Abstract

Tauopathy is accompanied by both loss of neurons and synapses. The neuronal loss is irreversible with very low chance of functional replacement therapy. However, lost synapses could be restored with proper stimuli. Perineuronal nets (PNNs) are serving as a protecting barrier for neurons, on the other hand they are significantly decreasing the synaptic plasticity. Temporary disintegration of the PNNs by enzymatic therapy might lead to rewiring and accelerate processes of memory and learning. Model of Cold Induced plasticity leads to the withdrawal of significant number of synapses across the brain. The recovery of these could be followed in healthy and diseased animals. Moreover, it can stimulate Cold shock protein dependent neuroprotective mechanisms. This master thesis is focused on these two forms of synaptic plasticity models; forced remodeling of PNNs and model of cold induced synaptic plasticity. Both will serve as a tool to modulate processes of memory and learning in the P301S tauopathy, in mice. In detail, the work will follow changes in the number of synapses at the region of CA1 of hippocampus and synaptic protein levels at level of whole hippocampus and behavioral recovery of pre-trained long-term memory task dependent on dorsal hippocampus.

Key words: Perineuronal nets, aggrecan, cold-induced synaptic plasticity, memory, hippocampus