acid treated hepatic microsomes, as determined by immunoblot analysis in a manner consistent with that of the enzyme activity levels. These results suggest that 18beta-glycyrrhetinic acid may act as a more specific suppressor for P4502E1 than P4501A1 and P4502B1/2 [This work was supported by Korea Research Foundation Grant (KRF-2000-041-F00314)].

[PA4-15] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

The Inhibitory Effects of Houttuynia cordata Extracts against Cadmium induced Cytotoxicity (VII)

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This study was carried out to evaluate the cytotoxicity of cadmium on NIH 3T3 fibroblasts and to develop the antidote on NIH 3T3 fibroblasts which was damaged by Cd50 of cadmium. The antitoxic activity of Houttuynia cordata extract in NIH 3T3 fibroblasts was evaluated by MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazoliumbromide) and SRB (sulforhodamine B protein) assays. The light microscopic study was carried out to observe morphological changes of the treated cells. These results were obtained as follows; The concentration of 10-2 mg/ml of Houttuynia cordata extract was shows significant antitoxic activity. The number of NIH 3T3 fibroblasts were antitoxic and tend to regenerate. These results suggest that the chloroform extract of Houttuynia cordata retains a potential antitoxic activity.

[PA4-16] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

Activation of cPLA2 Leading to Increase in Glutathione and Downregulation of iNOS May Make LLCpk1 Cells Resist to TNF-a-induced Cell Death

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Group IV cytosolic PLA2 (cPLA2) is known to be involved in hydrogen peroxide-induced cytotoxicity in LLC-pk1 kidney epithelial cells. However, the precise mechanism by which cPLA2 is implicated in TNF-a-induced cell death is not fully elucidated. Here we found that cPLA2-overexpressed LLC-pk1 cells, but not vector-cells, is resistant to TNF-a-induced cell death. Treatment of TNF-a to vector-LLCpk1 cells, but not cPLA2-overexpressed LLC-pk1 cells, provoked a DNA fragmentation and change in nuclear morphology as detected by 4,6-diamidino-2-phenylindole staining. There was a significant increase in the level of glutathione in the cPLA2-overexpressed LLC-pk1 cells. Treatment of TNF-a for 24 h up-regulated the inducible nitric oxide synthase (iNOS) in vector-LLCpk1 cells, but not cPLA2-overexpressed LLC-pk1 cells. In contrast, arachidonic acid (AA), the product of cPLA2, induced more cell death in cPLA2-overexpressed cells than in vector-LLCpk1 cells and the enhancement in cell death in cPLA2-overexpressed cells was not blocked by any inhibitor of cyclooxygenase and lipoxygenase. Our results suggest that the sustained release of AA by action of cPLA2 may make LLCpk1 cells resist to TNF-a-induced cell death through antioxidant defense system and iNOS-related pathway.

[PA4-17] [04/20/2001 (Fri) 10:30 - 11:30 / Hall 4]

cDNA Microarray Analysis of the Gene Expression after Ad5CMV-p16INK4a Gene Transfer in the Non-Small Cell Lung Cancer Cells