In the second part of the book "*Alice in the wonderland*" entitled "*Through the looking glass*" by Lewis Caroll, Alice meets the Red Queen. They both run very fast; however, when Alice stops exhausted she notices that they are still in the same place. Then the Red Queen explains to Alice: "*Here, you see, it takes all the running you can do to keep in the same place*." This sentence was used by scientists to explain that all species have to adapt constantly to changing conditions in order to avoid extinction. In the context of immunology, it means that both hosts and their pathogens need to improve constantly their defence and virulence mechanisms, respectively. This is also known as the "Red Queen Hypothesis" or "Evolutionary arm-race" between a host and a pathogen.

Insects have developed efficient strategies to protect themselves against infection. They rely only on innate mechanisms, which means that they do not possess T and B cells or antibodies. When infected, hemolymph cells called hemocytes may phagocytise intruders or capture them in multicellular structures called nodules or capsules. Furthermore, after activation of signalling pathways, they synthesise and secrete so-called antimicrobial peptides to the hemolymph, which directly kill infecting microorganisms, mostly by destruction of their membranes. On the other hand, pathogens have developed strategies that are able to force insect's protective mechanisms, get inside the insect body, degrade host's antimicrobial peptides, and subsequently colonise the insect body. Hence, the body of an infected insect can be perceived as the front line, where the fight for death and life takes place. The one whose defence or virulence mechanisms appear to be more effective will survive.

In this context, we plan to investigate the interaction between the insect host, i.e. the greater wax moth *Galleria mellonella*, and the relatively newly found entomopathogen *Pseudomonas entomophila*. This is a unique bacterium among the *Pseudomonas* genus, which can naturally infect insects. This Gram-negative bacterium isolated from the fruit fly *D. melanogaster* can be found in the soil, water, and rhizosphere; yet, it is not pathogenic to plants. When ingested by *Drosophila*, it causes gut damage leading to insect's death. The insect model is a wax moth living in beehives, or, more often in slices of stored wax. Its larvae feed with honey, pollen, and wax, causing *galleriose*, and can be considered as pests.

The submitted project focuses on investigations of host-pathogen interaction during the infectious process. We will study the activation of immune response of the greater wax moth, e.g. the appearance of antimicrobial activity in insect hemolymph, expression of genes encoding immune-related polypeptides. We will also follow changes in the body of infected larvae. We will learn which organs are colonised and eventually destroyed by infecting bacteria. By the analysis of the hemolymph of infected larvae, we will learn what antimicrobial peptides are synthesised by the insect as defence molecules acting against intruding bacteria. Since we will infect with a relatively new pathogen, we expect to find new immune polypeptides. Preliminary studies indicate that, indeed, proteins that are not yet known appear in the hemolymph of infected insects. Therefore, we may find new bioactive molecules and test their spectrum of activity. Additionally, we plan to investigate immune priming phenomenon. This touches the exciting issue of insect immunity - a kind of "*immune memory*" in animals that do not possess mechanisms of classically understood acquired immunity. It is an element of adaptation to recurring infections.

The output from realisation of the project may bring us information about new defence strategies undertaken by the host. We may identify new bioactive molecules with antimicrobial activity. The results will be of interest for biologists and biotechnologists working in different areas such as innate immunity and host-pathogen interactions, biopesticides and antimicrobial drugs.