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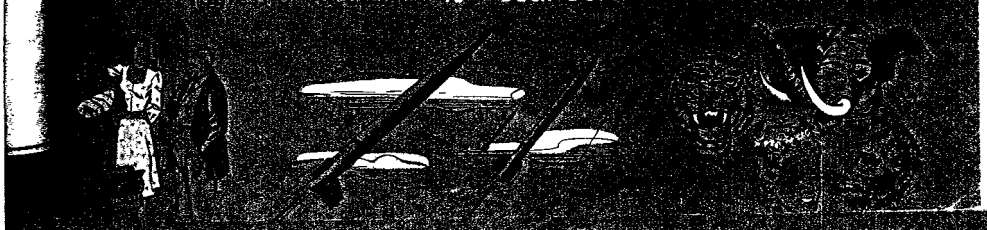
what this changing world means to you



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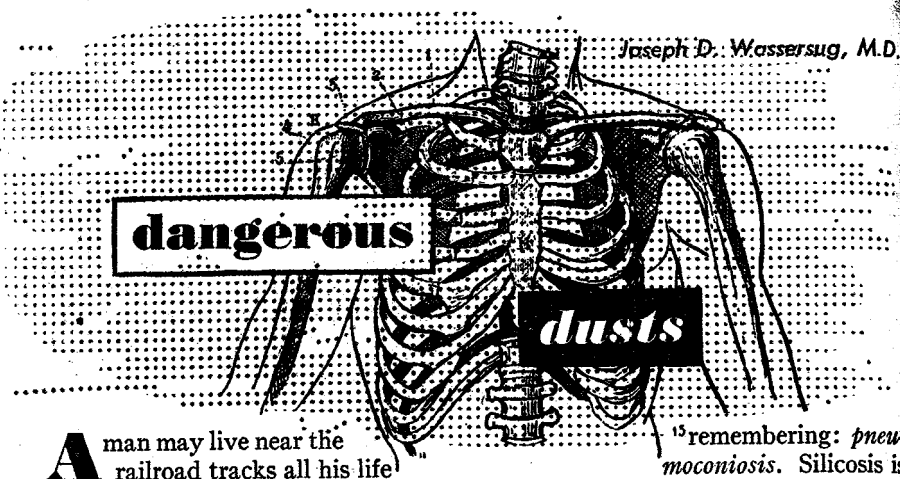
dangerous dusts



A man may live near the railroad tracks all his life and inhale the coal-black soot of the passing trains day after day without any damage to his lungs.

Carbon from train smoke may make his lungs black as pitch and yet they will continue to function normally. He may live till the age of eighty or ninety only to die of some disease of his heart or intestines or some other organs. In spite of their blackness, the vitality of his lungs may be spared.

On the other hand there are many instances where coal miners—who are equally exposed to a carbon-laden atmosphere—have succumbed to serious impairment of their lungs at the age of forty or fifty. If the lungs of these men are examined post-mortem, they too will be found to be heavily stained with coal-black pigment.

Why do these men die? What is the difference between the carbon at the railroad tracks and the carbon in the coal mines? The answer can be found in one word—*silica*. It is not the carbon that kills the coal miner, it is *silicosis*.

Doctors have a name for these dust diseases of the lungs and, although it is a tongue-twister, it is a name worth [15]remembering: *pneumoconiosis*. Silicosis is but one of them. Over one million workers in the United States today are exposed to one sort of dust or another. Pneumoconiosis is a major hazard in many industries.

Silicosis is the most common—and perhaps most lethal—form of pneumoconiosis. But don't think that it is a disease exclusively of coal miners. No indeed. Possible victims of the disease are those persons who are engaged in mineral grinding, rock quarrying, stone cutting, tunnel driving, fire brick manufacturing, foundry work, and the manufacture of tile and abrasive soap powders.

There is hardly a state in the Union in which the problems associated with this disease have not presented themselves. In the Coeur d'Alene mining district of Idaho, in the fire brick industry of Kentucky, in the anthracite coal mines of Pennsylvania, and in the granite industry of Vermont, silicosis is a serious problem.

The dangerous dust that causes silicosis is silica—ordinary sand! Chemically it is represented by the formula SiO_2, which simply means silicon dioxide in chemist's shorthand. But here

again we run into some problems. The beachcomber doesn't get silicosis and he may have sand blown at him all day. The windows at a Coast Guard station may be etched and scratched by particles of sand carried by the wind and yet silicosis is not a health hazard of the Coast Guard.

What is the difference between the sand inhaled by the granite cutter and the sand inhaled by the beachcomber? The answer is size.

The sand (silica) in the stone quarry is very fine, like dust; the sand on the beach is coarse. Furthermore, and equally important, the man working in a stone grinding or polishing shed or in a mine is working in a closed space where the dust particles may accumulate in the air to astounding proportions. Unlike the beachcomber he does not have the advantage of pure, clean air, swept in from the ocean.

Particles larger than four-millionths of an inch in diameter cause no harm since they are actually trapped within the nose and throat and expectorated. It is only when the dust particles are one-third of this size or smaller that they get by the natural protective barriers of the nose and throat and enter the lungs themselves.

On the basis of repeated surveys and examinations it has been discovered that there have to be about five million particles of silica dust per cubic foot in the air—or more—before it can be regarded as a health hazard. If the atmosphere is less heavily laden with dust, a normal man can apparently work at his job for years without impairing his health.

The dangers from silicosis come from two sources—from the disease itself and from a complicating infection with tuberculosis. Silica irritates the lungs, causing scarring which interferes with the normal passage of air in and out of the lungs. This results in shortness of breath, coughing, and sometimes, feelings of weakness and fatigue. If the disease is extensive enough it may cause progressive disability and death.

For some unknown reason, lungs that are affected with silicosis are more susceptible to TB. Statistics from various sources, as a matter of fact, indicate that about 60 per cent of the patients with silicosis develop TB as a complication. It is much more difficult to cure patients with silico-tuberculosis than uncomplicated cases. Besides, for them, the symptoms are usually more severe, the cough is worse, the expectoration more annoying.

Until recently the only method that doctors had for controlling silicosis was dust control. When possible "closed" spaces were eliminated. In other cases, blowers and vents rid the atmosphere of the dangerous dusts. Masks and other protective devices were invented.

Doctors found too that by routine x-ray examinations they could detect evidences of the disease—especially silico-tuberculosis—before it had advanced too far. But, from a strictly medical standpoint, there was very little that could be done to cure the patient of his disease or arrest its terrible progress. There was no *specific* medicine for the victim of silicosis.

And then, in 1937, a group of Cana-

dian researchers hit upon a brand new idea. They found that if they made experimental animals inhale finely powdered aluminum, it would protect them against silicosis. At the same time, in this country, Dr. Leroy U. Gardner and his colleagues were working on the same problem at the Saranac Laboratory using aluminum *hydroxides* or aluminum *hydrates* instead of metallic aluminum.

"Quite independently," says Doctor Gardner, "we discovered that hydrated alumina would also protect animals against quartz."

Preliminary tests conducted on human subjects confirmed the fact that aluminum had a beneficial protective action. This was the first step in a long series of experiments that are still under way, in an effort to demonstrate that aluminum has a curative as well as a protective action against this terrible dust disease.

At present it seems as if both powdered metallic aluminum and aluminum hydroxide are equally effective. Statistics released not long ago by Dr. J. W. G. Hannon reveal that of 143 disabled silicotic workmen, 135 improved with aluminum treatment. Of 104 additional workmen whose x-rays showed silicosis but who were not as yet disabled by the disease, 93 improved.

In the disabled patients, treatment with aluminum brought about a decrease in the cough, less shortness of breath, and a diminution of chest pain.

Why aluminum protects against silicosis is not known for certain. There is some evidence that the aluminum coats the dangerous silica particles with an infinitesimal layer of aluminum and thus prevents the irritation of the delicate lung tissues. Harmless—so far, no serious toxic effects have been noted—aluminum stands as a protective barrier between dangerous silica and the lungs.

Aluminum, even if it does prove to be effective, is no substitute for dust control. Industry should continue to make every effort to provide the worker with a dust-free atmosphere or, at least, one that does not contain excessive amounts of dangerous dusts.

But silicosis is only one form of pneumoconiosis. Another bad one is *asbestosis*. The mineral, asbestos, from which we get so much of our insulating materials, does not contain free silica, but it is rather a complex *silicate* containing iron, magnesium, and calcium.

In cases of asbestosis, if the lungs are examined under the microscope, tiny particles of asbestos can be easily identified in the lung tissues. The asbestos scars up the air passages, causes them to narrow and shrink in size to a fraction of their normal calibre. The result is difficulty in breathing, shortness of breath, and disability.

In one state alone—North Carolina—there are about 500 persons engaged in the asbestos industry, and according to one authority, many of them have already been found to be affected.

Silicosis and asbestosis both have a marked tendency to get worse in time even if the person is removed from exposure to them. Of course the amount of damage done varies from person to person and with the duration of the exposure. Some men can work in

mines all their lives without developing any disease; others will get a bad case of silicosis or asbestosis after a few years. The reason for this variability is still a medical mystery.

Often, especially during the war, electric arc welders were seen complaining of irritation of their nose and throat. A few of the men believed they had been "poisoned" by the fumes or had contracted TB. In most of these cases, x-rays of the lungs showed absolutely nothing abnormal.

As soon as these men were able to take a brief vacation or were able to get adequate rest or shorter working hours, their symptoms disappeared. The welding fumes had merely caused a *local* irritation of their upper respiratory tract but no real damage to their lungs.

On the other hand, there were other welders whose lungs did show abnormal changes on the x-ray. When their chest films were studied it looked almost as if someone had thrown a handful of sand at them or had peppered them with a very fine buckshot.

These men had "*siderosis*"—a condition caused by inhaling fine particles of *iron*. These particles had become lodged within the lungs. That was why the lungs had a "peppered" appearance. But actually this excess iron had not made any of these men sick.

Iron, unlike silica or asbestos, does not irritate the lungs, does not cause them to scar up. In this way it is more like carbon. If the welding is done in open rooms, the fumes do not become concentrated enough to cause changes within the lungs. If the welding electrodes are coated or if the welders employ protective masks, siderosis is prevented.

But even if siderosis does occur it does not lead to progressive disability. Recently, for example, Dr. O. A. Sander of Milwaukee reported that a group of patients with siderosis whom he had followed for years showed very little change in their x-rays during that period. Apparently none of them had *suffered* as a result of the iron within their lungs.

Cotton dust, strangely, may be irritating to the lungs and may be responsible for some of the respiratory ailments of cotton mill workers. "Cotton-mill fever," as the disease is called, first begins as a mild respiratory infection.

A curious point about the disease is that patients recover somewhat over the weekends, when they are not employed, and that symptoms are aggravated on return to work on Mondays.

For this reason the disease has also been called "Monday-morning fever." The cough grows worse, and there is progressive disability unless the patient changes his employment to some out-of-door work. Like so many of the dangerous dusts, cotton dust disease can be better prevented than it can be treated.

There are some strange dusts that can make a person sick in ways that are still baffling and inexplicable. Such a disease is the one that is called "*bagassosis*."

Bagasse is the product remaining after sugar is extracted from sugar cane. This material is commonly baled and if not used immediately may re-

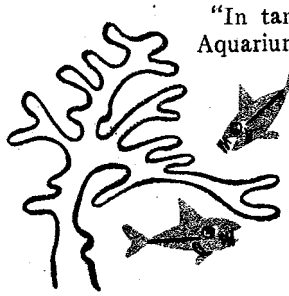
main exposed in the field for some months before being converted, by processing, into insulating and acoustic board. And it is the workers who are employed in breaking these bales that may become stricken with the disease.

Since the first case of bagassosis was reported in medical literature only as recently as 1941, scientists have not yet had an opportunity to determine the exact nature of the illness. First it was thought that it was a fungus contaminating the dust, and not the dust itself that was responsible. Other researchers have believed that it was due to allergy.

According to the latest bit of evidence from Drs. W. A. Sodeman and R. L. Pullen of New Orleans, it is "spicules" of the dust that get into the lung to cause the disease. Tiny slivers or "spicules" of bagasse can, apparently, be very irritating to a person's lungs.

In spite of ample provisions from Nature that guard our lungs from injury by dangerous dusts, these provisions are not always adequate. In the highly industrialized and mechanical life of modern society it is not always possible to avoid these dusts and to breathe "pure" air constantly. Yet present-day industrial engineers and modern industrial physicians are increasingly aware of their responsibility for eliminating dangerous dusts from the atmosphere or of finding some effective treatments for those workers whose lungs are damaged.

COWARD KILLER FISH



The piranha fish is probably the most vicious and bloodthirsty creature of its size—some 6 or 7 inches usually. Piranhas have reduced a 400-pound hog to a skeleton in ten minutes. Their teeth will nick hard steel. They are called "the fish that eat men."

Yet this same piranha lives a life filled with terror, or whatever a fish feels in place of terror, according to Christopher W. Coates, curator of the New York Zoological Society's aquarium, in *Animal Kingdom:*

"In tanks of the New York Aquarium we have attempted to rear several Piranhas at a time, allowing them space far disproportionate to their size, but we have found that invariably, sooner or later, they tear each other to bits—or else some of them die of what appears to be fright. They have been found dead with no marks of fighting or injury.

"Even when kept in the same tank but separated by a sheet of glass, it is long before they learn not to dash into hiding as soon as another pokes his head from behind a stone or cluster of plants."