WAYNE STATE UNIVERSITY

The WSU Program for Traumatic Brain Injury Research

presents a Special Topic Seminar

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"The Implications of Traumatic Axonal Injury for Retrograde Neuronal Cell Death and Downstream Target Deafferentation and Repair"

Abstract: Traumatically-induced axonal injury or diffuse axonal injury (DAI) has long been appreciated to be a significant feature of human traumatic brain injury where it plays a major role in any ensuing morbidity. While progress has been made in our understanding of the pathogenesis of this traumatically-induced change and its potential therapeutic modulation, little is known regarding the consequences of this axonal injury for the upstream, sustaining neuronal cell body of origin or the downstream target neurons deafferentated by this same traumatic axonal injury. In this presentation I will focus upon recent studies using various YFP-expressing mice to critically follow DAI-injured neuronal populations to ascertain the fate of their cell bodies of origin while also exploring the consequences of target deafferentation. These studies incorporate advanced bioimaging with multiple electrophysiological and molecular approaches to confirm that the traumatically-induced axonal injury does not result in neuronal cell death. Rather, the neurons maintain their electrophysiological viability, although they do show evidence of atrophy over time. Similar bioimaging and electrophysiological approaches also demonstrate that once disconnected, the downstream targets are diffusely deafferentated, leading to local circuit disruption and the reduction of excitatory postsynaptic potentials. Over time post injury, there is little evidence of adaptive synaptic rearrangement in damaged thalamic targets, although the neocortex demonstrates adaptive neuroplasticity with the recovery of function over a several week posttraumatic course. Importantly however, this neurological recovery is subject to failure upon secondary challenges elicited by other forms of neocortical circuit disruption. Collectively, these DAI-induced changes do not support evidence of posttraumatic cell death. Rather, they strongly support evidence for enduring circuit disruption and/or failure in multiple brain sites. In our estimation, these findings most likely explain the patterns of circuit disruption recently described using functional connectivity MRI in traumatically brain-injured humans.

Date: Tuesday December 11th, 2012
Time: 12:00pm – 1:00 pm
Location: Blue Lecture Hall – Scott Hall
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