Chapter 15
Dysbiosis, Small Intestinal Bacterial Overgrowth, and Chronic Diseases: A Translational Approach

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ABSTRACT

Dysbiosis is characterized by an alteration in quantity and quality of intestinal microbiota composition. In the presence of dysbiosis, enterocytes will have difficulty in maintaining the integrity of the mucosal barrier, leading to increased intestinal permeability. These events are recognised to be linked to several chronic diseases. One of the consequences of dysbiosis is the manifestation of small intestinal bacte-

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rial overgrowth (SIBO), which is associated to a variety of chronic diseases. Single food nutrients and bioactive molecules, food additives, pre- and probiotics, and different dietary patterns may change the composition of the intestinal microbiota. Low FODMAPs diet has been a reference in SIBO treatment. This chapter intends to describe how the intestinal microbiota, dysbiosis, and SIBO can be related; to define dysbiosis food and nutrients influence; and to offer some nutritional therapy strategies for applying the low FODMAPs protocol, enabling better adherence by patients in order to increase their wellbeing.

1. INTRODUCTION

Microbiota corresponds to the community of microorganisms that inhabit a specific environment of the human body. It can be found in skin, genito-urinay tract, mouth and intestine. Each microbiota is composed of bacteria that varies not only according to its environment, but also throughout individual's life. Regarding to human gastrointestinal tract, there are approximately 100 trillions of bacteria, classified according to phyla, classes, orders, families, genus and species. There are more than 1000 different species identified [1]. They are clustered in six phyla, namely *Firmicutes, Bacteroidetes, Actinobacteria, Verrucomicrobia, Proteobacteria* and *Fusobacteria.* Approximately 60% of the bacteria belong to the *Bacteroidetes* and *Firmicutes* phyla [2, 3]. For each of these phyla, there are several classes of bacteria.

Gut mucosa consists of an external intestinal barrier and an inner immunological barrier. Intestinal barrier is composed by commensal gut microbiota, mucous layer and intestinal monolayer. It is responsible for two fundamental functions for the individual's survivance: allowing nutrients absorption and defending the entry of foreign molecules to the organism. The inner layer barrier consists in immune cells organized in Gut-associated lymphoid tissue (GALT). GALT depends on the dendritic cells and the M-cells present in the Payer's patches to interact with luminal antigens [4]. The interaction between commensal bacteria and mucosal immune system is essential for immune function.

Integrity of these structures is necessary for maintenance of normal intestinal barrier function. The microbiota produces bacteriocins and short-chain fatty acids (SCFA), including butyrate, acetate and propionate, which inhibit the pathogenic growth of microorganisms; and defensins, which control bacteriocins and SCFA. On the other hand, the mucosal immune system produces immunoglobulin A (IgA), preventing pathogenic bacteria from entering in the epithelium [5].

There is a mutual benefit between the microbiota and host organism during homeostasis. While prebiotics ingested by the individual are the necessary substrate for its growth, bacteria provide maintenance of mucosal barrier integrity; synthesis of vitamins B (B1, B2, PP, biotin, pantothenic acid, folate, and B12) and K, amino acids, neurotransmitters (e.g. serotonin) and SCFA; promote a better absorption of other vitamins and minerals; promote lymphocyte maturation; and prevent entry of pathogens [5, 6].

After being produced by bacteria, SCFA are released in the intestinal lumen, quickly absorbed and used as energy mainly by colonocytes, specially butyrate. In its turn, acetate and propionate may be carried into the bloodstream and become available to a variety of different organs [7]. SCFA regulate countless processes, regarding to appetite and weight management; inflammatory responses from immune system; lipid oxidation; and thermogenesis in brown adipose tissue. In fact, butyrate is crucial to