Proteins

Th2 Cells
Th2-specific cytokines play a central role in the cellular response to allergens. These cytokines promote the production of IgG antibodies by B cells, induce the survival and recruitment of mast cells and eosinophils, promote edema, hyperviscosity, and increased vascular permeability, and stimulate structural changes in the airways. Regulatory Th1 cells also produce cytokines, such as IL-10 or IFN-γ, that can inhibit the allergic response by Th2 cytokines, indicating the potential for this cell type to be involved in regulating the pathogenesis of asthma.

Eosinophils
Eosinophils, like mast cells, are activated following allergen-induced IgE-FcεRI complex formation. Activated eosinophils can release proteases, cytokines, and other molecules that contribute to the inflammatory response in the late phase asthmatic reaction.

Basophils
Basophils, like mast cells, can also be activated by allergens. They are more sensitive to IgE-FcεRI cross-linking than mast cells and can quickly degranulate upon allergen exposure.

Mast Cells
Mast cells are the primary effector cells of the early allergic reaction. Cross-linking of IgE-FcεRI complexes on the surface of mast cells with allergen leads to the release of histamine, cysteinyl leukotrienes, and other inflammatory mediators.

Neutrophils
Neutrophils play an important role in the late phase reaction. They are attracted to the site of inflammation by chemokines produced by other immune cells. Neutrophils are activated by chemokines and can release proteases and other cytokines that contribute to tissue damage.

Allergy & Asthma
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Exposure to inhaled allergens, such as pollen, dust mites, molds, or animal dander can initiate an acute immune response in allergen-sensitized individuals that leads to airway inflammation. Persistent inflammation is associated with chronic responses in allergic individuals that lead to airway inflammation. R&D Systems offers a wide variety of research reagents for the characterization of allergic disease.

The Immune Response to Inhaled Allergens: Pathogenesis of Asthmatic Inflammation

- Basophils undergo degranulation to release histamine, and cysteinyl leukotrienes. These mediators cause smooth muscle contraction, which triggers the release of pro-inflammatory molecules at these sites by eosinophils, infiltrating basophils, neutrophils, and Th2 cells that recruit and activate naïve T cells to differentiate into T helper type 2 cells (Th2).

- The immediate response is the early, acute phase reaction in which mast cells and basophils undergo degranulation to release inflammatory mediators.

- The late phase asthmatic reaction occurs several hours after the initial reaction and is characterized by eosinophil infiltration, thickening of the airway, and increased mucus production.

- Th2 lineage commitment is established by IL-4-dependent expression of GATA3 which induces the expression of Th2 cytokines including IL-4, IL-13, IL-5, IL-9, and IL-13. Mast cells and basophils bind to the FcεRI receptors on mast cells, basophils, eosinophils, and neutrophils. Th2-lineage allergic response is mediated by IL-4 and IL-13 which induce B cells to produce allergen-specific IgE antibodies.

- IgE antibodies cross-link FcεRI receptors on mast cells and basophils to release mediators that cause immediate hypersensitivity (early phase asthmatic reaction; green arrow).

- Several hours later the late phase asthmatic reaction occurs. During this phase, Th2- and mast cell-derived cytokines stimulate eosinophil activation and leukocyte recruitment to the sites of allergic exposure.

- The release of pro-inflammatory mediators at these sites by eosinophils, infiltrating basophils, neutrophils, and Th2 cells plays a critical role in promoting chronic inflammation and airway remodeling.

For a complete listing of R&D Systems products available for Allergy and Asthma research, please visit our website at www.rndsystems.com/go/allergy.