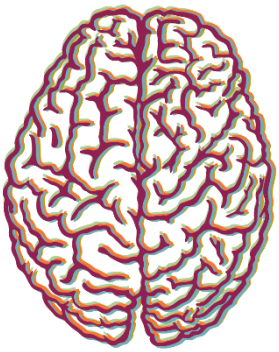

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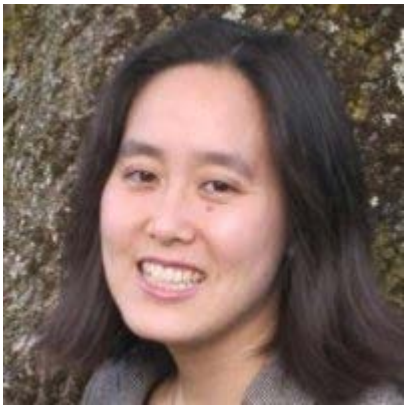

**Joy Sebe, Ph.D.**
**Postdoctoral Fellow**
**University of Washington, Seattle**

# "Calcium Homeostasis and Glutamatergic Transmission at the Mechanosensory Hair Cell Synapse"

**February 09, 2017**
**3:30pm – 4:30pm**
**Kelly Hall 310**

Calcium has a dose dependent role in cellular function serving as both an important signaling molecule and as a cytotoxic agent at low vs. high cytosolic concentrations. Cytoplasmic $Ca^{2+}$ levels are tightly regulated by ion channels that limit entry, organellar $Ca^{2+}$ sinks such as the mitochondria and endoplasmic reticulum (ER) that act as buffers, and pumps that extrude $Ca^{2+}$ from the cell. When $Ca^{2+}$ homeostasis is disrupted, cytotoxic levels of $Ca^{2+}$ are thought to cause neuronal damage and trigger cell death in a host of neurological diseases. Using a combination of electrophysiological, molecular biology and *in vivo* $Ca^{2+}$ imaging approaches, I am examining two aspects of $Ca^{2+}$ homeostasis in a larval zebrafish mechanosensory synapse. First, using a combination of genetically encoded $Ca^{2+}$ indicators targeted to the cytoplasm, mitochondria and ER, I am examining $Ca^{2+}$ flow across cellular compartments in sensory cells in response to physiological levels of mechanical stimulation. Second, the bulk of my work has examined glutamate receptors that mediate transmission across the mechanosensory hair cell synapse and identified a potential $Ca^{2+}$ mediated mechanism for excitotoxic damage and neuronal protection. Lastly, I will present my plans to investigate chronic changes in cellular metabolism that are shaped by activity as well as the role of glutamate receptors in sensory coding. The long-term goal of this work is to identify molecular mechanisms that protect cells and circuits from excitotoxic damage thereby conferring resilience during times of stress.

**Contact Anne Wailes for more information: awailes@vt.edu**