Summary
Synapses have the remarkable ability to adaptively modulate synaptic strength in response to perturbations that would otherwise destabilize neurotransmission, referred to as homeostatic synaptic plasticity. Homeostatic signaling systems have emerged as robust and potent regulators of neural activity, enabling stable synaptic function while permitting the flexibility necessary for learning and memory, yet the molecules and mechanisms involved remain poorly understood. We have pioneered forward genetic approaches in Drosophila to identify genes required for homeostatic synaptic plasticity. We will first discuss an enigmatic protein complex that has emerged from this screen, which is associated with schizophrenia, and the role of this complex in synaptic function and homeostatic plasticity. We will then present data about how an individual synapse adapts to conflicting homeostatic perturbations to stable synaptic function. Finally, we are developing new tools, including translational profiling and light sheet microscopy, to reveal homeostatic adaptations to synaptic function, which may be linked to sleep, and ancient, essential, and fundamental homeostatic signaling system shared by all animal life.

Bio
Dion Dickman was born in Hawaii and did his undergraduate work at Washington University in St. Louis, studying synaptogenesis at the mouse neuromuscular junction in the lab of Joshua Sanes. He went to Harvard for graduate work and UCSF for his postdoctoral studies, performing electrophysiology-based, forward genetic screens in Drosophila, identifying new genes involved in synaptic development, function, and plasticity. He has recently started his own laboratory at the University of Southern California, where his group investigates how synaptic transmission is kept within stable physiological ranges in the nervous system, while still permitting the flexibility necessary for learning and memory. Using Drosophila as our model system, we are interested in the genes and molecular mechanisms that achieve and maintain the homeostatic control of synaptic strength, and how dysfunction in this process may contribute to neuropsychiatric disease. We are using a combination of genetic, electrophysiological, imaging, and behavioral approaches to gain insight into this complex and fundamental form of neural plasticity.