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WORK
IS DANGEROUS TO YOUR HEALTH

A Handbook of Health Hazards in the Workplace and What You Can Do About Them

Jeanne M. Stellman, Ph.D.
Susan M. Daum, M.D.
With Contributions by
James L. Weeks, M.S.
Steven D. Stellman, PH.D.
Michael E. Green, PH.D.

With illustrations by Lyda Pola



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DUSTS

Dusts usually cause disease only in the lungs. Lung disease that results from dusts is called *pneumoconiosis,* derived from the Greek *pneumo-,* meaning "lung," *coni-,* meaning "dust," and *-osis,* meaning "reaction." The development of pneumoconiosis depends on the type of dust and the susceptibility of the individual worker, and is also affected by duration of exposure and by exposure to other chemicals or to cigarette smoke at the same time. Particle size affects the toxicity of dusts: the smaller the particle, the farther down into the lungs it can go, and also the greater the amount of dust that will be retained by the body. A general rule is: The more dust retained, the more severe the resulting illness. Dust particles may be so small they are visible only by microscopic analysis. Particles this size behave essentially like air, passing practically unimpeded through the protective barriers of hair and mucus in the upper airways. The air sacs and small air tubes therefore receive a very large dose, and the intensity of lung reaction is greater than if the same quantity of larger particles had been inhaled.

Inorganic Mineral and Chemical Dusts

Silica. Inhaling the free, finely divided dust of silicon dioxide causes a form of pneumoconiosis called *silicosis.* The dust may be in crystalline form, as in quartz, cristobalite, and tridymite, or in non-crystalline form, as in opal. The smaller and more compact crystalline forms (quartz, tridymite) cause the greater lung scarring since they have the most dangerous particle size: 0.05 microns to 5 microns (1 inch contains 25,400 microns).

Silicosis may develop either rapidly or slowly. The rapid-developing variety occurs most commonly among workers in the manufacture or packing of abrasive soap powders,

among sand blasters, especially those working in enclosed tanks), and among tunnel workers using high-power drills on rock. These workers are exposed to tremendous amounts of very finely divided crystalline silica. The chemical interaction between silica dust and lung tissue may be heightened by the presence of alkali or acid.

In rapid-developing or acute silicosis, symptoms may appear as soon as 8 to 18 months after the first exposure. The main symptom is shortness of breath, at first occurring only during physical activity, but soon appearing after less and less exertion, until eventually the victim is short of breath even while at rest. This is caused by the many small round lung scars that develop from irritation by silica dust. These hard, inelastic scars—just like those on the skin that result from an operation—make the lungs stiff, so that it takes more work to inflate them with air. The scars also thicken the walls of the air sacs, blocking the transfer of oxygen into the blood, low blood oxygen is a characteristic finding in silicosis. The area surrounding each scar becomes stretched and distorted, breaking down the normally tiny, delicate air sacs so that they form larger, thicker-walled sacs—a form of localized emphysema. Further reaction to the silica may cause the scars to join into larger scars; some may occupy the entire lung. This process, *progressive massive fibrosis,* is frequently accompanied by increased susceptibility to tuberculosis and other infections. Finally the heart, which must pump blood through these stiff, inelastic lungs, becomes weakened and enlarged and fails to pump effectively.

The course of chronic or slowly progressive silicosis is very similar to that of acute silicosis, except that rather than developing quickly, it arises after many years of exposure and may take many more years to become worse.

The legal standard for silica exposure depends on the form of the dust. Protective apparatus is useful only for emergencies, and would be difficult to work in for a full working day.

The prevalence of silicosis in workers exposed to silica dust is probably very high. A survey showed that in the period 1950–1958 there were 12,000 cases in 26 states. This may be a gross underestimate of the true numbers, as the study was based mainly on workers over 50 and already disabled.

Another variation of rapidly progressive silicosis results from exposure to diatomite, a non-crystalline silica used in filters, insulating materials, absorbents, and polishes. It produces a very fine dust capable of causing severe illness. *Shaver's disease* is a similar, rapidly progressive disease caused by inhalation of bauxite fumes. These fumes are emitted by electric furnaces in the production of corundum or refining of aluminum ores, and consist of extremely small particles (0.02–0.05 microns).

Coal dusts. Exposure to soft-coal dust causes *simple pneumoconiosis*, or *black lung*. Soft (bituminous) coal contains little silica. Large amounts of the black dust are deposited in the lungs, especially at the end of the small air tubes just before they enter the air sacs. This peculiar location and the properties of the dust itself give the disease the characterestics both of bronchitis and emphysema and of silicosis. Like emphysema, it shows up late or not at all on chest X-rays and in most simple lung-function tests. Like silicosis, it leads to lung scarring, which makes the lungs stiff and may lead to heart strain.

"Complicated pneumoconiosis" results when the small scars of simple pneumoconiosis join together to form large masses of scar tissue. These masses, which are stony hard, may occupy more than one-half of the lung. As the scars pull together, they rip the air-sac walls behind them and cause severe emphysema. Respiratory infections, and especially tuberculosis, may be a fatal complication of black lung. Among workers exposed to coal dust, smoking cigarettes appears to make black lung more frequent and more severe.

Anthracite, or hard coal, which contains more silica,

causes a lung disease more like silicosis. It shows up on X-rays fairly early and leads to symptoms of respiratory disease only years later. Large scars taking up almost the whole lung are a particularly common effect of hard-coal dust.

For many years, doctors thought the amount of lung scarring or disease that resulted from coal dust depended on the exact silica content of the dust. But ample information shows that silica-free coal can produce both the very small scars and the very large ones. Some doctors, many of whom work for the compensation insurance companies or the coal companies, still do not believe that bituminous coal causes any illness. This shows that doctors are not immune from bias in the interpretation of scientific facts, especially when the facts have economic consequences for major industries.

The standard for coal dust, under the Coal Mine Safety Act of 1969, is 3 million particles per cubic foot of air. However, the Bureau of Mines has not conducted enough inspections to ensure that this limit is maintained, not even the weekly spot inspections of "especially hazardous mines" required by law. It has either not given fines, or has failed to collect on fines it has given. With this sort of enforcement, it is not surprising that coal miners are dying from accidents at a rate of approximately one man every other working day, a rate higher than that of 1969, before the Coal Mine Safety Act went into effect. Young miners exposed to coal dust in mines where inspectors never come will develop black lung that will show up at least 10 years from now.

Carbon black is produced by burning creosote oil, tar oils, or petroleum distillates in a shallow iron pan and then collecting the resulting soot. Slight variations in the process produced **channel black, furnace black,** and **acetylene black.** In England the word "carbon black" is used for the same products as the word "soot." (Most of the research on the health effects of this dust comes from England.) In this country, "carbon black" refers to the specific product of the

incomplete burning of natural gas or oil: essentially pure carbon, containing less than 1 percent mineral impurities and *no* silica. Exposure to large quantities, as in the manufacture of rubber or inks or the production of carbon black itself, causes pneumoconiosis identical to that produced by soft coal, with emphysema, abnormalities of the small air tubes, and lung scarring that may lead to progressive massive fibrosis.

Graphite, also called plumbago, has numerous industrial uses as a lubricant, a conductor of heat and electricity, and a carbon carrier in steels. Natural graphite can contain from 2 to 10 percent silica after it is freed of the rock from which it is mined. Synthetic graphite contains less than 1 percent silica and often none at all. Pneumoconiosis can result from both natural and synthetic graphite. The illness and the medical findings are similar to those found in black lung. Synthetic graphite and graphite with low silica content produce lung disease like that caused by soft coal. Graphite with higher silica content produces a disease more like silicosis.

Asbestos is a fibrous mineral that, like cotton, can be made into thread and cloth. Unlike cotton fibers, its fibers are as strong as piano strings. It is virtually indestructible—heatproof, fireproof, and resistant to most chemicals. Because of these properties, it has found its way into more than 3,000 products, from potholders and children's toys to welding rods and industrial and household insulation. No factory or home is without asbestos, and almost no group of workers is without potential exposure.

Asbestos fibers are extremely fine. Fibers that can be seen with the naked eye or under an ordinary light microscope actually consist of thousands of smaller fibers called fibrils. It takes a specially powerful microscope, called an electron microscope, to magnify enough to make individual asbestos fibrils visible, as they are so small that there are 1 million of them to an inch. (For comparison, there are approxi-

mately 630 human hairs, side by side, to an inch.) When such very fine fibers get into the air, they float like water vapor and never settle. They are not trapped by the mucus or hairs of the nose or air passages, and can enter the air sacs of the lungs without any hindrance. Based on the usual method of dust counts, which measure only fibers larger than 5 microns and leave uncounted the majority of smaller and more dangerous fibers, it is estimated that in an 8-hour day, at the legal limit of 5 fibers per cubic centimeter of air, a person breathes 15 million fibers. Many of these remain trapped in the lungs. Once they are inside the body they remain as indestructible as they were outside it. An asbestos fiber you breathe when you are 18 years old may stay in your lungs until you die.

Although the body's mechanisms cannot destroy the asbestos fiber, they try very hard to isolate it. Over a period of 10 to 20 years individual fibers or groups of fibers may be surrounded with a thick wall of scar tissue. When these scars form in the air-sac walls, the lungs become inelastic, hard and contracted, smaller than normal. This means they hold less air and the body must work harder to fill them. The heart must work harder to pump blood through them. Since the scars are situated in the delicate air-sac walls, they block the transfer of oxygen to the bloodstream. Low blood oxygen and decreased lung volume are what doctors find in a patient with asbestosis. The main symptom is shortness of breath. Frequent respiratory infections are an additional problem, as the slightest infection can cause severe shortness of breath and even death in a person with asbestosis. Cigarette smoking appears to increase the frequency and severity of asbestosis.

Extensive studies have been made of asbestosis in asbestos-insulation workers, most of whom spend only about half their working time handling asbestos. This is a low exposure compared with workers who handle asbestos all the time. In spite of this "low" exposure, 1 out of every 10 asbestos-

insulation workers dies of asbestosis. Occupational groups that may have generally higher exposures to asbestos dusts include shipyard workers, asbestos-manufacturing workers, and asbestos-textile workers. The British Threshold Standard for asbestos is 2 fibers per cubic centimeter. The American standard is 5 fibers per cubic centimeter, to be reduced to 2 in 1976. There is already scientific evidence that these standards are inadequate to protect workers from asbestosis.

Asbestos dust has another, even more serious effect: it causes body cells to turn cancerous. Scientists do not know exactly why this happens. The reason may be the long residence of the dust in the lungs, or the ability of asbestos to adsorb other harmful chemicals onto its surface. The surface of asbestos fibers may hold carcinogenic (cancer-producing) chemicals in contact with the body's cells, which become malignant many years later. Workers develop cancers after at least 20 to 30 years have elapsed.

Asbestos can cause cancer in several parts of the body. *Mesothelioma* is a cancer of the membrane lining of the chest or abdomen. Normally this membrane is microscopically thin and acts to lubricate the walls of the chest and abdomen so that the lungs and intestines can move without rubbing or friction. In mesothelioma the lining becomes thickened and the chest or abdomen fills with fluid. Eventually the cancer overgrows the body, the way a weed overgrows a garden, and kills the victim. There is no known cure, and the cancer usually cannot be removed surgically. Mesothelioma is believed to occur only after exposure to asbestos. Prior to the introduction of asbestos into our society at the turn of the century, mesothelioma was so rare that it was unheard of by most doctors. Then the only cases appeared to result from exposure to natural deposits of asbestos, or to asbestos-rich soil in farming country. Mesothelioma can result from small doses of asbestos, well below the current threshold limit. There are reports of mesotheliomas

resulting from only one day of work in a shipyard, or developing in asbestos workers' wives who were exposed when washing their husbands' work clothes. Insulation workers get mesothelioma whether they smoke or not. One in 10 asbestos-insulation workers dies of mesothelioma.

Asbestos also causes cancer of the air tubes in the lungs. Lung cancer occurs mainly in asbestos workers who also smoke cigarettes. An asbestos worker who smokes has 92 times the chance of dying of lung cancer that a person has who neither smokes nor works with asbestos. One out of every 5 asbestos workers dies of lung cancer, and about 1 out of every 30 smokers dies of it. Cancers of the intestinal

Table 16. CAUSES OF DEATH AMONG ASBESTOS-INSULATION WORKERS

CAUSE	PERCENTAGE OF TOTAL CAUSES OF DEATH
Cancer, all types	40.4
Lung cancer	17.3
Mesothelioma	3.3
Cancer of the intestines	11.1
Other cancers	7.7
Asbestosis	5.5
All other causes	54.1

tract are also common in asbestos workers. They have about 3 times as many cancers of the stomach, large intestine (colon), and rectum as the general population.

Because asbestos is so common and so hazardous, special methods of preventing exposure must be used. The important and necessary goal of these methods is that *no* asbestos

dust should be released into the work or general environment, and none should get into anyone's lungs.

One way to prevent a harmful substance from causing disease is not to use it. Numerous substitutes for asbestos have been tried. Each has its own hazards. Some are touted as harmless, but they have been in use for too short a time for long-term effects to be seen. A general rule seems to be that any substance with the properties of asbestos, any substance as strong and indestructible, does not belong in your lungs and will do harm if it gets in. Substitutes now in use are fibrous glass, rock wool, sodium silicate, and polyurethane. The rules for handling fibrous glass are identical to the rules for handling asbestos. The British have experimented with asbestos-free calcium silicate block and pipe covering; this substitute is not generally available in the United States.

Modifications of asbestos may prevent some dust exposures. Various coatings have been designed to keep installed asbestos—or fibrous glass—from flaking. These should be used on all asbestos blocks, and especially on the asbestos linings of air-conditioning ducts. (Contamination of the air in urban office buildings from asbestos-lined ducts is being studied in several cities.) Dust suppressants are also used in asbestos cloth such as that in insulation blankets. (Fibrous glass has been substituted for asbestos as the filler in the blanket.) These blankets are not only cleaner to install but also cleaner to rip out.

Work practices specifically designed to release little or no dust into the air have been introduced for processes requiring asbestos. Their use for all operations involving asbestos (or other hazardous substances) will allow workers to control the safety of their own workplaces, and free them from dependence on all-too-infrequent inspections. If the correct, safe work practices cannot be followed, the job should not be done.

Isolation of a hazardous process is an important way of

limiting the number of people exposed Isolation of asbestos processes has an added avantage: it puts most of the work in shops equipped with proper ventilation and with tools designed for the special problems of asbestos. Shops can be equipped with strong general ventilation and hoods that even the best-ventilated field equipment cannot rival. Prefabrication can be done in such shops, or in the factory from which the asbestos supplies come. (The factory, of course, is really a giant shop and must also have properly designed and functioning equipment and ventilation.) Prefabricated blocks can be shipped, wet, in plastic bags labeled with the exact area where they will be used. Insulation blocks can be bought prescored, or scored in the shop with a properly ventilated gang saw. Pads and blankets for valves and machinery can also be made up in the shop. The fibrous glass or asbestos-cloth blanket is unrolled, wetted, and rerolled. Thoroughly dampened by this procedure, the blanket is then cut to shape in a well-functioning lateral-draft hood.

Repair work can also be done in the shop. Convenient-sized sections of pipe assembly can be brought to the shop, the insulation stripped, the pipes or insulation fixed, and new or repaired lagging put on. Only small repair jobs should be done at the job site.

Unfortunately, all work cannot be brought to the shop. Demolition and some removal operations must be performed on the job site. These operations too must be isolated, both from other workers and from the general community. Other workers must be kept off the job site during the operations. Trade crafts not directly involved in the rip-out should not be scheduled to do any work while the area is contaminated. The surrounding community can be protected by plastic tenting that completely encloses the work area and is equipped with a portable industrial vacuum cleaner to create a negative-pressure exhaust ventilation within it. All of the dust liberated within the tent stays

there, because of the inward pull of air created by the exhaust system.

The spraying of insulation cement must also be isolated on the job site. Many cities are now abolishing the use of asbestos-containing cement for spraying operations. However, work rules for the use of substitutes such as fibrous glass are the same. Only materials thoroughly mixed with water should be used. Mixing must be done in an enclosed system, such as a plastic bag with a valve for a hose. Cement shot dry from the nozzle of the sprayer and then passed through water onto the steel is dangerous because it sprays the entire workplace. It should not be used The spray area should be sealed off from the outside and from the rest of the building with large plastic or plastic-coated tarpaulins. The tarpaulins must reach through window areas into the building to prevent any loss of hazardous dust at the bottom of the enclosure, and must seal off elevator shafts and stairwells. No other workers should be in the area while it is contaminated. During spraying operations, air levels of asbestos may be higher than 100 fibers per cubic centimeter, and the sprayer must wear a powered air-purifying or air-supplied respirator. Wet cement that falls on the floor should be immediately shoveled or vacuumed up and disposed of.

Large power tools, such as band saws and gang saws, can be equipped with good local ventilation. The usual system for a fixed installation shop is a high-volume exhaust system that draws the dust into a baghouse. Special tables for cutting asbestos have perforations, and a down-draft pulls the dust down through the holes into a disposable container. Larger pieces of asbestos debris go into a plastic bag at the side of the table.

Some of this equipment has been successfully modified for field use. Smaller power tools, such as saber saws or rotary saws for cutting cement board, can be fitted with an industrial vacuum cleaner that creates a high-airflow, low-volume draft leading to a bag. The air stream can be moved

past the saw blade at a rate three times faster than the saw blade moves.

One word of caution: these ventilated power hand saws are not as safe as the larger ventilated shop tools, and should be used only for those jobs that cannot be brought to the shop. They should never be substituted for band saws, which can be attached to dust collectors of much greater efficiency. The perforated down-draft worktable can also be operated on the job site with an industrial vacuum cleaner creating the draft.

Whether such dust collectors are installed on power equipment or are part of hoods or of a general ventilation system, they must be tested with dust counts to see if they are safe. If they are found to be operating properly, a pressure gauge can be installed on the exhaust duct to indicate whether adequate ventilation is being supplied for each job. All the workman has to do then is see if the pressure as shown on the meter is at a safe value. If it is, the job is being done in a safe environment. If not, the job should wait until the exhaust equipment is working properly.

Asbestos cement can be prepackaged in convenient-sized plastic bags with a valve for the nozzle of a hose. Water is introduced and the cement is kneaded right in the bag. (If the water is introduced the night prior to use, no kneading will be necessary.) Very little dust is liberated by this procedure.

Housekeeping is another important part of preventing exposure. Good housekeeping begins before the job starts, with the laying of plastic dropcloths. At the end of the day, all debris can be wrapped up and thrown away. In between, the smaller dust should be vacuumed up with industrial vacuum cleaners into strong, single-use inner bags that can be removed and sealed. Asbestos or other harmful dusts should never be swept up by hand. Cleanup can be performed either by the workers doing the job or by special cleanup crews.

Disposable work clothes, made of material such as resin-impregnated paper, are recommended. No one else has to handle them, and they can be placed in plastic bags at the end of the day. They help prevent the spread of contamination outside the work area. The clothes should be removed and thrown out before leaving the work area, or in a special "dirty" locker room, so that street clothes do not come in contact with them. In field operations, the work van can have a "clean" locker room for street clothes and a "dirty" one for work clothes. Asbestos brought home on work clothes has been responsible for disease in wives and children of workers.

All these special procedures are to be used in addition to the general procedures of industrial hygiene discussed in Chapter 9. Safe use of a substance as harmful as asbestos shows that both simple and sophisticated techniques can be developed and applied to protect workers.

Mineral wool, rock wool, and fibrous glass are synthetic materials (silicates) with properties similar to asbestos, which makes them both useful and harmful. There are over 70 varieties of fibrous glass. Since diseases resulting from these substances probably take a long time to show up, and the substances have only been in widespread use for 10 to 15 years, it is not surprising that little disease due to them has actually been reported. Studies on animals, however, show that they can cause lung scarring and also some cancers. This should be warning enough. Fibrous glass should be handled as carefully as asbestos and the same protective measures applied to its use. It also causes skin irritation, and may cause acute bronchitis and asthma if inhaled.

Talc is very similar to asbestos. Its particles are mostly flat, like little plates, but may also be fibrous. It is usually found in deposits mixed with asbestos, and it is not clear if the health effects of talc are from the asbestos or from the talc itself. (American cosmetic talc tends to have less mixed asbestos than French.)

The effects of talc are similar to those of asbestos, although they have not been as carefully studied. Talc is also a very fine dust, with some particles so small that they cannot be seen with the usual optical microscope. These smaller particles, the most dangerous ones to the body, are not counted by routine dust counts. Once in the lungs, talc stays there and cannot be removed by body mechanisms. The effects show up 20 or 30 years after exposure.

Talc causes lung scarring similar to that in asbestosis Mesotheliomas have also been reported, and cancer of the lungs and the intestinal tract may also occur, though there have been too few studies for definite proof. Talc has found its way into some foods. Scientists, concerned that talc may cause intestinal cancer, have requested that talc additives in foods be banned by the Food and Drug Administration.

Beryllium causes lung, skin, liver, and kidney disease, however, the effects on the lungs are the most prominent and disabling. *Acute berylliosis* is a severe, pneumonia-like lung inflammation that can occur after brief, intensive exposure, or prolonged exposure to low concentrations. It may result from extremely low exposure in some workers, depending on individual susceptibility. The typical symptoms are cough with phlegm that can be bloody, severe shortness of breath, and weight loss over the first few weeks. The chest X-rays may be normal initially, even though the worker is seriously ill. The lung inflammation may be severe enough to block oxygen transfer into the blood. This disease is frequently fatal, and those who recover may have sustained permanent damage to the lungs.

Chronic berylliosis may develop after 1 year or even up to 20 years of exposure. Chest X-rays may be abnormal for many years before symptoms begin. At first the symptoms may be mild, slowly progressing to a more severe and disabling illness in which chronic inflammation makes the lungs stiff and unable to transfer oxygen to the bloodstream. The heart is affected by years of straining to pump blood

through the inelastic lungs. The final stage of the disease is similar to asbestosis.

Iron, barium, tin. *Siderosis* (iron), *baritosis* (barium), and *stanosis* (tin) are caused by compounds such as iron oxide, tin oxide, and barium sulfate, which give rise to dusts that are insoluble in lung tissue but not irritating. Because these dust deposits are visible on chest X-rays, they are included under pneumoconiosis, but they do not cause symptoms and apparently do not produce disability.

Organic Dusts

Organic dusts may contain plant fibers or fungi, bacteria, and their products. They act upon the lungs in a different manner from mineral dusts. The diseases they cause are frequently the result of an allergic reaction to the inhaled dust.

For example, the disease called *byssinosis* or *brown lung* is caused by an allergy-like reaction to cotton dusts (and also to the dusts of flax and hemp). The severity of reaction depends on the susceptibility of the individual worker to the allergy-producing qualities of the dust.

Although a susceptible cotton worker may develop this reaction at any time in her or his working life, it usually begins after only a short exposure. The first reaction generally occurs when the worker returns to the mill after a few days off—a weekend or a vacation. Because of this pattern of reaction, the initial stages have been called "Monday fever." The first reaction is like an asthma attack, with shortness of breath, severe air hunger, chest tightness, and a dry cough. These symptoms are caused by the narrowing of the medium and small air tubes and by the accumulation of excessive amounts of thick mucus in the tubes. The air has difficulty moving *out* of these narrowed tubes, and makes a wheezing sound. A single such attack is extremely frightening and uncomfortable, but causes no permanent lung damage. Repeated attacks, however, may damage the lungs.

In the initial stages, these attacks occur only on the first