Bacteria from genus *Salmonella* are widespread pathogens, which primarily colonize the gastrointestinal tract and one of the main causes of gastrointestinal symptoms. Currently, *Salmonella* infections are considered as a serious epidemiological problem worldwide.

Infections caused by *Salmonella* are referred as zoonoses, as consumption of animal products is the major source of disease in humans. The major etiological factor responsible for 99% of salmonellosis cases is *S. enterica* subspecies *enterica*, which includes more than 1500 serovars. *S.* Typhimurium is the most frequently isolated serovar from human and animals infections, responsible for food poisoning and invasive infections. Millions of gastroenteritis cases are reported each year and, in addition, increasing amount of systemic infections (bacteremia) with a fatal outcome is observed. Due to increasing amount of antibiotic resistant *Salmonella*, treatment of salmonellosis is impeded. For this reason, salmonellosis is considered a serious threat to public health and socio-economic development in many regions of the world.

Pathogenesis of salmonellosis is a multistage interaction of bacteria with host cells. Successful establishment of *Salmonella* infection depends on the initial stages – adhesion to and invasion of the intestinal epithelium. Adhesion prevents mainly mechanical removal of microorganisms from gastrointestinal tract, facilitating bacteria survival and intensive multiplication. The key role in this stage of infection is played by number of cellular structures referred as adhesins. In *Salmonella* strains they are both chromosome- and plasmid-encoded, and many of them are clustered together in chromosome, and called pathogenicity islands.

Limited knowledge regarding factors involved in infection process, raise need for high-throughput research focused on identification of genes that affect the level of adhesion to and invasion of intestinal epithelial cells by *Salmonella*. Screening studies conducted at the Department of Biochemistry and Molecular Biology have enabled identification of several genes not previously associated with adhesion and invasion and one of the most promising of them is a *sanA* gene, which encode SanA protein.

The aim of the proposed study is to determinate the role of the SanA in the pathogenesis of *Salmonella* spp. It is planned to generate *S.* Typhimurium *sanA* deletion mutant, which will give us opportunity to investigate contribution of SanA to adhesion and invasion of the intestinal epithelium. The mutants will be subjected to functional assays using human epithelial cell lines. Additionally, in order to verify the role of SanA in the virulence of *Salmonella* in animal model, it is planned to conduct *in vivo* experiments with use of the mouse model. Taking into account previous scientific reports on the contribution of SanA to vancomycin tolerance, and therefore its potential role in cell wall synthesis, investigated *Salmonella* strains will be tested for tolerance to a number of xenobiotics. The final stage of the study includes the analysis of subcellular localization of SanA protein in a bacterial cell using rabbit polyclonal antibodies and, super- resolution microscopy.

The results of this project will allow to verify the biological function of SanA in *Salmonella* Typhimurium in the context of infection and will provide new and valuable information about interaction of *Salmonella* and host. This knowledge is crucial in the context of a detailed understanding of the bacterial pathogenesis and consequently, in the development of new methods for prevention and treatment of salmonellosis based on the elimination or hindrance of adhesion to host cells. The significance of the results is supported by the increased amount of antibiotic-resistant *Salmonella* isolates, which leads to the difficulties in the treatment of salmonellosis.