

Analysis of Polysaccharide and Biofilm Production of the Emerging Cystic Fibrosis Pathogen *Stenotrophomonas maltophilia*

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Stenotrophomonas maltophilia is an emerging Cystic Fibrosis (CF) lung pathogen, which displays high intrinsic resistance to a number of different antibiotics. Additionally, *S. maltophilia* is thought to increase antibiotic resistance by forming biofilms during infection. Biofilm disruption could promote clearance of the microorganism from CF-infected lungs. However, the genetic, biochemical and immunological mechanisms underlying *S. maltophilia* biofilm formation are not well understood. Secreted polysaccharides have been proven to form a vital component of the matrix that surrounds and protects biofilm bacteria. Hundreds of *S. maltophilia* mutants were generated by transposon mutagenesis and screened for strains with reduced polysaccharide secretion. These strains were screened through the use of agar plates containing the polysaccharide-binding dye, Congo red. Experimental controls included a *S. maltophilia* wild-type strain (positive control) and a confirmed polysaccharide deficient mutant (negative control). A Congo red liquid binding assay was utilized to identify the amount of Congo red bound in the samples, which confirmed the amount of polysaccharide present in them. A total of 1,728 mutants were screened with 61 mutants showing reduced polysaccharide production. The mutants were further narrowed down to 8 samples showing the most consistent phenotype. Arbitrary-primed polymerase chain reactions (AP-PCR), followed by sequencing, will be performed on the selected samples in an effort to identify the genes mutated in polysaccharide deficient strains. We will also perform immunological assays in an effort to understand underlying immune responses to *S. maltophilia*. In order to determine the effects of the deletion of *gpmA* in *S. maltophilia*, we will be performing an in-vitro co-culture assay using the wild-type strain and the *gpmA* mutant strain. These studies will yield evidence to the molecular process involved in polysaccharide production, which could lead to mechanisms to disrupt biofilm formation in CF patients.