

obtained active compound (referred to VD-2 and VD-6) from the dichloromethane extract. IC50 of VD-2 and VD-6 was shown to be 75 μ M and 18.8 μ M, respectively. Also, they activated caspase-3 enzyme of NIH-3T3 cells during apoptosis. These results indicated that Korean mistletoe has highly cytotoxic compound, VD-2 and VD-6, against tumor cells, and the compounds were velutin and betulinic acid.

[PC1-16] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Sauchinone, a Lignan from *Saururus chinensis*, Inhibits Staurosporine-induced Apoptosis in C6 Rat Glioma Cells

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Neuronal apoptosis may contribute to the pathological neuronal loss in certain disease states such as neurodegenerative diseases. Staurosporine (ST), a nonselective protein kinase inhibitor, has been shown to induce apoptosis in a variety of cells including nerve cell lines. In this study, we investigated the neuroprotective effect of sauchinone, which is a unique lignan from *Sauchinone Chinensis*, on ST-induced apoptosis in C6 rat glioma cells. Here, we show that sauchinone attenuated ST-induced apoptosis of C6 glioma cells as evidenced by DNA fragmentation. We also provide evidence that the protective effect of sauchinone on ST-induced apoptosis involves a dose-dependent upregulation of an anti-apoptotic protein, Bcl-2. Mounting evidence reveals that the activation of caspases, especially caspase-3, triggers the apoptotic process. To examine the involvement of caspase-3 in the protective effect of sauchinone, we measured the activity of caspase-3 of ST-treated C6 cells upon sauchinone treatment. We show that sauchinone decreased the activity of caspase-3, suggesting that sauchinone protected C6 glioma cells from apoptosis by ST in a caspase-3-dependent manner.

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Anticoagulant Activity of Acharan Sulfate In Vivo

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We previously reported that acharan sulfate from the African giant snail *Achatina fulica* showed the anticoagulant activity in vitro, but it was much less than that of heparin. In present study, the anticoagulant activity of acharan sulfate was investigated in vivo. Intravenous administration of acharan sulfate prolonged the clotting time (APTT) in mice and rats in a dose-dependent manner. Although the activity was low in rats, it could be maintained over 5 h after administration of AS (30mg/kg). In contrast, the activity of heparin (5mg/kg) was restored to the normal level at 3 h. In a thrombin-induced lethality model in mice AS (20 mg/kg) protected the lethality by 80 percent, while heparin (20 mg/kg) did not show any protective activity after 3.5 h of administration of drugs. We could also determine the plasma concentration of AS even 5 hours after administration to rats. These results show that the longer duration of AS in blood has a possibility to interact with coagulation factors. Further study is required for the study of anticoagulant and antithrombotic mechanism of AS in vivo.