

The ability to rearrange neuronal networks in response to environmental stimuli is one of the most remarkable and fascinating features of the brain. Modifications in neuronal networks are critical for learning and memory processes. However, the aberrant plasticity mechanisms are also basis of several neurological diseases such as chronic pain, schizophrenia, mental retardations or epilepsy. It is therefore crucial to understand the mechanisms which drive aberrant changes in neuronal connections.

Neuronal plasticity largely depends on modifications in the shape of neurons. Modifications in structure of neurons enable formation of new connections or disruption of existing ones. Changes in the neuronal shape are tightly regulated on the molecular level. There is number of proteins involved in this process. One of proteins recently implied in modulation of neuronal morphology is Lipocalin-2. Interestingly, it has been shown that this protein is produced in the brain during multiple pathological states including inflammations, spinal cord injury, chronic stress or epilepsy. Moreover, research conducted in our laboratory showed that the level of Lipocalin-2 is increased in the brain during chemically induced pathological neuronal plasticity. In this project we hypothesize that Lipocalin-2 is involved in formation of aberrant neuronal connections. To test this hypothesis we will check the influence of Lipocalin-2 on the plastic changes observed during development of epilepsy. We will also identify the mechanism of the Lcn-2 mediated alterations in neuronal morphology. Consequently the project will contribute to the better understanding of processes occurring during a number of brain pathologies.