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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.

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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.

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.

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 of itself, may cause disability and death and which carries with it a definite 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).

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

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

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-

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

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.

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

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

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

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

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 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.

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.

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

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

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, nor in the light of clinical knowedge, is it possible to account for the excess mortality on the score of damage to the lung tissue caused by dust.

The relationship between dust inhalation and acute pulmonary disease 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.

REFERENCES

(1) HOFFMAN, F. L.: The mortality from consumption in the dusty trades, Bull. 79, U. S. Bur. Lab., November, 1908.

(2) HOFFMAN, F. L.: The mortality from respiratory diseases in the dusty trades, Bull. 231, U. S. Bur. Lab. Stat., 1918.

(3) HOFFMAN, F. L., Chairman: Preliminary report of Committee on Mortality from Tuberculosis in Dusty Trades, Working Conditions Service, U. S. Dept. Lab., 1919.

Second preliminary report of Committee on Mortality from Tuberculosis in Dusty Trades, Natl. Tuberc. Assn., 1919.

(4) WHITNEY, JESSAMINE S.: Death rates by occupation, Natl. Tuberc. Assn., June, 1934.
(5) Registrar-General's Decennial Supplement: England and Wales 1921, Part II, Occupational mortality, H. M. Stat. Off., London, 1927.
(6) Joint Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1929.
(7) Occupation Study, Actuarial Society of America and The Association of Life Insurance Medical Directors, 1938.
(8) SAYERS, R. R., Chairman: Report of the Committee on the Prevention of Silicosis through Medical Control, U. S. Dept. Lab., November, 1936.
(9) SAYERS, R. R., BLOOMFIELD, J. J., DALLAVALLE, J. M., JONES, R. R., DREESSEN, W. C., BRUNDAGE, D. K., AND BRITTEN, R. H.: Anthraco-silicosis among hard coal miners, Pub. Health Bull. 221, U. S. Pub. Health Service, 1936, p. 85.
(10) National Tuberculosis Association: Framingham community health and tuberculosis demonstration, Framingham Monograph No. 10, July, 1924, p. 69.
(11) SYDENSTRICKER, E., AND BRITTEN, R. H.: The physical impairments of adult life, Am. J. Hyg., 1930, *11*, 89, 100.
(12) BRITTEN, R. H., AND THOMPSON, L. R.: A health study of ten thousand male industrial workers, Pub. Health Bull. 162, 1926, p. 161.
(13) GARDNER, L. U.: Reaction of the living body to different types of mineral dusts with and without complicating infection, Tech. Pub. No. 929, Am. Inst. Min. & Met. Eng., May, 1938.
(14) PEDLEY, F. G.: Asbestosis, Canad. J. Pub. Health, November, 1930.
(15) MILLER, J. W., AND SAYERS, R. R.: The physiological responses of the peritoneal tissue to dusts introduced as foreign bodies, Pub. Health Repts. 49, January 19, 1934.
(16) COLLIS, E. L., AND YULE, G. U.: The mortality experience of an occupational group exposed to silica dust, compared with that of the general population and an occupational group exposed to dust not containing silica, J. Indust. Hyg., 1933, *15*, 395.
(17) MACKLIN, E. L., AND MIDDLETON, E. L.: Report on the grinding of metals and cleaning of castings with special reference to the effects of dust inhalation upon the workers, U. K. Home Off., H. M. Stat. Off., 1923.
(18) COLLIS, E. L.: Industrial pneumoconiosis with special reference to dust phthisis, Milroy Lectures, 1915; reprinted by H. M. Stat. Off., 1919.
(19) DRINKER, P.: Causation of pneumoconiosis, Harvard School of Public Health, 1937.
(20) CARLETON, H. M.: The effects produced by the inhalation of hematite and iron dusts in guinea pigs, J. Hyg., 1927, *26*, 227.
(21) Health protection in welding, Industrial Health Section, Metropolitan Life Insurance Co., (b) 513 L.W.
(22) DOIG, A. T., AND MCLAUGHLIN, A. I. G.: X-ray appearances of the lungs of electric arc welders, Lancet, London, 1936, *230*, 771.
(23) ENZER, N., AND SANDER, O. A.: Chronic lung changes in electric arc welders, J. Indust. Hyg. & Toxicol., 1938, *20*, 337.
(24) LANDIS, H. R. M.: The relation of organic dust to pneumokoniosis, J. Indust. Hyg., 1925, *7*, 1.
(25) SAYERS, R. R., AND MCFRIWETHER, F. V.: Miliary calcifications of the lungs due to unknown cause. Reprint 1431 from Pub. Health Repts. 45. December, 1930.
(26) FAWCITT, R.: Fungoid conditions of the lung, Part I, Brit. J. Radiol., New Series No. 99, March, 1936; Part II, Brit. J. Radiol., New Series No. 102, June, 1936.
(27) DICKSON, E. C.: Coccidioides infection, Arch. Int. Med., 1937, *50*, 1029.

(28) Iron foundry workers show highest percentage of deaths from pneumonia, Statistical Bulletin, Metropolitan Life Insurance Co., March, 1928, *9*, 3.

(29) BLOOMFIELD, J. J.: Engineering aspects of industrial epidemiology, Indust. Med., 1938, *7*, 7.

(30) KIBBEY, C. H.: Pneumonia and tuberculosis among industrial workers, Am. J. Pub. Health, 1937, *27*, 6.

(31) HAYTHORN, S. R., AND MELLER, H. B.: Pneumonia, Ibid., 1938, *28*, 483.

(32) THOMPSON, L. R., BRUNDAGE, D. K., RUSSELL, A. E., AND BLOOMFIELD, J. J.: The health of workers in dusty trades. I. Health of workers in a Portland Cement Plant, Pub. Health Bull. 176, U. S. Pub. Health Service, April, 1928.

(33) RUSSELL, A. E., BRITTEN, R. H., THOMPSON, L. R., AND BLOOMFIELD, J. J.: The health of workers in dusty trades. II. Exposure to siliceous dust (granite industry), Pub. Health Bull. 187, U. S. Pub. Health Service, July, 1929.

No commitments of any kind have been made by the Temporary Organization Committee as to the future policies or program of a permanent organization if and when one is formed, except that it has been arranged so that contributions and membership can become known or can remain unknown as may be elected by the individual contributor.

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In conclusion -

A large number of people have said that they believe that there should be a central clearing house, - or co-ordinating body - to represent all industry in connection with the industrial dust problem.

A Symposium has been held in Pittsburgh on January 15th, 1935.

A Temporary Organization Committee was elected to prepare a plan and recommendation as to a permanent organization.

This plan has been prepared and submitted (on February 6th, 1935).

Whether a permanent organization shall be formed, and how it shall be formed, and what shall be its program is now in the hands of those to whom this "narrative" is being sent.

Roger A. Hitchins
As Chairman -
Temporary Organization Committee.

MARKOWITZ

Egilman - By Plaintiff - Redirect

Anything else?

MR. BERRY: No, I think that is all I have, your Honor.

MR. CONSTANTINE: Nothing further, your Honor.

THE COURT: All right. You may step down.

(Whereupon the witness was excused.)

MR. PLACITELLA: I call Dr. Gerald Markowitz to the stand.

G E R A L D M A R K O W I T Z, Ph.D. residing at 160 West 97th Street, New York, New York, called as a witness by the Plaintiff, having been first duly sworn was examined and testified as follows:

DIRECT EXAMINATION

BY MR. PLACITELLA:

Q. Where do you currently live?

A. I live in New York City, Manhattan.

Q. Did you grow up in New York?

A. Yes, I did.

Q. Where?

A. In the Bronx.

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They wanted to do something about the health of workers in these dusty trades involved with silica and asbestos.

Q There is nothing wrong with that?

A Absolutely not.

Q That was a good motive?

A Absolutely.

Q Was there also a private purpose or private agenda?

A Yes. I think that the private agenda was they wanted to do something about these lawsuits. They wanted to find a way to combat these lawsuits which were hurting them financially, and that is what their private concern was, and that they did not emphasize so much in their public statements.

Q You have in front of you a document which has been marked for identification as Plaintiffs' Exhibit Number 139. Is this the same document?

A Yes.

Q Where did you find that document?

A I found this document in the National Archives, Washington.

Q The fact it has got a seal on it, what does that mean?

A It means that the Archive says that this is a true copy of the document in the National Archives.

Q What generally, don't get specific because we don't have time, what generally is the document about?

A The document is about the founding of the Air Hygiene Foundation.

Q That is one of the documents you relied upon in reaching your opinions?

A Most definitely.

Q Is that the kind of document that an historian like yourself would rely upon?

A Yes.

Q Do you know whether or not Owens-Corning Fiberglass was ever a member of the Industrial Hygiene Foundation?

A Yes, it was a member.

Q How do you know that?

A Well, in their membership lists that were published periodically by the Industrial Hygiene Foundation, they listed as a member, they say beginning in 1953.

Q You don't know about if there were other