Plant parasitic nematodes are small roundworms comprising above 4000 species, which attack the plant roots and cause serious crop yield losses worldwide. It was estimated that damages caused by plant parasitic nematodes exceeds $US 100 billion every year. Substantial part of the losses is caused by sedentary endoparasitic root-knot and cyst nematodes. These nematodes are able to manipulate plant cell metabolism by injecting effector molecules into the plant cells. Nematode effectors are proteins or sometimes other chemical molecules, which are able to suppress pathogen recognition and defence response of susceptible plants. It allows for the induction and development of nematode feeding sites. Unfortunately, different plant protection methods (traditional, chemical, biological) against nematodes are expensive, environmentally unfriendly and ineffective. Breeding for nematode resistance based on natural host resistance genes (*R*) is complicated due to the low number of identified and cloned nematode resistance genes. These genes usually originate from wild relatives and were introgressed into crop genomes by the crossbreeding. Unfortunately, resistance based on R genes is often overcome under field conditions. Only three R proteins against plant parasitic nematodes have typical R protein domains (NBS-LRR, nucleotide-binding site – leucine-rich repeat). Specific R proteins are activated by specific pathogen effectors, what leads to interaction called effector-triggered immunity (ETI), which limits pathogen proliferation. Other proteins, called pattern-recognition receptors (PRR) that are in the first line of plant immune system, can also recognize nematode pathogens. PRRs are leucine-rich repeat kinases located at the extracellular surface and they are activated by pathogen-associated molecular patterns (PAMPs). Activation of PRRs induces defence response called PTI (PRR-triggered immunity), which inhibits pathogen proliferation. PTI defence in susceptible host plants is insufficient to suppress the infection and colonization of pathogens. ETI or PTI are triggered at the early stages of pathogen infection, however to date there is no data allowing identification of plant genes responsible for the loss of plant defence.

Therefore, the aim of this project is to find out, which genes have substantial importance for plant susceptibility and to define their role in the plant-nematode interaction, through the analysis of differential gene expression during early stages of the nematode parasitism. Obtained results will be confirmed by examination of mutant and overexpressor plants. To achieve this goal we chose a widely accepted and well established experimental model consisting of the susceptible plant *Arabidopsis thaliana* and its pathogen, beet cyst nematode (*Heterodera schachtii*). *A. thaliana* is an excellent genetic model plant, which genome was sequenced and excessively examined.

The knowledge concerning basic molecular mechanisms of the interaction between plant and parasitic nematode may provide tools to develop new mechanism of plant resistance or tolerance to parasitic nematodes, which could be implemented in plant resistance breeding in the future.