The Role of FTL_1228 in Erythrocyte Invasion by *Francisella tularensis.*

Dylan Anderson, Joseph Horzempa, Department of Natural Sciences and Mathematics, West Liberty University, West Liberty, WV 26074

Francisella tularensis is a pathogenic bacterium that can cause tularemia in mammals through several routes of infection including bites from infected arthropods, through contaminated water or aerosols, and by physical contact with infected animals. Tularemia is a deadly disease with a mortality rate of 60% in untreated cases. Due to the high mortality rate and ease of dissemination, *F. tularensis* could potentially be used as a bioterrorism agent. During infection, *F. tularensis* replicates in phagocytic cells of the immune system, such as macrophages. This bacterium also invades erythrocytes, a phenomenon that has recently been shown to increase tick colonization following acquisition of a blood meal. Identifying factors necessary for transmission and pathogenesis of this bacterium could lead to the development of novel therapeutics. We previously hypothesized that bacterial genes that mediate erythrocyte invasion may exhibit increased expression in the presence of these host cells. RNA-seq revealed that among other genes, FTL_1228 was induced in the presence of erythrocytes. FTL_1228 is predicted to encode the iron-sulfur activator complex subunit, SufD, which was previously shown to be a virulence-associated factor. We are currently in the process of generating both a disruption and a deletion mutant to determine the role FTL_1228 in erythrocyte invasion. ***(Supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence).***