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Effect of CV-3988, a specific antagonist against platelet activating factor, on homologous passive cutaneous anaphylaxis in the mouse ear.

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PAF caused a potent increase in vascular permeability in the mouse ear. The potency was slightly lower than that of serotonin (5HT) but higher than those of histamine (Hi), LTC₄, LTD₄, PGE₁ and PGE₂ on a weight basis. The increased vascular permeability caused by PAF was inhibited by CV-3988 in a dose-dependent manner. CV-3988 did not affect the increase in vascular permeability caused by Hi or 5HT. IgG₁ antibody-mediated PCA in the mouse ear was inhibited by CV-3988, although it did not affect IgE antibody-mediated PCA. These results suggest a possibility that PAF might be involved in IgG₁ antibody-mediated PCA in the mouse.

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The effects of thromboxane A₂ inhibitors (OKY-046 and ONO-3708) and leukotriene inhibitors (AA-861 and LY-171883) on CCl₄-induced chronic liver injury in mice.

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The effects of OKY-046, a selective thromboxane A₂ (TxA₂) synthetase inhibitor, ONO-3708, a novel TxA₂ receptor antagonist, AA-861, a selective 5-lipoxygenase inhibitor and LY-17883, a peptide leukotrienes (p-LTs) receptor antagonist on the chronic liver injury were investigated in mice. The chronic liver injury was induced by the injection of CCl₄ twice a week for 12 weeks in mice. In chronic liver injury models, administration of OKY-046, ONO-3708, AA-861 and LY-17883 for 12 weeks suppressed the histopathological changes in the liver and the extensive elevation of serum GOT and GPT levels. These results suggest that TxA₂ and LTs inhibitors are effective for the onset and development of chronic liver injury in mice.

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Morphological studies on nephrotoxic serum nephritis accelerated with rabbit IgG in mice.

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Mice were injected with subnephrotoxic dose of nephrotoxic serum (NTS) after preimmunization with rabbit IgG. In order to characterize the induced glomerulonephritis, light, electron and immunofluorescence microscopic studies were carried out 15 days after NTS injection, the time when increases in urinary protein and serum cholesterol and a decrease in serum albumin were apparent. The results obtained indicate that this nephritic model shares a common pathology with human membranoproliferative glomerulonephritis type 1 and crescentic glomerulonephritis and can be considered an appropriate model for producing severe nephritis for short periods.