Table 15-6 Lung Diseases Caused by Air Pollutants

Agent	Disease	Exposure
Mineral Dusts		
Coal dust	Anthracosis Macules Progressive massive fibrosis Caplan syndrome	Coal mining (particularly hard coal)
Silica	Silicosis Caplan syndrome	Metal casting work, sandblasting, hard rock mining, stone cutting, others
Asbestos	Asbestosis Pleural plaques Caplan syndrome Mesothelioma Carcinoma of the lung, larynx, stomach, colon	Mining, milling, manufacturing, and installation and removal of insulation
Beryllium	Acute berylliosis Beryllium granulomatosis Lung carcinoma (?)	Mining, manufacturing
Iron oxide	Siderosis	Welding
Barium sulfate	Baritosis	Mining
Tin oxide	Stannosis	Mining
Organic Dusts That Induce Hypersensitivity Pneumonitis		
Moldy hay Bagasse Bird droppings	Farmer's lung Bagassosis Bird-breeder's lung	Farming Manufacturing wallboard, paper Bird handling
Organic Dusts That Induce Asthma		
Cotton, flax, hemp	Byssinosis	Textile manufacturing
Red cedar dust	Asthma	Lumbering, carpentry
Chemical Fumes and Vapors		
Nitrous oxide, sulfur dioxide, ammonia, benzene, insecticides	Bronchitis, asthma Pulmonary edema ARDS Mucosal injury Fulminant poisoning	Occupational and accidental exposure

ARDS, Acute respiratory distress syndrome.

classification is presented in Table 15-6. Where implemented, regulations limiting worker exposure have resulted in a marked decrease in dust-associated diseases.

Although the pneumoconioses result from well-defined occupational exposure to specific airborne agents, ambient air pollution also has deleterious effects on the general population, especially in urban areas (Chapter 9), and can have serious, sometimes fatal effects on those with COPD or heart disease. Pollution increases the risk of asthma, especially in children. Efforts to reduce air pollution have been effective in the west, but industrialization in other parts of the world, particularly China, has produced dangerous levels of air pollution. Even in the U.S. improvements are possible, as some data suggest that even low levels of air pollution can have deleterious effects on health.

Pathogenesis. The development of a pneumoconiosis depends on (1) the amount of dust retained in the lung and airways; (2) the size, shape, and buoyancy of the particles; (3) particle solubility and physiochemical reactivity; and (4) the possible additional effects of other irritants (e.g., concomitant tobacco smoking). In most cases, these particles stimulate resident innate immune cells in the lung, leading to the diseases discussed later. The following general principles apply to pneumoconioses:

- The amount of dust retained in the lungs is determined by the dust concentration in ambient air, the duration of exposure, and the effectiveness of clearance mechanisms. Any influence, such as cigarette smoking, that impairs mucociliary clearance significantly increases the accumulation of dust in the lungs.
- The most dangerous particles are from 1 to 5 μm in diameter, because particles of this size may reach the terminal small airways and air sacs and settle in their linings.
- The solubility and cytotoxicity of particles, which are influenced to a considerable extent by their size, modify the pulmonary response. In general, small particles composed of injurious substances of high solubility may produce rapid-onset lung damage. Such particles are more likely to cause acute lung injury. Larger particles are more likely to resist dissolution and may persist within the lung parenchyma for years. These tend to evoke fibrosing collagenous pneumoconioses, such as is characteristic of *silicosis*.
- Other particles may be taken up by epithelial cells or may cross the epithelial cell lining and interact directly with fibroblasts and interstitial macrophages. Some may reach the lymphatics by direct drainage or within