Hydrogen peroxide induced repression of icaADBC transcription and biofilm development in Staphylococcus epidermidis

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PMID: 18942741 DOI: 10.1002/jor.20758

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Abstract

Expression of the icaADBC-encoded polysaccharide intercellular adhesion by Staphylococcus epidermidis promotes biofilm formation and represents an important virulence factor in biomaterial-related infections following orthopedic surgery. Biofilm development by the pathogen can be viewed as a protective reaction to environmental stressors including osmotic stress, thermal stress, and antimicrobial chemotherapy. Oxidative stress, arising from the release of toxic oxygen radicals by polymorphonuclear cells, is encountered by bacteria entering the body perioperatively. Evasion of this and other cell-mediated immune responses by pathogenic bacteria plays an important role in the development of chronic biomaterial-related infection. Here we investigated the impact of sublethal oxidative stress induced by H_2O_2 (<18 mM) on S. epidermidis biofilm formation. S. epidermidis grown in brain heart infusion (BHI) media supplemented with 5 mM H_2O_2 or 10 mM H_2O_2 formed significantly less biofilm ($p < 0.01$ and $p < 0.001$, respectively) than bacteria grown in BHI alone. Consistent with this, using reverse transcription-polymerase chain reaction expression of the ica locus was also shown to be reduced by subinhibitory concentrations of H_2O_2. Furthermore, diminished ica operon expression correlated with increased expression of icaR, which encodes a repressor of icaADBC. Thus, these data suggest that mild oxidative stress downregulates biofilm development by S. epidermidis and may have potential in a therapeutic context.