Horizontal gene transfer, i.e. the lateral transmission of genetic material between cell of various strains and species, is regarded as a driving force of bacterial evolution. One of the most important mechanisms observed in bacteria which influences the occurrence of such phenomena is the activity of restriction-modification (R-M) systems. This term is used to define various genetic modules, ubiquitous in bacterial genomes, which determine two enzymatic functions. The first of them—a nucleolytic (restrictive) activity—is responsible for cutting specific sequences within foreign DNA which penetrate into the cell. Thus their prevent the stabilisation of a new mobile genetic element therein (e.g. a plasmid or bacteriophage DNA). To preclude similar host's DNA degradation, the other enzymatic activity is indispensable, viz. the ability to modify the mentioned specific sequences by the introduction of methyl moieties to nitrogenous bases in the DNA. Consequently, the host's genetic material is protected from cutting in contrast to the unmodified invasive DNA. In the most widespread type II R-M systems the two enzymatic activities are conferred by a pair of proteins: a methyltransferase (MTase) and a re-striction endonuclease (REase), which are specific against the same short DNA sequence. Due to their ability to cut DNA selectively, REases are universally applied in genetic engineering.

As it can be concluded from the consideration of the nature of the described modules, the expression of MTase and REase genes must be a subject of a strict regulation so that no undesirable damage of the cell's DNA would happen. It may come to pass if the R-M genes are lost while a residual pool of the proteins remain within the cell's cytoplasm. Along with the decrease in the MTase level it ceases to methylate all target sites in the host's DNA which will render them prone to the REase-mediated cutting. In consequence the cell may suffer death or a genomic rearrangement—if DNA repair mechanisms prove to be efficient. Both phenomena determined by R-M systems influence the structure of bacterial populations. Therefore, the research on the mechanisms of action of the described genetic modules provides insight into the explanation of the course of bacterial evolution. That includes also pathogenic bacteria, in which R-M systems may indirectly modulate the production of virulence factors.

Our project encompasses studies of two R-M systems carried by plasmid pP62BP1—an extrachromosomal genetic element harboured in an Arctic strain of *Psychrobacter* sp. DAB_AL62B. The results of our initial research suggest that the activity of both systems is subjected to a complex regulatory mechanism, which consists of the function of the MTases encoded therein as well as the influence of putative regulatory RNA. Similar phenomena are known for other R-M systems, however their joint activity has never been observed before. Moreover, the studied systems are unique as for their genetic organisation. The level of identity of pP62BP1-encoded proteins is as high as ca. 97%, nevertheless considerable differences between the systems are found in their regulatory elements.

The aim of the project is thus to establish and confirm experimentally a model for a novel type of a regulatory network of R-M systems. To achieve this, we intend to conduct complex analyses of the gene expression profile for both modules in various experimental set-ups as well as to investigate the activity of the proteins encoded therein. The obtained results will also allow for the elucidation of mutual interactions between both 'twin' R-M systems of plasmid pP62BP1.