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The Effects of the Toxicity of (Fe (so4).7H2o) on the Isolated Mitochondria from the Brain of Rat

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Abstract

Introduction: Iron, through the reaction of Fenton, generates free radicals such as active oxygen radicals and activates the oxidative stress pathway. The oxidative stress due to the increased iron level in the brain regions plays an important role in creation of neurodegenerative diseases.

Methods and Results:In this study, the mitochondria of the brain tissue of Wild Wistar Rat isolated from various centrifuge rounds and with the concentrations of Fe (so4).7H20 were incubated at 30 and 60 minutes. To determine IC50 Fe (so4).7H20, the mitochondrial survival ratio was measured by MTT test. Mitochondrial suspension with the concentration of 0.5 mg protein/ml at various concentrations of Fe (so4).7H20 was placed in a shaker incubator at 37° C for 30 and 60 minutes. Then the activity of mitochondrial complex 2 and the formation ratio of reactive oxygen species was investigated. The results showed that IC50 ratio for Fe (so4).7H20 was 20 and 5 μ g/ml at 30 and 60 minutes, respectively, and mitochondria incubation isolated from the brain tissue of the rat with Fe (so4).7H20 can disrupt be the electron transfer chain and significantly increases the formation of reactive oxygen species compared to the control group (P <0.001).

Conclusions: The findings of this study indicate that Fe (so4).7H2o disrupts electron transfer chain in the mitochondria and causes increasing ROS production. This excessive increase of ROS can activate the oxidative stress pathway and ultimately activate the cell toxicity pathways.

Key words: Mitochondria, Fe (so4).7H2o, Oxidative stress, Brain, Rat

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