

Molecular features of nosocomial *S. epidermidis*

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Staphylococcus epidermidis is one of the most prevalent causes of nosocomial infections in immunocompromised patients with implanted biomaterials. The capacity to form thick multilayered biofilm on a wide variety of polymer and metal surfaces is an important virulence factor that allow *S. epidermidis* to evade host immune defence mechanisms, antibiotic therapy and to develop device-related infections. The formation of a biofilm involves several distinct stages, including initial attachment, cell-to-cell adhesion and proliferation, maturation, and finally, detachment. Although several factors are reported to influence accumulation of bacteria on the surface, production of a polysaccharide intercellular adhesin (PIA) is the main biofilm mechanism in staphylococci. PIA is synthesised by enzymes encoded by the *ica* (intercellular adhesin) operon.

Using several molecular typing methods for nosocomial *S. epidermidis* strains such as multilocus sequence typing (MLST), pulsed field gel electrophoresis (PFGE) and other genetic techniques, we have analysed a number of strains from various spatiotemporal origin. We could show that *S. epidermidis* strains circulating in the hospital environment differ from commensal isolates. Typical features of nosocomial *S. epidermidis* strains obtained from device-associated infections are presence of the *ica* operon, biofilm formation and carriage of additional genetic information such as mobile DNA elements. Furthermore, they are mostly resistant to methicillin and to other antimicrobial classes. By means of MLST we were able to find a limited number of closely related *S. epidermidis* clones in hospital settings that are responsible for the majority of device-related infections.

In summary, biofilm formation, acquisition of resistance traits and a pronounced phenotypic and genetic diversity seems to facilitate the survival and successful establishment of *S. epidermidis* in hospital environments as a pathogen.