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EDITORIAL

Pro and anti-inflammatory diets as strong epigenetic factors in inflammatory bowel disease

Adele Rostami, Kristen White, Kamran Rostami

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Abstract

Inflammatory bowel disease (IBD) is the consequence of a complex interplay between environmental factors, like dietary habits, that alter intestinal microbiota in response to luminal antigens in genetically susceptible individuals. Epigenetics represents an auspicious area for the discovery of how environmental factors influence the pathogenesis of inflammation, prognosis, and response to therapy. Consequently, it relates to gene expression control in response to environmental influences. The increasing number of patients with IBD globally is indicative of the negative effects of a food supply rich in trans and saturated fats, refined sugars, starches and additives, as well as other environmental factors like sedentarism and excess bodyweight, influencing the promotion of gene expression and increasing DNA hypomethylation in IBD. As many genetic variants are now associated with Crohn's disease (CD), new therapeutic strategies targeting modifiable environmental triggers, such as the implementation of an anti-inflammatory diet that involves the removal of potential food antigens, are of growing interest in the current literature. Diet, as a strong epigenetic factor in the pathogenesis of inflammatory disorders like IBD, provides novel insights into the pathophysiology of intestinal and extraintestinal inflammatory disorders.

Key Words: Inflammatory bowel disease; Epigenetic; Anti-inflammatory diet; Immunogenetics; Microbiome; Polymeric diet; Elemental diet

Core Tip: This editorial highlights the implication of environmental factors, in particular diet, as epigenetic factors in pathogenesis of inflammatory bowel disease (IBD). The concept of epigenetic factors involved in the genesis of IBD brings new insight into the identified risk factors and future targeted approaches, as a guide to the prevention and treatment of IBD.

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INTRODUCTION

Epigenetic represent alteration in an organism's phenotype that persists through mitosis and meiosis[1], for example how the environment can change the way the body reads DNA sequences without changing the DNA itself. These factors are mainly involved in regulating innate and adaptive immunity, as well as maintaining intestinal epithelial barrier function [2]. The term "epigenetics" was introduced in 1942 by Waddington[3], to explain how a phenotype might be produced by the interaction between genes and environmental influences.

DIET AS EPIGENETIC FACTOR IN INFLAMMATORY BOWEL DISEASE

As highlighted in a study by Marangoni *et al*[4] published in *World J Gastroenterol*[4], DNA methylation is an essential remodeling process in the control of genetic information, which contributes to the epigenetics by regulating gene expression. DNA methylation and histone modifications are two central epigenetic mechanisms that impact gene transcription and cell fate. The authors emphasize the role of diet, gut microbiota composition, and exercise in activation and modification of epigenetic mechanisms through the individual's genetic inheritance[4,5]. An anti-inflammatory diet, by acting on gut microbiota composition, can induce phenotype changes through gene expression without changing the genetic sequence. Nutrition is a powerful convertible factor, acting directly on DNA methylation pathways. For example, diets deficient in methyl donors and proteins may cause global DNA hypomethylation, or high-fat diets may result in changes in DNA methylation[6].

Nutrition affects the epigenetic regulation of DNA methylation in several possible epigenetic pathways: Mainly, by altering the substrates and cofactors that are necessary for proper DNA methylation; additionally, by changing the activity of enzymes regulating the one-carbon cycle; and lastly, by playing a role in several possible mechanisms related to DNA demethylation activity[7].

Histone modifications are highly dynamic and respond to various environmental cues, such as dietary compounds, and have been found to alter the epigenome which impacts gene expression. A pro-inflammatory diet disrupts the balance between histone acetyltransferase and histone deacetylase activities, and when this balance is disrupted, it has a repressive action on the gene expression regulation network in cancer and inflammation[8,9].

Marangoni *et al*[4] also highlights the impact of non-coding RNA molecules that play a crucial role in the gene transcription and translation by non-coding RNAs[4]. Non-coding RNAs are RNAs not involved in protein translation, and they are divided into two categories by size, which include short and long non-coding RNAs[10]. They have essential roles in epigenetic modifications, regulating gene expression and chromatin remodeling. It is also envisaged that silencing of repeats in the genome is mediated by small RNAs[11].

In a study by Glória *et al*[12], DNA global hypomethylation profile[12] was increased in rectal mucosa of active and inflamed ulcerative colitis (UC) patients, supporting epigenetic and kinetic changes that might predispose these individuals to develop colorectal neoplasms[12]. This explains why inflammatory bowel disease (IBD) is at high risk for developing malignancy long term. Dysbiosis, following exposure to an inflammatory diet and other environmental factors, leads to the activation of IBD genes *via* hypomethylation and histone modification. Westernized dietary risk factors like deficiencies in micronutrients, and being rich in ultra-processed foods, additives, and emulsifiers[13] are implicated in reducing bacterial diversity and promoting an inflammatory response[4,14] (Figure 1).

The DNA methylation is a widely studied, heritable epigenetic alteration in animals, involving the covalent transfer of a methyl group to the C-5 position of the cytosine ring of DNA-by-DNA methyltransferases. Gene expression will inactivate, either because proteins bind to the methylated cytosine phosphate guanin island and initiate DNA compensation and inactivation, or methylation itself blocks the DNA sequence and transcription factors are unable to bind[15].

The consequences of dysbiosis are often systemic immune dysregulations in the form of pathogenic autoantibodies *via* activation of chronic inflammatory cells that ultimately result in a wide range of clinical manifestations, including skin rashes and arthritis in inflammatory conditions[16].

An emerging body of evidence suggests nutritional epigenetics as a novel mechanism underlying gene–diet interactions. The bioactive compounds of nutrients impart advantageous environments, such as homeostatic inflammation, through differential gene expression[17]. Dietary habits therefore shape the gut microbiota and influence the interaction with the immune system depending on the diet component. For instance, medical nutrition therapy using an elemental

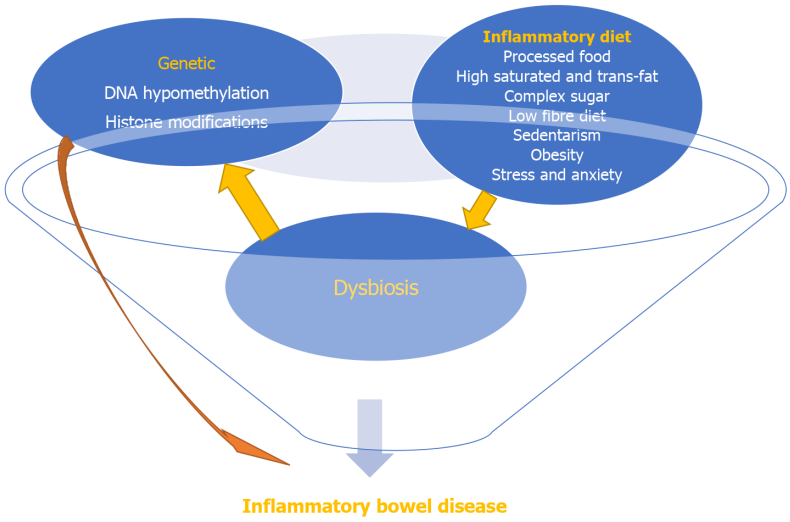

Figure 1 Epigenetic factors turning the inflammatory bowel disease genes on.

formula in conjunction with a LOFFLEX[18,19] diet in active Crohn's disease (CD), restores the intestinal microflora by preventing the growth of potentially pathogenic bacteria, thus promoting an anti-inflammatory action. However, this is not valid with polymeric formula due to its exponentially high fat (long chain triglycerides which is considered colitogenic)[20], refined sugar content[21] and other antigenic components, such as whole protein casein and soy[22], with colitogenic and antigenic properties[23]. Based on studies and evidence above, polymeric diet seems unsafe to use in active CD (Figure 2).

Environmental factors like industrialization, urbanization and antibiotic/nonsteroidal anti-inflammatory drugs usage are not only involved in the development of Inflammatory bowel disease, but also the course of prognosis and severity of IBD manifestation. These factors are implicated in rising incidence of CD in newly industrialized countries like Africa, Asia, and South America[24]. Environmental factors promote histone methylation or histone demethylation resulting in epigenetic modifications, that have the power to reduce or bolster gene expression, especially because of altering chromatin structure[2,25].

A histone is a protein that helps to comprise the structure of chromatin, which is composed of DNA-wrapped protein octamers[2]. Various amino acids on the histone tails, namely lysine, arginine, serine, and threonine, are epigenetically altered by enzymes, which then influences if a gene is accessible for binding by transcription factors and the RNA polymerase II machinery[15].

A rise in IBD in the Western world during the 20th century has been reported[26]. In Asia, Africa, and Latin America, few IBD were reported in the 20th century[26], but this has steadily increased during the 21st century[24] and does not appear to have peaked yet[27]. Environmental exposures associated with the westernization of societies are found to be the primary factor for these trends[28]; especially early life exposures that can alter the diversity, composition and function of intestinal microbiome that may lead to the development of IBD later in life[28].

In addition to diet, other environmental factors described in the literature include mode of childbirth, breastfeeding, urban environments, air pollution, and use of antibiotics/contraception and nonsteroidal anti-inflammatory drugs[29-31]. This may explain why one member of identical twins develops IBD and the other member may stay IBD-free despite sharing the same genetic background. Epigenetic modifications influenced by environmental factors, might help to understand the increasing IBD incidence.

Where patients have genetic susceptibility for IBD, modulating these environmental exposures can potentially prevent the development of IBD in the future[27].

For instance, diets high in fibre[32] and low in ultra-processed foods[13,33], exercise and mindfulness are all protective against gut inflammation and can potentially turn off the manifestation of disease in genetically predisposed individuals.

In a consensus statement produced by a group of experts from the Organization for the Study of Inflammatory Bowel disease, recommendations also include screening, at diagnosis and during flare-ups, for a patient's mental well-being and excluding psychosocial stressors and symptoms of depression and anxiety[34]. Regular physical exercise and healthy weight maintenance, as well as screening for obesity and nutritional deficiencies[34], are also advised. Tobacco smoke and long term, frequent use of high dose non-steroidal anti-inflammatory drugs should be avoided. Breastfeeding is encouraged[34], as breastfeeding for more than 12 months was found to be 7 times more protective against the development of UC[27]. As reported by Ng *et al*[24], a New Zealand study found breastfeeding to be protective against both UC and CD[24]. Evidence-based anti-inflammatory diets, in form of nutrition therapy of active IBD, should be encouraged[34], as this plays a significant role in removing possible food antigens with pathogenic potential in IBD and improves the long-term prognosis[18].

Westernized diets high in trans and saturated fats, refined carbohydrates and animal proteins are found to cause an imbalance in gut microflora with an increase in pathogenic bacteria[35]. Ultra-processed foods, additives and emulsifiers can also increase inflammatory mechanisms through increased intestinal permeability and a reduction in bacterial diversity[14]. In contrast, fresh, whole foods produce short chain fatty acid (SCFA) bacteria, that promote gut health and

Elemental	Polymeric
Simple amino acids – absorbed through simple diffusion → no antigen presentation to lamina propria	Whole proteins – in the form of **casein (milk)** and **soy** increases rate of transcytosis → **increase antigen load to lamina propria** → inflammation
Monosaccharides	**Polysaccharides - corn syrup *via*** depletion of luminal short-chain fatty acids
Lower fat content – makes up 2%-3% of calories	**Higher fat content** – makes up **30% of calories** (long chain triglycerides) ↑Faecal endotoxin ↓Mucin production
Restore the Microbiota	Colitogenic
Polymeric diet seems to be unsafe in active Crohn's disease	

Figure 2 Elemental *vs* polymeric.

protect against malignancy[14].

We should emphasize that similarly, high fibre diets also produce SCFA through the fermentation of dietary fibre by gut bacteria and have been shown to reduce inflammation, modulate gut microbiota, improve gut barrier function and thus protect against IBD[32,36].

Studies on smoking and antibiotic use in the western world compared to Asia, have demonstrated that variation of populations and ethnicity may make some people more vulnerable to certain environmental factors, whereas, in contrast, other environmental factors may be preventative in others[27].

CONCLUSION

Epigenetic mechanisms mediate the interactions between genome and environment. Pro and anti-inflammatory diets are strong epigenetic factors implicated in IBD pathogenesis and treatment. Future studies are needed to enrich our insight into the manipulation of environmental factors for the prevention and optimal treatment of IBD.

FOOTNOTES

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