In very rare cases, enough dust has been inhaled to cause mechanical blockage of the air spaces. (Flour dust has been known to cause this condition.) Some dusts may be essentially inert and remain in the lungs indefinitely with no recognizable irritation, and a few (like limestone dust) may be gradually dissolved and eliminated without harm.

Silicosis is the most important lung disease caused by the inhalation of mineral dust. It is well-known in industries where crystalline free silica dust is present, such as foundries, glass manufacturing, granite cutting, mining, and tunneling in quartz rock. It is found throughout the world, and in the past it has had many names, such as miner's asthma, grinder's consumption, miner's phthisis, potter's rot, and stonemason's disease. The same occupational disease, however, is meant by all these names, and it is caused by dust from crystalline free silica, usually quartz.

Silicosis has been defined as "a disease due to breathing air containing silica characterized *anatomically* by generalized fibrous changes and the development of miliary nodulation in both lungs, and *clinically* by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic X-ray findings."*

Silicosis has been known to manifest itself after widely differing periods of exposure to silica dust. Apparently, development of the disease depends upon

1. The amount and kind of dust inhaled
2. The percentage of free silica contained in the dust
3. The form of the silica
4. The size of the particles inhaled
5. The duration of the exposure
6. The powers of resistance of the individual concerned
7. The presence or absence of a complicating process such as infection

Many theories have been advanced over the years to explain why crystalline free silica acts as it does in the lungs. It is now believed that the fibrosis produced is caused not by the hardness or sharpness of the particles, but by a combination of slight solubility with a physiochemical effect and an immunological effect—but no one is certain of the exact mechanism of the disease. Experimental work on the reasons for the development of silicosis is still going on in various parts of the world. If the precise mechanism of silicosis could be determined, better medical preventive measures might be developed and possibly a cure could be found.

Amorphous free silica differs from crystalline free silica in physical structure and in physiological effects. In the amorphous state, molecules of silica are randomly oriented and may be naturally converted to opal and diatomaceous earth (kieselguhr) or artificially converted into such forms as silica gel, silica fume, and fused silica or quartz.

If amorphous silica is heated to a high temperature, as in calcining, forms of crystalline free silica called cristobalite and tridymite result, intermediate forms of amorphous silica are known as crypto-crystalline (ultra-microcrystalline). Inhalation of these crystalline forms can readily cause diatomite pneumoconiosis.

When diatomaceous earth is calcined, particularly in the presence of a trace of alkaline flux, appreciable quantities are converted to cristobalite. As a result of studies made by the U.S. Public Health Service, it has been recommended that the threshold limit value for crude or amorphous diatomite be placed at 20 mppcf (million particles per cubic foot), but that the atmospheric concentration for dust containing cristobalite be kept under 5 mppcf.

Various commercial products containing particles of silica under 1 μ in size are available. The physiological effects of these products have not been well defined. Until more experience with human beings is available, these products should be handled with care.

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*American Public Health Association, 1790 Broadway, New York City. "Report (Joint) of the Committee on Pneumoconiosis and the Committee on Standard Practices in Compensation of Occupational Diseases." *Year Book*, 1933.