

response to particular allergens include those encoding the major histocompatibility complex (MHC) and certain chains of the T-cell receptor (TCR- $\alpha\delta$). The complexity of atopy and the associated diseases includes susceptibility, severity, and therapeutic responses, each of which is among the separate variables modulated by both innate and adaptive immune stimuli.

The induction of allergic disease requires sensitization of a predisposed individual to a specific allergen. The greatest propensity for the development of atopic allergy occurs in childhood and early adolescence. The allergen is processed by antigen-presenting cells of the monocytic lineage (particularly dendritic cells) located throughout the body at epithelial surfaces that contact the outside environment, such as the nose, lungs, eyes, skin, and intestine. These antigen-presenting cells present the epitope-bearing peptides via their MHC to T helper cells and their subsets. The T-cell response depends both on cognate recognition and on the cytokine microenvironment provided by the antigen-presenting dendritic cells, with IL-4 directing a T_H2 subset, interferon (IFN) γ a T_H1 profile, and IL-6 with transforming growth factor β (TGF- β) a T_H17 subset.

Allergens can induce an epithelial alarmin response, with expression of IL-25, TSLP, and IL-33, which stimulate group 2 innate lymphoid cells, which can generate large quantities of IL-5 and IL-13.

Allergens also contain pattern recognition ligands that facilitate the immune response by direct initiation of cytokine generation from innate cell types such as basophils, mast cells, eosinophils, and others. The T_H2 response is associated with activation of specific B cells that can also present allergens or that transform into plasma cells for antibody production. Synthesis and release into the plasma of allergen-specific IgE results in sensitization of Fc ϵ R1-bearing cells such as mast cells and basophils, which become activated on exposure to the specific allergen. In certain diseases, including those associated with atopy, the monocyte and eosinophil populations can express a trimeric Fc ϵ R1, which lacks the β chain, and yet respond to its aggregation.

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