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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 LTD₄-induced contractions of tracheal muscle. However, none 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 LTD₄.

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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.