LPS (10 ng/ml) or IFN-r (100 U). MRC and CRC in starch-loaded groups also increased NO production in the LPS activation. Changes of serum enzyme activities of glutamic oxaloacetic transaminase (GOT), glutamic pyruvic transaminase (GPT) were also investigated. Acarbose appeared to be greater (59, 78) than those of control (32, 51) when compared with GPT, GOT in both maltose-loaded groups. The level of liver glycogen after treatment MRC (108.05±12.05 dl/ml) or CRC (95.00±22.02 dl/ml) was not significant from that of control (115.83± dl/ml).

[PB4-11] [04/18/2003 (Fri) 09:30 ~ 12:30 / Hall P]

Protective effects of Ginsan against Cyclophosphamide-induced immunosuppression in mice

Shim Jiyoung°, Ahn Jiyeon, Han Youngsoo, Jung Insung, Yun Yeonsook, Song Jieyoung*

Laboratory of Immunology, Korea Institute of Radiological and Medical Sciences, KAERI, 215-4 Gongneung-dong, Nowon-Ku, Seoul, 139-706, Korea

The immunomodulator Ginsan has been found previously by us to stimulate the secretion of high levels of IL-1, IL-6 and TNF-alpha in irradiated mice. These cytokines are known to induce proliferation and differentiation of hematopoietic progenitor cells from the spleen and bone marrow and to protect mice from DNA-damaging agents. The present studies were evaluated as a cytoprotective agent against toxicity of the alkylating drugs. Sublethal dose (250 mg/kg) of cyclophosphamide (CP) caused neutropenia, decreased cellularity of bone marrow and inhibited Natural Killer (NK) cell activity in Balb/c mice. A single injection of Ginsan (2mg/mouse) at 24 h after CP treatment accelerated recovery of blood neutrophils and bone marrow cellularity and restored NK activity in CP-treated mice.
Moreover, ginsan protected these animals from the lethal effects of high doses of CP. These protective effects were demonstrable only when ginsan was administered to mice 24 h after CP treatment. To assess the big difference in survival rate between pre- or post administration of Ginsan, we analyzed the cell cycle progression of spleen and bone marrow cells in CP-treated mice. CP, given alone, affected apoptotic cell death and caused deregulation of the cell cycle in the spleen and bone marrow. Ginsan, applied 24h after CP administration, resulted in a suppressing effect on apoptosis and rapid recovery from the cell cycle perturbation triggered in normal bone marrow cells by the alkylating drug. The result may be useful for therapeutic application of ginsan with CP therapy.

[PB4-12] [04/18/2003 (Fri) 09:30 ~ 12:30 / Hall P]

Platycodon grandiflorum enhanced macrophages function and NK and LAK cell mediated cell lysis.

Choi ChulYung°1, Kim Ji young2, Jeong HyeGwang2

1Division of Food Science, Jinju International University, Jinju, Korea. 2Department of Pharmacy and Research Center for Proteineous Materials, Chosun University, Kwangju, Korea.

The immunostimulatory and host resistance effects of the Platycodon grandiflorum A. DC, changkil (CK) and inulin (CKI) isolated from CK were investigated in rats. SD rat were exposed to CK or CKI by gavages for 7days and isolated peritoneal macrophages and splenocyte were used for these studies. CK and CKI significantly enhanced peritoneal macrophages activities such as ROS production and phargocytosis. Lymphokine-activated killer cell (LAK) and natural killer (NK) cell activation in splenocyte were measured by MTT assay and these activities were significantly

enhanced by administration of CK and CKI.

[PB4-13] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Anti-Inflammatory Effect on Rat Microglia by UDCA

Joo SeongSoo, Oh WonSik, Lee Dolk°

Department of Immunology, College of Pharmacy, Chung-Ang University, Seoul 156-756, Korea

Ursodeoxycholic acid (UDCA) has been known commonly improving hyperbilirubinemia and excretion abnormality of bromsulphalein, which are appeared in the liver, and reducing the release of cholesterol from bile duct. In our study, UDCA was aimed to know if the agent can inhibit the pathogenesis of AD by suppressing the microglial activation when stimulated particularly by Aβ peptide, which is known a major cause of AD. For the study, we selected proinflammatory cytokines such as TNF-α and NO released from the brain microglia. In the study, 1day Sprague Dawley-rat(SD-rat) was used for the culture of microglia. Microglia taken from the isolated period were co-incubated with LPS or beta-amyloid to activate microglia at various concentrations of UDCA during scheduled times. From the experiment, we obtained very interesting results that UDCA plays a role in suppressing the inflammatory parameters of microglia. In conclusion, a new research area for AD by UDCA is worthy of studying by suppressing microglia at early stage of plaque formation in brain, and can be a basic knowledge in a future clinical approach between UDCA and AD.

[PB4-14] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Involvement of Phosphodiesterase Isozymes in Osteoclast Formation

Yim Mijung°

College of Pharmacy, Sookmyung Women's University

cAMP acts as a second messenger in the functional responses of various cells to hormones, cytokines and other agents. In turn, this nucleotide also modulates the signal transduction processes regulated by a range of cytokines and growth factors.
The intracellular level of cAMP is regulated by a G protein coupled adenylyl cyclase and degradation is mediated by the phosphodiesterases (PDEs), a superfamily of enzymes that catalyze the hydrolysis of cAMP.
In osteoblast, activation of adenylyl cyclase by parathyroid hormones (PTH) or prostaglandins (PGs) has been found to increase osteoclast formation via expression of TRANCE, a key molecule for osteoclast differentiation and activation. However, whether PDEs in osteoblast regulate osteoclastogenesis is unknown.
In this study, RT-PCR analysis of mouse primary osteoblast revealed the presence of PDE1, PDE2, PDE3, PDE4, PDE5, PDE7, PDE8, and PDE9. Furthermore, the general PDE isozyme inhibitor enhanced osteoclastogenesis in a dose dependent manner in co-culture system. These results indicate that PDE isozymes in osteoblast are involved in the modulation of osteoclast differentiation.

[PB4-15] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Production and characterization of a PPARgamma-specific monoclonal antibody Pγ 48.34A