SPEAKER: Jürgen Sandkühler
Center for Brain Research - Medical University of Vienna, Vienna, Austria

TITLE: “Neurogenic neuroinflammation: potential relevance for pain”

ABSTRACT:
Injuries that develop into chronic pain often involve strong excitation of primary afferents, including C-fibres. The initial phase is followed by a prolonged afferent barrage from nociceptors innervating the injured deep tissues. Brief high-, or low frequency discharges in peptidergic C-fibres induce long-lasting increase in synaptic strength (long-term potentiation, LTP) at C-fibres. LTP in nociceptive pathways is considered a cellular model of pain amplification (Sandkühler, 2009). It has long been thought that LTP is induced by Ca²⁺-dependent signalling cascades triggered in superficial spinal dorsal horn neurons in response to the activation of NMDA receptors. Recent evidence suggests, however, that many more cellular players are involved, including microglia, astrocytes, mast cells, T-cells and endothelial cells (Sandkühler, 2013). We recently suggested that the concerted and finely tuned actions of neurons, immune-competent cells and vasculature in response to enhanced neuronal activity constitutes the phenomenon of “neurogenic neuroinflammation” (Xanthos and Sandkühler, 2014). In case of an injury neurogenic neuroinflammation might contribute to the induction of LTP at C-fibre synapses and to pain amplification. Later phases leading to the resolution of neurogenic neuroinflammation may be involved in eventually turning down the spinal pain amplifier.


NEXT UPCOMING SEMINAR: Thursday May 21, 2015, h. 12:00 – Enrico Cherubini, SISSA and EBRI, Dynamics of GABAergic synapses: from transmitter release to receptor activation and plasticity
http://laboratoriobiologia.sns.it/neuroscience-seminars/