

DANDRITE Lecture

Wednesday 14 June 2017
at 10.15 – 11.00

Building 1170, room 347 (Aud. 6), Aarhus University

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Seminar on “The good and the bad of NMDA receptor induced calcium signaling”

The dialogue between the synapse and the nucleus controls activity-driven gene transcription and is vital for virtually all adaptive responses in the nervous system including the build-up of a neuroprotective shield, the formation of memories, but also unwanted adaptations such as chronic pain or addiction. Calcium signals generated by synaptic activity and the opening of synaptic NMDA receptors and voltage-gated calcium channels serve as initiators of this communication pathway. They also mediate the propagation along the synapse-to-nucleus axis, although additional protein-based transport processes, such as the ERK-MAP kinase cascade, play a role (Hagenston and Bading, 2011). Nuclear calcium transients represent an important signaling endpoint in synapse-to-nucleus communication and function as master switch for adaptations-associated transcription. Blockade of nuclear calcium signaling in hippocampal neurons eliminates ‘acquired neuroprotection’, an activity-driven form of adaptation in which neurons that have been electrically activated are more resistant to harmful, cell death-inducing conditions. Similarly, the consolidation of memories and their extinction, as well as the development of chronic pain in mice is critically dependent on nuclear calcium signaling (Bading, 2013). In my presentation I will outline the features of the synapse-to-nucleus communication axis, discuss its genomic targets, and summarize how in neurodegenerative conditions this transcription-promoting axis is being antagonized by a cell death promoting signaling pathway activated by extrasynaptic NMDA receptors (Hardingham and Bading, 2010; Bading, 2017).

Host: Group Leader Poul Henning Jensen, DANDRITE, Dept. of Biomedicine, Aarhus University