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Cadmium is heavy metals that cause vascular lesions such as artherocslerosis and hypertension. However, less information is available concerning for its toxicity. The results from previous in vitro and in vivo studies demonstrate that cadmium cation can induce an oxidative stress in various tissues. Oxidative stress has been shown to be involved in the mutagenicity and apoptosis of mammalian cells treated with cadmium.
In this study, we investigated whether cadmium cause cell death in vascular endothelial cells. We also examined the effect of cadmium on mitochondrial function by MTT assay. Cell death was quntitatively determined by measuring lactate dehydrogenase (LDH) activity, propidium iodide(PI)-uptake and by observing morphology in CPAE cells.
In CPAE, a significant decrease was observed in mitochondrial function 24, 36 hours after the treatment with 10-100μM CdCl2. Cadmium-induced cell injury was also observed morphologically by microscope. We also observed cadmium induced increase in PI-uptake.
In conclusion, our results suggest that cadmium can cause mitochondrial dysfunction and subsequent cell death in vascular endothelial cells.

[PA3-3] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Cardiotoxic effect of carbofuran in rat.

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Of all pesticides, carbamates are known to be most common, since alternatives such as organophosphates have long lifetime and are extremely toxic to produce a delayed neurotoxic effect. Among the available carbamates, carbofuran is the most widely used one in agriculture and forestry as a broad-spectrum systemic insecticide. Although a number of studies about toxicity of carbofuran have been reported, its cardiovascular toxicity has not yet been studied.
In the present study, we investigated its cardiovascular toxic effect in isolated Langendorff rat heart and in anesthetized rat in vivo. Isolated rat heart, carbofuran (10μM) caused a significant depression in the left ventricular developed pressure (LVDP), indicating contractile dysfunction by carbofuran. Carbofuran (10μM) also decreased coronary flow rate (CFR) in isolated heart, indicating carbofuran-induced coronary dysfunction. In anesthetized rat model, carbofuran (10 mg/kg) significantly reduced blood pressure and heart rate, and altered ventricular component of electrocardiogram. These results suggest that carbofuran can cause cardiac dysfunction in rat in vivo and vitro.

[PA3-4] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

A Cancer Risk Assessment of Di(2-ethylhexyl)phthalate in Powdered Milk for Infant Exposure

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The United States Environmental Protection Agency(EPA) characterized the cancer hazard of di(2-ethylhexyl)phthalate(DEHP) as a B2 group(probable human carcinogen) and proposed "Guidelines for Carcinogen Risk Assessment". This guidelines proposed alternative methods for analyzing carcinogen