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

neighboring programs, and the Berlin Brain Days have become an important forum for information exchange.

Berlin has already had a good tradition in fostering common activities in the neurosciences: the Berlin Neuroscience Forum has been organized every other year since 1997 and is a common activity of all programs and collaborative research centers (Sonderforschungsbereiche, Forschergruppen, Graduiertenkollegs, etc.). It regularly attracts over 200 neuroscientists to a small resort outside of Berlin, Liebenwalde.

The success of the Berlin Brain Days inspired a previous keynote speaker from Japan to solicit a similar activity in Fukuoka. Last year, Professor Mami Noda joined the Berlin Brain Days with a group of her students from Japan, and this year she organized the first Kyushu Brain Days – and even obtained funds for Berlin students to join in the event. In 2009, we can welcome another interesting group of visitors: the Berlin School of Mind and Brain awarded a women's travel grant to 20 neuroscience, linguistics and philosophy students from Canada, USA, Israel, France, Britain, Spain, Turkey, and Germany. We welcome them to Berlin and look forward to seeing their research as they too will attend the Berlin Brain Days and present posters.

It is in our best interest that we join forces, interact closely, and develop Berlin as a hotspot for research across the neurosciences. With this in mind, I am convinced that we will have a very interactive and successful meeting that will result in new collaborations within the Berlin neuroscience research community.

Helmut Kettenmann, Conference Chair

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Contribution of KCNQ5 to the medium and slow after-hyperpolarization currents in hippocampal neurons

P24

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Benign familial neonatal convulsion (BNFC) is a neurological disorder caused by mutations in the potassium channel genes KCNQ2 and KCNQ3, which contribute to the medium afterhyperpolarization current (ImAHP) and slow afterhyperpolarization current (IsAHP) in hippocampal neurons. KCNQ5 is not yet linked to any human disease but is broadly expressed in the brain similar to KCNQ2 and KCNQ3.

To investigate the role of KCNQ5 in the brain we generated a KCNQ5 dominantnegative (Kcnq5dn/dn) mouse. Histological analysis did not reveal structural brain abnormalities. Western blots of total brain proteins revealed that there is no detectable influence of the mutated KCNQ5 protein on overall KCNQ2, KCNQ3 and KCNQ5 levels. In addition, the subcellular localization of KCNQ2 and KCNQ3 is not altered in Kcnq5dn/dn mice. Using electrophysiological analysis we could show that KCNQ5 contributes to the ImAHP and IsAHP in a subset of hippocampal neurons where KCNQ5 is highly expressed. Therefore, our study is a direct demonstration that in addition to KCNQ2 and KCNQ3, KCNQ5 channels contribute to the ImAHP and IsAHP.