Pannexin

Pannexins may be involved in the process of tumor development. Particularly, PANX2 expression levels predict post diagnosis survival for patients with glial tumors.

Probenecid, a well-established drug for the treatment of gout, allows for discrimination between channels formed by connexins and pannexins. Probenecid did not affect channels formed by connexins.

Increasing evidence suggests that the large-pore ion channels, pannexin 1 (Panx1) and 2 (Panx2), are involved in epilepsy and brain development.

Li et al., investigated the expression of Panx1 and Panx2 in surgical samples from patients with FCD type Ia (FCDIa), type IIa (FCDIIa), and type IIb (FCDIIb) and in age-matched autopsy control samples. We found Panx1 mRNA and protein levels were both increased in all these FCD samples. Immunohistochemical analyses revealed that Panx1 was mainly distributed in microcolumn neurons, dysmorphic neurons (DNs), balloon cells (BCs) and reactive astrocytes. Double-labeled staining showed that the Panx1-positive neurons were mostly glutamatergic DNs and occasionally GABAergic normal-appearing neurons. Importantly, the protein levels of Panx1 positively correlated with the frequency of seizures. Intriguingly, the Panx2 mRNA and protein levels were only upregulated in FCDIIb lesions and characteristically expressed on SOX2-positive multipotential BCs. Immunofluorescent experiments identified that Panx2-positive BCs mainly expressed the neuronal differentiation transcription factor MASH1 but not the immature glial marker vimentin. Taken together, our results established a potential role of the specific expression and cellular distribution patterns of Panx1 and Panx2 in FCD-associated epileptogenesis and pathogenesis ¹⁾.

1)

Li S, Zang Z, He J, Chen X, Yu S, Pei Y, Hou Z, An N, Yang H, Zhang C, Liu S. Expression of pannexin 1 and 2 in cortical lesions from intractable epilepsy patients with focal cortical dysplasia. Oncotarget. 2016 Dec 28. doi: 10.18632/oncotarget.14317. [Epub ahead of print] PubMed PMID: 28036289.

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