Helicobacter pylori is a bacterium that commonly inhabits humans and causes chronic gastritis. Untreated infections can lead to ulcer disease and even stomach cancer. *H. pylori* attacks stomach epithelial cells using a variety of virulence factors. Among them, proteins that allow adhesion to host cells (adhesins), proteases that destroy connections between epithelial cells, and vacuolating toxin VacA can be distinguished. These virulence factors are proteins that are transported from the cytoplasm (synthesis site) across the inner membrane to the periplasm by means of Sec, a special translocation machinery, to reach the target location (bacterial outer membrane or extracellular environment). The SecA protein plays a very important role in this process, as it introduces the protein precursor into the Sec transmembrane channel and actively participates in its transfer, utilizing energy from the ATP hydrolysis. Because transported proteins leave the Sec channel in an unfolded state, they are prone to formation of incorrect interactions which may lead to the formation of aggregates harmful to the cell. This phenomenon is counteracted by the action of the extracytoplasmic protein quality control system (EPQCS), which groups chaperones, folding catalysts and proteases. Together, these proteins prevent protein aggregation, protect proteins during their travel to the outer membrane or extracellular environment, facilitate achievement of the native structure by periplasmic substrates, and remove incorrectly folded and abnormal proteins through their degradation. It is also believed that some components of EPQCS may also interact with the Sec machinery in the process of releasing proteins from the transport channel, which could increase export efficiency from the cytoplasm. Translocation of proteins across the inner membrane, followed by their folding or transit into or across the outer membrane is relatively well described in the model bacterium *Escherichia coli*. However, in the case of *H. pylori*, this process is very poorly understood. The EPQCS system has not been characterized in *H. pylori*. Most of the components of this system were selected on the basis of *in silico* analyzes of the genomic sequences of these bacteria, while there are no genetic, proteomic, biochemical and functional analyzes of EPQCS. It should be noted here that *H. pylori* is significantly different from *E. coli* and it is not possible to assign functions to the putative EPQCS components in *H. pylori* by analogy with their *E. coli* counterparts. Therefore, the purpose of this project is to investigate functioning of EPQCS in *H. pylori*. In particular, we plan to obtain mutated *H. pylori* strains, unable to synthesize selected EPQCS components, and determine the effects of mutations. We will check whether deletions of individual genes or their combinations will affect bacterial growth under physiological and stress conditions. We will examine the translocation efficiency of Sec using model reporter proteins, determine the changes that occur in the outer membrane of mutated cells, compare the protein composition of mutated to non-mutated cells. We will attempt to identify proteins that are substrates of selected periplasmic chaperones. We will examine the physiological effects of mutations in SecA, the motor protein of the Sec machinery, and we'll check whether reducing SecA activity affects EPQCS. Finally, we will check how disorders in EPQCS and translocation efficiency affect *H. pylori* virulence. The last task will be carried out in cooperation with Professor Silja Wessler from the University of Salzburg (Austria), who is an indisputable specialist in the field of *H. pylori* virulence and host-bacterial pathogen interactions. We expect that the successful implementation of the planned research tasks will enable to propose a model of functioning of EPQCS and its interaction with the Sec machinery in *H. pylori*. Characteristic properties of selected periplasmic chaperones and determination of their substrates should be also provided. Expanding knowledge in this area should lead to a better understanding of the mechanisms of transport and maturation of virulence factors in this bacterium. To our knowledge, this project is the first attempt to define partners, substrates and functions of the periplasmic chaperones in *H. pylori* and to link them to the functioning of the Sec translocation machinery and virulence of these bacteria.