The aim of the project is to assess the onco -BCG vaccine in terms of the possibility of limiting the development of infections caused by *Helicobacter pylori* (*H. pylori*) bacteria, neutralizing the inflammatory reaction in the gastric epithelium, induction of the first line immune cells and inducing repair processes. In the research we will use BCG-onco vaccine bacilli.

Gram-negative *H. pylori* rods, described in 1983 by Warren and Marshall, colonize the gastric epithelium of humans. On average, about 50% of the population is infected with these bacteria, although the infection rate can be as high as 80-90%. About 20% of *H. pylori* carriers develop chronic gastroduodenal inflammation, ulcers, and even cancer. If the infection is not treated with two or more antibiotics combined, the infection becomes chronic. Its effects depend on the susceptibility of the host, virulence factors of the pathogen, and environmental conditions. A typical symptom of infection is an excessive inflammatory reaction which results in the impairment of the barrier function of the gastric epithelium. Erosions promote the movement of toxins and components of *H. pylori*, as well as soluble mediators of inflammation into the epithelium, contributing to the development of systemic inflammatory reaction.

The increasing antibiotic resistance of clinical isolates of *H. pylori*, as well as the high frequency of infections in the population, pose a real threat to people susceptible to symptomatic infection with these bacilli. A correlation has been demonstrated between the increase in *H. pylori* infections in the population and the increased frequency of isolation of antibiotic-resistant strains. For this reason, new biologically active preparations are sought to assist in the treatment of such infections and eliminate their negative health effects locally and peripherally.

Our previous research shows that these bacteria destroy gastric epithelial cells inducing the process of apoptosis, as well as inhibit the activity of immune cells, which allows for permanent colonization. Therefore, we plan two-way studies using both gastric epithelial cells and cells of the monocytic-macrophage lineage to determine their potentially beneficial effects in the context of *H. pylori* infection.

We will evaluate selected biological preparations in terms of their impact on the gastric epithelium, in reducing oxidative stress, secretion of pro-inflammatory interleukin (IL) -8, and pro-repair IL-33. We will take into account the ability of preparations to enhance the phagocytosis process and intracellular killing of macrophages whose activity is inhibited by *H. pylori*, as well as the ability of these cells to secrete pro-or anti-inflammatory cytokines, TNF-α and IL-10, respectively, which direct the development of a specific immune response. Assessment of the enhancement of the cytotoxic activity of lymphocytes is also planned as well as expansion of antigen-specific T-lymphocytes. We will use infrared spectroscopy in Fourier modification (FT-IR) to assess quantitatively serum molecular biomarkers related to *H. pylori* infection.

Using *in vivo* model of *Caviae porcellus* susceptible to *H. pylori* infection, we will check whether oral administration of biomodulators will reduce the development of *H. pylori* infection and the accompanying inflammatory reaction, activate innate immune response, pro-regenerative signaling, and whether it is followed by quantitative changes with selected markers correlated with *H. pylori* infection.