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NEUROSCIENCE SPEAKER SERIES



Karen Ting Chang

**Keck School of Medicine
University of Southern California**

Molecular mechanisms regulating activity-induced synaptic remodeling: a role for secretory autophagy

**Wednesday, April 9th
4:30 PM**

**Seaver Commons RM 102
Pomona College**

Abstract: The ability of neurons to rapidly remodel synaptic structure and strength in response to neuronal activity is highly conserved across species and is essential for complex brain functions such as learning and memory. However, the mechanisms that coordinate these acute, activity-dependent structural changes across synapses remain poorly understood. I will describe our work using the *Drosophila* neuromuscular junction as a model to decipher the molecular pathways regulating activity-induced synaptic remodeling. Using an RNAi screen targeting genes affecting nervous system functions in humans, we uncover that while autophagy is necessary for both synapse development and activity-dependent remodeling, a specialized form of autophagy – secretory autophagy – plays a critical role in synaptic plasticity. This discovery unveils a previously unrecognized paradigm governing synaptic plasticity and provides new insights into how neurons communicate to orchestrate activity-dependent changes.