NILS SEMINAR

"New aspects of calcium-related proteins in neurodegenerative diseases"

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To date, a number of evidences suggest that perturbations of intracellular calcium homeostasis and calcium-related proteins play an important role in synaptic dysfunction and neuronal cell death in neurodegenerative diseases. Calsenilin, a member of the neuronal calcium sensor family of calcium-binding proteins, has been shown to have multiple functions in different subcellular compartments through serving as a presenilin-interacting partner, a calcium-dependent transcriptional repressor (DREAM) and a direct modulator of A type potassium channels (KChIP3). Calsenilin has been implicated in the regulation of presenilin-mediated calcium signaling, ER calcium signaling, amyloid precursor protein (APP) processing, and apoptotic signaling in neuronal cells. Our recent findings also demonstrate an important role of calsenilin in PS1/γ-secretase-mediated N-cadherin processing and in β-catenin nuclear signaling. Peptidylarginine deiminases (PADs) are known to be directly affected by calcium homeostasis and convert peptidylarginine to peptidylcitrulline (protein citrullination) in a calcium-dependent manner. Abnormal accumulation of citrullination and/or upregulated PAD2 have been reported in a number of human diseases including multiple sclerosis, rheumatoid arthritis, and Alzheimer's disease. Recently we have reported that abnormal elevation of PAD2 and citrullinated proteins were involved in pathogenesis of the brains from scrapie-infected mice and sporadic Creutzfeldt-Jakob disease patients. Thus, these findings suggest that abnormal calcium-mediated processes by the calcium-dependent signaling molecules may lead to neuronal dysfunction and/or cell death in the central nervous system.

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