

Tioredoxins and Glutaredoxins System Proteins: Immunolocalization in The Rat Central Nervous System. An Atlas

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The oxidoreductases of the Thioredoxin Family of proteins play a role in the maintenance of the cellular response to oxidative stress. Redox imbalance is a major feature of brain damage. If considering the neuronal damage and glial reaction induced by a hypoxic-ischemic episode is highly related to glutamate excitotoxicity, oxidative stress and mitochondrial dysfunction. Most animal models of hypoxia-ischemia in the central neural system (CNS) use rats to study the mechanisms involved in neuronal cell death, however, no comprehensive study on the localization of the redox proteins in the rat CNS was available today.

The aim of this work was to study the distribution of the following proteins of the thioredoxin (Trx) and glutathione/glutaredoxin (Grx) system in the rat CNS by immunohistochemistry: Trx1, Trx2, TrxR1, TrxR2, Txnip, Grx1, Grx2, Grx3, Grx5, γ -GCS, Prx1, Prx2, Prx3, Prx4, Prx5 and Prx6. We have focused on areas most sensitive to a hypoxia-ischemia insult: cerebellum, striatum, hippocampus, spinal cord, substantia nigra, cortex and retina.

Previous studies claimed that these proteins may be distributed in most cell types and regions of the central nervous system. In our work, we describe several remarkable differences in both abundance and regional distribution that lead us to think the existence of a complex interplay and crosswalk between the proteins of these family and other molecules of the cell. We consider that this data might be helpful to reveal new insights into the role of thiol redox pathways in the pathogenesis of hypoxia-ischemia insults and others disorders of the central nervous system.

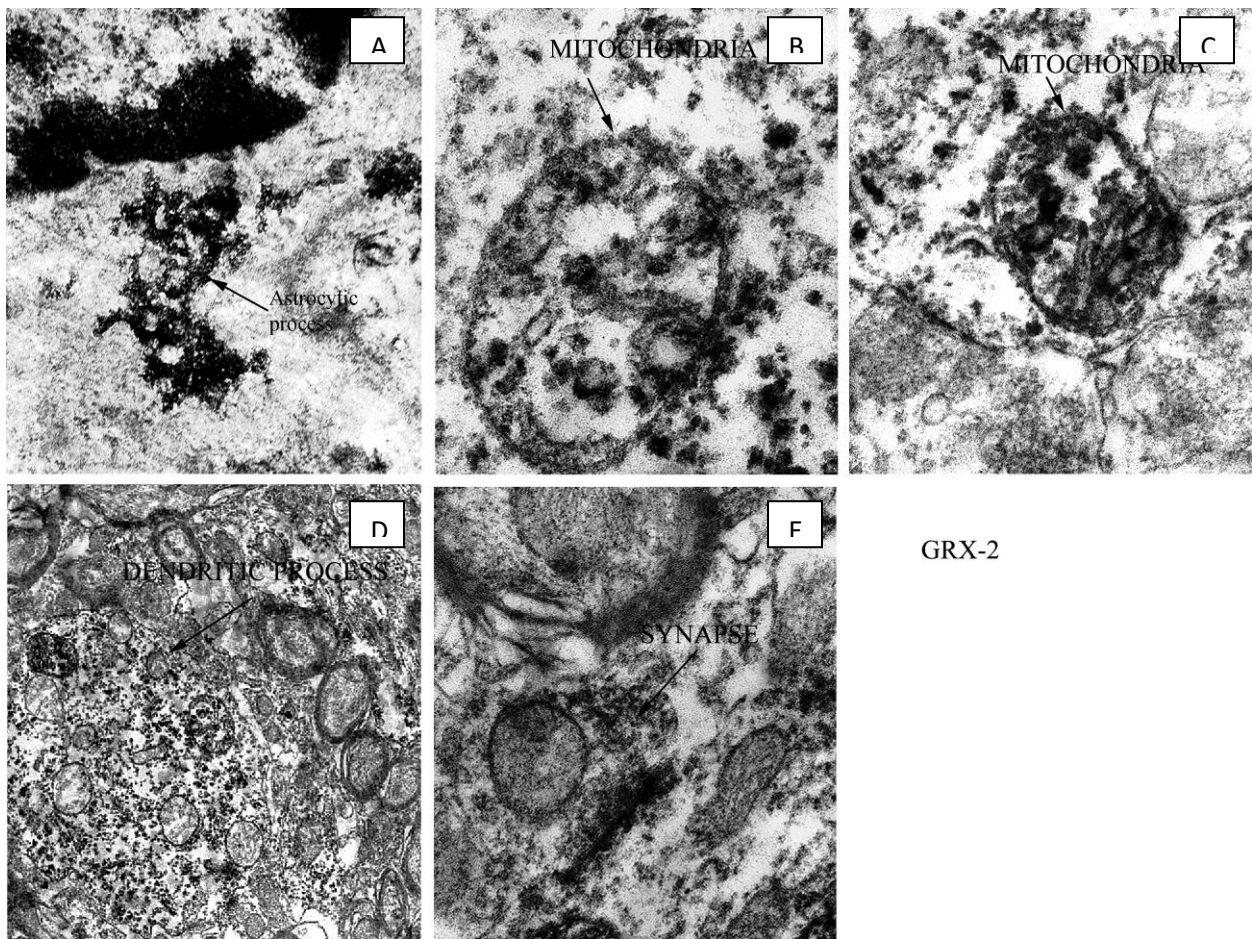


FIG. 1: Grx-2 protein. EM: cytoplasmatic immunostaining in Central Nervous System samples: neurons and small cells. A. Astrocytes process. B-E. Neurons. Mitochondria, dendritic process and synapse.

References

- (1) María Laura Aón Bertolino et al, *Biochim Biophys Acta*. 1810 (1). 93-100. (2011)
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