**Abstract**

Alzheimer's disease is a chronic, progressive, neurodegenerative disease. It belongs to the most common type of dementia and worldwide it is statistically the fifth cause of mortality. The most common morphological markers are insoluble β amyloid plaques, hyperphosforylated tau proteins and formation of neurofibrilar tangles. Among the manifestations of the disease is amyloid angiopathy, synaptic transmission disorders and subsequent apoptosis, deterioration of cognitive functions and brain atrophy. Studies have shown that administration of mesenchymal stem cells (MSC) has an immunomodulatory effects and it can reduce the production and storage of β amyloid and thus improve cognitive functions. In preclinical studies, which are conducted in transgenic mice and often use xenografts, administration of immunosuppresion may lead to variety of positive or negative effects which can affect the results of the experiment. The subject of the master‘s thesis was to determine the effect of immunosuppression on experimental therapy with MSC in various time windows of AD progression (model 3xTg). At which scale and combination of immunosupression will influence the cell therapy's effects, the length of graft survival, mortality of experimental animals and changes at the cellular level. We have also assessed whether immunosuppression is essential for cell therapy. Our result showed that repeated administration of Wharton Jelly MSC in combination with immunosuppression did not increase cell survival in host ventricles, cells alone reduced β amyloid plaque formation in animals 9-12 months old and reduced incidence of TAU-5 protein in animals 13-15 months old. These changes did not improve memory in Morris Water Maze. Furthermore we observed the effect of cell therapy and immunosuppression on the expression of inhibitory synaptic density (PSD 95) and the expression of excitatory synaptic markers were not affected . The overal mortality of immunosuppressed animals was 30-40 % and represents a burden for transgenic animals that is not balanced by better results.

**Key words:** Alzheimer‘s disease, β amyloid, tau, mesenchymal stem cells, imunosuppression, transgenic model, memory deficit