OP33

STAPHYLOCOCCUS EPIDERMIDIS BIOFILM LIFECYCLE AND ITS VIRULENCE: FROM PLANTKONIC GROWTH, TO BIOFILM STRUCTURE AND SYSTEMIC DISSEMINATION

Ângela França[1]; A. I. Freitas[1]; V. Carvalhais[1]; B. Perez-Cabezas[2]; A. Correia[2]; C. Cywes-Bentley[3]; T. Maira-Litrán[3]; M. Vilanova[2]; G. Pier[3]; N. Cerca[1]

[1]*University of Minho*
[2]*University of Porto*
[3]*Harvard Medical School*

Staphylococcus epidermidis has been recognized as a leading cause of several clinically relevant infections, with particular association with the use of medical devices. This is related with its ability to colonize the surface of these devices and form biofilms. The major clinical implications of biofilm formation are the high resistance to antimicrobials and the host immune system, resulting in the development of chronic infections. S. *epidermidis* biofilm lifecycle is divided into 3 stages: initial adhesion, accumulation and maturation, and biofilm disassembly. Despite its impact in the development of acute infections, biofilm disassembly is the less studied of all stages, and little is known about the phenotype and the interaction of the released cells with the host immune system.

To uncover the mechanisms by which biofilms evade the host immune system and cause chronic infections, a transcriptomic analysis of *S. epidermidis* biofilms exposed to human blood was performed. Our results revealed an extensive remodelling of the transcriptome suggesting a quick adaptation to the new environment.Genes involved in amino acids biosynthesis, as well as iron uptake were strongly affected, indicating these mechanisms as important factors in bacterium survival and virulence.

Furthermore, to understand the particularities and virulence associated with biofilm disassembly and the development of acute infections, biofilm-released cells (Brc) were characterized by the following parameters: total protein and gene expression profiles, antimicrobial susceptibility, initial adhesion, CLSM analysis of surface biomarkers, opsonophagocytic killing assays, and finally, the interaction of brc with the host immune system was assessed using a murine infection model. Our results revealed that S. *epidermidis* Brc are unique in their phenotype and virulence potential, sharing some features with planktonic cells, such as expression of *psm*β, but simultaneously displaying features similar to biofilms, such as high antibiotic tolerance.The phenotypic differences were translated to differences in the immune response. Brc elicited higher amounts of IL-6 and KC cytokines, and lower amounts of MCP-1 than biofilm cells showing, concomitantly, higher colonization of the spleen and liver. Thus, targeting of particular properties of the Brc could present new opportunities to effectively prevent the pathologic events associated with dissemination of cells from a biofilm to more distant sites.