ELSC-ICNC Seminar: Arthur Konnerth

January 9, 2013

On the topic of: "Abnormal activity in cortical and hippocampal circuits in Alzheimer's disease"

ELSC & ICNC cordially invite you
to the lecture given by:

Arthur Konnerth
Institute of Neuroscience, Technical University Munich, Germany

On the topic of:

"Abnormal activity in cortical and hippocampal circuits in Alzheimer's disease"

The lecture will be held on Wednesday, January 9, 2013
at 17:00, at ELSC-ICNC: Silverman Bldg., 3rd Wing, 6th Floor, Edmond J. Safra Campus

Light refreshments at 16:45

Abstract:

The accumulation of amyloid-beta in the brain is an essential feature of Alzheimer's disease (AD). However, the impact of amyloid-beta-accumulation on neuronal dysfunction on the single cell level in vivo is poorly understood. The neurodegeneration observed in AD has been associated with synaptic dismantling and progressive decrease in neuronal activity. We tested this hypothesis in vivo by using two-photon calcium imaging in the frontal cortex of a mouse model of AD. Although a decrease in neuronal activity was indeed seen observed in some cortical neurons, a substantial fraction of neurons displayed an unexpected increase in the frequency of spontaneous calcium transients. These "hyperactive" neurons were found exclusively near the plaques of amyloid beta-depositing mice (Busche et al., Science, 2008). When analyzing the visual cortex, we found that a progressive deterioration of neuronal tuning for the orientation of visual stimuli occurs in parallel with the age-dependent increase of the amyloid-beta load (Grienberger et al., Nat. Comm, 2012). As in cortical neurons, there was a marked increase in the fractions of both silent and hyperactive neurons also in the plaque-bearing CA1 region of the hippocampus of old transgenic mice (Busche et al., PNAS, 2012). However, in the hippocampus of young mice, we observed a selective increase in hyperactive neurons already before the formation of plaques, suggesting that soluble species of Abeta may underlie this impairment. Indeed, we found that acute treatment with the gamma-secretase inhibitor LY-411575 reduces soluble Abeta levels and rescues the neuronal dysfunction. Thus, we conclude that hippocampal hyperactivity is a very early functional impairment in AD transgenic mice and that soluble Abeta is crucial for hippocampal hyperactivity.

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