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Monthly Life Insurance Department

JUNE 8, 1940

Underwriting Aspects of the Industrial Dust Hazard

By A. J. Lanza, M.D., Assistant Medical Director, and R. J. Vane, Statistical Bureau, Metropolitan Life

Address Before Medical Section of American Life Convention, Colorado Springs, Colo., June 6

OUR knowledge of health hazards resulting from exposure to various kinds of dust in industrial occupations is based on observation and clinical studies of workmen exposed to these dusts, and upon noting the results of the action of these same dusts upon experimental animals. The former method goes back for centuries and our earliest conceptions of the harmful properties of dust were based on first hand observation.

Many of these conceptions were erroneous. It is only in the past twenty years that anything like a comprehensive knowledge of dust diseases has been attained—due first to the perfection of the X-ray as a means of diagnosis, and the development of successful laboratory technique for conducting tests on experimental animals.

Now we have arrived at a point where intensive studies, both clinical and experimental, conducted in this country and in other countries, have resulted in a crystalization of our ideas. In the English speaking world, the outstanding contributions to our knowledge have come from the South African Institute for Medical Research and the Miner's Phthisis Prevention Committee in Johannesburg; from various official agencies in England and Australia, in Ontario, Canada, and in this country, especially the United States Public Health Service, the United States Bureau of Mines, and the Saranac Laboratory.

In presenting this discussion of industrial dusts, their effects on workmen exposed to them, and the bearing that this subject has on life insurance underwriting, we have attempted to summarize what should be the insurance attitude, based on the present state of our knowledge. Briefly, the present concept regarding industrial dust hazards is as follows: Organic dusts, of which the dust arising in textile operations is an example, do not cause any specific pulmonary disease. There is no clinical nor experimental evidence to the contrary. Coal, which is an organic substance, is considered in connection with mineral dusts. Organic dusts at times may cause an allergic reaction and also irritation of the upper air passages. They do not cause protracted disability nor death. In this classification may be included cotton, wool, silk, sugar, flour, starch, wood, tobacco, and leather.

It is true that some occupations in which is involved exposure to organic dusts manifest a tuberculosis mortality rate higher than the average. Formerly, it was thought that this undue prevalence of tuberculosis was due to the occupational dusts but the dust studies of recent years have demonstrated that, mostly, organic particles are too large to penetrate to the pulmonary tissue and when they do, they are absorbed without producing any specific inflammatory reaction. Nor is much credence given nowadays to the idea that organic dust particles convey tubercle bacilli to the lungs and thus cause tuberculosis.

Metallic Dusts

Here the situation is not unlike that of organic dusts. The prevalence of pulmonary disease among workers in certain occupations exposed to metallic dusts gave rise to the theory that metallic dust was a fruitful source of tuberculosis. Such diseases as "grinder's rot" were recognized clinically long before the era of modern diagnosis and laboratory research. Metallic particles in the form of fumes may cause such reactions as zinc chills or brass founder's ague, which are more in the nature of an allergic reaction. The pulmonary disease which afflicts grinders and buffers is silicosis and not in any way a result of exposure to metallic dust. Metallic dust particles are mostly large and heavy (that is, comparatively speaking), consequently do not penetrate to the lung, in many instances, and when they do they are either absorbed or remain fixed and inert in the tissues. From an underwriting standpoint then, the hazard of metal dust is the hazard of such silica dust as may be associated with it.

Coal

For many years there has been a belief that coal miners had less tuberculosis than the average for wage earners and, on the other hand, miner's asthma has been recognized as an occupational disease of coal miners. Here again, recent studies have clarified the situation to a considerable extent.

Bituminous or soft coal miners do not have an occuptional pneumoconiosis of a disabling quality. Their lungs become pigmented from coal dust, but they do not develop a true fibrosis. For anthracite miners, the picture is quite different and the reason is that anthracite mining includes exposure not only to coal dust but also to silica. Disabling miner's asthma was shown by the United States Public Health Service study of 1935 to be a type of silicosis, to which the term anthraco-silicosis is properly applied. Anthracite seams are often found in country rock which contains a considerable amount of free silica and, as in other silica dust exposures, the development of pulmonary fibrosis was found to be proportionate to the amount of silica in the coal dust and the length of exposure. The incidence of tuberculosis among anthracite miners is very high. Clinical pulmonary tuberculosis was found in 15 per cent of those with early anthraco-silicosis and in 43 per cent of those with advanced anthraco-silicosis. Service upwards of twenty to twenty-five years was associated with a tuberculosis rate of 14 per cent among regular miners and 37 per cent among those with a number of years as rock workers. Animal experimentation, with both bituminous and anthracite coal dust, free from associated silica, produced no specific reaction.

Non-Siliceous Minerals

There is no evidence that non-siliceous minerals produce any pulmonary disease. Gardner, at the Saranac Laboratory, tested upon animals such substances as diamond, aluminum oxide, rutile, marble, gypsum, galena, chalcopyrite, iron sulphide, zinc sulphate, fluorite, and dolomite. The sulphides and dolomite proved toxic but this affect is outside of our present discussion. Non-siliceous minerals proved inactive in animals and there is no clinical evidence of specific disease among workers exposed to this type of dust.

Artificial Abrasives

Experiments with artificial abrasives demonstrated the incorrectness of earlier ideas that dust caused injury to the lungs proportionate to the hardness and sharpness of the particles. Artificial abrasives are among the hardest substances known. Neither silicon carbide nor aluminum oxide produced any reaction resembling silicosis in experi-

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mental animals nor do workmen exposed to these dusts show any clinical symptoms or X-ray appearances suggestive of silicosis.

Silicates

A considerable number of silicate dusts have been studied and their effects upon experimental animals, both by inhalation and injection, noted. They produce little or no reaction in the body tissues and what little reaction some of them do produce does not tend to progress. An exception should be noted here regarding asbestos, which is considered separately.

Nor is occupational exposure to silicate dusts accompanied by any such clinical picture of disability and death as is found in silica dust exposures. The Public Health Service studied some talc mines in 1935. Only 66 men were exposed. Tuberculosis morbidity and mortality rates were very high in the county where the mines were located and the dust exposure was exceedingly severe, running into hundreds of millions of particles. Eight men showed advanced pneumoconiosis but tuberculosis was also present in most of them. It is conceivable that exposure to tremendous quantities of any mineral dust, regardless of silica content, might so overwhelm the lungs that they could not deal with the invading substance. Irritation, inflammation, and probably infection would follow with disability in proportion. Such isolated instances do not invalidate the conclusions stated in the previous paragraph.

Asbestos

Asbestos is the only dust of combined silica which is known to cause a definite pulmonary fibrosis which may result in disability and death. Most of the asbestos fabricated in the United States comes from the mines in Quebec. Asbestosis, however, is not found among asbestos miners, most of whom work in open quarries or pits, nor among men engaged in milling operations whereby the asbestos is prepared for shipment and bagged, but among workers in fabricating plants. Only recently has an explanation been offered for this anomaly. It would appear that not only is asbestos an exception in that it differs from other silicate dusts in its ability to cause structural changes in the lungs but its action appears to be mechanical and not chemical like the action of silica. If asbestos is crushed to fine particles, it will not cause asbestosis in experimental animals nor will asbestos produce any reaction elsewhere than in the lungs. If its action were in the nature of silica, the effect would be more pronounced with smaller particle sizes. Consequently, Gardner concludes that the relatively long spicules of asbestos produce their effect because the lungs are never at rest and the constant movement provokes the fibrous reaction.

Like silicosis, asbestosis develops slowly, presenting the characteristic symptom of shortness of breath. The number of cases of asbestosis that have come to autopsy is small but there are several cases on record in which death was due entirely to the asbestos and not to any associated infection or other condition. There does not seem to be any pronounced tendency to tuberculosis in asbestosis, but most of the fatal cases have been complicated by other disease such as cancer and diabetes so that the extent to which the asbestosis was responsible for death is not clear.

The pathology and X-ray appearance of asbestosis are entirely different from silicosis and in the absence of a definite history of exposure over a considerable period of time to asbestos dust, the diagnosis is difficult. A great deal of success has been attained in controlling dusts in the plants where asbestos is fabricated so that it is not likely that asbestosis will ever become an important industrial disease, as far as the number of cases is concerned.

The scarcity of clinical data on asbestosis makes the prognosis doubtful. We have had a small group of cases under observation for about ten years and some of these show a definite but slow progression as far as can be judged from the X-ray films. However, what the outcome may be remains to be seen. These men are all working and showing no signs of any disability.

Silicosis

It may be said that there is a very general accord among the leading investigators and research authorities in the opinions and conclusions presented in the international conferences on silicosis. Such differences of opinion as may be expressed from time to time do not seriously affect the general agreement on the basic factors affecting the cause, incidence, effects, and prognosis of silicosis.

The effect of silica upon the lungs depends on the nature and extent of exposure. That is to say, upon the quantity of dust in the air breathed by workmen, the amount of free or uncombined silica in the dust, the size of the dust particles, the presence of other substances in the dust which may modify the action of silica, and the length of time or duration of exposure. The individual himself must also be considered. Previously existing disease of the lungs, especially a tubercle infection, will influence his reaction to inhaled silica. There may be other phases of susceptibility about which we know little or nothing but in the main, it may be stated that individual susceptibility to the action of silica dust is an acquired rather than a congenital condition.

In industry we find different types of silica hazards. The severity of a silica hazard is estimated on the amount of dust in the air and this is expressed in millions of particles per cubic foot. The particles counted are those under ten microns in size, though the present opinion is that it is the particle between one-half a micron and three microns that is mostly responsible for causing the disease.

Exposure to excessive quantities of pure silica dust, that is, where the dust count is in the hundreds of millions of particles per cubic foot, is now rare. Isolated instances may be found as in the case of a sandblaster improperly protected. Such exposure produces a severe type of silicosis in upwards of five years, even sooner in extreme cases. With such extensive silica damage to the lungs, tubercle infection is almost inevitable, though cases will be seen occasionally that proceed to a fatal termination without infection.

Exposure to moderate amounts of silica dust where the particle counts are in excess of ten million does not occur frequently at the present time, though it is only in the last few years that silica industries have endeavored to cope with their dust problem and effectively reduce the amount. Such exposures produce definitely disease in from eight or ten to twenty or more years, in proportion to the actual quantity of dust. The incidence of tuberculosis is high and of those who die of their pulmonary condition, 75 per cent are tuberculous.

It is also possible to find degrees of silica exposure which, while producing definite silicosis evident on X-rays, will not produce disability or death in an average working lifetime. Mortality statistics are not broken down by industry to an extent sufficient to make available definite figures by degree of exposure. We must rely then on the clinical results obtained in industrial investigations which are conclusive enough for those industries studied. Our own impression is that even where the silica hazard is of a mild type, the incidence of tuberculosis will be higher than the average. It has been stated that where silica dust contains caustic alkalies also, the action of the silica is intensified very considerably. The evidence here is meager. Other substances apparently have some inhibiting effect upon the action of silica, affecting the pathological as well as the clinical picture.

Prognosis

The prognosis is fair for a silicotic whose lungs are not extensively damaged and who is free of infection. Such persons have an increased susceptibility to infection and if brought into intimate contact with a case of tuberculosis are very apt to become tuberculous also. The outlook for the tuberculo-silicotic is poor. Many cases are essentially chronic and may live for years. Others show rapid progression and die in from one to two years after their infection becomes active. The importance of tuberculous infection among persons exposed to silica dust brings up a very important underwriting consideration, that is, where the worker in a silica occupation is in contact in his home with an open case of tuberculosis.

The question is frequently asked whether men who had worked in silica dust at one time but later changed to a job in which there was no exposure to silica might be considered standard risks for life insurance if they passed the usual insurance physical examination. While there are no insurance statistics to prove the point one way or the other, there is every reason to believe that a group of such men will not give a standard mortality. The usual type of physical examination will not detect silicosis. The only way to be sure is to secure X-rays of the lungs of such applicants and have them interpreted by some one who has specialized in reading films of this kind.

Silicosis is gradually coming under control. The aggravated cases seen twenty years ago

(*Continued on Page 1417*)

SURPLUS FUNDS

1.	Metropolitan	$309,717,567	23.	National Life & Acc.	10,451,570	49.	Wash. National	1,253,833	75.	Amer. Mutual, Ia.	1,217,705
2.	Prudential	209,392,460	24.	Lincoln National	8,941,163	50.	Minnesota Mutual	1,066,705	76.	Colonial Life	1,161,364
3.	New York Life	125,639,022	25.	Equitable Life, Ia.	8,277,602	51.	Sun Life, Md.	1,045,994	77.	Industrial L. & H., Ga.	1,157,717
4.	Equitable, N. Y.	90,762,658	26.	State Mutual	8,625,381	52.	Columbus Mutual, O.	2,708,398	78.	Protective Life, Ala.	1,111,333
5.	John Hancock	37,961,337	27.	Phoenix Mutual	7,344,962	53.	Ohio National	2,672,773	79.	Country Life, Ill.	1,101,143
6.	Travelers	64,902,795	28.	Southwestern Life	7,340,000	54.	Guarantee Mutual	2,578,074	80.	American Standard	1,097,333
7.	Northwestern Mutual	57,186,343	29.	Kansas City Life	7,205,984	55.	Midland Mutual	2,570,341	81.	Indianapolis Life	1,512,305
8.	Mutual Life, N. Y.	51,852,202	30.	Fidelity Mutual	6,468,839	56.	Amer. United, Ind.	2,500,000	82.	Amicable Life	1,490,437
9.	Aetna Life	36,855,402	31.	Jefferson Standard	6,300,000	57.	North Amer. Reassur.	2,411,961	83.	Paul Revere Life	1,440,425
10.	Penn Mutual	30,944,720	32.	Bankers Life, Neb.	5,799,129	58.	Commonwealth	2,353,555	84.	Franklin Life, Ill.	1,370,002
11.	Mutual Benefit	30,314,767	33.	Pacific Mutual	5,672,956	59.	Western Life, Mont.	2,350,000	85.	Capital Life, Colo.	1,373,668
12.	Western & Southern	25,383,923	34.	Northwestern National	5,457,918	60.	Home Beneficial	2,348,435	86.	Union Labor Life	1,344,333
13.	Mass. Mutual	27,050,794	35.	Guardian Life	5,269,459	61.	Continental Amer.	2,321,712	87.	Oregon Mutual	1,313,523
14.	Provident Mutual	19,374,230	36.	Reliance Life, Pa.	5,171,696	62.	Mass. Sav. Bank	2,293,596	88.	Monarch Life, Mass.	1,309,124
15.	New England Mutual	19,010,803	37.	Great Southern	4,783,020	63.	Pilot Life, N. C.	2,285,976	89.	Loyal Protective	1,301,597
16.	Life Ins. Co. of Va.	16,956,930	38.	Monumental Life	4,569,137	64.	Pan-American Life	2,244,524	90.	Southland Life	1,301,167
17.	Amer. Nat'l, Tex.	15,533,244	39.	Home Life, N. Y.	4,300,635	65.	General American	2,244,449	91.	National Reserve	1,300,577
18.	Conn. General	14,423,687	40.	Occidental, Calif.	4,152,034	66.	Old Line, Wis.	2,211,278	92.	Illinois Bankers	1,274,333
19.	Conn. Mutual	13,020,498	41.	Life & Casualty	4,077,235	67.	Baltimore Life	2,154,771	93.	Volunteer State	1,269,179
20.	Union Central Life	11,348,155	42.	Continental, Ill.	3,922,443	68.	Ohio State Life	2,074,617	94.	Atlantic Life, Va.	1,256,692
21.	Bankers Life, Ia.	11,599,738	43.	Central Life, Ia.	3,878,551	69.	Presbyterian Min.	1,963,971	95.	Girard Life, Pa.	1,242,820
22.	National Life, Vt.	11,224,492	44.	Columbian National	3,529,068	70.	Alliance Life	1,859,775	96.	Great Amer., Texas	1,174,325
			45.	Acacia Mutual	3,412,797	71.	Berkshire Life	1,840,918	97.	United Life & Acc.	1,093,817
			46.	Calif. Western States	3,295,000	72.	Farmers & Bankers	1,834,668	98.	Northern Life	1,082,901
			47.	Provident Life & Acc.	3,283,576	73.	Business Men's, Mo.	1,808,949	99.	Mass. Protective	1,063,910
			48.	Mutual Trust	3,281,718	74.	New World Life	1,799,790	100.	Lutheran Mutual	1,052,766

*Includes $128,801,620 contingency reserve.

INDUSTRIAL DUST HAZARD

(*Continued from Page* 1413)

are rarely seen now and the clinical picture has changed with the progress of dust control measures. The trend is towards a lessened incidence of new cases of silicosis. This improvement depends upon the maintenance of constant vigilance in detecting unsuspected hazards and in supervising the efficiency of dust control methods after they have been put into effect. It cannot be expected that this improvement will be reflected in insurance mortality until a considerable period of time has elapsed.

The conclusion seems warranted from clinical and laboratory experience, then, that the only dusts which of themselves might have sufficient effect on the mortality of workers exposed to them, so great as to require an extra premium charge for life insurance, are silica and possibly, asbestos.

Life insurance men naturally will turn to mortality statistics for confirmation of a finding so at variance with the common belief, long held, that exposure to all kinds of dusts is productive of a high incidence of respiratory tuberculosis. To the layman it seems only reasonable to suppose that the difficulty in breathing he experiences when exposed to large quantities of dust must in some way affect his health, and that filling the lungs with foreign particles must have injurious effects on them. The writings of eminent students of occupational mortality statistics have served to strengthen this belief. Nearly all such statistics show the mortality from respiratory tuberculosis among workers exposed to a wide variety of dusts to be well above the average for all workers included in the study. But, when these figures are refined so far as is practicable to make allowance for factors other than dust, which may influence the mortality from tuberculosis of men employed in these occupations, the importance of many dusts as causative factors becomes less evident.

There is a surprisingly large number of factors, other than dust, any one of which may be responsible for the high mortality from tuberculosis among men in dusty trades. Some dusty occupations are carried on by men recruited from the lower income classes, among whom it is now known that apart entirely from exposure to a specific occupational hazard, the death rate from tuberculosis, is considerably higher than among persons in the higher income groups. The selection of certain kinds of work by the less robust workers is reflected in the high mortality from tuberculosis of men in other callings. Racial and nationality groups tend to enter certain occupations, with the result that the peculiar susceptibility or relative immunity to tuberculosis of these stocks, colors the mortality picture of men in such occupations. These are but a few of the many circumstances for which allowance must be made before conclusions can be drawn as to whether a particular dust is or is not the responsible agent in the high mortality found among men in a dusty trade.

Unfortunately there is, for the United States, no considerable body of mortality statistics sufficiently detailed to permit of evaluating separately some of even the most important conditions affecting the mortality of men in specific occupations. Perhaps the best statistical demonstration of the effect of different types of dust on mortality from tuberculosis is supplied by two British authorities, Dr. Edgar L. Collis and G. Udney Yule. These authors selected for study, from the occupations reported upon in the Registrar General of England and Wales' report on Occupational Mortality covering the years 1921-1923, two groups of occupations—one exposed to silica dust, and the other to non-silica inorganic dust. The groups were so chosen that each had, as far as possible, the same amount of dust exposure, physical effort, exposure to heat or to weather, or any underground environment. Part of the authors' comment on the results as regards respiratory tuberculosis is quoted:

"For all ages (20 to 65) the comparative mortality figure (this figure is defined as the number of deaths that would have occurred in the Standard Population at the rates ruling in the occupation) of the Silica Group is no less than 592.2 against 163.5 for the Standard Population, i. e., the mortality is more than three and a half times the normal. For the Non-silica Group, the Comparative Mortality Figure, although actually slightly higher than normal, at first sight hardly seems to differ significantly from the normal; nor would one's judgment be much affected if, instead of making comparison with the Standard Population, one had used the Social Groups III and IV, for which the respective Comparative Mortality Figures are 139.8 and 164.2. But the summary figures conceal interesting changes with age. A glance at the figures (shown on Table 4 of their study), shows that in the Silica Group the ratio of mortality to the normal, though greater than unity even in the lowest age group, rapidly increases as age advances. In the Non-silica Group, the comparative mortality is actually below normal up to age 35: it rises just above normal in the following age group; but as ages over 45, it is conspicuously above normal."

The facts regarding tuberculosis were not published in this report for each of the six silica occupational classes included in the study. We have, however, determined by reference to the report of the Registrar General, that each of the silica occupations had over twice the expected number of deaths. The extraordinary high ratio of 1,150 per cent was recorded for tin and copper miners, while sandstone masons, cutters, and dressers had a ratio of 441 per cent. On the other hand, only the limestone masons, cutters, and dressers of the five occupations considered in the non-silica group exhibited a high ratio, 191 per cent.

Like the English tin and copper miners, American metal miners are exposed to a serious silica hazard and have an inordinate mortality from respiratory tuberculosis as will be seen by reference to Table 1. In this table are given the ratios of actual to expected deaths from tuberculosis for a number of occupations exposed to dust and reported upon in the Joint Occupation Study, 1928, and in the Occupational Study, 1937, both published by the Actuarial Society of America and the Association of Life Insurance Medical Directors. In the period 1915-1926, the number of deaths from respiratory tuberculosis among metal miners was over eight times the number expected on the basis of death rates by ages prevailing among standard ordinary lives in the same years. In the latter study covering the years 1925 to 1936, the ratios were even higher—over 11 times as many deaths were recorded for metal miners as were expected on the basis of death rates among standard lives in these years.

While the actual number of deaths for each separate metal mining class was rather small in both investigations, and consequently the ratios are subject to a large margin of

(*Continued on Page* 1420)

INDUSTRIAL DUST HAZARD

(*Continued from Page* 1417)

error, nevertheless it is interesting to observe that they were quite uniformly high for copper miners, gold and silver miners, and lead and zinc miners.

The situation as regards iron miners is interesting because it was thought for a long time that they had little or no exposure to silica. More recent studies show that there is actually a moderate degree of silica hazard which produces silicosis in upwards of twenty years. However, in both insurance studies there were too few deaths from respiratory tuberculosis to permit definite quantitative conclusions therefrom.

A great many workers in mining occupations, it must be borne in mind, are common laborers among whom we should expect a much higher mortality from tuberculosis than among standard ordinary policyholders. From the limited data available for unskilled workers, from these studies it would appear that the tuberculosis death rate for this class of workers runs between two and three times the rate of standard ordinary business. Thus it is clear that even when allowance is made for the important factor of social-economic class, the death rates for the metal miners are extremely high.

Coal miners in different sections of the country present an interesting contrast. Among the bituminous miners, exclusive of the Pennsylvania miners, the mortality from tuberculosis was over 1½ times that on standard ordinary miners, a low ratio for this type of worker. Pennsylvania miners, on the other hand, most of whom were employed in the anthracite field, had a very high ratio of actual to expected deaths—well over 300 per cent. Anthracite miners, as we have stated, are exposed to an appreciable amount of silica dust, which accounts for their relatively unfavorable position.

No dusty trade has received more careful study in the United States than that of granite cutting. Long recognized as a dangerous trade, it has been found time and again to take a heavy toll in deaths from tuberculosis. The amount of free silica in the dust breathed by these workers is very large. In the years 1925 to 1936, the mortality from tuberculosis among granite and sandstone cutters was the highest recorded for any of the groups of insured lives studied. There were more than 25 times as many deaths from tuberculosis as expected, whereas in the earlier years, there were nearly ten times as many deaths as expected, based on a small exposure. Marble and limestone cutters did not show anything like as high a ratio as the granite and sandstone cutters. There were ten deaths where one was expected in the years 1925 to 1936.

It can be but regretted that there are so few mortality statistics for occupations exposed to dusts other than silica. Grinders of metals, most of whom for many years have been using composition wheels in place of the old sandstone wheels, and consequently have had a little or no exposure to free silica, experienced a relatively low extra mortality from tuberculosis in both studies, based, however, on a small exposure. An interesting point to be noted here is that this occupation also offers exposure to metallic dust. It would appear that neither the dust from the composition wheels nor the metal dust has produced excessively high death rates from tuberculosis. Buffers and polishers, a somewhat similar group, also appeared to have a low extra mortality from tuberculosis in the years 1915 to 1926.

Cement and Lime Workers

Cement, lime, and artificial stone workers were reported upon in both the Joint Occupation Study and the Occupational Study, but the number of deaths from tuberculosis was in each instance less than the number required for separate tabulation. This fact in itself is significant, although it does not mean necessarily that the death rate from tuberculosis was below the average. Nevertheless, from the facts presented in these studies, it is quite evident that the tuberculosis death rate for this occupational class was comparatively low.

Three groups of male workers exposed to organic dusts are included in the table—upholsterers, cotton mill operatives, and woolen mill operatives. The ratios for all of these classes run about 200 per cent, based on small exposures.

It must be admitted that these occupational mortality statistics are meagre and do not permit of very definite conclusions regarding the effects of specific dusts. So far as they go, they fully substantiate clinical and laboratory findings regarding the injurious character of silica dust. The death rates from respiratory tuberculosis for men in occupations where there is a serious exposure to silica dust are so high as to leave no room for doubt that silica is the offending agency.

Contrary to clinical and laboratory findings, however, the mortality statistics, presented by Collis and Yule, suggest that long continued exposure to non-siliceous inorganic dust has an injurious effect on the lungs as is indicated by the higher mortality from tuberculosis among exposed workers age 45. About all that can be said from the limited American life insurance company experience is that the tuberculosis death rates for grinders, metal polishers, and buffers who are exposed to inorganic dusts, most of which do not contain a large percentage of silica, are not high, having in mind the economic status of these workers as compared with the average ordinary policyholder. Lime, cement, and artificial stone workers had a quite favorable mortality. The very high ratio for marble and limestone cutters, however, may be suggestive of some untoward influence. Dr. L. U. Gardner at Saranac Lake, found that animals dusted with marble dust by inhalation, developed little reaction, but became temporarily more susceptible to infection with tuberculosis. The increased susceptibility was not marked.

The three occupations in which there is exposure to organic dust had from two to two and one-half times the number of tuberculosis deaths expected. Even allowing for the economic position of these groups as compared with ordinary policyholders generally, the mortality is high. It is difficult to see in the light of present clinical knowledge, how it is possible to account for the excess mortality on the score of damage to the lungs caused by such dust.

Mortality findings, then, are still not in complete harmony with clinical and laboratory findings. But from a practical underwriting standpoint, it may be said that the only dust which of itself might s... the total mortality, by increasing t... dence of tuberculosis, as to require an... premium charge for life insurance, is free silica. We have no mortality data for asbestos workers but, it may well be that exposure to asbestos dust in large quantities will also be found to produce a high mortality.

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the same physician, that it was impossible to come to any definite conclusion and say, "this man died of asbestosis," when his previous disability certificates showed that he had myocarditis or high blood pressure or cancer or diabetes or pulmonary tuberculosis.

The pathology and X-ray appearance of asbestosis are entirely different from silicosis and in the absence of a definite history of exposure over a considerable period of time to asbestos dust, the diagnosis is difficult. A great deal of success has been attained in controlling dusts in the plants where asbestos is fabricated so that it is not likely that asbestosis will ever become an important industrial disease, as far as the number of cases is concerned.

The scarcity of clinical data on asbestosis makes the prognosis doubtful. We have had a small group of cases under observation for about ten years — there were originally, I think, some sixty odd men in this group and we still have fifty of them where we can get at them — and some of these show a definite but slow progression as far as can be judged from the X-ray films. However, what the outcome may be remains to be seen. These men are all working and show no signs of any disability.

In the meantime the plants have been cleaned up so that these men are not exposed now to inordinate quantities of asbestos dust, so it is difficult to say what would have been the progress of their disease had their working conditions remained the same.

SILICOSIS

It may be said that there is a very general accord among the leading investigators and research authorities in the opinions and conclusions presented at the international conferences on silicosis (Johannesburg, August, 1930; Geneva, August and September, 1938.) Such differences of opinion as may be expressed from time to time do not seriously affect the general agreement on the basic factors affecting the cause, incidence, effects, and prognosis of silicosis.

The effect of silica upon the lungs depends on the nature and extent of exposure. That is to say, upon the quantity of dust in the air breathed by workmen, the amount of free or uncombined silica in the dust, the size of the dust particles, the presence of other substances in the dust which may modify the action of silica, and the length of time or duration of exposure. The individual himself must also be considered. Previously existing disease of the lungs, especially a tubercle infection, will influence his reaction to inhaled silica. There may be other phases of susceptibility about which we know little or

dust particles of one to two and three microns in their largest diameter, that those particles had no physical characteristics in the ordinary sense in which we apply them. Artificial abrasives are among the hardest substances known. Neither silicon carbide nor aluminum oxide produced any reaction resembling silicosis in experimental animals nor do workmen exposed to these dusts show any clinical symptoms or X-ray appearances suggestive of silicosis.

SILICATES

A considerable number of silicate dusts have been studied and their effects upon experimental animals, both by inhalation and injection, noted. They produce little or no reaction in the body tissues and what little reaction some of them do produce does not tend to progress. An exception should be noted here regarding asbestos, which is considered separately.

Nor is occupational exposure to silicate dusts accompanied by any such clinical picture of disability and death as is found in silica dust exposures. The Public Health Service studied some talc mines in 1935. Only 66 men were exposed. Tuberculosis morbidity and mortality rates were very high in the county where the mines were located and the dust exposure was exceedingly severe, running into hundreds of millions of particles. Eight men showed advanced pneumoconiosis but tuberculosis was also present in most of them. It is conceivable that exposure to tremendous quantities of any mineral dust, regardless of silica-content, might so overwhelm the lungs that they could not deal with the invading substance. Irritation, inflammation, and probably infection would follow with disability in proportion. Such isolated instances do not invalidate the conclusions stated in the previous paragraph.

ASBESTOS

Asbestos is the only dust of combined silica which is known to cause a definite pulmonary fibrosis which may result in disability and death. Let me make that point clear. When we talk of silicosis and the types of dust that produce silicosis, we mean dusts that contain silica in a free or uncombined state, SiO_2. When the silica is in the form of silicon carbide or magnesium silicate or iron silicate that is what we mean by combined silica. The combined silicate dusts, as I have just mentioned, do not produce any pulmonary disease, with one exception, and that one exception is asbestos, which is a hydrated magnesium silicate mixture, with a little iron in it. Most of the asbestos fabricated in the United States comes from the mines in

Quebec. Asbestosis, however, is not found among asbestos miners, most of whom work in open quarries or pits, nor among men engaged in milling operations whereby the asbestos is prepared for shipment and bagged, but among workers in fabricating plants. Fabrication, you will understand, being in the nature of a textile process. Only recently has an explanation been offered for this anomaly. It would appear that not only is asbestos an exception in that it differs from other silicate dusts in its ability to cause structural changes in the lungs but its action appears to be mechanical and not chemical like the action of silica. If asbestos is crushed to fine particles, it will not cause asbestosis in experimental animals nor will asbestos produce any reaction elsewhere than in the lungs. If its action were in the nature of silica, the effect would be more pronounced with smaller particle sizes. In other words, Gardner established that the intensity of a silica reaction upon animals depended upon the smallness of the silica particles, because, to sum it up briefly, the finer the silica particles, the greater is the surface exposure which acts upon the pulmonary tissue.

Asbestos particles, below a certain size, do not produce any effect in experimental animals. Furthermore, you can produce a silicotic reaction, the typical silicotic nodule, in any organ by injecting prepared silica. You can get silicosis of the liver or of the spleen or elsewhere. But you cannot produce any reaction in animals with asbestos anywhere except in the lungs, and then only if your particles are sufficiently large. Consequently, Gardner concludes that the relatively long spicules of asbestos produce their effect because the lungs are never at rest and the constant movement provokes the fibrous reaction.

Like silicosis, asbestosis develops slowly, presenting the characteristic symptom of shortness of breath. The number of cases of asbestosis that have come to autopsy is small but there are several cases on record in which death was due entirely to the asbestos and not to any associated infection or other condition. There does not seem to be any pronounced tendency to tuberculosis in asbestosis, but most of the fatal cases have been complicated by other disease such as cancer and diabetes so that the extent to which the asbestosis was responsible for death is not clear.

I made a rather exhaustive study a few years ago of all the insurance certificates that I could find in which asbestos was given as the cause of death and then followed up the previous history of these people and got their medical certificates when they were on disability. The diagnosis given on the disability certificates was usually so at variance with the diagnosis given on the death certificates, often by

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far exceed those found for men in other pursuits, as to leave little room for doubt that silica is implicated.

With regard to silicate and other inorganic dusts not containing free silica, American mortality data are very meagre. The relatively low death rate from tuberculosis among grinders in the small American insurance experience suggests that the effects of aluminum oxide, silicon carbide and other substances used in manufactured wheels are slight. British data, much more complete, show lower than average tuberculosis mortality up to age thirty-five, but substantially higher mortality after age forty-five for a group of men exposed to inorganic dusts other than silica dust. Whether this unfavorable situation later in life is due to the cummulative effect of such dusts with duration of exposure or whether it is due to the inclusion in these occupational groups of a substantial number of men who had been exposed also to silica dust, is not clear.

There are no mortality data for men exposed solely to metallic dusts.

Recent statistical studies, like the earlier ones, show higher than average death rates from tuberculosis among men employed in certain occupations or industries in which organic dust is generated. In no instance, however, does the rate for a group of this kind approach in magnitude the extremely high rates found among men employed in some of the occupations in which there is exposure to silica dust, [illegible] the light of clinical knowedge, is it possible to account for the excess [illegible]ity on the score of damage to the lung tissue caused by dust.

The relationship between dust inhalation and acute pulmon[illegible]ase remains a field for further investigation. A splendid contribution to our knowledge of this subject has already been made by the United States Public Health Service in their bulletins on *The Health of Workers in Dusty Trades* (32, 33). A greater volume of data of this kind and more detailed studies of occupational mortality are vitally needed to guide the work of industrial physicians and hygienists in this field.

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an unusually high mortality from pneumonia (28) and the same is true of certain occupations in the steel industry (29, 30). Exposure to dust is associated with some of these occupations and usually extremes of temperature are also encountered with such other conditions as would tend to cause dampness and chilling. So far as silica is concerned, a high mortality from pneumonia is associated with the silica trades, both in this country and abroad. Whether silica acts as a predisposing cause of pneumonia or due to the accompanying lung damage the prognosis is more unfavorable, cannot be shown from the figures available.

For some years, a series of interesting studies has been carried on in Pittsburgh by Haythorn and Meller and their associates in an endeavor to establish any possible connection between the prevailing high mortality rate from pneumonia in that city and atmospheric pollution. These reports are interesting and suggestive but not entirely conclusive. The following quotation is from a recently published report of these authors (31).

> It is further seen . . . that during the years from 1932 to 1935 when the depression was at its height, when air pollution from industrial flues was greatly decreased and when economic and living conditions were at their worst, there was a great decrease in the number of deaths from pneumonia. However, the decrease occurred in females as well as males so that the change cannot be attributed to conditions within the plants, such as overheating and rapid chilling of the employees.

SUMMARY

Early occupational mortality studies focussed attention on the importance of dust as a cause of respiratory diseases. Virtually all kinds of dusts were implicated. Present day clinical and laboratory studies point to the serious damage to the lung tissue caused by a few dusts, notably silica and asbestos, and to the relatively little damage to the lung tissue caused by many other dusts. The most complete recent mortality studies have been examined in the light of this clinical and laboratory knowledge. These mortality data, while yielding highly suggestive information, are very incomplete and permit only qualified general conclusions regarding the effects of dust exposure on the incidence of respiratory diseases for even the broad classes of dust with one exception—silica dust. The death rates from tuberculosis among men in occupations in which there is exposure to free silica so

In the American insurance experience, covering the years 1915–1926 (6), also, the ratio of actual to expected deaths was above average for several occupational groups exposed to organic dust. The ratios of actual to expected deaths for the more important of these were: cotton mill operatives, 176 per cent; woolen mill operatives, 134 per cent; upholsterers, 225 per cent; cigar makers and tobacco factory operatives, 174 per cent.

The occupations discussed here have been traditionally classified among the dusty trades. One may well question, however, the inclusion in such a list of boot and shoe and of tobacco factory workers. The great bulk of workers in these industries cannot be said to be exposed to appreciable quantities of dust. But taking the list as it stands, there is obviously no resemblance between the tuberculosis ratios for these occupations and the very high ratios for occupations with exposure to silica dust. Some condition connected with the work of persons in these trades obviously is associated with the above-average incidence of tuberculosis. It may be that the less robust workers are attracted to them. Whatever may be the explanation, clinical findings would suggest that dust is not an important factor in the high incidence of tuberculosis in these trades.

FUNGUS DISEASES OF THE LUNGS

Dust may convey to the respiratory tract various types of fungi. Some of these apparently have no clinical significance. Others are pathogenic and may produce either acute or chronic disease. The constantly increasing use of the roentgen-ray in diagnosis and in routine physical examinations has served to awaken interest in fungoid diseases about which too little is known and which are not uncommonly diagnosed as tuberculosis (25). Our knowledge of the subject is very incomplete but it is recognized that these fungus diseases are frequently occupational in origin and associated with dust inhalation and may result fatally. Mortality statistics are entirely lacking, but the reports of Fawcitt (26) and the recent studies of coccidioidal infection in California (27) indicate that such pulmonary diseases may be quite important.

PNEUMONIA

The effect of dust inhalation upon the incidence of and mortality from pneumonia has been the subject of much study without any clear-cut picture of the rôle of dust resulting (32, 33). The foundry industry has

of textile plants and he stated that there was no evidence of organic dust causing pulmonary diseases (24).

The mortality picture as regards organic dust and tuberculosis is confused. In the Registrar-General's Report (5), out of thirteen classes of textile workers, six had above average death rates from tuberculosis but in only one class—wool, worsted, card, comb or frame (not spinning frame) tenters—was the rate as much as 59 per cent above average. No

TABLE 4

*Standardized mortality (comparative mortality figures) from respiratory tuberculosis of males ages 20 to 65 years in occupations exposed to organic dust compared with that of all occupied and retired civilian males taken as 1,000, England and Wales, 1921–1923**

OCCUPATION	MORTALITY RATIO
All occupied and retired civilian males	1,000
Wool sorters	1,065
Cotton blow room operatives—skilled	750
Rag grinders, wool willowers, etc.	1,093
Cotton card and frame (not spinning frame) tenters	1,057
Wool, worsted card, comb, or frame (not spinning frame) tenters	1,591
Cotton strippers and grinders and card room jobbers	796
Cotton spinners and piecers	1,072
Wool and worsted spinners and piecers	898
Cotton—doublers, winders, warpers, beamers, etc.	869
Wool and worsted—doublers, winders, warpers, beamers, etc.	510
Cotton weavers	731
Woolen and worsted weavers	1,162
Weavers of other textiles	904
Boot and shoe clickers and cutters	1,820
Skilled boot and shoe operatives, not clickers or cutters	1,821
Bakers and pastry cooks	1,016
Tobacco factory operatives	2,002
Upholsterers, coach trimmers, and bedding makers	1,262
Drafters and brush makers	2,376

* Compiled from Registrar-General's Decennial Supplement, England and Wales, 1921, Part II. Occupational Mortality.

other class showed more than 16 per cent excess, while in several classes the rate was quite low. On the other hand, boot and shoe factory workers showed an 82 per cent excess; tobacco factory operatives had 100 per cent excess mortality, and brush makers and drafters, 138 per cent excess. The rate for upholsterers, coach trimmers, bedding makers was 26 per cent above the average. Table 4 gives the Standardized Mortality for the principal occupations exposed to organic dust.

dust was injected into the peritoneal cavity of animals, the reaction was inert.

In the process of welding, metals and metallic oxides are volatilized and inhaled into the lungs in a very finely divided state. Reports have been made of roentgen-ray films, taken during the physical examination of welders, which showed an appearance of nodulation resembling fine silicosis. This has given rise to the question as to whether silicosis might be contracted from welding (21, 22), although all these cases were symptom free.

One of the cases reported by Sander came to autopsy following an accidental death. No fibrous tissue was found and it was concluded that the nodular shadows on the film were caused by collections of iron and carbon pigment in the lymph channels of the lung (21, 23).

No satisfactory statistics are available on the mortality of men exposed solely to metallic dust. Perhaps there is as much dust of this character thrown off in grinding as in any other process and the comments regarding the mortality of grinders and of buffers and polishers, given under inorganic dust, are pertinent here. These mixed metal and other dusts from the composition wheels do not seem to have produced mortality rates from tuberculosis comparable in any way with those where the silica hazard is severe.

ORGANIC DUSTS

Until the action of various kinds of dusts upon the lungs was made the subject of experimental studies, the organic dusts, rising from industrial processes, were considered to be responsible for tuberculosis. Knowledge resulting from the extensive studies of silicosis has tended to minimize the possible action of organic dust, both on account of the large average size of the particles and their small numbers when compared with the concentration of inorganic dust particles found in various industries. The presence of pulmonary fibrosis or other structural change caused by organic dust and associated with infection has not been demonstrated among industrial workers. Nor is there evident, in connection with industrial processes involving exposure to organic dust, a clinical picture which could be compared with that associated with silica.

The late Doctor Landis of Philadelphia was one of a group of physicians who were among the first in this country to study tuberculosis among industrial workers. His studies included employees of a number

might have been expected and 18 deaths from influenza and pneumonia where 11 were expected.

In the 1925–1936 study (7) there were 10 deaths from tuberculosis among limestone and marble cutters where one was expected. Here again, as in the case of the British limestone masons, cutters and dressers, the question is raised as to whether some of these men had not worked on granite and other stones as well. Granite and sandstone cutters in the American insurance experience, as we have shown, had more than twenty-six times as many deaths from tuberculosis as expected.

American iron miners apparently have a different type of exposure from the British. We have included them in table 1, somewhat arbitrarily, with the occupations exposed to silica dust. There were 12 deaths from tuberculosis among them where only 1.4 were expected, or a ratio of 857 per cent. This very high ratio suggests that, in some American mines, there may be a greater silica hazard than had been thought. The number of deaths was small, however, and the result cannot be considered conclusive.

METALLIC DUSTS

Hoffman (2), in Bulletin 231, emphasized the danger of exposure to metallic dusts (pages 51–161), but the experience of recent years has not borne out his conclusions. There has not been defined in the various industries in which exposure to metallic dust occurs any definite or specific pulmonary disease resulting therefrom. All the indications are that, where pulmonary disease has been described in conjunction with metallic dust, the blame must be placed on coincidental exposure to silica dust. Macklin and Middleton (17), reporting in 1923, point out that silicosis is associated with the use of natural grindstones and not artificial grindstones and make no mention of ill effects from metal dust. Collis (18) had stated the same general conclusion in his well known Milroy Lecture in 1915 and stated further that the percentage of free silica is the index of harmfulness.

Drinker (19) states that there are no data to indicate that iron in the absence of silica caused pathology in any way comparable to silicosis. Carleton (20) states that hematite is relatively harmless compared with flint and other silica dusts; if inhaled over long periods of time, it might cause fibrosis and tuberculosis but further evidence is needed. Under experimental conditions, it is a relatively harmless dust.

In a series of experiments by Miller and Sayers (15), when iron oxide

deaths. The extraordinarily high ratio of 1,150 per cent was recorded for tin and copper miners, while sandstone masons, cutters and dressers had a ratio of 441 per cent. On the other hand, only the limestone masons, cutters, and dressers in the nonsilica group exhibited a high ratio, 191 per cent. Why these men should have so high a ratio is not clear. It may be that many of them had carried on their trade, at one time or another, in districts where granite or sandstone were cut and, consequently, had been exposed to silica dust.

So far as the occupations exposed to silica dust are concerned, as we have mentioned, these findings are in agreement with American insurance experience. Unfortunately, there are only a few occupations exposed to inorganic dust, other than those discussed under silica and coal dust, included in the American insurance experience and none of these is entirely free from complicating exposure to silica. In the 1915–1926 Medico-Actuarial Study (6), a small group of grinders of metals had twice the expected number of deaths from tuberculosis, while in a somewhat larger exposure in the period 1925–1936 (7), the number of deaths from tuberculosis was about one and a half times the number expected. In recent years, sandstone grinding wheels have largely been replaced by composition wheels throughout industry. It is a fair assumption, therefore, that most of the grinders were exposed to dust from composition wheels, silicon carbide, aluminum oxide, etc., although undoubtedly some grinders were employed where sandstone wheels are still in use. This comparatively favorable result is at variance with the result for grinders in England and Wales (5) who had a ratio of actual to expected deaths of 368 per cent. That study covered the years 1921 to 1923, and the proportion of workers using sandstone wheels may have been greater than in the later American insurance experience. The report of the Registrar-General brings out the important fact that grinders in the cutlery industry, where the sandstone wheel is much in use, have a much higher mortality than do other grinders. We have determined from the facts presented in this report that among the cutlery grinders there were over seven and a half times as many deaths from respiratory tuberculosis as expected, whereas among other grinders actual deaths were somewhat fewer than three times the expected.

Buffers and polishers of metal, a somewhat similar group, were represented in the 1915–1926 American insurance study (6) by 17,000 life years of exposure. There were 10 deaths from tuberculosis where 7

rapidly increases as age advances. In the Non-silica Group, the comparative mortality is actually below normal up to age 35; it rises just above normal in the following age-group; but at ages over 45, it is conspicuously above normal.

The report does not give the facts regarding the mortality from respiratory tuberculosis separately for each of the eleven occupations included in either the silica or the nonsilica group. Because of our special

TABLE 3

*Ratio of actual to expected deaths from respiratory tuberculosis among males ages 20 to 65 years, exposed to specified kinds of dusts, England and Wales, 1921–1923**

OCCUPATION	ACTUAL DEATHS	EXPECTED DEATHS	RATIO PER CENT
Silica Dust			
All selected occupations	654	179	365
Tin and copper mine—underground workers, not superintending staff	92	8	1,150
Potters' mill workers; slip makers; potters	105	37	284
Earthenware, china, etc., kiln and oven men, and kiln setters and placers	50	23	217
Metal grinders	221	60	368
Sandstone miners and quarriers	36	17	212
Sandstone masons, cutters, and dressers	150	34	441
Nonsilica Dust			
All selected occupations	162	150.3	108
Brick and plain tile makers, moulders, etc., furnace and crucible pot makers	34	33.7	101
Brick, tile, etc., kiln and oven men	15	22	68
Iron ore mine—underground workers, not superintending staff (Staffordshire and North Riding of Yorkshire)	13	24	54
Limestone miners and quarriers	39	38.6	101
Limestone masons, cutters, and dressers	61	32	191

* Compiled from: Registrar-General's Decennial Supplement, England and Wales, 1921, Part II. Occupational Mortality.

interest in the facts for this particular disease, we have calculated, from the original report of the Registrar-General (5), for each of the selected occupations, the number of deaths from respiratory tuberculosis which might have been expected on the basis of death rates prevailing among all occupied and retired civilian males between the ages of twenty and sixty-five. These results, together with the numbers of deaths which actually occurred, are presented in table 3.

Each of the silica occupations had over twice the average number of

INORGANIC DUSTS OTHER THAN SILICA, ASBESTOS AND COAL DUST

Many industrial dusts contain silica in combined form, as distinguished from free silica, such as combinations with magnesium, iron and aluminum. The effect of silicate dusts upon the lungs, with one exception (asbestos), is problematical. Where exposure to silicate dusts occurs in industrial establishments, under circumstances which might be thought hazardous, one does not see the clinical picture which is presented where free silica is involved nor is there evidence of disability and tendency to infection. Extensive animal experimentation with silicates, both by inhalation and intraperitoneal injection (15), fails to produce any disease akin to silicosis or other evidence of definite disease. It is doubtful whether there is a definite disease to which the term *silicatosis* can be applied, although such a term has been devised. Certainly, the last word has not been said with respect to silicate dusts.

Similarly the dusts of aluminum oxide and some other artificial abrasives, of hematite, and of other inorganic dusts have not been shown to be the cause of marked pulmonary damage. Mortality statistics tend to emphasize the distinction between the effects of dusts containing free silica and other inorganic dusts. This is very strikingly brought out in a study, based on the occupational mortality data of the Registrar-General of England and Wales (5), by Collis and Yule (16). These authors selected for study two groups of occupations—one exposed to silica dust and the other to nonsilica—so chosen that each had as far as possible the same amount of dust exposure, physical effort, exposure to heat or to weather, or any underground environment. Part of the authors' comment on the results as regards respiratory tuberculosis is quoted:

> For all ages (20 to 65) the C.M.F.[3] of the Silica Group is no less than 592.2 against 163.5 for the Standard Population, i.e., the mortality is more than three and a half times the normal. For the Non-silica Group, the C.M.F., although actually slightly higher than normal, at first sight hardly seems to differ significantly from the normal; nor would one's judgment be much affected, if instead of making comparison with the Standard Population, one had used the Social Groups III and IV, for which the respective C.M.F.'s are 159.8 and 164.2. But the summary figures conceal interesting changes with age. A glance at the figures shows that in the Silica Group, the ratio of mortality to the normal, though greater than unity even in the lowest age-group,

[3] Comparative Mortality Figure: This figure is defined as the number of deaths that would have occurred in the Standard Population at the rates ruling in the occupation.

employees; at ages 45 to 54, in 10 per cent; and at ages 55 to 64, in 20 per cent. No such rise with age occurred in any general population group for which comparable data are available.

The prevalence of tuberculosis was greatest among the rock workers. The next to the highest rate occurred among anthracite workers who had changed more than five years previously from very dusty to relatively non-dusty occupations in the industry. The third highest rate was exhibited among persons who had had appreciable exposure to harmful dusts in other industries. Among the regular miners working at the face, the rate was definitely higher than in the control group (men whose dust exposure averaged less than 5 million particles per cubic foot) which showed a prevalence rate of less than 1 per cent.

When the term of service exceeded 20 years, more than 2 or 3 of which involved exposure to heavy concentrations of rock dust, about 37 per cent of such employees [classified as rock workers] showed evidence of pulmonary tuberculosis. Service of 25 to 34 years was associated with a tuberculosis rate of 8 per cent among non-rock workers employed in the haulageways, of 14 per cent among the regular miners, but with a rate under 2 per cent among men exposed to less than 5 million dust particles per cubic foot of air.

Asbestos: Exposure to asbestos dust may produce a pulmonary fibrosis which, like silicosis, may cause disability and death but which does not appear to carry with it a predisposition to tubercle infection and which has a pathology quite distinct from silicosis. The circumstances under which asbestosis will occur are not too clearly defined and undoubtedly there has been a tendency to classify as asbestosis cases in which the causal relationship of asbestos to the condition present has been assumed rather than proved.

Our knowledge of asbestosis is based on individual reported cases. The actual number of fatal cases of asbestosis, supported by postmortem examination, is too few to have any statistical weight but the disease itself and its pathology have been clearly demonstrated. Gardner (13) believes that the action of asbestos dust, unlike silica, may be mechanical and not chemical. If this is so, it would well explain some of the confusing aspects of this disease. The cases described have originated in textile and other asbestos fabricating plants and not in connection with the mining of asbestos. Pedley (14) found that the tuberculosis mortality rate in Thetford Mines (whence 80 per cent of the asbestos used in the United States is derived) did not differ materially from the rate of the Province of Quebec as a whole.

ard lives. Compared with the death rate for laboring groups generally, therefore, the tuberculosis rate for bituminous miners is quite low, whereas the rate for anthracite miners is as high, if not actually higher, than the average for this class of workers. The number of deaths and the ratio of actual to expected deaths for coal miners is given in table 2.

The following quotation from the Public Health Service report (9), previously referred to, is of interest in showing the incidence of clinical tuberculosis among anthracite miners:

Several surveys have shown that tuberculosis of the lungs occurs among 1 to 2½ per cent of the general adult white male population of the country. In a

TABLE 2

Number of deaths and ratio of actual to expected deaths from tuberculosis of the respiratory system

Ordinary Department Mortality Experience of American Life Insurance Companies*

Coal Miners

OCCUPATION	1915–1926		1925–1936	
	Actual deaths	Ratio per cent actual to expected	Actual deaths	Ratio per cent actual to expected
Operatives not underground	14	159	†	—
Operatives underground				
Total	125	157	135	288
Pennsylvania (mostly anthracite)	†	—	115	334
Other localities (bituminous)	†	—	20	161

* Compiled from: Joint Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1929; and Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1938.

† Data not available.

study of tuberculosis in Framingham, Massachusetts (10), it was found that about 1 per cent were suffering from the disease in an active form, and another 1 per cent were classified as having arrested tuberculosis. Physical examination of 100,924 adult white males made by the Life Extension Institute (11) indicated a prevalence rate of about 1½ per cent when suspected cases were included. A somewhat higher percentage, namely 2½ per cent, was found by the Public Health Service (12) from the examination of 10,000 male industrial workers.

Among the anthracite workers examined, the clinical tuberculosis rate was below normal in the younger adult ages, but at ages 35 to 44 clinical pulmonary tuberculosis was diagnosed in about 5 per cent of the hard coal mining

high among certain groups of miners while among others the death rate from tuberculosis has been consistently low. Recent investigations have cleared up many points about the hazard of coal dust and have indicated certain differences between anthracite coal and bituminous coal. It is desirable, therefore, in studying the effects of coal dust to consider these two types of exposure separately.

In 1934, the Public Health Service (9) made a report on an investigation of pulmonary disease among anthracite miners. This report stated what had previously been suspected, namely, that anthracite mining had, under certain conditions, a silica hazard and many anthracite miners were exposed to the effects of coal and silica dust. The evidence tends to show that disabling miners' asthma is, in effect, silicosis, a silicosis modified by coal dust but nevertheless a silicosis.

Turning to the mortality record of coal miners, we find that the Medico-Actuarial Occupation Study (7) shows for the Pennsylvania miners (nearly all anthracite) the following figures, based on 144,535 life years, for the twelve-year period 1925–1936: There were 1,699 actual deaths where 613 were expected, giving a ratio of actual to expected deaths of 277 per cent. Of this excess, 37 per cent was due to accidents and 63 per cent to disease. The pneumonia and influenza death rate was five times the normal; tuberculosis, over three times the normal; and accidents, five times the normal.

Miners elsewhere (bituminous) presented the following figures: There were 52,522 life years and 330 actual deaths against 187 expected, a mortality ratio of 176 per cent, but of this excess mortality, 84 per cent was due to accidents and 16 per cent to disease. The death rate from accidents was five times the normal; the pneumonia death rate was normal; and the tuberculosis death rate, 61 per cent in excess of normal.

In the insurance occupation study (6, 7), as we have pointed out, the death rates for each occupation are compared with the rate for standard ordinary policy holders, a rigorous standard as compared with the rate for all occupied males. When considering the mortality of men employed in mining operations many of which are carried on by unskilled workers, it is especially necessary to bear this fact in mind. Apart from any specific occupational influence conducive to a high incidence of tuberculosis, we should expect a greater than average mortality from tuberculosis among them because of their economic status. Insured common laborers outside of the mining industry, it should be mentioned, have a death rate from tuberculosis about three times as great as that of Stand-

miners, but in the earlier study (6), covering the years 1915–1926, there were 11 deaths from tuberculosis in this group or eighteen times as many as expected. The ratio for iron miners, 857 per cent, is unexpectedly high inasmuch as it is thought that most of these workers are exposed to only moderate amounts of silica dust, except for a limited number who are working in hard rock. It might have been expected that their mortality would more closely approximate that of coal miners. The number of deaths is small, however, and the difference may be more apparent than real.

Among cutters of granite and sandstone, there were 38 deaths from tuberculosis in an exposure of 5,944 life years, compared with 1.4 expected, or more than twenty-six times as many as the expected number of deaths. Chippers of metal (exclusive of ship chippers) had 16 deaths from tuberculosis where only one was expected.

It will be observed that the ratio of actual to expected deaths from tuberculosis for each of the silica occupations is higher in the period 1925–1936 than in the period 1915–1926. The reason for this apparently lies in the difference in the trend of the death rates for men in these occupations and the rate for insured persons generally. While the figures are too small on which to base broad conclusions, they suggest that men in silica occupations have not shared in the general decline in the death rate from tuberculosis. It may be that there was actually an increase in the rates for the period 1925–1936 over that for the period 1915–1926.

Interesting confirmation of these high ratios by English data is presented in table 3. English tin and copper miners had a death rate from tuberculosis eleven and one-half times the average; sandstone masons, cutters and dressers, nearly four and one-half times the average; and metal grinders, about three and two-thirds times the average. Ratios such as these cannot be explained away on the ground of differing social classes or the selection of the occupation by physically weaker types of workers.

This evidence is in agreement with reports of silicosis studies from South Africa, Australia, Canada, and Great Britain as well as the United States and is supported by clinical experience of physicians in many parts of the world whose practice has been among workers exposed to silica dust.

Coal dust: It has long been known that coal miners are subject to chronic pulmonary disease characterized by dyspnoea and usually termed "miners' asthma," also that the death rate from respiratory diseases is

No useful purpose would be served by quoting extensively from the impressive volume of mortality data available to show the influence of silica dust on the incidence of tuberculosis. Virtually every one of the many studies is in agreement in showing extraordinarily high mortality rates from tuberculosis for industries and occupations in which large numbers of men are known to be exposed to a real silica hazard. These rates are so high, in fact, as to leave no room for doubt of the cause-and-effect relationship between the hazard and the high mortality. A few figures from the insurance investigations (6, 7) may be quoted. Table 1 shows the ratio of actual to expected deaths for the chief occupations exposed to silica dust.

TABLE 1

Number of deaths and ratio of actual to expected deaths from tuberculosis of the respiratory system

Ordinary Department Mortality Experience of American Life Insurance Companies*

Occupations exposed to silica dust

OCCUPATION	1915–1926		1925–1936	
	Actual deaths	Ratio per cent actual to expected	Actual deaths	Ratio per cent actual to expected
Stonecutters—granite and sandstone	16	976	38	2,639
Chippers of metal—(not shipbuilding)	8	615	16	1,667
Mine operatives—underground				
Copper mine operatives	24	913	29	1,381
Gold and silver mine operatives	9	804	11	940
Iron mine operatives	4	260	12	857
Lead and zinc mine operatives	11	1,833	3	—
Other and not specified mine operatives	†	—	10	1,020

* Compiled from: Joint Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1929; and Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1938.

† Data not available.

Among a group of underground miners employed in mines other than coal mines, nearly all of whom were employed in metal mines, there were 65 deaths from tuberculosis in the years 1925–1936 in an exposure of 25,000 life years where 6 were expected, or about eleven times as many deaths as the expected number. There were 13 deaths from pneumonia where 5 were expected. Tuberculosis deaths were fourteen times the expected among copper miners, nine times the expected among gold and silver miners, and eight and a half times the expected among iron miners. In this experience, there was only a small representation of lead and zinc

These are the Registrar-General's Decennial Supplement for England and Wales (5) and two studies made jointly by the Actuarial Society of America and the Association of Life Insurance Medical Directors (6, 7).

In the English study, the tuberculosis death rate for men in a given occupation, ages 20 to 65 years, is compared with that for all occupied and retired civilian males of the same ages. The method of analysis employed in the insurance studies was to compare the actual number of deaths which occurred among men engaged in a specific occupation with the expected number of deaths calculated on the basis of death rates by ages prevailing among standard lives, that is, persons who buy insurance on an annual basis in amounts of $1000 or more and who are not employed in hazardous occupations. Thus the insurance standard is a more rigorous one than that of "occupied males in the general population" since those in the lowest social-economic class are excluded, as are men who are employed in occupations where there is a serious exposure to dust, accident or other hazards. This should be kept in mind in interpreting the figures presented for insured lives in the following discussions.

INORGANIC DUSTS

Silica: Silica in the form of dust produces a definite, characteristic disease of the lungs, namely silicosis, a progressive fibrosis which, in itself, may cause disability and death and which carries with it a special disposition to tuberculous infection. Silicosis results from the inhalation of dust containing free or uncombined silica. Its pathology has been extensively studied, both clinically and in the experimental laboratory. It has been established that silica particles which penetrate the lungs are under ten micra in their largest diameter and are mostly from one to three micra. Not only must the silica dust be in a state of such fine subdivision, but the particles must be present in large amounts and the exposure of the individual must be prolonged. When such conditions are fulfilled the natural defenses of the body against inhaled dust break down and silicosis results.

The precise nature of the action of silica dust upon body tissues is not definitely known but it appears to be a protoplasmic poison and produces its effect chemically and not mechanically. A given dust is dangerous in proportion to the amount of free silica which it contains. The nature of the relationship between silicosis and tubercle infection has not been determined, but the fact of such relationship is attested by overwhelming evidence both clinical and statistical (8).

There are reasons, obvious to us to-day, why these mortality studies have not been an entirely accurate guide. For one thing, students of mortality did not have the benefit of the clinical and laboratory knowledge regarding dusts now available and, consequently, had to make their own classifications of dusts on a somewhat arbitrary basis. They were handicapped, too, by the fact that the existing occupational codes often brought together all the men in a whole industry. This did not permit of detailed studies of the mortality of men exposed to a single type of dust. Occupational mortality statistics still lose much of their value because of this same lack of refinement in methods of classifying occupations. Then again, factors other than dust, which have a marked influence upon the incidence of tuberculosis among men engaged in different occupations, were not as well understood and were not given due consideration in interpreting the results of mortality studies.

There are pitfalls in reasoning from cause to effect, especially where tuberculosis is concerned, and the error of *post hoc ergo propter hoc* is particularly to be guarded against. A high incidence of tuberculosis or other respiratory disease among men in a given occupation does not necessarily indicate the existence in that occupation of a definite occupational hazard. It is well established that people of the poorer economic classes, whether in industry or out of it, have a higher incidence of respiratory tuberculosis than do people better off financially. In Miss Whitney's (4) study of *Death Rates by Occupation in Ten States in 1930*, the standardized death rate for tuberculosis was over twice as high among unskilled workers as among all occupied males while in the Registrar-General of England and Wales' study (5), 1921–1923, the rate for unskilled workers was about two-fifths higher than the rate for all occupied and retired civilian males. Certain occupations, too, are more suited to the physically weak and are selected by them as a means of earning a livelihood. When the followers of an occupation are recruited from the ranks of either of these classes, it is to be expected that a high r incidence of tuberculosis mortality will be found among them. Failure to take cognizance of these factors as possible explanations of a high tuberculosis incidence in certain callings has led to much misinterpretation of the significance of mortality findings.

It is not our purpose to reassess the older statistical material to which reference has been made. We shall limit our discussion of statistics mainly to the results of the three most recent investigations in which tuberculosis death rates are obtainable for a number of occupations.

In 1908, Dr. Frederick Hoffman (1) wrote an article on the mortality from consumption in the dusty trades. Ten years later, he produced the well known Bulletin 231, *The Mortality from Respiratory Diseases in the Dusty Trades* (2), an important milestone in the hygiene of industry. In 1919, appeared the first and second preliminary reports of the Committee on Mortality from Tuberculosis in the Dusty Trades (3), of which Committee, Doctor Hoffman was Chairman. An indefatigable worker in many fields of public health, Doctor Hoffman's name is thus linked to the early authoritative publications in this country dealing with dust and pulmonary disease. The various reports of Governmental Commissions in South Africa, Australia, Great Britain and other countries have also stimulated industrial studies and laboratory research into the effects of many kinds of industrial dusts, especially those containing silica.

At the present time, while no one would state that we have adequate information about the effects of the inhalation of industrial dusts, we do know a great deal more than we did twenty years ago. Both clinical and laboratory studies have given us some knowledge of what kinds of dusts are dangerous, the circumstances under which they are dangerous, and the nature of their action upon the pulmonary tissue. We have also learned a great deal about the prevention and control of the dust hazard by engineering methods. But when we seek evidence of the effects of dust inhalation in mortality and morbidity records, we find that the statistical demonstration of mortality and morbidity due to the inhalation of dusts is anything but satisfactory or complete.

This situation is the more regrettable because comprehensive mortality and morbidity statistics would be of the greatest value in clearing up many doubtful points regarding the effects of specific dusts and would bring to light occupations in which there might be a real, but unsuspected, exposure to injurious dusts. Early occupational mortality studies, it must be admitted, gave the first broad clues to the extent of the dust hazard in industry and to the kinds of dust which are most injurious. These earlier mortality studies, valuable as they were, however, were in many instances misleading. Virtually all kinds of dusts were shown to be productive of tuberculosis rates higher than average, whereas recent clinical and laboratory experience points to the very considerable damage to the lung tissue produced by a few dusts, notably silica and asbestos, and to the relatively little evidence of harm done to the lung tissue by organic and many inorganic dusts.

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INDUSTRIAL DUSTS AND THE MORTALITY FROM PULMONARY DISEASE[1]

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Air and water are the two immediate vital necessities of our lives. We take extraordinary precautions to guarantee for ourselves not only an ample supply of water but one of defined purity, and vast engineering water-supply projects, costing many millions of dollars, are an accepted and commonplace fact in our times.

With respect to the air we breathe, we are more complaisant. Dust arising from industrial processes pollutes the atmosphere of working places, in mine, factory and mill, and the products of combustion, both industrial and nonindustrial, are liberated into the atmosphere of our communities with little restraint. In recent years, particularly, industrial dusts have received much attention and much effort has been put forth by industrial firms of all kinds to control dusty processes in their establishments.

Fortunately, nature has furnished us with a respiratory system which has not only a large margin of safety, but a fairly efficient protective mechanism. When that protective mechanism is subjected to severe stress for a sufficiently long period of time, it may fail. The extent to which such failure may be reflected in mortality experience is, within certain limitations, the subject of this discussion.

We are concerned here with industrial dusts other than those commonly recognized as poisonous. Lead, mercury, arsenic, manganese and other systemic poisons are excluded, together with the chemical poisons. Those dusts with which we are concerned are both organic and inorganic, and the latter, in turn, may be subdivided into metallic and nonmetallic. There are many possible subdivisions of these three classifications but for our purpose extensive subdivision is unnecessary. Indeed, it is difficult to get sufficiently ample statistical material to give us definite information under the general headings.

[1] Read as part of the Symposium on *Tuberculosis in Industry* at the joint session of the Pathological, Clinical, Social Work and Administrative Sections at the 34th annual meeting of the National Tuberculosis Association, Los Angeles, California, June 23, 1938.

[2] Me ropolitan Life Insurance Company, New York City.