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P1. THE CELLULAR MECHANISM OF MERCURY NEUROTOXICITY AND A CASE REPORT

Murat BÜYÜKŞEKERCİ [1*], Erol Rauf AĞIŞ[1]
[1]Department of Pharmacology, Ankara Occupational Diseases Hospital, TÜRKİYE
*drmuratbs@gmail.com

In vitro and animal studies performed in the last 40 years revealed that the neurotoxicity induced by Methylmercury (MeHg) involves a) depletion of intracellular antioxidants b) inhibition of critical enzymes c) modulation of transporter activity and neurotransmitter receptor activity. As a long term effect MeHg is reported to modify gene expression and change the cellular signalling. The neurotoxicity mechanism of MeHg mainly depends it's electrophylic property, as it reacts with low and high molecular weight proteins possesing thiol and selenol groups. This reaction is partly responsible for the depletion of anti-oxidant capacity and accumulation of reactive oxygen species. Oxidative stress which is defined as the deterioration of the equilibrium of pro-oxidant/anti-oxidant in favor of pro-oxidant state plays the major role in MeHg induced toxicity. As a result of the interreaction with thiols and selenols the intracellular Ca concentration is modulated and neurotransmitter release is either inhibited or augmented. MeHg also increases the intracellular Ca concentration via raising the extracellular levels of glutamate. MeHg induces the deterioration of glutamate homeostasis by reacting with the glutamate receptors at synaptic vesicles and plasma membranes. It shows higher affinity to seleno groups than thiol groups. Because of that MeHg binds to specific thiol groups but also binds more stably to anti-oxidant selenoproteins like glutathione peroxidase (GPx) and thioredoxin reductase (TrxR). GPx and TrxR are inhibited following MeHg exposure. Inhibition of these selenoenzymes (perhaps degeneration of other selenoproteins) is the primary pathway of MeHg induced oxidative stress.