While airway eosinophilia is a characteristic feature of asthma and allergic rhinitis, the accumulation of eosinophils in the airways is the terminal step of a sequence that involves complex cellular interactions, both in the airway itself and in the associated secondary lymphoid organs. It is now clear that T cells, specifically Th2 cells, play a critical role in the development of allergic airways inflammation by virtue of their ability to produce cytokines such as IL-4, IL-5 and IL-13. Indeed, these effector molecules can clearly mediate key processes such as eosinophilia, IgE isotype switching, goblet cell hyperplasia and bronchial hyperreactivity. However, the generation of a specific effector T cell subset is, in turn, dependent upon antigen presentation by professional antigen-presenting cells, most notably dendritic cells. Thus, understanding the tripartite interaction among, antigen antigen-presenting cells and T cells during primary and secondary immune responses is essential not only to elucidate the pathogenesis of allergic disease but also to develop novel intervention strategies. In this regard, the current intense research unveiling new co-stimulatory pathways coupled with the definition of their associated signal transduction factors is likely to lead to the discovery of promising molecular targets. Is there a need for novel therapeutic strategies for allergic diseases? It is, to some extent, paradoxical that despite the availability of a powerful therapeutic armamentarium, the prevalence of asthma and allied allergic diseases of the airways has not only decreased but significantly increased in the First World over the last 20 years. Indeed, it appears that currently available short-and long-acting bronchodilators as well as inhaled steroids have failed to reverse this trend. In the case of bronchodilators, these are effective medicines for symptomatic control but have limited anti-inflammatory activity. For steroids, which are powerful anti-inflammatory drugs, the reasons are not clear. In this presentation we will discuss the dilemma faced by inhaled steroid treatment: that by delivering steroids locally we may forfeit the opportunity to treat the sites where the immune response is generated and where antigen-specific T cells are stored. What are the prospects for genetic therapy? The introduction of genetic information provides unique opportunities to understand the role of specific genes in events that are key to the development of the allergic phenotype. That genetic information can be packaged in a variety of vehicles and introduced with different delivery systems may allow investigators to target specific cell types or immune sites. Moreover, the cloning of the most relevant allergens offers exciting opportunities to investigate the impact of concurrent administration of an allergen with an immuno-regulatory molecule. While locally delivered steroids will likely remain the treatment of choice to control inflammation in the effector organ one can foresee a possibly complementary role for gene transfer in the design of future immunotherapy strategies.