Effect of repeated antigen inhalation on airway inflammation and bronchial responsiveness to acetylcholine in interleukin-5 transgenic mice

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Bronchial hyperresponsiveness (BHR) is a characteristic feature of bronchial asthma, yet its precise mechanism remains obscure. Human studies have demonstrated that T helper 2 cytokines, including interleukin (IL)-4 and IL-5, are involved in the development of airway inflammation and BHR. In the present study, we examined the role of IL-5 in the onset and aggravation of bronchial responsiveness to acetylcholine in two strains of IL-5 transgenic (Tg) mice, derived from the C3H/HeN and BALB/c strains. Three inhalations of antigen (ovalbumin) caused an increase in the number of eosinophils in bronchoalveolar lavage fluid (BALF) and a significant elevation in serum IgE in wild-type mice. In contrast with wild-type animals, systemic overproduction of IL-5 resulted in massive airway eosinophilia, especially around the peribronchi and perivascular regions of the tissues, after repeated antigen provocation. In C3H/HeN background IL-5 Tg mice, repeated antigen provocation did not induce BHR similar to that of wild-type mice. In contrast, antigen-induced BHR was observed in BALB/c- background mice, but there were no significant differences between the magnitude of BHR in wild-type and IL-5 Tg mice. Furthermore, antigen-induced IL-5 production in BALF was detected in both C3H/HeN and BALB/c mice and was elevated to a similar degree in both wild-type and IL-5 Tg mice. These findings suggest that systemic overproduction of IL-5 or airway eosinophilia is not, by itself, important in the development or aggravation of antigen-induced BHR in mice.

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