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# Eosinophil recruitment in TNF-receptor-deficient mice

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## Keywords

Eosinophil recruitment, knockout mice, TNF receptor

# Context

Tumour necrosis factor (TNF) is increased in the airways of asthmatic patients; however, its contribution to allergic inflammation is unclear. Two different receptors (p55 and p75) in the mouse mediate different inflammatory responses to TNF-a. In this study TNF receptor gene knockout mice were used to investigate the role of TNF in allergic inflammation.

# Significant findings

Mice deficient in P55 or P55/75 demonstrated significantly reduced, though not abolished, alveolar eosinophilia in response to airway challenge with ovalbumin. This was despite allergic sensitisation demonstrated by immediate skin test hypersensitivity. In conjunction, studies measuring eosinophil adhesion and rolling on mesenteric venules showed significant reduction in eosinophil trafficking in p55-deficient mice. In contrast, bronchial hypersensitivity to metacholine was preserved, possibly reflecting the persistent, though reduced, alveolar eosinophilia.

# Comments

By comparing double- and single-TNF-receptor knockout mice in a model of eosinophilic inflammation, the investigators demonstrated an important contribution of p55 in eosinophil recruitment in allergic pulmonary disease. TNF is known to increase the expression of various cell adhesion molecules and this may explain the findings. Given the complex inflammatory milieu that exists in asthmatic airways it is not surprising that eosinophil inhibition was incomplete. This study, however, points to a fundamental role for TNF in the development and persistence of allergic inflammation.

# Methods

Gene knockout, BAL, airway resistance measurement, immunohistochemistry, eosinophil adhesion studies

# Additional information

## References

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