patients suffering from IBD have reduced levels of beneficial SCFA producers *Bacteroidetes*, *Lachnuspiraceace,* and *Ruminococcaceae* (Martinez & Chang, 2013). An Additional study shows that IBD patients show reduced levels of several other SCFA producing bacteria such as *Calibacterium praunsnizti* and *Roseburia* (Dolan, 2017). This shows a direct link between dysbiosis and IBD.

Another task of the gut microbiome is to maintain the mucus layer of the colon and the small intestine. If the mucus layer is intact the gut flora will not directly interact with the epithelial cells and inflammation will not occur. However, there is an adherent invasive strain of *E. coli* that is found in abundance in IBD patients compared with controls. These invasive bacteria can potentially invade epithelial tissue and induce granuloma formation during inflammation. A second group of invasive bacteria *Fusobacteria* is found in abundance in the colon mucosa of IBD patients. *Fusobacterium varium* have been found to cause colon mucosa erosion, indicating the possibility of the influence of *Fusobacterium varium* on the pathogenesis of IBD (Kostic, et. al. , 2013).

Bile Acid signaling is another point where host, microbe and even dietary factors all come together. Bile acids assist in digestion and emulsification of fats to be absorbed in the intestines. These bile acids are made in the liver from cholesterol. After undergoing conjugation to glycine or taurine, the bile acids are released in to the digestive tract. In the intestines, deconjugation of bile acids occurs through Bile Salt hydroxylase (BSH) enzymes, making the bile acids functional. These BSH enzymes are made exclusively by bacteria, making the bile acid composition in the intestine dependent on the microbiota composition. The two products of bile acid metabolism act as ligands for FXR (Farnesoid X receptor) and TGR5 (G protein-coupled bile acid receptor 1), which act as signals for anti-inflammatory and barrier function. It was found that IBD patients have increased levels of conjugated bile acids compared to the control group, making these patients prone to inflammation. Furthermore, when bile acids are increased in the gut, it can promote growth of pathobionts such as *Bilophila Wadsworth*, which is known to induce inflammation (Dolan, 2017).

On the other hand, many microbes perform protective tasks when enriched in the host. *Bacteroides* and *Clostridium* species have been shown to cause expansion of T-cells and reduce inflammation. Additionally, many bacteria can reduce intestinal inflammation by regulating nuclear factor (NF)κB activation. *Faecalibacterium praunsnizti* has anti-inflammatory properties and is underrepresented in patients with IBD. Many species, such as *Bifidobacterium, Lactobacillus* and *Faecalibacterium,* protect the host from mucosal inflammation by down regulating inflammatory cytokines or stimulating IL-10 anti-inflammatory cytokines (Kostic, et. al., 2013). Lastly, intestines rich in *Bacteroidetes* are shown to initiate host responses that protect the host from lethal infectious colitis (Brown, et. al., 2012).

Due to this connection between IBD and dysbiosis, there are many current treatments for IBD that are directed at the microbiota. Many antibiotics such as ciprofloxacin, metronidazole, the combination of both rifaximin and anti-tuberculous regimens are used to treat IBD (Nitzan, et.al., 2016). Normally antibiotics may cause thinning of the mucus layer, weakening the barrier function, leading to increased gut infections. Additionally, antibiotics tend to cause an increase in E. coli composition in the gut, which is a distinctive feature in intestinal inflammation. However, studies show that brief exposure to antibiotics causes slight changes to the microbiota in IBD patients that provides relief from symptoms. Alternatively, repeated exposure of the same antibiotic causes persistent changes to the microbiota leading to infection (Kostic, et.al., 2013). Due to this controversy of antibiotics, in the last few years methods to repopulate the gut with healthy beneficial microbes has been gaining popularity as a potential treatment for IBD. Evidence shows that Fecal Microbiota Transplantation (FMT) can be effective in replenishing a healthy microbiota. FMT, also known as a stool transplant, is the transplantation of gut microbes from the stool of a healthy individual into the recipient. There has been a high success rate for FMT in many other metabolic diseases such as relapsing *C. difficile* infection. Therefore, researchers are considering FMT for other gastrointestinal illnesses such as IBD. Additional studies are needed to determine the outcome of FMT on IBD patients (Kostic, et.al., 2013).

It is now clear that IBD is inherently linked with dysbiosis. There are many factors that can cause dysbiosis such as genetics, immune function, and diet. Genetic susceptibility is the most obvious factor. IBD patients carrying a mutation in a NOD2 related gene, have increased numbers of mucosa adherent bacteria, as well as significant alterations in bacterial composition (Kostic, et. al., 2013). Studies have shown that CD is common in the Ashkenazi Jewish population. Many studies were done using large families of Ashkenazi Jews to determine a genetic variant cause to CD. One study found the haplotype 268SJW1 on chromosome 16 to contribute to the risk of CD in Ashkenazi Jews (Sugimura, 2003). Although genetic susceptibility cannot be ignored, due to the increasing incidence rates of IBD, researchers believe that environmental factors are the core triggers.

Dietary Factors effecting the Microbiome

Diet is suspected to be the prime contributor to the pathogenesis of IBD. Dietary composition has been proven to be associated with intestinal inflammation by directly or indirectly modifying the gut microbiome (Reddavide, et al., 2018). So, current research is aimed at examining how diet changes the microbiome and how these changes affect the host, in order to determine foods that can cause or prevent inflammation (Kostic, et.al., 2013).

In a study done, subjects were put on a western-style diet for one month and showed a 71% increase in plasma endotoxins. The western diet is characterized by a high fat diet and therefore may cause endotoxemia by causing changes in the intestinal barrier function or microbiota composition (Pendyala, et.al., 2012). Another study showed that mice fed a high fat diet were associated with endotoxemia and systemic inflammation. These mice showed lowered amounts of *Bifidobacterium spp*, indicating that this species of bacteria have anti-inflammatory properties (Cani, 2007).

Variations in long term diet patterns change the ratios of *Bacteroides, Prevetolla,* and *Firmicutes*. However, modifications in short term diets have no significant influence. It has been determined that enriched protein and animal fat diets, common in the western diet, favored *Bacteroides*. Diets rich in carbohydrates, such as in agrarian societies, favored *Prevetolla* (Wu, 2011). Switching to a high fat diet causes a decrease in *Bacteroides* and an increase of *Firmicutes* and *Proteobacteria* (Hildebrandt, 2009). It has been proven that protein rich diets increase enzymes that produce toxic metabolites which trigger inflammatory response (Brown, DeCoffe, et.al., 2012).

Another microbe often associated with IBD inflammation is *Bilophila Wadsworth*, a sulfite producing bacteria. In a recent study when mice were fed a milk fat enriched diet, it led to a series of reactions that ultimately led to an abundance of *Bilophila Wadsworth*. Although there is no precise mechanism for how it causes inflammation, there are many hypotheses (Frank, 2007). Furthermore, an interesting study concluded that refined dietary glucose intake can change microbe composition and increase intestinal inflammation (Whitehead, 2011). A strict Vegan or Vegetarian diet led to a significant reduction in *Bacteroides spp, Bifidobacterium spp,* and the *Enterobactericeae* which helps manage inflammation of IBD (Enck, 2011).

A further connection between diet, microbes, and host inflammation has been discovered through studies of the intestinal function of the aryl hydrocarbon receptor (AhR). AhR is a nuclear receptor that activates certain metabolic genes. AhR also plays a key role in adaptive immunity through the regulation of T-cell activity. IBD patients have a decreased expression of AhR which may cause pro-inflammatory immune activation conditions. It has been discovered that certain gut microbes can produce AhR agonists. IBD patients contain less amounts of the microbes necessary to produce these agonists. An important dietary source of AhR ligands are found in cruciferous vegetables such as cauliflower, broccoli, brussels sprouts and bok choy. This may explain why these vegetables have a protective effect on IBD risk (Dolan, 2017).

One major role of the microbiota is to act as an immune system of the host. The gut flora protects the host from pathogens by maintaining the integrity of the intestinal mucosa. Dysbiosis can cause a disruption in the intestinal mucosa and allow access for pathogens which can lead to reactions which cause inflammation and eventually tissue damage. Many studies were conducted to determine the influence of micro and macro nutrients on immunity. Many micronutrients are essential for immunonutrition such as vitamins A, C, D, E, folic acid, zinc, and iron (Reddavide, et al., 2018). Deficiencies in vitamins A and D may reduce function of natural killer cells, while supplemental zinc and vitamin C can increase their activity. Vitamin D is important in intestinal function for it is known to suppress microbial invasion into the epithelium. 82% of IBD patients have been found to have vitamin D deficiencies which have been linked to a weakened epithelial barrier. Although studies have been done to determine the effects of vitamin D supplements on IBD patients, more research is needed. Furthermore, Iron deficiencies have been associated with faulty T cell response and weakened cytokine production. However, iron supplements have been shown to have a negative effect on IBD patients by increasing an individual's susceptibility to infections. It is evident that micronutrients can affect the immune system thereby causing dysbiosis and the onset of IBD (Aleksandrova, et.al., 2017). More research is needed to recognize optimal nutrient levels and therapeutic options.

These findings support the fact that the microbiota is directly related to food quality. Diet influences the composition of the microbiota and thereby controls IBD pathogenesis. The current diet is significantly different than the diet of previous generations when the prevalence of IBD was not as widespread. Countless experiments were performed to see the effects of diet on IBD. Dietary factors can have a negative effect on IBD and increase inflammation, or they can have a protective or even healing effect on patients with IBD.

Dietary Risk Factors of IBD

Refined and processed carbohydrates are risk factors for IBD, but complex carbohydrates, such as fruits, vegetables, or fibers can help manage IBD. A diet high in animal protein was correlated with a 3.3-fold increased risk in IBD (Reddavide, et al., 2018). Individuals that consumed milk products including milk, yogurt and cheese, had a lower chance of developing IBD. This suggests that dairy intake has no risk effect on IBD and may even have a protective effect (Malavia, 2017). Many studies were done on the effects of fat intake on IBD. Although it is known that a typical high fat western diet has a negative effect on IBD, it is unclear which specific fats cause that. One study showed

that an increase in poly-unsaturated fatty acids was positively associated with IBD (Parekh, 2015). Another study concluded that long term intake of trans-unsaturated fatty acids is associated with a trend towards IBD. Evidence shows that excessive consumption of omega-6 poly-unsaturated fatty acids increases the risk of IBD, while consumption of omega-3 poly unsaturated fatty acids is associated with a decrease in one's risk for the onset of the disease (Brown, et. al., 2012).

Dietary Management in IBD

Interestingly, research has shown that the Mediterranean Diet, due to its unique balance of fat, is directly correlated with decreased inflammation. The Mediterranean Diet (MD) is characterized by fruits and vegetables rich in fiber, vitamins, and antioxidants, whole grains, and nuts, as well as olive oil and oily fish that are high in poly and mono unsaturated fatty acids. Each item in the diet provides benefits to patients with IBD (Serra-Majem, 2009).

Legumes, a major part of the MD, contain soluble fibers that do not aggravate the gut microbes. Furthermore, legumes tend to have a prebiotic action and promote the growth of beneficial SCFA producing microbes which protect the gut mucosa. Fruits and vegetables have a naturally high fiber content and therefore using a juicer to extract the essential vitamins and minerals showed best results when treating IBD patients. Recent studies claim that olive oil has ani-inflammatory effects. This is due to the synergic action of its oleic acid with other antioxidant molecules. Grains that were proven to have the most beneficial effect were unmodified grains. These grains have low immunogenic impact and do not cause inflammation in the gut mucosa. Bluefish, which is popular in the MD, is shown to have anti-inflammatory effects due to its omega-3 fats EPA and DHA (Reddavide, et al., 2018).

An alternative diet that has shown promise in IBD management is the Specific Carbohydrate Diet (SCD). This diet works with the hypothesis that patients with IBD can not break down disaccharides and amylopectin. Large amounts of disaccharides can lead to an over growth of microbes and cause IBD related symptoms. Therefore, the SCD is a strict monosaccharides diet excluding disaccharides and most polysaccharides. The diet includes vegetables with a high amylose to amylopectin ratio. The real potential for this diet is in the maintenance of IBD patients in remission to help maintain a healthy gut microbiome (Reddavide, et al., 2018). A similar diet the FODMAP – Low Fermentable oligosaccharide disaccharide monosaccharide and polyols diet also showed significant improvement of symptoms in patients with IBD (Dolan, 2017). Although these findings clearly define the effects of diet on IBD, the role of diet does not stop there.

Dietary Factors and Epigenetics

New emerging evidence points to epigenetics as a major component of IBD pathogenesis. Epigenetics is the study of heritable changes in gene expression that do not involve changes in the actual DNA sequence. This leads to a change in phenotype without a change in genotype. Epigenetic modifications, such as histone modifications, influence DNA accessibility and chromatin structure, thereby effecting the regulation of gene expression. The body uses epigenetic modifications as natural ways to control gene expression as needed throughout one's life. However, epigenetic changes can also be induced by external factors including age, the environment, lifestyle, and disease state. Dietary factors have been shown to contribute to IBD in an indirect manner via epigenetics (Reddavide, et al., 2018).

Over time, researchers have discovered new methylation patterns emerging for IBD. Many therapeutic studies were done to try to counteract this methylation using drugs and other processes. However, researchers found it difficult to pinpoint the methylation in specific tissue and to predict drug response in clinical trials so other strategies were considered. Dietary factors have been known to alter epigenetics which can influence an immune response that can potentially offset inflammation. Therefore, scientists looked at dietary components for a solution to IBD methylation patterns. It was discovered that secondary plant metabolites, such as polyphenols, have been demonstrated to inhibit DNA methyltransferase activity. Furthermore, gut microbiome has been shown to alter host histone acetylation and methylation in human colon tissues. Interestingly, SCFA, which is generally produced from microbial fermentation of fibers, is shown to be important in epigenetic regulation of inflammatory responses. It was determined that a lack of SCFA can disturb chromatin effects. This deficiency of SCFA can be a result of a diet low in fiber or a decrease in beneficial SCFA producing bacteria. As mentioned previously, IBD patients have been found to have low amounts of *Bacteroidetes*, *Lachnuspiraceace*, *Ruminococcaceae*, *Calibacterium praunsnizti,* and *Roseburia,* SCFA producing bacteria, making IBD patients susceptible to epigenetic changes which can lead to inflammation. (Reddavide, et al., 2018).

Other Environmental Factors

Although diet has been presumed to be the primary trigger for IBD, there are many other environmental factors that can prompt IBD symptoms. Smoking, and stress are such factors that have also been shown to influence IBD pathogenesis. Interestingly, Smoking has been found to be negatively associated with UC but positively associated with CD. Patients who did smoke only developed UC after they ceased smoking. Additionally, male UC patients who smoked during disease progression, reported reduced symptoms and had lower hospitalization rates compared to non-smokers. Increased colectomy rates were seen in non-smokers and ex-smokers than in current smokers. Also, reduced rates of relapse were seen in patients who began smoking after the onset of UC. On the other hand, many

studies show an increased risk of CD in smokers compared to non-smokers. There is a 34% increased relapse rate for CD in smokers. Furthermore, severe lesions at the anatomic sight have developed more in smokers than non or ex-smokers in CD patients (Thomas, et.al., 2000).

There are many theories for the possible mechanism of the influence smoking has on IBD. Firstly, due to the interrelationship of gut microbes and immunity, researchers studied how smoking influences the immune system. Studies show that changes in T cells, such as increased levels of suppressor OKT8+ cells and a decreased ratio of OKT4+ to OKT8+ cells have been seen in heavy smokers. Remarkably, these changes revert to normal range once a patient terminates smoking. The low levels of IgA's in the saliva and intestinal secretion of smokers dictates an additional immune effect. The IgA antibody is a vital component of secretions that lubricate the mucosal surfaces which act as the first line of defense in the immune system. Therefore, patients who smoke will already have a weakened immune system. Another possible mechanism involves the inflammatory pathway. Nicotine, a substance found in most cigars and cigarettes, is known to effect cytokine production in the body, specifically by reducing pro-inflammatory cytokines. Also, certain eicosanoids, which are correlated with inflammation, are seen in decreased amounts in smokers. This may be the cause to the negative effect of smoking on UC patients. Nevertheless, smokers show lower levels of two interleukins, IL-1β and IL-8, which help regulate inflammatory response. Lastly, smoking may also influence IBD via intestinal mobility. One major symptom of IBD is issues with bowel movement and defecation. It has been found that nicotine can affect the intestinal motility at many sites. Nicotine relaxes the smooth muscles of the intestine and therefore reduces contractile activity during bowel movement (Bhatti, 1997). These mechanisms portray the direct influence that smoking may have on IBD.

Another environmental factor that researchers have discovered to be effective in the pathogenesis of IBD, is stress related influences. In earlier years IBD was classified as a psychosomatic disorder. Although this classification had been long disproved, it does represent the deep relationship between stress and IBD. In general, stress acts as a threat to an organism's homeostasis. Therefore, the body will exert mechanisms to maintain equilibrium in the face of any stress. There are two interconnected pathways in the body that govern stress related responses; the HPA axis - hypothalamic pituitary adrenal axis and the ANS - autonomic nervous system. The pathway of the HPA axis begins when stress stimulates the release of corticotropin releasing hormone from the hypothalamus. This causes the release of adrenocorticotropic hormone from the anterior pituitary gland which then stimulates the secretion of cortisol from the adrenal cortex. Cortisol then travels throughout the blood stream causing changes all around the body to help deal with stress. The pathway of the ANS involves the stress induced activation of pontomedullary nuclei, which control autonomic responses. As a response to stress, adrenaline and noradrenaline are released from the adrenal medulla via the stimulation of the sympathetic nervous system. Furthermore, The HPA axis and ANS are key modulators of the rich nerve supply of the gut, the ENS – enteric nervous system. The ENS is composed of 100 million neurons that regulate functions of the gastrointestinal tract. The term "Brain-Gut Axis" refers to this complex network of neurons of the HPA axis, ANS, and ENS (Mawdsley, 2005).

The actual pathway of the Brain-gut axis response to stress is complex. Various interconnected activities occur in both the gut and around the body in response to stress that can lead to inflammation. Specifically, in the gut, in response to stress, inflammatory cytokines, SCFA, and microbial products can alter the ANS and in turn lead to the secretion of cortisol and adrenaline. Also, under stress, the gut microbiota and mast cells can release various chemicals such as histamine or serotonin which are known to impair the intestinal secretion and intestinal mucosa, which can lead to inflammation. There are many stressors that can affect the intestinal mucosa's permeability, making a host predisposed for infections and inflammation (Brzozowski, 2016).

Due to this intimate correlation between the nervous system and the gut, it is obvious that stress can influence the pathogenesis of IBD. Chronic heightened stress has been determined to have an important role in predicting the relapse of IBD in patients in remission (Mawdsley, 2005). Also, mice that were induced with depression demonstrated an increase in inflammation which was modulated by an increase in pro-inflammatory cytokines secreted by macrophages (Brzozowski, 2016). Although researchers have not determined an absolute direct pathway for the control of stress on inflammation, there are many suggestions. Many propose that patients with IBD have altered HPA axis functioning, which may explain the relation of stress to IBD symptoms. Interestingly, cortisol, which is released under stress, is known to have anti-inflammatory effects. However, in patients with UC, levels of cortisol showed no correlation to the levels of inflammatory cytokines. On the other hand, others hypothesized that due to the high levels of inflammatory cytokines in the blood of active IBD patients, the response of the HPA axis and thereby the response of cortisol may be dampened. Likewise, stress is known to impact gastrointestinal motility, and water and ion secretion. While these are non-inflammatory effects, they may contribute to the non-inflammatory symptoms of IBD patients (Mawdsley, 2005). The above mechanisms demonstrate the correlation between stress and IBD, making psychological factors a key influence in the pathogenesis of IBD.

Conclusion

The intestinal microbiota performs vital jobs in host metabolism making its composition vital to the host's health. Dysbiosis,

which is found in IBD, can be caused by several different factors. While genetics and other environmental factors cannot be ignored, an altered microbiota resulting from diet-induced dysbiosis may also be a factor that contributes to the inappropriate inflammatory responses that occur. As our understanding of the microbiota continues to grow, promoting microbes to prevent or control inflammatory-mediated diseases through diet may represent an exciting therapeutic avenue.

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