AMPAR and NMDAR are two of the most important ionotropic glutamate receptors expressed on postsynaptic densities of the neurons, where they modulate synaptic transmission and plasticity. Many evidences indicate that the function and membrane trafficking of the two receptors are regulated by their subunit composition and posttranslational modifications. There are also plenty of data reveal that the spinal cord injury changes the expression pattern of two receptors and they are involved in controlling motor circuit and hindlimb locomotion.

Therefore, the aim of the project is to understand the roles of AMPAR and NMDAR in the recovery process after spinal cord transection. In the proposed project we plan to characterize the subunit composition and phosphorylation state of glutamatergic AMPA and NMDA receptors on postsynaptic membrane of spinal cord α-motoneurons (MNs) in rats with complete spinal cord transection (SCT) and after treatment of spinal animals with brain-derived neurotrophic factor (BDNF), an important regulator of neuronal development. We plan to build a scheme of the pharmacological treatment based on the understanding how the AMPAR and NMDAR are involved in changing excitability of spinal MNs under these two conditions. In order to verify presented hypothesis, I will quantify and visualize the distribution and protein level of the two receptors on selected pools of MNs.

In the word, many people suffering paralysis caused by impairment of spinal cord. Increasing number of kinds of accidents result in spinal cord injuries leading to permanent impairment of motor functions. Thus, the very important task of neuroscience is to understand and explore processes underlying motor dysfunction mechanisms and possible ways to limit it as it may serve implementation of new rehabilitative methods after spinal cord injury leading to improvement of the function. The proposed project should reveal (1) which subunit and distribution of AMPAR and NMDAR are the most affected by SCT and (2) whether we can counteract changes evoked by the lesion by manipulating of BDNF availability. Finally, the analysis of locomotion after SCT and after SCT followed by BDNF treatment will allow us to evaluate functional effects of this treatment indicating a novel therapeutical pathways.