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Poster Presentation

NCX Family as a Neuroprotective

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Abstract

In the mammalian, there are three NCX genes (NCX1, NCX2, NCX3), which their three proteins are differentially expressed in distinct regions of the central nervous system, where they might underlie different physiological and pathophysiological functions. NCX is a nine-transmembrane protein which is distributed in the brain widely. NCX operates in a bidirectional way, and exchanges Ca2+ and Na+ ions across the cell membrane in the central nervous system; thus it plays a relevant role in the maintenance of the intracellular balance of these two ions. A great number of reports have been published which studied on the effects of NCX modulation on cell damage, under anoxic conditions. The results of in vivo studies regard the ability of NCX activation to reduce the extent of brain infarct volume after permanent middle cerebral artery occlusion (MCAO). It is also reported that selective pharmacological blockade produced a worsening of the brain lesion, so it can be concluded that NCX antiporter plays a protective role during the events leading to brain ischemia. It is relevant to mention that after transient global ischemia in rats, NCX gene was upregulated. In the early phase of neuronal anoxic ischemia, [Na⁺]i increases in the cells, so the reverse mood of NCX operates to prevent cell swelling and death, because it contributes to a decrease in [Na⁺]i overload. On the contrary, when [Ca²⁺]i overload takes place in the later phase of neuronal anoxia, NCX forward mode of operation contributes to the lowering of [Ca²⁺]i, thus protecting neurons from [Ca²⁺]i-induced neurotoxicity. Studies show that NCX family has an impact on neuroprotection mechanism so it can be an important implication in the pathogenesis of stroke. It is hoped that novel cerebroprotective strategies may be developed for those at risk of stroke or in whom cerebral perfusion is electively reduced, perhaps at the time of surgery.

Keywords: NCX, Neuroprotective, Brain Injury.

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