Cortical inhibitory interneurons play an important role in cortical neuronal circuits in physiological state and probably also in the etiopathogenesis of epilepsy. The first part of my research work was focused on calretinin expressing (CR+) interneurons in a) non-malformed temporal neocortex in patients with refractory temporal lobe epilepsy associated with hippocampal sclerosis (nHSTN), b) in focal cortical dysplasias (FCD). FCD represent a prominent cause of pharmacologically intractable epilepsy. Main goal of our study was to show whether CR+ interneurons exhibit any change in human nHSTN and in various types of FCD and to compare it with previously well documented decrease of parvalbumin expressing (PV+) interneurons. We used immunohistochemical staining for CR. Brain tissue from 24 patients surgically treated for pharmacoresistant epilepsy as well as post-mortem acquired control neocortical samples of 9 patients were examined. CR+ interneuronal density in all types of samples was evaluated. In nHSTN, no change compared to controls was found in CR+ neuronal density. The density of CR+ interneurons was significantly decreased in FCD type I (to approximately 70 % of control values) and even more in FCD type II (to approximately 50 % of control values). The decrease of CR+ interneuronal density was shown to be less prominent in comparison with previously described decrease of PV+ interneurons. Besides
dysbalance of cortical excitation and inhibition, a functional dysbalance between individual subpopulations of cortical interneurons seems to play an independent role in epilepthogenesis. As a second part of my research work, I underwent qualitative and quantitative analysis of interneuronal populations in perirhinal cortex (PRC) of the rat brain. PRC plays an important role in propagation of epileptic activity in temporal lobe epilepsy. We used immunohistochemical staining for CR, PV and calbindin (CB).