

Ameliorating effect of melatonin on mercuric chloride-induced neurotoxicity in rats

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Abstract

Background: Mercury is a highly toxic metal. It induces its toxicity via production of reactive oxygen species. Brain tissues are more susceptible to oxidative damage. Melatonin and its metabolites are free radical scavengers. The aim of this work is to elucidate the neuroprotective effect of melatonin on mercuric chloride-induced neurotoxicity in rats.

Methods: Fifty male albino rats were used and divided into five groups. Group I acts as normal control. Group II (LD HgCl₂) received mercuric chloride at a dose of 2 mg/kg. Group III (HD HgCl₂) received HgCl₂ at a dose of 4 mg/kg. Rats in group IV (LD HgCl₂ MLT) received HgCl₂ 2 mg/kg Melatonin 5 mg/kg. Rats in group V (HD HgCl₂MLT) received HgCl₂ 4 mg/kg Melatonin 5 mg/kg.

Results: This study revealed that mercuric chloride decreased the activity of superoxide dismutase, catalase and glutathione peroxidase enzymes and increased malondialdehyde levels. Toxicity of mercuric chloride lead to upregulation of the gene expression level vascular endothelial growth factor.

HgCl₂ induced fragmentation of rough endoplasmic reticulum, ballooning of Golgi apparatus, nuclear and cytoplasmic degeneration of pyramidal neurones of rat cerebral cortex. This neuronal damage caused by HgCl₂ was significantly improved by melatonin.