eradicate pests and insects in Korea, providing potential for human exposure. It, however, could adversely affect the reproductive endocrine system if it has hormonal activity. Little is known about hormonal activity of bioallethrin throughout the world. This study investigated the potential estrogenic (antiestrogenic) activity of bioallethrin by immature rat uterotrophic assay and Calbindin–D9k (CaBP–9k) gene expression assay. In the uterotrophic assay using 18–day old SD rats, subcutaneous treatment of bioallethrin (5 to 800 mg/kg/day) for 3 days had no significant effects on uterine wet weights, compared with vehicle control group, but led to statistically—significant enhancements in E2–increased their weights at all doses tested. In addition, this effect was statistically significant at certain doses. This chemical also enhanced E2–induced CaBP–9k mRNA expression in Northern blot analysis, although not statistically—significant. In conclusion, our results suggest that bioallethrin might mimic estrogen. However, since its estrogenic (antiestrogenic) activity was not clearly elucidated in this experiment, more–detailed further studies with various screening test methods are needed.

[PA4-13] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Hepatoprotective Effects of the Acteoside on Carbon tetrachloride ?Induced Liver Damage in Mice

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The protective effects of acteoside, a phenylethanoid glycoside, on carbon tetrachlorideinduced hepatotoxicity and the possible mechanisms involved in this protection were investigated in mice. Pretreatment with acteoside prior to the administration of carbon tetrachloride significantly prevented the increased serum enzymatic activities of alanine and aspartate aminotransferase in a dose-dependent manner. In addition, pretreatment with acteoside also significantly prevented the elevation of hepatic malondialdehyde formation and the depletion of reduced glutathione content in the liver of carbon tetrachloride?intoxicated mice. However, hepatic reduced glutathione levels and glutathione-S-transferase activities were not affected by treatment with acteoside alone. Carbon tetrachloride?induced hepatotoxicity was also essentially prevented, as indicated by a liver histopathologic study. The effects of acteoside on the cytochrome P450 (P450) 2E1, the major isozyme involved in carbon tetrachloride bioactivation were also investigated. Treatment of mice with acteoside resulted in a significant decrease of P450 2E1-dependent p-nitrophenol and aniline hydroxylation in a dose-dependent manner. Consistent with these observations, the P450 2E1 expressions were also decreased, as determined by immunoblot analysis. Acteoside showed anti?oxidant effects in FeCl2?ascorbate induced lipid peroxidation in mice liver homogenate and in superoxide radical scavenging activity. These results suggest that the protective effects of acteoside against carbon tetrachloride-induced hepatotoxicity possibly involve mechanisms related to its ability to block P450?mediated carbon tetrachloride bioactivation and free radical scavenging effects

[PA4-14] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Platycodi Radix inhibits endothelial cell invasion and angiogenesis

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Herbal medicines are increasingly being utilized to treat a wide variety of disease processes. In

this study, we assayed the preventive and therapeutic effects of aqueous extract from the roots of Platycodon grandilflorum A. DC. Changil (CK) in human microvessel endothelial cell-1 (HMEC-1) angiogenesis. CK inhibited cell migration and in the presence of CK proliferation of HMEC-1 was inhibited in a dose-dependent manner. CK also inhibited the tube formation in a dose-dependent manner. In these assays, 40% inhibition was showed in high does of CK (800 ug/ml). These results demonstrated that prevention of angiogenesis by CK was mediated by inhibition of proliferation and migration of endothelial cells.

[PA4-15] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Hepatoprotective Effects of the Acteoside on Carbon tetrachloride ?Induced Liver Damage in Mice

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The protective effects of acteoside, a phenylethanoid glycoside, on carbon tetrachlorideinduced hepatotoxicity and the possible mechanisms involved in this protection were investigated in mice. Pretreatment with acteoside prior to the administration of carbon tetrachloride significantly prevented the increased serum enzymatic activities of alanine and aspartate aminotransferase in a dose-dependent manner. In addition, pretreatment with acteoside also significantly prevented the elevation of hepatic malondialdehyde formation and the depletion of reduced glutathione content in the liver of carbon tetrachloride?intoxicated mice. However, hepatic reduced glutathione levels and glutathione-S-transferase activities were not affected by treatment with acteoside alone. Carbon tetrachloride?induced hepatotoxicity was also essentially prevented, as indicated by a liver histopathologic study. The effects of acteoside on the cytochrome P450 (P450) 2E1, the major isozyme involved in carbon tetrachloride bioactivation were also investigated. Treatment of mice with acteoside resulted in a significant decrease of P450 2E1-dependent p-nitrophenol and aniline hydroxylation in a dose-dependent manner. Consistent with these observations, the P450 2E1 expressions were also decreased, as determined by immunoblot analysis. Acteoside showed anti-oxidant effects in FeCl2?ascorbate induced lipid peroxidation in mice liver homogenate and in superoxide radical scavenging activity. These results suggest that the protective effects of acteoside against carbon tetrachloride-induced hepatotoxicity possibly involve mechanisms related to its ability to block P450-mediated carbon tetrachloride bioactivation and free radical scavenging effects.

[PA4-16] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Effects of Platycodi Radix on Hepatic Fibrosis in Rats

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Herbal medicines are increasingly being utilized to treat a wide variety of disease processes. We previously reported that aqueous extract from the roots of Platycodon grandiflorum A. DC (Campanulaceae), Changkil (CK), had hepatoprotective effects against acetaminophen induced