Alzheimer's disease is a degenerative brain disorder. The incidence of the disease increases with age and every year the total number of affected persons is higher. The malfunction of various mental functions is a typical feature of Alzheimer's disease, including loss of short-term memory and development of personality changes. It is now generally accepted that the main cause of the disease is increased production of r3-amyloid fragments that mediate toxic effects and lead to r3-amyloid plaques formation. Alzheimer's disease and also normal aging are accompanied by a loss of cholinergic neurons and weakened cholinergic neurotransmission. In my thesis I dealt with changes in the brain chol inergic system during aging in control mice and in a mouse model of Alzheimer's disease (APPswe/PS1 dE9). In this context I also investigated in vitro influence of docosahexaenoic acid (w-3 essential fatty acid) changes in membrane cholesterol content on the expression of cholinergic phenotype in the NG108-1 5 cholinergic cell line. I also performed ex vivo experiments on rat brain cortex to investigate the characteristics of action of muscarin ic agonist xanomeline that was developed as a selective muscarinic agonist to strengthen signalization through the muscarinic M1 receptor in Alzheimer's disease. The experiments on APPswe/PS1dE9 transgenic mice producing increased amount of r3-amyloid demonstrated a decrease of some cholinergic markers and functional damage of muscarinic neurotransmission. These changes were already apparent in young animals at the time when increased production of r3-amyloid and plaque pathology just started to appear. The disorders of cholinergic markers were present in both presynaptic and postsynaptic compartments of cortical cholinergic synapses. The general neuroprotective effects of docosahexaenoic acid was confirmed in a model of cholinergic dedifferenciation in the NG 108-15 cell line cultured in medium without serum.