Resistance to antibiotic treatments coupled with the decline in antibiotic discovery has resulted in a steady increase in deaths caused by once “curable” bacterial infections. Developing new drugs is crucial to prevent more loss of life in the future. We discovered the compound resazurin exhibits antimicrobial activity against gram-negative bacteria including *Francisella tularensis* (*Ft*), however, certain strains of *Ft* have developed resistance to resazurin. Understanding how *Ft* develops resistance to resazurin will help with defining the mechanism by which resazurin elicits its antimicrobial effect. Whole genome sequencing of resazurin-resistant (*Rzr*) *Ft LVS* mutants revealed four mutations found in 93% of the isolates sequenced. Three mutations were within the coding regions of FTL_0421, FTL_0895, and FTL_1504 and the other mutation was 50 bp upstream of FTL_0445, likely disrupting expression of this gene. The focus of my project was to explore the role of FTL_0895 in resazurin susceptibility. To confirm this gene plays a role in the reduced susceptibility of the *Rzr* strains to resazurin, we cloned the wild-type copy of FTL_0895 into the *Francisella* vector pABST which contains the robust groE promoter of *Ft*. The resulting plasmid will be electroporated into one of the *Rzr* mutants and we will test the susceptibility of the complemented strain to resazurin, using time kill and agar dilution assays. If the susceptibility of the complemented strain to resazurin is restored, then it can be determined that FTL_0895 is a potential target of resazurin.