FP418. On the old story of the accidental pathogen: can transcriptomics clarify how *S. epidermidis* becomes virulent?

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Background
Staphylococcus epidermidis is a commensal inhabitant of healthy human skin and mucosae that can originate important infections such as medical device-associated bloodstream infections, often associated to patients with impaired or undeveloped immune systems. Of concern, the current inability to discriminate between true bacteraemia caused by *S. epidermidis* and contaminated blood cultures often leads to misdiagnosis, resulting in a significant increase in patient morbidity and in healthcare costs. Until now, several genetic and phenotypic approaches were not able to identify any specific marker that clearly distinguishes commensal from isolates that cause infection. Hence, our goal was to identify possible RNA-based molecular markers for the diagnosis of *S. epidermidis* infections, a strategy never reported before.
Method
The transcriptome of three clinical and three commensal isolates exposed to human blood was sequenced using high-throughput RNA-sequencing (RNA-seq). A bioinformatics analysis was used to compare the 6 transcriptomes and to select potential markers that could be used to differentiate true *S. epidermidis* infections from laboratory contamination. Several approaches were performed and the obtained data was further confirmed by qPCR. Biological confirmation was then accomplished by qPCR in a representative worldwide collection of 70 *S. epidermidis* isolates.
Results and Conclusion
We identified and selected 5 genes that where able to discriminate between the 3 clinical from the 3 commensal isolates. However, when testing a wider range of isolates, the discriminative power of the selected genes was no longer observed. This suggested that, in fact, both clinical and commensal isolates are able to adapt to human blood and use similar strategies when causing infection. To demonstrate this principle, the survival rate of all 70 isolates was assessed after incubation with human blood. The results showed that both clinical and commensal isolates shared the same survival capability in human blood. Together, our data reinforces the idea that *S. epidermidis* is an accidental pathogen.

Acknowledgments
The authors acknowledge the Doctoral Advanced Training [Norte 69-2015-15] funded by the European Social Fund under the scope of Norte2020, FCT project EXPL/BIA-MIC/0101/2013; strategic funding of unit UID/BIO/04469/2019; BioTecNorte operation (NORTE-01-0145-FEDER-000004).