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Alpha-lipoic acid mitigates toxic-induced demyelination in the Corpus Callosum by lessening of oxidative stress and stimulation of Polydendrocytes proliferation

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Multiple Sclerosis (MS), is a disease that degenerates myelin in central nervous system (CNS). Reactive oxygen species (ROSs) are toxic metabolites, and accumulating data indicate that ROSs-mediated apoptosis of oligodendrocytes (OLGs) plays a major role in the pathogenesis of MS under oxidative stress conditions. In this study, we investigated the role of endogenous antioxidant alpha-lipoic acid (ALA) as ROSs scavenger in the OLGs loss and myelin degeneration during cuprizone (cup)-induced demyelination in the experimental model of MS. Our results have shown that ALA treatment significantly increased population of mature OLGs (MOG+ cells), as well as decreased oxidative stress (ROSs, COX-2 and PGE2) and apoptosis mediators (caspase-3 and Bax/Bcl2 ratio) in corpus callosum (CC). Surprisingly, ALA significantly stimulates population of NG2 chondroitin sulfate proteoglycan positive glia (NG2+ cells or polydendrocytes), from week 4 afterward. Accordingly ALA could prevent apoptosis, delays demyelination and recruits OLGs survival and regeneration mechanisms in CC. We conclude that ALA has protective effects against toxic demyelination via reduction of redox signaling, and alleviation of polydendrocytes vulnerability to excitotoxic challenge.

Speaker Biography

Professor Mehdi Mehdizadeh has a PhD in Anatomical Sciences from Tehran University of Medical Sciences. He is a member of Cellular and Molecular Research Center, Faculty of Advanced Technologies in Medicine, Department of Anatomical Sciences, IUMS, Tehran, Iran. Professor Mehdizadeh worked as a Fellow at the German Research Center for Biotechnology on Transgenic Animals. He is author and co-author of over 100 peer-reviewed scientific articles, and has contributed numerous book chapters.

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