

JACOB PANCAKE¹, JENNIFER HICKMAN¹, DONALD PRIMERANO², JAMES DENVIR², and DEANNA M. SCHMITT¹. ¹Department of Natural Sciences and Mathematics, West Liberty University, West Liberty, WV, ²Genomics and Bioinformatics Core Facility, Marshall University, Huntington, WV. Determining the role of the *pilD* gene in *Francisella tularensis* susceptibility to resazomycins

Antimicrobial resistant strains (AMRs) of bacteria have become such a severe worldwide issue that the United Nations General assembly met in 2016 to discuss the topic of AMRs. Due to the increase in the number of antibiotic-resistant strains, new therapeutics against AMRs must be developed. Resazomycins are resazurin-based compounds that exhibit antimicrobial activity against *F. tularensis* as well as other Gram-negative bacteria including *N. gonorrhoeae*. The mode of action of these antibiotics is not understood. Approximately half of forty-eight resazurin-resistant *F. tularensis* isolates had a mutation in the genes FTL_0959 (*pilD*) and FTL_1306 (*dipA*). The objective of this project is to determine how *pilD* affects *F. tularensis* susceptibility to resazomycins. In *F. tularensis*, *pilD* encodes for the cytoplasmic membrane peptidase responsible for processing the prepilin subunits in type IV pilin assembly proteins. We are currently working to generate a *pilD* null deletion mutant using standard molecular genetic techniques. We will first confirm the sensitivity of the *pilD* deletion mutant to resazomycins. Understanding the role of *pilD* in resazomycin susceptibility would facilitate further development of these compounds as potential treatments for tularemia and gonorrhea.