IL-4 was detected in cryostat tissue sections of frozen effector cells in the early and late phase asthmatic reactions.

### Mast Cells
Mast cells are the primary effector cells of the early asthmatic reaction. Cross-linking of the IgE-FcRI receptors on mast cells unfolds a signaling cascade that leads to mast cell degranulation and release of histamine, serotonin, leukotrienes, and other inflammatory mediators.

### Basophils
Basophils, like mast cells, degranulate following allergen-induced IgE-FcRI cross-linking. The release of inflammatory mediators by these cells contributes to bronchial constriction in the early phase asthmatic reaction.

### Eosinophils
Eosinophils are the prime regulators of the late phase asthmatic reaction. These cells infiltrate the airways, and release cytokines and chemokines that attract inflammatory cells involved in the late phase asthmatic reaction.

### Neutrophils
Neutrophils play an important role in the late phase reaction. They express high levels of FcγRIIa receptors and release cytokines, and chemokines that contribute to airway constriction and mucous production. While infiltrating neutrophils are a characteristic of severe asthmatic reaction, neutrophil-mediated responses are not currently well understood.

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**About Lymphocyte Selection:**
- **CD14** in Human Lymph Node
- **CD4** and **CXCR4** in Human Lymphocytes
- **Cam4** and **CXCR4** in Mouse Lymphocytes

**About Intracellular Detection:**
- **Interleukin-5 (IL-5)** in Human Lymphocytes

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**Image Notes:**
- **Vector Labs**: A trademark of Vector Labs.
- **R&D Systems**: Tools for Cell Biology Research™
- **Allergy & Asthma**: www.RnDSystems.com
- **CD14**: Detection in Human Lymph Node.
- **CD40 Ligand**: Neutralization of CD40L Stimulation by Flow Cytometry.
- **IL-4**: Detection of STAT6 in Mouse DA3 Cells.
- **CXCR4**: Isolation of CXCR4+ Lymphocytes by FACS

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**Additional Resources:**
Exposure to inhaled allergens, such as pollen, dust mites, reptile or animal dander, can initiate an acute immune response in allergen-sensitive individuals that leads to asthma inflammation. Persistent inflammation is associated with airway hyperresponsiveness, increased mucus secretion, and thickening of the airway wall. The asthmatic response to allergens takes place in two phases. The immediate response is the early, acute phase reaction in which mast cells and basophils degranulate to release histamine and cytokines. These mediators cause smooth muscle contraction and mucosal edema, which are manifest as a shortness of breath and wheeze. The late phase reaction occurs several hours after the initial reaction and is characterized by excessive inflammation, infiltration of the airway by eosinophils, and neutrophils. This change in reactive airway remodeling.

The early and late phase asthmatic responses are initiated by the recognition and processing of allergens by dendritic cells which drive naïve T cells to differentiate into Th2 type 2 cells (Th2). Th2 lineage commitment is established by Th2 cytokines or expression of OX40, which induces the expression of Th2 cytokines including IL-4, IL-5, IL-9, IL-13, and GM-CSF (induction phase dark blue arrows). Together these cytokines direct the inflammatory responses to allergens (light blue arrows). The hallmark cytokine IL-4 promotes clonal expansion and, along with IL-13, drives Th2 cell differentiation towards a typical Th2 cell phenotype that promotes the release of pro-inflammatory molecules at these sites by eosinophils, infiltrating basophils, neutrophils, and Th2 cells. The allergic response to allergens triggers the release of mediators that cause immediate hypersensitivity (early phase asthmatic reaction; green arrows) and late phase asthmatic reaction (late phase reaction; purple arrows) which leads to 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/gor/Allergy.