Infections dominated by bacterial species that exhibit antimicrobial resistance are becoming an increasing concern to global health. *Pseudomonas aeruginosa* is a prevalent, multi-drug resistant pathogen that causes detrimental effects on those with Cystic Fibrosis (CF) by infecting and causing major inflammation in the lungs, eventually leading to respiratory failure. *P. aeruginosa* is known to employ multiple acquired, adaptive and intrinsic mechanisms to produce a complex antimicrobial resistance profile. Of interest is the intrinsic mechanism of producing antibiotic-inactivating enzymes such as β-lactamases, as well as the adaptive mechanism of transitioning from free swimming cells (planktonic) into organized, surface-attached communities called biofilms.

Although *P. aeruginosa* has been studied for many years, there are still many genes that code for proteins of unknown function that require characterization. PA2915 and PA0057 were identified using proteomics to be putative β-lactamases expressed in biofilm and planktonic cultures, respectively. This project aims to characterize these two putative β-lactamases biochemically and phenotypically. First, both proteins will be purified and analyzed for β-lactamase activity using both a chromogenic substrate and clinically applicable β-lactam antibiotics. Next, mutants will be generated in a lab strain and used to create antimicrobial susceptibility profiles for both PA2915 and PA0057. Finally, investigation will continue by examining the individual contribution of each protein to antimicrobial resistance in a highly virulent clinical isolate by using the same biochemical and genetic techniques.

This research will further our fundamental understanding of the *P. aeruginosa* resistance profile, providing novel insight into β-lactam resistance in biofilm and planktonic modes of growth.