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Lycopene prevents 3-nitropropionic acid-induced mitochondrial oxidative stress and dysfunctions in nervous system.

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Abstract

3-nitropropionic acid (3-NP), an irreversible inhibitor of succinic acid dehydrogenase (SDH), induces neurodegeneration similar to that observed in Huntington's disease (HD). The present study was designed to investigate neuroprotective effect of lycopene on 3-NP induced mitochondrial dysfunctions and oxidative stress. Rats administered with 3-NP (25 mg/kg, intraperitoneally) for four consecutive days exhibited deficits in cognitive and motor functions on day 15, whereas, lycopene (10 mg/kg, orally) administration for 15 days ameliorated 3-NP-induced neurobehavioral deficits. The activities of mitochondrial Complexes-II, IV and V were found to be significantly lowered in striatum along with the reduction in mitochondrial respiration. However, no significant change in Complex-I activity was observed in 3-NP treated animals. 3-NP administration increased the rate of reactive oxygen species (ROS) and nitrite production which was accompanied by increase in lipid peroxidation in mitochondria. Thiol content and superoxide dismutase activity were depressed in 3-NP treated brain. 3-NP treatment induced mitochondrial swelling with increased cytochrome c release. Expression of p53 and active caspase-3 were increased in 3-NP treated animals. On the other hand, lycopene administration exhibited protective effect on 3-NP induced mitochondrial dysfunctions and oxidative stress. The results of the present study provide evidence for effectiveness of lycopene in preventing mitochondrial dysfunctions in 3-NP-induced HD.

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