[Prostaglandins Leukotrienes Essential Fatty Acids, 48, 343-349 (1993)] [Lab. of Pharmacology]

The Effect of a Novel Thromboxane A_2 (TXA_2) Receptor Antagonist (S-1452) on the Antigen-Induced Bronchoconstriction and Airway Hyperresponsiveness in Guinea Pigs.

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The effect of a thromboxane A_2 (TXA_2) receptor antagonist, S-1452 on the antigen-induced bronchoconstriction and airway hyperresponsiveness in guinea pigs was studied. S-1452 clearly inhibited U-46619-induced pulmonary pressure increase. The agent inhibited the antigen-induced bronchoconstriction in passively sensitized guinea pigs. The agent also inhibited repeated antigen provocation-induced airway hyperresponsiveness, but did not affect the accumulation of inflammatory cells in bronchoalveolar lavage fluid.

[Prostaglandins Leukotrienes Essential Fatty Acids, 48, 447-453 (1993)] [Lab. of Pharmacology]

Pharmacological Model for Airway Hypersensitivity Produced by Propranolol and Reserpine in Guinea Pigs.

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Combined treatment with propranolol and reserpine enhanced acetylcholine-induced dose-response curves for bronchoconstriction in guinea pigs in vivo. Increased capillary permeability and increases in leukocytes in bronchoalveolar lavage fluid were not observed. The increased airway sensitivity to acetylcholine was inhibited by ketotifen and theophylline. Leukotriene receptor antagonists, MCI-826 and FPL-55712 clearly inhibited the increased airway reactivity. A thromboxane A_2 receptor antagonist, ONO-3708, and a thromboxane A_2 synthetase inhibitor, OKY-046, also inhibited the increased airway reactivity. These results indicate that this model is a valuable pharmacological tool for investigating a remedy.

[Life Sciences, 53, PL 243-247 (1993)] [Lab. of Pharmacology]

Effect of Anti-IL-5 Monoclonal Antibody on Allergic Bronchial Eosinophilia and Airway Hyperresponsiveness in Mice.

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The effect of pretreatment with rat anti-murine interleukin-5 (IL-5) antibody on antigen-induced bronchial eosinophilia and bronchial reactivity to acetylcholine in mice was studied. Three inhalations of an antigen by actively sensitized animals resulted in an increase in airway reactivity to acetylcholine. Twenty-four hours after the final inhalation, the number of leukocytes (mononuclear cells and eosinophils) and the amount of IL-5 in BALF increased significantly. Anti-IL-5 monoclonal antibody inhibited the antigen-induced increase of eosinophils with little effect on bronchial hyperreactivity.