adherence to surface immobilized FN. To this end, the involvement of defined Embp-modules in this process was tested by heterologous in trans expression of the native export motif fused to repetitive found in various architectures (FIVAR) or FIVAR-GA repeats, followed by anticipated cell wall anchor domains. Expression of FIVAR repeats alone or in combination with FIVAR-GA repeats resulted in increased binding of *Staphylococcus carnosus* to surface immobilized FN. By the use of biochemical methods the minimal structural unit of the FIVAR region and GA-module sufficient for FN binding were identified. Strikingly, *S. epidermidis* is incapable of recruiting soluble FN via Embp to its surface, suggesting the involvement of cryptic FN domains that are only accessible during resolution of the globular conformation of the FN molecule during fibrillogenesis. Since immobilization of a recombinant FN Type III subdomain (rFN12-14) strongly augmented bacterial binding, these domains are obviously sufficient for *S. epidermidis* - FN interactions. Protein-interaction-mapping suggests binding in FN12 apart from so far known binding mechanisms. In conclusion, we here provide molecular evidence demonstrating the crucial role of defined Embp modules in staphylococcal adherence to FN and we present first insights into a yet unknown FN binding mechanism in staphylococci.

OS-P01

S. epidermidis biofilm-released cells: the final frontier?

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Staphylococcus epidermidis is the most common CoNS associated with nosocomial infections, mainly due to its ubiquitous presence in human skin and mucosae and its remarkable ability to form biofilms on medical devices. Biofilm formation is associated with high antimicrobial tolerance and evasion from the host immune system, often leading to recurrent infections. Not surprisingly, biofilm formation has been extensively studied in the past decades. However, little is known regarding biofilm disassembly. Nevertheless, it has been shown that the cells released from biofilms are involved in the onset of acute infections, with bacteremia being one the major clinical manifestations. Since the investigation of biofilm disassembly requires more complex experimental set up, the study of the cells released from biofilms has lagged behind.

We have recently demonstrated that using the common microtiter plates system, in specific conditions, it's possible to collect cells disassembled from *S. epidermidis* biofilms. Transcriptomic and phenotypical characterization has shown that these cells present unique traits, as compared to biofilms or planktonic cells, including higher tolerance to antimicrobials and stimulation of a unique response from the murine immune system.
We first performed an RNA-Seq analysis that revealed that when exposed to human blood, biofilm-released cells presented major transcriptomic alterations, specially associated with biosynthesis and metabolism of amino acids and import and export of substances through ABC transporters, as well as biotin metabolism. These differences had an impact in how murine immune effectors react to infection: microarrays of mouse splenocytes and cytokines quantification in the serum indicated that biofilm-released cells are particularly effective at activating neutrophils, monocytes, T lymphocytes and antigen presenting cells. Finally, we tested the antimicrobial susceptibility to 10 different antibiotics and we observed that in the majority of the cases, biofilm-released cells presented high tolerance, which is classically associated with biofilms.
Overall, our data reveals that *S. epidermidis* biofilm-released cells present a particular phenotype and open the door for further research in the final stage of the biofilm life-cycle: biofilm disassembly.

OS-P02

Macrolide and lincosamide resistance genes in coagulase-negative staphylococci living in aquatic environments

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In contrast to clinical isolates, little is known about antibiotic resistance in *Staphylococcus* sp. living in aquatic environments. To investigate the resistance level against macrolides and lincosamides, 2249 coagulase-negative staphylococci obtained from sewage and receiving river waters were tested and the corresponding resistance genes identified by whole genome sequencing of 8 resistant isolates and pcr. 18.2 % of the isolates were resistant against the macrolide erythromycin, 4.3 % were inducibly and 3.5 % constitutively resistant against the lincosamide clindamycin. Clindamycin resistance was mainly encoded by *erm*(C). The *erm*(43) gene was not only