

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

### **Acute cocaine poisoning in a body packer**

**Park Meejung**<sup>o</sup>, Choi Sangkil, Son Haengja, Lee Jaesin, Kim Eunmi, Lim Miae, Chung Heesun  
*National Institute of Scientific Investigation*

A 35-year-old Peruvian 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, cerebellum and lung were taken and analyzed. Biological specimen were extracted by liquid-phase extraction using CH<sub>2</sub>Cl<sub>2</sub> : 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, ecgoninemethylester and benzoylecgonine 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), cerebellum (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**

**Chung Yung Chul**<sup>o</sup>, Choi Chul Yung, Kim Ji Young, Chun Hyo Kon, Gho Young Hee, Jeong Hye Gwang  
*Division of Food Science, Jinju International University, Jinju, Korea, Department of Pharmacy and Research Center for Proteinaceous Materials, Chosun University, Kwangju, Korea, Korea Research Institute of Bioscience and Technology, Taejeon, South Korea*

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<sub>2</sub> 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-κB, 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**

**Lee Kyung Jin**<sup>o</sup>, Shin Dong Weon, Chung Young Chul, Kim Young Sup, Ryu Si Yung, Roh Sung Hwan, Jeong Hye Gwang  
*Department of Pharmacy and Research Center for Proteinaceous Materials, Chosun University, Kwangju, Korea, Division of Food Science, Chinju International University, Chinju, Korea., Korea Research Institute of Chemical Technology, Taejeon, Korea, Department of R&D, Jangsaeng Doraji Co., Ltd., Chinju, Korea*

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