Abstract

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Coenzyme Q10 treatment ameliorates cognitive deficits by modulating mitochondrial functions in surgically induced menopause.

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OBJECTIVE: The mechanisms associated with cognitive decline in post-menopausal state driven by loss of ovarian function and reduced estrogen levels are not well understood. The aim of the present study is to investigate the role of mitochondrial dysfunctions in cognitive impairment in post-menopausal state and to evaluate the protective effect of Coenzyme Q10 (CoQ10).

METHODS AND RESULTS: A significant decline in cognitive functions was observed in mice after four weeks of ovariectomy as assessed by morris water maze and elevated plus maze. Administration of CoQ10 (10mg/kg body weight, orally) daily for 4weeks was found to reverse cognitive deficits observed in ovariectomized (Ovx) mice. The activity of mitochondrial electron transport chain components; NADH: cytochrome c reductase, succinate dehydrogenase and cytochrome c oxidase was significantly reduced in the brain of Ovx mice. This was accompanied by higher levels of ROS, protein carbonyls, lipid peroxidation, mitochondrial swelling and reduced activity of aconitase. The levels of GSH were observed to be significantly lowered resulting in reduced redox ratio (GSH/GSSG) in brain of Ovx mice. Activities of antioxidant enzymes; superoxide dismutase and catalase were also found to be reduced in brain of Ovx animals. CoQ10 supplementation to Ovx mice mitigated the mitochondrial dysfunctions and oxidative stress.

CONCLUSIONS: Thus, the data indicates that CoQ10 improves cognitive decline in post-menopausal state by modulating mitochondrial functions and oxidative stress.

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