Regulatory Effect of Atopic Allergic Reaction by Carpopeltis affinis

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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)- α secretion from the PMA- plus A23187- stimulated HMC-1. CA also dose-dependently inhibited the histamine and β -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-α-arabinofuranoside protects heart-derived H9c2 cells against oxidative injury through maintaining MMP

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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 quentitatively determined by measuring lactate dehydrogenase (LDH) activity. BSO-induced ROS level and mitochondrial membrade 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 μ M to 10 μ M of quercetin 3-O- α -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 μ M quercetin 3-O- α -arabinofuranoside. In conclusion, our results suggest that quercetin 3-O- α -arabinofuranosied 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)

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

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