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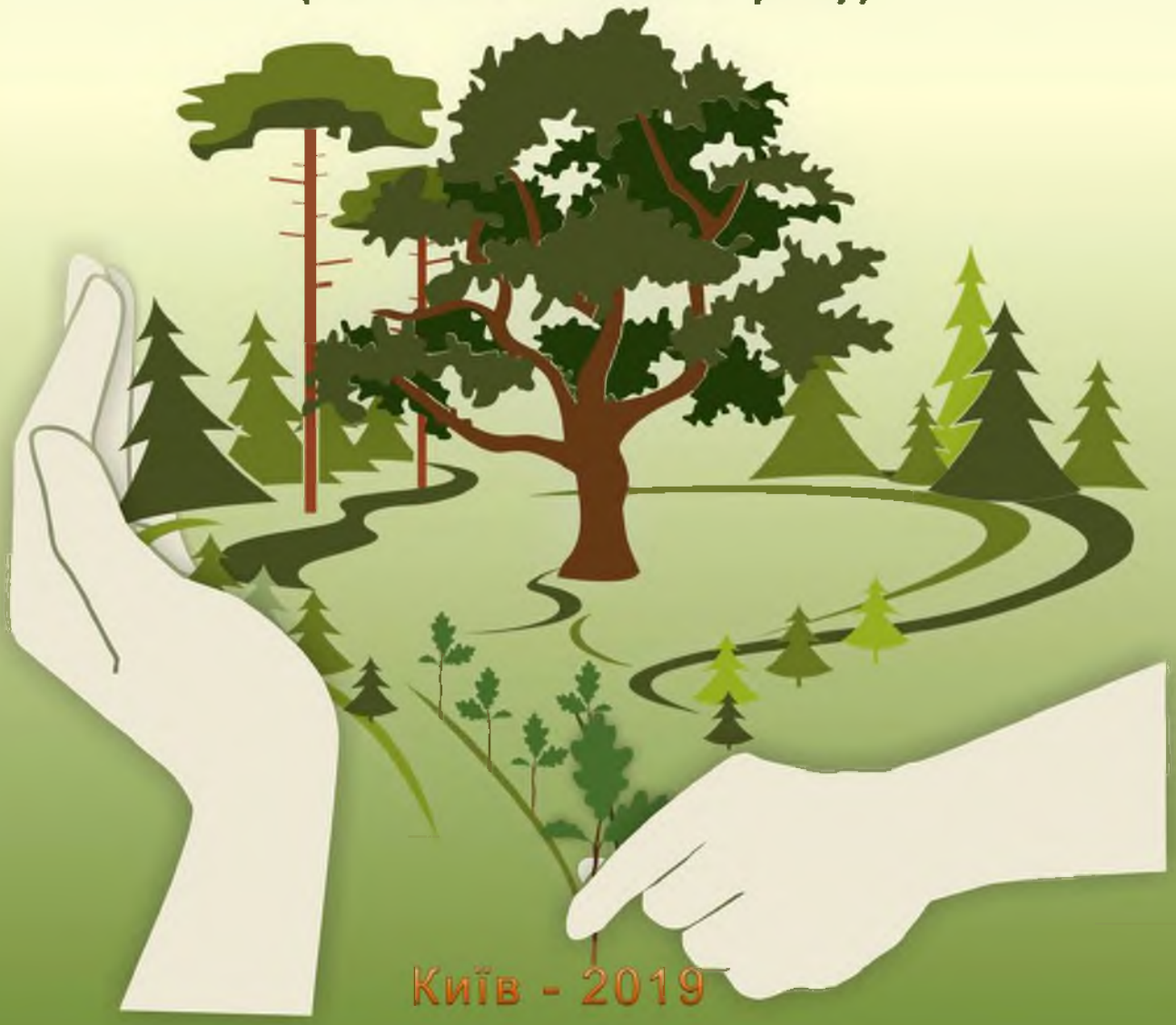

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PLANT POLYPHENOLS REGULATE FUNCTIONS OF ISOLATED RAT LIVER MITOCHONDRIA AND PREVENT MITOCHONDRIA IMPAIRMENTS *IN VIVO* AND *IN VITRO*

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There is a strong interest to compounds of plant origin as potential medicinal agents due to their effectivity, safety and wide range of biological activity. The present study was undertaken for further elucidation of the mechanisms of plant polyphenols biological activity, focusing on the antioxidative and protective effects in free radical-generating systems and mitochondria. The aim of the present work was to evaluate biochemical effects of plant polyphenols and terpenoids using isolated rat liver mitochondria *in vitro* and toxic liver damage in rats *in vivo*. Terpenoid ferutinn isolated from the plant *Ferula tenuisecta* considerably increased the permeability of artificial and cellular membranes to Ca^{2+}-ions. Ferutinin in a dose-dependent manner (5-27 μM) decreased the rate of ADP-stimulated oxygen consumption and resulted in uncoupling of respiration of isolated rat liver mitochondria. These effects depended on the presence of Ca^{2+}-ions in the media. In the presence of Ca^{2+}-ions, ferutinin induced considerable depolarization of mitochondrial membrane and permeability transition pore formation, the last effect is inhibited by cyclosporin A. We confirmed that mitochondrial effects of ferutinin were induced by stimulation of mitochondrial membrane Ca^{2+}-permeability as well as by transportation of other ions. The treatment of rats with cranberry flavonoids (7 mg/kg, 30 days) during chronic carbon tetrachloride-induced intoxication prevented mitochondrial damage, including fragmentation, rupture and local loss of the outer mitochondrial membrane. The treatment of rats chronically receiving ethanol (4 g /kg bw, 8 weeks) with cranberry polyphenols (daily, 4 mg /kg bw) partially prevented alcoholic liver damage, ameliorating steatosis and inflammatory signs in the liver, decreasing serum and liver triglyceride contents, ALT and AST activities. The polyphenols restored mitochondrial functional activity in these animals, inhibited Ca^{2+} - induced mitochondrial permeability transition in the liver, free radical generation, and membrane lipid peroxidation. *In vitro*, cranberry polyphenols effectively scavenged different types of free radicals and prevented lipid peroxidation and glutathione oxidation and considerably prevented mitochondrial ultrastructure oxidative impairments. In conclusion, the hepatoprotective potential of plant polyphenols could be due to specific prevention of rat liver mitochondrial damage.