

[Int. Arch. Allergy Appl. Immunol., 90, 137 (1989)]

Anti-asthmatic activity of newly synthesized calcium antagonists: 2-n-butyl-1 {N-methyl-N-[2-(N', N'-dimethylamino)ethyl]amino} -5, 6-methylenedioxy-indene and -indane.

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The anti-asthmatic activity of 2-n-butyl-1{N-methyl-N-[2-(N', N'-dimethylamino)ethyl]amino}-5,6-methylenedioxy-indene (MDI-C) and -indane (MDI-D), were investigated in guinea pigs. The agents inhibited the antigen-induced contraction of sensitized tracheal smooth muscle. In histamine and leukotriene D₄-induced contractions of tracheal smooth muscle, each agent showed antagonistic actions. These agents demonstrated potent calcium antagonistic actions. Both MDIs inhibited the antigen-induced release of histamine and SRS-A from sensitized lung tissue, and inhibited asthmatic respiratory disorders without affecting blood pressure in guinea pigs.

[J. Pharmacobio-pyn., 12, 164 (1989)]

Evaluation of anti-allergic effects of 1-substituted 2-n-butyl-methylenedioxyindenes.

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The effects of ten 1-substituted 2-n-butyl-5, 6-methylenedioxyindenes on passive cutaneous anaphylaxis (PCA) in mice and Schultz-Dale reactions in guinea pig tracheal muscle were investigated in the development of a new anti-allergic drug. 2-Butyl-1-[N-methyl-N[2-(N', N'-dimethylamino)ethyl]amino]-5,6-methylenedioxyindene (*I*) indicated the most potent anti-allergic activity. The effect of *I* on allergic reaction and Ca-induced contraction of tracheal muscle in these animals were compared with 2-n-butyl-3-dimethylamino-5,6-methylenedioxyindene, which showed a potent anti-allergic effect by interfering with the calcium(Ca) movement in the allergic reaction in guinea pigs. The present data indicate the superiority of *I* in both reactions.

(Inflammation, 13, 673 (1989))

Role of peptide-leukotrienes in liver injury in mice.

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The role of peptide leukotrienes (p-LTs), especially LTC₄ and LTD₄ in liver disease, was investigated in mice experimental liver injury models. The liver injury was induced by the injection of bacterial lipopolysaccharide (LPS) into *Corynebacterium parvum* pretreated mice. Carbon tetrachloride-induced liver injury in mice was used as a standard model. Significant elevation of LTC₄ was observed in both models 1 and 6 h after the onset of disease. Administration of AA-861 and LY-171883 suppressed the elevation of serum GOT and GPT levels and histopathological changes in both models, and authentic LTC₄ or LTD₄ injected into the mouse increased serum GOT and GPT and histopathological changes of the liver.