

CARLY CUNNINGHAM, RHIANNON MALCOM, AND JOSEPH HORZEMPA, Dept of Natural Sciences and Mathematics, West Liberty University, West Liberty, WV, 26074. Investigation of the role of Band 3 in red blood cell invasion by *Francisella tularensis*.

*Francisella tularensis* is a pathogenic gram-negative bacterium and the causative agent of the disease tularemia. As an intracellular pathogen, *F. tularensis* can infect and replicate within cells of the immune system, such as macrophages as well as non-immune cells including hepatocytes and epithelial cells. In all of the aforementioned cases, uptake is facilitated by the endocytic machinery and cytoskeletal rearrangements of the host cell. *F. tularensis* is also capable of invading erythrocytes - cells that are incapable of endocytosis or phagocytosis. Therefore, it is quite likely that red blood cell invasion occurs through a distinct mechanism. Unlike the cytoskeleton of the nucleated host cells, erythrocytes are supported through a meshwork of spectrin filaments that are tethered by small actin bundles. We previously showed that actin rearrangement is not required for invasion. However, treatment of red blood cells with a snake venom that inhibits the activity of spectrin abrogates erythrocyte invasion. This venom acts through binding and inactivating Band 3, an anion transporter that complexes with spectrin. We therefore hypothesized that interaction with Band 3 is required for erythrocyte invasion. To test this, we generated Fab fragments from monoclonal and polyclonal antibodies specific to Band 3. These fragments will be used to treat erythrocytes during in vitro assays to determine the role of Band 3 during red blood cell invasion by *F. tularensis*. (Supported by NIH Grant P20GM103434 to the West Virginia IDeA Network for Biomedical Research Excellence and 1R15HL147135).