Review

Western diet components that increase intestinal permeability with implications on health

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Abstract: Intestinal permeability is a physiological property that allows necessary molecules to enter the organism. This property is regulated by tight junction proteins located between intestinal epithelial cells. However, various factors can increase intestinal permeability (IIP), including diet. Specific components in the Western diet (WD), such as monosaccharides, fat, gluten, salt, alcohol, and additives, can affect the tight junctions between enterocytes, leading to increased permeability. This review explains how these components promote IIP and outlines their potential implications for health. In addition, we describe how a reduction in WD consumption may help improve dietary treatment of diseases associated with IIP. Research has shown that some of these components can cause changes in the gut microbiota, leading to dysbiosis, which can promote greater intestinal permeability and displacement of endotoxins into the bloodstream. These endotoxins include lipopolysaccharides derived from gram-negative bacteria, and their presence has been associated with various diseases, such as autoimmune, neurological, and metabolic diseases like diabetes and cardiovascular disease. Therefore, nutrition professionals should promote the reduction of WD consumption and consider the inclusion of healthy diet components as part of the nutritional treatment for diseases associated with increased intestinal permeability.

Keywords: permeability, western diet, tight junctions, gut microbiota, gut

Introduction

The intestinal barrier can regulate permeability through various components, including the immune system, the enteric nervous system, and intestinal epithelial cells with their respective intercellular junctions and cytoskeletons. Also, the intestinal microbiota plays an important role in intestinal barrier and permeability integrity through the regulation of epithelial repair, metabolism and maintenance, and inflammatory responses [1]. However, this barrier must be permeable for certain substances essential for life.

Intestinal permeability is the physiological property that allows the selective entry of nutrients, water, and ions for their subsequent arrival in the bloodstream [2]. The molecules passage of the intestinal epithelium occurs through two pathways: the transcellular pathway (via intracelullar) involves the transportation of molecules through intracellular transfer, used by most dietary components like glucose, amino acids, fatty acids, vitamins, and some ions, and the paracellular pathway (via intercellular junctions) as a permeation of hydrophilic molecules, ions and water. These intercellular junctions, made up of a multiprotein complex known as tight junctions (TJs), anchor junctions, and desmosomes, can separate and allow the entry of small molecules [3]. In some cases, nutrients in high concentrations in the lumen can cross the epithelium through this pathway [4]. Nevertheless, the breaking of these protein junctions can lead to an increase in intestinal permeability (IIP).

An IIP implies an abnormal displacement of pathogenic microorganisms, antigens, and toxins [5], generating intestinal deterioration or signs of inflammation and intoxication [4], which could have an important association with some pathologies. For example, recent studies have reported a prevalence of IIP in 30% of people with type 1 diabetes, 34.3% with Crohn's disease, 10.5–42.9% with ulcerative colitis, 34.3% with systemic sclerosis 35% with cirrhosis, and others [6]. Some of these diseases are related to the presence of variants associated with IIP. For example, the PTGER4 gene variants for Crohn's disease, which is related to an anormal redistribution of TJ and the cytoskeleton [7]. However, 4.6 to 6.8% of healthy people have been shown IIP [6], which can be explained by other causes, not by a specific disease.

Some factors can cause IIP. For example, studies have reported that infections alter IP. *Salmonella* penetrates the tissues and causes a rupture in the TJ [8]. Stress is another

https://doi.org/10.1024/0300-9831/a000801