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[Lab. of Pharmacology]

The effect of a thromboxane A₂ receptor antagonist BAY-u-3405 on experimental allergic reactions.

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The effect of a novel thromboxane A₂ receptor antagonist, BAY-u-3405, on experimental allergic reaction was studied in vivo. BAY-u-3405 inhibited the U-46619-induced increase in respiratory resistance (Rrs) and the aerosolized antigen-induced biphasic increase in Rrs in guinea pigs. BAY-u-3405 also inhibited repeated aeroantigen-induced airway hypersensitivity and airway inflammation in mice. Moreover, BAY-u-3405 inhibited the IgE antibody-mediated biphasic skin reactions in mice. These results demonstrated the efficacy of the drug on antigen-induced late-phase reactions in the airway and skin in guinea pigs and mice.

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[Lab. of Pharmacology]

Selective potentiation of IgE-dependent histamine release from rat peritoneal mast cells by stem cell factor.

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Effect of stem cell factor (SCF) on histamine release (HR) from rat peritoneal mast cells (MC) was studied. Although SCF did not evoke HR by itself, it potentiated HR from sensitized MC caused by antigen, anti-IgE and concanavalin A, but not compound 48/80, calcium ionophore A23187 and substance P. Potentiation of HR by SCF was transient, but those by phosphatidylserine (PS) and non-MC in the rat peritoneal cavity was incubation time-dependent. Potentiation by SCF was additive to that by PS or non-MC. These results indicate that SCF selectively potentiates IgE-mediated HR from MC, and that the mechanism is distinct from that of PS or non-MC.

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[Lab. of Pharmacology]

Characterization of purification-associated reduction in IgE-dependent histamine release from rat peritoneal mast cells.

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Histamine release (HR) from purified rat peritoneal mast cells (PMC) was examined and compared to that from a non-purified preparation (PEC). Although both PEC and PMC released similar amounts of histamine upon stimulation with compound 48/80, calcium ionophore A23187 and substance P, IgE-dependent HR from PMC was very low compared to that of PEC. The reduced IgE-dependent HR from PMC, however, was recovered when PMC was reconstituted with non-mast cells (NMC) present in the peritoneal cavity. The potentiating effect of NMC was observed even in the presence of excess amount of phosphatidylserine. These results demonstrated that IgE-dependent HR from rat PMC is upregulated by other cells present in the peritoneal cavity.