

SHANIA DAVIS, MARIAH CASHBAUGH, ELIO DELATORE & JOSEPH HORZEMPA Dept of Biomedical Sciences, West Liberty University, West Liberty, WV, 26074. Analyzing the contribution to pathogenesis by three putative transcriptional regulators of *Francisella tularensis* using the chicken embryo infection model.

*Francisella tularensis* is a highly pathogenic bacterium that can be used as a biological weapon. This bacterium causes tularemia which is also known as Rabbit Fever. Previously, our laboratory identified three *F. tularensis* loci predicted to encode transcriptional regulators (FTL\_0671, FTL\_1199, and FTL\_1665) that exhibited transcriptional changes in the presence of human erythrocytes. We hypothesized that because transcriptional modulations were associated with the presence of host cues, these potential transcriptional regulators may be important for pathogenesis. Therefore, mutants of these three loci and the isogenic wild-type bacterium (strain LVS) were used to infect chicken embryos. Healthy chicken embryos were injected with  $10^4$ ,  $10^5$ , or  $10^6$  colony forming units of each strain, and chicken embryos were candled daily for viability. The chicken embryos were housed in an egg incubator ( $\sim 37^\circ\text{C}$  with occasional rocking) and were observed over a span of 10 days. Neither FTL\_0671 nor FTL\_1199 affected the pathogenesis of *F. tularensis*. However, chicken embryos infected with the FTL\_1665 deletion strain exhibited mortality more rapidly compared to those infected with wild-type bacteria suggesting that this locus may be repressing pathogenesis. Future studies will evaluate the function of FTL\_1665 to determine if the protein encoded by this locus truly mediates transcriptional regulation.