Synaptic Plasticity Impairments in the Post-ischemic Brain

April 4

Tuesday, 12:30 pm

Billings Building—Rosedale Room and Zoom

SPEAKER:

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Host: Dianna E. Willis, Ph.D.

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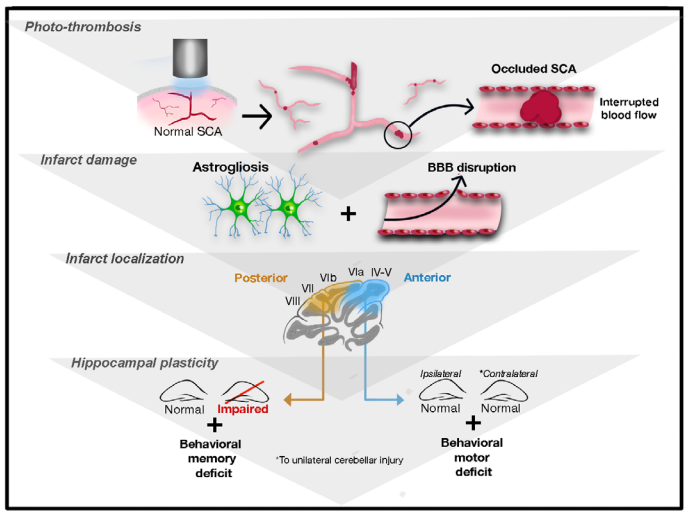
Abstract

Quillinan Laboratory



We are pre-clinical basic science lab that investigates excitability and plasticity changes that contribute to neurological deficits. Our overarching goal is to identify therapeutic strategies to improve neuronal network function that will improve neurological outcome after brain injury.

Ischemia research has largely focused on developing neuroprotective agents to reduce acute neuronal cell death. Our laboratory primarily focuses on dysfunction of surviving neuronal networks and improving synaptic plasticity to restore function at delayed timepoints after injury. We use a multidisciplinary approach that includes in vivo animal models, electrophysiology, molecular analysis of gene and protein expression and virus-mediated gene manipulation. Current projects in the lab are aimed at investigating motor and cognitive deficits after cerebellar ischemia and changes in functional connectivity of the cerebellum with forebrain areas. We are examining thalamic excitability and hippocampal synaptic function after cerebellar stroke using slice electrophysiology. We also study sex- and age-dependent mechanisms that contribute to neuronal injury and plasticity deficits in the hippocampus. We are particularly interested in how sex hormones contribute to injury and repair throughout the lifespan. Experiments utilize hormone manipulations, histological and morphological analysis of hippocampal neurons and viral-mediated gene silencing to interrogate the contribution of sex steroid receptors to cardiac arrest-induced memory deficits.

Publications:

1. *Differences in hippocampal plasticity and memory outcomes in anterior versus posterior cerebellar stroke.* Moreno M, Minjarez C, Vigil J, Orfila JE, Schmidt R, Burch A, Carter DJ, Kubesh M, Yonchek J, Dietz RM, Quillinan N. Neurobiol Dis. 2022 Jun 15;168:105701. doi: 10.1016/j.nbd.2022.105701. Epub 2022 Mar 23. PMID: 35337949

2. *Stepwise disassembly of GABAergic synapses during pathogenic excitotoxicity.* Garcia JD, Gookin SE, Crosby KC, Schwartz SL, Tiemeier E, Kennedy MJ, Dell'Acqua ML, Herson PS, Quillinan N, Smith KR. Cell Rep. 2021 Dec 21;37(12):110142. doi: 10.1016/j.celrep.2021.110142. PMID: 34936876

3. Calcium/Calmodulin-Dependent Kinase (CaMKII) Inhibition Protects Against Purkinje Cell Damage Following CA/CPR in Mice. Chalmers NE, Yonchek J, Steklac KE, Ramsey M, Bayer KU, Herson PS, Quillinan N. Mol Neurobiol. 2020 Jan;57(1):150-158. doi: 10.1007/s12035-019-01765-9. Epub 2019 Sep 13. PMID: 31520314