The human body is inhabited by microorganisms such as bacteria, fungi and protists, forming a microbiome. Their number may even exceed the number of cells building the whole human body. The microorganisms that are part of the microbiome are involved in the metabolism of carbohydrates, amino acids, drugs, and the formation of vitamins. Although they can cause illness under certain conditions, they have a positive impact on human health and development. Contact between the microbiome and human cells takes place on the epithelial barriers mainly through the compounds produced by the microbes, although in the event of epithelial rupture, direct contact also occurs.

Unfortunately, our current knowledge of the mechanisms of interaction between cells of the human immune system and the microbiome is fragmentary. The main goal of the presented project is to change this state. We suppose that both: the products of bacterial metabolism and direct contact with bacteria (pathogenic and beneficial) affect epigenetic processes (changes in expression resulting from permanent changes in chromosomes without disturbing the DNA sequence) in cells of the immune system, which leads to changes in a set of features called the phenotype of these cells. What is more, the interaction between the cells of the immune system and the microbiome can be disturbed by environmental factors such as pollutants which may foster conditions leading to autoimmune diseases in which the organism attacks its own cells. Our model organism will be *Staphylococcus aureus*, a pathogen that resides in the nasopharynx and on the skin, which is a common source of infection in people suffering from an autoimmune skin disease called psoriasis. Cells of the immune system that are involved in the inflammatory process of psoriasis will be Th17 lymphocytes and T regulatory cells. We will check how indirect (through toxins) and direct interactions of *Staphylococcus aureus* and human immune system cells affect gene expression and epigenetic processes that control these genes. Thus, we will determine whether such an interaction may affect the properties of the immunological cells that favor the occurrence of inflammation, which is present in psoriasis and other autoimmune diseases.

Our research will not only broaden knowledge of interactions between the microbiome and cells of the immune system and how these interactions affect gene expression and epigenetic processes during contact between these two systems, but will also allow the identification of potential molecular targets for direct interference with processes leading to the development of psoriasis and other autoimmunological diseases.