〔J. Immunol., **148**, 3513-3519 (1992)〕 [Lab. of Pharmacology]

Tyrosine Phosphorylation is Reqired for Mast Cell Activation by FcεRI Cross-Linking.

TOSHIAKI KAWAKAMI, NAOKI INAGAKI*, MASAO TAKEI, HIROMI FUKAMACHI,
K. MARK COGGESHALL, KIMISHIGE ISHIZAKA, TERUKO ISHIZAKA

The possible role of tyrosine phosphorylation in the activiation process of mast cells by cross-linking of cell-bound IgE antibodies was investigated. Activation of mast cells induced a marked increase in tyrosine phosphorylation of several proteins. Activation of protein-tyrosine kinase (PTK) upon cross-linking of FcεRI was demonstrated by an *in vitro* kinase assay. However, Ca ionophore or PMA failed to induce the activation of PTK. Present results suggest that activation of PTK is an early event upstream of the activation of phospholipase C, and that it is involved in transduction of IgE-dependent triggering signals to mediator release.

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Antiallergic Mechanisms of Beta-Adrenergic Stimulants in Rats.

NAOKI INAGAKI, TORU MIURA, HIROICHI NAGAI, AKIHIDE KODA*

Antiallergic mechanisms of beta-adrenergic stimulants were investigated in rats. Isoproterenol administered intravenously inhibited IgE-mediated homologous passive cutaneous anaphylaxis and histamine-induced cutaneous reaction elicited at the same time. Histamine release in the rat peritoneal cavity caused by intravenous antigen was inhibited by isoproterenol and salbutamol administered intravenously. When histamine release was caused by intraperitoneal antigen, these beta-stimulants administered simultaneously with antigen failed to inhibit the reaction. Furthermore, these drugs did not inhibit *in vitro* histamine release from peritoneal mast cells. Beta-adrenergic stimulants may prevent intravenously administered antigen from activating sensitized mast cells through affecting endothelial cells.

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Studies of the Anti-Allergic Mechanism of Glucocorticoids in Mice.

NAOKI INAGAKI, TORU MIURA, TAKASHI NAKAJIMA, KENJI YOSHIDA,
HIROICHI NAGAI, AKIHIDE KODA

The effect of actinomycin D on dexamethasone-caused inhibition of passive cutaneous anaphylaxis (PCA) and histamine-induced cutaneous reaction in the mouse ear was investigated. Tyrosine aminotransferase (TAT) activity in the liver significantly increased by the administration of dexamethasone. Actinomycin D in doses of 1 and 10 mg/kg abrogated the increase in TAT activity by dexamethasone. Treatment with 1 mg/kg of actinomycin D failed to affect the inhibition of PCA and histamine-induced cutaneous reaction by dexamethasone. These relults suggest that glucocorticoids exhibit their inhibitory action of PCA and chemical mediator-induced cutaneous reactions in mice through a mechanism resistant to actinomycin D treatment.