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Signaling and Transport Processes
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Curriculum vitae

- since 2009 Independent group leader, Max Delbrück Center for Molecular Medicine
- 2001 – 2008 Postdoctoral fellow, lab of Lily Jan, University of California, San Francisco
- 1997 – 1999 Undergraduate student in medicine, University of Hamburg
- 1995 – 2000 Graduate student in biochemistry and postdoctoral fellow (Thomas J. Jentsch), Center for Molecular Neurobiology (ZMNH), Hamburg
- 1990 – 1995 Undergraduate student in physics and economics, University of Hamburg

Research fields

The work of our group is aimed at understanding signal and transport processes on the molecular level. We focus on:

- Functional characterization of the recently identified TMEM16 family of ion channels
- Understanding the pathophysiology of diseases caused by mutations in this gene family, including ataxia, Scott syndrome, and muscular dystrophy
- Regulation and interaction partners of proteins
- Identification of “new” ion channels and other molecules involved in neuronal excitability and synaptic transmission.

Activities in the scientific community, honors, awards

- 2009/2011 Teaching, Helmholtz Graduate School, Max Delbrück Center for Molecular Medicine
- 2001/2004 Human Frontier Long-Term Fellowship Award

Selected publications

Schroeder, BC, Cheng, T, Jan, YN and Jan, LY. Expression cloning of TMEM16A as a calcium-activated chloride channel subunit. *Cell*. 2008; 134, 1019-29.

Estevez, R*, Schroeder, BC*, Accardi, A, Jentsch, TJ and Pusch, M. Conservation of chloride channel structure revealed by an inhibitor binding site in CIC-1. *Neuron*. 2003; 38, 47-59. | * equal contribution

Schroeder, BC*, Waldegger*, S, Fehr, S, Bleich, M, Warth, R, Greger, R and Jentsch, TJ. A constitutively open potassium channel formed by KCNQ1 and KCNE3. *Nature*. 2000; 403, 196-9. | * equal contribution

Schroeder, BC, Hechenberger, M, Weinreich, F, Kubisch, C and Jentsch, TJ. KCNQ5, a novel potassium channel broadly expressed in brain, mediates M-type currents. *J Biol Chem*. 2000; 275, 24089-95.

Kubisch, C*, Schroeder, BC*, Friedrich, T, Lutjohann, B, El-Amraoui, A, Marlin, S, Petit, C and Jentsch, TJ. KCNQ4, a novel potassium channel expressed in sensory outer hair cells, is mutated in dominant deafness. *Cell*. 1999; 96, 437-46. | * equal contribution

Schroeder, BC*, Kubisch, C*, Stein, V and Jentsch, TJ. Moderate loss of function of cyclic-AMP-modulated KCNQ2/KCNQ3 K⁺ channels causes epilepsy. *Nature*. 1998; 396, 687-90. | * equal contribution

Biervert, C*, Schroeder, BC*, Kubisch, C, Berkovic, SF, Propping, P, Jentsch, TJ and Steinlein, OK. A potassium channel mutation in neonatal human epilepsy. *Science*. 1998; 279, 403-6. | * equal contribution

Wollnik, B, Schroeder, BC, Kubisch, C, Esperer, HD, Wieacker, P and Jentsch, TJ. Pathophysiological mechanisms of dominant and recessive KVLQT1 K⁺ channel mutations found in inherited cardiac arrhythmias. *Hum Mol Genet*. 1997; 6, 1943-9.