Abstract

The aim of this thesis is to summarize *S. aureus* interactions with selected mechanisms of innate host immunity especially interactions with neutrophils and processes on the cell level which lead to host colonization. *S. aureus* surface proteins MSCRAMM interact with host cell surface proteins such as fibrinogen, keratin and thereby mediate adhesion to the host cell, which is an essential point for colonization of the host cell. The central mechanism of innate immunity against any *S. aureus* infection is the interaction of the pathogen with neutrophils, which produce neutrophil extracellular traps and phagocytes *S. aureus* cells. A crucial role in the elimination of bacterial cells in the phagosome of neutrophils is lysis by the antimicrobial peptides and degradation of bacterial biomolecules by the oxygen radicals. *S. aureus* defence mechanisms against action of immune system are considered to be virulence factors, due to its contribution to the establishment of the infection. These mechanisms are based on cell wall modification, inhibition of neutrophil chemotaxis, and production of enzymes that inhibit the effect of antimicrobial peptides, lysozyme, oxygen and nitrogen radicals. Expression of virulence factors of a particular *S. aureus* strain and host-specific risk factors can lead through successful colonization of the host to the origin of a local or severe systemic infection.

Key words:

Staphylococcus aureus, innate immunity, adhesion, phagocytosis, mechanisms of interaction, intracellular cycle