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Learning and memory processes are key issues of current neuroscience research. Such processes are accomplished via activity-dependent changes in the strength of synapses. Particularly, synapses can be either potentiated or depressed and such changes can last from hours to days (so called long term potentiation, LTP, or long term depression, LTD). In addition, very recently, structural changes at the synapses as well as changes in the number of dendritic spines (the site where excitatory synapses are made) have been correlated to the activity-dependent functional changes. Interestingly, also these structural changes can occur in a positive (growth of new synapses) or negative (loss of synapses) way correlated respectively to positive (potentiation) or negative (depression) functional changes. Taken together the functional and structural plastic changes at synapses are supposed to be the basic phenomena of long term memory storage.

The goal of our group is to understand the molecular and cellular mechanisms linking the functional changes to the structural modifications occurring during learning and memory processes. To this aim we use both in vitro and in vivo approaches to analyze the function as well as the morphology of neurons of the hippocampus, one of the main areas involved in memory storage. Using several mutant mice as well as transfection methods we analyze the effect of various molecules on both the function and the morphology of neurons.

A promising candidate molecule to link changes in function to the changes in structure of hippocampal neurons is the nerve growth factor BDNF (Brain derived neurotrophic factor). BDNF is released by neurons in an activity-dependent way and acts via a dual receptor system. Specifically, the TrKB receptor has been shown to mediate dendrite and spine growth during development as well as to control LTP in the mature brain. These observations indicate a crucial role of the BDNF/TrkB system in mediating "positive" activity-dependent changes at synapses. We could show that p75NTR, the other BDNF receptor, has a crucial role in controlling LTD and it has a negative effect on spine density and dendrite complexity in hippocampal pyramidal neurons. Since the p75NTR is also binding the amyloid-beta peptide, a molecule involved in the pathology of Alzheimer, these results might be of high relevance also for clinical research.