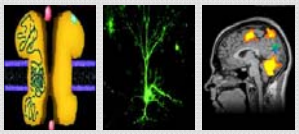

Sensory Processes: From Molecules to Cognition

April 12 - 13, 2016, Veranstaltungszentrum, Ruhr University Bochum

Tuesday April 12, morning (9:25 – 12:35)

Session 1 **Cellular Mechanisms**

TOBIAS MOSER

Institute for Auditory Neuroscience & InnerEarLab, University Medical Center Göttingen
MPIs for Biophysical Chemistry and Experimental Medicine, Göttingen
German Primate Center, Göttingen

Inner hair cells decompose auditory signals through active zones with different Ca^{2+}-influx

For each sound frequency, spiral ganglion neurons with different response properties are thought to collectively encode the wide range of audible sound pressures. How such dynamic range fractionation is implemented remains unclear. Heterogeneity of spiral ganglion neuron synapses with presynaptic inner hair cells is an attractive candidate mechanism. Here, we studied the heterogeneity of presynaptic active zones (AZs) by patch-clamp, confocal imaging of synaptic Ca^{2+}-influx, immunohistochemistry and STED microscopy of mouse inner hair cells. AZs held 20-270 Ca^{2+}-channels, mostly arranged in linear 60 nm x 100-600 nm clusters. AZs facing the ganglion exhibited larger ribbons and more Ca^{2+}-channels causing stronger maximal Ca^{2+}-influx. We found an opposite gradient for voltage-dependence of Ca^{2+}-influx: AZs that point away from the ganglion activated at weaker depolarizations. Disruption of the deafness gene GIPC3 in mice reversed the gradient of maximal Ca^{2+}-influx, caused a hyperpolarizing shift of activation and changed the firing properties of spiral ganglion neurons. We postulate that inner hair cells segregate Ca^{2+}-channel properties among AZs to generate complementary codes in functionally distinct spiral ganglion neurons from one receptor potential.