

GUEST LECTURES

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"A plasma membrane centric view to explain (some) cognitive
defects of the old: how gradual changes in neuronal plasma << membrane lipids lead to synaptic plasticity deficits"

Hosted by: Eleonora Cuboni

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Zoom Login

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Guest Lectures RTG 2413

Abstract

Aging determines the occurrence of changes in a panoply of biochemical / molecular hubs, each contributing to the typical deficits of age. In fact, aging comes with alterations in genomic and nongenomic activities, including among the latter, but not only, defects in mitochondrial function, intracellular trafficking, proteostasis, in the response to cellular stress and in the control of intracellular calcium. What mechanism could act upstream of these defects, responsible for each of them occurring? In other words: is there a "master" change with age, upstream of all (or some) defects that occur with age? A defect that could fulfill the "upstream" role is membrane signaling: it occurs early after cell maturation and follows a gradual course, it occurs in all cells of our organs and tissues and influences genomic and non-genomic activities. Although the bestknown example of defective membrane signalling with age is brain insulin resistance, age is also accompanied by resistance to thyroid stimulating hormone (TSH), to growth hormone, orexin, corticosteroid hormones, fibroblast growth factor and more. Moreover, defective membrane signalling is also evident for neurotransmitter signalling. Therefore, understanding the mechanisms underlying the changes in plasma membrane signalling with age could open avenues of intervention to reduce the sensory, motor and cognitive deficits that occur with age. In my talk I will present data that suggest that small but persistent changes in the lipid composition of the plasma membrane with aging cause different types of receptors to lose synaptic plasticity capacity while -for some of themfavouring survival.

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