

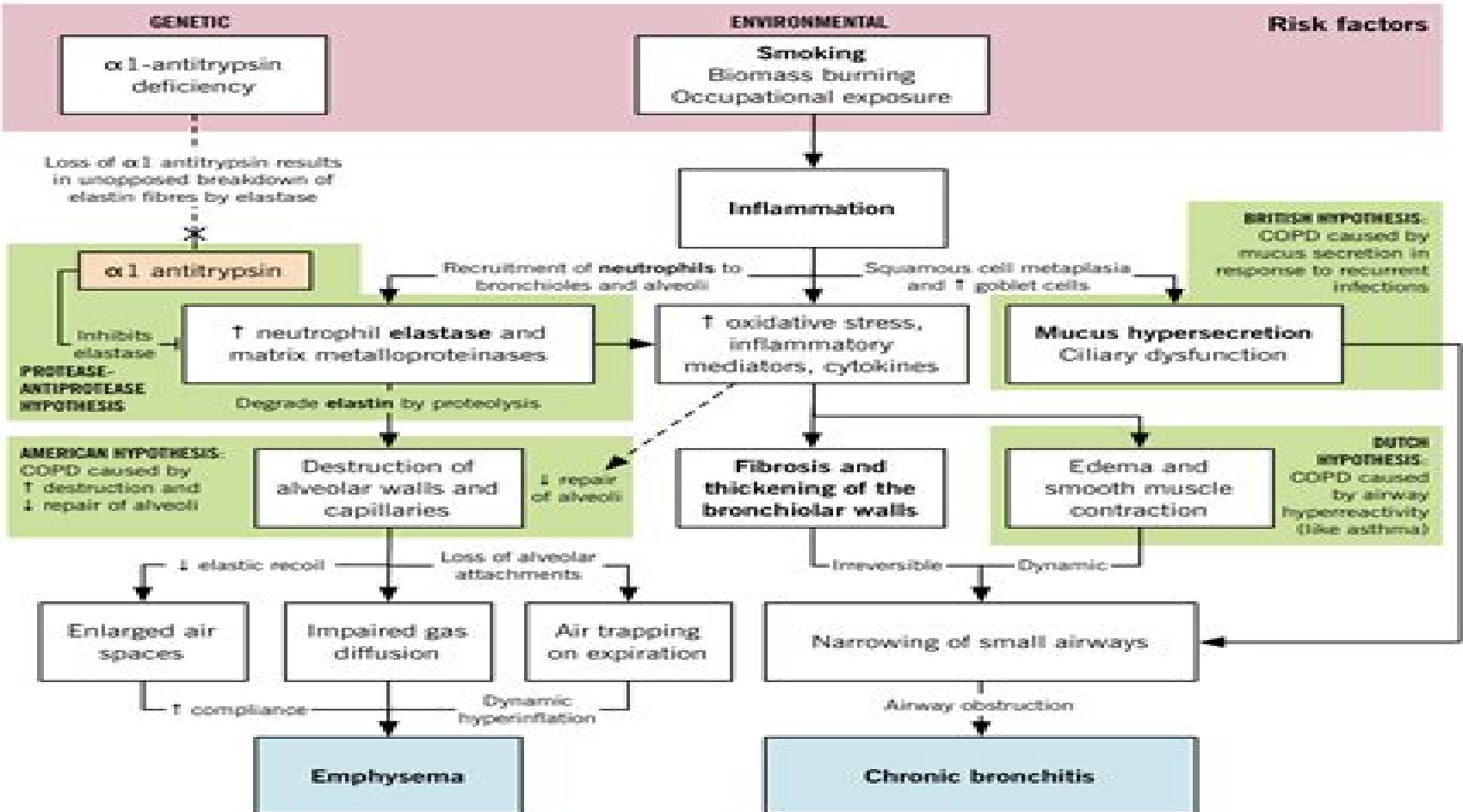
Pathogenesis of COPD

Sources: BMJ. 2006 May 20; 332(7551): 1202-1204.
Semin Respir Crit Care Med. 2005 Apr;26(2):142-53.

COPD arises from environmental exposures, particularly cigarette smoking, in genetically susceptible individuals. Both emphysema and chronic bronchitis arise from similar pathogenic mechanisms.

EMPHYSEMA: Smoking causes inflammation in the airways. Neutrophils and other immune cells are recruited to the small airways, releasing proteases and oxidative species. **Neutrophil elastase** breaks down elastin fibres that normally contributes to the elastic recoil during expiration. $\alpha 1$ antitrypsin is a protease inhibitor that keeps elastase activity in check. **$\alpha 1$ -antitrypsin deficiency** is the best known genetic predisposition to **emphysema**, especially in smokers with this genetic disorder. Impaired gas exchange and air trapping are also features of emphysema.

CHRONIC BRONCHITIS: Inflammation from smoke exposure also causes **fibrosis of the bronchiolar walls**, **mucus hypersecretion**, **airway edema**, and **bronchoconstriction**. These features make up the small airway disease component of COPD, known as **chronic bronchitis**.



Pulmonary Pathophysiology

Tobias Bleicker

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Table of Contents Pulmonary Pathophysiology

1. Understanding the eBook Pulmonary Pathophysiology
 - The Rise of Digital Reading Pulmonary Pathophysiology
 - Advantages of eBooks Over Traditional Books
2. Identifying Pulmonary Pathophysiology
 - 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 Pulmonary Pathophysiology
 - User-Friendly Interface
4. Exploring eBook Recommendations from Pulmonary Pathophysiology
 - Personalized Recommendations
 - Pulmonary Pathophysiology User Reviews and Ratings
 - Pulmonary Pathophysiology and Bestseller Lists
5. Accessing Pulmonary Pathophysiology Free and Paid eBooks
 - Pulmonary Pathophysiology Public Domain eBooks
 - Pulmonary Pathophysiology eBook Subscription Services
 - Pulmonary Pathophysiology Budget-Friendly Options

6. Navigating Pulmonary Pathophysiology eBook Formats
 - ePub, PDF, MOBI, and More
 - Pulmonary Pathophysiology Compatibility with Devices
 - Pulmonary Pathophysiology Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Pulmonary Pathophysiology
 - Highlighting and Note-Taking Pulmonary Pathophysiology
 - Interactive Elements Pulmonary Pathophysiology
8. Staying Engaged with Pulmonary Pathophysiology
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Pulmonary Pathophysiology
9. Balancing eBooks and Physical Books Pulmonary Pathophysiology
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Pulmonary Pathophysiology
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Pulmonary Pathophysiology
 - Setting Reading Goals Pulmonary Pathophysiology
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Pulmonary Pathophysiology
 - Fact-Checking eBook Content of Pulmonary Pathophysiology
 - 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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