Abstract

Overview

Over the course of the 2018 Spring Semester, I invested how the mammalian immune system defends against *Legionella pneumophila*, an intracellular bacterial pathogen. In order for *Legionella pneumophila* to replicate, it relies on effector proteins that are delivered into infected cells using a type IV secretion system which inadvertently has been demonstrated to enhance host defense against *L. pneumophila*. However, a loss-of-function mutation in the *legC4* effector gene results in increased replication of *L. pneumophila* with the presence of cytokines (IFN-Gamma) in mammalian lungs. My research this semester was geared to understand the function of *LegC4* and how ∆*LegC4* enhances replication of *L. pneumophila.*

Experiments

At the beginning of the semester using western blots, experiments were geared to see if Δ*LegC4* overexpressing *LegC4* could enhance the JAK-STAT pathway by using the primary antibody a-pSTAT Y701 compared to wild-type *L. pneumophila.* All strains were treated with IFN-G. After these experiments were conducted, a conclusion was made that treating each strain with IFN-G could lead to saturation in the pathway where any type of enhancement could not be seen nor registered. From this conclusion, another experiment was conducted to see if Δ*LegC4* could promote Stat-1 phosphorylation in the absence of cytokines. This was done by using western blots, immunofluorescence microscopy, and cytoplasmic and nuclear extractions. Through different strains, timepoints, MOIs, and primaries, a conclusion was made that Δ*LegC4* overexpressing *LegC4* could not promote Stat-1 phosphorylation in the JAK-STAT pathway in the absence of cytokines. With this conclusion being made, future experiments will include further investigations throughout different mechanisms from the bacterial replication and the

host cell’s defense. These future experiments include screening for transcript abundance (mRNA) and investigating other pathways dealing with antimicrobial defense.