Neurodevelopmental disorder is a group of conditions leading to a number of serious disorders such as schizophrenia, autism, and mental retardation. Often the disease is caused by alterations in the numbers and shapes of dendritic spines, structures where excitatory synapses' are located. The neuronal morphology is transcriptionally regulated. However, the precise mechanisms and transcription factors involved in the neuronal development are poorly understood.

The advancements of human genome sequencing led to the identification of single nucleotide polymorphisms in genes coding transcriptional activators MKL1 and MKL2. These modifications have been associated with greater susceptibility to neurodevelopmental diseases such as schizophrenia or autism. Also, the inhibition of MKL1 and MKL2 activities led to changes in neuronal morphology during development.

To elucidate the role of MKL1/2 in synapse development and its contributions to neurodevelopmental disorders, we will study transcriptomic signature underlying MKLs depletion and electrophysiological features of neurons *in vitro*, and finally, behavioral phenotypes of adult animals lacking MKLs expression. Our research may provide a molecular basis for the treatment of neurodevelopmental disorders in the future.