

apoptotic processes. The purpose of the present study is to elicit the molecular mechanism of tetrandrine induced apoptosis in HepG2 cells. Treatment of the cells with tetrandrine resulted in the activation of caspase-3 protease and the cleavage of PARP. The activation of caspase-8 was also observed at a concentration of 16 μ M. Tetrandrine didn't affect the release of cytochrome c from mitochondria into cytosol in a time and dose dependent manner. The treatment of HepG2 cells with tetrandrine did not influence the mitochondrial transmembrane potential (MPT) as has been determined by JC-1 staining. Caspase-9, a downstream caspase of cytochrome c release, was not activated by the treatment with tetrandrine. These results suggest that apoptosis of HepG2 cells induced by tetrandrine proceeds via cytochrome c independent and caspase-8 as well as caspase-3 dependent pathway.

[PA3-7] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Protection against hydrogen peroxide-induced oxidative DNA damage by galangin

Yang He Eun^o, Lee Seung Chul, Heo Moon Young

College of Pharmacy, Kangwon National University, 200-701, Korea

The effect of *Alpinia officinarum* extract and its major flavonoid, galangin (>10% in the 70% ethanol extract) on the oxidative DNA damages *in vitro* were evaluated to develop new chemopreventive agent against oxidative stress. In order to investigate the modulating effects of these compounds, 8-OH 2'dG and comet assay have been performed in CHL cell. Hydrogen peroxide (H_2O_2) and iron (II) induced 8-OH 2'dG formation and DNA damages in CHL cells were decreased by catalase, SOD and model antioxidants (Vit-E, Vit-C, BHT), including *Alpinia officinarum* extract and galangin. Galangin showed the most potent modulating effects on the oxidative DNA damages *in vitro*. These results suggest that *Alpinia officinarum* extract and its major flavonoid, galangin may be useful chemopreventive agents for protecting cellular oxidative DNA damage by oxidative stress.

[PA3-8] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Cardioprotective effect of *Lidera erythrocarpa* against oxidative stress-induced cell death

Kim Mi-Young^o, Jung Yi-Sook, Kim Young Ho¹, Lee Soo Hwan, Baik Eun Joo, Moon Chang-Hyun

Dept. of Physiology, School of Medicine, Ajou Univ, Suwon, #442-749, Korea

Recent evidence has suggested an intimate link between myocardial failure and an excessive generation of reactive oxygen species (ROS), such as O_2^- , $\cdot OH$, H_2O_2 . We investigated the effect of several extracts of natural products, *Hovenia dulcis*, *Koeleruteria paniculata*, *Sorbus comixta*, *Pedicularis resupinata*, *Lidera erythrocarpa*, *Sanguisorba officinalis*, *Boehmeria berchemiae*, *Euscaphis japonica*, against ROS-induced cell death. Previously it has been demonstrated that those extracts have anti-oxidant effect as shown by DPPH assay. Cell death was induced by using BSO, buthionine sulfoximine, which inhibit GSH level and subsequently increase ROS level. Cell death was quantitatively determined by measuring lactate dehydrogenase (LDH) activity, propidium iodide (PI)-uptake and morphology. Among those extracts, *Lidera erythrocarpa* has shown the most potent protective effect against BSO-induced cardiac cell death. From 0.3 μ g/ml to 10 μ g/ml of *Lidera erythrocarpa* reduced LDH release and PI-uptake by induced BSO, in a dose-dependent manner. We also observed cardioprotective effect of *Lidera erythrocarpa* morphologically by using microscope. In conclusion, our results suggest that *Lidera erythrocarpa* can produce cardioprotective effect against ROS-induced cell death.