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Poster Presentation

Mitochondria as a Target for Drug Therapy in Epilepsy

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Abstract

Introduction: Epilepsy is one of the most common neurologic disorders, characterized by unprovoked, recurring seizures that interrupt the nervous system and can induce mental and physical dysfunction. Mitochondrial dysfunction and oxidative stress play a central role in several neurological disorders, and recently mitochondrial dysfunction has been shown in acquired epilepsies. Mitochondrial dysfunction in repeated seizures can cause neuronal cell death which is an important characteristic of the drug-resistant type of epilepsy. Mitochondria are considered as targeted neuroprotective plans for a limited number of neurodegenerative disorders with mitochondrial pathologies such as amyotrophic lateral sclerosis and Chorea Huntington. The creatine/phosphocreatine system, adjusted by mitochondrial creatine kinase, plays an essential role in conserving energy balance in the brain. The buffering of neuronal energy levels by systemic creatine administration has been proposed for treatment. The supplemented creatine has been shown protective effect not only on neurodegenerative disorders but also on hypoxia-induced or traumatic brain injury. It was observed that creatin can reduce hypoxia-induced seizures but had the negative effect on temporal lobe epilepsy. Recent researches suggest that protection of mitochondria and decrease of oxidative stress-related events represent hopeful therapeutic methods for the treatment of various disorders. Metabolic antioxidants such as lipoic acid, N-acetyl cysteine, CoQ10 were tested for a potential neuroprotective effect in epilepsy. ketogenic diets particularly showed an increase in mitochondrial glutathione levels. Thus, current progress in developing mitochondria-targeted antioxidants is a promising approach for new therapeutic methods not only in ischemia/reperfusion but also in human epilepsy. **Conclusion:** Treatment with compounds possessing antioxidant properties and targeting mitochondrial dysfunction may in the future provide therapeutic strategies for the successful treatment of epilepsies.

Keywords: Epilepsy, Drug, Therapy, Mitochondria.

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