FENS Regional Meeting

Belgrade, Serbia, July 10–13, 2019

ДНС SNS Друштво за неуронауке Србије Serbian Neuroscience Society

National Neuroscience Society of Romania

Neuroscience Society of Turkey

Abstract Book

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Wednesday, July 11, 2019

11:30–13:10

Room Atlantic 2

SYMPOSIUM 08

TEMPORAL LOBE EPILEPSY: FROM CELLS TO MOLECULES

Organizers: Ivan Spasojevic (Belgrade, RS) and Aleksandar Ristic (Belgrade, RS)

HIPPOCAMPAL ANTIOXIDATIVE SYSTEM IN EPILEPSY

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Mesial temporal lobe epilepsy associated with hippocampal sclerosis (mTLE-HS) is probably the single most frequent human focal epilepsy. The involvement of redox processes in the pathological mechanisms of mTLE-HS has been implicated by mitochondrial dysfunction and oxidative damage, and by different metabolic abnormalities that have been observed in sclerotic hippocampi, such as altered metabolism of redox-active metals. The strongest proof came with the analysis of enzymatic antioxidative system. Sclerotic hippocampi show drastically increased activity and levels of hydrogen peroxide–removing enzymes – catalase and glutathione peroxidase/reductase. Catalase is located mainly in neurons in both, controls and HS. Sclerotic hippocampi are depleted of glutathione peroxidase-positive blood vessels that are present in control hippocampi. Pertinent to this, it has been documented that hippocampi of mTLE-HS patients show increased blood vessel density, but most of the vessels represent atrophic vascular structures. On the other hand, HS showes specific glutathione peroxidase-rich loci that are present in gyrus dentatus, CA regions, and alveus, and appear to represent bundles of astrocytes. These loci are probably sites of excessive (neuronal) production of hydrogen peroxide that is counteracted by astrocytes. Finally, protein levels of mitochondrial enzyme manganese superoxide dismutase are higher in HS than controls. Neurons with abnormal morphology and strong superoxide dismutase immunofluorescence are present in all neuronal layers in HS. In close, antioxidative system is upregulated in HS implying that epileptogenic hippocampi are exposed to oxidative stress. The involvement of redox alterations in the pathology of epilepsy may open new pharmacologic perspectives for mTLE-HS treatment.