

Asthma: Pathogenesis

Genetic factors

(i.e. HLA gene mutations, defects in bronchial airway epithelium)

Environmental factors

(i.e. excess hygiene, fewer siblings, antibiotics within the first two years)

Atopy:

predisposition to allergic hyper-sensitivity in airways

First exposure to triggers* sensitizes helper T cells

Stimulation of B-cells to produce IgE, which binds to mast cell surfaces

Activated Helper-T cells & IgE-sensitized mast cells now line the airways

Second exposure to triggers

Early response (0-2 hrs)

Allergens cross-link IgEs on mast cells

Mast cells release histamines, leukotrienes, and other inflammatory mediators

Vascular permeability → edema of airway mucosa

Goblet cell hyperplasia → ↑ mucus secretion

Bronchial smooth muscle contraction

Airway obstruction

Delayed response (3-4 hrs)

Activated mast cells & helper T cells release cytokines

Induce maturation of granular WBCs like eosinophils

Eosinophils migrate into:

Airways

→ Bronchiole constriction

Eyes

→ Conjunctivitis

Nose

→ Rhinitis

Note: Delayed response presents within 3-4 hrs, peaks within 6-8 hrs, and resolves within 24 hrs

Asthma:

Defined as airway hyper-responsiveness causing variable and reversible airflow obstruction

Triggers of airway hyper-responsiveness include:

Upper respiratory tract infections (URTIs)

Allergens (pollen, animal dander, dust, mold, etc)

Air pollution, cigarette smoke, other chemicals

Drugs (aspirin, NSAIDs, Beta-blockers)

Cold air

Exercise

Asthma

Pathogenesis And Therapy Of Bronchial Asthma

Clemens Wendtner



Pathogenesis And Therapy Of Bronchial Asthma:

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Table of Contents Pathogenesis And Therapy Of Bronchial Asthma

1. Understanding the eBook Pathogenesis And Therapy Of Bronchial Asthma
 - The Rise of Digital Reading Pathogenesis And Therapy Of Bronchial Asthma
 - Advantages of eBooks Over Traditional Books
2. Identifying Pathogenesis And Therapy Of Bronchial Asthma
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Pathogenesis And Therapy Of Bronchial Asthma
 - User-Friendly Interface
4. Exploring eBook Recommendations from Pathogenesis And Therapy Of Bronchial Asthma
 - Personalized Recommendations
 - Pathogenesis And Therapy Of Bronchial Asthma User Reviews and Ratings
 - Pathogenesis And Therapy Of Bronchial Asthma and Bestseller Lists
5. Accessing Pathogenesis And Therapy Of Bronchial Asthma Free and Paid eBooks
 - Pathogenesis And Therapy Of Bronchial Asthma Public Domain eBooks
 - Pathogenesis And Therapy Of Bronchial Asthma eBook Subscription Services
 - Pathogenesis And Therapy Of Bronchial Asthma Budget-Friendly Options
6. Navigating Pathogenesis And Therapy Of Bronchial Asthma eBook Formats

- ePub, PDF, MOBI, and More
 - Pathogenesis And Therapy Of Bronchial Asthma Compatibility with Devices
 - Pathogenesis And Therapy Of Bronchial Asthma Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Pathogenesis And Therapy Of Bronchial Asthma
 - Highlighting and Note-Taking Pathogenesis And Therapy Of Bronchial Asthma
 - Interactive Elements Pathogenesis And Therapy Of Bronchial Asthma
 8. Staying Engaged with Pathogenesis And Therapy Of Bronchial Asthma
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Pathogenesis And Therapy Of Bronchial Asthma
 9. Balancing eBooks and Physical Books Pathogenesis And Therapy Of Bronchial Asthma
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Pathogenesis And Therapy Of Bronchial Asthma
 10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
 11. Cultivating a Reading Routine Pathogenesis And Therapy Of Bronchial Asthma
 - Setting Reading Goals Pathogenesis And Therapy Of Bronchial Asthma
 - Carving Out Dedicated Reading Time
 12. Sourcing Reliable Information of Pathogenesis And Therapy Of Bronchial Asthma
 - Fact-Checking eBook Content of Pathogenesis And Therapy Of Bronchial Asthma
 - Distinguishing Credible Sources
 13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
 14. Embracing eBook Trends
 - Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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