Diphenyl Diselenide reverts the redox imbalance induced by Acetaminophen in a hepatotoxicity model in mice

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The acetaminophen (APAP) is employed frequently in the medicine due to the pharmacological properties, such as analgesic and antipyretic activity. However, when a high dose of APAP is ingested, it could induce the acute hepatic failure and concomitantly mitochondrial oxidative stress and increase in generation of reactive oxygen species. This effect is at least due the NAPQI generation, which impairment the antioxidant system cellular and mitochondrial. Since, studies have been focused in compounds that display an antioxidant capacity, in special the Diphenyl diselenide [(PhSe)2]. Therefore, the objective of this work was to investigated the ability of the (PhSe)2 revert the redox imbalance induced by APAP overdose. The animals received a dose of APAP 600mg/Kg injected intraperitoneally (IP), and one hour after receiving 15.6 mg/kg (PhSe)2 via IP. Three hours after the animals were sacrificed and then hepatic mitochondria were isolated and analyzed the oxidative damage markers. In this work, the (PhSe)2 reduced the production ROS, MDA levels, as well as carbonyl protein and depolarization of the mitochondrial transmembrane potential induced for acetaminophen toxicity. The (PhSe)2 maintains the glutathione levels after the hepatic injury. In conclusion, the present results provide evidences for that diphenyl diselenide is an effective tool in to modulate the oxidative stress markers, and enabled to reduce the mitochondrial dysfunction.

Keywords: Acetaminophen, (PhSe)2, Mitochondrial Dysfunction, Oxidative Stress.

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