Abstract :

Choline is being used in all mammalian cells as a precursor for synthesis of a major phospholipide phosphatidylcholine and as a donor of acetyl residues. Cholinergic neurons in addition require choline to synthesize neuromediator acetylcholine. The ability of cells to create choline via de novo synthesis is limited and therefore they need to transport choline from extracellular space. Limited availability of choline in brain leads specifically to diminished function of cholinergic neurons and in general to impaired reparation of biological membranes. Dysfunctions of cholinergic signaling in brain is characteristic for Alzheimer‘s disease.

Aim of this work was to investigate whether gene and protein expression of high-affinity cholinergic transporters is altered in 5-6 months old APPswe/PS1dE9 mouse model of Alzheimer’s disease. Expression of specific high-affinity cholinergic transporter CHT1 (responsible for transport of choline to be used for acetylcholine synthesis) and putative high-afinity choline transporter CTL1 (generally present in all cells and related to high affinity choline transport for phospholipide synthesis) in cerebral cortex was measured. Compared to non-trangenic littermates, no changes in the expression of both genes were detected at either mRNA (quantitative PCR) or protein (Western blot with antibody detection) levels. These findings indicate that the decrease in evoked acetylcholine release observed at this age is not due to reduced expression of these transporter proteins.

Keywords :

Amyloid β
Alzheimer’s disease
Choline
CTL1
CHT1
Neuroscience