Amyloid β peptide is cleaved from the amyloid precursor protein by β and γ secretases. According to the amyloid hypothesis i tis the main cause of the early pathogenetic events of Alzheimer's disease (AD) which is the most common neurodegenerative disease in the world without an effective treatment. The main pathogenesis of AD is considered to be the loss of synapses, disruption of neuronal plasticity and neurodegeneration. Amyloid β can bind directly to the membrane or mediate neuronal damage indirectly via toxic inflammatory mediators (e.g., reactive oxygen intermediates, nitric oxide and cytokines) by activating microglia and astrocytes. In addition to interacting with various membrane receptors, Aβ can also bind to the cell surface directly, disrupting membrane integrity or forming selective cation channels. This thesis summarizes key interactions with membranes of synapses and mechanisms of amyloid-induced toxicity through receptors.