[PA4-4] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Acute cocaine poisoning in a body packer

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A 35-year-old Perubian who suffered from grand mal seizures died in aircraft on his way from United States to Hongkong. When he boarded the aircraft, he was normal but later he suffered from hyperthermia, dizziness, abdominal pain, agitation and convulsion in the flight before dead. While performing the autopsy, 115 cocaine packs were found in the GI tract. To determine the concentration of cocaine and its metabolites, blood, urine, bile juice, gastric content, liver, spleen, heart, kidney, cellebrum and lung were taken and analyzed. Biological specimen were extracted by liquid-phase extraction using CH3Cl : IPA (= 9:1), derivatized with BSTFA and analyzed using GC/MS with selective ion monitoring mode. Dextromethorphan was used as internal standard. The purity of cocaine in 22 packs out of 115 ranged from 71.6 to 98.1 %. High levels of cocaine, egconinemethylester and benzoylegconine were found in the blood (0.96, 5.59 and 3.09 μg/ml), urine (32.85, 53.17 and 145.35 μg/ml), bile (2.96, 14.84 and 4.89 μg/ml), gastric content (3.19, 0.86 and 2.27 μg/ml), liver (0.12, 2.88 and 0.73 μg/ml), spleen (2.90, 0.18 and 0.28 μg/ml), heart (3.54, 0.31 and 0.32 μg/ml), kidney (4.46, 0.30 and 0.44 μg/ml), cellebrum (3.94, 0.31 and 0.18 μg/ml) and lung (3.44, 0.20 and 0.26 μg/ml), respectively.

[PA4-5] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Inhibition of cyclooxygenase-2 expression by Caffeoyl-4-dihydrocaffeoyl quinic acid in macrophages

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Inducible cyclooxygenase-2 (COX-2) has been implicated in the processes of inflammation and carcinogenesis. Thus, the potential COX-2 inhibitors have been considered as anti-inflammatory or cancer chemopreventive agents. In this study, we investigated the effect of Caffeoyl-4-dihydrocaffeoyl quinic acid (CDCQ) isolated from Salicornia herbacea on the expression of cyclooxygenase (COX-2) in lipopolysaccharide (LPS)-activated RAW 264.7 macrophages. When CDCQ was treated with LPS, the prostaglandin E_2 production and COX-2 gene expression induced by LPS were markedly reduced in a dose-dependent manner. Transient transfection experiments showed that LPS-induced increase in COX-2 promoter activities was suppressed by CDCQ. Moreover, transient transfection experiments using reporter vectors harboring deleted COX-2 promoters revealed that the transcriptional factor AP-1, but not NF-kB, between -574 and -51 in COX-2 promoter could be important for the inhibition of LPS-induced COX-2 mRNA by CDCQ. This study suggests that modulation of COX-2 by CDCQ may be important in the prevention of carcinogenesis and inflammation.

[PA4-6] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

An aqueous extract isolated from Platycodon grandiflorum reduced acetaldehyde-induced collagen and alpha-SMA expression in hepatic stellate cells

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The increased deposition of extracellular matrix by hepatic stellate cells following liver injury in a process known as activation is considered a key mechanism for increased collagen content of liver during the development

of liver fibrosis. In this study, we investigated the protective effects of an aqueous extract from the roots of Platycodon grandiflorum A. DC (Campanulaceae), Changkil (CK), on hepatic fibrosis in hepatic stellate cells. We report that CK reduces the accumulation of collagen in acetaldehyde-induced hepatic stellate cells. The accumulation and synthesis of collagen were measured by Marson-Trichrom stain and pulse-labeling with [^{3}H]-proline, respectively. As the results, CK inhibited collagen accumulation and incorporation of proline in a dose dependent manner. Furthermore, the effects of CK on expression of alpha-smooth muscle actin (a-SMA) and collagen type I were evaluated utilizing immunocytochemistry. CK reduced a-SMA and collagen type I expressions compared with acetaldehyde-induced hepatic stellate cells. These results suggested that the protective effects of CK on the hepatic fibrosis in stellate cells might, at least in part, be due to its ability to reduce the accumulation of collagen and blocked activation of hepatic stellate cells by acetaldehyde.

[PA4-7] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Effects of Mancozeb on cell-mediated immunity in mice.

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Mancozeb is a protective fungicide on plants and a polymeric complex of ethylene bisdithiocarbamate manganese with zinc salt. It is reported to induce teratogenic and carcinogenic effect in laboratory animals. But the immunomodulating effects of Mancozeb exposure have not been systemically evaluated. The purpose of this study was to investigate the effects of Mancozeb on cell-mediated immunity in mice. For ex vivo assessment, mice were orally exposed to Mancozeb dissolved in distilled water as concentrations of 2,500, 5,000, 10,000 mg/kg for single occasion (acute exposure) or 250, 1,000, 1,500 mg/kg/day 5 days a week for 30days(subacute). Splenocyte proliferation was significantly suppressed through T cell mitogen supplementation. IFN-γ production was decreased both acute and subacute exposure, IL-4 production was increased in subacute exposure. These results suggest that Mancozeb could alter cell-mediated immunity in mice.

[PA4-8] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

The Effect of 3-MCPD on Male Fertility and Sperm Parameters in Rats

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3-Monochloro-1,2-propanediol(3-MCPD) is a toxic compound, often present in different foods containing acid hydrolyzed(AH) protein, like seasonings and savory food products. In Korea, 3-MCPD is currently being a problem because of its toxicity in AH soybean sauce. The purpose of the present studies was to investigate the effects of 3-MCPD on male fertility, sperm and testosterone secretion. In vivo male fertility test was performed for observing the adverse effects of 3-MCPD on the function of male reproductive system and pregnancy outcome. 0.01, 0.05, 0.25, 1 and 5 mg/kg bw of 3-MCPD were given daily by gavage to groups of 15 adult male SD rats for 4 weeks. At the end of pre-treatment period, males were mated overnight with untreatment females. Following morning, males demonstrating successful induction of pregnancy were sacrificed on that day to assess sperm parameters and histopathology of reproductive organs. The resulting pregnant females were sacrificed on day 20 of gestation to evaluate pregnancy outcome. As a result, four-week paternal administration with 3-MCPD resulted in adverse effects on male fertility and pregnancy outcome without remarkable histopathological changes in testes and epididymides; sperm motility, copulation index and fertility index were markedly decreased in the treated group and numbers of live fetuses showed steep dose-response curves. Also, spermatogenesis was investigated in this experiment. However, no effect was observed on production of sperm in testes treated with 3-MCPD for 4 weeks. Hormone assay was performed for observing the effects of 3-MCPD on testosterone and luteinizing hormone(LH) in blood and tstes of male SD rats and cultured primary Leydig cell. In result, significant change of related hormones did not obsreved by treatment of 3-MCPD. These results indicated that paternal treatment with