DETERMINING THE EFFECTS OF THE TYPE SEVEN SECRETION SYSTEM IN *STAPHYLOCOCCAL* INNATE IMMUNE INTERACTIONS

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Staphylococcus aureus is an opportunistic pathogen that leads to upwards of twenty thousand deaths and five billion dollars in healthcare-related costs in the United States annually. While many virulence factors within *S. aureus* are well characterized, the type VII secretion system (T7SS), a protein secretion system found within many bacterial species, remains poorly understood. Studies within *Streptococcus intermedius* indicate the T7SS is responsible for secreting proteins that allow the pathogen to evade immune responses. To investigate whether the T7SS in *S. aureus* was important in survival to innate immunity, a *Drosophila melanogaster* sepsis model was utilized. A knockout in the *essC* gene encoding the T7SS ATPase was studied for differences in *D. melanogaster* survival. *D. melanogaster* infected with the *essC* knockout exhibited significantly increased survival compared to flies infected with wild-type *S. aureus*. Survival within a macrophage cell line was explored further to elucidate the mechanism behind the differential survival. RAW264.7 macrophages were infected with an *essC* knockout and wild-type *S. aureus* at a multiplicity of infection of 25. 24 hours post-infection, macrophages infected with the *essC* knockout exhibited a 1-log reduction in bacterial burden compared to macrophages infected with wild-type. To explore the differential macrophage survival, a reactive oxygen species (ROS) assay was performed, as ROS is a primary method of killing within macrophages. No significant difference in survival was found. The T7SS of *S. aureus* remains a poorly understood virulence factor that may play a pivotal role in bacterial survival to components of the innate immune system.