Th9 cells elicit bronchial hyperresponsiveness through eosinophil-independent mechanisms

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Th2 cells play an important role in the pathogenesis of bronchial asthma, because eosinophils migrated into the lungs in response to Th2 cytokines are considered to cause bronchial hyperresponsiveness (BHR). Recently, a newly identified T cell subset, Th9, was reported to induce the same responses as Th2 cells. We have demonstrated that antigen-induced airway eosinophil infiltration and BHR developed in mice transferred with Th9 cells, without an assistance of IgE/mast cell-dependent responses. However, differential mechanisms were implicated in BHR elicited by Th2 and Th9 cells. Th9 cell-mediated BHR was reproduced in eosinophil-deficient mice and resistant to glucocorticoid treatment, whereas Th2 cell-mediated BHR disappeared in those conditions. Identification of mediators responsible for not only eosinophil-mediated responses but also eosinophil-independent BHR may be useful for the development of new anti-asthma drugs, especially against steroid-resistant asthma.