[PA4-35] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Cadmium altered zinc homeostasis in the Neuronal Cell

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In this study, we investigated the effect of cadmium on genes expression related to zinc homeostasis in HT22 hippocampal neuron cell line by RT-PCR and western blotting technics. In the time-course effect, cadmium up-regulated the relative levels of MT-I and MT-II to㉥b-actin at 4 hr after treatment. These effects were consistent with MT-I/II protein contents by western blot analysis. But MT-III, a specific MT isoform in brain, was not affected by cadmium. In the dose-dependent effect, cadmium (0,5 - 2.0 ㎍M) up-regulated MT-I and MT-II levels by a dose-dependent manner. Cadmium dose-dependently down-regulated zinc transporter (ZnT-1), which serves as zinc efflux transporter. Our results suggest that cadmium can exert neurotoxicities (including degenerative neuron disease) via alteration of zinc metabolism in CNS.

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Effects of Dially Sulfide on Thioacetamide-induced Hepatotoxicity

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Effects of diallyl sulfide (DAS), a component of garlic, on thioacetamide-induced hepatotoxicity were investigated in male ICR mice. When mice treated subcutaneously with 100, 200 and 400 mg/kg of DAS in corn oil for three consecutive days, the activity of cytochrome P450 (P450) 2E1-selective p-nitrophenol hydroxylase was dose-dependently suppressed. In addition, the activities of P450 2B-selective benzyloxyresorufin O-debenzylase and pentoxyresorufin O-depentylase were dose-dependently induced by the treatment with DAS. To investigate a possible role of metabolic activation by P450 enzymes in thioacetamide-induced hepatotoxicity, mice were pre-treated with 400 mg/kg of DAS for 3 days, followed by a single intraperitoneal treatment with 100 and 200 mg/kg of thioacetamide in saline for 24 hr. The activities of serum alanine aminotranferase and aspartate aminotransferase greatly increased by thioacetamide were recovered in DAS-pretreated animals. Taken together, our present results indicated that thioacetamide might be activated to its hepatotoxic metabolite(s) by P450 2E1, not by P450 2B, in male ICR mice.

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The Influence of Bisphenol A on the Thyroid Hormone System in vivo

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