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ABSTRACT BOOK

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EFFECT OF CHRONIC CADMIUM EXPOSURE ON GLUTATHIONE PEROXIDASE AND GLUTATHIONE S-TRANSFERASE ACTIVITIES IN RAT: THE ROLE OF SELENIUM

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Cadmium (Cd) is an ubiquitous toxic metal that may induce oxidative damage by disturbing the prooxidant-antioxidant balance in the tissues. Among antioxidant micronutrients, selenium (Se) is an essential dietary trace element which plays an important role in a number of biological processess. The effect of cadmium on the activity of glutathione peroxidase (GSH-Px) and glutathione S-transferase (GST) which play an important role in the detoxification of xenobiotics, was studied in the liver, kidney and heart of rats. Furthermore, the role of selenium in the modulation of Cd toxicity with respect to GSH-Px and GST was also evaluated. In our experiments, cadmium exposure (15 mg Cd/kg body wt./day as $CdCl_2$ for 4 weeks) to rats (n=30, male Wistar rats, age 8 weeks) resulted in decreased GSH-Px and GST activity in all organs present in the order liver > kidney > heart. Oral administration of Se (0.5 mg Se/kg body wt./day as Na_2SeO_3 for 4 weeks) caused a significant increase in GSH-Px activity in the order liver > heart > kidney. Selenium administration caused an increase in total GST activity in liver but a decrease in kidney and heart. Simultaneous administration of Cd and Se resulted in an increase GSH-Px and GST activity in all tissues under study. Thus, the mechanism by which Se decreases Cd toxicity in rats seems to rely on the protection of the enzyme systems GSH-Px and GST in the three organs, possibly by forming non-toxic cadmium selenide. Selenium efficiently protects tissues from cadmium-induced oxidative damage.