The main topic of this thesis is the ARE subfamily of ABC transporters. The importance of the proteins of this subfamily lies in the fact that they confer resistance to several classes of clinically important antibiotics: macrolides, lincosamides, streptogramines and pleuromutilines and they do it in significant pathogens, as for example Staphylococcus aureus. Compared to canonical ABC transporters, the structure of ABC proteins lacks the transmembrane domain (TMD) and so far, there where not even found an integrating transmembrane protein. Due to these facts, the mechanism of resistance conferred by these proteins remains unclear. In the thesis, both suggested hypotheses of the mechanism of how these proteins work are discussed. The first hypothesis presumes the active efflux of antibiotics out of the bacteria. The second hypothesis suggests release of antibiotic from its binding site initiated by ARE proteins, followed by its passive diffusion out of the cell.

Keywords: ABC proteins, ARE proteins, resistance, MLS, Vga