PADOVA
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CENTER

31 OCTOBER 2024 3:00 pm

AULA RI

(Complesso Vallisneri, Viale Colombo 3, Padova)

PNC SEMINARS

A talk by Micaela Zonta
(CNR Neuroscience Institute)

FOCUS ON CA^{2+} SIGNALING DYSREGULATION IN ASTROCYTES IN THE PROGRESSION OF ALZHEIMER'S DISEASE

Alzheimer's disease (AD) is a chronic, incurable neurodegenerative disorder, characterized by progressive memory loss. Despite the advances in the research on neurodegenerative diseases, the molecular and cellular mechanisms underlying AD pathogenesis and the early events that anticipate the cognitive decline remain poorly understood.

Our research evaluates the involvement of astrocytes in AD pathogenesis, focusing on astrocyte Ca^{2+} signaling and its dysregulation during AD progression. Indeed, the widely recognized evidence that brain function requires dynamic interactions between neurons and astrocytes implies that both cell types can contribute to brain dysfunction.

We employ two-photon microscopy to perform Ca^{2+} imaging experiments in brain slices and *in vivo* preparations of somatosensory cortex (SSCx) astrocytes expressing GCaMP6f, and electrophysiological monitoring to assess the efficiency of long-term memory processes. We describe significant alterations in astrocyte activity in the familial AD model PS2APP, as well as in astrocyte-dependent long-term plasticity and reveal the molecular mechanisms underlying Ca^{2+} dysregulation. Most importantly, we describe a genetic strategy to rescue astrocyte signals and synaptic plasticity.

Our data identify the dysregulation of astrocyte Ca^{2+} activity as a functional hallmark of early AD stages, revealing a new target to recover AD symptoms.

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Biography

Micaela Zonta is Research Technologist at the Neuroscience Institute of the National Research Council in Padova. She obtained her degree in Biology at the University of Padova and her PhD under the supervision of the CNR neuroscientist Giorgio Carmignoto, investigating how calcium signal in astrocytes regulates gliotransmitter release and functional hyperemia.
Her research group at the CNR Neuroscience Institute in Padua investigates the role of astrocytes in Alzheimer's Disease mouse models and in the modulation of dopaminergic circuits.

Research interests
Our research interests focus on the characterization of Ca^{2+} signals in astrocytes from different brain circuits and in different brain pathological states. To pursue our aims, we combine 2 photon imaging, electrophysiological techniques and immunohistochemistry.
Our recent results reveal the contribution of astrocytes to synaptic modulation in the dopaminergic circuits of Ventral Tegmental Area, and the effects of early astrocyte Ca^{2+} dysregulation on somatosensory synaptic plasticity in a mouse model of Alzheimer's disease (B6.152H).
We are actually studying Ca^{2+} signals in hippocampal astrocytes from α7 nicotinic acetylcholine receptor knockout mice, characterized by an age-dependent Alzheimer's disease-like phenotype, and in parallel we are deepening Ca^{2+} signal characterization in astrocytes from the Ventral Tegmental Area.
In a recently funded Telethon project, we are exploring cerebrovascular function in the B6.152H Alzheimer's mouse model, to disclose possible links existing between the impairment observed in astrocyte Ca^{2+} signal and the different aspects of vascular dysfunction commonly reported in Alzheimer's disease.

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