(Trop. Med. Parasit., 38, 157 (1987))

Polyclonal B-Cell Activation and Autoantibody Formation during the Course of Mosquito-Transmitted *Plasmodium berghei* Infection in Mice.

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The time course of polyclonal B cell activation, as measured by titers of antibodies to DNP, FITC, and haemocyanin, as well as the time course of autoantibody formation, was followed in mice infected with *Plasmodium berghei* via *Anopheles stephensi*. IgM class antibodies to DNP, FITC, and haemocyanin appeared earlier than IgG class antibodies and persisted until death. Only IgM class anti-DNP peaked. Although IgM class autoantibodies also appeared earlier than IgG class, they peaked sharply at days 14 to 15, as did antibodies to mouse RBC. Polyclonal B cell activation, as measured by spleen plaque-forming cells using SRBC and TNP-SRBC in the direct test, peaked at day 13. This latter event could be correlated with the time course of hypergammaglobulinemia.

(Int. Archs Allergy appl. Immun., 84, 390 (1987))

Inhibition of Antigen-Induced Contracticon of Guinea Pig Isolated Tracheal Muscle with 2-n-Butyl-3-Dimethylamino-5,6-Methylenedioxy Indene (MDI-A), Indane (MDI-B) and 8-(Diethylamino) octyl-3,4,5-Trimethoxy Benzoate Hydrochloride (TMB-8).

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The effects of three intracellular calcium antagonists, MDI-A, MDI-B and TMB-8, on antigen-induced contraction of sensitized guinea pig tracheal muscle were investigated. These agents caused a concentration dependent inhibition of the contraction and showed antagonistic actions in histamine and LTD<sub>4</sub>-induced contractions of tracheal musle. However, non of these agents affected the antigen-induced release of histamine and SRS-A. These results suggest that MDI-A and MDI-B inhibit the antigen-induced contraction by interfering with the action of histamine and LTD<sub>4</sub>.

(Japan. J. Pharmacol., 43, 454 (1987))

Change in the Activity of the Cyclic AMP-Dependent Protein Kinase in Antigen-Stimulated Sensitized Mast Cells and Effect of Drugs Inhibiting Allergic Mediator Release.

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The activity of cyclic AMP-dependent protein kinase (protein kinase A) in the sensitized rat mast cell was decreased 2 min after antigen challenge when the histamine release exhibited a maximum. Drugs inhibiting allergic mediator release such as disodium cromoglycate, tranilast and theophylline significantly inhibited antigen-induced histamine release and reduced a decrease in the activity of protein kinase A. These results suggest that protein kinase A is involved in the histamine releasing process in the mast cell, and drugs inhibiting allergic mediator release cause their effects partially through the inhibition of protein kinase A.