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The Hepatoprotective Effects of Aquatic Extract of Levesticum Officinale against Paraquat Toxicity of Hepatocytes

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Abstract : Paraquat is widely used as a strong nitrogen-based herbicide for controlling of weeds in agriculture. This poison is extremely toxic for humans which induces several – organ failure by accumulation in cells and many instances of death occurred due to its poisoning. Paraquat metabolized primarily in the liver. The purpose of this study was to assess the effects of aquatic extract of levisticum officinale on oxidative status and biochemical factors in hepatocytes exposed to paraquat. Our results determined that hepatocytes destruction induced by paraquat is mediated by reactive oxygen species (ROS) production, lipid peroxidation and decrease of mitochondrial membrane potential were significantly ($P<0.05$) prevented by aquatic extract of Levisicum officinale (100, 200 and 300 µg/ml). These effects of paraquat also prevented via antioxidants and ROS scavengers (α-tocopherol, DMSO, manitol), mitochondrial permeability transition (MPT) pore sealing compound (carnitine).MPT pore sealing compound inhibited the hepatotoxicity, indicating that paraquat induced cell death via mithochondrial pathway. Pretreatment of hepatocytes with aquatic extracts of Levisticum officinale, antioxidants and ROS scavengers also blocked hepatic cell death caused by paraquat, suggesting that oxidative stress may be directly induced decline of mithochondrial membrane potential. In conclusion, paraquat hepatotoxicity can be attributed to oxidative stress and continued by mithochondrial membrane potential disruption. Levisticum officinale aquatic extract, presumably due to its strong antoxidant properties, could protect the destructive effects of paraquat on rat hepatocytes.

Keywords : hepatocyte protection, levisticum officinale, oxidative stress, paraquat
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