#### Author: Yan Yu Asthma: Pathogenesis Reviewers: Jason Raserman Jennifer Au Genetic factors **Environmental factors** Clara Hanly (i.e. excess hygiene, fewer (i.e. HLA gene mutations, Yonglin Mai (废涂用) defects in bronchial siblings, antibiotics within Naushad Hirani\* Asthma: the first two years) \* MO at time of airway epithelium) Defined as airway hyper-responsiveness publication causing variable and reversible airflow obstruction Alopy: predisposition to allergic hyper-sensitivity in airways Triggers of airway hyperresponsiveness include: First exposure to triggers\* sensitizes helper T cells Upper respiratory Allergens (pollen, Air pollution, Drugs (aspirin, Exercise: Cold tract infections animal dander, cigarette smoke, NSAIDs, Betanie. other chemicals blockers) dust, mold, etc) (URTIS) Stimulation of B-cells to produce IgE, which binds to mast cell surfaces Vascular permeability → Activated Helper-T cells & IgE-sensitized edema of airway mucosa. Mast cells release mast cells now line the airways histamines. Goblet cell hyperplasia Airway leukotrienes, and → T mucus secretion obstruction Allergens Early other inflammatory cross-link lgEs response mediators Bronchial smooth 10-2 hrs? Second on mast cells muscle contraction exposure to triggers Asthma Delayed Activated mast cells Induce maturation of & helper T cells granular WBCs like response (3-4 hrs) release cytokines. eosinophils Bronchiole constriction Airways Note: Delayed response Eosinophils presents within 3-4 hrs. Eyes Conjunctivitis migrate into: peaks within 6-8 hrs, and



Rhinitis

Note:

resolves within 24 hrs.

# **Guide For Pathophysiology**

Sue E. Huether MS, Kathryn L. McCance MS

## **Guide For Pathophysiology:**

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