

PH.D. DISSERTATION DEFENSE OF



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Mitochondrial Dynamics in the Demyelinating Cerebellum

Multiple Sclerosis (MS) is an autoimmune demyelinating disease of the central nervous system that leads to significant motor, cognitive and visual disability. Approximately 80% of MS patients have inflammatory demyelination within the cerebellum and present with tremors, impaired motor control, and loss of coordination. While most MS patients exhibit symptoms indicative of cerebellar dysfunction, the pathophysiology of cerebellar symptoms in MS is complex and remains to be elucidated. The purpose of my work was to determine the contribution of mitochondrial pathology in neurodegeneration in animal models of demyelination. Mitochondrial morphology, pathology, and function were assessed in both the autoimmune experimental encephalomyelitis (EAE) and cuprizone (CPZ) models of MS longitudinally throughout disease. We demonstrated a decrease in coupled respiration at peak EAE disease and a decrease in mitochondrial fusion at chronic CPZ. Understanding the pathophysiology of mitochondria dysfunction in mouse models of MS while also elucidating the mechanism of action of remyelinating drugs on mitochondria for neuroprotection will help us identify superior therapeutics and improve the quality of life for patients with MS.

Wednesday, September 8, 2021

10:00AM (PST)

<https://ucr-edu-hipaa.zoom.us/j/91468689589?pwd=aXNUNWpUMWZ1Y05Dc0tESW1qY0pJZz09>