

Presentation number: P1.16

Topic: Neuroanatomy

Characterization of LCN2 Receptor Expression and Signaling Pathways in Glial Cells under Healthy and Inflamed CNS Conditions

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Lipocalin 2 (LCN2), a 25-kDa secreted protein, possesses bacteriostatic properties and plays a crucial role in innate immunity. Recent findings show that LCN2 is secreted by activated astrocytes and significantly influences brain inflammation. Although six putative receptors for LCN2 have been identified, the mechanisms by which these receptors transmit and amplify signals remain poorly understood. We aim to characterize the expression patterns and signaling pathways of distinct LCN2 receptors in healthy and inflamed CNS.

We conducted a detailed analysis of existing research on LCN2 receptor expression and signaling. Given the limited knowledge on cell type-specific receptor expression within the CNS, we performed immunohistochemical staining in inflammatory animal models. Additionally, gene and protein expression studies were conducted on primary glial cells. To further elucidate the signaling mechanisms and functional relevance of the receptors NGALR and LRP2, shRNA was used to knock down their expression in astrocytes.

LCN2 receptors are expressed in various cell types and organs. In the CNS, we observed high expression of NGALR and LRP2 in glial cells. Astrocytes expressed these receptors at mRNA and protein levels. Inflammatory conditions did not alter their expression. Similar findings were noted in brain endothelial cells. shRNA experiments indicated that receptor expression might not be critical for astrocyte survival and metabolism.

LCN2 is involved in numerous biological processes and disease states, yet a significant gap remains in understanding how its receptors function. Further studies are needed to determine how NGALR and LRP2 signaling impacts inflammatory processes within the CNS.

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Topic: Neuroanatomy

High-resolution 3D mapping of cytoarchitectonic areas 44 and 45 reveals new anatomical details of Broca's region

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Broca's region is involved in key aspects of language processing. But its detailed functional diversity is in contrast to classical anatomical subdivisions of this region into two cortical areas (44 and 45). Previous work of our lab resulted in 3D cytoarchitectonic maps of areas 44 and 45, and later revealed further subdivision based on multiple neurotransmitter receptor distributions.

Based on the hypothesis that a more detailed parcellation of the two areas in receptor architecture would have a correlate in cytoarchitecture, we now mapped the cytoarchitecture of Broca's region in ten postmortem human brains using an observer-independent border definition approach.

We identified two subdivisions of area 44 (44a and 44p) and two of area 45 (45a and 45p) arranged in anterior posterior orientation. 3D maps of all four subdivisions were computed in common stereotaxic spaces (MNI 152, Colin 27). Interindividual differences were expressed through probabilistic maps of these areas. In addition, high-resolution 3D reconstructions of these areas in a microscopical template brain, the BigBrain, have been generated at 20-micron resolution isotropic, to reveal fine details of anatomy.

The maps will be provided with the Julich-Brain Atlas and made accessible on the EBRAINS infrastructure as FAIR data. They may serve for education purposes in brain anatomy, facilitate neuroimaging studies on structure-function relationships and inform modelling, simulation and brain stimulation.

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Topic: Neuroanatomy

Characterization of Bcl11a/b transcription factors in multi-protein complexes during corticogenesis

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Transcription factors orchestrate gene expression and interact with multi-protein complexes during corticogenesis. Bcl11a/b transcription factors have been shown to control diverse functions in the developing central nervous system. However, the molecular mechanisms through which Bcl11a/b regulate neocortical development remain unclear.

To uncover changes in gene regulatory network upon the knockout of Bcl11a/b, ATAC-seq was performed on conditional knockouts (cKO) of Bcl11a, Bcl11b, and double cKO (dcKO) at E14.5.