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Poster Presentation

Terminalia Chebula Attenuates Quinolinate-Induced Oxidative PC12 and OLN-93 Cells Death

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Abstract

Introduction: Quinolinic acid (QA) is a product of tryptophan degradation and its pathologic accumulation has been found to induce neuroinflammatory and demyelinating diseases such as multiple sclerosis via excessive free radicals generation. Recent studies showed Terminalia chebula has several pharmacological effects such as antioxidant, anti-inflammatory and neuroprotective properties. The aim of this study was evaluation of the protective effect of T. chebula alcoholic extract (TCAE) on oxidative PC12 and OLN-93 cells death induced by QA. **Materials and Methods:** The cells were pretreated with TCAE (6.25-50 µg/mL) for 2 h and then were subjected to QA (8 mM) for 24 h in which the same treatments were applied. Cell viability and the parameters of redox status including the levels of intracellular reactive oxygen species (ROS), lipid peroxidation and oxidative DNA damage were measured using 2-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium (MTT), 2,7-dicholorofluorecin diacetate (DCF-DA), thiobarbituric acid and comet assays, respectively. **Results:** Based on Folin-Ciocalteu method, the total phenolic compounds in TCAE were estimated about 1.18%. TCAE at concentration ranges of 6.25-50 µg/mL had no toxic effect on cell viability ($p>0.05$). Treatment with TCAE significantly increased cell viability following QA insult at concentrations above 25 µg/mL ($p<0.01$). Cytoprotective potential of TCAE also ameliorated ROS accumulation, lipid peroxidation and DNA damage induced by QA. **Conclusion:** These data suggest that TCAE exhibits neuroprotection and oligoprotection potential by means of alleviating oxidative stress parameters.

Keywords: Quinolinic acid, Terminalia chebula, PC12 cells, OLN-93 cells, Gliotoxicity, Neurotoxicity

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