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Neuroprotective effect of tangeretin against potassium dichromate induced acute brain injury in rats via modulating the Nrf2 signaling pathway and quenching the release of inflammatory mediators and apoptosis in rats

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Dotassium dichromate (PD) is an environmental xenobiotic commonly recognized as teratogenic, carcinogenic, and mutagenic in animals and humans. The present study was conducted to investigate the role of tangeretin (TNG) as a neuro-protective drug against PD-induced brain injury in rats. Thirty-two male adult Wistar rats were blindly divided into four groups (8 rats/group). The first group received saline intranasally (i.n.). The second group received a single dose of PD (2 mg/kg, i.n.). The third group received TNG (50 mg/ kg; orally), for 14 days followed by i.n. of PD on the last day of the experiment. The fourth group received TNG (100 mg/kg; orally) for 14 days followed by i.n. of PD on the last day of the experiment. Behavioral indices were evaluated 18 h after PD administration. Neuro-biochemical indices and histopathological studies were evaluated 24 h after PD administration. Results of the present study revealed that rats intoxicated with PD inducedoxidative stress and inflammation via an increase in malondialdehyde (MDA) and a decrease in nuclear factor erythroid 2-related factor 2 (Nrf2) signaling pathway and glutathione (GSH) levels with an increase in brain contents of tumor necrosis factor-alpha (TNF-a) and interleukin (IL-6). Pre-treatment with TNG (100 mg/ kg; orally) ameliorated behavior, cholinergic activities, and oxidative stress and decreased the elevated levels of pro-inflammatory mediators; $TNF-\alpha$ and IL-6 with a decrease in brain content of chromium residues detected by Plasma-Optical Emission Spectrometer. Also, the histopathological picture of the brain was improved significantly in rats that received TNG (100 mg/kg). Additionally, TNG decreased caspase-3 expression in the brain of PD rats. In conclusion, TNG possesses a significant neuroprotective role against PD-induced acute brain injury via modulating the Nrf2 signaling pathway and quenching the release of inflammatory mediators and apoptosis in rats.



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Biography

Ahmed A Sedik, An assistant Professor of Pharmacology, working at Medical Research and Clinical Studies Institute, National Research Centre, Egypt. He has earned my master and PhD studies from Cairo University at 2016 and 2020, respectively. Nowadays; he had more than 20 innovative scientific publications in highly cited journals and shared in more than 7 national and international projects. He have earned a scholarship from Campus France as a Post- doctoral Researcher, Université Clermont Auvergne, France from 1October 2024 to 3 April 2025

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