

## **Diazinon-induced oxidative stress and renal dysfunction in rats**

### **Abstract**

Diazinon (O,O-diethyl-O-[2-isopropyl-6-methyl-4-pyrimidinyl] phosphoro thioate), an organo-phosphate insecticide, has been used worldwide in agriculture and domestic for several years, which has led to a variety of negative effects in non target species including humans. However, its nephrotoxic effects and mechanism of action has not been fully elucidated so far. Therefore, the present study was aimed at evaluating the nephrotoxic effects of diazinon and its mechanism of action with special reference to its possible ROS generating potential in rats. Treatment of rats with diazinon significantly enhances renal lipid peroxidation which is accompanied by a decrease in the activities of renal antioxidant enzymes (e.g. catalase, glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase, glutathione S-transferase) and depletion in the level of glutathione reduced. In contrast, the activities of renal  $\gamma$ -glutamyl transpeptidase and quinone reductase were increased. Parallel to these changes, diazinon treatment enhances renal damage as evidenced by sharp increase in blood urea nitrogen and serum creatinine. Additionally, the impairment of renal function corresponds histopathologically. In summary, our results indicate that diazinon treatment eventuates in decreased renal glutathione reduced, a fall in the activities of antioxidant enzymes including the enzymes involved in glutathione metabolism and excessive production of oxidants with concomitant renal damage, all of which are involved in the cascade of events leading to diazinon-mediated renal oxidative stress and toxicity. We concluded that in diazinon exposure, depletion of antioxidant enzymes is accompanied by induction of oxidative stress that might be beneficial in monitoring diazinon toxicity. © 2010 Elsevier Ltd. All rights reserved.