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Underwriting Aspects of the Industrial Dust Hazard

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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-

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

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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 over 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[ignify] the total mortality, by increasing [evi]dence of tuberculosis, as to require a 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.

SURPLUS FUNDS

1. Metropolitan	$309,717,567	23. National Life & Acc.	10,453,570	49. Wash. National	3,263,533	75. Amer. Mutual, Ia.	[illegible]
2. Prudential†	209,392,460	24. Lincoln National	8,941,163	50. Minnesota Mutual	3,066,705	76. Colonial Life	[illegible]
3. New York Life	125,639,022	25. Equitable Life, Ia.	8,777,602	51. Sun Life, Md.	3,045,994	77. Industrial L. & H., Ga.	[illegible]
4. Equitable, N. Y.	90,762,655	26. State Mutual	8,625,351	52. Columbus Mutual, O.	2,705,395	78. Protective Life, Ala.	[illegible]
5. John Hancock	87,961,337	27. Phoenix Mutual	7,544,962	53. Ohio National	2,572,773	79. Country Life, Ill.	[illegible]
6. Travelers	64,902,795	28. Southwestern Life	7,540,000	54. Guarantee Mutual	2,578,074	80. American Standard	[illegible]
7. Northwestern Mutual	57,156,343	29. Kansas City Life	7,205,984	55. Midland Mutual	2,570,341	81. Indianapolis Life	1,512,306
8. Mutual Life, N. Y.	51,852,202	30. Fidelity Mutual	6,468,839	56. Amer. United, Ind.	2,500,000	82. Amicable Life	1,490,457
9. Aetna Life	36,555,402	31. Jefferson Standard	6,300,000	57. North Amer. Reassur.	2,413,961	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	[illegible]
11. Mutual Benefit	30,314,767	33. Pacific Mutual	5,672,956	59. Western Life, Mont.	2,350,000	85. Capital Life, Colo.	1,373,665
12. Western & Southern	28,383,923	34. Northwestern National	5,457,915	60. Home Beneficial	2,348,435	86. Union Labor Life	1,344,335
13. Mass. Mutual	27,050,794	35. Guardian Life	5,269,459	61. Continental Amer.	2,321,712	87. Oregon Mutual	1,318,523
14. Provident Mutual	19,874,280	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	4,788,020	63. Pilot Life, N. C.	2,285,976	89. Loyal Protective	1,301,507
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	1,300,377
18. Conn. General	14,428,687	40. Occidental, Calif.	4,152,034	66. Old Line, Wis.	2,211,275	92. Illinois Bankers	1,274,833
19. Conn. Mutual	13,020,498	41. Life & Casualty	4,077,235	67. Baltimore Life	2,154,771	93. Volunteer State	1,259,179
20. Union Central Life	11,848,155	42. Continental, Ill.	3,922,443	68. Ohio State Life	2,074,617	94. Atlantic Life, Va.	1,256,692
21. Bankers Life, Ia.	11,599,788	43. Central Life, Ia.	3,578,551	69. Presbyterian Min.	1,863,971	95. Girard Life, Pa.	1,242,320
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	1,834,663	98. Northern Life	1,082,901
		47. Provident Life & Acc.	3,283,576	73. Business Men's, Mo.	1,808,949	99. Mass. Protective	1,068,910
		48. Mutual Trust	3,231,715	74. New World Life	1,799,790	100. Lutheran Mutual	1,052,766

†Includes $128,801,620 contingency reserve.

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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 159.8 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

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