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International Conference on ALZHEIMER AND DEMENTIA

July 12, 2021 | Webinar

Evaluation of ala16valmnsod in epilepsy: Involvement of inflammatory, oxidative and metabolic biomarkers

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Epilepsy is a neurological disease that affects around 1% of world population, with neurobiological, neurochemical, cognitive and psychological consequences. Despite of the good prognosis, there is a high proportion of epilepsy patients who are refractory to antiepileptic drugs, reflecting the need of a better understanding about neurochemical disorders characteristics involving the disease. Inflammatory, apoptotic, DNA damage, and genetic processes are involved in epilepsy. Furthermore, metabolic syndrome (MetS) is another problem in epilepsy. Some studies demonstrate that genetic mutations, as manganese superoxide single nucleotide polymorphism (MnSOD Ala16Val SNP) is associated to inflammatory, oxidative, and

apoptotic pathways, as well as to metabolic disturbances, as obesity and dyslipidemia. However, the relation between MnSOD Ala16Val SNP and epilepsy is not well known, as well as the genetic mutation influence on inflammatory, oxidative stress, apoptotic and dyslipidemia. Epilepsy patients and volunteers were recruited to participate. Metabolic profile, cytokines (TNF-α, IL-1β, IL-6, AChE), caspases (1, 8 e 3), DNA damage, and SOD activity were evaluated. The results demonstrated increased proportion of VV genotype in epilepsy

group when compared to control group. Increased levels of TNF-α, AChE, Caspase-8 and Picogreen in epilepsy patients with VV genotype compared to AA and AV from epilepsy and control groups. An interesting correlation among biomarkers in epilepsy group with VV genotype and seizure type was already observed. A correlation between TNF-α vs. Caspase-8, and cholesterol vs. Triglycerides was oberved in epilepsy group with VV genotype. The results from the study suggest the MnSOD Ala16Val SNP contributing to the disease prognosis, in view that the genetic polymorphism demonstrated influence in the inflammatory, oxidative, apoptotic, and metabolic biomarkers, contributing to the maintenance of inflammation in epilepsy.

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