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Program#/Poster#: 359.15/K7

Title: Activity dependent potentiation governed by presynaptic calcium stores at a tripartite synapse

Location: San Diego Convention Center: Halls B-H

Presentation Start/End Time: Monday, Nov 05, 2007, 10:00 AM -11:00 AM

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We investigated activity dependent plasticity mechanisms in a presynaptic terminal that includes opening of intra-cellular calcium stores and the participation of astrocytes. Our modeling study was carried out with MCell3, a Monte Carlo simulator of cellular microphysiology, in a simplified 3D realization of a hippocampal glutamatergic tripartite synapse (a presynaptic terminal, a postsynaptic terminal and an enveloping astrocytic process). The model incorporates biologically realistic spatial distributions and concentrations of receptors and molecules as well as experimentally validated biochemical pathways and kinetic rate constants. Our simulations suggest that the activation of presynaptic metabotropic glutamate receptors (mGluRs) leads to Inositol Triphosphate (IP3) production and causes additional calcium to be released from the endoplasmic reticulum (ER). The mGluRs can be activated by either glutamate released from the presynaptic terminal or glutamate released from the astrocytes, leading to modulation of synaptic transmission. The consequential increase in presynaptic calcium can also lead to an increase in the release probability of vesicles. This novel form of potentiation can last for several seconds and, as far as we know, cannot be explained by any of the previous standard models for short term potentiation such as the 'residual calcium hypothesis' or the 'bound calcium hypothesis' at the presynaptic terminal.

Disclosures: **S. Nadkarni**, None; **T.M. Bartol** , None; **T.J. Sejnowski**, None; **H. Levine**, None.

Support: NSF PHY0216576
NSF PHY0225630
NIH P01-NS044306
NIH R01-GM068630
HHMI

[Authors]. [Abstract Title]. Program No. XXX.XX. 2007 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2007. Online.