ELLE ROBERTS & JOSEPH HORZEMPA, Department of Biomedical Sciences, West Liberty University, West Liberty, WV, 26074. Deletion of *FTL_0671* to determine the role of this gene in erythrocyte invasion by *Francisella tularensis.*

Francisella tularensis is a bacterium that causes the zoonotic disease tularemia. During infection, *F. tularensis* bacteria invade erythrocytes, a phenomenon that enhances the colonization of ticks after a blood meal. To gain insight into the mechanism of erythrocyte invasion, we hypothesized that transcription of bacterial genes important in red blood cell invasion would increase upon exposure to these host cells. An RNA-seq analysis revealed that transcription of 7% of *F. tularensis* genes increased when in the presence of erythrocytes. Of these, we identified three putative transcriptional regulators, namely FTL_0671, FTL_1199, and FTL_1665. The goal of this work was to determine the role of FTL_0671 in erythrocyte invasion by *F. tularensis* LVS. After successfully generating a ΔFTL_0671 strain by homologous recombination, we tested the ability of this mutant to invade erythrocytes using a gentamicin protection assay. As seemingly more ΔFTL_0671 bacteria invaded red blood cells than wild-type LVS, this preliminary experiment suggested that FTL_0671 may restrict erythrocyte invasion. In addition to being a potential transcriptional regulator, FTL_0671 is homologous to PanK, a protein required for CoA biosynthesis in some bacteria. To determine whether FTL_0671 functioned in this capacity, LVS or ΔFTL_0671 was cultured in a chemically defined medium, devoid of CoA. In this experiment, ΔFTL_0671 did not exhibit diminished growth, suggesting that either FTL_0671 does not encode an enzyme required for CoA biosynthesis, or that *F. tularensis* can utilize an alternative pathway to synthesize this critical coenzyme.