COLLOQUIA IN CELLULAR SIGNALLING

Venue: Medical University Vienna, Center for Physiology and Pharmacology,
Institute of Pharmacology, Währingerstraße 13a, 1090 Vienna, Leseraum, Hochparterre
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Friday **11.01.2013 11:00 c.t.** **Björn Falkenburger** **(host: H. Sitte)**

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"Transporter-mediated dopamine secretion, KCNQ2/3 channels, and phosphoinositide metabolism: a talk in two parts"

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Abstract:
Neurotransmitter transporters take up neurotransmitter secreted by exocytosis. In the first part of my presentation I will discuss our published work indicating that the dopamine transporter may, in addition, secrete dopamine from dendrites of dopaminergic neurons when glutamatergic inputs are activated. Since this interpretation has remained controversial, I will briefly review the literature on transporter-mediated neurotransmitter release.
In the second part I will present our mechanistic and kinetic characterization of the inhibition by M1 muscarinic receptors of KCNQ2/3 (Kv7.2/7.3) potassium channels. M1 receptors couple to Gq and deplete the plasma membrane lipid PIP2, a member of the phosphoinositide family of signaling lipids. It is the PIP2 depletion, not calcium rise or PKC activation, that mediates KCNQ2/3 channel inhibition. KCNQ2/3 current recordings were subsequently used to study abundances and turnover rates of plasma membrane phosphoinositides. We found that the steady-state between PIP2 and its precursor PI(4) is maintained on a rapid timescale and predict an independent function of PI(4)P for actin dynamics.