Free Radical Scavenging Activity of Silymarin with Refrence to Depleted Uranium Induced Mitochondrial Dysfunction

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ABSTRACT

Introduction: Recently depleted uranium is being widely used as antiarmour ammunition and at very high temperature, results in information of an aerosol of very small uranium oxide particles, which may be inhaled. It is alleged that these particles represent a new battlefield hazard because of the chemical toxicology and/or radioactivity.

Method & materials: Male Wistar strain albino rats were divided at random into 3 groups. In the first group normal saline was administrated intraperitioneally (i.p), this group served as controls. The second group were i.p injected with uranyl acetate 40 mg/kg. In the third group the rats received 100 mg/kg/day silymarin by i.p injection for 5 days and 1hr after the last injection; animals were injected with a single i.p dose of uranyl acetate (40 mg/kg). Silymarin was tested for its free radical scavenging activity and protective role against mitochondrial dysfunction in uranyl acetate stressed rats.

Results: Lipid peroxidation activity was increased and activity of mitochondrial enzymes (cytochrome-coxidases, NADH-dehydrogenase, α -ketoglutarate dehydrogenase and succinate dehydrogenase) and glutathione was decreased in the liver and kidney of rats intoxicated with uranyl acetate when compared to control rats. In traperitoneal administration of silymarin significantly reduced the lipid peroxidation, increased the activity of mitochondrial enzymes and increased glutathione to near control level.

Conclusion: These results suggest that the major components in silymarin (including silybin A and B, isosilybin A and B, cis-silybin A and B) play a protective role through their free redical scavenging properties.

Keyword: Silymarin, Depleted Uranium, Oxidative Stress, Mitochondrial Enzymes

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