

Using Amino Acid Derivatives to Inhibit *Pseudomonas aeruginosa* Biofilm Formation on Cystic Fibrosis Bronchial Epithelia Cells

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Cystic Fibrosis is a genetic disease caused by a mutation which inhibits the proper transport of sodium and chloride ions across epithelium. Improper ion transport results in the accumulation of thick mucus in critical organs such as the lungs, pancreas, liver, and intestines. The genetic mutation is incurable, but treating the symptoms can vastly increase life expectancy. CF patients are often afflicted with bacterial infections which colonize the excess mucus within the lungs. The most prevalent pathogen associated with CF lung infection is *Pseudomonas aeruginosa*, a Gram-negative bacterium found in soil and water. *Pseudomonas aeruginosa* exists in two forms: planktonic (free-swimming) and sessile (immobile within a biofilm community). The planktonic form is about 1,000x more susceptible to antibiotics and immune cells than the sessile form. Biofilm communities of sessile bacteria are protected by an exopolysaccharide layer outside of the cell wall. Small molecules which inhibit biofilm formation or initiate biofilm disassembly can dramatically increase the effectiveness of drugs and the immune system. In order to identify novel biofilm-inhibitory molecules, we assessed the activity of a library of small molecules in biofilm assays. Active compounds were then screened for activity on living Cystic Fibrosis bronchial epithelial cells infected with *Pseudomonas aeruginosa*. Compounds which successfully inhibit biofilm formation without affecting the Cystic Fibrosis bronchial epithelium cells can potentially be a new drug for treating Cystic Fibrosis infections.

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