

YOU ARE INVITED TO ATTEND THE
DEFENSE OF THE DOCTORAL
DISSERTATION

**“THE NUCLEAR HORMONE RECEPTORS OF *BRUGIA MALAYI* AND
HOW THEY INFLUENCE MOLTING”**

By

Tyler Eck
PhD Biomedical Sciences,
Molecular Biology, Cancer, and Genetics
Bachelor of Science, SUNY New Paltz, 2016
Master of Science, Montclair State University, 2019

Thesis Advisor: Dr. John Siekierka
Adjunct Assistant Professor RWJMS,
Department of Biochemistry & Molecular Biology

Wednesday, March 18th, 2026
10:00 AM
MSB, H609

Join Zoom Meeting:
<https://rutgers.zoom.us/j/91946899289?pwd=8F1EWBFt1rusc0YoSaAZ08tv09YId.1>

Meeting ID: 919 4689 9289
Password: 205801

Abstract

Lymphatic filariasis is a mosquito-borne parasitic infection which is a significant cause of morbidity in the tropical climates of Africa, Asia, and South America. Currently, there are few options available to eradicate disease in patients, and a lack of understanding of the parasite's life cycle impedes the search for novel therapeutics. Many researchers study the third larval (L3) stage of *Brugia malayi*, the laboratory model of Lymphatic filariasis, which gets introduced into the mammalian host, molts, and takes residence in the lymphatics to establish an infection. Therefore, understanding the critical factors involved in the L3 molt will identify new molecular targets to prevent primary infection and eradicate the disease. In a previous study, hormone response elements (HREs) were overrepresented in genes which were activated during the L3 molt implicating nuclear hormone receptors (NHRs) as molting-related transcription factors. Of the 27 NHRs in the *B. malayi* genome, we sought to filter them down to uncover true molting effector genes. First, sequence homology and annotation of P-box residues were used to determine binding to the validated HRE (AGGTCA) left with 13 genes that were expected to bind. Then analyzing public RNAseq databases and qPCR validation found that at least six of these NHRs have significant expression changes during the L3 molt. When these genes were subject to RNAi based knockdowns, we notice three genes (*Bm nhr-23*, *Bm nhr-25*, and *Bm unc-55*) significantly reduced the ability for worms to molt. Finally, we used an NR2F (*Bm unc-55* ortholog) and two rxr-1 modulators to ascertain the validity of using such molecules to reduce worm viability and molting efficiency. By achieving these aims, we have demonstrated the important role of certain NHRs in development and highlight a new therapeutic area for antiparasitics.