Yersinia enterocolitica is an important, human, gastrointestinal pathogen, an etiological factor of yersiniosis. It causes a range of human diseases from mild diarrhea to mesenteric lymphadenitis. The clinical manifestations of the disease can vary according to the age and health of the host. As an opportunistic pathogen *Y. enterocolitica* primarily causes yersiniosis in young children, older people, and those with reduced immunity. The incidence of *Y. enterocolitica* infection appears to be increasing worldwide. According to Scientific report of the European Food Safety Authority and the European Centre for Disease Prevention and Control, yersiniosis is third most commonly reported zoonosis in Europe, after campylobacteriosis and salmonellosis. *Y. enterocolitica* is a heterogeneous species with varying virulence. It synthesizes a wide variety of virulence factors, such as adhesins, invasin and many secreted toxins and effector proteins which enable it to invade the human host. Synthesis of virulence factors is tightly regulated in response to different environmental factors including temperature, osmolarity, pH, nutrient and ions availability. To adapt to changes in environmental conditions, bacteria tightly regulate their gene expression at both the transcriptional and post-transcriptional levels.

The importance of post-transcriptional regulation has only recently become a focus of interest with the discovery of numerous small non-coding RNAs (sRNAs). Trans-encoded sRNAs are expressed by loci that are separate from their target genes. These sRNAs are usually 50-150 nucleotides long and they modulate mRNA translation and/or stability by imperfect base-pairing interactions. The usual outcome of sRNA-target mRNA interaction is the silencing of gene expression. In *Escherichia coli* sRNA RyhB plays an important role in iron homeostasis by controlling over 50 genes involved in iron utilization and import. Recent studies demonstrate that this regulator might be involved in the virulence of pathogenic bacteria and could play a significant role in regulating an adaptative response during bacterial infections, making them important targets in the fight against pathogens. RyhB regulates gene expression by base pairing with target mRNAs and stimulating their degradation. The identification of *ryhB* homologs and RyhB-mediated regulation of putative targets has not been yet the subject of investigation in *Y. enterocolitica*. Bioinformatic analysis conducted in our research group revealed that *Y. enterocolitica* encodes two homologs of RyhB, termed RyhB-1 and RyhB-2. Also, we identified potential mRNA targets for RyhB-1 and RyhB-2, using TargetRNA2 and IntaRNA programs, which are genes encoding proteins involved in virulence and adaptive abilities of *Y. enterocolitica*.

The main scientific goal of this project is to determine the role of RyhB-1 and RyhB-2 in the pathophysiology of *Y. enterocolitica* 2/O:9. Firstly, mutants lacking the activity of one or both sRNAs will be constructed. Next, changes in selected gene expression profile in obtained mutants will be verified by RT-qPCR. Moreover, the research will be based on the characterization of the phenotype of the strains differed in RyhB-1 and/or RyhB-2 content, i.e. assessment of their swimming/swarming motility phenotype; estimation of susceptibility to various stress conditions; determination of adhesion/invasion ability in epithelial cell line; determination of survival within human macrophages and biofilm formation.

The anticipated findings of this project will shed light on the function of RyhB-1 and RyhB-2 in *Y. enterocolitica* which will broaden knowledge about the role of these homologs in the pathophysiology of yersiniosis, as well as in the infections caused by other bacteria in which these sRNAs have been identified. Moreover, the silencing of bacterial genes by sRNAs creates new possibilities in combating pathogenic bacteria. The search for novel methods to treat bacterial infections is a high priority due to the spread of antibiotic-resistant strains, which make antibiotic therapy increasingly ineffective.