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### **Computational modeling of bi-directional communication between neurons and astrocytes**

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According to the classical view of the nervous system, the numerically superior glial cells have inferior roles in that they provide an ideal environment for the function of neuronal cells. Their main function is supposed to be the clearance of extracellular space (ECS) from glutamate, potassium and others neurotransmitters released during synaptic activity. However, recent experimental findings revealed that glial cells are intimately involved in the active control of neuronal activity and synaptic neurotransmission. In particular, it was shown that astrocytes respond to external stimuli with a transient increase of the intracellular calcium concentration or can exhibit self-sustained spontaneous activity. Both evoked and spontaneous astrocytic calcium oscillations are accompanied by exocytosis of ATP and glutamate caged in astrocytes leading to the spread of intra-cellular calcium waves and paroxysmal depolarization shifts (PDS) in neighboring neurons.

We present a mathematical model of the interaction between astrocytes and neurons that is able to numerically reproduce the experimental results concerning the initiation of the calcium waves and PDS.

The timing of glutamate release from the astrocyte is studied by means of a combined modeling of a vesicle cycle and the dynamics of SNARE-proteins. The neuronal slow inward currents (SICs), induced by the astrocytic glutamate and leading to PDS, are modeled via the activation of presynaptic glutamate receptors.

The dependence of the bidirectional communication between neurons and astrocytes on the concentration of glutamate transporters and diffusion of ATP in ECS is analyzed, as well. Our numerical results are in line with experimental findings showing that astrocyte can induce synchronous PDSs in neighboring neurons, resulting in a transient synchronous spiking activity.

**Key Words:** astrocytes, neurons, synchronization, PDS, release of ATP