Weekly Colloquium
Tuesday, 5/2/2017, 12:30pm, Billings Building – Rosedale Conference Room

"Mitochondrial Structural and Functional Remodeling in Proinflammatory Microglial Activation"

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Research Abstract

Neuroinflammation is thought to contribute to pathology in most neurodegenerative disorders. Microglia, the innate immune cells of the brain, are activated to proinflammatory states in response to damage-associated molecular pathogen (DAMPs) released by injured cells. During activation, microglia undergo a bioenergetic shift from oxidative phosphorylation to glycolysis. Mitochondrial structural remodeling consistent with the possibility of fission accompanies this functional change. Drp1 is the main GTPase that promotes mitochondrial fission. We hypothesized that proinflammatory microglial activation requires mitochondrial structural and bioenergetic changes that depend on Drp1 activity. Mdivi-1, a drug characterized as a Drp1 inhibitor, suppresses proinflammatory markers of microglial activation in vitro. However, surprisingly, it does so without inhibiting mitochondrial structural or functional changes. This talk will describe new targets of mdivi-1 and identify biochemical mechanisms contributing to mitochondrial respiratory suppression during microglial activation.

Recent publications:

