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OXIDANT-INDUCED NEUROTOXICITY WAS BLOCKED BY ANTIOXIDANTS AND METAL CHELATORS IN MOUSE CEREBRAL NEURON CULTURES

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It is well known that oxygen radicals induce neuronal cell damage by initiation of lipid peroxidation chain reaction. Recent work has been also demonstrated that enzymatically generated free radicals cause the release of glutamate and aspartate from cultured rat hippocampal slices. In order to characterize the mechanism of oxidant-mediated neurotoxicity in mouse cerebral neuron cultured, cultured cells were exposed to 20m μ/ml glucose oxidase as H_2 O_2 generation system after 2 hours of preincubation with oxygen radical scavengers and metal chelators. Cell viability was determined by MTT assay and neurofilament ELISA assay. Glucose oxidase-induced neurotoxicity resulted in significant cell death in a time-dependent manner on cerebral neuron cultured. The neurotoxicity induced by oxygen radicals was blocked by superoxide dismutase(SOD)(1-60 μ g/ml), catalase(1-100 μ g/ml) and allopurinol(10-150 μ M) in a dose-dependent manner. Tetrakis(2-pyridymethyl) ethylene diamine(TPEN), a metal chelator(5-30 μ M) also showed positive effect against oxidant-induced neurotoxicity in mouse cerebral neuron cultures. These results indicate that selective antioxidants and metal chelators such as catalase and TPEN are effective in protecting oxidant-induced neurotoxicity in CNS.

keyword : Neurotoxicity, Anti-oxidant, metal chelator