Abstract #10

# Symposium Abstract: Ionic excitability of astroglia beyond (and towards) calcium in health and disease

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### Content

Astrocytes communicate by intra- and intercellular calcium signals. Calcium signalling, however, is not the only form of ionic excitability of astrocytes. There is now compelling evidence that astrocytes undergo activity-related changes in sodium, protons, and chloride, which directly alter their functions and modulate neuron-glia interaction. Our goal is to provide in depth coverage of these emerging aspects of astroglial signalling. We will present established knowledge and discuss new and exciting findings on the molecular pathways and mechanisms by which transient changes in monovalent ions are induced in astrocytes in different brain regions. We will show surprising experimental results on how ionic excitability beyond calcium directly feeds back onto and modulates the function of astrocytes and determines their interaction with surrounding neurons. Moreover, we will highlight the relevance of astrocytic ion changes for the pathogenesis and for the generation of neural damage in human disease and following ischemic stroke.

Christine R. Rose (Heine University Düsseldorf, Germany) will highlight sodium as a new signaling element, altering the astrocytes' capacity for clearance of glutamate and potassium, feeding back onto neuronal excitability. Sodium loading in the ischemic penumbra *in vivo* results in calcium loading through reverse $Na^+/Ca^{2+}$ exchange (NCX), contributing to ischemic damage. There is also tight link between sodium and pH. Dandan Sun (University of Pittsburgh, U. S. A.) will show that excessive activation of the $Na^+/H^+$ exchanger (NHE1) leads to sodium overload and astrocyte swelling. Deletion of astrocytic Nhe1 reduces infarct size and swelling and improves functional recovery after ischemic stroke, apparently affecting astrocytes as well as neurovascular function. Verena Untiet (University of Copenhagen, Denmark) will discuss new aspects of glial chloride homeostasis, which determines the driving force for GABA and glycine uptake as well as for chloride currents, and regulates volume changes and extracellular potassium buffering. New approaches of imaging chloride *in situ* and *in vivo* will be presented, advancing our understanding of chloride regulation and alterations in human diseases. Alexander A. Mongin (Albany Medical College, U. S. A). will explore the molecular nature and significance of astrocytic volume regulated chloride/anion channels (VRAC), which allow the movement of osmolytes such as taurine and modulate neuronal activity through chloride fluxes via glycine and GABA receptors. Chloride fluxes thus play an unexpectedly important role in neuron-astrocyte communication.

### Acknowledgement

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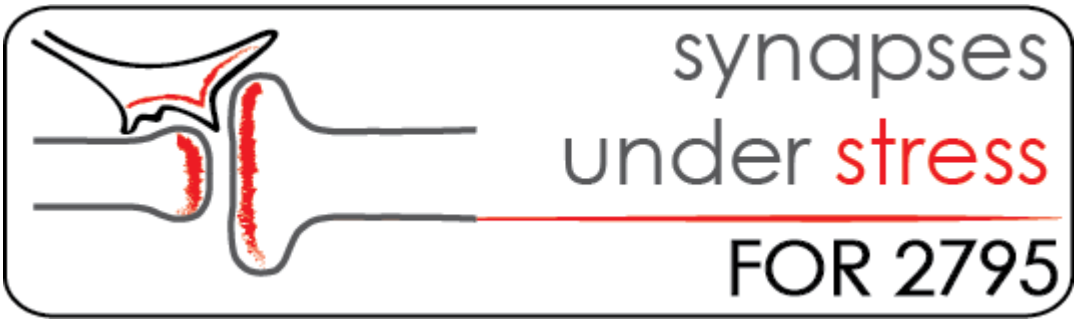


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