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Mechanism underlying Interneuron plasticity

A fundamental feature of the mammalian brain is its ability to acquire, store and recall novel information, which enables the individual organism to flexibly adapt to its changing environment. Memory formation dependents on the capacity of nerve cells to change the efficacy of their communication points, the synapses. These plastic changes depend on correlated neuronal activity of communicating neurons and are expressed as long-term potentiation (LTP) or the opposite long-term depression (LTD) of synaptic transmission. Almost 50 years ago synaptic plasticity was first identified at glutamatergic terminals in networks of excitatory principal cells (Hebb, 1949; Bliss & Lomo, 1973). Since then synaptic plasticity has been broadly accepted as the main cellular mechanism underlying learning and memory in the mammalian brain. Recent investigations, demonstrated that plasticity also exists at glutamatergic synapses targeting GABAergic inhibitory interneurons (INs). Moreover, an unexpected variety in the conditions and forms of IN plasticity has been identified, raising the possibility that IN plasticity could contribute to information processing in a very versatile, cell type- and synapse-specific manner. We aim to decipher the contribution of IN plasticity in the hippocampal dentate gyrus to learning and memory in mice which we aim to introduce in this talk. First, we provide evidence for the main cellular and molecular mechanisms underlying synaptic plasticity in in parvalbumin-expressing persioma-inhibitory (PVI) and somatostatin-expressing dendrite-targeting interneurons (SOMIs) of the dentate gyrus. Second, we show that in addition to functional, PVIs and SOMIs undergo structural plasticity upon spatial learning. Finally, we will demonstrate newly developed and tested molecular tools to interfere with some of the main molecular pathways underlying IN-type plasticity. The next logical step we aim to follow is, to use these tools and draw a causal link between IN plasticity, neuronal network activity and memory-relevant behaviour.