Sonderforschungsbereich/Transregio 31 “Das aktive Gehör”

# E I N L A D U N G

zum Vortrag im Rahmen des Seminars des SFB/TRR 31

**Freitag, 3. Dezember 2010, 14 Uhr c.t.**

im Raum W2 1-143 der Universität Oldenburg
und im Raum G26.1 – 010
Rechenzentrum der Universität Magdeburg
(per Videoübertragung)

## “Synaptic transmission, nitric oxide and homeostatic control of neuronal excitability by potassium channels”


## Prof. Ian Forsythe

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We have previously established that the principal neurones of the medial nucleus of the trapezoid body (MNTB) express nitric oxide synthase (nNOS), that this is activated by synaptic stimulation of the giant synapse, the calyx of Held; and that the NO acts as a volume transmitter, regulating excitability in surrounding neurones (Steinert et al., Neuron 60: 642-656, 2008). We first observed that Kv3 channels are suppressed over a timecourse of 10-30 mins, but on a slower timescale of 30-60 minutes, Kv2 channels show a reciprocal enhancement. This means that the delayed rectifier mediating action potential repolarisation shifts from Kv3 domination under periods of low activity, to Kv2 domination during periods of higher synaptic activity. This homeostatic shift in the dominant delayed rectifier favours maintenance of high firing rates and therefore information transmision across this relay synapse. It suggests that voltage-gated potasium channels are under rapid activity-depen dent control and our work elsewhere in the brains suggests that this may be a general mechanism of regulating neuronal excitability.