Immunotherapy by stimulating of autoantibodies is promising methodical approach for Alzheimer's disease treatment. Autoantibodies can be directed against β-amyloid peptide or against neuronal β-amyloid receptors. Some of scientists supposed that the prion is one of the possible β-amyloid receptors and upon the binding to prion β-amyloid begins to accumulate in the brain tissue leading to the neuronal death. We proposed that anti-prion antibodies will prevent the interaction between β-amyloid and the prion protein and will induce the therapeutic effect in the animal model of sporadic Alzheimer's disease. We studied the ability of keyhole limpet hemocyanine conjugated prion synthetic fragments 95-123 and 203-229 to induce an immune response and to protect memory in olfactory bulbectomized mice which demonstrate abnormalities similar to Alzheimer's disease. Vaccination only with conjugated fragment 95-123 was shown to rescue spatial memory tested in the Morris water maze and increase the β-amyloid level in brain tissue of bulbectomized mice. Antibodies against peptide 95-123 were revealed in blood serum and cerebrospinal fluid in the experimental animals. It was shown that immunization with fragment 95-123 improves the morphofunctional state of neurons in the temporal cortex and hippocampus in bulbectomized mice. Control compounds were inactive in all performed tests. We suppose that anti-prion antibodies could be used to develop new therapeutic agents for Alzheimer's disease treatment. The revealed therapeutic activity of anti-prion antibodies is an additional argument for possible role of prion protein in Alzheimer's disease pathology.