

INDUSTRIAL HYGIENE BULLETIN



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PHARMACOLOGY INFORMATION SHEET
DUSTS AND THEIR EFFECTS ON HEALTH

5.1 THE DEPOSITION OF DUST IN THE RESPIRATORY TRACT

All the dust in the inspired air does not ordinarily reach the air sacs in the lungs. Much of it is ordinarily caught by impingement on the moist linings of the upper respiratory passages, namely, the nasal passages, throat, windpipe or other tubes leading to the lungs. As impingement of dust particles is the result of chance impact against these moist surfaces of the respiratory tract, collection is more efficient with the larger particles which possess appreciable momentum, and less efficient as the particles approach sizes which move as an integral part of the transporting air. It has been ascertained that particles larger than 10 microns are caught by impingement in the upper respiratory passages, and sphere-like particles measuring less than 6 microns (red blood cell = 7 microns; human hair = 200 microns) reach the lungs in great numbers. Fibrous particles of various dimensions such as asbestos, can also reach the lungs in large numbers.

5.2 THE REMOVAL OF DUST FROM THE RESPIRATORY TRACT

The nasal passages, the windpipe and other air passages extending down to the lungs are lined with specialized cells with hairlike fingers which whip and carry upward dust which is deposited on these surfaces. This action, which is called ciliary action, is never stagnant during life and results in the upward propagation and removal of dusts and excess mucous from the respiratory passages leading to the lungs.

The air sacs of the lungs are not cleansed by ciliary action; instead they are supplied with vast hordes of other types of specialized blood cells (phagocytes and

microphages) which ingest the dust and, under their power of independent locomotion, remove the particles from the lungs. In addition to these methods of removing dusts from the respiratory tract, some dusts are removed by direct absorption into the blood.

5.3 SPECIFIC DUSTS AND THEIR EFFECTS FOLLOWING INHALATION

Certain dusts produce disabling injuries, while others are merely a nuisance. Several dusts in each of these classifications are discussed below.

5.3.1 SILICON DIOXIDE and ASBESTOS produce no injury to the tissues of the upper respiratory tract; however, these materials can produce serious changes in the tissues of the lungs and permanent disability. These changes consist largely of the conversion of the normal, healthy lung tissue to stringy, fiber-like structures -- a condition called fibrosis. Because of the formation of these fiber-like structures, the lungs lose some of their elasticity and the ability to breathe is impaired. Fibrotic conditions caused by inhaling excessive amounts of silicon dioxide are called asbestosis. There is no fully satisfactory explanation of the physiological mechanisms fundamentally responsible for the production of either silicosis or asbestosis. It is possible that these dusts exert a lethal action on the scavenger cells (phagocytes -- see preceding section), thus impeding efficient cleansing of the lungs. On the other hand, injury may be caused by some pronounced chemical or physical action exerted by these dusts directly on the tissues of the lungs.

Several months or years of continuous overexposure to dust of silicon dioxide or asbestos may be required before symptoms of serious respiratory difficulty become apparent. Therefore, X-ray is usually used to detect these conditions in their early stages of development. Increased susceptibility to tuberculosis is also associated with advanced cases of silicosis. Precautions should be taken to minimize inhalation of silicon dioxide and asbestos and sufficient ventilation should be used to maintain atmospheric concentrations of these dusts below the recommended safe level of five million particles per cubic foot of air.

5.3.2 ALUMINUM OXIDE, CARBORUNDUM (silicon carbide), MICA (below 5% free SiO_2), TALC, SOAPSTONE (below 5% free SiO_2), FULLER'S EARTH (below 5% free SiO_2) and SHALE (below 5% free SiO_2) produce no serious injury to either the upper respiratory tract, the lungs or other body organs. Although a few of the silicates have been reported to cause mild, transitory change in the normal structure of the lung tissue, these changes have been associated with several years of uncontrolled exposure to very high concentrations of these dusts. These dusts do not present any appreciable risk to workers, and exposures are ordinarily maintained well within tolerance levels (20 to 50 million particles per cubic foot of air) by minimal ventilating facilities.

5.3.3 CARBON BLACK and IRON are more or less biologically inert. They produce no significant irritation or injury to either the upper respiratory passages or the lungs. Chest X-rays of individuals who have been exposed to iron fume for several years are frequently suggestive of silicosis. However, the deposition of either iron or carbon black in the lungs has been observed over the years to produce no disability and no effect on longevity.

5.3.4 CALCITE, CALCIUM CARBONATE, GYPSUM, LIMESTONE, PORTLAND CEMENT, and most ACID and ALKALIN DUSTS encountered in industry do not normally produce injury to the lungs. Instead they exert an irritating action directly on the tissues of the upper respiratory tract -- the nose, throat or windpipe. These dusts usually cause coughing and sneezing; consequently, they ordinarily give warning when encountered in harmful concentrations in the air. When inhaled daily in high concentrations, these dusts may also produce irritation of the bronchi and cause a chronic bronchial cough.

5.3.5 FLUORIDES, CHROMATES, ALKALI BICHROMATES, OXMIUM TETRAOXIDE, CADMIUM and BERYLLIUM can produce serious irritation and damage to the lungs when breathed in excessively high concentrations. These dusts, which can also produce systemic intoxication following their absorption in the blood, are considered further under toxic dusts in section 5.3.6. Exposures to these dusts must be limited and all precautions must be taken to maintain atmospheric concentrations of these dusts below their respective threshold limit values.

5.3.6 The following are examples of toxic dusts which can be easily transferred from the inspired air to the blood.

Aldrin (see P.I.S. No. 6, Sect. 6.1.4)	Cyanides (see P.I.S. No. 4, Sect. 4.6)
Antimony	Dieldrin (see P.I.S. No. 6, Sect. 6.1.4)
Arsenic (see P.I.S. No. 6, Sect. 6.1.4)	Endrin (see P.I.S. No. 6, Sect. 6.1.4)
Beryllium	Fluorides
Cadmium	Lead (see P.I.S. No. 4, Sect. 4.1)
Barium	Organic Phosphates
	Phosphorus (yellow)
	Selenium Compounds

With fibrosis-producing dusts (see P.I.S. No. 5, Sect. 5.3.1) only particles small enough to enter the air sacs of the lungs are of importance; however, with these toxic dusts, all particle sizes are important because absorption can take place directly through the moist tissues lining the upper respiratory passages, as well as from the lungs. Consequently, greater systemic absorption and more severe injury can be caused by deposition of larger particles of these dusts on the tissues of the upper respiratory

passages than would occur from the deposition of finer particles in the lungs. Exposures to these dusts must be limited and all precautions must be taken to maintain atmospheric concentrations below their respective threshold limit values. NOTE WELL: There should be NO EXPOSURE TO BERYLLIUM.

5.3.7 SAWDUST, HOUSE DUSTS and GLASS WOOL DUST exert no particularly injurious effect on either the upper respiratory passages or the lungs. Because these dusts cause only mild and transitory degrees of local irritation to the lining of the nose or throat, they are generally classified as nuisance dusts rather than harmful dusts. Exposures are ordinarily maintained within tolerance levels (50 million particles per cubic foot of air) by minimum ventilating facilities.