

**ML141 and its Influence on Endothelial Cell and Macrophage Immune Response**

**An Honors Thesis (HONR 490)**

**By**

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May 2019

**Expected Date of Graduation**

May 2019

## **Abstract**

*Staphylococcus aureus* infections are a prevalent infection. Specific strains of *S. aureus*, such as MRSA (methicillin resistant *Staphylococcus aureus*), receive attention due to resistance to many first-line antibiotics, but this may only be part of the problem. Recurrent and persistent infections can be caused by *S. aureus* entering the bloodstream and migrating to secondary sites of infection. Establishment of these secondary sites of infection is caused in part by *S. aureus* invading host cells. Our lab previously demonstrated that ML141, a novel small molecule inhibitor with specificity for human protein CDC42, limited host cell invasion by *S. aureus*. Though CDC42-targeted therapeutics may present a way to limit intracellular *S. aureus*, inhibition of CDC42 may not be an obvious therapeutic option. CDC42 serves a wide variety of roles within human cells, including the regulation of the innate immune response. This work demonstrates that production of immune molecules in macrophage and endothelial cells is sustained in ML141 treated cells, suggesting short-term inhibition of CDC42 may provide a therapeutic approach to infection.

## **Acknowledgments**

I would like to thank Dr. McDowell for her continued support throughout my time within her lab. She has been a great mentor and has provided me with many opportunities that I would not have received if I did not have her mentorship. I would also like to thank Melissa Evans for allowing me to talk through some of the experiments with her.