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EVIDENCE FOR THE INVOLMENT OF STAPHYLOCOCCUS EPIDERMIDIS LPXTG SURFACE PROTEIN SESC IN BIOFILM FORMATION AND CATHETER-RELATED INFECTIONS

L. Khodaparast[1], L. Khodaparast[1], M. Shahrooei[1], J. Van eldere[1]

[1]Clinical immunology and microbiology, Kuleuven, Leuven, Belgium

Objective:

Staphylococcus epidermidis is a frequent cause of infections on indwelling medical devices due to its ability to form a biofilm. Surface proteins have been shown to play key roles in *S. epidermidis* biofilm formation. We have recently shown that active and passive immunization against *S. epidermidis* LPXTG surface protein SesC can decrease *S. epidermidis* biofilm formation. In this study, we present new evidence on the role of SesC in *S. epidermidis* biofilm formation and its potential as a suitable vaccine target.

Method:

The entire coding region of genes encoding *S. epidermidis* LPXTG proteins SesC and SesK were cloned and transformed into *S. aureus* strains lacking *sesC* and *sesK*. The effect of this transformation on biofilm formation and virulence was evaluated *in vitro* and *in vivo*.

Results:

Transformation of polysaccharide intercellular adhesin (PIA)-dependent biofilm-forming *S. aureus* strain 8325-4 with *sesK* had no any effect on the quantity or phenotype of biofilm. However, transformation with *sesC*: *i*) switched the mechanism of biofilm formation from PIA-dependent to proteinaceous in 8325-4 and another PIA-dependent biofilm-forming *S. aureus* strain MSSA4, *ii*) converted non-biofilm-forming *8325-4* isogenic *ica* mutant to a proteinaceous biofilm-forming strain, *iii*) converted biofilm-forming 8325-4 isogenic *srtA* mutant to a non-biofilm-forming strain but biofilm formation could be restored by complementation with *S. aureus srtA* gene, *v*) significantly increased catheter colonization and infection rates *in vivo* which could be significantly decreased in the presence of αSesC IgG's.

Conclusion:

Our new data suggest that SesC is directly involved in biofilm formation and infection *in vitro* and *in vivo*. It might indirectly intracellularly switch the mechanism of biofilm formation. SesC seems to be a promising target for vaccine development against *S. epidermidis* biofilm formation.