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 (^{1}H-, ^{13}C-NMR and GC/MS studies). The inhibition values of carvacrol at the concentration of 1.0 μg/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 normotensve rats and to establish the mechanism of action. Phenylephrine (an adrenergiα1-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 μg/ml), but not affected in low concentration of bornyl acetate (2.5 ~ 5 μg/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 μg/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 α1-receptors. β-Eudesmol also causes vascular relaxation in the isolated aortic strips of the rat via the blockade of adrenergic α1-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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