KRISTEN HAGGERTY, DEANNA SCHMITT, and JOSEPH HORZEMPA, Department of Natural Sciences and Mathematics, West Liberty University, West Liberty, WV 26074. The Role of *Francisella tularensis* PhoH-Like Protein in Erythrocyte Invasion and Pathogenesis

Francisella tularensis is the cause of the disease tularemia. This bacterium usually infects the host via arthropod bites. *F. tularensis* is able to invade mammalian erythrocytes which increases tick colonization following a blood meal. We hypothesized that bacterial genes involved in erythrocyte invasion would be upregulated in the presence of red blood cells. mRNA profiling experiments revealed that FTL_0885 was one of the most highly induced genes in the presence of erythrocytes. We initially attempted to generate deletion and disruption mutants of FTL_0885. However, these attempts were unsuccessful likely due to the proximity FTL_0885 to a neighboring essential gene. A transposon insertion mutation was available in the homolog to FTL_0885 in a related bacterium, *F. novicida*. *F. novicida* is not a human pathogen and its ability to invade erythrocytes is poorly characterized. Therefore, we are reconstructing this mutation in the *F. tularensis* LVS background so that we may study the effects of this gene on erythrocyte invasion. Also, because erythrocytes may contain cues important for pathogenesis, we also were interested in determining whether the FTL_0885 mutant is attenuated. To test this, the mutant bacteria will be evaluated for their ability to replicate in host macrophages in vitro. In addition, the chicken embryo infection model will be used to evaluate in vivo pathogenesis.