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DUST *in* INDUSTRY

The Economic and Hygienic Aspects of Silicosis

By ALBERT E. RUSSELL, M.D.¹

IT WOULD be difficult to estimate with any degree of accuracy the number of people in the United States who are engaged in dusty trades; in fact, with the present varied record systems of the different states, such an estimate would be practically impossible. This the author learned when he was preparing his paper for the International Silicosis Conference. However, an idea of the frequency of exposure to dust may be found from the statement that more than half a million workers are engaged in the coal-mining industry in the United States, and over 20,000 workers are employed in dusty occupations in New York City alone. It is evident that more people are exposed to dust, which constitutes the greatest single industrial hazard, than is generally supposed. Certain dusts contain poisonous elements, such as lead, arsenic, mercury, etc., which give rise to general conditions resulting from their absorption. This paper, however, will deal only with those dusts which are known to be direct factors in the production of pulmonary diseases.

Dust is possibly the oldest source of industrial hazard. Knowing as we do how dust is generated, we may assume that as far back as the stone age and farther, people have been affected by it. Certainly the Greeks, Romans, and Egyptians must have encountered this hazard in connection with their extensive construction work with stone. The harmful effects of dust are mentioned in Greek literature, but the Italian writer, Ramazzini, gave us in the 18th century the first detailed account of the action of this industrial factor upon the worker.

ECONOMIC ASPECTS OF SILICOSIS

The economic problem presented by silicosis is tremendous. The suffering and loss of life due to it cannot be adequately measured. In the gold-mining area around Johannesburg, South Africa, during the period 1911 to 1929, £11,208,015, or more than \$53,000,000, was paid in compensation alone. The costs of medical care, hospitalization, and legal proceedings further augmented this enormous figure.

Mines in Australia are reported to have been bankrupted by payment of silicosis compensation. The author knows of a company in our own country which has claims amounting to more than a million dollars for disability due to dust inhalation. There are other plants in the East which will be bankrupted if all the

claims which are filed against them are allowed. In most of the industrial states the number of claims filed has greatly increased in recent years.

It is clear that dust-prevention work, like any health-promoting activity, would pay many times its cost in money, good health, and efficient operation.

Strangely enough, only during the past 25 years has dust received much attention as an etiological factor of disease. The rapid development of industrialization within that period partly explains the impulse toward the study of this source of industrial hazard. The advances which have been made in all branches of industrial medicine have helped to center attention on dust and its effect upon the health, inasmuch as the number of workers in the dusty trades is larger than that of any other group exposed to a single industrial hazard; and an additional reason for increased interest in this subject is gathered from the study of mortality statistics, which reveal the fact that workers in the dusty trades have an excessive death rate from tuberculosis, as well as from other respiratory diseases.

The most notable studies of silicosis have been made in the gold mines of South Africa, where the disease, with its complications, has been most commonplace among the miners. It became necessary either to study the disease and devise methods for its control or else to abandon mining. Consequently funds, personnel, and clinical material were made available for the first time on a large scale. Investigators in South Africa have become foremost in this field of research; their scientific work is widely known and has served as a basis for legislation, compensation rates, and further study of the disease silicosis.

There are almost 300,000 workers in the South African mines. It was because of the excessive incidence of silicosis among the miners around Johannesburg that the International Labour Office of the League of Nations selected that city for the first International Silicosis Conference.

In the United States the first study of the effect of dust was made by Dr. A. J. Lanza, an officer of the Public Health Service serving with the Bureau of Mines. His observations, made on miners in the Joplin, Mo., district and published by the Bureau of Mines and Public Health Service, have been confirmed by subsequent studies. Agencies which have done and are now doing investigative work are the U. S. Public Health Service, the Bureau of Mines, and the Universities of Harvard, Yale, and Pennsylvania. Private persons have also contributed valuable information on silicosis.

The word "pneumoconiosis" is of Greek origin

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and means "dusty lungs." It is an inclusive term and is being used now more than previously with a designation as to the type of dust responsible for the pathology. There are few instances where a pure dust, i.e., one unmixed with other elements, occurs in industry.

The intelligent consideration of the relation of dust to the etiology of pulmonary disease must include the following:

- 1 Chemistry and petrography of dust
- 2 Condition of worker at beginning of exposure
- 3 Length of exposure, concentration, and size of particles
- 4 Effects produced.

The dusts most frequently met with—and, incidentally, the most harmful—in the industries are inorganic. It has been shown conclusively that silica, one of the most common dusts encountered, alone produces more permanent pulmonary damage than all other causes combined.

CHEMISTRY AND PETROGRAPHY OF DUST

In the earlier days all dusts were thought to be capable of producing serious damage. Subsequent investigation showed that this was not the case, and that no two types of dust produced the same results. Clinical and experimental work has proved that silica is the arch-offender. Therefore any paper on the subject of pneumoconiosis must necessarily be concerned principally with silicosis. The first theory relative to the effect of dust was that its harmfulness is due to sharp, angular particles. This theory has been disproved by several investigators. Gardner's work on animals showed that carborundum dust, a harder substance than silica, would not cause the massive pulmonary fibrosis that silica produces. The size of dust particles will be discussed later, but it might be said here that only the very small particles reach the alveoli of the lung, many of them as small as bacteria and of unimportant physical hardness. Collis² stated that, "Generally speaking, dusts are more injurious as their chemical composition differs from that of the human body or from the elements of which the body is normally composed, whence it follows that animal dusts are less injurious than others."

With this in mind, it may readily be seen that dust from calcium, carbon, etc., common constituents of the human body, would not be expected to produce the same effect as silica or quartz, an uncommon constituent in our make-up. Research has shown that silica in its free or uncombined state is capable of producing serious pulmonary damage, whereas in its combined state, as in silicates, it is relatively innocuous. Obviously, it is important when studying a dust to know the percentage of quartz or free silica in its composition. This is accomplished by petrographic analysis. The chemical analysis of cement dust indicates the presence of about 22 per cent of silica, while petrographic analysis shows a content of only about 1 per cent of quartz. Likewise, the chemical

analysis of Barre granite indicates the presence of approximately 70 per cent of silica, while by petrographic analysis only 35 per cent of quartz is found. It is not surprising that chronic conditions of the upper respiratory tract may be produced by the physical or mechanical action of dust and that possibly a chronic bronchitis may result, but only silica produces massive pulmonary fibrosis and a strong predisposition to infection with tubercle bacilli.

The theory that silica becomes soluble after remaining in the tissues has been generally accepted, and it was also agreed at the International Silicosis Conference that "there is experimental evidence that the solubility of silica in the tissues is an essential factor in the causation of silicosis." It is generally accepted that the silica particles are removed from the alveoli by the macrophages, which are sometimes called "dust cells," and are stored in the various parts of the lung. Apparently, the gradual dissolution of the silica stimulates an excessive production, with time, of fibrous tissue. Kettle³ contrasts this slow action of silica in the lung tissue with the more rapid reaction which occurs when silica is injected into the subcutaneous tissues, in which case an inflammatory reaction is produced and in a short time necrosis takes place.

It is also well known that tubercle bacilli grow well in a medium rich in colloidal silica. Gardner⁴ showed that guinea pigs inoculated with a low-virulent strain of tubercle bacilli and exposed to siliceous dust of known quantity and concentration developed clinical tuberculosis with a dissemination of the infection, whereas the controls inoculated with the same strain and dosage of bacilli failed to develop the disease to such an extent. Obviously, the effect of silica in the tissues aids in the growth and proliferation of tubercle bacilli.

Apparently, there is not the same effect in the action of other bacteria in the pulmonary tissues that is found in the case of tubercle bacilli. Likewise it has not been shown that the growth of tubercle bacilli is augmented by the presence of other types of dust. An excessive amount of other respiratory diseases can be accounted for by the lowered local resistance caused by the presence of dust and the excess of bacteria carried into the lungs by the dust itself.

CONDITION OF WORKER AT BEGINNING OF EXPOSURE

Reference has been made to the manner in which a tuberculous infection is augmented by the presence of silica. It follows that a worker having a tuberculous infection, either latent or active, will suffer more from the effects of dust than a person who is not so infected. It is highly necessary for workers to be examined thoroughly before being permitted to engage in a dusty trade. Experience with miners in South Africa has shown that a worker having a latent tuberculosis or one in whom such infection was not demonstrable, or if he was of the so-called "phthinoid" type, he developed

² E. L. Collis and Major Greenwood, "Health and the Industrial Worker."

³ E. H. Kettle, "Experimental Silicosis," *Jl. Ind. Hygiene*, vol. 8 (1926), pp. 491-495.

⁴ L. U. Gardner, *American Review of Tuberculosis*, December, 1929.

silicosis more rapidly and a tuberculous complication earlier than did the normal worker.

LENGTH OF EXPOSURE, CONCENTRATION, AND SIZE OF PARTICLES

The length of exposure to dust is evidently a great factor in the production of disability. It has been found that the occurrence of silicosis is in direct relation

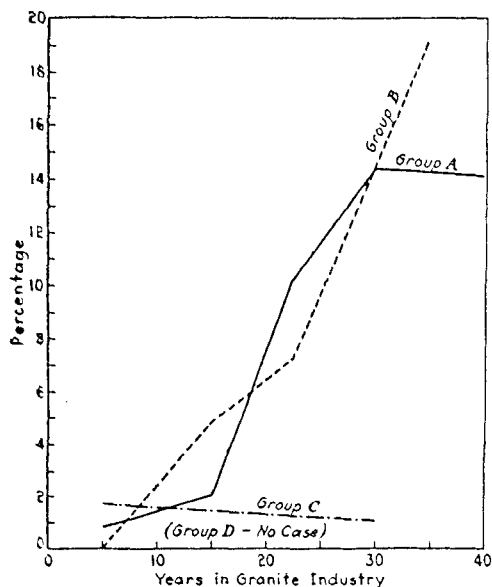


FIG. 1 PREVALENCE OF TUBERCULOSIS (ACTIVE CASES) BY LENGTH OF SERVICE AND DUST-COUNT GROUPS (Percentage of workers diagnosed on physical examination as having tuberculosis.)

to length of service and concentration of dust. This is illustrated in Fig. 1.

EFFECTS PRODUCED

In order to determine the effects produced by dust inhalation, a number of studies were made. These observations included a study of the sickness of workers with especial reference to respiratory diseases, physical examinations supplemented by X-ray and laboratory tests, and a study of the mortality records.

Morbidity. Prior to the discussion of morbidity rates, a brief reference will be made to the extent of dustiness to which groups of workers have been exposed. The principal information presented in the morbidity studies was taken from observations made on a group of granite workers exposed to a known concentration of dust. These workers were classed in four general groups according to intensity of exposure per cubic foot of atmosphere, as follows: Groups A and B, 30 to 60 million particles per cubic foot; Group C, 20 to 30 million particles per cubic foot; Group D, less than 20 million particles per cubic foot (Fig. 1).

The frequency of disability lasting longer than one week, exclusive of that from accidents and from respiratory diseases in specified industries, is shown in Fig. 2. The lower portion of the graph shows the incidence of

respiratory diseases; it may be seen that these rates are not in the same ratio as the total amount of sickness.

COURSE OF SILICOSIS AMONG WORKERS NO LONGER EXPOSED TO SILICA DUST

In the study of silicosis, one of the most important questions which has arisen is the health history of those individuals who have worked in the granite industry for a number of years and then left, to find occupations in other trades where there was no further dust exposure. It was possible during the course of the study at Barre to observe 24 such cases.

The latency of silicosis has been referred to by other investigators. The South African workers found that "a steady fall over a period of years in dust concentration is not associated with a corresponding fall in the silicosis incidence."⁶ Dr. Watkins-Pitchford also records cases of miners who had been exposed to dust for a long period of time, apparently sufficient to develop silicosis, but who went into the war apparently not silicotic. He asserted that such cases were liable to

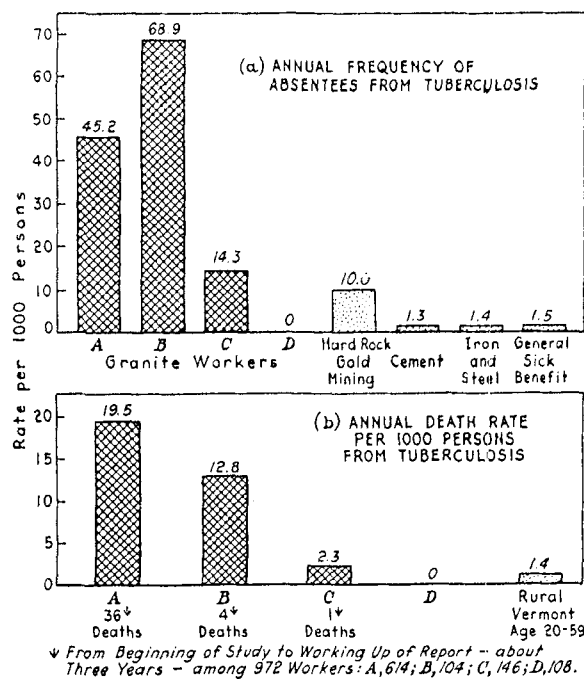


FIG. 2 (a) ANNUAL FREQUENCY OF ABSENCE FROM TUBERCULOSIS (EIGHT DAYS AND MORE). (b) ANNUAL DEATH RATE PER 1000 PERSONS FROM TUBERCULOSIS

develop tuberculosis. Dr. Pancoast presents a case of advanced pneumoconiosis in a quartz miner who had been exposed to dust for eight years. He had been out of the mining industry for 10 years, yet the X-ray showed extensive pneumoconiosis with irregularities of diaphragm, and by fluoroscope he found the diaphragm restricted on each side. There was, perhaps, a tuberculous infection intervening at the time.⁶

⁶ South African Institute Medical Research, Annual Report for year ending Dec. 31, 1925.

⁶ Henry K. Pancoast, "Roentgenological Studies and Other Fibrosing Conditions of the Lungs," *Analysis of Clinical Medicine*, July, 1923.

Britton⁷ reports two cases of workers who had been exposed to dust between seven and eight years. They changed occupations and had been away from siliceous dust for eight and nine years. They developed pulmonary symptoms and were found to be suffering from silicosis and tuberculosis. Tattersall⁸ also observed cases of latent silicosis in his studies: "Some of the men (rock drillers), moreover, had changed their occupation for various reasons quite apart from health, but in due course the inevitable dyspnoea came on. One man, for instance, worked eight years regularly with rock drills from 1906 to 1914, then joined the Army, was passed as A-1; but in spite of his open-air life, dyspnoea came on in 1918, and from then until his death six years later his illness was a typical case of silicosis."

MORTALITY

The influence of dust on mortality from tuberculosis is clearly indicated in the following table. New methods

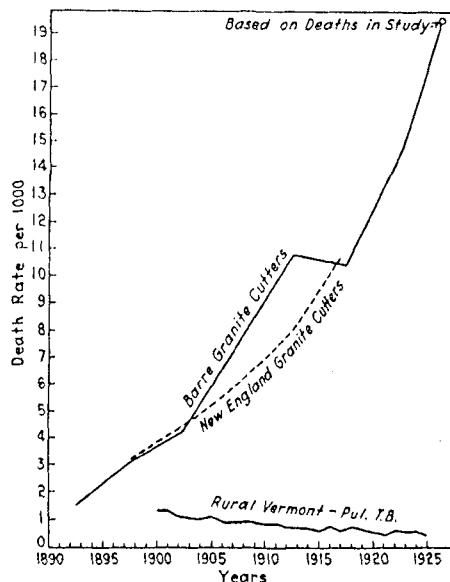


FIG. 3 TREND OF MORTALITY FROM TUBERCULOSIS AMONG GRANITE CUTTERS COMPARED WITH THAT IN RURAL VERMONT

of manufacturing stone which created excessive dust by the use of pneumatic tools, were introduced in the granite industry about the beginning of the present century, and the tuberculosis rate has increased rapidly with their use. The rate has risen in direct proportion to the length of time during which they have been employed, as follows:

1.5 per 1000.....	1890-1894
10.8 per 1000.....	1910-1914
19.5 per 1000.....	1924-1926*

* During period of our observations.

A consideration of the mortality statistics of Barre,

⁷ James A. Britton, M.D., "Silicosis, a Modern Factory Hazard," *Jl. Ind. Hygiene*, September, 1924.

⁸ Norman Tattersall, M.D., "The Occurrence and Clinical Manifestations of Silicosis Among Hard Ground Workers in Coal Mines," *Jl. Ind. Hygiene*, November, 1926.

Vermont, shows that there has been an excessive death rate from pneumonia and other respiratory diseases (tuberculosis excluded) during this period.

Fig. 3 shows the age curve of mortality of workers using hand pneumatic tools as well as that of males in rural Vermont. The rate of the latter may have been influenced by the fact that many disabled cutters move to

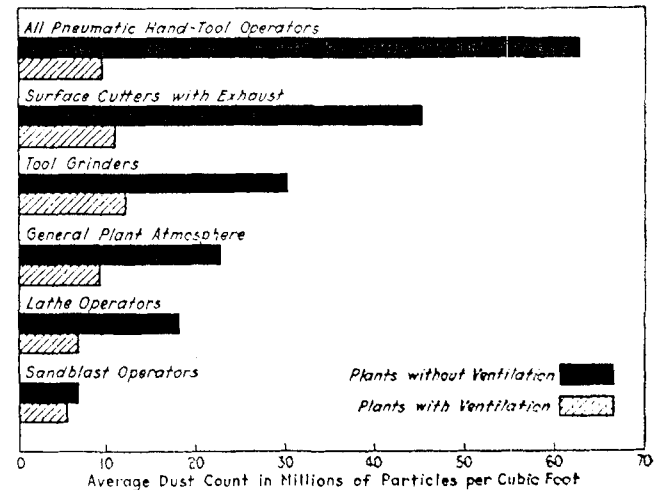


FIG. 4 SHOWING THE DIFFERENCE IN ATMOSPHERIC DUST CONDITIONS BETWEEN A GRANITE PLANT EQUIPPED WITH EFFICIENT LOCAL EXHAUST VENTILATION AND PLANTS NOT SO EQUIPPED

rural districts. However, the contrast between the two rates is great.

SUMMARY

Morbidity and mortality records indicate that workers exposed to dust have excessive rates of respiratory diseases.

Inorganic dusts produce more pulmonary disability than organic dusts.

Silica (SiO_2) of the crystalline variety (quartz) is the element in dust which produces the greatest pulmonary damage, and it is present in varying extents in most industrial dusts.

Silicosis is insidious in its onset and progress, and presents but few symptoms or physical signs in its early stages.

Silicosis is almost invariably complicated by tuberculosis after a number of years of exposure, which time depends upon the concentration of the dust and the percentage of free silica which it contains. Latent tuberculosis is usually activated after shorter exposure to siliceous dust, and has more characteristics of this disease in persons not in dusty trades.

Masks frequently give a false sense of protection. Removal of dust at its source is preferable (Fig. 4).

There has been a great reduction in the death rate from tuberculosis among the general population during recent years, while among workers in dusty trades there has been a great increase during the same time in the death rate from this cause.