# Biomedical Research Seminar Series

## *Speaker Announcement*

**Friday, March 8, 2019 @ 3:30 pm**

**Domenici Hall, Room 109**

Refreshments served at 3:00 pm

## *NMSU Faculty Spotlight: Jennifer Curtiss, PhD*

*Associate Professor, Department of Biology*

### *Using fruit flies to probe the conserved roles of M13 peptidases in animal lipid and carbohydrate metabolism*

In mice and humans, activity of the metalloendopeptidase Neprilysin correlates with obesity and diabetic complications, but little is known about the underlying mechanisms. The fruit fly *Drosophila melanogaster* is an excellent model organism for studying metabolism and metabolic disorders such as diabetes. Flies have an organ that stores nutrients as triglycerides and glycogen, and they produce insulin-like peptides that regulate nutrient storage. Flies lacking the *Nepl15* gene (*Nepl15*$^{ko}$), which encodes a Neprilysin, feed normally but have reduced triglycerides and glycogen compared to controls. Additional data suggest insulin signaling is reduced in *Nepl15*$^{ko}$ flies. Whereas human Neprilysin is membrane-bound and functions at the cell surface, Nepl15 appears to be secreted and catalytically inactive, but may still be able to bind peptide substrates. Another fly Neprilysin, Nep1, appears to be a typical membrane bound, catalytically active Neprilysin. Interestingly, Nep1 has the opposite effect to Nepl15: flies with CRISPR-generated mutations in *Nep1* have increased triglyceride and glycogen levels compared to controls. We hypothesize that Nepl15 can bind target peptides but cannot cleave them, sequestering them from other Neprilysins, thus explaining the opposite phenotypes of *Nepl15* and *Nep1* mutants. Future experiments will help us better understand the role of Neprilysin in human metabolic disorders.

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For more information or to meet with the speaker please contact Ryan Ashley at ryashley@nmsu.edu