BMC Neuroscience

Oral presentation

Open Access

A bitable synaptic model with transitions between states induced by calcium dynamics: theory vs experiment

Michael Graupner*[1] and Nicolas Brunel[2,3]

Address: [1]Department of Cognitive Studies, Ecole Normale Supérieure, INSERM, Paris, 75005, France, [2]Laboratory of Neurophysics and Physiology, Université Paris Descartes, Paris, 75270, France and [3]CNRS, UMR 8119, Paris, 75270, France

Email: Michael Graupner* - Michael.graupmer@ens.fr

* Corresponding author

from Eighteenth Annual Computational Neuroscience Meeting: CNS*2009
Berlin, Germany. 18–23 July 2009

Published: 13 July 2009

BMC Neuroscience 2009, **10**(Suppl 1):O15 doi:10.1186/1471-2202-10-S1-O15

This abstract is available from: http://www.biomedcentral.com/1471-2202/10/S1/O15

We present a model of a single synapse submitted to trains of pre- and postsynaptic spikes evoking calcium transients. The synaptic efficacy is taken to be proportional to the phosphorylation level of a kinase (e.g. CaMKII) and has two stable fixed points at resting conditions, endowing the system with bistability. Changes of the synaptic efficacy are driven by de- and phosphorylation processes which themselves are driven by calcium elevations. De- or phosphorylation are activated as long as the calcium concentration stays above the de- or phosphorylation threshold, respectively. The response of the synapse model to various experimental protocols known to induce synaptic plasticity experimentally is studied numerically and analytically.

We show that the proposed simple yet biologically founded synapse model reproduces qualitative plasticity results by virtue of two opposing pathways-protein de- and phosphorylation-that activate at distinct calcium levels. In particular, the model explains how: (i) low frequency pre-synaptic stimulations induce LTD while high frequency stimulations induce LTP; (ii) pre before post spike-doublets at intermediate frequency induce LTP while post before pre doublets induce LTD; (iii) only LTD is induced at low frequency by post-pre doublets, and doublets at high frequency induce LTP independent of pre-post timing; (iv) there is an asymmetry between pre-post-pre and post-pre-post triplets, i.e. our model shows potentiation for post-pre-post triplets and no change for pre-post-pre triplets. In a step towards more realistic activity patterns, we investigate synaptic changes induced by pre- and postsynaptic Poisson firing at different rates.

In all these scenarios, we present analytical calculations of transition probabilities between the two stable synaptic states, which allow us to understand in detail how experimental plasticity outcomes are related to the underlying synaptic machinery. We can furthermore make predictions about how changes in the calcium dynamics or the de- and phosphorylation pathways affect synaptic plasticity results. The model demonstrates that synaptic changes driven by calcium transients evoked by pre- and postsynaptic activity patterns reproduce naturally the nonlinearities of observed plasticity outcomes.