Extracellular ATP in asthma airway inflammation

A role for ATP as a pro-asthmatic mediator has previously been suggested from in vitro studies. This group investigated whether extracellular ATP and purinergic signalling are important mediators of airway inflammation in asthma, using experimental mice models and human subjects.

Rapid accumulation of ATP was observed in bronchoalveolar lavage (BAL) fluid after allergen challenge in subjects with allergic asthma and in experimentally sensitised mice. In the mouse model, ATP binding to purinergic receptors drove inflammatory chemotaxis and bronchospasm. Prevention of this increase in ATP using treatment with the ATP-hydrolysing enzyme apyrase to degrade ATP, or blockade of ATP effects using broad spectrum purinergic P2 receptor antagonists, down modulated all of the cardinal features of the asthmatic response. In addition, administration of exogenous ATP to mice enhanced Th2-type sensitisation to inhaled antigen, indicating that ATP may contribute to the development of asthmatic sensitisation by recruitment of Th2-inducing lung myeloid dendritic cells to mediastinal lymph nodes.

The authors conclude that ATP and purinergic signalling are important in the pathogenesis of asthma. Further studies are needed, but this may be an important therapeutic target for the future.


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