Effects of overexpression of IL-10, IL-12, TGF-beta and IL-4 on allergen induced change in bronchial responsiveness

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摘要

Abstract

BACKGROUND: An increasing prevalence of allergic diseases, such as atopic dermatitis, allergic rhinitis and bronchial asthma, has been noted worldwide. Allergic asthma strongly correlates with airway inflammation caused by the unregulated production of cytokines secreted by allergen-specific type-2 T helper (Th2) cells. This study aims to explore the therapeutic effect of the airway gene transfer of IL-12, IL-10 and TGF-beta on airway inflammation in a mouse model of allergic asthma. METHODS: BALB/c mice were sensitized to ovalbumin (OVA) by intraperitoneal injections with OVA and challenged by nebulized OVA. Different cytokine gene plasmids or non-coding vector plasmids were instilled daily into the trachea up to one day before the inhalatory OVA challenge phase. RESULTS: Intratracheal administration of IL-10, IL-12 or TGF-beta can efficiently inhibit antigen-induced airway hyper-responsiveness and is able to largely significantly lower the number of eosinophils and neutrophils in bronchoalveolar lavage fluid of ovalbumin (OVA) sensitized and challenged mice during the effector phase. Furthermore, the effect of IL-10 plasmids is more remarkable than any other cytokine gene plasmid. On the other hand, local administration of IL-4 gene plasmids before antigen challenge can induce severe airway hyper-responsiveness (AHR) and airway eosinophilia. CONCLUSION: Our data demonstrated that anti-inflammatory cytokines, particularly IL-10, have the therapeutic potential for the alleviation of airway inflammation in murine model of asthma.