Anti-asthmatic activity of newly synthesized calcium antagonists: 2-n-buty1-1{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-buty1-1{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.

Evaluation of anti-allergic effects of 1-substituted 2-n-buty1-methylenedioxyindenes.

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The effects of ten 1-substituted 2-n-buty1-5, 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-Buty1-1{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-buty1-3-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.

Role of peptide-leukotrienes in liver injury in mice.


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.