Abstract:

Species of the *Bordetella* genus cause the highly contagious whooping cough disease in humans (*B. pertussis*, *B. parapertussis*) and related respiratory diseases in other mammals (*B. bronchiseptica, B. parapertussis*). One of the virulence systems of *Bordetellae* is the type III secretion system (T3SS) employed for translocation of effector proteins directly from bacterial cytosol into the cytosol of host cells. The T3SS protein BopN protein has been categorized as a *Bordetella* effector protein. Nevertheless, the homologous proteins in other gram-negative bacteria function in establishing the secretion hierarchy through T3SS and some of them block T3SS secretion in high calcium environments before bacteria-host cell contact has been established. In this thesis I examined the function of the BopN protein and the role of calcium ions in T3SS activity of *B. bronchiseptica*. Two independent methods have been used for determination of T3SS secretion activity. Addition of 2 mM calcium ions into bacterial media decreased secretion of the T3SS reporter, while no such effect was observed in a *B. bronchiseptica* strain lacking the bopN gene. Mass spectrometry data confirmed the inhibition of T3SS activity in the presence of calcium ions. Enhanced calcium levels resulted in decreased mobilization and secretion of the T3SS translocator protein BopD, while secretion of the BteA effector protein was unaffected. Deletion of the bopN gene then reversed the effect of calcium ions on T3SS secretion activity. Our data suggest a regulatory role of BopN protein in the function of *B. bronchiseptica* T3SS.