Abstract:

CRF receptor type (CRHR) 1 exerts neuroregulatory control on associative learning processes such as fear and anxiety like behaviour. Using hippocampal slices, we investigated the neuronal excitability in mice lacking CRHR1 (Crhr1(-/-)). Compared to wild-type mice, long-term potentiation (LTP) elicited by 100 pulses at 100Hz was not different. Unexpectedly, at lower frequencies (1, 5 or 10Hz), the resulting synaptic changes in CA1 neurons of Crhr1(-/-) were systematically shifted towards long-term depression (LTD). Furthermore, testing paired-pulse paradigm revealed a GABA receptor-dependent decrease of paired-pulse ratio in Crhr1(-/-). It might be assumed that a lack of CRHR1 induce developmental changes which resulted in altered GABAergic activity, producing attenuated synaptic potentiation after repetitive stimulation and thus favouring LTD in principal neurons. Since CRHR1 are located in GABAergic somata, axons and boutons the activity of these receptor types rather might contribute to the development of the neuronal ability for plasticity like processes on the level of NMDAR subunit composition and GABAergic activity.