Seewald *et al.: Intrinsic Activity*, 2019; **7**(Suppl.1):A3.43
doi:10.25006/IA.7.S1-A3.43
published online: 11 September 2019

INTRINSIC ACTIVITY

Joint Meeting of the Austrian Neuroscience Association (16th ANA Meeting) and the Austrian Pharmacological Society (25th Scientific Symposium of APHAR)

Innsbruck, 25–27 September 2019

MEETING ABSTRACT

A3.43

Plasticity of amygdala intercalated cell microcircuits in fear learning

Anna SEEWALD[1,*], Ayla AKSOY-AKSEL[2], Ingrid EHRLICH[2] and Francesco FERRAGUTI[1]

[1]Department of Pharmacology, Medical University of Innsbruck, Austria; [2]Hertie Institute for Clinical Brain Research and Centre for Integrative Neuroscience, University of Tübingen, Germany

Background: The amygdala plays a crucial role in attaching emotional significance to environmental cues. Its intercalated cell masses (ITC) are tight clusters of GABAergic neurons, which are distributed around the basolateral amygdala complex (BLA) and appear to be involved in the acquisition as well as extinction of conditioned fear responses. As their ablation results in a deficit of the expression of fear extinction, ITC have been the subject of intense investigations. The aim of our study is to characterize neuron subtypes and plastic properties of pre- and postsynaptic partners of ITC neurons within the medial paracapsular cluster (mpITC).

Methods: To address the question whether changes in AMPA and NMDA distribution and density correlate with functional synaptic changes observed during different fear states, a detergent-digested freeze-fracture replica labelling approach was used. This allows us to investigate the spatial distribution as well as density of ionotropic glutamatergic receptors from thalamic inputs within postsynaptic specializations in mpITC neurons. Furthermore, we combined whole-cell patch-clamp recordings and biocytin labelling to anatomically reconstruct mpITC neurons and analyse their synaptic contacts.

Results: Electrophysiological studies showed that thalamic inputs to mpITCs are modified by fear learning. Here, we suggest that functional changes in the AMPA/NMDA ratio are attributable to changes in the expression of AMPA receptors. Furthermore, we confirm the presence of heterogeneous mpITC projection subtypes. Our data suggest that projections to the amygdalostriatal transition zone (AStria) and, hence, potentially to the striatum, might be a major pathway of the mpITC.

Discussion: The mpITC is most likely implicated in a much more complex microcircuit than originally proposed, functioning only as a relay station between the BLA and the main output station of the amygdala, the central amygdala. Together, our results further a circuit-based understanding of how ITC activity can contribute to high and low fear states.

Acknowledgements: Funded by D-A-CH I2215 grant from the FWF (F.F.) and DFG (I.E.).

Keywords: fear – amygdala – neuroanatomy – plasticity

*Corresponding author e-mail: *anna.seewald@i-med.ac.at*