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Poster Presentation

Mitochondrial Dysfunction and Oxidative Stress in Epilepsy

Elham Shiri[1], Akram Shiri[2], Maryam Borhani-Haghighi[1, 3*], Fatemeh Alipour[3]

[1]Department of Anatomy, School of Medicine, Tehran University of Medical Science, Iran
[2]Faculty of Nursing, Baqiyatallah University of Medical Sciences, Tehran, Iran
[3]Shefa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran

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Abstract

Introduction: Mitochondria have essential functions such as the generation of ATP, metabolite/neurotransmitter synthesis, fatty acid oxidation, calcium homeostasis, control of cell death and they are the primary source of reactive oxygen species (ROS) production. Mitochondrial dysfunction and oxidative stress play a pivotal role in several neurological disorders and recently the changes in mitochondria function have been shown in acquired epilepsies. An acute increase in mitochondrial oxidative stress and following damage to cellular macromolecules have been demonstrated in repeated seizures i.e. status epilepticus (SE). On the other hand, mitochondrial dysfunction and oxidative stress reported in other disorders such as hypoxic-ischemic insults, traumatic brain injury, viral infection, and hyperthermia which can lead to chronic acquired epilepsies. In addition, epilepsy has been seen in disorders led by mutations in mtDNA and nuclear genes. Mitochondrial dysfunction during chronic epilepsy is characterized by a decrease in ETC complex I and IV activity, increase in complex II activity, and a decline in mitochondrial membrane potential in the CA1 and CA3 regions of hippocampus one month after pilocarpine-induced SE. It can be proposed that inhibition of complex I in mitochondria can lead to a rise in ROS production and/or RNS (Reactive nitrogen species) which may cause not only neuronal injury demonstrated in a model of seizures but also epileptogenesis. In recent years, a paramount of fundamental research has been directed toward developing pharmacologic approaches to restore mitochondria function. **Conclusion:** Evaluation of mitochondrial damage in epilepsy and the specific targeting of mitochondrial oxidative stress, malfunction, and bioenergetics may be novel strategies for decreasing epileptogenesis and seizure initiation.

Keywords: Epilepsy, Mitochondrial Dysfunction, Oxidative Stress.

Corresponding Author: Maryam Borhani-Haghighi
E-mail: *Borhanihm@gmail.com*

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