

## S3 SEMINAR - ONLINE

### *Calcium signals in glial cell astrocytes modulate synaptic transmission and plasticity in cerebral cortices*

Tuesday May 24, 2022 – 11.00

ONLINE: <https://meet.goto.com/772284917>

#### Speaker

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#### Abstract

Astrocytes, the most abundant glial cells in the brain, have long been recognized for their crucial role in regulating brain tissue development, metabolism, ionic and water homeostasis as well as defense from pathogens or injuries. Information processing was considered to be exclusively a neuronal function in the brain. Recent studies however revealed that astrocytes are very active players that modulate synaptic activity and plasticity, affecting information processing, cognitive functions and behavior. A crucial mechanism for this dynamic astrocyte-neuron interplay is represented by intracellular calcium transients in astrocytes in response to neuronal activity, evoking the release of different molecules from astrocytes that in turn strongly modulate synaptic transmission. Indeed each astrocyte contacts thousands of synapses with ultra-thin protrusions that intimately enwrap synaptic elements, sensing and modulating their activity. Until recently,  $Ca^{2+}$  signaling in fine processes could not be studied in detail due to technical limitations. With the advent of genetically encoded calcium indicators (GECI) and 2-photon laser scanning microscopy (2P-LSM), it is now possible to study  $Ca^{2+}$  events that occur in astrocytic thin processes, the so called calcium microdomains. We used 2P-LSM to study  $Ca^{2+}$  signals in cortical astrocytes in combination with electrophysiological recordings, optogenetics and chemogenetics. We also used new genetic tools to selectively inhibit astrocyte  $Ca^{2+}$  signals in order to unveil their functional contribute to synaptic plasticity and memory retention. Our results show that  $Ca^{2+}$  signals in fine processes are dynamic and complex signals that show specific intrinsic forms of plasticity and play a crucial role in the modulation of synaptic plasticity and memory.

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