7. Summary

"Synaptic plasticity in the temporal lobe and its alterations in a model of MK-801-induced acute schizophreniform psychosis"

The hipppocampal formation, a base of our consciousness, is the major relay station of the information flow from the outer world to the cortex where it is finally stored. Thus it is most important for processing, storage and retrieval of information or, in other words, for cognition, learning and memory. In the hippocampus the area CA1receives two major inputs to area CA1. The first, the trisynaptic loop, reaches area CA1 via Schaffer collaterals in stratum radiatum after it is processed through different hippocampal areas. The second input projects from the entorhinal cortex directly (direct cortical input, dCI) to stratum lacunosum-moleculare of area CA1.

In this work, in-vitro-electrophysiological study at the horizontal combined hippocampo-entorhinal cortex slice preparation of the rat elucidates the electrophysiological phenomenon of “synaptic plasticity” in area CA1. Synaptic plasticity describes the capacity of neurons to change the efficacy of synaptic transmission and is regarded as the substrate for learning and memory formation. This study is focused on the dCI that, despite of its importance for the function of area CA1, is not well characterized by electrophysiological examination.

Wash-in studies showed that low-frequency stimulation (LFS) of the dCI can elicit a $GABA_A$- and kainate receptor dependent long-term depression (LTD), which is modulated by the activity of local interneurons. Simultaneously, this LFS of the dCI induces mGluR and $GABA_B$ receptor dependent LTP in the unstimulated SCI. NMDA receptor dependent interneuron-plasticity regulated by kainate receptor activity contributes to this effect.

Treatment with MK-801 is an accepted animal model of acute schizophreniform psychosis. In this work studies with MK-801 treated rats show that MK-801 acutely interferes with HFS-induced synaptic plasticity in area CA1 and can induce long-lasting alterations. These alterations may contribute to symptoms seen in an acute psychotic episode as well as to longer-lasting deficits in human.