Gram-negative bacterium *Helicobacter pylori* is one of the most common human pathogens worldwide. It is found in approximately 50% of the entire human population. Infection with this bacterium causes inflammation of the stomach, which can lead to the development of ulcers and in severe cases to stomach cancer. Gram-negative bacteria are surrounded by two membranes (inner and outer membrane), which are separated by a space called the periplasm. Successful infection by bacteria is associated with the secretion of virulence factors into the outer membrane or outside the cell. However, before these proteins reach their final destination, they must be transported from the cytoplasm to the periplasm, across the bacterium inner membrane. The majority of proteins are transported across this membrane by Sec translocon, whose key element is the SecA protein. SecA delivers the precursor protein to the membrane channel and actively participates in its movement, using energy from ATP hydrolysis. The function of the Sec translocon is very well described in the model bacterium *E. coli*, however, in case of *H. pylori* bacterium is very poorly understood. Taking into account numerous differences between these two bacterial species, including low homology between the components of the export systems, we cannot predict mechanisms of regulation of the Sec translocon basing on analogy with these in *E. coli.*

Sec translocon transports proteins in an unfolded form (primary structure), so as they enter the periplasm, they must acquire their correct structure (tertiary structure). The periplasmic protein quality control system (PPQCS), which includes proteases and chaperone proteins, is involved in this process. In addition, this PPQCS has an important function in the process of maintaining cellular homeostasis, which can be disrupted when the cell is exposed to stress conditions (e.g., elevated temperature, non-physiological pH). It has been suggested that PPQCS cooperate with the Sec translocon during translocation of precursor proteins. Therefore, the aim of this project is to investigate the effect of disruption of cellular homeostasis on the levels of expression of the genes encoding Sec translocon components. For this purpose, using qPCR method, we will study the expression level of the selected Sec genes under stress conditions affecting the outer membrane and periplasm, leading to accumulation of misfolded proteins.

The knowledge obtained during the project will result in a better understanding of the transport mechanisms in *H. pylori* and will expand the knowledge of its physiology. Better understanding of this mechanism may, in the future, provide the basis for developing new strategies to combat pathogenic bacteria.