Introductory Remarks to Symposium 2

Mechanisms of neuronal and synaptic plasticity in epilepsy

Jochen Meier and Günter Schwarz, Braunschweig and Cologne

Epilepsies are chronic neurological syndromes that degrade life quality due to sudden occurrence of seizures. Focal epilepsies are a serious health problem since antiepileptic drugs often become ineffective over time. Most focal epilepsies have no discernable genetic component, suggesting that they depend on environmental factors and involve disease-promoting mechanisms of neuronal plasticity which shall be discussed during the symposium. Günter Schwarz will discuss genetic mechanisms of epilepsy and focus on the gephyrin, the major postsynaptic synaptic GABA type A and glycine receptor anchoring protein (Dejanovic et al., 2014 and 2015). Marta Zagrebelsky will focus on the molecular mechanisms regulating synaptic plasticity at glutamatergic synapses and how they relate to cognitive function and learning and memory processes (Delekate et al., 2011; Kellner et al. 2016). Jochen Meier will discuss the role of glycine receptor RNA editing and reveal how presynaptic expression of this gain-of-function receptor variant affects GABAergic and glutamatergic synaptic transmission, network excitability and cognitive function including learning and memory (Winkelmann et al., 2014 and Caliskan et al., 2016). Nicola Maggio will provide insights into how life stress affects synaptic, cellular and network mechanisms of epilepsy (Maggio et al., 2013 and Maggio et al., 2012). The two student talks by Felix Beinlich and Esin Candemir will provide insights into the contribution of CLC-3 on the acidification of glutamatergic synaptic vesicles using fluorescence lifetime imaging microscopy and discuss neuronal nitric oxide synthase PDZ - interactions in schizophrenia-like behavior, respectively. Altogether, this symposium will provide an in-depth analysis of current “from molecule-to-behavior” discussions of mechanisms of neuronal and synaptic plasticity and their involvement in health and disease.