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Dynamical patterns in brain parenchyma : modelling approach and Issues

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Recent advances in understanding of complex interactions between the processes that maintain and control the physiological state of brain parenchyma open new possibilities and deliver new challenges for modelling studies on topic. Now it is clear that any significant deviation from normal conditions as well as natural alternations of activity of cortical neurons (say, during sleep-wake cycle) are accompanied by measurable changes of physiological parameters, like extracellular ionic concentrations, astrocytic calcium signalling, or vascular tone of adjacent blood vessels. The interaction pathways within the so called neural-glial-vascular unit not only drive the local responses of the cells, but form the spatially extended networks at different functional layers.

Being normally on the sidelines, these physiological mechanisms become dominant during the extreme physiological behaviours of the brain cortex, such as cortical spreading depression, migraine waves, or spreading depolarization events with stroke or injures. There is a clear room and need for relevant modelling studies aimed to better understanding of how all this activity is orchestrated. However, this task is very challenging due to the extreme complexity and heterogeneity of system under study.

The presented study addresses the selected aspects of above problem. Being focused on dynamical patterns, we still keep the balance between the physiological details and dynamically tractable generalized modelling. First, we discuss the dynamical consequences of inclusion of extracellular potassium concentration as a state variable in neuron models. Specifically, this additional variable provide both the self -sustained depolarization, and the additional pathway for cell-to-cell signalling which is also known as “volume connection”. Second, we extend this potassium driven model to the mathematical model of cortical spreading depression which counts the effects of neurovascular coupling and cerebral blood flow redistribution. Our most notable finding here is that the combination of vascular-mediated spatial coupling with local regulatory mechanisms results in the formation of stationary Turing-like patterns during a course of spreading depolarization.

Third, we describe a way to incorporate in model the recently revealed details of astrocyte morphology and functions. This approach includes the algorithms for the creation and computational treatment of spatial patterns resembling the real astrocyte networks.

In conclusion, as an outlook, we discuss how the dynamical redistribution of extra- and inter-cellular volumes could affect the reported dynamics, as well as formulate the issues related to adequate modelling of vascular-mediated spatial interaction.

Primary author: Prof. POSTNOV, Dmitry (Saratov State University, Russia)

Presenter: Prof. POSTNOV, Dmitry (Saratov State University, Russia)

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