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, 23. Mai 2014, 14 Uhr c.t.

im Raum H28 / R 2.31 des Med. Campus Magdeburg
und Raum W2 1-143 der Universität Oldenburg
(per Videoübertragung)

"Epigenetic control of auditory cortical plasticity enables the acoustic specificity of reward-related memory"

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Memory-related auditory cortical plasticity requires transcription, which is the expression of genes that enable stable changes in neuronal identity, function, structure, connectivity, and ultimately animal behavior. A powerful new avenue to investigate the molecular mechanisms underlying the formation of/ /auditory memory is through epigenetic control of transcription. One of the best-studied epigenetic mechanisms controlling transcription is histone acetylation and deacetylation, which primarily promotes or represses gene expression, respectively. Recent studies have shown that genetic or pharmacological inhibition of histone deacetylase 3 (HDAC3) can transform an experience (that does not normally lead to memory formation) into a robust long-term memory. This demonstrates that HDAC3 is a critical negative regulator of memory formation (McQuown et al., 2011; Malvaez et al., 2013). A key open question is whether such molecular mechanisms can likewise gate the transformation of a transient auditory sensory experience into an enduring auditory memory. This talk will show new findings that HDAC3 controls the threshold of memory induction for specific auditory details (e.g., sound frequency and sound intensity) of an auditory associative learning experience /via /regulation of auditory cortical remodeling.

Pharmacological inhibition of HDAC3 in rats learning to associative sound with reward produces associative memory with highly specific auditory content as well as a signal-specific reorganization of the primary auditory cortex (A1) that each reflects the increased acoustic specificity of reward-related memory formation. These findings potentially explain the mechanism by which HDAC3 can enhance the robustness of memory. They also introduce a novel niche for investigating mechanisms of adult sensory cortical plasticity in the service of the learning and memory that may underlie abilities important for communication and comprehension.