1125-92-2792 **Joe Latulippe*** (jlatulip@norwich.edu), **Derek Lotito** and **Donovan Murby**. *A mathematical model for calcium signaling in neurons in the presence of Amyloid Beta.* Preliminary report.

Alzheimer's Disease (AD) is the leading cause of Dementia and can ultimately lead to death. It is believed that the onset of AD occurs due to intermittent interruption of synaptic transmission between neurons. Regulation of intracellular calcium signaling plays a critical role in neuronal signal transmission. The accumulation of Amyloid-Beta proteins may trigger an increase in intracellular calcium levels by disrupting the regulatory mechanisms within the neuron. Steadily elevated intracellular calcium levels disrupt normal calcium signaling leading to synaptic dysfunction, synaptic loss, memory loss, and neurodegeneration as seen in AD. We present a mathematical model for calcium regulation in neurons in the presence of Amyloid-Beta. A detailed description of the model components is given and compared to experimental data. By investigating the mathematical structure of the model, we systematically determine the key parameters and how they affect calcium regulation. We use numerical simulations to further investigate the model and to determine what it tells us about the biological mechanisms involved in AD. (Received September 20, 2016)