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Chemomodulatory effect of betanin against paracetamol and diclofenac induced neurotoxicity and endocrine disruption in rats

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Paracetamol and diclofenac are two of the most popular analgesics and anti-inflammatory medications. Despite of their several therapeutic benefits, their over consumption led to subsequent cellular damage. Their cytotoxicity is attributed to reactive radical generation. Betanin has antioxidant and anti-inflammatory properties. The protective effects of betanin against paracetamol or diclofenac induced neurotoxicity or endocrine disruption has not been investigated before. Therefore, this study aims to explore the protective potential of betanin against paracetamol or diclofenac neurotoxicity and endocrine disruption in a rat model. In brain, paracetamol (400 mg/ kg) and diclofenac (10mg/kg) enhanced DNA fragmentation and lipid peroxidation level. A depletion of GSH content concomitant with a reduction in the activities of antioxidant enzymes (HOX-1, POX-1, CAT and SOD) were detected. Serotonin, nor-adrenaline and dopamine levels were markedly reduced after paracetamol and diclofenac challenge. In serum, a significant reduction of testosterone, TRH, TSH, T3 and T4 were associated with the enhanced oxidative damage. Co-treatment of rats with betanin (25mg/kg) by gavage for 28 consecutive days ameliorated most of the biochemical and histopathological changes induced by paracetamol or diclofenac. In conclusion, betanin exerted a potential chemomodulatory effect against paracetamol or diclofenac overconsumption induced neurotoxicity and endocrine disruption.

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