

The Regulatory Protein AlgR Influences *Pseudomonas aeruginosa* Pathogenesis on Airway Cells **Shubham Chakravarty**¹ and Gregory G. Anderson¹

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One of the primary causes of high mortality in patients with Cystic Fibrosis (CF) is lung infection caused by the Gram-negative bacterium *Pseudomonas aeruginosa*. In the lungs of such patients, this bacterium forms lifelong infections, characterized by biofilm formation. Our goal is to identify factors that influence *P. aeruginosa* biofilm formation in the CF airways. Using a novel model of biofilm formation on cultured human CF airway cells, we found that mutation of the gene *algR* resulted in significant reduction in the ability to form biofilms as well as adhere to CF airway cells. When *algR* gene activity was restored in these deletion mutant strains, by an *algR* complementation plasmid, adherence improved to a level similar to that of the wild type *P. aeruginosa* strain, and biofilm production was also restored significantly. Additionally, we observed the effects of AlgR on transcription of the Type III Secretion System (T3SS), and found that AlgR might influence regulation of T3SS activity through the magnesium transporter protein MgtE. Altogether, our results point to a role for AlgR in biofilm formation on CF airway cells through modulation of T3SS as well as adherence. Through this and additional studies, such as investigation of cross-talk between AlgR and other genes such as MgtE, which is a putative virulence modulator in *P. aeruginosa*, we aim to get a more lucid understanding of the molecular mechanisms responsible for persistence of lifelong *P. aeruginosa* biofilms in lungs of CF patients.

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