〔Prostaglandins, **38**, 439 (1989)〕

The role of thromboxane A_2 (TxA_2) in liver injury in mice.

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The role of thromboxane A_2 in CCl_4-induced liver disease was investigated in mice. Significant elevation of TxB_2 in the liver was observed 6 hours after the injection of CCl_4. Administration of OKY-046 (10 and 50 mg/kg) and ONO-3708 (0.5, 1 and 2 mg/kg) suppressed the elevation of serum GOT and GPT levels and histological changes of the liver. In addition, OKY-046 inhibited the elevation of TxB_2 in the liver. When U-46619, a stable TxA_2 mimetic was injected i.v. into the mice, clear elevation of serum GOT and GPT levels and histopathological score of the liver were observed. These results suggest that TxA_2 play a role for the onset of CCl_4-induce liver injury in mice.

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Effect of OKY-046 and ONO-3708 on liver injury in mice.

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The effect of OKY-046, a selective thromboxane A_2 (TXA_2) synthetase inhibitor, and ONO-3708, a novel TXA_2 receptor antagonist, on liver disease were investigated in mice. The liver injury was induced by either an injection of anti-basic liver protein antibody (a-BLP) into DBA/2 mice immunized previously with rabbit IgG or by an injection of bacterial lipopolysaccharide (LPS) into *Corynebacterium parvum* pretreated DDY mice. Administration of OKY-046 and ONO-3708 suppessed the elevation of serum GOT and GPT levels and histopathological changes in both of the models. Indomethacin inhibited the injury caused by a-BLP but not by LPS. Prostaglandin I_2 inhibited the a-BLP induced injury and showed the tendency to inhibit the LPS-induced injury.

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Inhibitory effect of β-adrenergic stimulants on increased vascular permeability caused by passive cutaneous anaphylaxis, allergic mediators, and mediator releasers in rats.

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The effect of isoproterenol, salbutamol, theophylline, and forskolin on IgE antibody-mediated homologons passive cutaneous anapylaxis (PCA) and on skin reactions caused hy allergic mediators and mediator releasers were investigated in rats. The results obtained indicate that β-adrenergic stimulants inhibit the increased vascular permeability caused by allergic mediators, and suggest that this activity of β-adrenergic stimulants might play an important role in their antiallergic actions. Inhibition of increased vascular permeabillty might be mediated via β-receptors and may be related to the increased in intracellular cyclic AMP levels.