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[P19] MODULATION OF *S. AUREUS* QUORUM SENSING BY *P. AERUGINOSA*

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S. aureus and *P.aeruginosa* are frequently found together in CF airway polymicrobial infections and we have determined the genetic basis of adaptation in the transmissible, CF-adapted *P. aeruginosa* DK2 lineage (PADK2). Here, we investigate whether and how the genetic changes in PADK2 have re-modeled its ability to interact with other CF-airway microorganisms such as *S. aureus*.

Infecting bacteria can grow as aggregates on airway surfaces, therefore we examined the *in vitro* transcriptional pattern of SA and PADK2 grown on a solid surface for 24 hours as monoculture or coculture spots. RNA-seq was performed and a pairwise analysis was conducted for each strain. RNA-seq data was validated by RT-qPCR and transcriptional *lacZ* fusions.

In coculture, SA showed a general tendency of downregulation of gene expression, including reduced expression of many genes involved in virulence and regulated by the Agr Quorum Sensing system such as proteases, leukocidins, leukotoxins and haemolysins. Interestingly, the *agrD* gene encoding the precursor of the autoinducing peptide – AIP– regulating QS, was also downregulated in coculture, pointing to inhibition of the Agr QS system. Consistent with this model, protein A was upregulated in these conditions. Conversely, PADK2 showed upregulation of the *pqsD* gene – involved in biosynthesis of the QS molecule HHQ – and a general pattern of enhanced gene expression.

Our *in vitro* study shows that microbe-microbe interactions can modulate expression of important virulence genes which – in the case of *S. aureus* – results in a phenotype compatible with a persistent infection and enhanced survival in the host. *P. aeruginosa* DK2 modulates *S. aureus* gene expression via the Agr QS system by a yet to be identified mechanism.