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THE DEVELOPMENT OF KNOWLEDGE ABOUT ASBESTOS DISEASE

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The Development of Knowledge About Asbestos Disease

Part I. Asbestosis

The earliest recorded historical recognition of the hazards of asbestos goes back to the time of Christ.

> "Strabo, the Greek geographer and historian (63 B.C. - 24 A.D.), described the dangers of asbestos weaving, and Pliny the Younger (61-113 A.D.), in his description of the diseases of slaves, called asbestosis an occupational disease. Both writers stated that the use of asbestos in the manufacture of handkerchiefs, headcloths, and tablecloths was common, and that the cost of these articles was equal to the pearls of India." [1]

Pliny the Younger, a Roman historian, referred to the use of respirators to avoid inhalation of the dust by the slaves.[2] I infer from Pliny's report that the Romans realized that slaves weaving asbestos were first becoming disabled to the point that they could not exert themselves at all in their work, and then they died as their breathing difficulties became severe. The Romans instituted the use of respirators in order to extend the lives of these slaves, much as one might oil a machine to keep it from wearing out too quickly. The Romans did not have pathologists with microscopes or the science of radiology available to them, but nonetheless they saw the gross effects of asbestos inhalation on their workers.

The modern asbestos industry began in the 1870's, and commercial production of asbestos insulation products was recorded as of 1874.[3] Selikoff and co-workers wrote in 1965:

> "Asbestos, as a mixture of fiber and sodium silicate, was first used as an insulation material in 1866 and as asbestos cement about 1870. Magnesia with asbestos as a binder soon followed and air-cell covering, made with corrugated asbestos paper, was introduced in 1898. These materials are still in use, with others."

The first modern reports of asbestosis were in asbestos textile workers in England and France.[4,5] One of these, Murray's case, was not published in the general medical literature, but it was referred to in a 1918 bulletin published by the U.S. Department of Labor's Bureau of Labor Statistics (Hoffman).[6] Murray had presented his case report to the Departmental Committee on Industrial Diseases. Diagnosis was verified by a post mortem examination. Hoffman wrote:

> "In reply to a question by the Chairman of the Committee as to whether, in view of the fact that there is something characteristic in the earlier stages of dust phthisis* in the predominance of shortness of breath before physical signs become very obvious, such a condition had been observed in the case of the asbestos worker under treatment by Dr. Murray, and under observation for 14 months, the doctor said that it had been noticed in the case in question and that, in other words, there was a definite relation between the course of phthisis and the physical incapacity resulting from the inhalation of asbestos dust." (Emphasis added.)

Hoffman cited figures from the Prudential (insurance) Company that attributed 3 of 13 deaths of asbestos workers to tuberculosis. Of the 3 who died from tuberculosis, two died between the ages of 35 and 44 years, and one was between 25 and 34 years of age at death; two others died of pneumonia. Nine of the 13 asbestos

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*Phthisis is an old term for tuberculosis. Its use with a modifier, whoever, refers to a pneumoconiosis (e.g., stone cutter's phthisis).

workers died before age 45. Hoffman said that the mining and "dressing" of asbestos

> "unquestionably involve a considerable dust-hazard, but the hygienic aspects of the industry have not been reported upon. It may be said, in conclusion, that in the practice of American and Canadian life insurance companies asbestos workers are generally declined on account of the assumed health-injurious conditions of the industry."

He continued,

> "It is regrettable that there should be no further information available regarding the asbestos industry in its various branches, including the utilization of by-products of manufacture, on account of the self-evident injuriousness of asbestos dust as a predisposing cause of pulmonary tuberculosis." (Emphasis added.)

His style is cumbersome, but Hoffman appears to have been raising the question of health hazards in trades where asbestos products were used, such as insulation work.

That 3 of these asbestos workers' deaths were reportedly caused by tuberculosis and 2 were reportedly caused by pneumonia raised concern over the possible role of asbestos trapped in the lungs in causing these deaths. The data pointed to one or more of the following as possible roles for asbestos: 1) asbestos dust in the lungs might reduce a person's resistance to tuberculosis and/or pneumonia infections; 2) asbestos dust in the lungs might reduce a person's resistance to the progress of such infections, once they are contracted; 3) asbestos "phthisis" might be killing people itself and passing as tuberculosis and/or pneumonia. Although these are small numbers, it is significant indeed that life insurance companies were already refusing asbestos workers, and that Murray had identified a distinct

disease as the cause of death of an asbestos worker. There had also been a British government report on an excessive incidence of tuberculosis in the production of asbestos mattresses and the use of dust controls in 3 factories.

> "The relation of asbestos dust to pulmonary tuberculosis is reported upon at some length in the Annual Report of the Chief Inspector of Factories and Workshops for England and Wales for 1910. The investigation was made by Dr. Collis, who states, in part, that--
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> Following up information received from the registrar-general, it was found that five deaths of persons suffering from phthisis had occurred in five years among a staff of under 40 workers employed at a factory where asbestos is woven. The process which appeared most dangerous is the production of asbestos mattresses. These mattresses, which are composed of bags of woven asbestos filled with short asbestos fiber, are placed on a table and beaten out flat by a man with a wooden flail, from which process much dust arises. Women who sew the mattresses into sections with asbestos threads worked close to the man who beat the mattresses and of necessity inhaled the dust. The reorganization of this process with the application of localized exhaust draft was called for, and an annual medical examination of the workers by the certifying surgeon has been instituted in the hope of detecting and removing from exposure to dust those showing early signs of respiratory disease. Weaving asbestos has only become an important industry during the last 15 years. Two other large asbestos factories were visited, each of which was found to have its own specialty in the production of which dust prevention is required."

The Department of Labor bulletin contained numerous pleas for research on health hazards in the growing asbestos industry.

> "The rapidly increasing development of industries using asbestos, as ascertained from domestic or foreign sources, suggests the urgency of more qualified medical consideration than has heretofore been given the subject."

Hoffman also noted that there was no medical literature available on asbestos-related diseases from the Surgeon General's Library, the U.S. Geological Survey, or the state of Georgia where asbestos was mined.

"There is evidently an urgent need for a more qualified and extensive investigation of the health aspects of asbestos manufacture . . . ", he said, in the paragraph describing Murray's case report. The paragraph ends,

> "It is therefore to be anticipated that the conditions of asbestos workers will attract more qualified attention in this country in the future than it has in the past."

Also in 1918, Pancoast and co-workers reported finding fibrosis in the chest X-rays of 15 asbestos workers.[7]

The first report of asbestosis in the British medical literature was by Cooke in 1924.[8] In 1927 Cooke published additional details of his case, a 33-year-old woman.[9] Another case of asbestosis in Britain was reported by Seiler in 1928.[10]

In 1929, Haddow reported 4 fatal cases of asbestosis whose average age at death was 41.[11] He also had four "advanced" cases under treatment, women whose average age was 35.

> "I know that many workers had drifted away from the asbestos factory because they believed it was unhealthy, whilst many women had ceased work to attend to their homes, and any fibrosis present would readily be overlooked in later years when death overtook them."

Thus, the asbestos workers were apparently able to see (from the impaired condition and deaths of those longest employed at the factories, no doubt) that grave dangers were attendant upon

dusty jobs in asbestos plants--they determined this without the aid of sputum cytology, pathology, epidemiology, or radiology.

In 1928-1929 Merewether and Price of the Factory Department in Great Britain investigated the health of workers exposed to asbestos. The asbestos textile industry was a logical place to conduct the study, because these workers' exposure was to pure or nearly pure asbestos. (No other potentially fibrogenic dusts were present.) In addition, dust exposure did not fluctuate as much as in other asbestos trades; and exposure in the asbestos textile industry was substantial. Further, such mills were centers of relatively large groups of workers. They had employed many individuals for long periods of time, thus giving the researchers an opportunity to observe the effects of prolonged exposure.

Merewether and Price examined 363 workers and found that 95 (26%) had asbestosis and 21 others had "precursive signs" of it.[12] Based on these findings, the authors wrote,

> " . . . it appears that the general incidence rate of fibrosis of the lungs amongst those employed in these sections of the industry is rather less than 1 in 8, or, excluding those employed under 5 years, rather less than 1 in 3."

These investigators analyzed the workers' exposures by lengths of time and dustiness of job, and summarized their findings thusly:

> "While it seems necessary for the production of generalised fibrosis of the lungs that a definite minimal quantity of dust must be inhaled, the lower the concentration of dust in the air breathed, the longer the lapse of time before the fibrosis is fully developed, and within a certain limit, the higher the concentration of dust, the sooner the fibrosis becomes fully developed and the more intense the involvement of the lung tissue.

> "If this hypothesis is correct, and the evidence points to it, the practical inferences are of very great importance, since it follows that the application of measures resulting in the reduction of the concentration of dust in the air in the neighbourhood of dusty asbestos processes will cause, first a great increase in the length of time before workers develop a disabling fibrosis, and secondly, the almost total disappearance of the disease, as the measures for the suppression of dust are perfected."

J. C. McVittie, of Great Britain's Ministry of Pensions and National Insurance, described the reaction to this report, which contained specific recommendations for improved ventilation and dust suppression:[13]

> "Under section 79 of the Factory and Workshops Act of 1901 the Secretary of State issued a certificate to the effect that 'the manipulation of asbestos and the manufacture and repair of articles composed wholly or partly of asbestos and processes incidental thereto are dangerous.' The section in the Workmen's Compensation Act of 1925, which provided for the application of ~~that Act~~ to workmen suffering from silicosis was extended to certain processes involving exposure to asbestos dust, and ~~in 1931 asbestosis became~~ a compensable disease. Compensation cover was fairly wide and the compensatable disease was defined as 'fibrosis of the lungs due to asbestos-dust or that disease accompanied ~~by tuberculosis.~~'"

Regulations for exhaust ventilation, dust control, and housekeeping measures in asbestos plants were made effective on March 1, 1932.

In 1930, the same year the above-mentioned report was issued in Great Britain, Merewether published a lengthy article in the American publication Journal of Industrial Hygiene.[14] He described his investigation of the British asbestos textile industry. The article began with a description of Murray's 1906 asbestosis case report, from which a grim picture of the early asbestos textile plants emerges:

> "The patient, a male aged 33, came under the care of Dr. Montague Murray at the Charing Cross Hospital in the beginning of 1899. He had worked with asbestos for 'some 14 years,' 10 years as a cardroom hand and the remainder in some other room of the factory 'where there was less dust.' He volunteered that, of the 10 people working in the cardroom when he went into it, he was the only survior, and that all the others had died somewhere about 30 years of age . . . He was treated in the Charing Cross Hospital for two months, and then returned to work. After a few months, however, he became ill again, and was re-admitted to the Hospital in April, 1900, where he died. The post mortem examination confirmed the clinical diagnosis of extensive pulmonary fibrosis. There was no evidence of pulmonary tuberculosis . . . "

He described the terminal stages of asbestosis:

> "As the disease progresses, if no acute illness has caused a fatal termination, a stage is reached when the lungs can do little more than maintain life; and the shortness of breath is extreme. Even in its terminal stages, the disease, deceitful to the last, may masquerade as chronic bronchitis, pulmonary tuberculosis, bronchopneumonia, or the like."

Merewether noted the limitation of studies based on those still healthy enough to be found at work in the factories. He pointed out that workers with asbestosis who develop a tuberculous infection would have cause to go to a doctor. Such workers would likely be told to quit their dusty jobs:

> "Thus, there tends to be a drift of such cases from the fibrosis-producing industry.
>
> "During the course of the inquiry, information was obtained of a number of persons, previously employed in asbestos, who were either at home or in sanatoriums, suffering from chest complaints. Where an examination of workers is confined to those at work, a certain number of advanced cases of fibrosis, and a certain number of pulmonary tuberculosis, with or without an asbestos fibrosis, in persons who have either given up work, or who are off work temporarily, will be missed."

In presenting his statistics, Merewether said that comparatively good exhaust ventilation,

> "which has been applied in some degree for more than seventeen years, has been a positive factor in minimizing the production of fibrosis--probably in the direction of lengthening the period before the fibrosis becomes fully developed."

The statistical highlights of Merewether's findings were also reported in the Journal of the American Medical Association on June 28, 1930, at the time when Lord Russell had just introduced a bill in Parliament to extend the silicosis provisions of the Workmen's Compensation Act to cover asbestosis as well.[15]

> "Supporting the bill, Lord Russell for the government said that many processes in silica working could not show a mortality approaching that for the asbestos industry, in which 14 fatal cases had now been reported and in addition 3 deaths from asbestosis associated with tuberculosis."

The same journal had run its first article on asbestosis in January, 1928, to bring Cooke's case to the attention of American physicians.[16] In 1930, the Journal of the American Medical Association noted Mills' report of an asbestosis case. This report had been published in the journal Minnesota Medicine, about a fatal case in a man who had worked in an asbestos mine in South America prior to 1898:[17]

> "Concerning the relation of physicians of the United States to this industry, Mills pointed out that asbestos is mined and manufactured in many parts of this country, and that pulmonary asbestosis surely will be encountered."

Also in 1930 Pedley published the first Canadian paper on asbestosis.[18] He remarked that the asbestos mining and milling industry was 40 years old in Canada, but that

> "[t]o our knowledge, however, no case of pneumoconiosis has ever been reported from asbestos in Canada, and the general impression among miners is that the dust is not hazardous."

The writer acknowledged that asbestosis had been recognized by the British authorities as a compensable disease, citing the notice on Lord Russell's bill before Parliament in the Journal of the American Medical Association. He pointed out that

> "Mining is carried out in open quarries and is not usually a very dusty job, but in the milling a great deal of fine dust is created, and men who work there are heavily exposed to whatever hazard may exist."

He provided no figures on the numbers of people employed in mining and in milling asbestos. He acknowledged that

> "it is quite possible that the condition in mines may have gone unrecognized, for mines are usually situated in the country, whereas factories are apt to be in the city where facilities for autopsies are better."

Note that mills* are usually located adjacent to mines, since it is most economical to separate the fiber from the host rock and sort it by grade (length) before transporting it. Pedley suspected that

> "if the miners were seriously affected by asbestos the effects should be reflected in a high tuberculosis death rate, provided the mining population was fairly stationary and did not go to sanitoria or elsewhere to die."

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*These mills process ore and are not the same as textile mills, where manufactured goods are made.

He went on to compare the 1926-1928 tuberculosis death rate from Thetford mines, Quebec, an asbestos mining center, to the death rate for the Province of Quebec as a whole--and found they were very nearly the same. The numbers of deaths on which the rates were based were not given.

Pedley then described the asbestos bodies that had been reported in published autopsy examinations of the lungs of asbestotics (variously referred to in the early literature as "asbestosis bodies" and "curious bodies").

> "Unquestionably these 'curious bodies' have stimulated much interest in the disease, asbestosis. Pathologists, whose interest in such a disease might languish, come alive at the sight of the 'curious bodies.' From the public health standpoint, however, it seems hardly likely that asbestosis will become of importance either from the standpoint of morbidity or mortality."

Pedley, in dismissing asbestosis as a pathologist's disease, was compounding slipshod research with wishful thinking. As he noted, the population at greatest risk of asbestosis was the mill workers. Yet he did not find out how many of these people there were. If the mill workers made up only 1-2 percent of the town's entire population, which is likely, then there is little chance that their mortality from tuberculosis, no matter how bad, would show up as an excessive tuberculosis rate for the whole town. Another serious error was the use of tuberculosis rates as a sole index: asbestotics usually died of heart failure, pneumonia, chronic bronchitis, or tuberculosis.

The failure of physicians in this Canadian mining town to recognize asbestosis among miners and mill workers is typical

of the situation the world over. The Americans had just begun to learn about asbestosis in textile mill workers from the British in spite of the long history of the modern asbestos manufacturing industry in England and in this country. And even the British doctors likewise took a very long time to recognize and investigate the threat of a fatal occupational disease faced by asbestos workers since the 1870's. Finally, one can hardly overlook the plight of the small-town physician who discovers that the industry the town depends on is killing people. If he makes an announcement to that effect, he might find himself branded an enemy of the people.

Pedley listed in his bibliography 17 articles on asbestosis, including Simson's 1928 paper on two cases in Southern Rhodesia. In the text Pedley mentioned the existence of reports of asbestosis in "four Rhodesian miners." Simson's two cases were men who had worked in a mill processing chrysotile asbestos fiber from a nearby mining operation. Chrysotile asbestos, by far the most abundant of the commercial asbestos minerals, is the same type of asbestos that is mined in Canada. Pedley did not attempt to explain how milling chrysotile in Canada presented no asbestosis hazard, while milling this same mineral in Rhodesia and weaving Canadian asbestos in England could both cause asbestosis. Whether Pedley actually read all of the papers in his bibliography is questionable--especially the last one, Merewether's 1930 publication in the Journal of Industrial Hygiene.

In December, 1930, Dr. J. V. Sparks of London journeyed to Los Angeles to present a major paper on asbestosis at the annual meeting of the Radiological Society of North America. The

paper was published the following year in the American journal Radiology.[19] Sparks remarked that "practically no literature on [asbestosis] has been published in America" from 1925 to 1930. Sparks called attention to the "striking . . . preponderance of workers who have been employed in the [British] asbestos factories for a period of four years or less," referring to Merewether's finding that fibrosis begins to show signs in some cases after the first four years of exposure:

> "It seems reasonable to suppose that after some years of such employment the workers may become affected by the inhalation of asbestos dust and seek other industries. The fact that they cease to be exposed to the dust does not, however, prevent the disease, when once established, from progressing."

Thus, the radiologists were the first to establish the startling fact that in some cases asbestosis became more serious as time passed, even for workers who had been removed from workplaces where they were exposed to asbestos. Not only did these workers fail to recuperate, but their health continued to deteriorate as the asbestos fibers trapped in their lungs produced a steadily increasing fibrosis.

Sparks said,

> "It is too early in the study of this disease to say very much on this subject, but once the asbestos bodies appear in the sputum the course of the disease would appear to be progressively downwards, nor does cessation of exposure to the dust avail to check its spread . . . Prophylaxis is all-important and the only hope for the asbestos worker lies in adoption of the proper means of protection against the risk attendant on the inhalation of the fibers."

At the end of 1930, Dr. W. B. Soper published a case report of asbestosis from Connecticut, in the American Review of Tuberculosis.[20] Noting the dearth of American medical literature

on asbestosis and acknowledging the flurry of British publications on the subject, he wrote:

> "Just as certain other dusts took their toll over a long period before the medical profession awoke to the true facts, so asbestos dust to a lesser degree appears to be taking its toll without sufficient recognition of the fact on the part of physicians."

In 1925 radiologists Pancoast and Pendergrass of Philadelphia reported X-ray abnormalities among asbestos workers they examined.[21] They published "A Review of Pneumoconiosis" in 1931.[22] In addition to some cases mentioned above, they described asbestosis case reports from 1927 to 1929 by Oliver and by Wood and Page, as well as the extensive study by Merewether and Price. They noted the progressive nature of asbestosis:

> "The condition is apparently progressive even after cessation of occupation. Merewether and Price state that with continued exposure to high concentration dust fibrosis may be fully developed in from seven to nine years, and death may result in about thirteen years. With less concentration, fibrosis may not be fully developed for 15, 20, or 25 years. Inhalation of dust in high concentration results in a more marked degree of fibrosis in a shorter time than when the concentration is lower."

Another case report was published in 1931 by Lynch and Smith in Charleston, South Carolina.[23] The report began,

> "For a number of years, one of us (W.A.S.) has observed in the chest clinic at the Roper Hospital (Charleston) and at clinics held at certain sections of Charleston County, cases of respiratory disease which possessed characteristics not ordinarily encountered . . . The significance of the relationship of the occupation and the pulmonary disability was realized when in the fall of 1927 an adult male, about 40 years of age, who had worked for 17 years in an asbestos plant, was seen in consultation with a local physician. The man exhibited the characteristic picture of the terminal stage of respiratory failure."

A literature review and the case report followed. In their

survey of the available literature the authors counted a total of 172 cases of pulmonary asbestosis.

Stewart and co-workers in Philadelphia reported two more cases of asbestosis, in 1931.[24]

> "The two cases cited illustrate nicely the extremes and some of the features of the disease. In the first case, as far as the patient's health and life were concerned, the presence of pulmonary asbestosis in the lung was relatively unimportant. He had symptoms doubtfully attributable to it. It was only through the history, which was confirmed by postmortem examination, that the possibility of such a condition was suggested. It is, however, important to point out two considerations in this case. First, it is a question whether the asbestosis had anything to do with his emphysema and cough. As an etiologic* factor, the asbestosis cannot be ignored. The other point is one that has to do with preventive medicine. The exposure was for nine years in a comparatively well ventilated factory in which precautionary appliances were used, and yet the patient developed a minor grade of the disease. It would seem that further precautionary means are necessary in this occupation to obviate a disease in itself so preventable.
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> "In the second case, the patient worked in an asbestos factory for a short time, but without a mask. This was long enough to permit his lungs to become densely filled with the dust. Obviously the symptoms did not result from the asbestos per se, but were due to the marked fibrosis resulting from the deposition of the pigment. This demonstrates that the symptoms of a pneumokoniosis may come on years after a short exposure and emphasizes again the absolute necessity for wearing masks in even the less dusty atmospheres."

Once again, the insidious progress of the disease after cessation of exposure is apparent, especially for the second case, a very severe one. This report showed that high asbestos

*etiologic: causative.

exposure for 9 months could later prove fatal, and that 9 years of textile "spinner" work under conditions of ventilation and respiratory protection regarded as good for those times did not prevent asbestosis. The result of a year or less of high exposure was not observable to Merewether, since the asbestos workers he examined with one year's exposure had first been exposed to asbestos only one year before he saw them.

Stewart and co-workers ended with the statement,

> "From the histories there appears to be a further need for study along the lines of prevention of this disease."

Wood and Gloyne published a paper on the histology of asbestosis in 1930.[25] As many others would note, they found that the fibrosis was most marked at the bases of the lungs.

A later paper by Gloyne describes damage to the pleura as well, and even one case of pleural cancer with asbestosis.[26] Stewart et al.[24] remarked in 1931:

> "The marked fibrosis of the pleura, particularly of that between the base of the lung and the diaphragm, is characteristic."

In Germany, similar findings to those in England were reported on the clinical course of asbestosis in 1931-1932. A summary of early German literature appeared in the British journal Lancet[27] in 1932. Kruger and co-workers reported, among 48 workers examined, 30 cases of "definite lung changes" and "cough in 13 of the cases," and dyspnea "the commonest initial complaint."

> "The statistical tables appear to indicate that moderately severe asbestosis takes some five years to develop, while none of the workers examined who had had ten years or more exposure was free from signs of the disease." (Dresden, published 1931.)

Gerbis and Ucko examined 33 asbestos workers in another German factory in Berlin, and found that "[n]early all the cases showed symptoms." These symptoms were shortness of breath.. especially on exertion; cough with sputum; lack of appetite; loss of weight; pain in breathing; palpitation of the heart; faintness; and increasing pallor.[27]

> "The authors noted that the severity of the disease did not appear to be dependent on the age of the worker, but rather on the number of years he had been in the factory, and on the amount of asbestos dust inhaled, which varies considerably in different processes. Whenever a patient had been working for more than ten years, asbestosis could be demonstrated radiologically." (1932)

In a series of only 8 cases from Dresden, it was noted that the radiograms in the early stages of the illness were "especially obvious" in the lower lobes of the lungs.[27] These authors found that

> "the more serious cases did not necessarily tend to get worse after removal from their harmful surroundings--in which their experience appears to have differed from the early observations of W. Barton Wood in this country."

Wood later reported (1934) that he had seen asbestosis cases that did not worsen after cessation of exposure, though many others did.[28]

In the July 1933 issue of the American publication Journal of Industrial Hygiene, Dr. Philip Ellman described the first case of asbestosis in an insulation worker to appear in the medical literature:[29]

> "A ~~typical lesion of~~ mild activity, with an early stage of pulmonary asbestosis, was present in one of my cases who had been exposed to asbestos dust for 10 years; but the work, which consisted in coating lead pipes, did not entail exposure to high dust concentrations."

Ellman's description of the asbestotic was vivid:

> "[A] case of simple silicosis often looks well, declares that he is well, and is even offended when any reflection is cast upon the soundness of his lungs. In contrast to this, a case of asbestosis is cyanotic, emaciated, anxious, and often obviously going downhill. Such then, is the clinical picture of sufferers from asbestosis.
>
> "Only by an extension of meaning could the word 'sufferer' be applied to anyone with simple silicosis." (Original emphasis.)

Ellman described the progressiveness of the disease:

> "An interesting feature of this disease is the length of time which may elapse between exposure to the dust and a fatal termination, and the fact that this period is only one-half of that in silicosis . . . The dust particles, once they have gained access, continue to injure the lungs, and the disease is a progressive one, which, if sufficient dust is present, ends fatally, the end being determined by some inter-current complication, such as acute bronchopneumonia or phthisis."

He also noted that diagnosticians would find the earliest development of asbestos fibrosis at the bases of the lungs.

The following year he again described the case of asbestosis in an insulation worker in a paper published in the British Journal of Radiology.[30] Ellman's first paper on case reports of asbestosis had been issued several years before.

In October, 1933, a report of an asbestosis case appeared in the Medical Bulletin of the Veterans' Administration.[31] The patient died at the age of 29 after 8 years of work in the dusty "carding" operation at an asbestos textile mill.

That same month Donnelly presented a paper at the annual meeting of the American Public Health Association in Indianapolis, which was later published in the American Journal of

Public Health.[32] Donnelly was a physician at the Mecklenburg Sanitorium in Huntersville, North Carolina, and had the opportunity to examine many workers from nearby asbestos textile plants.

> "Having had occasion some years ago to familiarize myself with the conditions in asbestos factories, I have since been intensely interested in determining the extent of industrial hazard in this type of work. For that reason I have attempted to obtain X-ray films of the lungs of all asbestos workers who have been referred to the clinic because of respiratory symptoms. Of the films taken, only 3 failed to exhibit some definite evidence of the condition called pulmonary asbestosis."

Donnelly collected 15 cases of asbsestosis over a 3 to 4 year period. He warned,

> "That exposure to the inhalation of this dust for even a comparatively short time is a definite and serious industrial hazard, has been too frequently indicated to be open to doubt. The fact that the condition when once acquired is permanent and more or less rapidly progressive is most important from a public health viewpoint."

He continued,

> "It also seems to be the consensus of opinion, not only among writers on the subject, but also among the asbestos workers, that the protective devices now in use in many plants are most inadequate."

He went on to contrast the British Asbestos Industry Regulations with their requirements for dust suppression and annual medical examinations of workers, with the lack of regulations in the United States and the lack of recognition of asbestosis in North Carolina's compensation laws. After acknowledging the great usefulness of asbestos, he said,

> "[T]he workers should be provided with every facility for the protection of their health, so that they may earn a livelihood for themselves and families, for at least an average period of time, at the occupation in which they are most skilled. Unfortunately, every worker handicapped by pulmonary asbestosis becomes almost invariably physically incapable of engaging in any occupation, and many of them become an expense to the welfare agencies of the community. Although the number of asbestos workers is much less than that in many other industries, their occupation is extremely hazardous, and they are amply justified in expecting whatever protection it is possible to give them."

He closed with a warning to factory owners:

> "Furthermore, the fact that efficient protective devices in this industry, in spite of the added expense, will effect a substantial financial saving, is becoming more apparent. The workers themselves are becoming informed of the danger to health, and many civil suits for damages against factory owners are the result."

In December, 1933, Merewether published a lengthy description, [33] "A Memorandum on Asbestosis". He posed the question, "Does asbestosis shorten life?" and answered it, "emphatically, 'Yes.'" He presented figures showing that of 42 fatal cases of asbestosis, the average age at death was 41. The youngest of the asbestosis cases died at age 26. One of the asbestosis cases died only 2 days after last exposure to asbestos.

> "The average duration of employment sufficient to cause a fatal degree of asbestosis was only 15.2 years, as compared with 40.1 years for all cases of silicosis."

He not only presented statistics, but also interpreted them based upon his experience:

> "While these figures show clearly the increasing risk with continued employment, it would be wrong to assume that the opposite inference which the figures appear to support--that so long as the period of exposure does not exceed five years the risk of contracting asbestosis is almost negligible--is correct.

"Unfortunately, that inference is wholly untenable. The fact is that work in a dense concentration of asbestos dust over a comparatively short period will lead inevitably to the development of a profound fibrosis, provided that the worker lives long enough for it to develop. It has been shown that experimentally in guinea-pigs fibrosis due to inhaled asbestos commences after about five hundred days' exposure, and the inhaled dust trapped in the respiratory bronchioles remained localised there for at least two-and-a-third years (the length of the experiments up to the time of the report).

Of the fatal cases in Great Britain of which full particulars are available, in two, examination over fourteen years after the last exposure to asbestos revealed numerous asbestosis bodies. Since these bodies are merely altered asbestos fibres and still contain an internal core of asbestos, and since unaltered asbestos fibres also can be found in the lungs years after the last exposure, it is evident that asbestos dust trapped in the lungs remains and continues to exert its fibrosis-producing powers for at least many years."

Merewether described the variable maturation period of asbestosis:

"It follows, therefore, and this is important from the practical point of view of prevention, that a certain minimum 'fibrosis-producing amount,' as it were, of asbestos dust must be trapped in the lungs in order to produce a potentially disabling or serious amount of fibrosis, and also that a certain 'maturation' period must elapse before that amount of fibrosis is developed . . . "

"Particulars of cases seem to show that with high concentrations of dust the minimum period of time which must elapse between the commencement of exposure and the production of a serious degree of asbestosis is approximately seven years--which include not only the trapping period of the fibrosis-producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap.

"The existence of this, which may be referred to as the fibrosis-producing period, i.e., the

period which must elapse from the date of commencing work before a serious degree of asbestosis can be produced, accounts for the fact that it is not until the second five years of employment is reached that appreciable numbers of cases of asbestosis are discovered.

"This period of seven years is, of course, the minimum, and few cases mature in this minimum period; in successive years, however, depending on the dustiness of the process in which employed, more cases mature. In the more dusty processes the fibrosis-producing period, as defined above, is commonly eleven years.

"At this point, i.e., when fibrosis of serious import has matured--the worker is unduly short of breath on any extra exertion, has a tinge of cyanosis of the lips, and a little dry cough, mostly in the mornings. He is disinclined to climb stairs or walk up hills and he may even have changed from an upper flat or room to a lower one to avoid climbing the stairs. He still, however, remains at work and usually is not anxious about the state of his health; many refuse to admit any deterioration in health at this stage . . . "

"Examination of the average length of employment of cases of asbestosis, of the radiograms, and of the particulars of the recorded fatal cases, also indicates that not only are 'spinners' less likely to contract asbestosis, but when they do, it takes longer to develop; in other words, in 'spinners' the maturation period of the fibrosis is longer."

In light of this recognition of the progressive nature of asbestosis, he held out little hope for the victims of asbestosis:

"The ultimate prognosis in those with developed asbestosis or in those who have incarcerated in their lungs a fibrosis-producing amount of asbestos dust, even though no appreciable fibrosis or only a slight amount can be detected, is bad. Death from the disease is inevitable sooner or later, if life is not cut short by accident or by some unconnected disease."

In 1934, Wood and Gloyne published a review of the first 100 cases of asbestosis they had seen.[28] Among the 67 women in their series, "The shortest exposure was six months (2 cases) . . . " Most of the men and all of the women in this series worked at the same factory. "Among the men the shortest exposure was 11 months . . . " Thus, these authors reported seeing at least 3 cases of asbestosis resulting from less than one year of occupational exposure to asbestos. Presumably, these individuals worked at jobs with heavy exposure to airborne asbestos fibers.

The case with the longest exposure is also interesting:

> "That even serious damage to the lungs is not incompatible with many years of tolerably comfortable existence is proved by such an example as that of a patient who was still able to carry out administrative duties at a factory when nearly 40 years' exposure to the dust had caused a fibrosis involving all except the apical regions of the lungs."

It is reasonable to consider the import of this case with regard to the asbestosis risk of insulation workers. This case is described later in the paper as having had "about 40 years (clerical and administrative work)" at an asbestos factory, as of 1934. Would he have had any higher exposure to asbestos than an insulation worker?

The authors provided brief, individual discussions for only a handful of cases. They noted two that resulted from outdoor exposure to asbestos:

> " . . . a van boy, aged 18, whose spare time during two and a half years' employment had been spent in mixing powdered asbestos in an open yard . . . "
>
> " . . . a man who had been employed handling asbestos mattresses in the open air at an aerodrome.*"

Again, one wonders how these exposures compared with the exposures of insulators. Insulators worked in the open air at times, but at other times worked in very confined enclosures where the airborne dust concentrations could "accumulate" in the course of doing a job.

The next example is the most relevant of all to insulation work:

> " . . . a middle-aged boiler riveter who had served his apprenticeship as a youth in a shop where asbestos was used for lagging pipes . .. "

It was not stated that this man ever applied insulation to pipes himself, but if he did it could have been only intermittently during his (several-year?) apprenticeship for the boiler riveting trade. He did work in close proximity to insulation workers during his apprenticeship and thus was exposed to the dust this work created. He may never have done insulation work himself, but he probably did a little. He may have been exposed to dust left in the air around boilers from which the old insulation had just been torn off. Or he may have worked mostly or exclusively in the manufacture of

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*"aerodrome--Brit. for airdrome; a landing field for airplanes which has permanent or extensive buildings, equipment, shelters, etc." The American College Dictionary, Random House, New York, 1956.

new boilers, thus having little or no opportunity to be exposed to asbestos after his apprenticeship. But whatever the facts were, one conclusion can be drawn: This boiler riveter surely was exposed to less asbestos than an insulator who spent his entire working life on boilers--tearing off old, dry insulation, outfitting boilers with asbestos "mattresses," and mixing and applying asbestos-cement powders for new insulation. Much of the insulation of boilers and pipes was done in confined areas; it was not outdoor work. It is an inescapable truth that if this boiler riveter could develop asbestosis, so could many insulation workers.

The authors noted that by 1934 they had seen some cases of the disease which did not worsen after exposure had ceased:

> "In a previous article we stated that the cessation of exposure to asbestos dust did not avail to check the spread of the disease. This is certainly often true, but we have now seen patients whose condition appears to have remained stationary since stopping work at the factory."

This report mentions that two of the asbestosis cases in the series also had carcinoma of the lung. The following year Gloyne issued a separate report on these two cases of lung cancer.

The asbestos industry produced a study of its workers in 1935, conducted by Lanza and co-workers of the Metropolitan Life Insurance Company.[34]

> "In 1929, the Metropolitan Life Insurance Company was approached by officials representing the asbestos industry in the United States, who were desirous of ascertaining whether asbestos dust was an occupational hazard in their establishments and, if so, what was the nature of this hazard and what should be done to prevent or control it."

The investigation was conducted during the period ending in January 1931, but was not published until four years later.

> "X-ray films were made of 126 persons (108 men, 18 women) working in asbestos plants in the United States. All but five of these were given physical examination. The cases were selected more or less at random from among those having more than 3 years of employment in the industry . . . . "

No further clarification was given on the meaning of "selected more or less at random." Thus, the asbestos industry's role in the selection process remains obscure, and it is not possible to draw inferences on the health status of the workers industry-wide from this report.

> "The (X-ray) films were read conservatively, taking into account the physical examination and the age of the individual, and were classed as positive only when there was no major disagreement (among the examiners of the X-rays) . . . "

> "The films classified as negative for asbestosis were further subdivided into doubtful and negative."

They went on to say without elaboration,

> "Only time can tell whether the individuals classed as doubtful are progressing toward a definite asbestosis."

This last remark disregards the extensive work of Merewether and others. Though allusion is made to "several articles in the English medical journals," the only British paper actually cited was Cooke's 1927 paper on his historic case.

The authors went on to say that the definite cases of asbestosis were broken down into first stage and second stage, the latter being the more extensive development of the disease.

> "Of the total of 126 X-ray examinations, 4 were diagnosed as second degree asbestosis, 63 as first degree, 39 as doubtful, and 20 as negative."

In other words, 53 percent of the workers had asbestosis by a conservative reading of the X-rays, and another 31 percent had some X-ray signs of disease ("doubtful"). Only 16 percent had no signs of asbestosis on X-ray. Most of the persons classified by X-ray as "doubtful" and "negative" complained of cough and dyspnea.

> "Of the 64 persons given physical examination and diagnosed as having positive asbestosis, only 8 were entirely free from symptoms, while 10 out of 37 with doubtful and 7 out of 20 with negative diagnoses were free from symptoms."

Referring to those that were classified as "negative" by X-ray who complained of symptoms, the authors said, "too much emphasis should not be placed on statements of subjective symptoms."

The authors minimized their finding of widespread asbestosis in American asbestos workers by noting that, in the cases selected for examination, none had "marked disability." The threat of progressive maturation of fibrosis noted by many previous authors was not discussed; one of these authorities, Dr. Pancoast at the University of Pennsylvania, had even been consulted by the Metropolitan Life investigators in the interpretation of X-ray films.

The Met Life study ignored the fine statistics of Merewether and offered no data of its own to support the emphasized part of the following statement:

> "[W]e are not justified in assuming that because available information suggests that asbestosis is a milder disease than silicosis, the threshold of permissible dust counts is higher." (Emphasis added.)

The authors did not recommend that the asbestos industry should be regulated and did not mention that regulations were in effect in England. The insurance company officials neglected to mention that asbestosis had been declared a compensable disease several years before in England. They said this:

> "[T]he experience so far does not warrant an attempt to define a standard of dustiness for asbestos dust."

A. J. Lanza, who directed the Met Life study, maintained cordial relations with the asbestos industry until he died in the early 1960's, and his 1963 book, The Pneumoconioses, has a chapter on asbestosis that was contributed by K. Smith, Medical Director of the Johns-Manville Corporation.[36] In Lanza's book terminal asbestosis was referred to as "respiratory embarrassment".

I point this out to make clear that this report was the result of collaboration between the insurance company and the asbestos companies in an attempt to downplay the published findings of hazards to workers in the asbestos industry. Even so, the report's findings were very worrisome. The paper was presented by the authors as a preliminary report, saying "[a] more extensive study of the asbestos industry is now under way." To the best of my knowledge, the more extensive study, if it was completed, was never published. Given the absence of regulations and workers' compensation, the authors' recommendation "that the industry seriously face the problem of dust control in asbestos plants" seems too mild. I think the authors were well aware of the seriousness of the hazards in the industry; moreover, civil suits and compensation cases were already beginning to bubble forth.

In 1934 the Department of Labor and Industry of Pennsylvania began a study of the health of workers in four asbestos fabricating plants of that state.[37] Airborne dust concentrations were sampled for the various industrial processes. Of 57 asbestos workers examined, 14 (25%) had asbestosis, and 3 more had "doubtful" asbestosis. The proportion of asbestosis cases was highest among the workers in the dustiest jobs. The degree of asbestosis likewise tended to be more extensive among the workers in the dustiest jobs. The asbestosis cases were classed as "slight" and "moderate," no "advanced" cases were found. The report, published September 20, 1935, contained a bibliography with 125 entries, almost all of which dealt specifically with asbestosis.

On May 23, 1934, the Supreme Court of North Carolina (McNeely v. Carolina Asbestos Co.) decided that asbestosis

> "contracted unexpectedly and gradually during many months of exposure, if due to the employer's failure to provide means of prevention, is an injury 'by accident' and, as such, was compensable under the Workmen's Compensation Act as it then read--which decision injected elements of uncertainty into the law that rendered the hazards of occupational diseases, particularly of silicosis and asbestosis, often uninsurable at practicable rates." [38]

The McNeely case led to the addition of provisions to the North Carolina Workmen's Compensation Act, effective March 26, 1935, which "specially regulate the liability to compensate for silicosis or asbestosis." The McNeely decision rendered "the rates then in effect grossly inadequate for all classifications with asbestosis, silicosis, and other serious occupational disease hazards," according to F. R. Jones,

General Manager for the Association of Casualty and Surety Executives in New York.[38] "Manual rates" for workmen's compensation nearly doubled, going from $2.86 to $5.48 per $100 payroll for asbestos goods manufacturing.

Jones' April, 1936, article also discussed workmen's compensation rates in Massachusetts, where the first U.S. disability claim for workmen's compensation was filed in 1927:[35]

> "The stock company rates for [the asbestos industry] vary from 7 1/2% to 10%, where strict precautions are taken, with a special rate of 12.5% for the making or repairing of asbestos mattresses."

In New York, two categories of asbestos products manufacturing were established, with separate rates for each, effective September 1, 1935. The "Dust Occupational Disease Coverage" for the New York rating plan was more expensive than the corresponding plan for North Carolina. For non-moulded asbestos products, asbestosis insurance cost $6.95 per $100 payroll, compared to $3.47 for moulded asbestos goods production in New York and $2.98 for the asbestosis element of workmen's compensation insurance in North Carolina asbestos goods manufacturing. But even these high rates were recognized as insufficient by the insurance companies:

> "High though these rates may seem, their adequacy under the present New York law, is generally doubted, with the consequence that the private insurance carriers are rejecting many risks, whereas the State Fund (which is not bound by manual rates) is exacting yet higher charges, especially for risks rejected by the companies."

The asbestos companies were clearly aware of the threat of occupational disease insurance to their profitability.

> "As a consequence of these very high rates, and the inability of some industries to get any kind of insurance at a price that permits continuance of operations, many establishments

> are laying off workmen and either closing down or sending their hazardous work out of the state and an insistent demand has arisen for drastic amendment of the law in so far as it relates to silicosis and other dust diseases."

In 1935, D. S. Egbert of the Yale University Medical School reported a fatal case of asbestosis in a 30-year-old man.[39] The author noted,

> "That the asbestos industry is growing rapidly and that, as a result, pulmonary asbestosis will continue to become more important is seen from the fact that total world production of asbestos has increased from 96,490 tons from 1910 to 434,938 tons in 1929."

He included a table, "Summary of reported fatal cases of pulmonary asbestosis," in which the reference, age of the victim, time exposed before onset of symptoms, total time exposed, and principal findings were recorded. Total time of exposure was only 9, 12, 15, 24, and 28 months for some of the cases which Dr. Egbert listed.

In 1935, Donnelly of the Mecklenburg Sanatorium in Huntersville, North Carolina, presented another paper at an annual meeting of the Industrial Hygiene Section of the American Public Health Association in Milwaukee. It was published the following year.[40] Donnelly's opening remarks called attention to the public health implications of the rapidly rising demand for asbestos products:

> "That the serious industrial hazard of this type of work has not previously received sufficient attention is becoming more apparent. The manufacture of asbestos products has increased more than four-fold in the last 20 years, and hence, because of the greater number of workers exposed, and the more frequent recognition as a pathological entity of the resulting pulmonary condition, the subject of asbestosis has rapidly assumed greater importance."

He doubted that the controls then in use would completely protect against asbestosis, noting that he was still finding fatal cases of asbestosis from one plant in spite of the installation of protective devices in 1916 in that plant.

> "It is indicated that the owners of asbestos plants, and the workers themselves, are beginning to realize that exposure to asbestos dust is a serious occupational hazard, and it also is apparent that these workers must be protected against the hazard as effectively as is possible. That this protection can be made complete seems very doubtful. It seems to be the opinion of engineers that the most effective types of suction apparatus now in use will remove a maximum of not over 90 per cent of the dust, but the remaining 10 per cent may be definitely injurious. In 1916 protective devices of the suction type were placed in a certain asbestos mill, and yet 15 years later cases of asbestosis with fatal termination were reported among workers from this plant. Since the most efficient type of protective machinery known apparently does not completely protect, although the number of workers affected is probably somewhat reduced, and the time required for the production of an advanced type of disease is probably lengthened, the problem becomes a matter for serious consideration."

Introducing his own data, he said,

> "I have seen no statistics in the literature in regard to the incidence of asbestosis among the workers in the industry. It would seem to be a matter of some importance to determine the average number of employees who may be now, or who are likely to become, disabled by the condition."

Donnelly examined the X-ray films of 151 workers and found definite evidence of asbestosis in 52 (34.4%). Of those with asbestosis, 48 had worked in the asbestos industry for five years or more, 3 had worked for four to five years, and 1 had worked for less than four years in the industry. Donnelly reported several cases in which the asbestosis had progressed after the

cessation of occupational exposure to asbestos, confirming this important fact which was first noted by Sparks:

> "One of the most important problems in the consideration of pulmonary asbestosis is whether or not the disease is progressive, even after an individual handicapped by it ceases to work in the industry. Information on this question is of considerable moment in rating the eventual degree of disability of a worker, either in the assessment of damages in civil suits or in claims under State Compensation Laws."

He noted that shortness of breath, in the more advanced cases, precludes any form of muscular exertion:

> "[T]he victim of asbestosis, as a rule eventually becomes totally disabled from engaging in any form of labor."

His closing paragraph was a plea that the best available dust controls be installed by the owners of the asbestos textile mills, and he warned that "in the final check up" the installation of such controls would be cheaper than the numerous damage suits and claims for compensation:

> "An industrial worker is entitled to every protection that may safeguard his health, so that he may earn a livelihood for himself and family for at least a reasonable period of years in the work in which he is most skilled. If he is prevented from continuing in such work because of impairment of health through no fault of his own, he is entitled to some remuneration for his loss of earning power. That protection of asbestos workers has been woefully lacking in the past has been definitely shown. It is imperative that such protection, as nearly complete as possible, be provided by mill owners. Efficient protective devices will be far less expensive in the final check up than the aggregate of numerous claims for compensation and frequent damage suits. That complete protection can be afforded by the devices in use at the present time seems to be somewhat doubtful, but workers are entitled to the highest type of protection which the engineers familiar with the hazard can provide."

In the same issue of the Journal of Industrial Hygiene and Toxicology, S.B. McPheeters of Charlotte, North Carolina, reported on the clinical examination of 210 asbestos textile workers seen at work.[41] None of the workers had been exposed for more than 20 years.

> "[O]f 9 persons exposed for more than 15 years to dust concentrations effective in producing disease, four, 44 percent, escaped asbestosis; of 32 persons exposed for more than 10 years to similar concentrations, fourteen, 43 percent, escaped; of 70 persons so exposed for more than 5 years forty-five, 64 percent, escaped, and of 28 persons exposed for more than 2 years and less than 5, twenty four, 85 percent, escaped."

McPheeters noted that individual differences in susceptibility to asbestosis appeared considerable, judging from the different responses to exposure to various amounts of dust seen in this work force. He recognized the limitations of his study, just as Merewether had:

> "To make a scientific appraisal of the effect on the employees in this plant of exposure to asbestos dust, it is necessary to know how many employees during the 15 to 20 years covered in the study have died or quit and how many of these had asbestosis. This knowledge is not available. Certainly it might add greatly to the gravity of the picture. The most that could be done was to note the effect on those now present in the plant, after varying periods of exposure."

In his opening paragraph, the author said:

> "While it employs a relatively small number of persons, the asbestos industry has had a remarkably rapid expansion and by reason of the widespread and varied uses of its products which include 'matches, filter pads, paints, roofing, high-pressure jointing, electrodes, brake linings, clutch rings and insulating material in a great variety,' the industry occupies an important, increasing, and permanent place in our economic establishment."

Soon after McPheeters' paper was released, the radiologist J. R. Shull published another report from the same city:[42]

> "During the latter part of 1934 it was my privilege to examine the chests of 71 of 100 workers who had been dismissed from local asbestos plants. All had undergone physical examination before they were referred for roentgenologic study and were found to be physically disabled . . .
>
> "The average age was 34.4 years and the time of exposure varied from 16 months to 21 years."

These 71 cases of asbestosis were classed as slightly advanced (16), moderately advanced (35), and markedly advanced (20).

Shull re-examined 21 of his patients a little more than a year after the first examination, by which time one had died of asbestosis. Of 5 markedly advanced cases, 3 showed increased fibrosis on X-ray, 1 showed no change, and 1 had improved. Of 7 moderately advanced cases, 2 showed spread of the disease and 3 showed improvement. Of 9 slightly advanced cases, 1 showed increased fibrosis and 5 had improved. His results showed that over a short post-exposure period of observation, progression of the disease occurred in all three groups, more commonly in the more severe cases; and conversely, the condition remained unchanged or improved more frequently in the milder cases, once these people were removed from asbestos exposure. Longer term follow-up would have been necessary to verify any lasting improvement among those whose conditions were seen to have improved during the year between examinations, in light of the previous literature. Shull's conclusion that "asbestosis is not primarily a progressive disease" emphasized the least worrisome of his findings.

Shull mentioned the McNeely case and the fact that the North Carolina legislature had recognized asbestosis as a compensable

The U.S. Public Health Service was requested by the health and compensation authorities in North Carolina to make an engineering and medical study of the conditions in the state's asbestos textile industry. The results were published in detail as a Public Health Bulletin in August, 1938, and in more concise form in the American Journal of Public Health in 1939. 43,4

In all, 242 dust counts were made to measure workers' exposure. The counts employed standard methods for that time, and all particles in the air were counted, not just asbestos fibers. The unit of measurement of airborne dust concentration was 1 million particles per cubic foot (mppcf), and the maximum value observed was 74.3 mppcf.

> "Medical examinations were made of 541 men and women representing practically all of the employees at the time of the study... The three factories studied had been in operation from 6 to 16 years. About 15 months before the study, approximately 150 workers were replaced by new ones with little or no asbestos experience. As a consequence, there was an abnormally large percentage of workers with less than 5 years' employment in the asbestos textile industry and an abnormally small percentage who had worked 10 years or more in the industry."

The Public Health Service reported,

> "The percentage of persons with asbestosis increases greatly with increasing length of employment . . . [A]lmost three fourths of the workers employed for more than 15 years have the early stages, at least, of a disabling disease, asbestosis. Since there is no satisfactory way of locating and examining persons who drop out of an industry because of disability incurred therein, the percentage values of the foregoing tables are necessarily minimal evidence of the prevalence of asbestosis."

> " . . . 16.3 percent of the 69 former asbestos workers [who had been traced] and 378 men and women employed at the time of the study in three asbestos textile factories had asbestosis. (Persons who were not exposed to appreciable quantities of asbestos dust have not been included.)"

(The non-exposed persons may have been office workers and other non-production personnel.)

> "Eight percent of the 378 workers employed at the time of the study had asbestosis. This does not completely represent the incidence of asbestosis because about 150 employees were discharged from these factories 15 months before the present study was made. Many of the discharged workers had asbestosis, as the reports of Shull, Donnelly, and McPheeters show. Although an attempt was made to examine as many as possible of these discharged cases, only 69 could be examined, 43 persons of whom had asbestosis according to the diagnostic standards employed in this study."

The investigators went on to estimate that another 50 cases of asbestosis among the recently discharged workers had "escaped observation." In other words, there were 30 cases of asbestosis in the factories by the time they were inspected by the Public Health Service, and an estimated 93 more asbestotics had been fired by the mill owners shortly before the Public Health Service conducted its survey.

By firing employees with long-term exposure, especially in the least dusty processes, the companies wiped out most of the data base the Public Health Service needed in order to develop any good dose-response data on asbestosis. Only 5 workers in the study who were considered at risk of disease had been exposed to levels under 5 mppcf for over 10 years.

> "None of the 39 persons exposed to dust concentrations below 2.5 million particles per cubic foot had a case of asbestosis, although, as a matter of fact, only 6 persons had been employed more than 5 years."

There were 3 "doubtful" or borderline cases of asbestosis found among the workers exposed to 2.5 to 4.9 mppcf. Despite the paucity of data on the risk among workers exposed to less than 5 mppcf, a tentative threshold value for asbestos dust exposure of 5 mppcf was designated. This value stood as the recognized standard in the U.S. for the next 30 years.

The Public Health Service investigators were assigned the difficult task of prescribing a limit that would be safe and demonstrably attainable with available process controls. They cited the 1935 Pennsylvania study that showed that incidence of asbestosis increases with the concentration of airborne dust and that found no safe threshold:

> "In their report of a similar study carried on in Pennsylvania asbestos textile factories, Fulton, Dooley, Matthews, and Houtz found that 8 percent of the workers exposed to an average dust concentration of 5 million particles per cubic foot had asbestosis; 22 percent of the 17-million particle group; and 57 percent of the 44 million particle group had asbestosis."

The conflict between the Pennsylvania findings and the 5 mppcf threshold set by the Public Health Service study was not resolved in the Service's report.

One attractive feature of the 5 mppcf limit was that it could be achieved, and in fact was being achieved in every process except "picking" at one of the North Carolina asbestos textile mills in the study. (The use of pitchforks to feed the picker machine was also described.) However, the exhaust equipment used in that plant could not get dust concentrations below 2 mppcf for most of the processes in the plant. The exhaust fan flow rates were by no means large, handling air flows of only 625-2570 cubic

feet per minute (CFM). A 1948 manufacturer's table on design of local exhaust system fans starts at 2520 CFM and goes up to 15,120 CFM.[45] A 2520 CFM fan would have a power rating of about 2 horsepower. The use of more high-powered fans and better process enclosures than had been voluntarily applied by this textile mill were clearly technologically feasible, but the Public Health Service was unwilling to draw conclusions about the need for dust controls it did not justify in its own study.

The British and American literature on the progressive nature of asbestosis was disregarded, though both Merewether and Donnelly were referenced. The Public Health Service investigators did cite the British statistics showing that asbestotics generally died nearly 15 years earlier than silicotics, after an average duration of employment only about one third as long as that of silicotics. They remarked that:

> "more than 90 percent of the raw material used in the manufacture of asbestos textiles in this country and Great Britain is Canadian chrysotile, indicating that very little difference is to be expected from the standpoint of biological reaction in the two countries. As near as dust concentrations in the various operations of asbestos manufacture of British practice can be compared with results obtained in this country, no greater differences are found than exist in the inherent limitations of the dust-sampling devices. It would seem, therefore, that quite negligible differences exist in the manifestations of the disease here and abroad with the possible exception of associated or complicating tuberculosis."

Nonetheless, the Public Health Service proposed a standard based solely on its own health and engineering study:

> "Because of the importance of the problem, it seems to be desirable to use such data as are at hand to define tentative safe working conditions that may serve as standards for the guidance of factory managers and engineers until more complete data are

> "Because clean-cut cases of asbestosis were found only in dust concentrations exceeding 5 million particles per cubic foot, and because they were not found at lower dust concentrations, 5 million particles per cubic foot may be regarded tentatively as the threshold value for asbestos dust exposure until better data are available.
>
> In order to find out the levels to which asbestos dust concentrations have been reduced in practice, an engineering study was made in an asbestos textile factory in which dust control equipment has been recently installed. This shows that means are already available for reducing the dust exposure of a majority of asbestos textile workers to less than 5 million particles per cubic foot."

The investigators were optimistic that no new cases of asbestosis would appear if asbestos dust concentrations in workplace air were kept below 5 mppcf--and followed by describing 5 mppcf as a "tentative threshold" that could be achieved with available methods of dust control.

The Public Health Service study had failed to develop the technical basis for establishing a threshold exposure level to protect workers from asbestosis:

> "Ideally, a threshold concentration of dust should be the highest dust concentration that would not produce pneumoconiosis in originally healthy workmen during their entire working life."

Unfortunately, nothing was published after 1938 that led to the determination of a threshold for worker exposure that would protect against asbestosis--for the next 30 years. Worldwide sales of asbestos fiber from 1938-1968 rose from 492,000 tons per year to 3,315,301 tons per year, with the U.S. in 1968 consuming about 25 percent of world output.[46,47]

In 1949, Wyers, medical director of the Cape Asbestos Company, reported on 115 fatal cases of asbestosis.[48] The average age at death

was 40.8 years, and the average duration of exposure was 10.4 years. The author said that a few of these persons had begun work with asbestos after the implementation of dust controls in factories imposed by the Asbestos Industry Regulations took effect in 1931.

> "The reduction in the intensity of the dust cloud seems to be producing a more chronic type of disease. Merewether's investigation of 1929 indicated a period of seven years' exposure for the disease to develop, whereas this series, which commenced in 1931, indicates that an exposure of ten years is necessary."

The progressive development of asbestosis is evident from the long post-employment survival of the women in this series, 9.8 years on the average:

> "No doubt many women ceased work to marry and symptoms of asbestosis did not appear until some time later."

The author pointed out that asbestosis was crucial in causing women's deaths in childbirth:

> "Pregnancy was associated with the deaths of two women suffering from asbestosis. The disease does not seem to be adversely affected by pregnancy, but the strain of labour may tip the balance in favor of right heart failure."

In 1968, the British Occupational Hygiene Society evaluated data on 290 men at work in an asbestos factory, data provided by the factory owners. As a result, a new standard was set, based (as it had been in the U.S.) on a study with a "note-

worthy . . . preponderance of individuals employed less than 20 years." The standard required no application of controls for exposures below 2 fibers per cubic centimeter (cc) of air.[49,50] For asbestos textile plants, this reduction (from 5 mppcf) was about 15-fold.[51] The British Occupational Hygiene Society predicted that the risk of having earliest signs of asbestosis would be less than 1% for a cumulative lifetime exposure of 100 fiber-years/cc (2 fibers per cc for 50 years of work, 4 f/cc for 25 years, etc). However, the workers in the same factory where the Society did their research on which the new standard was based showed a greater prevalence of X-ray abnormalities four years later.[50]

The debate over the "safe threshold" of airborne asbestos dust concentration that will protect a worker from asbestosis is still going on. In December, 1976, the Public Health Service (National Institute for Occupational Safety and Health) recommended a standard of 0.1 f/cc which it said would "protect against the non-carcinogenic effects of asbestos" and "materially reduce" the risk of asbestos-induced cancer.[50] For asbestos textile plants, the reduction from 5 mppcf to 0.1 f/cc is about 300-fold. Whether workers can tolerate even 0.1 f/cc of asbestos and not develop asbestosis is not yet known. However, the evidence is continuing to unfold:

> "In a study of 232 former insulation plant employees, Selikoff (1976) reported positive X-ray findings among individuals having exposures to asbestos known to be as short as one day." 50

There were a few papers of interest published after World War II about the asbestosis risk of insulation work. These studies showed that 1) insulation workers are still dying from asbestosis, although death rarely occurs within 20 years of the onset of the workers' exposure; 2) exposure of insulation workers, whose work has not changed very much for decades, appears to be somewhat less than 5 mppcf of dust particles in their workplaces on a daily average basis; and 3) the major cause of death resulting from insulators' occupational hazards is cancer. (Indeed, this latter fact is true for all asbestos workers today.)

In 1945 U.S. Navy doctors took X-rays of 1,074 men who were working as pipe coverers in Navy yards.[52] They also found that exposure to asbestos dust was below 5 mppcf in most of these workers' tasks, but that level was exceeded in some measurements. This study had several deficiencies in its methods. First, they did not monitor the dustiest job of all in ship insulation work--the removal of old insulation. Second, only 51 of the men had been doing insulation work for more than 10 years, and only 3 cases of asbestosis were identified. Of these three, two had worked for 22 and 30 years at pipe covering and the other had worked in an asbestos factory before coming to work in the Navy yard. Third, the authors ignored the experience of many previous investigators that a complete clinical examination is necessary to diagnose asbestosis accurately. Symptoms of cough and shortness of breath may be pronounced in cases where the X-ray shows little sign of pneumoconiosis.

The main shortcoming of the study was that very few workers had been exposed long enough for cases of asbestosis to "mature." Only 125 workers had worked for 5 to 10 years in the pipe covering industry; only 176 had been exposed for 5 or more years. Yet, in spite of these limitations, the authors cited a ridiculous statistic and concluded that the trade was safe:

> "The incidence of asbestosis among pipe coverers in the shipyards studied was low, 0.29 percent or 3 cases out of 1074 . . .
>
> "Since each of the 3 cases of asbestosis had worked at asbestos pipe covering in shipyards for more than 20 years, it may be concluded that such pipe covering is not a dangerous occupation."

These authors' ignorance of the literature on asbestosis is suggested by their lack of discussion of asbestosis per se and by their brief bibliography, which contained only three references--a book by Lanza, the Public Health Service study, and a Navy manual on ships and planes.

In Denmark, the insulation workers' union of Copenhagen approached a university clinic for occupational diseases for a clinical study of the health of union members.[53] Workers with less than 20 years experience were not even included for study:

> "After consultation with the union officials, we began examinations in 1953 by selecting workers who had been in the trade for twenty years or more ...
>
> The average age of the subjects was 52 years, ranging from 41 to 70; the period of exposure

> averaged 27 years, ranging from 20 to 45 years. Six of them complained of shortness of breath, which they considered abnormal for their age. All of them were working full time."

The researchers, who were radiologists, found "definite signs of pulmonary asbestosis" in 9 of the 31 workers, usually associated with pleural abnormalities.

> "In no less than 19 cases abnormalities of the pleura were found. These changes ranged from obliterated sinuses and pleural adhesions to extensive thickening of the pleura with calcification ...

Some workers' chest x-rays were so whited-out by pleural calcification that there was no way to get a look at the condition of the men's lungs

> In a few cases the abnormalities of the pleura were so widespread that no definite diagnosis could be made regarding the conditions of the lungs."

The authors considered the pleural damage to be a reliable sign for judging asbestosis among these insulators.

In their introduction, they referred to a 1950 Danish case report of asbestosis in an insulation worker.

In 1964, Marr reported a fatal case of asbestosis in an insulator at Long Beach Naval Shipyard who had had 10 years' employment as a pipecoverer and insulator.[54] Marr noted that ship insulation material and work methods had "remained essentially the same" since 1945.

Selikoff and co-workers reported in 1964 a follow-up study of the cause-specific mortality of asbestos insulation workers.[55] The work force studied was the membership of New York and New

Jersey insulation workers' unions, among the oldest such unions in the United States. The investigators traced a group of insulators employed as of December 31, 1942, and others who joined the union between 1942 and the end of 1962. They observed that among 392 workers with more than 20 years' exposure, "the very large majority had x-ray evidence of pulmonary asbestosis."[3] In general, severe clinical symptoms of asbestosis did not appear until after 20 years or more after the start of their asbestos work. By 1962, 255 workers in the group who had had 20 or more years of insulation work had died. Of these, 12 died of asbestosis. Another 95 died of cancer, and some of these would have later died of asbestosis had they not developed cancer.

McVittie reported that 41 percent of the cases of asbestosis recognized between 1955 and 1963 by the Pneumoconiosis Medical Panels in Great Britain were insulation workers.[13] "Two of the insulating workers who entered in 1956 had only four years exposure."

In 1968, Harries reported on new asbestosis cases certified by the Pneumoconiosis Panel from Devonport Dockyard.[56] These cases included men from the following occupations "who are NOT recognized asbestos workers":

Boilermaker	Plumber
Electrical Fitter	Rivetter
Engine Fitter	Shipwright
Engineer	Shotblaster
Iron Caulker	Stoker
Joiner	Welder

According to industry estimates, worldwide asbestos fiber output soared to 5,708,000 tons in 1976.[57] A scarcity of the "magic mineral" has been forecast by the year 2000 by the U.S. Bureau of Mines.[58]

Part 2. Cancer.

Two of the earliest reports of cancer with asbestosis were made by the British pathologist, Gloyne. He wrote at length of the damage to the pleura and lungs in asbestosis, and reported the first published case of pleural cancer with asbestosis in 1933.[26] In 1935 he reported 2 cases of lung cancer in female workers with asbestosis.[59] He considered the pleural cancer a complication unrelated to asbestosis. He suggested the possibility of industrial origin of the lung cancer, though he realized that there was insufficient evidence to "make out a case for an aetiological association of these two diseases."

One of Gloyne's cases was a 35-year-old woman who had worked as a spinner for 8 years and had not worked with asbestos for 9 years since, making 17 years from onset of exposure until death. The other woman died at 71, 15 years after periods of 6 and 13 months' exposure "in the mattress and opening departments of the factory." These lung cancers were not recognized during the lives of the patients, but were discovered at atopsy. For both cases,

> "The growth was in a portion of lung in which the asbestosis was fairly advanced. This was particularly clearly marked in Case 2."

Gloyne suspected that even small lung tumors in non-vital places could precipitate death in asbestosis:

> "At this point an interesting question arises. The asbestosis, though fairly advanced, was not sufficiently advanced to cause death. The neoplasms were small and had involved no vital parts. Indeed, compared with the squamous carcinoma of the lung in general, as it reaches the post mortem room, the growths were unusually small. A tentative suggestion may be made that in asbestosis a small tumour turns the scale, just as bronchitis with early brochopneumonia will do, probably as a result of toxaemia." *

Also in 1935, Lynch and Smith of South Carolina published the first case of asbestosis lung cancer in the American literature.[60] Lung cancer was an uncommon disease in 1935, and the occurrence of even one case of asbestosis with lung cancer was noteworthy:

> "As worthy of record in the interest of both disease, as well as their possible relationship, the following case of pulmonary asbestosis with associated carcinoma of the lung is reported."

The man had worked in an asbestos textile mill for 21 years, toward the end of which time he developed cancer.

The authors noted that the cancer had developed after there was fibrosis of the lung. They concluded,

> "A conception of (the tumor's) origin by reason of chronic bronchial irritation is compatible with the current view of the etiology of such tumors."

---

*toxemia - entry into, and persistence in, the bloodstream of bacterial toxins absorbed from a local lesion, from which these poisons are borne by the circulation to

The state of medical knowledge as of 1935 was recently described by Dr. Irving Selikoff, who has achieved prominence from his research on asbestos diseases.[61]

> "Thus, by 1935, the main directions of the problem were known. Chrysotile asbestos, virtually the only fiber then used, could produce wide-spread disease, this disease could be fatal and malignancy might be a result of exposure."

In 1936, Dr. Gloyne reported another case of asbestosis lung cancer.[62] His first two cases had had squamous cell tumors, this one had an oat cell tumor. The tumor was in the lower lobe of the lung. It was well known by 1936 that the more extensive fibrosis of the lungs in asbestosis cases is likewise found in the lower lobes.

The occupational history was presented:

> "[The patient] began work as a packer in an asbestos works in 1912 and eighteen months later was promoted to be a foreman in the stores department."

The man died 21 years after starting work in the asbestos plant. He was at work there when he developed chest pains, 6 months before he died.

Lubert and Geiger at the Yale University School of Medicine published another case report of asbestosis with lung cancer in July 1936.[63] The patient was a man who worked as a weaver in an asbestos factory for 18 years. He had to leave his job when he developed a disabling pain in his back, he died shortly afterward with lung cancer.

The authors concluded:

> "That the irritating effects of the inhaled asbestos particles may in this case have been a significant factor concerned in the development of the primary lung cancer seems sufficiently plausible to be worthy of consideration."

The authors also referred to Gloyne's 1933 report of asbestosis with pleural carcinoma; they had not seen the 1935 reports of lung cancer with asbestosis at the time their paper was submitted for publication.

In 1938, Nordmann in Germany published a paper entitled, "The Occupational Cancer of Asbestos Workers."[64] He reported 2 new cases of lung cancer with asbestosis, in a 35 year old woman and a 55 year old man. These two were asbestos textile workers. Nordmann reviewed the cumulative literature and was the first to propose that lung cancer be recognized as an occupational disease among asbestos workers.

He tabulated statistics from all of the earlier reports except Gloyne's 1936 case, which he apparently missed. For the six cases, he looked at a number of factors for signs that they were different from lung cancer then seen in the general population. He also looked for the specific signs of occupational cancer.

Nordmann noted that 3 of the 6 asbestosis lung cancer cases were young compared to the usual age of people dying with lung cancer (two were 35 and one was 41). In the

older cases, entry into the asbestos industry had occurred at an older age. A consistent finding for all cases was the time elapsed from the start of asbestos work until the time of death: no less than 15 years and no more than 21.

In 5 of the 6 cases, the tumors developed in the lower lobes of the lungs, whereas in the general population lung cancer was usually in the upper lobes.

The relatively frequent finding of multifocal cancers -- the development of more than one primary malignancy in the lungs of one person -- also suggested that occupational exposure to a carcinogen was the cause.

The microscopic appearance of the tumors in the group of asbestosis patients also was different from the pathology of lung cancer usually seen in the population. There were frequent findings of squamous cell carcinomas and adenocarcinomas with asbestosis, while the most frequent type seen in general was round cell carcinoma.

Finally, Nordmann assembled the scant autopsy data on asbestosis cases to reveal that an unusually high fraction of these cases had cancer. Referring to Wood and Gloyne's report of 100 cases of asbestosis, he noted that of 12 autopsied cases 2 (17%) had lung cancer. In Germany, he knew of 12 autopsied cases of asbestotics, and 2 of these also had lung cancer.

Though there were few reported cases in which an autopsy of an asbestosis patient had been done and determina-

tion of the presence or absence of cancer made, Nordmann still considered asbestos a proven carcinogen. He favored the view that fibrotic changes of asbestosis preceded development of cancer.

Hornig also wrote about one of the cases reported by Nordmann, the 35-year-old woman.[65] He considered the evidence sufficient to establish beyond doubt the causal connection between asbestosis and lung cancer. He urged the recognition of the secondary disease as an occupational disease.

E. W. Baader in Berlin wrote in March 1939 that the government regulations on compensation did not provide for the combination of asbestosis and lung cancer.[66] However, Baader said the (state) insurance carriers in Germany had readily recognized and compensated light asbestosis with fatal lung cancer as an occupational disease.

Thus, in 1939 the German government recognized that life could be cut short by the development of lung cancer in a worker whose asbestosis had not become severe. It is possible that such a worker would have later died of asbestosis, and that it had taken no less asbestos exposure to cause lung cancer than to produce an ultimately fatal asbestosis. But the more likely inference is that the German government recognized that workers who had not been exposed to enough asbestos to die from asbestosis had nonetheless been exposed to enough asbestos to develop lung cancer (and die from it).

It is also noteworthy that the existence of asbestosis diminishes the likelihood that lung cancer, once found, can be successfully operated upon. The asbestosis destroys the reserve breathing capacity that is necessary if one is to survive the removal of a cancerous lung. Thus, asbestos wrokers' life expectancy was known in 1939 to be lessened by two fatal "competing risks," the diseases asbestosis and lung cancer. Moreover, asbestotics in general had less chance of surviving lung cancer surgery than others in the general population who developed lung cancer.

In England, the Senior Medical Inspector of Factories reported that a total of 12 cases of cancer of the lung had occurred in 103 autopsied cases with asbestosis on record as of 1938 (11.6%).[67]

The same year, Lynch and Smith of South Carolina reported another fatal case of asbestosis with lung cancer in a 50-year-old man.[68] The man had begun work with asbestos 15 years before he died, and had worked with asbestos for 13 years. The malignancy of the lungs was so extensive that no definite focus of origin could be positively determined.

The authors reviewed other literature on lung cancer with asbestosis, noting that Nordmann considered lung cancer with asbestosis an occupational disease. The authors reported that 2 of the 7 cases of lung cancer they had seen at autopsy over a 12 year period were in people with asbestosis. Such people were a very small minority of the total number of autopsy cases:

> During the past twelve years in 2343 consecutive necropsies we have encountered 7 cases of primary lung carcinoma including . . . the two asbestosis cases. Among these necropsies are 35 showing some degree of asbestosis. In the majority this is a minor finding and not the cause of illness or death; in a few the condition is advanced.
>
> Including all necropsies and all lung cancer cases, the evidence of primary pulmonary carcinoma in our necropsy service over this period is 0.3 percent . . . .
>
> Among the asbestosis cases (35), the incidence of lung carcinoma (2 cases) is approximately 6 percent.

The authors did not conclude that the figures constituted significant evidence that the lung cancer rate was higher among asbestotics, due to the small number of cases involved. Their finding of an unusually high rate of lung cancer with asbestosis was strengthened by the fact that similar observations had been reported in Germany and England.

In 1940, Koelsch in Germany reported two cases of asbestosis with lung cancer, giving no details of occupational histories.[69] Two more were cases reported by Linzbach and Wedler in 1941, also in Germany.[70] One was in a man of age 61 exposed for at least 3 years to asbestos. No details of the other case were known. Also in 1941 two more cases of asbestosis with lung cancer were reported in Quebec, in men who were 50 and 57 and had each worked with asbestos for over 20 years.[71] In both cases from Canada, the authors described the

tumors as "epithelioma pleuro-pulminaire", though they were believed to have originated in the lungs, not the pleura. In both cases there was extensive malignancy of the pleura.

In 1942, the lengthy book Occupational Tumors and Allied Diseases was published in the United States by Dr. W. C. Hueper.[72] Hueper had begun his research on cancer in 1925 in Chicago. By 1942, he had established himself as one of the leading American authorities on experimental toxicology and carcinogenesis with dozens of studies and books on a wide range of substances used in industry. Hueper appraised the evidence on asbestosis and lung cancer thusly:

> "An evaluation of the evidence presented reveals certain features connected with these cases, which are suggestive of an occupational causation. There is an incidence of lung cancer in asbestosis of the lung which is definitely excessive. The relatively small number of necropsies on which this conclusion is based represents an urgent indication for more extensive post mortem examinations in cases of asbestosis; especially considering that pulmonary malignancies seen in previously reported cases were, in several instances, very small and were coincidental observations. A second point favoring an industrial origin is the young age of an appreciable portion of the cases thus far recorded, and the relationship existing between the time of exposure to asbestos dust and the manifestation of the neoplasms throughout the entire series. The third factor consists in the shift of the histological types observed ... and in the multicentric development of two (Nordmann; and Gloyne) and possibly even three (Lynch and Smith) of these pulmonary neoplasms.

> The relatively small number of asbestosis carcinomas of the lung reported does not disprove an occupational origin of this condition. Previous experience with other industrial neoplasms (lung cancer of the Joachimsthal miners; and lung cancer of the chromate workers) has shown that such isolated cases may represent the first warning of a subsequent appearance of large numbers of similar tumors of undeniable occupational derivation."

He went on to discuss medico-legal and social aspects and to urge wide ranging studies of the cancer risks of asbestos work:

> Asbestosis carcinoma of the lung is not included in any group of occupational tumors recognized by any country. The evidence presented is sufficiently serious, and the number of persons exposed to the suspected causative agent large enough to indicate a thorough and extensive clinical, statistical, and experimental investigation of the incidence and causative interrelation of asbestosis and pulmonary carcinoma. In view of the fact that several of the states with many large asbestos plants do not include asbestosis among compensatory occupational diseases, this study is even more urgent. Such a condition militates against an effective hygienic control of an important industrial hazard, and impedes the collection of pertinent and essential information in regard to the incidence, nature, and potentialities of an occupational disease growing steadily in general significance. Workers dying from asbestosis should be subjected to a postmortem examination including a histological study of the pulmonary lesions, as multiple neoplastic manifestations in asbestosis may be mistaken for tuberculosis (Lynch)."

Holleb and Angrist in New York reported two more

cases of asbestosis with lung cancer in 1942. The deceased

were men aged 52 and 58 and both had worked applying insulation to pipes for over 20 years. The older one experienced progressively worse disability beginning 15 years before his death, although for the last 10 years of his life he was employed as a salesman and was not exposed to asbestos dust.

> "(Dyspnea) progressed slowly but steadily so that during the last few years of life he was almost completely exhausted after climbing only one flight of stairs. He had frequent bouts of coughing which were especially severe in the morning but occurred throughout the day and night.
>
> The patient had been employed by a manufacturer of asbestos products since the age of 23 years until a few months before his death."

The authors wrote that in a city hospital over a 5-year period, they had seen 59 brochigenic carcinomas, comprising 2.4 percent of all routine autopsies. They had seen only two cases of pulmonary asbestosis, and in both there was lung cancer present as well.

This appears to be the earliest report of asbestosis and lung cancer among insulation workers.

On January 29, 1943, the German government declared "asbestosis in combination with lung cancer" a compensable occupational disease, acknowledging that lung cancer occurs in clinically slight cases of asbestosis as well as in well developed asbestosis. Thus, the citations of this health

hazard in the medical literature since 1938 by Nordmann, Hornig, Baader, Linzbach and Wedler -- resulted in the German government formally recognizing this occupational disease within a period of 5 years. Germany's tradition of industrial disease compensation stretches back to 1884 (German Industrial Insurance Act).[72] The U.S., with private insurers and 50 different state worker's compensation laws, has still not fully caught up with the standard of compensation that Baader described in practice in Nazi Germany in 1939. Hueper wrote in 1942 that 24 states in the U.S. still did not hold employers liable for diseases workers contracted as the result of conditions of their employment.[72]

In 1943, Hueper was no longer uncertain about the occupational origin of lung cancers with asbestosis. He had no way of knowing that the German government had formally recognized lung cancer with asbestosis as a compensible disease. This is from an article on occupational and environmental cancer in the Bulletin of the American Society for the Control of Cancer:[75]

> "Asbestosis cancer of the lung is the most recent newcomer among occupational cancers of this organ. First described in 1935, there are now 18 cases of this industrial cancer on record observed among asbestos workers in England, Germany and the United States. The latter contributed 5 cases. Inasmuch as the asbestos industry is most extensively developed in this country asbestosis cancer of the lung has for us a special hygienic and sociologic significance."

Hueper urged that industry take action to control occupational cancer saying among other things:

> "Industry should devote considerably more effort than heretofore in determining the cancerous or noncancerous nature of their numerous products which may be suspected on general grounds in cancerigenic respects ...
>
> Industry should make serious attempts to eliminate all potentially cancerigenic agents from further use by the development of suitable substitutes."

To the asbestos insulation manufacturers in particular and the asbestos industry in general he might as well have said, "Go into some other business for the sake of your workers and customers."

H. W. Wedler in Heidelberg filed a report in 1943 which mentioned pleural tumors with asbestosis in the same breath with lung tumors as an occupational disease.[76] He reported that in Germany, a total of 29 necropsies had been performed on workers with asbestosis, and there were 4 with lung cancers and 2 with pleural cancers. Wedler recalled that Gloyne had also reported finding a case of pleural cancer with asbestosis in 1933.

In a survey of the world literature, Wedler found a total of 92 reported necropsies of patients with asbestos disease, in which 14 malignant lung and pleural tumors were found (16%). This far exceeded the prevalence of lung cancer normally found in the general autopsy statistics (2-6%), according to Wedler. There were probably no figures available for

the incidence of primary pleural tumors at autopsy, since these were extremely rare. No pleural tumors would have been expected in a random series of 92 autopsies.

Wedler noted that the histology of lung cancers among asbestotics was different in general from the histology of lung tumors usually seen; the tumors appeared most often in the lower portions of the lungs, where asbestotic changes are also most pronounced; the period from onset of exposure to asbestos dust until evidence of cancer appeared was 12-42 years; the youthful ages of some of those afflicted was striking. And an unusually high proportion of females in the series also pointed toward an occupational disease, since women comprised a majority of the workforce in asbestos plants; ordinarily, lung cancer was 3 to 4 times as prevalent among men as among women. Wedler concluded that cancer of the lung in asbestosis was a legally justified disease for insurance claims.

It is significant that Wedler and Hueper submitted papers that were published at nearly the same time in Germany and the United States (August 6 and June, 1943, respectively). They were obviously not corresponding with each other during the Second World War. Yet, both authors surveyed the world literature and came to the same conclusion: lung cancer with asbestosis was an occupational disease.

A summary of Wedler's article by Gloyne was published the following year in the British journal, Bulletin of Hygiene.[77]

Homburger at Yale published a report of three more cases in the American Journal of Pathology in 1943.[78] The deceased were men aged 43 and 45, and a woman of age 49. One had been exposed to asbestos for 5 years, one for 20 years, and the last for an unknown period. Homburger's paper contained an excellent tabular presentation of the literature on asbestosis with lung cancer. Homburger reported that from 1918 to 1938 his laboratory had conducted thousands of autopsies and found asbestosis in 8: lung cancer was found in 4 of them, a "remarkably high ... association of the two conditions."

The Journal of the American Medical Association carried an editorial, "Environmental Cancer", on November 25, 1944.[77] The editorial warned that

> "The largest and most important group (of environmental cancers) is represented by occupational cancer elicited by exposure to chemical and physical agents in the course of regular occupations ... The agents known or suspected to cause occupational cancer are ... asbestos ..."

The article said that 5000-9000 cases of occupational cancer had been recorded, and that there was clearly under-reporting of cancers attributable to occupation. Organized investigation of occupational cancer was urged:

> "The benefits from such efforts will be not only the early detection and improved prognosis of cases of occupational cancer but also the prevention of industrial cancer by the elimination of exposures to cancerigenic agents."

In 1947, Kennaway and Kennaway published a report on lung cancer in Great Britain.[80] From a search of death certificates of lung cancer in males between 1921 and 1938, they found 8 with asbestosis listed as well. They were unable to obtain satisfactory figures on the number of persons occupationally exposed to asbestos but believed the number of lung cancers was probably greater than would be expected for the number of workers exposed. One of the cases they found was a "boiler and pipe asbestos coverer."

The following year the British pathologist Cureton reported a case of lung cancer with asbestosis in a woman who died at the age of 37.[81] This woman had worked for 7 years in "a factory making asbestos pipe covers," starting at the age of 15. That asbestos insulation manufacturing was hazardous was supported by the additional note that the patient's aunt had worked in the same factory and died at 40 of asbestosis. The author considered the woman to be unusually young compared to others that die of squamous cell carcinoma of the lung.

Merewether, Chief Inspector of Factories, produced cumulative statistics for autopsied cases of asbestosis in England from 1924 to 1946, in his annual report for the year 1947.[82] In a total of 235 cases, "cancer of the lungs or pleura" was present in 31, an incidence of 13.2%. This was contrasted with the incidence of such cancers in silicotics (1.32%) and the general

adult population (1.0%). In other words the expected number of lung cancers in a series of 235 would be 2 or 3 (0.01 x 235) and instead, 31 occurred. The chance that as many as 31 lung cancer cases in 235 would occur if their real likelihood were only the same as that of the general population (1%) is remote -- less than 1 in a million.

This Merewether report was often cited in later papers. The large number of asbestosis cases with lung cancer pointed unmistakably to an occupational origin of these cancers.

Lynch in South Carolina updated his figures on asbestosis in 1948 with a report on 40 autopsied cases.[83] In the 5 new asbestosis cases, one had lung cancer. The total incidence of lung cancer was thus 3 out of 40, or 7.5%. This was contrasted with the 1% general incidence of lung cancer seen in the authors' previous ten years' experience. The authors also reported that they had identified another case of lung cancer in a patient still living:

> "in a man who had been exposed to asbestos dust irregularly for twenty years ..."

All three deceased cases of asbestosis with lung cancer had moderate and advanced fibrosis of the lungs. Most of the 40 asbestosis cases had pleural fibrosis, for all grades of asbestosis.

A 1949 Editorial in the Journal of the American Medical Association ("Asbestosis and Cancer of the Lung") discussed Merewether's findings.[84] The Editorial cited epidemiological and experimental evidence suggesting that asbestos workers were at

increased risk of lung cancer. It characterized this risk as "probable," and stated that workers in asbestos-consuming industries should be considered at risk along with manufacturing plant workers:

> "Since some 20,000 workers are employed in the asbestos-producing industries of this country and Canada and many additional thousands in various asbestos-consuming industries, increased attention to this probable occupational hazard of cancer of the lung by the medical profession is desirable."

Wyer's report on 115 deaths from asbestosis in England also offered statistics on the incidence of accompanying lung and pleural cancer.[48] There were 17 such cancers in all, at least one of which was a pleural cancer. Lung and pleural cancer were referred to as one entity: "The proportion of pulmonary cancers in the series was therefore 14.8 percent." Seven others died with "cancer of other organs." No doubt about it, the author considered pleural cancer in the same class with lung cancer: an occupational disease. His discussion concerned how asbestos caused cancer, not whether it did.

It was striking that the pulmonary cancer cases had the longest exposure to asbestos and died the oldest of any subgroup in the series. As previously noted, the average age at death for the entire 115 cases was 40.8 years and the average duration of asbestos exposure was 10.4. For those with pulmonary cancer the average age at death was 52 years, and the average duration of exposure was 17.3 years. One possible explanation is that these individuals had the least dusty jobs, and that they therefore lived long enough to develop cancer because they were not killed off sooner by asbestosis. This thought might have occurred to a thoughtful student of the

Hueper, Chief of the Environmental Cancer Section of the National Cancer Institute, wrote in 1951 that most researchers had concluded that there was a causal relationship between asbestosis and lung cancer:[85]

> "Although Warren rather recently maintained that the connection between asbestosis and lung cancer is of coincidental nature, the actual existence of a causal relation appears to be very likely (Lecoeur; Koelsch; Saita; Hueper; Teleky; Editorial; Gross)."

That year, Tabershaw of New York published a list of occupational diseases covered by social insurance in West Germany.[86] One was "Asbestosis with diminished capacity to breathe and impaired circulation or associated with cancer of the lungs." Coverage was not limited to any group of asbestos-exposed workers.

Another fatal case of asbestosis with lung cancer was reported in New York in 1951 in a 40-year old-man.[87] He had worked as a pipe coverer starting in 1940.

> "For the next four and a half years he continued to work as a pipe coverer in a shipyard where he again used asbestos only. During this time he was told to wear a mask while working but neglected to do so and complained that the smell always made him sick."

Aside from illustrating the problems of implementing respiratory protection in the early 1940's, this story shows that at least in one shipyard in the United States at that time, the authorities knew that asbestos insulation work was hazardous.

The cause of death was lung cancer "with the possibility of multicentric origin", with metastases to the kidney, brain, and liver. The authors (one of whom had reported 2

earlier cases of these complications in insulators) introduced their report with a good review of earlier literature. They concluded that it was time to compensate asbestos workers for occupational cancers:

> "The importance of this association of carcinoma in cases of asbestosis is indicated from the review of the literature presented.
>
> This association emphasizes the hazards of industrial exposure, the compensability of the cancerous process as well as asbestosis and the need of careful preventive measures."

Owen in England reported another case of asbestosis with lung cancer in 1951.[88] The victim died at age 39, and his only exposure to asbestos had been a 12-month job in a London asbestos factory twenty years before.

A tabular presentation of the literature on cases of lung cancer with asbestosis was published in 1952 by Behrens in Germany.[89] He reviewed the cumulative world literature for the frequency of lung cancer with asbestosis and compared that with the reported rate of lung cancer with silicosis. For asbestosis, he found 509 autopsy case reports in which 44 (14.2%) had lung cancer. For silicosis, there were only 35 cases with lung cancer out of 2,475 autopsy reports (1.4%) known to the author.

That year, Cartier reported 2 cases of pleural mesothelioma among Canadian asbestos miners and mill workers.[90]

Hardy of the Massachusetts Institute of Technology referred to prior case reports of asbestosis with lung cancer in an insulation worker and in a man who "worked for seven years sorting and carding asbestos fibers intended for insula-

> "These cases have particular significance since there are nearly 10,000 workers exposed to varying amounts of asbestos in American industry, many of them in some operations not properly safeguarded against a potential carcinogen."

Hardy used the term "potential carcinogen" though she seemed convinced by Merewether's 1947 statistics. The use of such an expression does raise the point that, whether one was inclined to accept the carcinoginicity of asbestos as proven or await further data before drawing a conclusion, one had to already acknowledge the immediate and pressing need for providing warnings and improved safeguards to protect asbestos-exposed workers such as insulators.

Isselbacher and co-workers reported another case of asbestosis with lung cancer in a 41-year-old asbestos mill worker, in 1953.[92] The man was a "chain smoker". They extensively reviewed the literature. They pointed out that in Merewether's 1947 report of 31 cases, 9 (29%) were women. This was contrasted with general population rates of 4 to 10.3%.

> "The higher figure in asbestosis supports the theory that asbestos particles act as carcinogens."

A second case of lung cancer with asbestosis was noted as an addendum to the article.

> "The patient was a 46-year-old contractor's helper whose work since age 17 consisted of cutting and sawing asbestos board to insulate pipes, boilers, and refrigerators."

The man was a 1 pack/day cigarette smoker.

Weiss in Germany reported a case of pleural cancer with asbestosis in a man who had been an asbestos insulation worker.[93] The man's exposure spanned 22 years, the latter part of it in a supervisory role. Thirty-one years elapsed

The author referred to 2 earlier cases of pleural cancer with asbestosis seen by Wedler and noted that tumor tissue from his new case contained asbestos bodies. Weiss considered pleural cancer confirmed as an occupational cancer of asbestos workers.

In 1954, Leicher in Germany reported a case of peritoneal mesothelioma with asbestosis in an asbestos factory worker.[94] The man began asbestos work in 1919 and died 33 years later. Asbestos dust was positively identified in the tumor. Leicher considered the cancer to be of occupational origin, noting that such tumors were very rare in the general population.

Earlier reports had listed "metastatic tumors" in the pleura and peritoneum in cases of asbestosis with lung cancer. Reports of primary tumors of these sites suggested that some of these earlier cases may have been misdiagnosed primary tumors, not metastases.

Bonser and co-workers, reporting in 1955 on post-mortem examination of 72 factory workers with asbestosis in the U.S., found 2 pleural and 4 peritoneal cancers.[95] Lung cancer was also common:

> "Cancer arising primarily in the pleura, lungs, or bronchi was present in 26.1 percent of males and 8.7 percent of females. This significantly higher incidence in males may be accounted for by the fact that 7 women died before the cancer age ... It was noted that the degree of fibro-

> sis was rather less in the lungs with cancer than in those without."

Also in 1955, Doll in England reported that of 105 asbestos textile workers examined at necropsy from 1935-1953, 18 had lung cancer.[96] Follow-up study of 113 men with at least 20 years' exposure showed that 39 deaths had occurred, whereas only 15.4 would have been expected based on general mortality rates. The excess was entirely due to lung cancer (11 against 0.8 expected) and other respiratory and cardiovascular disease (22 against 7.6 expected). Doll noted that all of the workers with 20 years or more exposure had been first exposed before the implementation of the asbestos industry regulations. Though he had no data on the fate of workers with 20 years exposure under the improved conditions, Doll believed the cancer risk of these asbestos workers had become greatly reduced after 1933. As noted by Dressen and co-workers in 1938, the British asbestos textile mills used the same asbestos that was predominantly used in the American asbestos industry: the chrysotile asbestos from Canada.

Lynch and Pratt-Thomas in South Carolina reported details for 2 cases of asbestosis with lung cancer in 1955.[97] In a discussion of the paper, Pratt-Thomas was asked,

> "What is the present industrial compensation status of the coexistence of these two diseases?"

The reply was that the courts were beginning to recognize that lung cancer was an occupational hazard of workers who developed asbestosis:

> "[We] have seen cases in which at least partial compensation has been allowed. It was thought they might have not died quite as fast if they had not gotten carcinoma and that the evidence for asbestosis producing cancer, though not proven, was sufficinetly questionable so the cases were judged in favor of the plaintiff, or at least partially so."

Hueper authored a Public Health Service monograph [98] on environmental causes of lung cancer in 1955. By this time, he noted that there were 99 cases of asbestosis cancer of the lung on record, though there may have been some duplication of cases reported by the various authors. Hueper was also struck by the large proportion of women with asbestosis and lung cancer:

> "Equalization of carcinogenic exposure as represented by asbestosis, for the two sexes, thus resulted in a trend toward equalization of liability to lung cancer."

He went on to declare that many asbestotics had died before they had time to develop cancer, citing Merewether's 1947 figures:

> "It is of importance to note that the mean age of 128 noncomplicated cases of asbestosis was only 44.2 years (Merewether).* One may conclude from this observation that some of these individuals apparently died from asbestosis before their lung cancer had a chance to develop (Linzbach and Wedler).

Thus Hueper, who was quite familiar with the lung latency period before tumor development from occupational exposure to carcinogens, recognized the significance of the older average age at death of asbestotics with lung cancer compared with other asbestotics. It is noteworthy that Hueper credited Linzbach and Wedler with having made similar observation in their 1941

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* The average age of the 31 cases of asbestosis with lung cancer reported by Merewether was 52.1.

article. Bonser and co-workers had also alluded to one fourth of the women in their series dying "before the cancer age" of other causes.

One could infer from Hueper's statement that, as conditions in asbestos factories were improved so that workers would not die of asbestosis before they reached age 50, their lung cancer rate might go up instead of down. This questions could have been resolved by a mortality study of least-exposed asbestos workers. A relatively large group of such workers had long existed, and their exposure to asbestos had changed little since the turn of the century: asbestos insulation workers.

Two doctors in Pennsylvania reported that over 16 years they had seen 27 cases with asbestosis at autopsy or in surgery.[99] These appear to have all been asbestos textile workers, and 13 of them (about half) also had lung cancer. In all of these, the primary site of tumor growth was in the lower lobes of the lungs.

Another case of asbestosis and lung cancer was reported in an insulation worker in 1960, also in the U.S.[100] This man had been a heavy smoker, and the authors suggested that more attention be paid to smoking histories, and the possible role of smoking in the lung cancer mortality of asbestos workers.

In 1960, Wagner of South Africa reported 33 cases of pleural mesothelioma.[101] A history of occupational or environmental exposure to crocidolite asbestos in the North West Cape Province, where it was mined, was established for 32 of the 33 cases. Most of these people had not been occu-

pationally exposed to asbestos, but had lived near the mines and roads where asbestos was hauled and mine tailings were used in road construction.

Late that year, Keal reported on 23 women and 19 men treated as in-patients with asbestosis in one hospital in London.[102] Of the 15 men known dead, 10 had died with lung cancer and 1 was thought to have had pleural mesothelioma. The men with lung cancer included 1 "asbestos lagger" and 2 "boiler coverers." The 4 women in the series that developed lung cancer were all non-smokers. The most surprising finding was that 8 of the women had cancer of the peritoneum.

Keal noted that the lapsed period from onset of exposure was ~~longer~~ shorter on the average for the lung cancer cases (under 25 years) than the cases with peritoneal cancer (30 years). He also called attention to two cases in which cancer developed long after cessation of occupational exposure, and in which there were no symptoms or radiological changes to indicate asbestosis. Both had asbestos bodies in their sputum, but showed no other sign of asbestosis. Keal pointed out that these cases had definite medico-legal interest. "The asbestosis in these two cases was very slight yet these persons appeared to have died from occupational cancers. Keal also noted that 10 of the 42 cases in his series had first been exposed to asbestos after 1930, when asbestosis was recognized as an industrial disease and measures were taken to prevent it.

In 1962, the British began to develop a register of mesotheliomas of the pleura and peritoneum.[103] In calling for information, Smither and co-workers acknowledged that,

> "There appears to be no correlation between the severity of any pulmonary asbestosis and the occurance of these tumors. In a number of cases the exposure to asbestos dust appears to have been minimal, and the only histological evidence of asbestos exposure is the presence of a few asbestos bodies and fibres in the lung tissue. However, a detailed occupational history has, in nearly all cases, revealed some contact with asbestos fibre."

McCaughey in Belfast had first reported 15 cases of pleural mesothelioma in 1958, which he investigated further in 1962.[104] Lung sections revealed numerous asbestos bodies in only one of these cases. Detailed occupational histories were obtained for 9 of the 12 cases in which asbestos bodies were present in the lungs. In 4 of the 9 there was "a clear cut history of intermittent exposure to asbestos". In the other 5 there was no known asbestos occupation, but

> "all had jobs in the shipyard which inevitably brought them into intermittent contact with asbestos work -- for example, shipyard plumbing and furnace repairing."

The authors went on to describe a more recent case of mesothelioma in a 62-year-old man.

> "For all his working life he had been an engine room fitter and had worked for periods each year cleaning down and dismantling machinery which had been embedded in asbestos insulation."

The asbestos exposure in shipyards arose from the use of insulation, and this report suggested that not only insulation workers but workers in other trades were at risk of developing mesothelioma of the pleura.

In a study of workers employed in an Ohio asbestos plant as of 1938-1939 followed through 1960, Mancuso and Coulter found an excess of deaths from asbestosis, lung cancer, and cancer of the digestive organs and peritoneum.[105] There were 19 lung cancer deaths compared with 5.6 expected (based on general population mortality data). There were 18 deaths from cancers of the digestive organs and peritoneum, compared with 8.1 expected. There were 3 deaths certified as peritoneal cancer (0.08 expected) and two others were identified (by examination of available histological slides) out of a total of only 195 deaths in the work force by 1960. The authors considered their findings conservative estimates of excess risks, in part because the government data base on the workers did not distinguish production workers from office staff, and so the total plant population had to be taken as the exposed group. The government data base was also insufficient to characterize the workforce by duration of exposure. The authors noted that over 2/3 of the workers were aged 15-34 in 1938, and the War and related events "affected the pattern of continuity and duration of employment" of the workers.

By the end of 1963, the Ministry of Labour had knowledge of 584 persons in Great Britain whose death certifi-

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cates mentioned asbestosis. The proportion with lung cancer also listed had risen from 13.2% in Merewether's 1947 report to 25% in 1963.

In 1964 Selikoff and co-workers reported on the mortality of building trades insulation workers.[55] There were substantially more deaths observed in these workers than would be expected from general population mortality figures:

> Of 632 insulation workers who entered the trade before 1943 and were traced through 1962, forty-five died of cancer of the lung or pleura, whereas only 6.6 such deaths were expected. Three of the pleural tumors were mesotheliomas; there was also one peritoneal mesothelioma. Four mesotheliomas in a total of 255 deaths is an exceedingly high incidence for such a rare tumor. In addition, an unexpectedly large number of men died of cancer of the stomach, colon, or rectum (29 compared with 9.4 expected)."

The authors noted that smoking habits alone could not account for the high rate (17.6%) of lung and pleural cancer deaths in the workers.

Subsequent study by Selikoff and Hammond showed that asbestos workers who smoke cigarettes are about 8 times as likely to develop lung cancer as they would be if they did not work with asbestos.[107] It was calculated that asbestos workers who smoke cigarettes are 92 times as likely to get lung cancer as they would otherwise be. This makes lung cancer far more common among insulators who smoke than those who do not. The risk of mesothelioma and cancer of the lower gastrointestinal tract is high in asbestos workers, regardless of smoking habits.[61]

Newhouse and Thompson reviewed the records of London Hospital through 1964 on mesothelioma.[108] Eighty-three cases were confirmed by necropsy or biopsy, and 76 full residential and occupational histories were obtained by interviews with surviving relatives. Forty (52.6%) had a history of occupational or domestic (living in the house with an asbestos worker) exposure. In comparison, only 9 out of 76 (11.8%) "control" patients from the same hospital suffering from other diseases had such exposure. Among those with no evidence of occupational or domestic exposures, 11 of the mesothelioma cases had lived within half a mile of an asbestos factory, compared with 5 of the control series. The associations of occupational, domestic, and neighborhood exposures to asbestos and the fatal disease were all statistically significant. The mean interval between first asbestos exposure and death varied from 29.4 years among the factory workers to 48.6 years among the neighborhood cases. Out of the 31 patients with occupational exposure only 10 were in jobs covered by the Asbestos Industry Regulations of 1931. There were 8 mesothelioma cases classed as "laggers and insulators."

The findings of household and environmental cancer from asbestos illustrate the seriousness of the hazards of asbestos, making it all the more perplexing that the occupational hazards were not more carefully studied long ago. Environmental hazards, such as exist in modern buildings with sprayed-on asbestos ceilings, would never have come into e tence had the occupational asbestos problem been properly

studied in the 1930's, 1940's, or 1950's. Likewise, the families of asbestos workers would have been less exposed in the past had the hazards in the workplace been better recognized. Anderson and co-workers recently counted 37 reported cases of household contact mesothelioma in the literature, and added 4 more.[122]

The occupational hazards of handling asbestos are so great that even one month or less of work in an asbestos plant has been shown to cause cancer deaths among workers.[109] Seidman and co-workers have recently reported on the lung cancer mortality of workers briefly employed in a plant making asbestos insulation. The plant opened in New Jersey in 1941, and those employed between 1941-1945 were followed up through the end of 1974. The ratio of observed to expected deaths from lung cancer was 2.24 for workers with less than one month exposure, 3.47 for those with one month exposure, 6.15, 3.57 and 5.52 for those with 2, 3-5, and 6-11 months exposure, respectively, and reached 7.84 for workers with duration of employment of one year.

> "The excess of lung cancer deaths was first observed 10-14 years after onset of work, and increased significantly with longer observation."

Overall, there were 157 cancer deaths in this work force through 1974, compared to 50 expected cancer deaths.[61]

It is now becoming apparant that the proliferation of asbestos in society has resulted in widespread hazards to workers only incidentally exposed to asbestos. Menck and Henderson have recently identified excessive rates of lung cancer among clothing ironers, plasterers, dry wall workers, electricians, and plumbers; they suspect that asbestos[110]

of recognized "asbestos workers" alone is enormous. The present situation was assessed recently by Dr. Selikoff: [111]

> "Cancers now being seen are a legacy of past inadequacies. The USPHS has estimated that there are some 1,000,000 Americans who are either currently working with asbestos or previously regularly worked with the material ("asbestos workers"). (In addition many millions of other workers have or have had less intimate but still substantial exposure, as in the construction industry, shipyards, brake repair work, etc.) Unless we learn to intervene in what seems to be the inevitable development of asbestos cancers among those exposed in the past and assuming that asbestos cancer rates remain about the same, approximately 400,000 (40%) will die of cancer in the course of the next 45 years or so, including an estimated 200,000 of lung cancer, and perhaps 50,000 of pleural or peritoneal mesothelioma."

Development of Literature on Excess Cancer Risks - by Site of the Primary Cancer

As asbestosis caused the deaths of a decreasing fraction of asbestos workers at early ages (before age 50 or so), the "competing" cancer risks became increasingly evident. As noted, lung cancer was first reported in 1934 among asbestotics and it was recognized as an occupational disease of asbestos workers as early as 1938 in Germany and 1943 in the U.S. Pleural mesothelioma was first reported with asbestosis in 1933, and was recognized as an occupational disease by the early 1950's in Germany. Peritoneal mesothelioma was first reported with asbestosis in 1954. An excess of gastrointestinal cancer among a defined population of asbestos workers was reported in 1963, and was firmly established by

about 1973. In 1973, reports appeared that linked occupational exposure to asbestos with excessive incidence of cancer of the larynx.[112,113] There appear to be other sites where asbestos workers also have an excess risk of developing cancer, though the sites of major excess risk have by now been identified. It is remarkable that a group of workers could have so many statistically significant excess rates of so many fatal diseases -- competing risks, as the epidemiologists say.

It is obvious that a worker who is at risk of 6 fatal diseases recognized at different times in history should have been warned as soon as there was cause to suspect that he or she was at risk of the first fatal disease. Whether an asbestos insulation worker dies of asbestosis, mesothelioma, or cancer of the larynx should not alter the manufacturers' liability for the occupational disease.

What Could American Asbestos Companies Reasonably Be Expected to Have Known, and When?

Pliny's writings tell us that the Romans discovered the disease asbestosis in slaves who weaved asbestos. The managers of asbestos factories at the turn of this century should likewise have been able to see that their workers were becoming stricken with a progressive, fatal respiratory disease.

The literature shows that the American asbestos industry realized health problems existed in U.S. factories before a case of asbestosis was published in the American literature (1930). In the introduction to their paper published in 1935, Lanza and co-workers said that their investigation had been requested in 1929 by "officials representing the asbestos industry in the United States."

From Wood and Gloyne's 1934 description of asbestosis one can visualize the senior workers in the asbestos industry at that time:[28]

> "The distress occasioned by constant breathlessness is so great that the disease might be described as monosymptomatic. It is the patient's first and last complaint. In the earlier stages dyspnoea* is only apparent during exertion -- 'I found I could not hurry to work in the morning!' -- in the later stages the act of undressing or even the effort required for speech may cause the patient to pant for breath ... Malnutrition is very evident in the terminal months when the patient may pass into a condition of extreme cachexia.**"

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* "dyspnea (dyspnoea) -- difficult or labored breathing." The American College Dictionary, Random House, New York, 1956.

** "cachexia -- general ill health with emaciation, due to a chronic disease, as cancer." Ibid.

In 1973 U.S. government scientists reported a much increased death rate from both lung cancer and non-malignant respiratory disease among asbestos manufacturing workers.[114] The majority of these deaths occurred within 5 years of termination of employment in the asbestos industry. "Twenty-six out of 45 lung cancer deaths and 41 out of 52 pneumoconiosis deaths occurred within this five year interval.

The report, which was introduced into the Congressional Record by Senator Taft of Ohio, continued:

> "[A] breakdown of pneumoconiosis deaths which occurred within five years since termination of employment may be seen in Table 6. The majority of these respiratory deaths occurred within one year since termination of employment including 17 deaths which at an average age of 53.8 years occurred within six months of termination ...
>
> "Lung cancer and pneumoconiosis deaths which occurred within six months of termination of employment are evaluated further according to one-month intervals since termination of employment (Table 7). The majority of both lung cancer and pneumoconiosis deaths which occurred within six months took place within three months of termination. Note that five lung cancer deaths and eight pneumoconiosis deaths occurred within one month."

The workers in this study manufactured asbestos textile, friction, and packing products in Pennsylvania, during the period 1940-1962, and their mortality was "followed up" through the end of 1967. Of the 13 lung cancer and pneumoconiosis deaths that occurred in this work force in the 1940's, 9 occurred within six months of termination of employment.

In the days before workers' compensation, unemployment compensation, and welfare, and before formal recognition of the hazards of asbestos, there was great economic pressure for asbestos workers to stay at their jobs as long as it was physically possible for them to work. Taking the statistics for the 1940's from the above study as a guide, one can only conclude that many of the senior workers in the asbestos factories of the 1920's and the years of the Great Depression were severely afflicted with asbestosis, and some worked virtually until the day they died. Many of them died in their forties, many others in their thirties. It is hard to imagine that the condition of these people and their deaths went unnoticed by their employers, regardless of the extent of the medical literature in the early part of this century.

The world medical literature has long been accessible in this country, of course. Foreign journals were carried in the better medical libraries around the country. Though some of the better literature on asbestos cancer was German, that should have presented little obstacle to American scientists, who were encouraged in college to learn German so that they would be able to read German scientific papers and patents, etc.

From 1927 until the early 1950's, the index of the world literature was called "Quarterly Cumulative Index Medicus". This was published by the American Medical Association, in Chicago. Much of the material published in the medical journals worldwide, though not all of it, was listed by subject in the Index.

For example, Wedler's important paper on asbestosis with cancer of the lung and pleura in August, 1943 was listed in the Index about one year later. The Index also recorded that the paper had also been summarized in another journal. Thus at the height of World War II, an American reader could look up "asbestosis" in the Index, be referred to "pneumoconiosis", and find the reference for Wedler's paper of the year before in the German Medical Weekly.

Aside from the Index, the best sources of references to medical papers were other, more recent, publications. Many of the authors, even some presenting a single case report, would present a fairly good review of earlier work in the field.

Finally, the 1936 report by Jones shows that asbestos manufacturing firms were jumping state lines to avoid workman's compensations laws. Thus, in a number of states the cost of running an unsafe workplace was becoming a major operating expense for the asbestos industry.

The asbestos industry has not only failed to make reasonable efforts to discover and control the hazards of its products. It has forcefully opposed such efforts. North Carolina asbestos textile firms sabotaged the Public Health Service study in the late 1930's by firing 150 out of less than 600 workers, workers suspected of having asbestosis.[115]

The cozy relationships that the American asbestos industry has set up with universities and government officials were described by New Yorker writer Paul Brodeur as the "medical-industrial complex," in his book Expendable Americans.[116] More

than half of Rachel Scott's chapter, "Science: The Drab and Slut of Industrialism" was spent discussing the publication of "red herrings" in the scientific literature to "buy time" for the asbestos industry.[123] Two other scathing accounts on the asbestos industry in the U.S. and Canada have been published by the Health Policy Advisory Center of New York, in its Bulletin.[117,118]

> "The granddaddy of occupational health coverups, however, is the coverup by the asbestos industry of asbestos hazards ...
>
> A historical review of the research papers on the subject since 1900 reveals industry's strategy: Ignore the problem, then minimize or deny it and when all else fails try to shift the blame."

An example of an "industry study" is a paper by Enterline and Henderson at the University of Pittsburgh of retired asbestos factory workers, including those from a plant in Manville, New Jersey.[119] This paper is described in a new document on asbestos from the National Institute for Occupational Safety and Health (part of the U.S. Public Health Service):[50]

> "Of 802 deaths only one mesothelioma had been recorded in the several plants investigated. In contrast, a subsequent investigation by Borow et al (1973) found 70 cases of mesothelioma from only one of these plants. The discrepancy was due to methodological variations, for example Enterline and Henderson (1973) had limited their investigation to men of 65 or over, while many of the mesothelioma cases reported by Borow et al (1973) had died before that age."

In the medical literature, the asbestos industry has been accused of adversely influencing two separate attempts to investigate the health hazards in the mines and mills of

Canada.[1,45]

In my own experience of urging government agencies to provide increased public and worker protection from asbestos, the industry has bitterly opposed any regulation of its activities. A recent example is a petition to the Consumer Product Safety Commission which I helped draft for the Natural Resources Defense Council.[120] The petition asked the agency to ban the use of asbestos in widely sold dry-wall taping and spackling compounds. In spite of the fact that the use of such products entails long term indoor contamination and short term exposures to asbestos levels in excess of those now allowed in asbestos plants, and despite the fact that these products can be made without asbestos and the use of asbestos in these products constitutes only 1 percent of the sales of asbestos in the U.S. -- the asbestos industry trade association (Asbestos Information Association of North America) asked the government to deny the petition.[121] This is only one recent example of the asbestos industry's refusal to accept a decline in sales to protect the health of its customers.

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