Cystic fibrosis (CF) is an autosomal recessive genetic disorder that results from a mutation in the cystic fibrosis conductance regulator (CFTR) gene. The most common CFTR mutation, ΔF508, is present in approximately 89% of Canadian CF patients. *Pseudomonas aeruginosa* is the predominant pathogen in the lungs of adult cystic fibrosis (CF) patients. This opportunistic pathogen can cause infections using an extracellular or an intracellular mode of survival. The extracellular mode of survival primarily studied in this research will be in the form of biofilms. I hypothesize that the occurrence of extracellular or intracellular modes of survival of *P. aeruginosa* will require distinct mechanisms of bacterial pathogenesis and host response that can be detected using proteomics. These changes will help us understand the bacterial factors that result in the extracellular or intracellular mode of survival and how the host responds. To test this hypothesis, I will complete the following three objectives: 1) identify proteomic changes that occur during biofilm formation on bronchial epithelial cells, 2) identify proteomic changes following invasion of *P. aeruginosa* within bronchial epithelial cells, and 3) determine if intracellular *P. aeruginosa* can exit bronchial epithelial cells and adopt an extracellular lifestyle. This research will aid in understanding the mechanisms of *P. aeruginosa* survival to aid in the development of treatment options specific to each mode of survival.