Introductory Remarks to Symposium 29

Brain dysfunction upon energy failure: new insights into the role of astrocytes

Sara Eitelmann, Duesseldorf

Ischemic stroke is one of the leading causes of death and disability. In this condition, the brain's energy demands vastly surpass its availability, causing cells to suffer a high level of metabolic stress. Ultimately, this can lead to permanent brain damage, and the main mechanisms of the underlying cell death are well described. Nonetheless, the initial implications of energy deprivation on neuronal and glial function and on the interaction between the two cell types are poorly understood.

In this symposium, researchers of the DFG-funded Research Unit "Synapses under stress" (FOR2975), supported by an international expert, will provide new insights into these early events upon metabolic stress at murine glutamatergic synapses, with a special focus on astrocytic function.
Stefan Passlick will open the symposium by introducing how extracellular dynamics and spread of synaptically released glutamate are altered during acute metabolic stress in the mouse hippocampus. Thereby, he will present results obtained by combining multiphoton imaging of extracellular glutamate with electrophysiology. Tim Ziebarth will proceed by reporting on glutamate events with "plume-like" characteristics in organotypic slices, which appear to drive extracellular glutamate accumulation during chemical ischemia. To complement this, Hil Meijer will present a computational model for ion fluxes at the tripartite synapse. The modelling allows him to relate experimental data of glutamate signals to changes in permeability strengths. His model simulations further suggest some critical factors for energy deprivation to cause pathological cell swelling. Next, Sara Eitelmann will focus on the dynamics of astrocytic gap junctional coupling upon acute energy failure in the mouse neocortex. Using an electrophysiological technique, she uncovered unexpected, rapid changes in astrocytic coupling strength following changes in ion homeostasis. Finally, Ákos Menyhárt will highlight the mechanisms underlying ischemia-induced brain edema formation *in vitro* and *in vivo*. Thereby, he will discuss the correlation between pathophysiological astrocyte swelling and lesion growth upon acute ischemic stroke.

Symposium 29

Friday, March 24, 2023
13:00 -15:00, Lecture Hall 105

Chair: Sara Eitelmann, Duesseldorf

13:00 **Opening Remarks**

13:05 Stefan Passlick, Bonn
SYNAPTIC AND PERISYNAPTIC GLUTAMATE SIGNALING DURING THE ONSET OF METABOLIC STRESS (S29-1)

13:30 Tim Ziebarth, Bochum
SF-IGLUSNFR IMAGING REVEALS "PLUME-LIKE" GLUTAMATE EVENTS DURING CHEMICAL ISCHEMIA IN MOUSE CORTICAL BRAIN SLICES (S29-2)

13:45 Hil Meijer, Enschede, The Netherlands
BIOPHYSICAL MODELLING OF ION DYNAMICS AT THE ENERGY-DEPRIVED TRIPARTITE SYNAPSE (S29-3)

14:10 Sara Eitelmann, Duesseldorf
RAPID CHANGES OF ASTROCYTIC GAP JUNCTIONAL COUPLING DURING ENERGY DEPRIVATION (S29-4)

14:35 Ákos Menyhárt, Szeged, Hungary
NEUROTOXIC ASTROCYTE SWELLING AND DYSFUNCTION UPON THE ACUTE PHASE OF ISCHEMIC STROKE (S29-5)