Cystic Fibrosis (CF) is the most common autosomal recessive disorder in the Caucasian population, with F508del as the most common disease causing mutation. There is considerable variability in the clinical phenotype of CF patients homozygous for F508del. To address this variation, a genome-wide association study was done by Sun et al, discovering the electrogenic amino acid transporter, SLC6A14, as a top modifier of CF (p<1.28x10^{-12}). Li et al found that single nucleotide polymorphisms in the putative promoter region of SLC6A14 were significantly associated with the severity and age of first *Pseudomonas aeruginosa* lung infection in CF patients. From this, experiments were aimed at testing the hypothesis that SLC6A14-mediated amino acid uptake is regulated by bacterial pathogens and in turn regulates bacterial growth and biofilm formation. To test this hypothesis, arginine uptake from the airway surface fluid of non-CF and CF primary bronchial cells, after treatment with purified flagellin from *Pseudomonas aeruginosa*, was measured. In the presence of flagellin, uptake of arginine significantly increased by 26.4% (n=5; p<0.01) and 16.6% (n=6; p<0.0204), in non-CF and CF cultures respectively. This uptake was inhibited by the SLC6A14 blocker alpha-methyl-DL-tryptophan. Additionally, a significant increase in expression of *SLC6A14* was observed using qRT-PCR, following treatment with flagellin in airway epithelial cultures, supporting the hypothesis that infection modulates the expression of this transporter. Currently, the impact of SLC6A14 function on *Pseudomonas aeruginosa* growth on primary airway cultures is being investigated, to better understand the role which SLC6A14 plays in modulating infection and innate immunity in CF airways.