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Three days after a single exposure to ozone, the mechanism ...

Ozone causes persistent airway hyperreactivity in humans and animals. One day after ozone exposure, airway hyperreactivity is mediated by release of eosinophil major basic protein that inhibits neuronal M₂ muscarinic receptors, resulting in increased acetylcholine release and increased smooth muscle contraction in guinea pigs.

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(apoE) is an endogenous negative regulator of airway hyperreactivity (AHR) and mucous cell metaplasia in

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experimental models of house dust mite (HDM)-induced airway disease.

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Airway hyperreactivity is a common feature of asthma and is characterized by exaggerated airway narrowing in response to a variety of stimuli . It is well established that airway insults (e.g., viral infection, ozone inhalation, antigen challenge) can precipitate or worsen asthma-like symptoms, including airway hyperreactivity, in normal . Three days after a single exposure to ozone, the mechanism ...

Ozone causes persistent airway hyperreactivity in humans and animals. One day after ozone exposure, airway hyperreactivity is mediated by release of eosinophil major basic protein that inhibits neuronal M₂ muscarinic receptors, resulting in increased acetylcholine release and increased smooth muscle contraction in guinea pigs.

Sara A. Mathie - ResearchGate

Innate epithelial-derived mediators are sufficient to drive airway hyper-reactivity and remodeling in response to environmental insults in the absence of overt Th₂-type inflammation in a model of

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Mediators of inflammation can be classified into those that exist preformed in cells and those that are rapidly generated following stimulation. Vasoactive amines, lysosomal enzymes, neuropeptides

CHEMICAL MEDIATORS OF INFLAMMATION

CHEMICAL MEDIATORS OF INFLAMMATION

Definition: any messenger that acts on blood vessels, inflammatory cells or other cells to contribute to an inflammatory response.

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Mast cells also release inflammatory cytokines, chemokines, and proteases that contribute to airway inflammation. 24,25,28 These mediators stimulate an inflammatory response in the epithelium, directly damage the airway, and recruit more leukocytes to the airway.