

## Regulatory Effect of Atopic Allergic Reaction by *Carpopeltis affinis*

Na HoJeong<sup>o</sup>, Moon PhilDong, Hong SeungHeon, Seo YoungWan, Kim HyungMin

Department of Oriental Pharmacy, Wonkwang University, Division of Ocean Science, Korea Maritime University, Department of Pharmacology, College of Oriental Medicine, Kyung Hee University

We studied the effect of methanol extract of *Carpopeltis affinis* (CA) on atopic allergic reaction. CA dose-dependently inhibited interleukin (IL)-8 and tumor necrosis factor (TNF)- $\alpha$  secretion from the PMA- plus A23187- stimulated HMC-1. CA also dose-dependently inhibited the histamine and  $\beta$ -hexosaminidase release from mast cells. CA had no cytotoxic effect. These results suggest that CA has the inhibitory effect of atopic allergic reaction and this might be useful for clinical application to treat several allergic diseases such as atopic dermatitis.

[PA1-38] [ 04/17/2003 (Thr) 14:00 - 17:00 / Hall P ]

### Quercetin 3-O- $\alpha$ -arabinofuranoside protects heart-derived H9c2 cells against oxidative injury through maintaining MMP

Kim Mi-Young<sup>o</sup>, Jung Yi-Sook<sup>1</sup>, Kim Young Ho<sup>2</sup>, Baik Eun Joo<sup>1</sup>, Lee Soo Hwan<sup>1</sup>, Moon Chang-Hyun<sup>1</sup>

<sup>1</sup>Dept. of Physiology, School of Medicine, Ajou Univ., Suwon, #442-749, South Korea, <sup>2</sup>Coll. of Pharmacy, Chung Nam natl. Univ., Daejeon, #305-764, South Korea

In this study, we investigated whether the cardioprotective effect shown by quercetin 3-O- $\alpha$ -arabinofuranoside extracted from *Lindera erythrocarpa* against ROS-induced cell death in H9c2 cardiac myocytes. Cell death was induced by BSO, buthionine sulfoximine, which inhibits GSH level and subsequently increase ROS level. Cell death was quantitatively determined by measuring lactate dehydrogenase (LDH) activity. BSO-induced ROS level and mitochondrial membrane potential (MMP) were measured using 2,7-dichlorofluorescein diacetate oxidation and rhodamine 123.

In H9c2 cells exposed to BSO 10 mM for 24h, LDH release was remarkably increased by 73% compared to that in control (18.7%). From 1  $\mu$ M to 10  $\mu$ M of quercetin 3-O- $\alpha$ -arabinofuranoside reduced LDH release and ROS level induced by BSO, in a dose-dependent manner. Cells exposed to BSO showed an early loss of MMP. This decrease in MMP was significantly reversed by treatment with 10  $\mu$ M quercetin 3-O- $\alpha$ -arabinofuranoside. In conclusion, our results suggest that quercetin 3-O- $\alpha$ -arabinofuranoside can produce cardioprotective effect against ROS-induced cell death through antioxidant effect. This study was supported by a grant of Ministry of Health & Welfare, Republic of Korea. (00-PJ2-PG1-CD02-0018)

[PA1-39] [ 04/17/2003 (Thr) 14:00 - 17:00 / Hall P ]

### The Joins (SKI 306X) study : Effects on Arachidonic acid metabolism pathway and other inflammatory mediators

Ryu KeunHo, Jung Kiwon, Han ChangKyun, Kwak WieJong, Cho YongBaik

Life Science Research Center, SK Chemicals

Joins (SKI 306X) is now clinically used for the treatment of osteoarthritis (OA). In previous

reports. Joins a natural herbal product extracted from three herbs *Clematis Radix*, *Trichosanthes Radix* and *Prunella Flos*, was shown to have good analgesic and anti-inflammatory effects in several *in vivo* models, e.g., acetic acid-induced pain, carrageenan-induced paw edema and adjuvant-induced arthritis. And Joins has the cartilage protective effects on rabbit articular cartilage explants culture, *in vitro* OA model, and collagenase-induced experimental OA model. In this study, Joins and its components were further examined to investigate the mechanism. To study the mode of action of Joins effects on arachidonic acid metabolism pathway and inflammatory mediators (NO, TNF- $\alpha$ ) were investigated. In arachidonic acid metabolism pathway, the effects on cyclooxygenases (COX) and lipoxygenase (LO) were examined. The generation of prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) and leukotriene B<sub>4</sub> (LTB<sub>4</sub>) in cultured murine macrophage cell line RAW 264.7 medium were determined by their ELISA assay kit. COX-2 protein in cultured cell was determined by western blot analysis. Nitrite accumulation, an indicator of NO synthesis (NOS), in the culture medium and TNF- $\alpha$  in heparinized human whole blood, very powerful inflammatory mediators, were measured. In arachidonic metabolism, Joins showed the inhibitory effects of lipopolysaccharide (LPS)-induced PGE<sub>2</sub> production and COX-2 gene expression, but no effects on COX-1 and COX-2 activity. Joins also inhibited the A23187-induced LTB<sub>4</sub> production concentration-dependently. In addition, Joins inhibited LPS and INF- $\gamma$ -induced NO production. Joins inhibited LPS-induced TNF- $\alpha$  expression. Above results indicate that Joins interfere the arachidonic acid metabolism pathway by inhibiting both 5-LO activity and COX-2 expression. Two important inflammatory mediators, NO, TNF- $\alpha$  were also suppressed by Joins. The results represents that Joins can modulate arthritis by interfering important mediators related with arthritis such as arachidonic acid metabolites, NO and TNF- $\alpha$ .

[PA1-40] [ 04/17/2003 (Thr) 14:00 - 17:00 / Hall P ]

### Development of hangover settlement materials from natural products

Kwon so yeon<sup>o</sup>, Kim shung hee, Kwon hyun jung, Lee chang hwan, Sim kyu jung, Jung se young

Department of hygienic chemistry college of Pharmacy Kyunghee university

Hangover is associated with ethanol metabolism in body after the ingestion of an alcoholic beverage. Especially, The metabolism in liver is focused by many researcher because, alcohol (approximately 90%) is metabolized by the liver. Ethanol metabolism in liver involves both liver alcohol dehydrogenase (ADH) which catalyzes the oxidation of ethanol to acetaldehyde, and liver aldehyde dehydrogenase (ALDH) which metabolized rapidly acetaldehyde, product of ethanol oxidation, to acetate. It has been known that hangover is caused by increasing acetaldehyde concentration in blood after intaking alcoholic beverage. Thus, it is valuable that researchers will effort to study substrates which decrease rapidly this generation. So, Fifteen samples ( Inositol, RICEO, RI-AX Sweet chest nut rose, Kum Quat, oyster, Kohki, Gurume-K, Gurume-J, Gurume-P, Carambora Lotus seed, lakanka F, Phytic acid and Chlorophyll) which had been determined *in vitro* by researcher in Yonsei University were tested *in vivo*. The samples were administrated through oral route thirty minutes before alcohol administration. The sample and alcohol dose were 200mg/Kg and 3g/Kg, respectively. Then, blood collected at 1, 3 and 5 hour after alcohol administration. Ethanol and acetaldehyde concentration in blood were measured by using commercially available measurement kit (so called F-kit). Values of these two concentrations were compared with one of control and effect of hangover settlement is evaluated by degree of decreasing ethanol and acetaldehyde concentration in blood.

from the results, the samples could be categorized into three according to their biological effect on the enzymes. Samples in the first category make ethanol and acetaldehyde concentration in blood decreased, which is explained that both ADH and ALDH activity are enhanced. The samples are sweet chest nut rose, Kohki, Kum Quat, oyster, Gurume-K, Gurume-J, and Carambora.

And second part's ones make only acetaldehyde concentration in blood decreased, which is