Abstract

Bordetella pertussis is strictly human pathogen that causes severe infection of the respiratory tract known as whooping cough, which is currently on the rise. *B. pertussis* was considered as an extracellular pathogen for a very long time. Recently it was shown the ability of *B. pertussis* to survive inside early endosomes of macrophages. This ability is studied in the human monocytic cell line THP-1 and also in primary macrophages from human donors. This diploma thesis is focused on THP-1 infectious model and mainly for the early phase of infection. A previously performed transcriptomic study showed significantly affected genes of *B. pertussis* during intracellular survival in THP-1 macrophages. In this study, we selected genes that are in some way related to intracellular survival inside human macrophages or have significantly effect for intracellular survival. The effect of the mutation in these genes was tested both on the level of cytotoxicity to THP-1 cells and the related number of surviving bacteria inside the macrophages. The deletion strain in two genes for cysteine dioxygenase (*BP2871* and *BP3011*) and the mutant strain allocated in the Bvg+ phase were less cytotoxic than the control strain. Monitoring the effect of opsonization to intracellular survival have not such clear results. The effect of serum to *B. pertussis* seems to be more complex. However, we optimized the infection model, on which other conditions or other mutant strains can be tested.

Key words: *Bordetella pertussis*, THP-1 cell line, infection model, intracellular survival, virulence factors, opsonization.