6th International Ph.D. Symposium

Berlin Brain Days

2009/*dec.* 9–11

1 **Program & schedule** *of the symposium*

2 **Abstract** *of the opening lecture by Jörg Geiger*

3 **Abstracts** *of the six keynote lectures*

4 **Curricula vitae** *of the keynote speakers*

5 **Abstracts** *of all Ph.D. conference talks*

6 **Abstracts** *of all presented Ph.D. posters*

7 **Information** *on the organizing Ph.D. programs*

Max Delbrück Center for Molecular Medicine Berlin-Buch

Welcome to the Berlin Brain Days 2009

The Berlin Brain Days are an activity of doctoral students across several independent Berlin institutions. Initiated in 2005 by faculty and students in Medical Neurosciences (a doctoral school at the Charité), it has subsequently grown year-by-year as the neuroscientific research and training environment has rapidly developed within the city.

The growth in the number and variety of new doctoral programs within Berlin is quite remarkable. Two research training groups (Graduiertenkollegs) of *Deutsche Forschungsgemeinschaft*, on "Learning and Memory" (GRK 1123 – Cellular Mechanisms of Learning and Memory Consolidation in the Hippocampal Formation) and on "Neuroinflammation" (GRK 1258 – The Impact of Inflammation on Nervous System Function), were established in 2005 and 2006. In this time the Bernstein Center for Computational Neuroscience was launching its own comprehensive doctoral program in computational neuroscience. Also in 2006, as part of the *Excellence Initiative* for German universities, the Berlin School of Mind and Brain was established to foster transdisciplinary research at a doctoral level across the mind and brain sciences. And there have been a second and third acquisition of the *Excellence Initiative*: the excellence clusters "NeuroCure" and "Languages of Emotion", both with funding for doctoral programs.

In December 2008, we very successfully joined our forces for the first time. The Berlin Brain Days 2009 are again a common activity of all these programs. Students and faculty alike are highly motivated to learn about the activities of

Patricia Seja, G. Spitzmaul, C. Pfeffer, and T. J. Jentsch

P63 The physiological role of KCC2 in the cerebellum

Physiology and Pathology of Ion Transport, Max Delbrück Center for Molecular Medicine and Leibniz Institute for Molecular Pharmacology

Cation chloride cotransporters mediate a coupled electroneutral movement of Cl^-, K^+ and Na^+ across plasmamembranes in many cells. The neuron-specific KCl cotransporter KCC2 is thought to lower the intracellular Cl^- concentration below its electrochemical equilibrium potential by using the outwards directed gradient of K^+ as a driving force. This low intracellular Cl^- concentration is required for the fast inhibitory action of GABA which is mediated by the GABAA receptor, a ligand-gated anion channel.

The activation of GABAA receptors drives the membrane potential of a cell towards EGABA, the reversal potential of GABAergic currents. In immature neurons, GABA is excitatory, as EGABA is above the resting membrane potential. The expression of KCC2 correlates with the drop of EGABA below the resting membrane potential, and thereby the switch from excitatory to inhibitory GABA signaling.

The KCl cotransporter KCC3 is expressed more broadly and involved in cell volume regulation. Nevertheless, while KCC2 may be the key regulator of neuronal $[Cl^-]_i$, a similar role was proposed for KCC3. We investigate the physiological role of KCC2 and KCC3 in the murine cerebellum at cellular level via the patch clamp technique in acute slices. We showed that knocking out KCC2 in cerebellar Purkinje cells results in a shift of EGABA, which could be correlated with motor learning deficits.