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Sarcoglycan sub-complex in WAG/Rij rats, a model of absence epilepsy: an immunofluorescence study

Giuseppina Cutroneo, Giovanna Vermiglio, Debora Di Mauro, Giuseppina Rizzo, Elena Florio and Emanuele Magaudda

Department of Biomorphology and Biotechnologies, University of Messina, Messina Italy

It is known that even in Central Nervous System the Dystrophin Glycoprotein Complex (DGC) exists but differs in composition from the DGC core present in muscle for the presence of several isoforms of dystrophin and for the existence of a sarcoglycan sub-complex which is made up only for ε- and ζ-sarcoglycans; for these reasons it was called "DGC-like". Although that, in our previous studies we have found that all sarcoglycans are present in human cerebral cortex and in different regions of rat's brain, suggesting that the composition of the brain DGC is not different from the muscle DGC but differing just for the function. In fact, our previous data showing the colocalization between sarcoglycans and GABAA Rε receptors in rat's cerebral and cerebellar cortex, thalamus and hippocampus suggested us that in brain sarcoglycans could be associated with synaptic neurotransmission. To better understand which kind of relationship between sarcoglycans and GABAA receptors exists, we aim to investigate α-,β-,γ-,δ-,ε- and ζ-sarcoglycans and the GABAA Rε in the brain of the WAG/Rij rats, a model of absence epilepsy, using immunofluorescence techniques; we have observed cerebral cortex, thalamus and hippocampus, the structures mainly involved in absence epilepsy, comparing this to normal rat's brain. Results show that the GABAA Rε receptors staining pattern is different from the normal rat's brain presenting an abnormal fluorescence distribution consisting in discontinuous clusters around the cellular body. Sarcoglycans, instead, have shown the typical "spot-like" staining pattern around the cellular body, sometimes colocalizing with GABAA Rε receptor; moreover, they seem to have an higher staining pattern than normal rat's brain. In our opinion these results, showing an alteration of GABAA Rε receptor pattern distribution and a normal or increased sarcoglycans staining pattern, allow us to hypothesize that the sarcoglycans in absence epilepsy are likely to compensate for the alteration in GABAA Rε receptor distribution. That support the opinion about the involvement of sarcoglycans in cellular signalling and receptor assembly regulating indirectly synaptic neurotransmission both in normal and in pathological condition.

Keywords: Sarcoglycan, absence epilepsy, GABAA Rε receptor, rat's brain.

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