

## Airway inflammation: Mechanisms and therapeutic implications

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It has been recognised for many years that asthma is an inflammatory disease of the airways and that suppression of inflammation is associated with marked clinical benefit. Recently interest has focused on airway inflammation in COPD. There is now evidence that airway inflammation is important in the pathogenesis of COPD and that reduction of airway inflammation is associated with clinical improvement.

The characteristics of airway inflammation in asthma are of CD4 positive T-cells, eosinophils and mast cells together with structural changes such as epithelial disruption and thickening of the reticular basement membrane. Treatment with inhaled corticosteroids causes resolution of these inflammatory changes in mild to moderately severe asthma with the exception that normalisation of the thickened reticular basement membrane has not been seen in all studies. While there are theoretical reasons to believe that long-acting beta 2 agonists have additional anti-inflammatory effects it has been difficult to demonstrate this in clinical asthma.

In COPD the characteristic inflammation is of CD8+ T-cells and macrophages with neutrophils being prominent in sputum or lavage. There is evidence that once this pattern of airway inflammation has been set up even smoking cessation does not lead to its resolution. Inhaled steroids only have a minor effect on the inflammation of COPD but, in contrast, the combination of an inhaled steroid and a long-acting beta 2 agonist has been shown to have a broad spectrum anti-inflammatory effect is likely that this explains many of the clinical benefits of combination therapy in COPD, particularly with respect to reduction of exacerbations, reduction of mortality and reduction in rate of decline of lung function.

New anti-inflammatory agents are being developed for both asthma and COPD and are in clinical development at present.