

[*Life Sci.*, 67, 1869-1879 (2000)]

[Lab. of Pharmacology]

The Effect of Am-80, One of Retinoids Derivatives on Experimental Allergic Encephalomyelitis in Rats.

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The effect of Am-80, a synthetic retinoid, on EAE in DA rats was examined. Am-80 diminished the clinical symptoms and infiltration of inflammatory cells in a dose-dependent manner. After stopping administration, however, EAE recurred. Transcriptional levels of IL-6, IFN- γ and TNF- α were parallel with the clinical symptoms of the disease in Am-80-treated rats. Present results suggest that inhibition of EAE by Am-80, in part, related to the inhibition of IL-6 production.

[*Pharmacology*, 61, 230-237 (2000)]

[Lab. of Pharmacology]

Effects of an Amphoteric Antiallergic Agent, HSR-609, on Antigen-induced Late Phase Nasal Eosinophilia in Brown Norway Rats.

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An experimental rhinitis model was established in BN rats, and the effect of a newly synthesized compound, HSR-609, on the rhinitis was investigated. Repeated exposure to antigen resulted in the increase in the number of inflammatory cells in the nasal cavity lavage fluid. Antigen-specific IgG and IgE were detected in the sera. HSR-609 inhibited the increase in the number of eosinophils in the nasal cavity lavage fluid.

[*Allergol. Int.*, 49, 253-261 (2000)]

[Lab. of Pharmacology]

Interleukin-4 Is Involved in Allergen-induced Airway Eosinophilic Inflammation and Bronchial Hyperresponsiveness Independent of Genetic Background.

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The role of IL-4 in the development of antigen-induced airway inflammation and bronchial hyperresponsiveness was examined in IL-4-gene-deficient BALB/c and C57BL/6 mice. In BALB/c mice, allergen challenge caused airway eosinophilia, bronchial hyperresponsiveness and increased level of serum IgE. In C57BL/6 mice, allergen-induced eosinophilia and hyperresponsiveness were moderate and serum IgE was not detectable. IL-4 gene disruption in both strains of mice abolished the allergen-induced eosinophilia, hyperresponsiveness and IgE production.

[*Int. Arch. Allergy Immunol.*, 123, 365-368 (2000)]

[Lab. of Pharmacology]

Inhibition of Passive Cutaneous Anaphylaxis-associated Scratching Behavior by μ -Opioid Receptor Antagonists in ICR Mice.

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Effects of opioid μ -receptor antagonists on the scratching behavior in ICR mice were investigated. Scratching behavior was induced by IgE-mediated PCA, compound 48/80 injection and histamine injection. Naloxone and nalmefene reduced the incidence of scratching behavior associated with PCA. Naloxone also inhibited the induction of scratching behavior caused by compound 48/80 and histamine. Naloxone did not affect the increase in vascular permeability caused by PCA and compound 48/80 injection.