



**BIOMEDICAL  
SCIENCES**  
PhD PROGRAM

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# DISSERTATION DEFENSE

## MARIE WALTERS

### PhD Candidate

**“Calcium Imaging of Parvalbumin DRG Neurons Provides New  
Tool to Study Proprioceptive Function and Reveals Abnormal  
Calcium Homeostasis After Peripheral Nerve Injury”**

**Thursday, February 28<sup>th</sup>, 2019**

**2:00 p.m.**

**101 White Hall (Gandhi)**

***Advisor: David Ladle, PhD  
Department of Neuroscience, Cell Biology & Physiology***

**Marie Walters, Biomedical Sciences PhD Program  
Wright State University, 2019**

Recovery from peripheral nerve injury (PNI) is dependent on the restoration of proprioception, a sensory modality that provides feedback to fine-tune movement. After PNI, persistent proprioceptive abnormalities limit improvement, leaving patients with disability, social and economic hardships, and decreased quality of life. The goal of this study is to better characterize the mechanisms responsible for proprioceptive deficits caused by PNI. We investigate calcium homeostasis in healthy proprioceptive neurons by using a transgenic mouse model to target expression of a genetically-encoded calcium indicator (GECI) to neurons containing parvalbumin (PV), a calcium-binding protein present in proprioceptors and low-threshold mechanoreceptors. We then utilize this technique to assess calcium homeostasis in two models of PNI known to produce proprioceptive deficits: sciatic nerve crush and sciatic nerve transection and resuture. Our results delineate the average parameters of calcium transients from PV DRG neurons at a population level, describe the diversity in calcium dynamics between cells, animals, and sexes, and illustrate that GECI calcium transients provide sufficient resolution to discern information about the activity of specific subclasses of calcium regulatory mechanisms. Injury experiments expose abnormalities in calcium homeostasis at multiple time-points following PNI and also reveal differences in calcium handling between injury models. Thus, this study establishes aberrant calcium homeostasis as an additional source of proprioceptive dysfunction following PNI. By enhancing our understanding of the mechanisms that prevent complete recovery from PNI, we can better inform research strategies aimed at treatment and improve outcomes for patients who have suffered PNI.