Plasmodium is a protozoan parasite that causes malaria, an infectious disease transmitted by Anopheles mosquitoes. Like other eukaryotes, Plasmodium possesses microtubules, a component of its cytoskeleton, that are involved in motility, vesicular transport, as well as cellular shape and stability. A prominent shape of *Plasmodium* is the ookinete, a motile, banana-shaped zygote that forms inside the mosquito and facilitates infection of the insect vector. The polarized banana-shape is generated and regulated by microtubule-associated proteins (MAPs). Thus far, very few MAPs have been identified for the malaria parasite. Our lab has identified a novel gene that is a putative MAP: the MAP6-related STOP Axonemal protein-1 (SAXO1). Using a heterologous expression system, we demonstrate that *Plasmodium* SAXO-1 does bind to microtubules.

Utilizing CRISPR/Cas9, our lab successfully generated a mutant Plasmodium strain that lacks the saxo-1 gene (SAXO-1KO), indicating that the gene is not essential. Here we characterize the SAXO-1KO phenotype. We first isolated individual clones, clone A and clone B, and determined their growth curves as well as the morphology of parasites during their development in the mouse blood. Our initial observations show significant morphological differences in the schizont stage, which appear highly disorganized in the SAXO-1KO parasites when compared to the parental wild type parasites. Our follow-up investigation focuses on the organization of the parasite microtubules during development in red blood cells. Our data give new insights into the organization and regulation of the cytoskeleton of the malaria parasite. This will increase our understanding of the parasite’s biology and may lead to novel approaches to combat this terrible disease.

### References


### Hypothesis

We hypothesize that SAXO1 binds to and stabilizes microtubules in the malaria parasite.

### Summary

The SAXO-1 gene is an important microtubule gene related to the cellular shape of the *Plasmodium* parasite. With regards to infections, KO-infected mice tend to reach higher parasitemia than their WT counterparts. However, this conclusion must be further researched as the wildtype parasitemia did not exhibit regular *Plasmodium* infection patterns. There is currently a second trial to account for this difference where a P0 parasite was used for infections. Apart from this, this second trial will also use the tail-vein injection technique to infect mice. Further steps will include staining the microtubules and using fluorescent microscopy to visualize the microtubules. The knockout parasites will also be studied in their sexual stages using live feed techniques to infect mosquitoes. These mosquitoes will later be dissected so that the sexual stage can be studied closer.

Most malaria studies come to the parasite at generating resistance against current treatment options. Further understanding of the cytoskeletal elements of this parasite will aid in future research and finding potential new target sites for antibiotics.

### References


### Acknowledgments

I would like to thank Loyola’s Center of Experiential Learning for the ASPIRE Scholarship, which has helped fund this research. I would also like to thank the generous donors of Loyola’s endowed scholarships, such as the funders of the Reinke Scholarship. Lastly, I would like to thank my fellow labmates for continuous support, namely Lara Ladney, Manuel Widuch, and Aiden Luers. I would also like to thank Dr. Kanzok for his support and mentorship.