Summary

Photodynamic inactivation (PDI) is a method utilizing a combined action of photosensitizing compound, light of a proper wavelength and oxygen. As a result of the simultaneous action of these three elements, a generation of local oxidative stress occurs, which leads to the damage of biological structures. PDI is a potent tool to combat multiresistant human pathogens. This work describes the analysis of mechanisms engaged in response of *Staphylococcus aureus* to photodynamic inactivation. Among those elements, the alternative sigma factor σ^B has been identified. Factor σ^B is engaged in *S. aureus* response to environmental stress. Disruption of genes coding σ^B and proteins regulating its activity, encoded in *sigB* operon, has led to increased susceptibility of analyzed strains to photodynamic action of protoporphyrin IX diarginate, zinc phthalocyanine and rose bengal. Sequencing of *sigB* operon genes in clinical *S. aureus* strains resulted in identification of various mutations, such as insertions, deletions and nucleotide substitutions. Functional analysis revealed that identified mutations correlated with decreased σ^B activity in 33% among 15 analyzed clinical isolates. Particular accumulation of mutations has been observed in gene *rsbU* coding the main activator of σ^B. In terms of biochemical analysis, all mutants were characterized by the inhibition of staphyloxanthin synthesis. This membrane pigment of antioxidant properties has been assessed as significant for *S. aureus* response to PDI. As a common feature of strains susceptible to PDI, a high bacterial membrane fluidity has been described. Analysis of bacterial catalase engaged in detoxification processes revealed that this enzyme plays no significant role in *S. aureus* response to PDI with the use of chosen photosensitizing compounds. Moreover, no correlation between σ^B and catalase activity has been observed. Conducted research indicates a particular role of RsbU-dependent σ^B factor, staphyloxanthin content and membrane fluidity in survival of *S. aureus* clinical isolates upon photooxidative stress conditions.