

The central nervous system of higher vertebrates, in contrast to the peripheral one, doesn't regenerate. That is because of the presence of many growth inhibitors produced by a glial scar and oligodendrocytes; the most important inhibitors are MAG (myelin-associated glycoprotein), OMgp (oligodendrocyte-myelin glycoprotein) and mainly Nogo protein. Nogo-A is one of three isoforms of the Nogo protein located primarily in the brain and the spinal cord where it causes the degradation of growth cones, inhibits the growth of neurites, restricts the neuroplasticity and prevents the regeneration of injured axons in adulthood. The Nogo receptor complex serves for a reception of signals and the following signal cascade causes the destabilisation of actin filaments. There are also other receptors for Nogo-A, e. g. the PirB receptor. During the development, Nogo-A is highly expressed by neurons but in adulthood, the main producers are oligodendrocytes. It is noteworthy, that neuronal expression of Nogo-A doesn't decrease after birth in structures with high plasticity, e. g. in the hippocampus which is important especially for spatial learning and memory. In the hippocampus, Nogo-A keeps a balance between the synaptic plasticity and stability and restricts the long-term potentiation. Therefore, this bachelor's thesis presents information about the topics mentioned above and also about some behavioral methods focused on learning and memory in Nogo deficient models and animals with an acute inactivation of Nogo-A protein; namely Morris water maze, two-way active avoidance learning, water T-maze, prepulse and latent inhibition tasks. These tests have brought interesting results and also indicate a possible association of the Nogo deficiency with schizophrenia.