Abstract

Introduction: According to previous studies, phosphine (PH3) gas released from aluminum phosphide can inhibit cytochrome oxidase in cardiac mitochondria and cause free radicals, alter oxidative stress in the antioxidant defense system, and Be cardiac toxicity. Accordingly, it can be predicted that antioxidants and mitochondrial protective agents are a rational strategy to prevent aluminum phosphide induced cardiac toxicity. On the other hand, the available evidence suggests that cannabinoids have protective effects in reducing oxidative stress, mitochondrial damage and are cardiovascular. The aim of this study was to evaluate the effects of trans-9-tetrahydrocannabinol (THC) on aluminum phosphide induced toxicity in isolated cardiomyocytes and mitochondria.

Method: Mitochondria isolated from rat heart were treated with different concentrations of THC (10, 50 and 100 μM) and IC50 of aluminum phosphide, then using flow cytometric and biochemical methods of mitochondrial toxicity parameters such as ROS formation, lipid peroxidation, mitochondrial swelling, mitochondrial membrane potential collapse, and changes in mitochondrial function.

Conclusion: Exposure to aluminum phosphide alone increased mitochondrial oxygen species (ROS) production, lipid oxidation, mitochondrial membrane potential collapse,and mitochondrial swelling compared with controls. However, introduction of THC (10, 50 and 100 μM) attenuated aluminum phosphide induced changes in all of these parameters in a THC-dependent manner. Interestingly, the results showed significant protective effects of THC by reducing the various parameters of mitochondrial toxicity and oxidative stress induced by aluminum phosphide in mitochondria. This is the first report to show the protective effects of THC against aluminum phosphide toxicity, and these effects may be related to the presence of cannabinoid receptors in the mitochondria or the antioxidant potential of THC.

Keywords: Aluminum Phosphide, Tetrahydrocannabinol, Decongestants, Mitochondria, Rice Tablets, Cardiac Toxicity