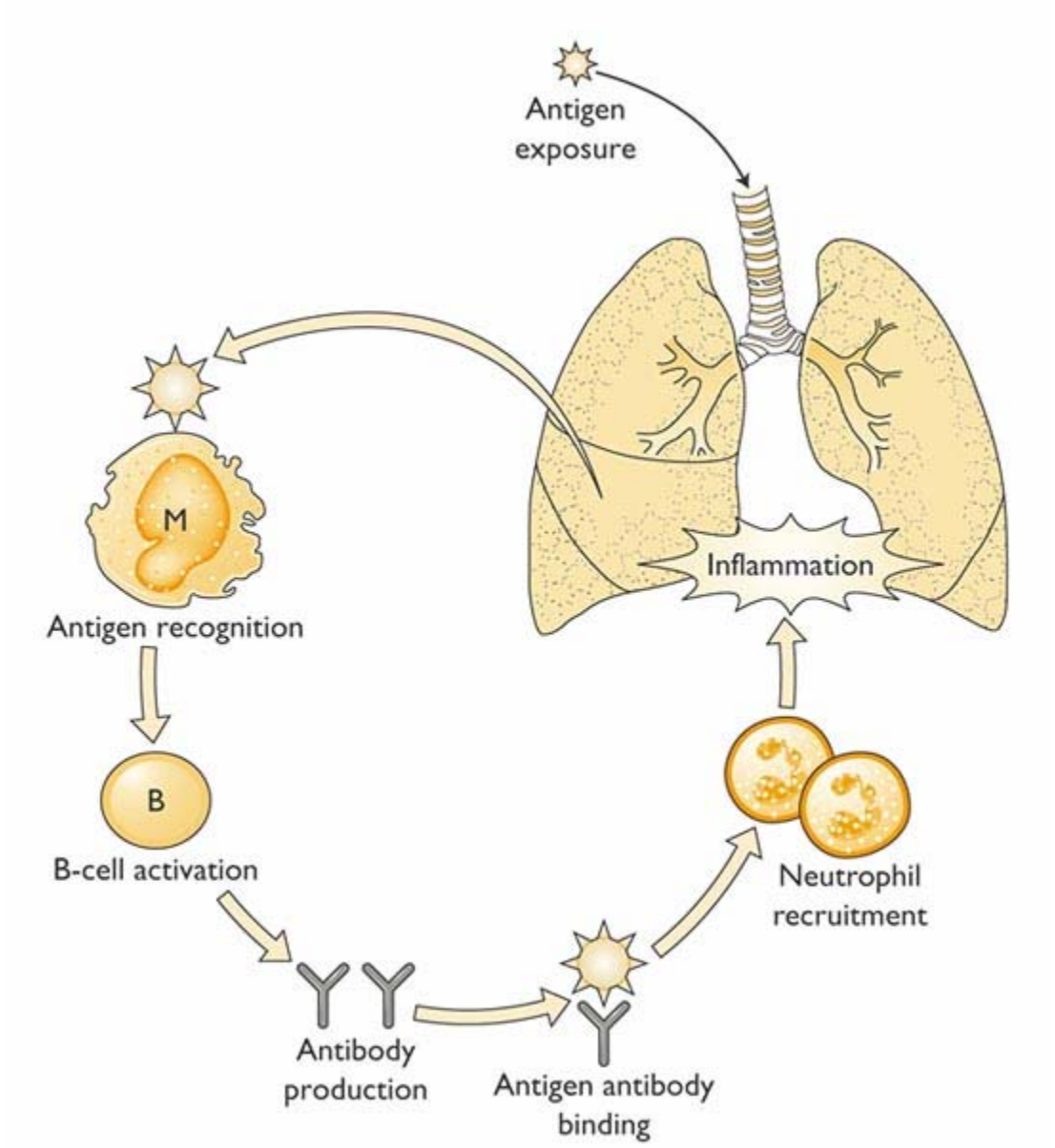
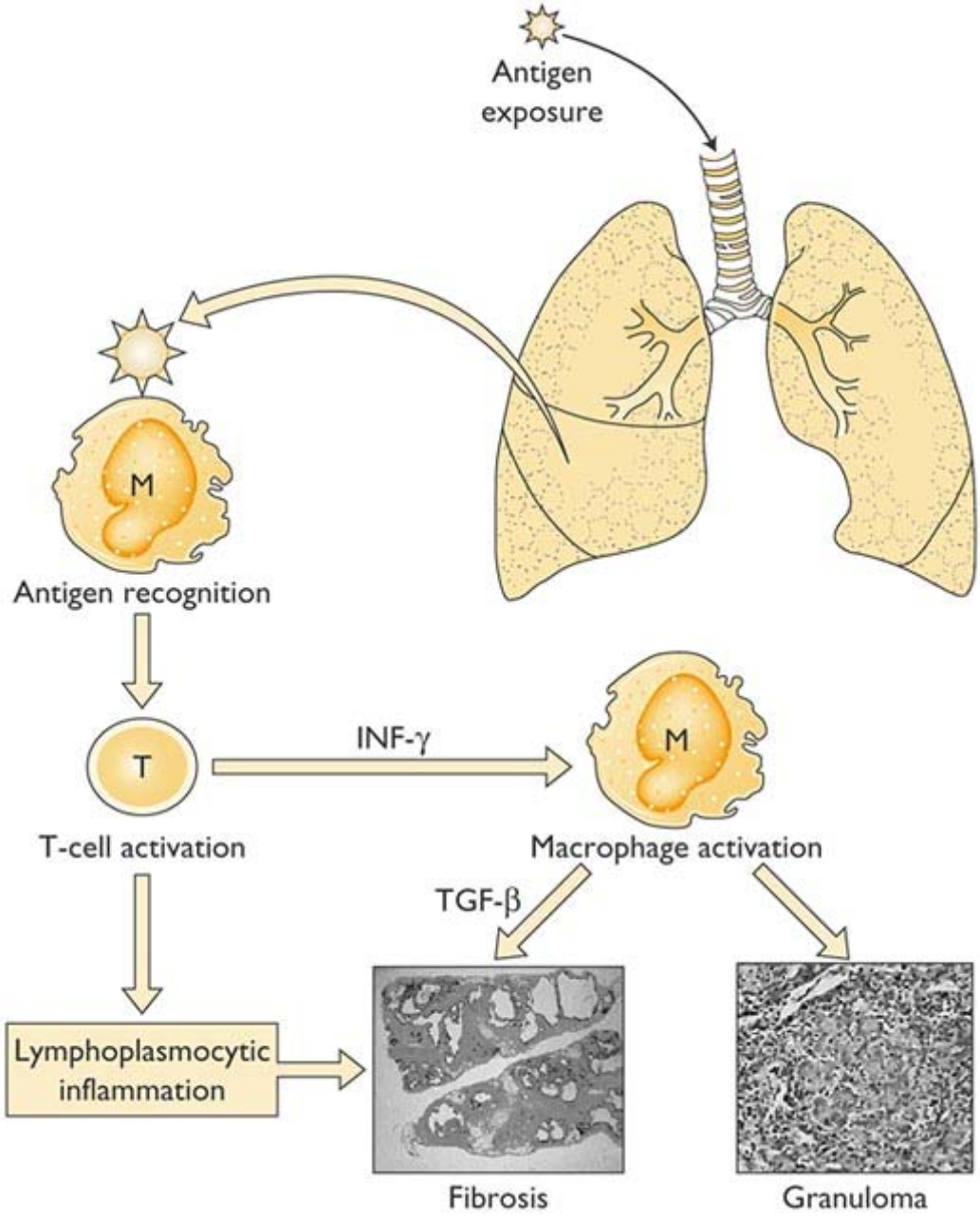


Pathophysiologic schema for hypersensitivity pneumonitis

Pathophysiologic schema for acute hypersensitivity pneumonitis. Acute hypersensitivity pneumonitis is thought to represent a primarily humoral immune response to an inhaled antigen (to be distinguished from the cellular immune response of the more chronic forms) [ref],[ref],[ref]. Inhaled antigen is recognized by antigen-presenting cells such as the alveolar macrophage (M) and presented to B cells (B). This results in B-cell activation, clonal proliferation, and antibody production. Antigen-

antibody immune complexes result in complement activation, recruitment of neutrophils, and generation of reactive oxygen species that result in acute inflammation. Equal antigen exposure to potential causative agents does not result in disease in all individuals. It is likely that a genetically based variation in an individual's antigen processing is important to disease pathogenesis [ref].



Pathophysiologic schema for subacute/chronic hypersensitivity pneumonitis

Pathophysiologic schema for subacute/chronic hypersensitivity pneumonitis. Subacute and chronic hypersensitivity pneumonitis are likely caused by a T-cell-mediated response to inhaled antigen [ref],[ref] Antigen presentation leads to activation of $CD4^+$ and $CD8^+$ T cells. $CD4^+$ cells elaborate T helper-1 cytokines such as interferon (INF)-γ, which promote macrophage (M) activation and granuloma formation [ref]. $CD8^+$ cells are critical to the development of delayed-type hypersensitivity and represent the lymphoplasmacytic inflammation characteristic of this disease. With continued exposure, activated macrophages release proliferative cytokines such as transforming growth factor (TGF)-β. This, in combination with ongoing lymphoplasmacytic inflammation, eventually leads to fibrosis.

Bone's Atlas of Pulmonary Medicine 2005