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[Lab. of Pharmacology]

**Augmentation of apoptosis in bronchial exudated rat eosinophils by cyclosporin A.**

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Cyclosporin A (CyA) and dexamethasone (Dex) enhanced the eosinophils (Eos) in bronchoalveolar lavage fluid from sensitized rats after inhaling an allergen apoptosis at concentrations of more than 0.1mM. Apoptosis in Eos was suppressed in the presence of culture supernatant of activated splenocytes as a source of various cytokines. Even in the presence of culture supernatant of activated splenocytes, Cys and Dex facilitated apoptosis in Eos. These results suggest that apoptosis death of activated Eos is augmented with Cys and that accelerated apoptosis in Eos of the airway may account for the inhibitory effect of Cys on Eos.

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[Lab. of Pharmacology]

**Selective growth of human mast cells induced by Steel factor, IL-6 and prostaglandin E<sub>2</sub> from cord blood mononuclear cells.**

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To establish the method for generating a large number of mature human mast cells, we cultured cord blood mononuclear cells in several conditions in the presence of steel factor (SF). The mast cells cultured in the presence of SF, IL-6, and PGE<sub>2</sub> for > 10 wk were 99% pure, and seemed to be functionally mature. Electron-microscopic analysis revealed that some of the cultured mast cells are morphologically mature since they filled with scroll granules and contained crystal granules.

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[Lab. of Pharmacology]

**Effect of a novel leukotriene synthesis inhibitor, BAY x1005, on antigen- and LPS-induced airway hyperresponsiveness in guinea pigs.**

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BAY x1005 is a selective inhibitor of leukotriene synthesis. The effects of BAY x1005 on the antigen- and bacterial lipopolysaccharide (LPS)-induced airway hyperresponsiveness (AHR) in guinea pigs were studied. Six times provocation of aeroantigen caused biphasic increases in respiratory resistance (Rrs) which peaked at 1 and 4 hr. It also caused AHR to acetylcholine. BAY x1005 inhibited antigen-induced increase in Rrs at 1 and 4 hr after the last antigen challenge. Simultaneously, BAY x1005 inhibited the antigen-induced AHR and airway eosinophilia and LPS-induced AHR. These results suggest that BAY x1005 is a potent anti-asthmatic agent.