## Department of Molecular and Cellular Biology Graduate Seminar MCB\*6500

Friday, Jan. 19, 2018 in SSC 1511 @ 12 noon

presented by:

## **Madison Turner**

(Co-Advisors: E. Guzman / R. Merrill)

## "Identification and characterization of toxic factors associated with disease in *Apis mellifera*"

The European honeybee, Apis mellifera, was globally domesticated for honey production. The contribution of honeybees to food security has recently been recognized, which has raised concerns about hive health. Current bee populations are declining due to a variety of factors, including the parasite, Varroa destructor, and the bacterial disease, American foulbrood. V. destructor is a mite that feeds on the haemolymph, or circulatory fluid of the bee, weakening its host. Overall, the mite reduces hive vitality, potentially leading to colony collapse if left untreated. Interestingly, a synergy between mite infestation and honeybee viruses has been noted. This led to the hypothesis that an immunosuppressive agent is present in mite saliva, which forms the basis of a major objective of this research proposal. American foulbrood is caused by a Gram-positive, bacterial pathogen, Paenibacillus larvae. This highly contagious infection targets the colony brood, killing progeny, which can lead to colony death. Exotoxins produced by *P. larvae* have been identified and characterized, including the mono-ADP-ribosyltransferase toxin, C3larvin. This toxin was deemed non-functional, due to a truncated N-terminus resulting in a loss of membrane-translocating machinery. A related C3larvin toxin, with an extended N-terminus, has recently been identified through genomic sequencing of another *P. larvae* genotype. It is proposed that this toxin is enzymatically active and has the necessary machinery to enter target cells and interfere with cytoskeleton assembly and maintenance. This research project aims to characterize causative agents associated with honeybee disease to facilitate disease management and improve hive health.