

Bacterial pathogens cause significant losses in worldwide plant production. Due to increasing global temperature, import of tropical fruits and vegetables as well as intensive tourist traffic, we have to be aware of possibility of unusual pathogenic bacteria strains transmission. To develop new and effective methods for plant protection we need better understanding of molecular processes determining pathogens virulence and plant resistance. *Pseudomonas syringae* is a frequent causal agent of bacterial diseases of plants. It is equipped with a syringe-like structure for injection of proteins called effectors directly into the host cell. Usually, the effectors interfere with components of plant defence mechanisms, therefore, are highly dangerous for plants. Each strain of *P. syringae* employs a repertoire of effectors, which controls the infection course and determines the host range.

Within the scope of these proposal we will analyse the uncharacterised effector family - HopAG1.

Our initial observation indicates, that this protein may act in different sectors of the plant cell, which suggests, that it may misregulate various vital processes. Bioinformatics predictions revealed, that HopAG1 is divided into three regions, which may exert different enzymatic activities *i.e.* ADP-ribosyltransferase, kinase, nudix hydrolase. Additionally, HopAG1 possibly undergoes modifications in the plant cell, which we hypothesize may act as a molecular switch changing the structure of the effector and thereby turns on/off its various activities.

In the proposed project, we will verify, if HopAG1 is modified within the plant cell. Next we will investigate, what are the consequences of this modification and introduced substitutions within the predicted motifs for HopAG1 cellular destination, its interaction with other proteins, its enzymatic activity and finally virulence of *P. syringae*. We will characterise HopAG1 interaction with the already identified proteins and we aim to find novel plant proteins targeted by this effector. Also, we will undertake an effort to elucidate a mode of HopAG1 action.