O639

Abstract (oral session)

Loss of MDR efflux systems in Salmonella enterica serovar Typhimurium results in an inability to form competent biofilms due to repression of curli biosynthesis

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Objectives: Mulitidrug resistant efflux pumps can confer low level resistance to a variety of antibiotics, detergents and dyes. Salmonella is known to have nine MDR efflux systems belonging to four families of efflux pumps; resistance-nodulation division (RND) family, multidrug and toxic compound extrusion (MATE) family, ATP binding cassette (ABC) family and major facilitator superfamily (MFS). Here, we show that genetic inactivation of these efflux systems results in an inability to form a competent biofilm. We have used the well characterized AcrAB-TolC system as a model to investigate the hypothesis that inactivation of efflux systems changes the expression of other biofilm related genes. Methods: A panel of 17 single and double mutants lacking components of the major MDR efflux systems of Salmonella were investigated. Biofilms were quantified using various models and under various conditions, the most useful and high throughput of these is the crystal violet biofilm assay. Various phenotypic assays were used to visualise curli (proteinaceous filaments) and cellulose (polysaccharide) production, important components of a Salmonella biofilm extracellular matrix. Differences in expression of biofilm related genes between wild-type and efflux mutants were determined using qRT-PCR. Results: Sixteen of the 17 efflux mutants formed significantly less biofilm than wild-type in all biofilm assays tested. The reason for this biofilm defect was a lack of curli expressed on their cell surface. qRT-PCR showed that this defect in curli expression was as a result of repression at the transcriptional level. In addition to genetic inactivation of the multidrug resistance efflux pumps, chemical inactivation with efflux pump inhibitors (EPIs) PA beta N, CCCP and chlorpromazine, also inhibits the formation of biofilms in the wild-type strain. Inactivation or inhibition of MDR efflux alters global regulatory pathways which mediate repression of curli. Conclusions: These data suggest that inactivation of acrB or tolC triggers a regulation pathway that ultimately decreases transcription of the curli operons and inhibits the formation of mature biofilms. It also shows that this phenomenon is not specific to AcrAB-TolC or even RND efflux pumps but is a generic effect seen with four different classes of multidrug resistance efflux pump.