Contribution ID: **247** Type: **Poster Presentation**

Mathematical model for the effects of Aβ on calcium signalling

Monday, 9 July 2018 19:45 (15 minutes)

Alzheimer's disease (AD) is a devastating illness affecting over 40 million people globally. The accumulation of AD associated Amyloid beta (Aβ) oligomers can trigger aberrant intracellular calcium signals by disrupting calcium regulatory mechanism within neurons. These disruptions can cause changes in homeostasis levels that can have detrimental effects on cell function and survival. Although studies have shown that Aβ can interfere with various calcium fluxes, the complexity of these interactions remains elusive. In order to better understand the impact of Aβ on calcium dynamics, we use a mathematical model to simulate calcium patterns under the influence of Aβ. More specifically, we assume that Aβ affects individual flux contributions through inositol triphosphate receptors, ryanodine receptors, and the plasma membrane. We show that the inclusion of Aβ can increase regions of mixed-mode oscillations leading to aberrant signals under various conditions. We use single and double parameter bifurcation structures to predict model solutions for various levels of Aβ. We further demonstrate that controlling certain biophysically relevant parameters can help control aberrant signalling. These results can be used to suggest possible targets for establishing therapeutic strategies in AD pathology.

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Session Classification: Poster Session

Track Classification: Disease - non-infectious