AUDREY BLUST & JOSEPH HORZEMPA, Department of Biological Sciences, West Liberty University, West Liberty, WV 26074. Targeted deletion of FTL_1711 to evaluate its role in erythrocyte invasion by *Francisella tularensis.*

Francisella tularensis is a pathogenic intracellular bacterium and the causative agent of the zoonotic disease tularemia. Due to its low infectious dose and capacity to produce severe pneumonic infection, this pathogen has been categorized as the highest priority for national security and public health. Humans can acquire infections caused by this bacterium through exposure to blood-feeding arthropods and contact with diseased animal hosts. Prior research from our laboratory demonstrated that FTL_1199, a Fur-family transcriptional regulator in the Live Vaccine Strain, is required for efficient erythrocyte invasion. Transcriptomic analysis revealed that FTL_1711 is significantly downregulated in the ΔFTL_1199 mutant strain, associating this reduction in gene expression with reduced erythrocyte invasion. Based on these findings, we hypothesize that FTL_1711 contributes to erythrocyte invasion. To evaluate this hypothesis, a targeted deletion of FTL_1711 is being generated using a markerless allelic exchange strategy. DNA flanking the gene of interest was introduced into an unstable plasmid to facilitate homologous recombination, followed by I-SceI–mediated resolution of the merodiploid intermediate. Successful mutagenesis will be confirmed by PCR analysis. Construction of the ΔFTL_1711 strain will enable functional assessment of its role in red blood cell invasion and further define the regulatory network governed by FTL_1199. [This work was supported by the National Institutes of Health, National Heart Lung and Blood Institute (1R15HL147135) and an Institutional Development Award (IDeA) from the National Institute of General Medical Sciences (P20GM103434) which funds the WV-INBRE program].