Inflammatory bowel disease (IBD) is a group of chronic inflammatory conditions with multifactorial and unexplained etiologies that includes two main forms: Crohn's disease and ulcerative colitis. Both forms of IBD are autoimmune diseases that result from an abnormal response of the immune system to the body's own tissues, but the exact cause is unknown. The diseases are thought to result from a combination of genetic, environmental and immunological factors and are characterized by recurrent and unpredictable episodes of exacerbations. In addition, there is no targeted treatment, so the goal of therapy is to treat symptoms, reduce inflammation and improve the lives of people with these diseases. It should be noted that more than 6.8 million people worldwide suffer from IBD, and the number of diagnosed cases is steadily increasing every year.

In IBD, damage to the intestinal barrier is observed as an important part of the disease. The intestinal barrier functions as an effective defense of the underlying tissues against toxins, bacteria, fungi and pro-inflammatory agents (endotoxins) present in the intestinal contents. When IBD is exacerbated, there is increased damage to the intestinal barrier, which in turn increases inflammation. In addition, when the intestinal barrier is impaired, endotoxins can reach various internal organs through the bloodstream and play a crucial role in the pathogenesis of non-intestinal diseases. The key elements are tight junction proteins, which determine the tightness of the barrier by regulating the permeability of the intestinal epithelium and transmitting signals between cells. A significant reduction in the expression of tight junction proteins is observed in patients with IBD and in animal models of inflammatory bowel disease. In addition, during IBD exacerbations, there is an increase in programmed intestinal epithelial cell death, which also reduces the tightness of the intestinal barrier.

Therefore, it is important to search for easily accessible nutritional factors that accelerate repair and/or protect against greater damage to the integrity of the intestinal barrier damaged by inflammation. This is especially important during periods of exacerbation, but also aims to accelerate and prolong the remission period. Among such potential agents are beta-glucans from oats, the polysaccharides consisting of D-glucose molecules linked by specific glycosidic bonds, which gives they linear structure. Due to their specific structure and belonging to the dietary soluble fiber fraction, beta-glucans exhibit many biological activities, such as anti-diabetic, prebiotic, cholesterol-lowering and immunomodulatory properties. The results also indicate that the mechanisms and strength of activity depend on the molar mass of these polysaccharides. There are no reports in the world literature on the molar mass-dependent effects of oat beta-glucans on the integrity of the intestinal barrier altered by inflammatory bowel disease, especially considering the different stages of IBD development. In light of the above, it is extremely valuable to demonstrate that a simple dietary intervention using oat beta-glucans can effectively improve the tightness of the intestinal barrier damaged by inflammation.

The main objective of the planned research will be to assess and verify the impact of dietary administered low or high molar mass oat beta-glucans on integrity of intestinal barrier damages by chemically induced *colitis.* The impact will be studied at three time points of the inflammation development including exacerbation and remission periods. An analysis of the intestinal barrier integrity markers expression will be performed on colon samples frozen and stored at low temperature.

In colon samples, obtained previously from an in vivo animal model experiment, the mucosal cells expression of selected tight junction proteins will be determined by immunohistochemistry and Western blot technique. The intensity of programmed colon epithelial cells death will be also assessed.

The results of the planned study will indicate whether oat beta-glucans normalize the integrity of the intestinal barrier damaged by inflammation, and what are the difference in this effect depending on the molar mass of these polysaccharides.

The results of planned project are extremely important from social dimension point of view, not only in the context of the increasing incidence of IBD associated with the intestinal barrier damage, but also in the context of other intestinal disorders in which disruption to the intestinal barrier is also observed.