PROGRESS IN NEUROSCIENCE PINS

Seminar Series of the
Brain & Mind Neuroscience Institute
Weill Cornell Medical College (WCMC)
&
The Graduate Program in Neuroscience of
WCMC and Sloan Kettering Institute

Thursday, 02/12/15, 3:45 PM
Weill Auditorium

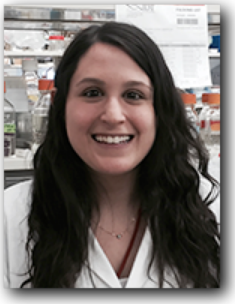
Presentation 1: Reconsolidation update: a novel approach to enhancing fear regulation across development

Dave Johnson, Neuroscience Graduate Student

Fear extinction learning is a context sensitive and highly adaptive process in which an organism learns to regulate cue-driven responding when the predictive value of a cue changes from danger to safety. When this learning process is functionally compromised in humans, it can lead to dysregulated fear expression long after a threat has passed and can set the stage for the emergence of anxiety disorders. Here I will present evidence for diminished extinction learning during adolescence, a developmental stage when anxiety disorders are often first diagnosed. Next, I will present results from a study that successfully tested a behavioral method based on the principles of memory reconsolidation to prevent the recovery of conditioned fear in this developmental group.

Presentation 2: The Role of Estrogen Receptor β in Brain Mitochondrial Permeability Transition

Suzanne Burstein, Neuroscience Graduate Student



The ability of mitochondria to buffer calcium is a critical aspect of neuronal viability, especially in conditions such as ischemic brain injury, in which neurons are challenged with large calcium influxes. We have found that estrogen regulates mitochondrial calcium handling via estrogen receptor β in brain mitochondria, suggesting a novel role of estrogen receptor β in regulating mitochondrial function. We are currently investigating the mechanisms of this regulation as well as its function in neuronal injury models.

Presentation 3: Chaperone Dynamics Perpetuate Parkinson's Disease in human iPSC-derived midbrain dopamine neurons

Sarah Kishinevsky, Neuroscience Graduate Student

Midbrain dopamine neurons selectively degenerate in Parkinson's Disease, yet the underlying environmental and genetic stressors that cause the disease are largely not cell-type specific. Numerous studies suggest that heat shock proteins may perpetuate neurodegenerative events. My goal is to understand the specific role of heat shock proteins in midbrain dopamine neurons during Parkinson's disease progression. I apply the protocol developed by the Studer lab to differentiate human pluripotent stem cells into midbrain dopamine neurons and the chemical tools developed by the Chiosis lab to assay the role of heat shock proteins in these cells.

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