Time course of early pathophysiological changes and biomarkers in Alzheimer´s disease and related disorders- implications for preventive therapy.

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Research Abstract
Alzheimer’s disease (AD) the most common form of neurodegenerative disorder is characterized by slowly progressive memory loss and independency. There is presently no cure of AD. Amyloid (Aß) is a key factor in the histopathological features of AD but other pathological

processes as neuroinflammation, tau pathology most probably pay an import role. The focus of this application is to characterize and further understand the pathophysiology, possible relationships and their influence on neuroplasticity and regeneration of brain. A further understanding of time course of evolution of Aß forms, inflammatory processes and influence on neurotransmission, and cognition will help us to understand and to develop new earlier diagnostic biomarkers and disease modifying and preventive therapy. Longitudinal multi-tracer PET studies in subjects belonging to families with high risk to develop AD show presence of fibrillar Aß in brain years before onset of symptoms and even much astrocytosis is detected. A major goal of our research is to understand the underlying mechanisms and to develop new PET biomarkers and drugs that may protect, regenerate nerve cells and thereby increase the neuronal communication in brain. Multi-tracer mikroPET studies in transgenic AD mice as well as CSF are used to understand the processes further. New strategies are warranted to promote neurogenesis and to utilize cellular and AD animal models to investigate factors regulating neurogenesis.

Further information available at:

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