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Beta-Carotene Attenuates Cognitive and Hepatic Impairment in Thioacetamide-Induced Rat Model of Hepatic Encephalopathy via Mitigation of MAPK/NF-kB Signaling Pathway

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Abstract: Liver fibrosis is a severe worldwide health concern due to various chronic liver disorders. Hepatic encephalopathy (HE) is one of its most common complications affecting liver and brain cognitive function. Beta-Carotene (B-Car) is an organic, strongly colored red-orange pigment abundant in fungi, plants, and fruits. The study attempted to know B-Car neuroprotective potential against thioacetamide (TAA)-induced neurotoxicity and cognitive decline in HE in rats. Hepatic encephalopathy was induced by TAA (100 mg/kg, i.p.) three times per week for two weeks. B-Car was given orally (10 or 20 mg/kg) daily for two weeks after TAA injections. Organ body weight ratio, Serum transaminase activities, liver's antioxidant parameters, ammonia, and liver histopathology were assessed. Also, the brain's mitogen-activated protein kinase (MAPK), nuclear factor kappa B (NF-kB), antioxidant parameters, adenosine triphosphate (ATP), adenosine monophosphate (AMP), norepinephrine (NE), dopamine (DA), serotonin (5-HT), 5-hydroxyindoleacetic acid (5-HIAA) cAMP response element-binding protein (CREB) expression and B-cell lymphoma 2 (Bcl-2) expression were measured. The brain's cognitive functions (Spontaneous locomotor activity, Rotarod performance test, Object recognition test) were assessed. B-Car prevented alteration of the brain's cognitive function in a dose-dependent manner. The histopathological outcomes supported these biochemical evidences. Based on these results, it could be established that B-Car could be assigned to treat the brain's neurotoxicity consequences of HE via downregualtion of MAPK/NF-kB signaling pathways.

Keywords: beta-carotene, liver injury, MAPK,NF-κB, rat, thioacetamide

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