BIOENGINEERING FALL 2020 Seminar

Date: Thursday, December 3, 2020
Time: 12:00 pm - 1:00pm
Location: Virtual
Join Zoom Meeting—https://gmu.zoom.us/j/92554249038?pwd=V2p1ZUdqM1Y2RnBCcWhDU0V0T2FZZz09
Meeting ID: 925 5424 9038 Passcode: 640851

Inbal Israel, Ph.D.

Biography: Dr. Israely carried out her PhD research at the University of California, Los Angeles, in the laboratory of Drs. Xin Liu and Alcino Silva, where she demonstrated that loss of a neural specific cell adhesion protein linked to disease, delta-catenin, results in cognitive dysfunction. Dr. Israely joined the Tonegawa lab at MIT for her postdoctoral training, where she used two-photon imaging and glutamate uncaging to uncover the learning rules associated with long lasting structural and functional changes at individual synapses. In 2009, she started her own group at the Champalimaud Foundation, and in 2016, the lab relocated to Columbia University, in the Taub Institute for Research on Alzheimer's Disease and the Aging Brain, within the departments of Pathology and Cell Biology and Neuroscience. Her lab has been uncovering dynamic interactions between spines during synaptic plasticity, such as cooperation and competition, that are critical for the long term storage of information in the brain. Dr. Israely's laboratory uses genetic, electrophysiological, optical and molecular approaches to study how activity dependent structural changes shape neural circuits, and how when impaired, these contribute to neurodevelopmental disorders, including autism, and neurodegeneration.

Title: Activity Driven Spine Structural Dynamics

Abstract: Brain circuits can be structurally rearranged with experience, and synaptic connections can grow and be eliminated, even in adults. Dendritic spines are highly dynamic structures whose morphology and lifespan are modified as a response to synaptic efficacy changes between neurons. We combine the precise stimulation of defined inputs with whole cell electrophysiological recordings and imaging, in order to understand how activity influences synaptic structure and function. Using two-photon glutamate uncaging and imaging, we have shown that activity at specific inputs can lead to the production of new proteins, promoting either long lasting growth of single spines, or cooperation and competition between multiple synapses following potentiation. This led us to predict that synaptic competition for newly made proteins constrains the number of inputs that can undergo structural changes during activity, a process that may become dysregulated in several neurodevelopmental disorders. By inducing and following mGluR plasticity at defined spines, we are elucidating interactions that also occur during synaptic depression. Further, we are determining with high precision whether abnormal synaptic competition contributes to the altered micro-circuitry in Fragile X Syndrome, and consider whether this represents a core mechanism of dysfunction across Autism Spectrum Disorders (ASDs). Our goal is to gain an understanding of how diverse forms of activity drive spine interactions, and how these processes influence the refinement of local neural circuits both in health and disease.