

Introduction

During the evolution, the microbiome maintains a relationship of symbiosis with the gut environment. The intestinal mucosa immune system constitutes the largest immune component in vertebrates, functioning closely with the intestinal microbiome. The balance and homeostasis of the intestinal mucosa immune system plays an important role in the host homeostasis and defense.

Objectives

This work aims to study the relationship between microbiota diversity and the health of the immune system due to the special interest in cancer patients for a better response to treatment.

Methods

A revision of the literature about microbiota diversity, dysbiosis, immune system, modulation factors, precision medicine, cancer treatment, etc., was made using the main databases, pubmed and scopus.

Results

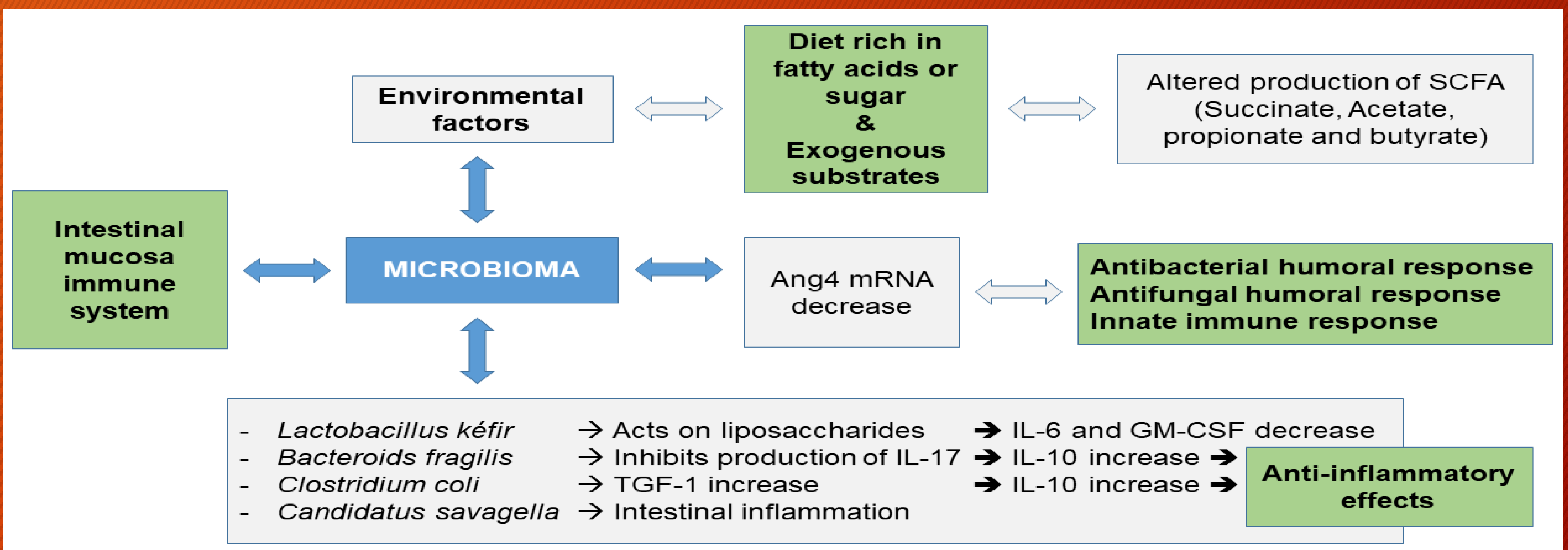
The intestinal microbiome plays an important role in the formation of mucosal immunity. A diet rich in fatty acids and sugar change the diversity and composition of the gut microbiome in mice, resulting in altered productions of acetate, propionate, succinate and butyrate by several type of bacteria in the gut. In healthy individuals, the microbiome mainly consists of 4 groups of bacteria: Bacteroidetes, Firmicutes, Actinobacteria and Proteobacteria. Some authors have reported that liposaccharides caused a significant decrease in the production of IL-6 and GM-CSF in mice treated with *Lactobacillus kefir*, indicating that this bacterium is an important factor in the inflammatory bowel malnutrition. *Bacteroids fragilis* is a symbiotic bacterium that can inhibit the production of IL-17 improving the activity of intestinal regulatory T cells (TREGS) to produce polysaccharide A with anti-inflammatory effects. Polysaccharide A regulates the conversion of CD4+ into Foxp3+ TREGS T cells in a TLR2-dependent manner. TREGS T then produces IL-10 anti-inflammatory to defend against inflammatory damage. The colonization of *Clostridium coli* cluster IV and XIVa increased the TGF-1 level and promoted Foxp3+ TREG T with IL-10 expression. Segmented Filamentous Bacteria, such as *Candidatus savagella*, are closely related with intestinal inflammation. Recent studies have shown that one of the important genes that regulate the pathogenesis of inflammatory bowel disease (IBD), member 9 of the caspase recruitment domain family, is responsible for mediating intracellular signals to trigger inflammation. In knockout mice for CARD9, the structure of gut microbes changes and they become susceptible to intestinal fungal infections, which indicates that the lack of CARD9 causes IBD malnutrition. In patients with mild to moderate colitis, IL-22 produced by ILC3 increases when exposed to excrement, suggesting that the gut microbiota is involved. The inhibition of SOCS3, MiR-19b regulates the intestinal epithelial cells to produce cytokines, thus inhibiting the inflammatory response and finally preventing the onset of Eosinophilic Colitis (EC). Changes in microbiota diversity were found in IBD, reflected in the decrease in symbiotic bacteria (such as Sclerotium and Bacteroides) and the increase in harmful bacteria (such as Proteus and Actinomycetes). Since gut microbiota can produce immunogenic substances, active bacterial products can regulate the inflammatory response of IBD. In this way, complex lipopolysaccharides on the cell surface of gram-negative bacteria can produce fecal immune responses. In some cases, immunogenic substances can pass through the intestinal wall, mainly when the barrier breaks down causing more damage. In addition, *Bacteroides fragilis* found in the human gut plays an important role in the regulation of the human immune system. Attacks of the gut microbiome can trigger autoimmune diseases. Some studies have shown that antinuclear antibodies, anti-bicatenary DNA and rheumatoid factor in systemic lupus erythematosus, and citrulline anti-protein antibodies in rheumatoid arthritis, can be detected prior to the onset of the disease, indicating the important role of the microbiome in the development of autoimmune diseases, mainly by *Porphyromonas gingivalis*. Recently, it was found that the proportion of ciliates/bacteroids in the intestine of Sweet' syndrome (STE) patients is reduced, indicating the low immune function of the mucosa. The analysis of RNA 16S have reported that *Lactobacillus* is increased in the fecal microbiota of patients affected of rheumatoid arthritis (RA). Moreover, Prevotella was found to be elevated in patients receiving long-term RA treatment. A significant reduction in purine synthesis was observed in these patients.

Conclusions

Gut microbiota is required for mucosal immunity. The intestinal mucosa is the main location for microbiome-host interactions. The dynamic interaction between microbiome and environmental factors modulate mucosal and systemic immunity. The absence of dysbiosis contributes to the good health of the immune system, leading to a better response to treatment in cancer patients.



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THE ABSENCE OF MICROBIOTA DYSBIOSIS IS ESSENTIAL TO MAINTAIN A HEALTHY IMMUNE SYSTEM IN CANCER PATIENTS