Abstrakt (anglický jazyk)

The most common senile dementia, Alzheimer disease (AD), is characterized by a decline of memory and high cognitive functions. Typical post-mortem brain lesions are extracellular amyloid deposits, intracellular neurofibrilary tangles and ruined cholinergic and other neurotransmitters systems. Connection between damaged central cholinergic system and beta-amyloid accumulation remains obscure. We examined parietal cortex of young adult (7-month-old) female APPswe/PS1dE9 double transgenic mice which develope beta-amyloid fragments at high rate. Cholinergic synapses of these mice demonstrate functional presynaptic (stimulated acetylcholine release) as well as postsynaptic (muscarinic receptor-induced G-protein activation) deficits and reduction of cholinergic markers. The mRNA levels of choline acetyltransferase, vesicular acetylcholine transporter and M_1 to M_4 subtypes of muscarinic receptors were determined in transgenic and littermate controls using qPCR. Obtained experimental data does not show any changes in measured mRNA levels. These observations indicate that reduction of cholinergic synaptic markers and function is due to posttranscriptional events.