

1135-34-1428

**Joe Latulippe\*** (jlatulip@norwich.edu), Department of Mathematics, 158 Harmon Dr., Northfield, VT 05663, and **Joseph Minicucci**. *A mathematical model for Amyloid beta influenced calcium signaling through the  $IP_3$  receptors*. Preliminary report.

Amyloid beta ( $A\beta$ ) peptides have been linked to the pathogenesis of Alzheimer's disease by altering intracellular calcium regulatory mechanisms. In this study we use mathematical modeling and analysis to study the effects of  $A\beta$  on calcium regulation by specifically tracking the contribution through the 1,4,5-Inositol-triphosphate ( $IP_3$ ) receptor. Our goal is to better understand how various levels of  $A\beta$  affect the  $IP_3$  production cascade and leads to subsequent calcium release through  $IP_3$  receptors on the endoplasmic reticulum. We develop a closed-cell calcium model and show how we calibrated our model to existing experimental data. We also use global sensitivity analysis to understand how model parameters affect solutions. We further investigate the behavior of the model under various parameter regimes and explore how different levels of  $A\beta$  affect model solutions. By linking experimental data with model solutions, we seek to precisely identify the physiological processes in the  $IP_3$  production mechanism that are directly influenced by  $A\beta$ . These results can then be used to better understand the role that  $A\beta$  plays in altering intracellular calcium signals. (Received September 22, 2017)