# Synaptic plasticity at the *C. elegans* Neuromuscular Junction

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**4:00 PM**
**Seminar Room**

# Workshop Seminar

## Abstract

Although *C. elegans* has been utilized extensively to study synapse formation and function, relatively little is known about synaptic plasticity in *C. elegans*. During the course of my talk I will show that a brief treatment with the cholinesterase inhibitor, aldicarb induces a form of presynaptic potentiation whereby ACh release at neuromuscular junctions (NMJs) is doubled. This aldicarb-induced potentiation is eliminated by mutations inactivating a single neuropeptide (NLP-12). I will go on to describe work that suggests that NLP-12 mediates a mechanosensory feedback loop that couples muscle contraction to changes in presynaptic ACh release, thereby providing a mechanism for the control of locomotion.

During the second part of my talk I will describe a novel function for a *C. elegans* immunoglobulin superfamily protein, RIG-3. Mutants lacking RIG-3 have an exaggerated paralytic response to aldicarb. The heightened drug responsiveness in *rig-3* mutants is caused by an increase in post-synaptic acetylcholine receptor (AChR) abundance. Mutants lacking RIG-3 also have defects in the axonal polarity of ALM neurons. RIG -3's effects on AChR trafficking and ALM polarity are both mediated by changes in Wnt signaling, and in particular by the Wnt receptor CAM-1. Collectively, these results identify RIG-3 as a new regulator of Wnt signaling, and suggest that RIG-3 has an anti-plasticity function that prevents activity-induced changes in post-synaptic receptor fields.