Abstract

Bordetella pertussis is a Gram-negative strictly human pathogen and the major causative agent of whooping cough or pertussis. The incidence of this highly contagious respiratory disease in developed countries has increased in the last decades. One of the less characterized virulence factors of *B. pertussis* is the type three secretion system (TTSS) which is responsible for the secretion of the effector proteins into host eukaryotic cells. This diploma thesis sheds light onto factors influencing TTSS *in vitro* activity. Although TTSS of laboratory strain Tohama I was induced by biologically active compounds present in blood (e. g. complement proteins), TTSS of recent clinical isolate B1917 seems to be induced permanently. Furthermore, *BB0302* encoding a GntR family transcription regulator in *B. bronchiseptica* RB50 (homologous to *BP0209* of Tohama I) was studied, however, the deletion of this gene did not affect the TTSS functionality. Serum resistance is a factor that plays a key role in the pathogenesis of *B pertussis*. We show that Czech recent isolates (2008–2015) are significantly more resistant to serum killing *in vitro* than the original vaccine strains (1954–1965). This phenomenon seems to result from the adaptation of global *B. pertussis* population to its human host. In addition, this diploma thesis documents and discusses the obvious differences between the laboratory strain Tohama I and recent isolates of *B. pertussis*.

Key words: *Bordetella*, clinical isolate, virulence, gene expression, T3SS, resistance to serum killing, complement, adaptation, vaccination