Synaptic plasticity on different stages of processes of Alzheimer disease

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Abstract: Objective Alzheimer disease (AD) is the most common form of dementia which is pathologically characterized by extracellular β amyloid (Aβ) accumulation and intracellular neurofibrillary tangles caused by phosphorylation of tau. The learning and memory deficits are featured clinical performances, which is probably due to synaptic plasticity dysfunction. Since the synaptic plasticity is considered the cellular mechanism of memory formation, it is necessary to clarify the checkpoint of synaptic plasticity decline in early stage of AD process. Methods We used 1 month-old, 2 month-old and 6 month-old 5XFAD mice to detect long-term potentiation (LTP) in dentate gyrus (DG) of hippocampus. Results The attenuation of LTP in DG of 5XFAD mice was found at 2 months of age, at which Aβ deposition was not detectable both in cortex and in hippocampus of the mouse model. While LTP at 6 months of age in DG of the AD mouse model cannot be induced. The basic synaptic transmission and presynaptic transmitter release in hippocampus of these AD mice at any month old showed no changes compared to wild type mice. Conclusion The decline of postsynaptic strength may be a leading event that precedes the Aβ accumulation in AD process.

Keywords: Alzheimer disease, 5XFAD mouse model, synaptic plasticity, LTP