〔Int. Archs Allergy appl. Immun., 86, 157 (1988)〕

Effects of Different Drugs on Passive Cutaneous Anaphylaxis Elicited in the Mouse Ear at 1.5 Hours.

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IgGl antibody-mediated homologous passive cutaneous anaphylaxis at 1.5 h (1.5-hour PCA) was elicited in the ears of mice and the effects of different drugs were studied. The reaction, as measured by the amount of extravasated dye, was inhibited by antihistamines, antiserotonins, cyclic AMP-elevating agents, tranilast and ketotifen but not by an SRS-A antagonist, lipoxygenase inhibitors, cyclooxygenase inhibitors and disodium cromoglycate. These results suggest that the pharmacological profile of 1.5-hour PCA resembles that of 48-hour PCA mediated by IgE antibody in the mouse ear.

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Comparative Study of IgGl and IgE Antibody Mediated Homologous PCA in the Mouse Ear. Lack of Cross-Desensitization of IgGl Antibody Mediated PCA to IgE Antibody Mediated PCA.

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To characterize IgGl antibody and IgE antibody mediated homologous PCA in the mouse ear, cross-desensitization was studied using a double-sensitizing technique. Mice were sensitized by injecting into their ears a mixture of 2 kinds of antibodies with distinct antigen specificities, and PCAs were elicited twice with specific antigens at an appropriate interval. In PCA mediated by IgGl antibodies, elicitation of the first reaction significantly inhibited the second reaction. Similar results were obtained in the case of IgE antibody mediated PCA. However, IgGl antibody mediated PCA did not affect the following reaction caused by IgE antibody.

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Inhibition of Vascular Permeability Increase in Mice. An Additional Anti-Allergic Mechanism of Glucocorticoids.

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The effects of glucocorticoids on IgE antibody-mediated 48-hour homologous PCA and skin reactions caused by mediator releasers and vascular permeability increasing factors were investigated. PCA and skin reactions were evoked in the mouse ear. Hydrocortisone, prednisolone and dexamethasone inhibited the PCA significantly, and the maximum inhibition was obtained when administered 8 h prior to the antigenic challenge. Dexamethasone significantly inhibited the skin reactions caused by compound 48/80, Ca ionophore A23187, hypotonic salt solution, histamine, serotonin, platelet-activating factor, leukotrienes C_4 and D_4, and bradykinin. The maximum inhibitory effects of dexamethasone on these skin reactions were observed when administered 12-6 h before.