

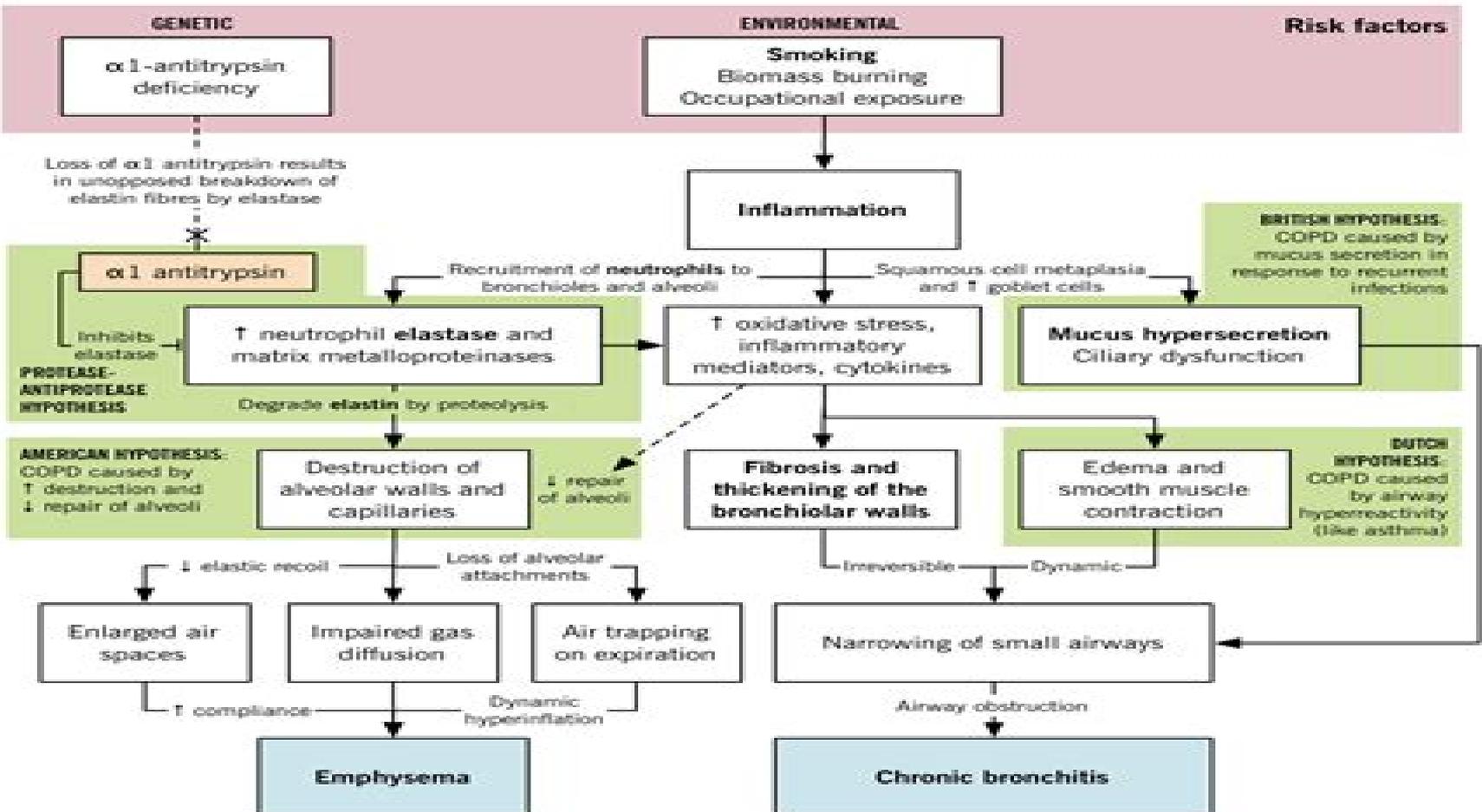
Pathogenesis of COPD

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

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