

activity using washed rabbit platelets *in vitro*. The biologically active constituent of *T. dolabrata* sawdust was isolated by silica gel column chromatography and HPLC and characterized as carvacrol by various spectral analyses (^1H -, ^{13}C -NMR and GC/MS studies). The inhibition values of carvacrol at the concentration of 1.0 $\mu\text{g}/\text{mL}$ for collagen, arachidonic acid, or PAF-induced platelet aggregations were 21, 92, and 2%, respectively. However, carvacrol at the same concentration no affected thrombin (0.1 unit/mL)-, calcium ionophore A23187 (2 μM)-, or PMA (20 μM)-induced platelet aggregation. These results suggest that carvacrol isolated from *T. dolabrata* sawdust may be useful as a lead compound and new agents for inhibiting platelet aggregation induced by arachidonic acid.

[PA1-27] [04/17/2003 (Thr) 14:00 – 17:00 / Hall P]

β -EUDESMOL CAUSES VASODILATORY EFFECT IN THE NORMOTENSIVE RAT

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β -Eudesmol is one of various compounds derived from the bark of *Magnolia obovata* Thunberg, a medicinal plant. It has been shown that β -eudesmol also markedly alleviated muscle fasciculation, tremor and convulsion induced by diisopropylfluorophosphate and prolonged the time to death in mice (Chiou et al., 1995). Actually, the extract of magnolia bark has been shown to have depressant actions on the central nervous system (Watanabe et al., 1973). Recently, it has been reported that the crude extract of magnolia bark, an herbal drug, inhibited the secretion of catecholamines from bovine adrenal chromaffin cells stimulated by acetylcholine in a concentration-dependent manner (Tachikawa et al., 2000). Therefore, the present study was conducted to investigate the effects of β -eudesmol on arterial blood pressure and vascular contractile responses in the normotensive rats and to establish the mechanism of action. Phenylephrine (an adrenergic α_1 -receptor agonist) and high potassium (a membrane-depolarizing agent) caused greatly contractile responses in the isolated aortic strips, respectively. These phenylephrine (10-5 M)-induced contractile responses were depressed in the presence of high concentrations of bornyl acetate (10 ~ 20 $\mu\text{g}/\text{ml}$), but not affected in low concentration of bornyl acetate (2.5 ~ 5 $\mu\text{g}/\text{ml}$). Also, high potassium (5.6 x 10⁻² M)-induced contractile responses were greatly inhibited in the presence of β -eudesmol (2.5 ~ 20 $\mu\text{g}/\text{ml}$) in a dose-dependent fashion. β -eudesmol (1 ~ 10 mg/kg) given into a femoral vein of the normotensive rat produced a dose-dependent depressor response, which is transient (data not shown). Interestingly, the infusion of a moderate dose of β -eudesmol (3 mg/kg/30 min) made a significant reduction in pressor responses induced by intravenous norepinephrine. Collectively, these results obtained from the present study demonstrate that intravenous β -eudesmol causes a dose-dependent depressor action in the anesthetized rat at least partly through the blockade of adrenergic α_1 -receptors. β -Eudesmol also causes vascular relaxation in the isolated aortic strips of the rat via the blockade of adrenergic α_1 -receptors, in addition to the unknown direct mechanism.

[PA1-28] [04/17/2003 (Thr) 14:00 – 17:00 / Hall P]

Enantio-Selective Inhibition of (1R,9S)- and (1S,9R)- β -Hydrastines on Dopamine Biosynthesis in PC12 Cells

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The inhibitory effects of (1R,9S)- and (1S,9R)-enantiomers of β -hydrastine (BHS) on dopamine biosynthesis in PC12 cells were investigated. (1R,9S)-BHS decreased the intracellular dopamine content with the IC_{50} value of 14.3 μ M at 24 h, but (1S,9R)-BHS did not. In these conditions, (1R,9S)-BHS inhibited TH activity mainly in a concentration-dependent manner (33% inhibition at 20 μ M) and decreased TH mRNA level. (1R,9S)-BHS at 10–50 μ M also reduced the intracellular cyclic AMP level and Ca^{2+} concentration. In addition, treatment of L-DOPA at 20–50 μ M for 24 h increased the intracellular dopamine content to 198–251% compared with the control value in PC12 cells, however, the increase in dopamine levels induced by L-DOPA was significantly reduced when L-DOPA (20–50 μ M) was associated with (1R,9S)-BHS (10–50 μ M). These results indicate that (1R,9S)-BHS, but not (1S,9R)-BHS, inhibited dopamine biosynthesis and L-DOPA-induced increase in dopamine content, in part, through the inhibition of TH activity and TH gene expression in PC12 cells: thus, (1R,9S)-BHS proved to have a function to regulate dopamine biosynthesis.

[PA1-29] [04/17/2003 (Thr) 14:00 – 17:00 / Hall P]

Inhibitory Effects on Dopamine Biosynthesis and Protective Effect on L-DOPA-induced Neurotoxicity of liriodenine in PC12 cells

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The effects of liriodenine, an aporphine isoquinoline alkaloid, on dopamine biosynthesis and L-DOPA-induced neurotoxicity in PC12 cells were investigated. Treatment of PC12 cells with liriodenine at 10 μ M showed 33.6% inhibition of dopamine content decreased at 3 h and reached a minimal level at 12 h after the exposure to liriodenine at 10 μ M. Under these conditions, tyrosine hydroxylase (TH) and aromatic amino acid decarboxylase (AADC) activities were also reduced inhibited at 10 μ M of liriodenine by 10.1% and 20.2% relative to control, respectively. However, TH mRNA level was not altered by liriodenine treatment. Intracellular level of cAMP and $[Ca^{2+}]_i$ were also decreased by liriodenine at 10 μ M. Liriodenine induced a time- and concentration- dependent decrease in cell viability. Liriodenine at non-cytotoxic concentration (10 μ M) significantly inhibited L-DOPA-induced the decrease in cell viability. These results suggest that liriodenine contributes partially to the decrease in dopamine biosynthesis and L-DOPA-induced increase in dopamine content by inhibition of TH and AADC activities in PC12 cells, which may be due to inhibition of cAMP production and $[Ca^{2+}]_i$. In addition, liriodenine may attenuate decrease the L-DOPA-induced death of PC12 cells.

[PA1-30] [04/17/2003 (Thr) 14:00 – 17:00 / Hall P]

Antimicrobial and Antioxidative Activities of Cornis fructus Extracts

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