

“Repeated exposure to inorganic mercury produces histological pathogenesis similar to EAE model of multiple sclerosis in C57B/6 mice”

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Mercury exposure produces detrimental effects such as neurotoxicity to human body. Human exposure to mercury could happen through environmental contaminant, food contamination or during medical healthcare (amalgam or thimerosal). Multiple Sclerosis (MS) is an autoimmune neurodegenerative disease in CNS. Many human studies suggest the aggravation of clinical symptoms in MS patients who have been exposed to mercury. In this study we aimed to investigate the additive effect of repeated oral administration of mercuric sulfide on the behavioral, molecular and histological findings in experimental autoimmune encephalomyelitis (EAE) model of MS in C57BL/6 mice. Neurobehavioral scores, immune cell infiltration, demyelination, axonal injury and the expression of inflammatory cytokines was investigated. Forty female C57B/6 mice were randomly selected in four different groups. The control group was given vehicle. In EAE groups, MS symptom was developed by subcutaneous injection of MOG35-55 followed by intraperitoneal injection of pertussis toxin. In other EAE group, mice were given oral mercuric sulfide at the dose of 1g/kg/day for seven consecutive days. The mercury group was intact mice that received merely oral mercuric sulfide for seven consecutive days. After 28 days, mice were anesthetized by isoflurane and the spinal cord was removed and the upper section was fixed for histological staining. The lower section was stored at -70°C for real-time RT-PCR experiments. Treatment with mercuric sulfide during disease onset significantly increased the neurobehavioral scores of EAE. This effect was accompanied by increased inflammation, demyelination and axonal damage. However, no significant

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change in the inflammatory cytokines was observed during administration of mercuric sulfide. Our results showed that repeated treatment of mercuric sulfide following induction of experimental model of multiple sclerosis in mice significantly increased the severity of the movement disabilities and degeneration of spinal cord neurons. This could lead us to seek for comparable mechanism of toxic actions in multiple sclerosis and in mercury poisoning.

Keywords: EAE; Multiple sclerosis; Mercuric sulfide