Can we boost our learning and memory? Why adult brain becoming less plastic with age? Our research project will attempt to answer this intriguing questions. The main goals of the proposal is to characterize detailed cellular mechanisms of 1) expression of the plastic change induced by sensory deprivation within somatosensory barrel cortex and 2) beneficial action of one of the most essential neurotransmiter in the central nervous system, acetylcholine, in adult neuroplasticity. Whisker-barrel cortex system is an attractive model for studying neuroplasticity because of well-defined connections, in which every whisker on the snout has its own cortical representation. Acetylcholine is known to stimulate neuroplastic processes but its precise cellular mechanism of action remains elusive. Processing of information in neocortex requires the activity of exctitatory cells, which are strongly modulated by inhibitory interneurons. One of the class of inhibitory cells, namely somatostatin (SST) interneurons, which role was undervalued for a long time, now are thought to play a crticial role in many cortical processes. We will determine whether selective shutting off the somatostatin interneurons with state-of-the-art chemogenetic technique will influence the expression of the plastic change in somatosensory cortex in adult animals. Moreover, using viral overexpression approach we will determine if SST interneurons mediate cholinergic improvement of adult neuroplasticity in barrel cortex. We decided to tackle this scientific problems because current state of the art within this field is highly limited, so our experiments may fill the gaps in understanding of detailed mechanisms of barrel cortex neuroplasticity induced by sensory deprivation. What is more, our result may have far-reaching translational implications allowing for discovery of efficient drugs supporting for example rehabilitation after injury or stroke.