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Editorial

Imaging AD Pathology and What can be Concluded

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The correlation between Alzheimer´s disease (AD) and the occurrence of extracellular amyloid-β (Aβ) plaques remains controversial, however, it is agreed that the plaques are not beneficial for brain's health [1]. To observe protein aggregation pathology in AD brains several imaging methods have been used, among them positron electron tomography (PET) and multiproton imaging. By PET studies using [18F]2-fluoro-2-deoxyglucose (FDG) a well-defined pattern of glucose hypometabolism in Alzheimer's disease (AD) was shown [2]. Multiphoton *in vivo* imaging of transgenic mouse models of AD enabled researches to follow pathological aggregation of proteins and the subsequent neuropil neurodegeneration and recovery after therapeutic interventions. It has been concluded that synapse loss, rather than Aβ plaques or tau made neurofibrillary tangles seems more predictive for cognitive decline in AD brain [3].

To further enlighten this issue, high-Resolution 3D Reconstruction (HR-3D) volumetric imaging was applied [4] comparing Tg19959, wild-type, and Tg19959-YFP mice models of AD as a function of age. Thioflavin S staining and antibodies against Aβ, and other structural and synaptic neuronal proteins were applied to show fibrillar deposits and other pathological features. The volumetric imaging of hippocampus of the AD transgenic mice showed intraneuronal onset of Aβ42 fibrillization within cell bodies, neurites, and synapses before plaque formation. Early fibrillar Aβ could be observed within synaptic compartments. Fibrillar Aβ aggregates could be seen piercing the cell membrane. These data support the notion that Aβ fibrillization leads to disruption of cytoarchitecture and degeneration of spines and neuritis [4].

Recently, not only Aβ aggregates but also hyperphosphoryllated tau aggregates prevention by targeting a critical kinase, has been shown as a way forward towards therapy of AD (paper in Science Reports (Site-specific phosphorylation of tau inhibits amyloid-β toxicity in Alzheimer's mice: by Ittner a et al.)

So, it is likely that both these proteins (Aβ and tau) aggregation should be targeted to stop disease progression.

References

1. Glenner GG, Wong CW, Quaranta V & Eanes ED. The amyloid deposits in Alzheimer's disease: their nature and pathogenesis. Appl Pathol. 1984; 2: 357-369.
2. Carbonell F, et al. beta-Amyloid is associated with aberrant metabolic connectivity in subjects with mild cognitive impairment. J Cerebr Blood F Met. 2014; 34: 1169-1179.
3. Kopeikina KJ, et al. Synaptic alterations in the rTg4510 mouse model of tauopathy. J Comp Neurol. 2013; 521: 1334-1353.
4. Capetillo-Zarate E, et al. High-Resolution 3D Reconstruction Reveals Intra-Synaptic Amyloid Fibrils. American Journal of Pathology. 2011; 179: 2551-2558.

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