The aim of this thesis is to summarize the mechanisms of resistance of *Staphylococcus aureus* to MLS_B antibiotics (macrolides, lincosamides and streptogramins B type) which are used to treat respiratory infections in cystic fibrosis patients. This pathogen evolved during time many various strategies of resistance to these proteosynthesis inhibitors. The most common mechanisms are target site modification, modification of the antibiotic itself or antibiotic eflux out of the bacterial cell. Apart from these mechanisms based on acquisition of genes, a mutation of specific genes can also result in resistance of the strain. In the lungs of CF patients, long-term antibiotic treatment together with immune system defects result in development of a unique niche. It is colonized (besides other bacteria) by *S. aureus*, which is well adapted to this environment and also uses different mechanisms of resistance as hypermutation or switching to dwarf phenotype (small colony variants) enabling intracellular persistence. MLS_B antibiotics as well as beta-lactams are being applied as the treatment of choice for respiratory infections in CF patients. Studying the mechanisms of MLS_B resistance is therefore of extraordinary importance.