Expert Report

of

Gerald Markowitz PhD and David Rosner PhD

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Introduction:

We have been asked to conduct a historical review of the relevant medical, scientific, industrial hygiene and industrial literature, as well as other relevant publically available information and provide our expert opinions, expressed to a reasonable degree of certainty, in answer to the following questions:

1. When was it known and therefore knowable that breathing dust containing asbestos could cause disease?
2. When was it known and therefore knowable that breathing dust containing asbestos could cause cancer?
3. Who was known to be at risk for contracting asbestos-caused diseases and when was that known?

Background, Qualifications and Experience:

David Rosner is Ronald H. Lauterstein Professor of Sociomedical Sciences History at Columbia University and Co-Director of the Center for the History of Public Health at Columbia’s Mailman School of Public Health. An elected member of the National Academy of Sciences’ Institute of Medicine, he received his BA from CCNY, his MPH from the University of Massachusetts and his PhD from Harvard in the History of Science. Until moving to Columbia in 1998, he was University Distinguished Professor of History at the City University of New York. In addition to numerous grants, he has been a Guggenheim Fellow, a recipient of a Robert Wood Johnson Investigator Award, a National Endowment for the Humanities Fellow and a Josiah Macy Fellow. He has been awarded the Distinguished Scholar’s Prize from the City University, the Viseltear Prize for Outstanding Work in the History of Public Health from the APHA and the Distinguished Alumnus Award from the University of Massachusetts. He has also been honored at the Awards Dinner of the New York Committee on Occupational Safety and Health and he and Gerald Markowitz have been awarded the Upton Sinclair Memorial Lectureship “For Outstanding Occupational Health, Safety, and Environmental Journalism by the American Industrial Hygiene Association.” He is author and editor of eleven books including A Once Charitable Enterprise (Cambridge University Press, 1982, 2004; Princeton University Press, 1987), “Hives of Sickness,” Epidemics and Public Health in New York City (Rutgers University Press, 1995), and Health Care in America: Essays in Social History (with Susan Reverby). In addition, he has co-authored and edited with Gerald Markowitz numerous books and articles, including Deadly Dust: Silicosis and the Politics of Occupational Disease in Twentieth Century America, (Princeton University Press, 1991;1994; University of Michigan, 2005), Children, Race, and Power: Kenneth and Mamie Clarks’ Northside Center, (University Press of Virginia, 1996; Routledge Press, 2001); Dying for Work, (Indiana University Press, 1987) and “Slaves of the Depression,” Workers’ Letters About Life on the Job, (Cornell University Press, 1987). He and Gerald

Gerald Markowitz, PhD is University Distinguished Professor of History at the John Jay College and the City University Graduate Center of the City University of New York. At CUNY he is also on the faculty of the PhD Program in Public Health. He is also Adjunct Professor of Sociomedical Sciences at Columbia University’s Mailman School of Public Health. He received his BA from Earlham College and his MA and PhD from the University of Wisconsin. He is the recipient of a Robert Wood Johnson Investigator Award, a National Endowment for the Humanities Research Grant two grants from the National Science Foundation and others from the Milbank Memorial Fund. He has been awarded the Viseltear Prize for Outstanding Work in the History of Public Health from the APHA. He has also been honored at the Awards Dinner of the New York Committee on Occupational Safety and Health and he and David Rosner have been awarded the Upton Sinclair Memorial Lectureship “For Outstanding Occupational Health, Safety, and Environmental Journalism by the American Industrial Hygiene Association. He is author and editor of eleven books including, with David Rosner, *Deadly Dust: Silicosis and the Politics of Occupational Disease in Twentieth Century America* (Princeton University Press, 1991; 1994; University of Michigan, 2005), *Children, Race, and Power: Kenneth and Mamie Clark’s Northside Center* (University Press of Virginia, 1996; Routledge Press, 2001); *Dying for Work*, (Indiana University Press, 1987) and “Slaves of the Depression,” *Workers’ Letters About Life on the Job*, (Cornell University Press, 1987), *Deceit and Denial: The Deadly Politics of Industrial Pollution* (University of California Press/Milbank, 2002) and *Are We Ready? Public Health Since 9/11* (University of California Press/Milbank, 2006). In 2008 he edited *The Contested Boundaries of Public Health*, (with James Colgrove and David Rosner) from Rutgers University Press in 2008. His newest book (with David Rosner), titled *Lead Wars*, (University of California Press, forthcoming) details the recent conflicts at Johns Hopkins over studies of children placed in homes with low level lead exposure.

Our qualifications are more fully laid out in Exhibits A and B which contain our CVs.
Opinions and Bases for Opinions

Our opinions to a reasonable degree of certainty are:

1. In the early 1900s it was known that breathing asbestos dust was hazardous and by 1930 it was well recognized.
2. Beginning in the 1930s there was a suspected link between breathing asbestos dust and cancer and by the mid-1940s it was acknowledged by many medical researchers. It was confirmed by the epidemiological studies of Sir Richard Doll and by publications in JAMA and elsewhere by 1955.
3. It was known in the early 1900s through the 1930s and 1940s that the people at risk were the people inhaling asbestos.

The bases for these opinions are expressed below. We have reviewed and relied upon the references cited throughout this report as well as those contained in Appendix C, the Annotated Bibliography.

We reserve the right to supplement this report upon review of additional information or if asked to answer other questions.

There is an enormous literature that documents the relationship between asbestos exposure and disease. While some have traced this knowledge back to the ancients, the modern literature can be safely said to date to the beginning of the last century. Factory reports in England from the first decade of the 20th century note the poor health of weavers and others in manufacturing establishments using asbestos fibers. By the teens insurance industry statisticians noted the high mortality of these workers and in the late 1920s and early 1930s the medical community became acutely aware of clinical reports and major studies of asbestos workers who suffered what became known as asbestosis. By this time it was known that workers other than those directly handling asbestos were at risk for asbestosis. Bystanders and others who breathed dust containing asbestos...
fibers also contracted the disease. Methods for reducing the risk of asbestos disease, including educating the workers and industrial engineering controls, were known. By the middle of the Depression exposure to asbestos was reported to be associated with lung cancer risks and by the mid- to- late 1940s this association was confirmed by many case studies that appeared in the medical literature and by reports in medical texts that made the link explicit. By the mid-1950s epidemiological studies confirmed this relationship.

While lung cancer was the first of the tumors associated with asbestos exposure other, rarer tumors in other parts of the body began to be identified in the 1950s and 1960s. By the mid 1960s mesotheliomas were identified and confirmed in many case reports and surveys of workers and by-standers. The range of diseases and cancers associated with asbestos exposure were widely acknowledged and documented by the 1960s.

Asbestos-related diseases in the early 1900s were rooted in work and factory production in the growing industrial economies of Britain, the United States and other industrial societies. In this new industrial environment where large portions of the workforce were now working indoors, in pits or underground, in jobs that entailed blasting, drilling, carding, weaving, cutting, and other dust-producing processes, medical researchers and clinicians, insurance actuaries and government officials observed that these dusts endangered the workforce. The variety of lung conditions produced by the “dusty trades” as they became known were called the “pneumoconioses,” i.e. dust-diseases of the lungs, and were all characterized by shortness of breath (dyspnea), long latency periods, irreversible symptoms and increasingly severe disability often resulting in death years or decades after exposure. Initially, the major focus was on silica dusts produced in the sandblasting, mining, granite cutting, foundry and construction trades. Silica dust produced an
unusual disease of the lungs, silicosis, which appeared years – even decades – after exposure and provided the medical and research community a new model of chronic lung disease.

In 1898 the Annual Report of the Chief Inspector of Factories and Workshops in Britain noted “the evil effects of asbestos dust” in her examination of the “dusty occupations.” It stood out “on account of their easily demonstrated danger to the health of the workers, and because of the ascertained cases of injury to bronchial tubes and lungs medically attributed to the employment of the sufferers.” The Lady Inspector singled out “asbestos sifting and carding, silk opening” and noted that there were few precautions taken “to subdue the dust [and] no sort of ventilation being applied.” The link between inadequate ventilation, unsafe working conditions and dust were henceforth identified as of particular concern. In this early report the dangers from asbestos dust were ascribed to “the sharp, glass-like jagged nature of the particles [which when they] remain suspended in the air of a room, in any quantity … have been found to be injurious, as might have been expected.”¹ The following year the Inspectors noted that the “jacketing for pipes, involving use of asbestos” was injurious to the workforce.² Also in the early century these factory inspectors suggested that the prevention of disease could be attained by improved ventilation and engineering controls.³ In Britain, the most distinguished medical personnel remarked that asbestos had been identified as dangerous by ancient cultures.⁴ The first clinical case of asbestos disease was documented in 1907 Dr. H. Montague Murray who reported on a 33

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In the second decade of the twentieth century the Labour Department in Canada investigated “the health of those employed in the asbestos industry.” They cited “One of the oldest medical practitioners in Thetford [who] expressed the view that the asbestos dust floating in the atmosphere of the cobbing room had a weakening effect on the lungs of those employed.” They suggested the use of engineering controls and respirators to keep the dust down.6

The British studies and reports on the dangers of asbestos dust were widely available in the United States when H.K. Pancoast, T.G. Miller and H.R.M. Landis published the first “Roentgenologic Study of the Effects of Dust Inhalation Upon the Lungs,” in the Transactions of the Association of American Physicians in 1917. In the discussion of their oral presentation at the meeting a Dr. Kober said, that “persons exposed to the habitual inhalation of dust, sooner or later would develop some chronic inflammation of the respiratory system.”7

The dangers from asbestos were well documented by Frederick Hoffman, the Vice President and actuary of the Prudential Life Insurance company in a major report produced in 1918 for the U.S. Bureau of Labor Statistics on “Mortality from Respiratory Disease in Dusty Trades (Inorganic Dusts).” This report detailed the variety of mineral dusts that potentially posed a threat to the workforce and gathered the existing statistics from the Prudential records of mortality. He remarked on the variety of occupational groups that were at risk of asbestos disease

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4 Oliver, Thomas, Dangerous Trades, New York: E.P. Dutton, 1902, p.25.
including workers who covered pipes, furnaces, plastered walls, used asbestos in roofing, insulated electrical equipment, and engaged in the construction of storage rooms of refrigerating plants. Asbestos workers were seen by the insurance industry as being at particular risk for early death and thus it was “the practice of American and Canadian life insurance companies [that] asbestos workers are generally declined on account of the assumed health-injurious conditions of the industry.”

Hoffman quoted from the 1910 Annual Report of Chief Inspector of Factories and Workshops, which included a report by Collis on the asbestos industry that stressed the need for dust protection for the workforce because of the deaths of asbestos workers. Hoffman also quoted Collis who noted that it was not only those who directly produced asbestos dust who were at risk of disease but also by-standers in the vicinity: “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.”

Hoffman concluded that there was an “urgent need for extensive investigation of health effects of asbestos manufacture.”

In the same year of Hoffman’s study academic medical researchers began to perform more sophisticated surveys of the workforce, using the new technology of the x-ray to identify pathological changes in the lungs of workers exposed to dusts, including asbestos. Pancoast and

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his colleagues published a survey of 137 workers including 15 asbestos workers. They described the three stages of pneumoconiosis and noted that the second stage “is characterized by a more or less uniformly arranged mottling throughout the lung structure due to depositions of dust in the lymph structures, cells, and fibrous tissue interspaces, with the addition of a certain amount of localized fibrosis....It occurs comparatively early in coal miners, cement workers and certain metal grinders, somewhat later in potters and asbestos workers....”

Animal experiments reported in the British Medical Journal, an esteemed journal that was available internationally, confirmed that asbestos dust caused fibrosis. More animal experiments would be performed in the coming years and decades.

Evidence accrued that documented the long latency of asbestos-related diseases. In 1925, Pancoast and his colleagues used the x-ray to show that of 17 asbestos workers, two “showed first stage changes and the other 15 definite second stage appearances.” The men who had worked longest at their jobs (17 and 14 years) “showed very definite diffuse, ‘soft’ spots throughout both lungs and another showed about the same appearance after fourteen years’ occupation. Very slight nodular shadows were found in one man after only two years’ occupation.” Cooke and his co-investigators used microscopy to look at the cells of workers in an asbestos factory where “a very

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considerable amount of dust is generated.” They concluded that asbestos dust produced a unique pathology in the lungs of these workers and that they had “never seen anything parallel to this in pneumoconiosis due to other dusts....”16 This British research was reported on by the most widely distributed medical journal in the United States, the Journal of the American Medical Association (JAMA), in an article titled “Pneumoconiosis Caused by Asbestos Dust.”17 S. Roodhouse Gloyne, in an article titled, “The Presence of the Asbestos Fibre in the Lesions of Asbestos Workers,” remarked that the term “asbestos bodies” should replace the then-current term, “curious bodies” since it was clear that asbestos was the cause of the pathological change.18 Thomas Oliver’s report on “Pulmonary Asbestosis in its Clinical Aspects,” was published in the American Journal of Industrial Hygiene in 1927 (as well as in the British Medical Journal) and JAMA also reported on it.19

The attention to asbestosis continued to accrue in the American medical literature in the late 1920s. An editorial in the widely distributed JAMA remarked on Cooke’s 1924 report, noting that “many other cases seem to have occurred” and that while they hoped that factory conditions were improving, “asbestosis, because it its dangers and its unique pathologic features, deserves

17 “Pneumoconiosis Caused by Asbestos Dust,” JAMA, 89 (July 23, 1927), 304.
19 Oliver, Thomas, “Pulmonary Asbestosis in its Clinical Aspects,” Journal of Industrial Hygiene, (November 1927), 483-485. (Also printed as “Clinical Aspects of Pulmonary Asbestosis,” in The British Medical Journal, 2, No. 3491, (December 3, 1927), 1026-27. Also reported in JAMA, 89 (1927), 2285.
more attention than it has had.”

Cooke himself, in an article in the prestigious British Medical Journal noted that “Pulmonary asbestosis has become a question of international interest and importance.”

In 1929, the British Medical Journal noted that asbestos had become such a significant problem in the UK that the government was considering making it a compensable disease. Authors suspected that a “wider recognition of this disease” would lead to more cases being identified and that “the mortality statistics [would therefore] probably be swelled.” The latency of this disease also made recognition difficult for it was usually first recognized more than five years following exposure. It was clear that asbestosis was a dread disease for which there was little treatment and no cure. As W. Burton Wood remarked in 1929, “when the diagnosis is established, the prognosis appears to be grave.”

By 1930 when the first clinical report of an asbestos victim was reported in the United States a huge literature had developed that documented the importance of asbestos as a cause of the pneumoconiosis, asbestosis. Asbestosis was documented by clinicians, by government inspectors and insurance officials, by researchers in university laboratories, by roentologists and pathologists, by editors of leading medical journals in both the United States and the UK and by industry executives themselves through their trade publications. It was even being considered for

inclusion in workmen’s compensation legislation in Britain. The first claim recorded in the United States was in 1927 in Massachusetts.\(^{25}\)

The first clinical reports in the United States were by Mills, Lynch and Smith, and Soper in 1930, the latter two in the national publications, *JAMA* and the *American Review of Tuberculosis*. In Mills’ first paper published in a state medical society journal, *Minnesota Medicine*, he reflected on the growing attention to this disease in the United States during the 1920s, although, he claimed, he was the first to document a case of what he called “an incurable disease.” He opined that because “asbestos is mined or manufactured in many portions of this country,” he was reporting the case “in the hope of arousing more widespread interest in the disease and establishing it as an entity with which American physicians surely will have to deal.”\(^{26}\) Indeed, *JAMA* picked up on Mills’ report bringing it to the attention of physicians all across the United States, pointing out that “asbestos is mined and manufactured in many parts of this country and that pulmonary asbestosis surely will be encountered.”\(^{27}\)

The scope of knowledge about the dangers from asbestos was captured in an authoritative epidemiological study of 363 asbestos textile workers, about a quarter of who showed signs and symptoms of fibrosis of the lungs in England. Organized by E.R.A. Merewether, the Medical Inspector of Factories and C.W. Price, the Engineering Inspector of Factories for the British

\(^{25}\) Lanza, A.J., “Asbestosis,” *JAMA*, 106 (February 1, 1936), 368: “While Hoffman called attention to the possible harmfulness of asbestos dust in 1918, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen’s compensation in Massachusetts.”

government, the report synthesized the huge literature that had developed over the previous decades. Citing the U.S. British, South African, French, Italian and German literature, it laid out the broad dimensions of the problem not just in the textile industry but in a broad range of other industries as well. The dangerous trades they identified included paper manufacturing, tile manufacturing, the production of insulation materials, the use of asbestos in brake and clutch linings, and its use as a sheathing for electrical conductors, cables and wiring. The scope of the problem led them to conclude “that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs.” They also identified various engineering controls that were necessary for the suppression of dust in industry. In his report he notes that among the “preventive measures” should be educating the worker “to a sane appreciation of the risk.”

Merewether also reported on his work in the United States publishing that same year in the *Journal of Industrial Hygiene*. Cross-sectional epidemiological studies were also initiated at the same time by American investigators. Between 1929 and 1931, A.J. Lanza and his colleagues at Metropolitan Life Insurance Company performed a study of Canadian asbestos miners and fabricators in both

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27 “Pulmonary Asbestosis,” JAMA, 95 (November 8, 1930), 1431.
28 Merewether, E.R.A., and C.W. Price, *Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*, London, Her majesty’s Stationary Office, 1930. A few years later, Merewether was even more explicit: asbestos was used in “a vast number of articles, ranging from matches to filter pads, from paints to roofing tiles, from high pressure jointing to electrodes, and from brake-linings to insulating materials in great variety. The phenomenal expansion of the motor car industry in the past twenty years has caused a corresponding increase in the demand for asbestos fabric brake linings, asbestos composition clutch rings, and latterly of moulded asbestos brake linings. Many other examples will come to mind.” See: Merewether, E. R. A., “A Memorandum on Asbestosis,” *Tubercle*, 15 (November, December, 1933 and January 1934) 69-81; 109-118; 152-159 at 71.
Canada and the U.S. for the asbestos industry which they published in 1935. They found that the longer a worker was exposed to asbestos the greater the chance that his or her X-ray would show that “prolonged exposure to asbestos dust caused a pulmonary fibrosis of a type different from silicosis.”

The widespread acknowledgement of the dangers of asbestos can be gleaned from the fact that in 1930 alone there were reports about asbestos disease by the International Labor Organization, the Monthly Labor Review, the Canadian Public Health Journal, the British Medical Journal, The Lancet as well as in other medical journals and at an international conference (see appendix C).

The concern with asbestos disease in the United States corresponded to the growth in the uses of asbestos in a wide variety of industries. Production increased dramatically in the United States in the first three decades of the twentieth century from “96,490 tons in 1910 to 434,938 tons in 1929.” Henry Pancoast and Eugene P. Pendergrass pointed out, in their review of the

31 Lanza, A. J., William J. McConnell, and J. William Fehnel, “Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers,” Public Health Reports, 50 (January 4, 1935), 1-12. See also Page, Robert C. “A Study of the Sputum in Pulmonary Asbestosis,” American Journal of Medical Science, 189 (1935), 44-55. Here the authors point out the short length of time necessary for the appearance of the disease: “The length of exposure to dust has varied within wide limits, but it is clear that gross disease may follow even as short an exposure as 18 months, provided the dust concentration has been sufficiently high.”


33 Egbert, Dan S., “Pulmonary Asbestosis,” American Review of Tuberculosis, 31 (1935), 25-34 at p.25. See also, for uses: “Memorandum on the Industrial Diseases of Silicosis and Asbestosis,” London, His Majesty’s Stationary Office, 1932: The processes and industries included among others: “crushing, opening of crude asbestos, blowing, mixing and carding of it”; “making insulating mattresses and slabs or sections composed wholly or partly of
field in 1931, the long latency and high incidence that characterized this chronic disease: “As the number of years at work grows for a worker, they show fibrosis. For those working 20 years and over 80.9% showed fibrosis.” They also pointed out the tragic consequences of prolonged exposure without respiratory protection: “Prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection....”34 The growing documentation of asbestosis in the early 1930s led observers to remark on the difficulty of keeping up with the literature. One observer in the Lancet remarked on this phenomenon: “During the past two or three years observations on the asbestosis body have been published with such frequency that it is very difficult to keep track of them all.”35 The growing attention to the pneumoconioses in general and the documentation of asbestosis in the medical literature led industry to sponsor research at the prestigious Saranac Laboratory in upstate New York. Leroy Gardner and Donald Cummings began their toxicological, inhalation and animal studies of asbestosis in January, 1928 and because “so much interest has recently been manifested in the subject of asbestosis, a preliminary report [was] submitted” in 1930. They concluded, “The physical and chemical properties of asbestos are so unlike those of any other dust previously studied that, when inhaled,

35 Gloyne, S. Roodhouse, “The Asbestosis Body,” The Lancet, 2 (1932), 1351. See also, Simson, F.W., and A. Sutherland Strachan, “Asbestosis Bodies in the Sputum: A Study of Specimens from Fifty Workers in an Asbestos Mill,” The Journal of Pathology and Bacteriology, 34 (1931), 1-4. In this survey “asbestos bodies… were present [in the sputum] in 48 out of 50 workers examined.” Editorial, “German Work on Pulmonary Asbestosis,” The Lancet, 2 (July 9, 1932), 93: “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 the signs of the disease.”
particles of this substance provoke an unusual type of reaction in the lung.”36 During this period research increased on the biological mechanisms by which asbestos created fibrosis in the lungs (see appendix C). Also, during this period recognition that the lungs were not the only organ affected by asbestos began to accrue. Medical literature showed that organs far from the site of primary contact ended up diseased by asbestos: “In asbestosis the lung is not the only organ invaded by asbestosis bodies. As we have shown, the spleen and the lymph nodes may harbor large asbestosis bodies.”37 Industrial engineering journals recognized the potential hazards of dust and asbestos specifically pointing out that “Asbestos and every operation in which it is used” is “seriously harmful.”38 These uses were expanding. The asbestos industry in Pennsylvania, for example, “consists mainly of several fabricating plants engaged in the making of asbestos cloth, brake lining, insulating tape, asbestos rope and wick, and other miscellaneous products.” It was also used in textiles, weaving, gasket making as well as insulation in steam pipes, asbestos cements, shingles, lumber, molded fittings, and household appliances.39 In England, by 1932, asbestosis was included as a compensable disease40 as it was in Wisconsin for a worker whose “occupation was that of cleaning and restoring the asbestos on pipes.”41

By 1932 while there was uncertainty as to the hazards of many mineral and organic dusts, silica and asbestos were singled out as the two about which there was no question. For example, Advisory Committee on Employment of Minors in Hazardous Occupations issued a report in *Monthly Labor Review*, the U.S. Department of Labor’s official publication, “that Minors under the age of 18 [should] be excluded [from] occupations involving exposure to free silica dust, asbestos dust, or other dusts in injurious quantities.”42 In addition, an article in *Industrial Medicine* cited one author who noted that “aside from silicosis and asbestosis, exact knowledge is not possessed on other dust diseases of the lungs.”43 Alice Hamilton, among the leading figures in occupational medicine, also highlighted the singular importance of the diseases silicosis and asbestosis in her summary of the activities of the Industrial Hygiene Section of the APHA: “Problems presented by industrial dusts are probably the most absorbing and important, and these particularly revolve around silicosis and asbestosis.”44

The medical, engineering, and social dimensions of asbestosis were well recognized by the early 1930s. C.O. Sappington captured the multidimensional nature of the problem thusly: “Asbestosis bodies are demonstrable in the sputum, in the lung tissue, and more recently, have been found in the lymph nodes and the spleen. The treatment of asbestosis apparently has been as unsuccessful as that of silicosis. It is believed that removal from exposure does not impede the progress of the disease, when once it is fully established. Prevention depends upon the same

procedures as in silicosis, with the added recommendation that mechanical devices be substituted for dusty hand work and that wet methods be used instead of dry.”

Industry, insurance, and government agencies were aware of this problem. The “Konicide Club” (Killer Dust Club) was composed of leading industrial hygienists and researchers concerned with the growing crisis over silicosis and pneumoconiosis in general. While “the liability crisis” around silicosis spurred its development, asbestosis also was a concern. At its 1933 meeting Leroy Gardner of Saranac Laboratory “spoke briefly on ‘What is Known About Asbestosis,’ an early contribution to a subject that looms so large today in the occupational health field.” By 1935 industry formed the Air Hygiene (later Industrial Hygiene Foundation). At its founding meeting of some 200 representatives of the “dusty trades” formed committees, one of which was to look specifically at asbestos, cork and non-metallic dusts. A representative of the Johns Manville Corporation, Vandiver Brown, was one of seven people nominated for the Temporary Organization Committee. In his report about the meeting to another asbestos company, Brown noted that “only two forms of dust, namely, free silica and asbestos, are definitely known to produce disabling fibrosis of the lung.”

45 Sappington, C.O., “Silicosis and Other Dust Diseases,” Industrial Medicine, 1 (December 1932), 158-68
46 Beyer, David S. , The Mechanical Control of occupational Diseases,” National Safety News, August 1933. 21-22, 34: “an analysis of approximately $300,000 in losses of one insurance company doing a general workmen’s compensation and liability business, for recent industrial disease claims, showing 54 per cent for cases involving pneumoconiosis (including silicosis, asbestosis, etc.) ....”
49 Brown to M.F. Judd, Secretary Raybestos-Manhattan, Inc, Bridgeport, Ct, January 22, 1935.
By the mid-1930s, asbestos was used in a wide variety of products, exposing workers, bystanders, and others to its ill-effects. One author noted, “the asbestos industry has had a remarkably rapid expansion and by reason of the widespread and varied uses of this product 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.”50 With this developing knowledge of the dangers of asbestos came attempts to define means of protecting the workforce. In the mid-to-late 1930s authors suggested levels of exposure to asbestos that could be “thresholds” of danger, above which harm was likely to occur. Dreesen and his colleagues, in a report for the U.S. Public Health Service in 1938, suggested that workers not be exposed to more than 5 million particles of asbestos per cubic foot (mppcf). He argued that workers breathing in asbestos dust resulted in the “initiation of fine, interstitial, pulmonary fibrosis” whose symptoms were dyspnea, variable cough “which sometimes raises blood streaked sputum and loss of weight.”51 Despite the quantification of this exposure limit Warren Cook, an industrial hygienist for Zurich General Accident and Liability Insurance Company observed that the “toxic limit for asbestos is five million particles of dust per cubic foot of air.” But he pointed out that “this is a very small concentration, so small in fact that the condition may look good even to a critical eye and still present an exposure greater than this low limit.” Thus, if asbestos dust in the air was

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visible, it was clearly above the threshold.\textsuperscript{52} The American Conference of Governmental Industrial Hygienists adopted this 5 mppcf threshold.\textsuperscript{53}

Researchers had always showed interest in the relationship of asbestosis to other lung diseases such as silicosis and tuberculosis. But, by the early 1930s, it was established that asbestosis alone was a serious disease that slowly killed its victims. In the mid-1930s troubling observations that some asbestos victims appeared on autopsy to also suffer from cancer of the lung arose. In 1934, a British study of one hundred workers exposed to asbestos for between six months and 15 years, fifty three of whom (thirty-four women and nineteen men) had asbestosis, revealed that two of the asbestos victims also had carcinoma of the lung and one had “a few deposits of growth in the pleura.”\textsuperscript{54} The issue of the relationship between cancer and asbestos exposure was “still an open question” which would occupy researchers for much of the next decade.\textsuperscript{55} In 1935, researchers in the United States began to look seriously at the relationship. Kenneth Lynch and W. Atmar Smith reported in the \textit{American Journal of Cancer} on a weaver in an asbestos factory who died from “pulmonary asbestosis with associated carcinoma of the lung.” The authors considered that it was “worthy of record in the interest of both diseases, as well as their possible relationship.”\textsuperscript{56} Anthony Lanza of Metropolitan Life reviewed the insurance company’s files and observed that of six asbestosis victims confirmed on autopsy, one had

\textsuperscript{55} Gloyne, S. Roodhouse, “Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis,” \textit{Tubercle}, 17 (October 1935), 5-10.
carcinoma.\textsuperscript{57} That same year, 1936, Dan Egbert and Arthur Geiger specifically addressed the possible relationship of cancer and asbestosis in an article in the \textit{American Review of Tuberculosis}. They believed that their report on a Hungarian immigrant weaver in an asbestos factory was “especially interesting since it is apparently the first to describe primary carcinoma of the lung as an associated condition.” They argued “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.”\textsuperscript{58} Gloyne, who had published the first piece linking asbestos and cancer in 1935, followed up this article with another case of “an oat cell carcinoma” a year later.\textsuperscript{59} In the late 1930s, reports began to cite this early literature, bringing attention to a possible relationship.\textsuperscript{60} In 1938 the issue was of enough concern that A.J. Vorwald, who would become the director of Laboratories at Saranac Laboratories, conducted a study of “Postmortem examination of 3739 individuals exposed to dust, as compiled from the literature and from our own series” which “revealed 30 individuals with pulmonary carcinoma, or an incidence of 0.8 per cent.” He concluded at the time that “this incidence [was] lower than that reported in routine autopsy examinations of the general populations” leading him to conclude that “Inhaled dusts, therefore, except those containing recognized carcinogenic substances such as radium and tar, cannot in general be considered as

\textsuperscript{57} Lanza, A.J., “Asbestosis,” \textit{JAMA}, 106 (February 1, 1936), 368-69.
\textsuperscript{58} Egbert, Dan S. and Arthur J. Geiger, “Pulmonary Asbestosis and Carcinoma,” \textit{American Review of Tuberculosis}, 34 (1936), 143 - 150.
\textsuperscript{60} Anderson, C.S. and J. Henry Dible, “Silicosis and Carcinoma of the Lung,” \textit{Journal of Hygiene}, 38 (1938), 185-
etiological factors in the development of primary pulmonary carcinoma.”\textsuperscript{61} JAMA reported on Vorwald’s study and results.\textsuperscript{62} The International Labor Organization (ILO) was more cautious in its interpretation of this limited evidence. It noted that exposure to asbestos was shown to result in “four main complications” -- bronchitis, pneumonia, TB, emphysema – but also to two others “which are less common” including carcinoma. In 1938 “the number of cases … described [was] small and it [was] not yet established that there is any aetiological connection between the two diseases,” yet there was “sufficient evidence to warrant careful observation in the future.”\textsuperscript{63} The ILO warned that “a practical maxim of the greatest value is that every translation of fiberised asbestos in the factory produces dust which, if not controlled, is dangerous.”\textsuperscript{64} J.V. Sparks picked up on Gloyne’s research and published in the British Journal of Radiology. This was, in turn, reported on in the American Journal of Industrial Hygiene and Toxicology in 1939 where industrial physicians and hygienists learned that among the chief complications of pulmonary asbestosis was carcinoma.\textsuperscript{65}

Between 1938 and 1942 there were at least six additional articles reported in the American and British literature about the relationship of asbestos and cancer. Some of these were reports or abstracts of German-language studies, including a 1938 article by Nordmann who reported on the

\textsuperscript{61} Vorwald, Arthur J. And John W. Karr, “Pneumoconiosis and Pulmonary Carcinoma,” American Journal of Pathology, 14 (January 1938), 49-58. He also exposed 3,338 animals to various dusts and found a lower-than-expected carcinoma rate.
\textsuperscript{62} “Pneumoconiosis and Pulmonary Carcinoma,” JAMA, 110 (June 18, 1938), 2068.
\textsuperscript{63} International Labour Office, Occupation and Health: Encyclopedia of Hygiene, Pathology and Social welfare,” ILO, 1938, pp.9-10.
\textsuperscript{64} International Labour Office, Occupation and Health: Encyclopedia of Hygiene, Pathology and Social welfare,” ILO, 1938, p.14.
\textsuperscript{65} Sparks, J.V. “Asbestosis,” British Journal of Radiology, 11 (June 1938), 371 - 377. Reported in Journal of Industrial Hygiene and Toxicology, 21 (1939), 54.
high number of cancers revealed on autopsy of workers who had asbestosis.\textsuperscript{66} American researchers Lynch and Smith cited Nordmann and others as providing further documentation of the link between asbestos, asbestosis and cancer. They reported on another case but acknowledged that the evidence-base was too small to establish a causal relationship. By 1939 there were a total of eight documented cases linking asbestos to lung cancer.\textsuperscript{67} By 1942 the number of reported cases had increased to ten and that inhalation of asbestos caused “irreparable damage” even in workers employed for long periods of time where “exposure to asbestos dust was relatively low.”\textsuperscript{68}

Wilhelm Hueper, the eminent student of industrial disease, published the classic, comprehensive study of \textit{Occupational Tumors and Allied Diseases} in 1942, noting the growing number of case reports in England, Germany and the United States. In this massive tome Hueper lays out the basis for connecting lung cancer deaths to exposure to asbestos at the workplace. First, he noted that the “incidence of lung cancer in asbestosis of the lung … is definitely excessive.” Second, “the young age of an appreciable portion of the cases … and the relationship existing between the time of exposure to asbestos dust and the manifestation of the neoplasms” was “suggestive” of such a relationship.\textsuperscript{69} The following year Hueper reported that “Asbestosis

\textsuperscript{68} Holleb, Herbert B and Alfred Angrist, “Bronchiogenic Carcinoma in Association with Pulmonary Asbestosis,” \textit{American Journal of Pathology}, 18 (1942), 123-131 at p. 129.
\textsuperscript{69} Hueper, W.C., \textit{Occupational Tumors and Allied Diseases}, Springfield IL: Charles C Thomas, 1942, p.404. See also, Davis, Edwin, “Chemical Carcinogenesis, Drugs, Dyes, Remedies and Cosmetics with Particular Reference to
cancer of the lung is the most recent newcomer among the occupational cancers of this organ” in an article published in The Bulletin of the American Society of the Control of Cancer. He noted that “asbestosis cancer of the lung has for us a special hygienic and sociologic significance” since “the asbestos industry is most extensively developed in this country.”

During World War II the medical literature on both sides of the Atlantic continued to follow each other’s reports of cancer and asbestos exposure. Specifically, articles in the American literature, including the New England Journal of Medicine, the Journal of Industrial Hygiene and Toxicology, and the Industrial Hygiene Foundation’s Industrial Hygiene Digest, cited German studies that continued to document the link. The latter two abstracted an article by H.W. Wedler which noted that the “German and foreign literature” clearly documented the relationship: “These figures [that twenty percent of autopsied asbestosis victims had lung cancer] clearly show that lung cancer is the most common complication encountered in cases of asbestosis, with the exception of agonal pneumonia and cardiac insufficiency. Even tuberculosis, whose rate of incidence is generally far higher than that of cancer, is considerably lower than for lung cancer in these cases. This constitutes conclusive evidence of the close correlation between asbestosis and lung cancer.”

Also, during the war the official publications of the major medical and public health journals continued to follow each other’s reports on the subject. For example, an article in the Journal of Urology, 49 (1943), 14-27, cited H. W. Wedler, “Lung Cancer in Asbestosis Patients,” Dtsch. Arch. Klin. Med., 191 (1943), 189-209. (In German but cited several times in subsequent literature.)
health societies in the United States, the American Medical Association and the American Public Health Association, as well as the International Labour Organization, reported on the suspected link between asbestos and cancer.\textsuperscript{73}

Shortly after the war ended Wilhelm Hueper published articles in \textit{JAMA} and the journal, \textit{Occupational Medicine} about this relationship. In the latter Hueper was especially vivid in his description of what occupational cancers, including those caused by asbestos, did to American workers: “The continued occurrence of occupational cancers,... represents a challenge not only to the intelligence but also to the social conscience of human society, because industrial cancers may be compared with a biologic bomb having a delayed time fuse which may be placed in the body of the victim without his knowledge and realization and which may display its deadly effect many years later when the conditions connected with its introduction are often forgotten.”\textsuperscript{74} The responsibility of employers and sellers alike to warn anyone who came in contact with hazardous materials, including dusts, was recognized by the leading trade association for the chemical industry in 1945: “The education of employees regarding chemical hazards is, and must remain, the direct responsibility of their employers. However, such hazards are not confined to employees alone, and information concerning them should, so far as practical, reach every person using, transporting, or storing chemicals. The most practical means for the seller to disseminate this


information appears to be by labels affixed to containers of hazardous chemicals, bearing appropriate precautionary statements and instructions stated as simply and briefly as circumstances permit.”75

The already documented cases of cancer of the lung and asbestosis spurred others to investigate this further. Following the war many more such cases were reported. In 1949, the publication of the “Annual Report of the Chief Inspector of Factories for the Year 1947” quantified the relationship between asbestosis and lung cancer, noting that between 1924 and 1946 13.2 percent of a sample of 235 autopsies of asbestosis victims also had lung cancer. This was ten times higher, the report noted, than the lung cancer rate among silicosis victims confirming that asbestos was indeed a cause of lung cancer. The average length of exposure for those victims with lung cancer was 16.5 years.76 Studies in the United States also noted the high incidence of asbestos-related cancer.77

While there was some dissent,78 by 1949, leading American publications accepted this data and wrote that asbestos caused lung cancer. An editorial in JAMA, “Asbestosis and Cancer
dust and to asbestos.”

78 See, for example, Smith, William E., “Survey of Some Current British and European Studies of Occupational Tumor Problems,” Archives of Industrial Hygiene, 5 (1952), 242 – 263 and the controversy found in “Cancer of the
of the Lung,” remarked on the sea-change that had overtaken medical thinking. “Until recently the coexistence of asbestosis and cancer of the lung was considered by many investigators a coincidence…. A causal relation between asbestosis and cancer of the lung is supported by the following observations: the incidence rate of cancer of the lung in this group is excessive, since the normal death rate from cancer of the lung among adults examined at necropsy at present is about 1 per cent of all necropsies.” This was particularly important “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.”

Among these “asbestos-consuming industries” were “the manufacture of fire-fighting suits, safety curtains, and boiler mattresses. Asbestos enters into the composition of lagging material for steam pipes, jointing for steam pipes, panelling [sic] of rooms, tiles, the lining of chemical pans, the coating of bulkheads of ships and marine piers and perhaps most significantly into the brake linings and clutch rings of motor cars.” An article in Scientific American also listed asbestos as a human carcinogen. The number of articles documenting this relationship cascaded in the early 1950s (see appendix C).

The studies in the early 1950s by Hill and Doll Sir Richard Doll, confirmed for virtually all serious scientists the relationship between cancer and asbestos. In his article, “Mortality from

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Lung Cancer in Asbestos Workers,” published in the British Journal of Industrial Medicine, he looked at the autopsy reports of 113 men who worked in asbestos factories and found “an increased mortality from lung cancer, other respiratory diseases and cardiovascular diseases, in association with asbestosis.” “From the data [which showed 11 lung cancer deaths when only 0.8 were expected] it can be concluded that lung cancer was a specific industrial hazard of certain asbestos workers.” He went on to say “that the average risk among men employed for 20 or more years has been of the order of 10 times that experienced by the general population. The risk has become progressively less as the duration of employment under the old dusty conditions has decreased.”

Doll’s work was cited by the prestigious British Medical Journal in an editorial on lung cancer and occupation. That same year Wilhelm Hueper, the chief of the Environmental Cancer Section of the National Cancer Institute in the United States published an editorial in the American Journal of Clinical Pathology, in which he concluded that “the epidemiologic and pathologic evidence supporting a causal relationship between asbestosis and cancer of the lung ... is quite substantial” and that “the available evidence is adequate for recognizing asbestosis cancer of the lung for medicolegal reasons as an occupational disease.” By the end of the 1950s asbestos was generally cited as a cause of lung cancer in American medical textbooks. This was

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confirmed by much evidence including prospective cohort studies of workers in a number of different trades. It was found that “asbestos workers had the second highest observed lung cancers … of the 10 occupations.”\textsuperscript{86}

Although the risk of asbestos disease in bystanders was known, this issue, including the risk of both asbestosis and cancer, increased during the 1960s. In 1962, Milton Scheinbaum reported in the \textit{American Industrial Hygiene Association Journal} that there were serious threat from airborne asbestos particles to “anyone working within fifty feet” of someone applying asbestos cement plaster with a pneumatic tool.\textsuperscript{87} By the mid-1960s Wilhelm Hueper warned that “asbestosis and asbestos cancer hazards related to an inhalatory exposure to asbestos exist not only for asbestos workers proper … but also for the large number of individuals who may sustain such contacts on an incidental basis. Such persons may be employed permanently or temporarily in or near operations where asbestos and asbestos products are produced or handled and where they inhale…”\textsuperscript{88}

The scope of the asbestos problem expanded dramatically in the post World War II era as the number of potential victims increased with the growing number of uses of the material. At a major conference on asbestos at the New York Academy of Sciences in 1964, Wilhelm Hueper stated that world-wide production of asbestos increased from 500 tons in 1880 to 330,000 tons in 1925 and quadrupled from 446,000 metric tons in 1938 just before the War to over 2,000,000 in

1958, two decades later. By 1964, production was over 3 million tons. This was especially important in the United States as it was “the principal consuming country.” At the same conference the keynote speaker, J.C. Gilson, the British epidemiologist, told his fellow scientists that “Asbestos can truly be called the 20th century mineral. Its output has increased over a 1000-fold in 60 years, compared with a mere 50 fold for oil – an industry often regarded as the symbol of industrial growth.” By 1964 observers noted the wide range of uses for asbestos and the “multiplicity of circumstances under which asbestosis can be encountered.” In a report of the Section on Nature and Prevalence Committee on Occupational Diseases of the Chest of the American College of Chest Physicians it was noted that there were – “3,000 specific conditions in which asbestos serves uniquely as a protecant against excessive heat, cold and corrosion.” Major uses of asbestos included textiles, cement products, friction materials and gaskets, paper, floor tile, paints, roof, caulks, plastics. But G.W.H. Schepers “stressed that the medical and health problem originating through exposure to asbestos dust may come about equally through minor uses,” as well. Another author noted the increasing dangers of asbestos produced for use in wide variety of products, including blankets, clothing, threats, rope, tape, brake linings, wall paper, wallboard, shingles, floor covering, plastics, insulation for pipes, boilers, wire, heating pads. Like

92 Hendry, N.W., “The Geology, Occurrences, and Major Uses of Asbestos,” *Annals of the New York Academy of Sciences,* 132 (1965), 12 – 22 and Schepers at p.21. See also, “Complications of Asbestosis,” *British Medical Journal,* 1 (April 30, 1960), 1345 - 1353. P. 1350 Comment by Dr. Hugh-Jones: “Asbestosis is a pneumoconiosis arising from the inhalation of asbestos dust during the manufacture of asbestos goods, such as asbestos sheeting, fireproof clothing, brake linings, lagging for boilers and pipes, etc.”
Schepers and others before him, H.B. Eisenstadt remarked that “due to the rapid expansion of its use, an ever-increasing number of craftsmen and laborers are exposed to the mineral.” He even cited a case of an auto mechanic who acquired asbestosis “while undercoating vehicles.”

The first U.S. case reports of cancers other than those of the lung was recorded in 1947 when Tracy Mallory, Benjamin Castleman and Edith Parris reported in the *New England Journal of Medicine* “case 33111” from the Massachusetts General Hospital. “Case 33111” was a 37 year old worker from Sweden whose work was cutting asbestos insulating board. He was diagnosed with mesothelioma of pleura and pericardium. The physicians noted the unusual character of this case for “a number of papers have been written to the effect that there is no such tumor as mesothelioma of the pleura, that the cells lining the pleura do not form tumors and that these tumors really arise from a small focus in the lung.” They had “held a similar opinion for a long time.” But the case forced them to reevaluate their belief. “This is perhaps the first case in which we believed that there was actually such a tumor.”

J.C. Wagner, C. A. Sleggs, and Paul Marchand documented the relationship between asbestos exposure and mesothelioma in a 1960 article published in the *British Journal of Industrial Hygiene* where they detailed “33 histologically proven cases” of mesothelioma, which they described as being “regarded as an uncommon tumour” during the previous four years. They

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reviewed the existing literature and concluded, “Our findings suggest that mesothelioma occurs 20 to 40 years or more after exposure to dust.” As early as 1962 researchers noted that “mesothelioma is now accepted by the majority of pathologists as an entity and in recent years diffuse or malignant mesothelioma has been shown to be associated with pulmonary asbestosis in Canada, Holland, South Africa, and Germany.” It was noted that not only workers were at risk but those “who lived in areas where asbestos was mined or treated” were also at risk. He also warned that there were “increasing number of people manufacturing, handling, or using these products.” He worried that the “widespread… use of asbestos today” has resulted in “mesothelioma of the pleura and peritoneum in people who have occupations in which association with asbestos is not suspected.”

Evidence continued to mount regarding the risk to bystanders and consumers of products using asbestos fiber. In their summary of their findings, Dermot Hourihane argued, “the association of these tumours [mesotheliomas] with asbestos bodies in lung tissue is confirmed.” He pointed out that “many of the cases gave no history of industrial exposure, and it is possible

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95 Wagner, J.C., C. A. Sleggs, and Paul Marchand, “Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province,” British Journal of Industrial Medicine, 17 (1960), p. 267: “In 1924 Robertson denied the existence of primary malignant tumours of the pleura and considered them to be secondary in origin. Since then, on the one hand, Willis (1948, 1953) and Smart and Hinson (1957) have supported Robertson’s views, while on the other hand, primary neoplasms of this nature have been described by many authors in recent years. These include Tobiassen (1955) in Sweden, Belloni and Bovo (1957) in Italy, Godwin (1957) in the United States, and McCaughhey (1958) in Britain. Evidence of the mesothelial origin of these tumours can be found in the tissue culture experiments of Stout and Murray (1942) and Sano, Weiss, and Gault (1950). They also noted Doll’s study which included one case of mesothelioma.


97 Thomson, J.G., “Mesothelioma of Pleura or Peritoneum and Limited Basal Asbestosis,” South African Medical
that temporary or relatively trivial exposure may have occurred.” The severity of the disease was grave as “once symptoms from the tumours have arisen, the prognosis appears to be measured in months rather than years.”^98 In the United States, Irving Selikoff built on the British and South African literature with an extensive epidemiological study of insulation workers in the building trades who had “relatively light, intermittent, exposure to asbestos.” He found that of 632 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.” Remarking on the mesothelioma findings, Selikoff wrote “the incidence of more than 1% of deaths from pleural mesothelioma is strikingly high for a tumor which is generally considered to be extremely rare.” This augured poorly for other building trades occupations: “insulation workers undoubtedly share exposure with their workmates in other trades; intimate contact with asbestos is possible for electricians, plumbers, sheet-metal workers, steamfitters, laborers, carpenters, boiler makers, and foremen.”^99 Selikoff also expressed concern that mesothelioma specifically was more than an occupational hazard and was one which “will be found to be a community problem” as well.^100 Others worried about the growing dangers of asbestos exposure. A British report noted that “(1) carcinoma of the lung, and (2) diffuse

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mesothelioma of the pleura and peritoneum,” were clearly recognized and that “there is some suggestion of an association also with gastro-intestinal carcinoma, and possibly ovarian tumours.” Because of the long latency of the disease, “further cases of these associated tumours are expected to occur for many years to come, even if dust exposures are now greatly reduced.”

Wilhelm Hueper summarized the state of knowledge and the depressing implications of the information available in a paper presented at the New York Academy of Sciences in 1964:

“Numerous reports by many investigators from various countries establish the fact that an occupational or nonoccupational inhalation of dust of different chemical and physical types of asbestos results in the development of asbestosis of the lungs which, depending upon the type of exposure may be designated as an occupational, neighborhood or household pneumoconiosis. Since 1935 an increasing amount of epidemiologic, clinical and pathologic evidence, moreover, incriminates this health hazard as one of the environmental sources of cancers of the lung, and more recently also of mesotheliomas of the pleura and peritoneum, although some commercially interested parties and their medical guardians and protectors still prefer for their own reasons and motives, to deny

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the existence of these dangerous and usually fatal sequelae of a respiratory contact with asbestos dust. The obvious fallacy of such allegations is well demonstrated by the remarkable and a progressive rise in the number of cases of asbestosis and cancers of the lung, pleura, and peritoneum reported from various countries during the past two decades.”¹⁰²

For the next four decade researchers would build on these basic observations. By the 1970s the federal government had incorporated the expanding literature on asbestos, asbestosis and cancer into the regulations adopted by the newly-formed federal Occupational Safety and Health Administration. In 1972, shortly after its formation, the National Institute of Occupational Safety and Health issued a “Criteria Document for a Recommended Standard … Occupational Exposure to Asbestos” that warned that “The recommended standard [was] designed primarily to prevent asbestosis,” and that “for other diseases associated with asbestos, there is insufficient information to establish a standard to prevent such diseases including asbestos-induced neoplasms by any all-inclusive limit other than one of zero.”¹⁰³ That same year, OSHA adopted the NIOSH recommendation for a “standard of 8 hour TWA airborne concentrations of asbestos dust not exceeding five fibers longer than five micrometers per millimeter.” OSHA “concluded that there should be one minimum standard to asbestos applicable to all workplaces exposed to any kind, or

mixture of kinds, of asbestos.”¹⁰⁴ NIOSH revised its recommendation in 1976, summarizing the consensus that had evolved through the scientific, epidemiological and medical studies of the dangers from asbestos to workers, by-standers and the population at large:

“All available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have occurred following exposure to chrysotile, crocidolite, amosite, and anthophyllite. Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have developed also in individuals living in the neighborhood of asbestos factories and near crocidolite deposits, and in persons living with asbestos workers…."¹⁰⁵

Animal studies confirmed the observation that “all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or IP injection.”¹⁰⁶ The available data indicated that asbestos had no safe level where workers or others would be protected from cancers. “There are data that show that the lower the exposure, the lower the risk of developing cancer,” NIOSH suggested.

But “excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a ‘safe’ level of asbestos exposure.” The standard that was recommended, 100,000 fibers $>5\mu$m in length/m$^3$, was intended to “protect against the noncarcinogenic effects of asbestos,” and also to “materially reduce the risk of asbestos-induced cancer.” But, NIOSH explained that “only a ban can assure protection against effects of asbestos….”

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Appendix C
Chronological List of Some Relevant Articles
(revised February 13, 2013)

1898

P. 171: “Of all the dusty occupations which specially came under observation in 1898 three ... stand out on account of their easily demonstrated danger to the health of the workers, and because of the ascertained cases of injury to bronchial tubes and lungs medically attributed to the employment of the sufferers. These occupations were asbestos sifting and carding, silk opening and combing and hemp spinning.... In the case of one particular asbestos works, which I visited with Miss Deane, far from any precaution having been taken the work (sifting, mixing and carding) appeared to be carried on with the least possible attempt to subdue the dust ... no sort of ventilation being applied.”

P. 172: Miss Deane reports on the abundant evidence she has had of the evil effects of dust: – “In the majority of cases the evil is very insidious, and the general symptoms produced by dust on the various respiratory organs are to the lay mind so similar to those produced by other causes that it is not always easy to trace the connection..... The evil effects of asbestos dust have also attracted my attention, a microscopic examination of this mineral dust which was made by H.M. Medical Inspector clearly revealed the sharp, glass-like jagged nature of the particles, and where they are allowed to rise and remain suspended in the air of a room, in any quantity, the effects have been found to be injurious, as might have been expected.”

1899

P. 264: “Among other dusty processes which engaged the attention of Miss Patterson and myself in1899 were those in works for the manufacture of non-conducting jacketing for pipes, involving use of asbestos (the injuriousness of which substance was demonstrated in last year’s report ....” need action to remove dust.

1902
Oliver, Thomas, Dangerous Trades, New York: E.P. Dutton, 1902.

P.25: “In the great civilizations of antiquity, whether in the East, West, or in Europe generally, there was sufficient concentration of the forces of labour to produce the intensest forms of the maladies classed by Pliny as the ‘diseases of slaves.’ Some of the most injurious processes known to us now are extremely
ancient. To mention but a few: lead and quicksilver mining, the potters’ craft, and the textile processes of preparing and weaving asbestos and flax.”

1906  
**Great Britain Factory Department, Lady Inspectors, Annual Report of the Chief inspector of Factories and Workshops for the Year, 1906, London, His Majesty’s Stationary Office, 1907.**

P. 219: “Of all injurious dusty processes of which I have again in 1906 received repeated complaints, some, I believe surpass in injuriousness to the workers the sieving, preparing, carding and spinning processes in manufacture of asbestos.... As we reported in 1898, microscopic examination of this mineral dust shows the sharp jagged nature of the particles, and wherever they are allowed in this manner to remain suspended in the air, injury, more or less serious, ensues for the respiratory organs of the workers.”

P. 220: Miss Deane re-visited recently for me the asbestos works on which I specially reported to you in 1898 on account of marked injury at that time to the health of the worker. There a greatly improved installation of exhaust ventilation was applied to the carding machines and Miss Deane reports now that there is far less complaint of injury to health.”

1907  

P. 127: Q – Have you any doubt in your mind that asbestos dust does cause fibrosis? “I think there is no doubt it did in this one case.” Man was 33 years old and had worked for 14 years, first ten in carding room. First sent to him in 1899.

1910  
**J.M. Beattie** conducts animal experiments showing fibrosis of lungs following exposure to asbestos dust. [noted in Merewether, “Report” 1930, p.5]

1912  
**“Effects of Asbestos Dust on Workers’ Health in Asbestos Mines and Factories,” Labour Gazette (Can.), 12 (1912), 761-672**

P. 761: Investigation by Labour Department on the “dusty processes connected with the manufacture of asbestos...particularly to the effect of the processes concerned on the health of those employed in the asbestos industry.”

P. 762: Use of respirators and then fans and dust conveyors to keep dust down. “Studying new method by which all the offensive dust will be collected by means of electric magnets.” “One of the oldest medical practitioners in Thetford expressed the view that the asbestos dust floating in the atmosphere of the cobbing room had a weakening effect on the lungs of those employed.”

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p. 249: Problem of ignorance of hazards at work: “It is recognized that the education of employees is one of the most important features in safety work, but the large number of transient men makes this educational feature a difficult problem. Experience also indicates that a greater effort should be made to instruct the new man as to the hazards of his occupation before he is permitted to be placed in a hazardous position.”


Pp. 102-3: cite asbestos dust as cause of pneumoconiosis; 1st description in medical literature on x-ray changes from asbestos; studies asbestos workers

In discussion Dr. Kober: “Safely said that persons exposed to the habitual inhalation of dust, sooner or later develop would develop some chronic inflammation of the respiratory system.”


P. 176: Aside from the mining of asbestos, in which the exposure is rather limited, numerous processes involving the conversion or remanufacture of the materials which are distinctly more serious in their effects on health and longevity.”

Uses: cover pipes, furnaces, wall plaster, roofing, electricians, construction of storage rooms of refrigerating plants.

P. 177 dust exposure in spinning and weaving of asbestos.

P. 178: “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.”

P. 179: need more information; quotes from Annual Report of Chief Inspector of Factories and Workshops, 1910 a report by Collis on the asbestos industry that need dust protection because of deaths of workers exposed to asbestos dust. Noted that there was a by-stander hazard: “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.”
P. 180: “urgent need for extensive investigation of health effects of asbestos manufacture.”


P. 129: Studied the chests of 137 individuals “engaged in various dusty occupations” including 15 asbestos workers.

P. 136: The 2nd stage of pneumoconiosis “is characterized by a more or less uniformly arranged mottling throughout the lung structure due to depositions of dust in the lymph structures, cells, and fibrous tissue interspaces, with the addition of a certain amount of localized fibrosis....It occurs comparatively early in coal miners, cement workers and certain metal grinders, somewhat later in potters and asbestos workers....”


  p. 365: “Tests in cement clinker grinding, asbestos carding rooms, and flint mills, and pottery factories showed a great reduction in the amount of dust in the air brought about by installation of efficient suction dust removal at the point of production. This also resulted in a marked decrease in the percentage of smallest sized particles and these we feel are the ones most likely to reach the lungs of the workers and cause fibrosis.”

  p. 366: “In the tests in mineral dust the best conditions were found in the oil grinding room of the ball bearing factory and the next in the best hooded asbestos carding room, but in general the pottery industry was least dusty. Some of the potters examined by Dr. Pancoast showed very marked fibrosis in the lungs but these were men who had been working many years in the industry, starting when no attention was paid to minimizing or removing dust. The worst conditions were in the cement, and the next in the steel ball grinding room, with the unprotected asbestos crushing and screening room almost as bad.”

1919  Proceedings of the National Safety Council, Eighth Annual Safety Congress, Cleveland Ohio, 1919

  p. 22: “The Library and bureau of information at our headquarters has grown steadily with the growth of the Council. During the past year valuable additions have been made covering industrial relations subjects. Our efficient Librarian, Miss Mary B. Day, has visited the best libraries in the East and has gathered much valuable data along new lines. Through weekly news-letters and other means the Council’s members are being constantly encouraged to avail themselves of the services of the Library.”
1924  Cooke, W. E. “Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust,” The British Medical Journal, 2, No. 3317, (July 26, 1924), 147

Cites J. M. Beattie previous work that shows that asbestos dust causes fibrosis in guinea pigs; Article shows that asbestos dust causes fibrosis in woman who worked in asbestos factories since the age of 13 and died at age 33.


Mentioned in July issue a case in Rochdale “where an employee of an asbestos plant had died from asbestos poisoning, and this was later made the basis of a suit against Turner Brothers Asbestos Company, Limited. A somewhat similar case has occurred in America, concerning James Nixon an employee of the Southern Asbestos Company at Charlotte, N.C. In a $30,000 damage suit instituted by Hattie Nixon, it is claimed that James Nixon contracted tuberculosis from dirt, trash and other particles flying thru the air like a snowstorm at the plant, going into the nostrils of the employees.”


P. 385:“In dusty factories, such as asbestos and cement works, cleanliness and ventilation are essential, although the actual danger in these two industries seems to be comparatively slight.”

Pp. 419 - 420: Asbestos Workers. “This is another very dusty industry. Asbestos is a magnesium silicate, and the rock that is crushed contains more or less free silica, to which the workers may be exposed all the way through the process of manufacturing to the finished product. ... With Miller and Landis we examined 17 asbestos workers, 2 of whom showed first stage changes and the other 15 definite second stage appearances. Of the men longest at work, one after seventeen years’ occupation showed very definite diffuse, ‘soft’ spots throughout both lungs and another showed about the same appearance after fourteen years’ occupation. Very slight nodular shadows were found in one man after only two years’ occupation.”


Same as above on  P. 22 and p. 155.

P. 1024: Cites earlier case from 1900 of man confirmed dying of asbestos fibrosis also at 33 years. All other 9 men who started with him had died presumably from occupational disease.

“During the carding process, and to a less extent in all the processes, a very considerable amount of dust is generated.”

P. 1025: “we have never seen anything parallel to this in pneumoconiosis due to other dusts....”


Case study – microscopic appearances of the dust were identical with those of the particles found in the lungs.

“Pneumoconiosis Caused by Asbestos Dust,” *JAMA*, 89 (July 23, 1927), 304.

Reports on paper presented by Cooke and Hill at a conference of the Royal Microscopical Society.

Oliver, Thomas, “Pulmonary Asbestosis in its Clinical Aspects,” *Journal of Industrial Hygiene*, (November 1927), 483-485. (Also printed as “Clinical Aspects of Pulmonary Asbestosis,” in *The British Medical Journal*, 2, No. 3491, (December 3, 1927), 1026-27. Also reported in *JAMA*, 89 (1927), 2285.

P. 1027: “Pulmonary asbestosis ... resembles silicosis of the lungs in the marked shortness of breath on slight exertion, deficient respiratory capacity, physical debility, ....”


Diagnosis of asbestos fibrosis.


“Pulmonary Asbestosis. An important article on this subject appeared in the British Medical Journal of December 3rd....”

P. 119: The Cooke case of 1924 “only the second reported [but] many other cases seem to have occurred.” Conditions are better now – factories remove more dust. “Nevertheless, asbestosis, because it its dangers and its unique pathologic features, deserves more attention than it has had.”


“Pulmonary asbestosis has become a question of international interest and importance.”


P. 321: Not mention asbestos specifically: Although it is possible to say that some dusts are less dangerous than others, all should be held under suspicion until they are proved innocent.”


Case reported – worked in asbestos industry – carding, weaving, etc.


P. 885: Several Cases in South Africa: “It has been known for some time that workers exposed to the dusty atmosphere arising from some processes involved in the preparation of asbestos materials suffer from pulmonary disability.”


1929 [Haddow] in “British Medical Association: Occupational Diseases – Section of Occupational Diseases – Asbestos Dust,” *The Lancet,* Aug. 3, 1929, pp.230-231. “Dr. A.C. Haddow (Leeds) described the clinical aspects of asbestosis, and quoted a case in which the curious bodies were found in a man who was
not employed in the industry, but who lived next door to an asbestos factory.” (p.231).

1929


UK considering putting asbestos under workers compensation.

1929


Fatal cases of asbestosis – average age 41 – average time in asbestos manufacture – 20 years;

P. 581: Asbestos produces fibrosis; usually first recognized after more than 5 years – but if more “systematic examination” might reveal it earlier; “the mortality statistics will probably be swelled by the wider recognition of this disease.”

1929


P. 1147: “A dust-free industry may be ideal, but it should be our goal.”

1929


More on nature of asbestos bodies in lungs.

1929


P. 407: “Asbestos fibres can be demonstrated in the corns from which the workers suffer, in the upper respiratory tract, in sputum in the lung, post mortem, and in animal tissues. The so-called ‘curious bodies’ are derived from asbestos fibres. It is suggested that asbestos bodies would be a better name for them than ‘curious bodies.”

1929


1929


   P. 356: “When the diagnosis is established, the prognosis appears to be grave.”


   P. 19: Asbestosis is unique among the minerals in being fibrous, and the dust generated during the manufacturing process is also unique.”


   P. 495: First U.S. Case. “Interest in the injury to the lungs produced by the inhalation of asbestos dust has been aroused in the last six years, and several reports of cases have appeared in the medical literature of Great Britain. Aside from an occasional editorial or incidental mention, little has been written in this country about the condition. Apparently this is the first case reported from the United States. Nevertheless asbestos is mined or manufactured in many portions of this country. Undoubtedly injury to the lungs of some of these numerous workers has occurred and may have been directly or indirectly responsible for death without the real condition being recognized. This case is reported in the hope of arousing more widespread interest in the disease and establishing it as an entity with which American physicians surely will have to deal.”

   P. 496: Contracted in South America where he had drilled asbestos bearing rock.

   P. 498: “Perhaps the most valuable feature of this case is the extreme length of time that elapsed between the exposure to the dust and death.... In short, asbestosis is an incurable disease, and one from which the patient may or may not die.” “This report corresponds reasonably well with reports from Great Britain.”

P. 571: case report of a worker in Connecticut in an asbestos factory – first symptoms 12 years after first exposure. Asbestosis “would appear to much more widespread than at present suspected.” Not been sufficiently recognized by MDs

P. 578: Prognosis grave.

P. 583: “when there has been exposure to asbestos dust, the presence of pulmonary asbestosis should always be suspected.”


P. 659: “Until recently asbestosis has been assumed to be essentially silicosis....” “It has come to attention within the last three or four years that there is a peculiar characteristic in the state of asbestosis, not found in silicosis.”

No report in American medicine and our purpose “that particular interest may be stimulated in its study in the extensive asbestos industry.”


Case in Italy


Detailed and authoritative epidemiological study of 363 asbestos textile workers, about a quarter of whom showed signs and symptoms of fibrosis of the lungs.

p. 6: “Asbestos is very largely used in industry, being an important constituent of many different products. The diversity of industries concerned made it essential that some restriction of the field of investigation should be decided upon.”

P. 9: “The examination of the data collected in this investigation leads to the conclusion that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs.”

P. 11: documents impact of length of employment.

P. 13: “risk falls heavily on those longest employed and on those engaged in the more dusty processes.”

P. 17: need suppression of dust. Also, educate the workers “to a sane appreciation of the risk.”

10
P. 31: dangerous trades: textiles, millboard, paper, sheets, tiles, insulation materials and articles, brake and clutch linings, covered electric conductors, cables and wiring.

Need much less dust – exhaust, enclosure, wet methods
Dust produce by sawing, grinding, and other abrading.


Comprehensive examination of asbestos industry in England.

P. 200: “The result of this investigation did not conclusively prove that asbestos possessed injurious properties, but it pointed to the probability that such was the case.”

P. 204 repeats views about the wide range of industries – “the diversity of industries which nowadays find its use necessary, either in the form of the raw material, or as manufactured articles.” looks at 16 cases.


P. 190: “Worker engaged in the extraction and manufacture of asbestos is constantly exposed to danger from dust.”


P. 27: “Asbestos damaged more rapidly than silica.”


Cites recent Oliver article.


List of 17 articles on asbestosis.

“Some attention is being given by the U.S. Bureau of Labor Statistics of the Department of Labor to Pulmonary Asbestosis, a disease resulting from exposure to asbestos dust. The Bureau urges the establishment of efficient exhaust systems and the introduction of other safety methods. This disease has been noted in England, and much has been written concerning it but this is the first time, to our knowledge, that it has been officially discussed in this country.” Statement refers to article from London in Bulletin of Hygiene in December on 15 cases of asbestosis among workers in an asbestos factory. “It is said that the asbestos dust causes a pulmonary fibrosis, attacking the bases of the lungs, and, like silicosis, it is frequently complicated by tuberculosis.”

1930  
Report on Merewether and Price and also advocates extending workers compensation to include asbestos.

1930  
“Pulmonary Asbestosis,” JAMA, 95 (November 8, 1930), 1431.  
Comment on 1st case in US in 1930 reported by Mills, who “pointed out that asbestos is mined and manufactured in many parts of this country and that pulmonary asbestosis surely will be encountered.”

1930  
“Pulmonary Asbestosis,” The Lancet, April 19, 1930, pp. 870-871.  
“The introduction of that extremely useful substance asbestos into industry has unfortunately given rise to a new occupational risk and to yet another from of chronic pulmonary fibrosis.” Been known since Greek and Roman times. Article’s main point is to cite Merewether and Price study above and “fact that a serious, even fatal degree of fibrosis can be produced by other dusts has not been generally appreciated.”

1930  
Stewart, M. J., “Asbestosis Bodies in the Lungs of Guinea Pigs After Three to Five Months Exposure in an Asbestos Factory,” Journal of Pathology and Bacteriology, 33 (1930), 848.  
Asbestos in lungs of guinea pigs who were left in an asbestos factory.

1930  

1931  
Draft scheme of workers comp under consideration by employers and
workers associations. A Mr. Clynes “was impressed by the urgency of the
question, and hoped to reach a final settlement by the end of next month.”

1931 Collis, Edgar, “Recent Views on Pneumoconioses,” Proceedings of the
Royal Society of Medicine, 24 (March 1931), 531-542.

P. 531 Abstract: “Up to the present, investigation of dusts from certain
silicates has shown them to be of great importance. Of these, asbestos has been
found to be most definitely injurious....”

1931 Gardner, Leroy U. And Donald E. Cummings, “Studies on Experimental
Pneumonokoniosis. VI. Inhalation of Asbestos Dust: Its Effect upon Primary
Tuberculosis Infection,” Journal of Industrial Hygiene, 13 (February 1931),
65 - 81. Conclusion – JIH, 13 (March 1931), 97 - 114.

P. 65: Saranac begins toxicological studies in January 1928. Not yet
completed “but since so much interest has recently been manifested in the subject
of asbestosis, a preliminary report is submitted at this time [1930 submitted].”

P. 103: “The physical and chemical properties of asbestos are so unlike
those of any other dust previously studies that, when inhaled, particles of this
substance provoke an unusual type of reaction in the lung.”

1931 Gloyne, S. Roodhouse, “The Presence of Asbestosis Bodies in the Faeces in a
Case of Pulmonary Asbestosis,” Tubercle, 12 (1931), 158-59.

1931 Gloyne, S. Roodhouse, “Method of Staining Asbestosis Bodies Found in the
Sputum,” Journal of Industrial Hygiene and Toxicology, 13 (1931), 85-86.

Tubercle, 12 (June 1931), 399-401.

1931 Lynch, Kenneth M. and W. Atmar Smith, “Pulmonary Asbestosis II:
Including the Report of a Pure Case,” American Review of Tuberculosis, 23
(1931), 643 - 660.

“These experiences led to the conviction that prolonged exposure to asbestos dust
presented a very definite health hazard, but until recently the matter had not been
the subject to proof.”
Had worked in the plant for 11 ½ years. “This is undoubtedly a pure case of long-standing asbestosis.”


Pp. 667-68: “Asbestosis is a pneumonoconiosis which advances to a fatal end without the supervention of any characteristic infection.”


On ways that asbestos acts as a “pathogenic agent.”


P. 585: Growth of asbestos industry; Since 1928 a considerable number of deaths in factories because of asbestos.

P. 588: As the number of years at work grows for a worker, they show fibrosis. For those working 20 years and over, 80.9% showed fibrosis.

P. 592-3: “Prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection....”

P. 609: “Pneumoconiosis is a more or less necessary risk of commercial development in the progress of civilization, but its progress and serious aspects may be lessened by certain preventive measures in most instances.”

“The importance of the condition and its differences from other forms of pneumoconiosis seem to warrant a general acceptance of the term ‘asbestos,’”


P. 756: The fibrosis is probably a result of the toxic action of the asbestos, a combined silicate, and not a mechanical effect.”

1931 Seiler, H.E. and Mary D. Gilmour, “A Case of Pulmonary Asbestosis,” The British Medical Journal, 1, No. 3677 (June 27, 1931), 1112-12.

1932 International Labour Organization, Silicosis: Records of the International Conference Held at Johannesburg, 13-27 August 1930, (Geneva: ILO by King and Son, Ltd. London, 1930). With papers and participants from a variety of researchers, including the U.S., Britain, Germany, South Africa, Australia, Japan,


P. 4: “The inhalation of asbestos dust in high concentrations leads to the appearance of asbestosis bodies in the sputum in a large percentage of the workers exposed thereto. They were present in 48 out of 50 workers examined.”

1931 Sparks, J.V., “Pulmonary Asbestosis,” Radiology, 17 (1931), 1249-1257.

P. 1253: “... the only hope for the asbestos worker lies in the adoption of the proper means of protection against the risk attendant on the inhalation of the fibers.”

P. 1256: what shows on the x ray.

1931 Stewart, Harold L. And Carl J. Bucher, “Asbestosis: Report of Two Cases,” Archives of Pathology, 12 (1931), 909-916

P. 915: 2nd case “patient worked in an asbestos factory for a short time, but without a mask....”

P. 916: In asbestosis the lung is not the only organ invaded by asbestosis bodies. As we have shown, the spleen and the lymph nodes may harbor large asbestosis bodies.”

1931 Willson, Frederick, “The Very Least an Employer Should Know About dust and Fume Diseases,” Safety Engineering, 62 (November 1931), 317-318

P. 317: “the employer who has at heart the welfare of his workmen and the vest monetary interests of his company should wish to have some information on
this subject [of dust and fumes] that he may not err through ignorance of the
danger of dust and fumes or of the liability under the law which he must always
face.”

“No employer can observe reasonable care unless he has at least a little
knowledge about the nature of dust and fume hazards.”

P. 318: “We do know, however, that breathing of dust under the following
conditions is seriously harmful.... Asbestos and every operation in which it is
used.”

1931 Wood, W. Burton and S. Roodhouse Gloyne, “Pulmonary Asbestosis
Complicated by Pulmonary Tuberculosis,” The Lancet, 2 (October 31, 1931),
954 – 956.

1932 Editorial, “German Work on Pulmonary Asbestosis,” The Lancet, 2 (July 9,
1932), 92-93.

P. 93: “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 the signs of the disease.”

1932 Farrell, Andrew J., “Silicosis,” Industrial Medicine,” 1 (October 1932), 35 -
37.
P. 37.

“Throughout this article ‘silicosis has been mentioned.... there are a number
of similar diseases: asbestosis, which results from asbestos dust.”.... It might have
been proper to entitle this article ‘The Legal Aspects of “Dust”’ because it is
intended to refer to all kinds of dust exposure. Because sickness from dust
exposure is commonly referred to as silicosis, there are many industries having a
dust hazard that are not cognizant of the fact and remain in ignorance of it until
they are greeted with a summons.”

P. 1351: “During the past two or three years observations on the asbestosis
boy have been published with such frequency that it is very difficult to keep track
of them all.”

1932 Kettle, E.H. “The Interstitial Reactions Caused by Various Dusts and Their
influence on tuberculosis Infections,” Journal of Pathology and Bacteriology,
35 (1932), 395-405.
P. 403: Silica and asbestos cause pronounced pulmonary fibrosis, out of all proportion to the amount in which they may be present in the lung....”


P. 638: Presents case of workers with asbestosis “because of the rapidly growing interest in the condition.”

P. 639: discovered “asbestos bodies in 48 out of 50 asbestos” miners.


Summary of Proceedings: “Dr. J.V. Sparks read a paper on pneumonoconiosis in asbestos workers....”

1932 “Recent Studies of Pulmonary Asbestosis in Germany,” *Monthly Labor Review*, 35 (September 1932), 541-42.

Summarizes article in *The Lancet* of July 9, 1932.


P. 11: “Asbestosis is a disease of the lungs produced as a result of the inhalation of asbestos dust usually over a period of several years.” resembles and differs from silicosis. ...

The disease occurs in workers in occupations associated with production of asbestos dust in a fine state of division; for example ...”crushing, opening of crude asbestos, blowing, mixing and carding of it; “making insulating mattresses and slaps or sections composed wholly or partly of asbestos.”

P. 12: Processes – “... “and the sawing, grinding and turning in the dry state of articles composed wholly or partly of asbestos such as motor car brake and clutch linings, electric conductors and packings and jointings.”

Need suppress the dust.

“Compensation is payable in cases of death or disablement caused by asbestosis or asbestosis accompanied by tuberculosis under a special Scheme.”


P. 180 Albert Russell, Bureau of Mines, shows x-ray of man with asbestosis. “His occupation was that of cleaning and restoring the asbestos on
pipes in one of our government hospitals.” Worked for about 6 years. Had disability and government compensation.

1932


P. 1319: Recommends that Minors under the age of 18 be excluded from a wide range of occupations including: “III. Occupations involving health hazards.... In occupations involving exposure to free silica dust, asbestos dust, or other dusts in injurious quantities.”

1932


Case 1 – worked in an asbestos factory from 1915-1928 (except for 1923-25); Case 2 worked in mattress making in asbestos factory for 27 years.

1932

“Silicosis and Other Dust Diseases,” Industrial Medicine, 1 (November 1932), 94 - 95

p. 95: cites talk by Sappington (below): “attention was drawn to the fact that aside from silicosis and asbestosis, exact knowledge is not possessed on other dust diseases of the lungs.”

1932

“Review of Present Status,” Industrial Medicine, 1 (November 1932), 108.

Presents summary of paper by Alice Hamilton on activities of the Industrial Hygiene Section of the APHA: “Problems presented by industrial dusts are probably the most absorbing and important, and these particularly revolve around silicosis and asbestosis.”

1932

Sappington, C.O., “Silicosis and Other Dust Diseases,” Industrial Medicine, 1 (December 1932), 158-68.

P. 167: “Asbestosis” “Asbestosis occurs with considerably less frequency than silicosis. Although enough clinical and pathological material is now available for specific study. The disease may be defined as a specific form of pneumonoconiosis due to breathing air containing asbestos dust, based on concentration and length of exposure; it is characterized anatomically by a generalized pulmonary fibrosis – clinically by shortness of breath and decreased lung expansion, and by x-ray finding which differ from those of silicosis.”
“Lynch and Smith survey all available literature on the subject in 1931 and collected 172 cases of pulmonary asbestosis. Apparently the first American case recorded was by Soper in December 1930.”

P. 168: “Asbestosis bodies are demonstrable in the sputum, in the lung tissue, and more recently, have been found in the lymph nodes and the spleen. The treatment of asbestosis apparently has been as unsuccessful as that of silicosis. It is believed that removal from exposure does not impede the progress of the disease, when once it is fully established. Prevention depends upon the same procedures as in silicosis, with the added recommendation that mechanical devices be substituted for dusty hand work and that wet methods be used instead of dry.”


P. 352: For the 1933 meeting [of the Konicide Club] (November 11th and 12th at the X-Ray Department, Hospital of the University of Pennsylvania, Philadelphia, Pa.) .... At the end of the second day, Dr. Gardner spoke briefly on ‘What is Known About Asbestosis,’ an early contribution to a subject that looms so large today in the occupational health field.”

P. 352: “Later in 1936 (November 23rd and 24th), ... at [Met Life]. Fehnel discussed a dust control program in an asbestos plant.”

P. 353: “On January 22, 1939 another meeting was held at the University of Pennsylvania .... The program was devoted to the health problems of the asbestos industry.”


P. 1275: “The manufacture of asbestos products in the last few years has assumed a somewhat greater importance in the industrial world. ... The conditions surrounding the greater proportion of the employees constitute a distinct and serious industrial hazard, and often sufficient devices for protection have not been provided.... [mills] Naturally, the longer the Service of an employee, the more certain is more or less extensive pulmonary damage.” Been recognized in last ten years. 1st reported case in 1900.

P. 1281 – 1931 England passed Asbestos Industry Regulations. “Although the number of asbestos workers [in the US] 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. ... many civil suits for damages against factory owners....”
1933  

On clinical features of asbestosis.

P. 252: “Pulmonary asbestosis had come to be recognized as a distinct clinical entity, in which, owing to the diffuse nature of the resulting fibrosis, the degree of respiratory embarrassment was usually greater than that of an equally advanced case of silicosis, and which was in fact a more severe disease than the latter.”

1933  

P. 34: “... within the last three or four years asbestos dust has also been shown to produce another specific type of reaction attended by symptoms differing in some respects from those of silicosis.

P. 35: “Asbestos dust, perhaps because of its fibrous structure is not transported very far within the lung. It tends to lodge along the walls of the finer tubes and little of it is removed by the lymphatic drainage system. The arrangement of the scar tissue in asbestosis is quite different from the nodular collections in silicosis because of the difference in localization of the foreign particles.”

1933  

P. 21: “an analysis of approximately $300,000 in losses of one insurance company doing a general workmen’s compensation and liability business, for recent industrial disease claims, showing 54 per cent for cases involving pneumoconiosis (including silicosis, asbestosis, etc.) ....”

Talks about the importance of invisible dust. Need remove the dust; exhaust systems; need eliminate or control the dust (p. 34)

1933  

1933  

P. 451: “The result of dust inhalation in pulmonary asbestosis is different from that seen in other dust diseases owing to the peculiar long needle-like shape of the asbestos particles. The fibrosis is diffuse, and the nodules characteristic of silicosis are not seen.”

20
“The variation of conditions in the asbestos industry renders it probably that as time goes on unusual forms of asbestosis will occur.”


   p. 445: “The history of the discovery of the asbestosis body itself and the study of the disease is now so well documented that further reference to it is unnecessary. Bibliographies will be found in the work of Merewether, Pancoast and Pendergrass, Gardner and Cummings, and Gloyne.”

   p. 446: “There are workers in whose industrial history asbestos exposure has been an inconspicuous, even unrecognized, event, and in whom lesions of pulmonary asbestosis, generally slight, are only detected quite by accident. In this class may also be placed workers who make a habit of [447] migrating from one industry to another, a practice commoner in America than in this country…. in another, a boiler riveter, asbestosis bodies were found in the sputum years after his short exposure.”


   “Medical research has shown us, however, that the dust that is most dangerous, through its deep inhalation into the lungs is so small that the individual particles are invisible and can only be seen by the naked eye if they are gathered in a dense cloud, which may have the appearance of a gray fog.”


   P. 621: “It is well known that the inhalation of certain industrial dusts is an important factor in causing occupational disabilities... other dusts, such as asbestos, cement, slate, etc., are stated to produce a varying degree of fibrosis.’


   P. 69: “This industrial disease of the lungs due to the inhalation of asbestos dust, although only recognized and accepted recently as an entity of serious import, has been, during the past few years, the object of much careful investigation in various countries.”

   Pp. 69-70: “In the case of asbestosis, evidence already available is amply sufficient to demonstrate conclusively the necessity of particularizing it as a
compensable industrial disease and of enacting regulations for the industry designed to control the incidence of the disease and ultimately abolishing it.”

P. 70 history of asbestos industry. 1880 – 500 short tons produced; 1900 – 35,000; 1920 – 200,000 1929 – 430,000.

Pp. 70-71: “The field of utility of asbestos products has rapidly expanded and to-day [71] is very large: constantly new uses for asbestos are being found. The mineral, the yarn, or the fabric, composes, or is incorporated in, a vast number of articles, ranging from matches to filter pads, from paints to roofing tiles, from high pressure jointing to electrodes, and from brake-linings to insulating materials in great variety. The phenomenal expansion of the motor car industry in the past twenty years has caused a corresponding increase in the demand for asbestos fabric brake linings, asbestos composition clutch rings, and latterly of moulded asbestos brake linings. Many other examples will come to mind.”

P. 74: Why recognition of asbestosis delayed: “it appears, therefore, that this focusing of energy on the all-important problem of silicosis, and the production of proof that the inhalation of free silica was responsible for the production of fibrosis of the lungs amongst workers in so many industries, tended to obscure the possibility that dusts other than those containing free silica might be injurious.” also size of industry relatively small.

P. 75: “Asbestosis – the pulmonary fibrosis of asbestos workers – is insidious in its onset, irregular in its course, and variable in its mode of termination.’

P. 152: “As noted above, the risk from asbestosis in the asbestos industry is no less grave than the most serious risks form silicosis in the silicosis producing industries.”

P. 158: “Evidence from all quarters, therefore, confirms the risk of pulmonary fibrosis attendant on the inhalation of asbestos dust. While, no doubt, the risk varies in different places and different works, depending on the local conditions and the amount of dust produced, the risk is general wherever more than minimal amounts of asbestos dust are produced in a manufacturing process.”

“... the imperative and immediate need of adequate preventive measures is apparent. The aim of these preventive measure is, of course, to make it impossible for new entrants into the industry to develop the disease, and the main agencies by which that goal can be attained are the triad which have served industrial hygiene so faithfully – localized exhaust ventilation, enclosure of dusty operations, and wet methods of manufacture. ... The practical engineering difficulties are great, but can be surmounted.”

1933 McCord, Carey P., “Silicates and Silicosis,” Industrial Medicine, 2 (July 1933), 4 - 12.

P. 4: “In general, it may be maintained that silicate dusts are harmful, and may lead to a fibrosis of lung tissues, but on comparison with silica the magnitude
of hazard is low, with asbestos in the leading position and providing an appreciable deviation from the common tendency.”

P. 5: “‘Asbestos’ – Is temolite, actinolite, chryotile, or amianthus, all of which are essentially magnesium silicates.”

Cites a number of articles on asbestos and other silicates causing disease.

1933

“Dusts and Their Effects,” Industrial Medicine, 2 (July 1933), 56.

Cites McCord article, above; “The two forms of pneumoconiosis about which we have fairly definite scientific knowledge are silicosis and asbestosis.”

1933


P. 128: “Roentgenologists and others have found that appearances differ quite materially [in asbestosis] from those of practically all phases of true silicosis, which is easy to understand when we realize the differences in physical as well as chemical characteristics of the inspired dust.”

P. 132: “Compensation laws must be passed by each state as nearly uniform in essential details as possible in order to avoid any effects leading to unjust competition.” Need medical boards.

Shows Roentgenogram of chest of a female aged 27 years and a asbestos worker for 6 ½ years. (P. 125)

1933

Smith, Clayton S. and Helen L. Witkoff, “The Silica Content of the Lungs of a Group of Tunnel Workers,” AJPH, 23 (December 1933), 1250 -1254.

1933


p. 139: Gardner presented a paper on “the Pathologic Reaction in Various Pneumoconioses.” Present knowledge allows a classification of the reaction to inhaled dust in three categories: nodular, linear perilymphatic and diffuse parenchymatous.... parenchymatousis produced by asbestos.”

1933


“In the year ending June 30, 1931, there were reported to the Connecticut board of Health 14 cases of silicosis due to dust of free silica .... and 2 cases of asbestosis, one of which was compensated.”
Stock, George A. “Pulmonary Asbestosis,” Medical Bulletin of the Veterans’ Administration, 10 (1933). 126-129.

P. 126: “Comparatively little has been written in the United States about a condition known as pulmonary asbestosis, although asbestos is mined or manufactured in many parts of this country. Yet, asbestosis, because of its dangerous and its unique pathologic features, deserves more attention than it has received.”

“Undoubtedly, asbestosis cases are admitted to hospitals in certain parts of the country with a diagnosis of Tuberculosis of the lungs and die without the real condition being recognized.” Describes a case at a veterans’ Administration facility.

P. 129: “asbestos workers are exposed to a serious pulmonary condition because of the dust generated in the manufacture of asbestos products, unless factories are equipped with modern devices for removing this dust.”


P. 25: “The only other type of dust [other than silica] which is generally recognized as a cause of severe pulmonary injury is asbestos, a silicate of magnesium.”


P. 351: “It has been the belief that the inhalation of dusts composed of silicates (combined silica), in comparison with silica (free silica in the form of quartz), was innocuous. Of recent years many investigators have become cognizant of the hazards of silicate dust exposure. Asbestosis, a fibrosis of the lungs cause by the inhalation of asbestos dust, has already been described in its clinical and roentgenographic manifestations by investigators in other countries, but to date no studies have been reported in the United States.”


Discussion in Parliament of asbestosis.

P. 277: “Pulmonary asbestosis is a form of pneumonoconiosis which constitutes a grave danger to workers in asbestos factories.”

P. 282: “Since 1927 tremendous strides have been made on the subject of pulmonary asbestosis, and a vast amount of literature has spring up.”

P. 284: “These highly characteristic golden-yellow ‘curious’ bodies, termed ‘asbestos bodies’ in 1929 by Stewart and Haddow, are found in the sputum and fibrosed lungs of asbestos workers.”

P. 293: “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.”

P. 295: “Pulmonary asbestosis, once established, is a progressive disease with a bad prognosis; its treatment can only be symptomatic.”

1934 Hatch, Theodore and C.L. Pool, “Quantification of Impinger Dust Samples by Dark-Field Microscopy,” Journal of Industrial Hygiene and Toxicology, 16 (May 1934), 177-191.

Various references to asbestosis in these proceedings.


P. 507: asbestos handled in 8 plants operated by 6 companies. “The principal products manufactured are woven and molded brake linings and clutch facing, and insulated wire for which asbestos is used as the insulation.” Need to provide dust control; need be aware not just visible dust but invisible too.


“Breathing the dust of this substance is bad for the health just as the breathing of almost any other foreign substance is deleterious.’ Workers should avoid breathing; “It is quite probably that the disease resulting from the breathing of asbestos dust will be similar to that resulting from silica. It that be so, manufacturers or handlers of asbestos products might begin here and now to prevent trouble because of the fact that, like silicosis, the disease can be prevented – there is no cure.”

P. 329: “There are only two definite dust diseases in industry which are clearly recognized at the present time, namely, silicosis and asbestosis.... A great deal more is known about silicosis than asbestosis, particularly regarding the postmortem and pathological findings.”


P. 1383: 53 cases (19 male; 34 female) of pulmonary asbestosis, “including 2 with carcinoma of the long and 1 with a few deposits of growth in the pleura.”

P. 1384 – All of the women worked in same factory. Shortest exposure was 6 months; longest 15 years; most of men worked at same factory but one was “a middle aged boiler riveter who had served his apprenticeship as a youth in a shop where asbestos was used for lagging pipes.

1934 The ILO Yearbook, 1834-1935, Volume 5, International Labour Office Geneva: 1934, p. 149: “Another disease very similar to silicosis, namely asbestosis, due to asbestos dust, has also been the subject of very close study, with a view to arriving at means of diagnosis and prophylactic measures.” (Japan was a permanent member of the ILO at least since 1919 when the League of Nations was formed.)

1934 Egbert, Dan S., “Pulmonary Asbestosis,” American Review of Tuberculosis, 31 (1935), 25-34.

P. 25: “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 the total world production of asbestos increased from 96,490 tons in 1910 to 434,938 tons in 1929.”

P. 34 – Provides summary of 28 fatal cases with “fatal termination.”


p. 24: On dangers of silica. “If the silica is combined with bases in the form of silicates, it rarely produces such active and progressive changes. The silicate of magnesium, asbestos, does cause the formation of scar tissue in experimental animals and in the human lung, but most other silicates are not generally recognized as irritating.”

p. 25: The only other type of dust which is generally recognized as a cause of severe pulmonary injury is asbestos…. This in itself is a cause of fibrosis or scarring of lung tissue.”

Includes bibliographical references from 1556 to December 31, 1933, subject index, authors index and state compensation laws. Includes many references to asbestosis. Describes itself as “a reference book designed for the permanent convenience of physicians, surgeons, lawyers, industrial executives, librarians, journalists, engineers, trade association officials, student and research workers.”


See notice and “Book Review” in *American Ceramic Society, Ceramic Abstracts*, 13 (September 1934), 246. “Industrial executives, industrial boards, state commissions, engineers, and research workers will find this book indispensable.”

See Review in *Illinois Manufacturers’ Association, Industrial Review*, 9 (October 1935), 4; review of Book no. 2 from 1934. Refers to usefulness for experts and the ordinary reader. “Between these two groups intervene ‘Doctor, Lawyer, Merchant Chief,’ not only individuals, but also corporations, commissions, insurance companies, and legislatures.... By ingenious indexes of authors and subjects anything and everything can be traced down at a moment’s notice.”

1935  Founding of the Air Hygiene (later Industrial Hygiene Foundation)


Enclosed in Robert Hitchins, “Report and Recommendation of the Temporary Organization Committee which was elected ...[at the above]. Industries represented at meeting “groups themselves into seven general classes and the Nominating Committee report consisted of the nominations for a Temporary organization Committee, with one representative from each of the following Groups of Industry: Groups of Industry I. Asbestos, Cork and Non-metallic....” nearly 200 people attended including representatives from asbestos industry.

Program, Symposium on Dust Problems, January 15, 1935, University Club, Pittsburgh, Pennsylvania. At the meeting a nominating committee nominated a “temporary organization committee. Among the 7 people nominated was Vandiver Brown of Johns-Manville.

Brown writes to M.F. Judd, Secretary Raybestos-Manhattan, Inc, Bridgeport, Ct, January 22, 1935: encloses memorandum he wrote about the meeting, shown that “only two forms of dust, namely, free silica and asbestos, are definitely known to produce disabling fibrosis of the lung.” silica more “serious” because predisposes to TB. Says on Temporary Committee, but agreed to serve on
condition could withdraw.” I pointed out that members of the Asbestos Industry did not care to be associated in the minds of the public or of employees with those industries whose problem was silicosis and that for this reason I felt there might be some opposition to having a representative of the Asbestos Industry working with them. I did indicate, however, that I realized numerous aspects of our problem were the same and that if cooperation could be worked out without an undue amount of publicity, Johns-Manville Corporation and perhaps the Asbestos Industry as a whole, would in all likelihood be willing to cooperate.”

1935  

P. 5: “although suggestions have been made from time to time ... that carcinoma of the lung is liable to supervene upon pneumonokoniosis of long standing, the precise interrelation of the two diseases is still an open question. Cases – 2 women

P. 9 – The carcinoma was in a portion of the lung in which the asbestosis was fairly advanced.

1935  

1935  

p. 565:”there is a sinister ghost that stalks every industry – a ghost whose name itself is forbidding. It is called ‘pneumoconiosis,’ and it is quite as dangerous as it sounds;” describes asbestosis. Fibrosis as serious as silicosis, but “not so nearly frequent in occurrence.”

1935  

P. 1 – 1929 Met life “approached by officials representing the asbestos industry int eh 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.”

P. 2 – investigation from October 1929 to January 1931 – study of dust conditions in asbestos mines and mills in Canada and in fabricating plants along Atlantic Seaboard in US. Also did physical examinations of asbestos workers, including x-rays
P. 10: “Prolonged exposure to asbestos dust caused a pulmonary fibrosis of a type different from silicosis and demonstrable on X-ray films. Clinically, from this study, it appears to be of a type milder than silicosis.”

P. 11: “It is recommended – 1. That the industry seriously face the problem of dust control in asbestos plants. 2. That new employees be examined physically, including X-ray examination of the chest, and rejected for employment if they show tuberculosis or pneumoconiosis .... 4. That the industry sponsor studies on known cases of asbestosis, as well as studies on effects of asbestosis on the heart and circulation.”

1935

P. 241: “Exposure to asbestos dust, chemically an hydrated silicate of magnesium, in time results in a somewhat similar disease [to silicosis] known as asbestosis.”

1935

P. 257:”The fibrosis-producing dusts are of particular interest at the present time because of the multitude of personal injury suits and claims in every section of the country growing out of industrial hazards and alleged diseases .... Terms such as silicosis and asbestosis are descriptive of diseases caused by the inhalation of silica and asbestos dusts, respectively.”

1935

P. 90: “the preponderance of medical opinion is that, of the inorganic dusts constantly generated in industry, only dust of free silica ... and perhaps asbestos dust – and those dusts only when in minute particles – are harmful, that is, harmful in the sense and to the extent of causing specific disabling diseases of the lungs.”

1935

Mentions asbestos as one of dusts that needs to be controlled

1935
In the manufacture of asbestos goods, particularly dusty atmospheres are created in the break-out, ... and picking operations.” Also crushing and grinding, spinning and weaving;


P. 56: “As worthy of record in the interest of both diseases, as well as their possible relationship, the following case of pulmonary asbestosis with associated carcinoma of the lung is reported.”

Weaver in an asbestos factory – asbestos mill weaver.

P. 64: “the pulmonary carcinoma appeared to originate from one of the branches of the bronchus to the right lower lobe, where squamous metaplasia of lining epithelium was observed. The duration of the carcinoma cannot be specified, it was apparently of considerable time but certainly did not antedate fibrosis of the lung. A conception of its origin by reason of chronic bronchial irritation is compatible with the current view of the etiology of such tumors.”


P. 44: “So much has been written on pulmonary asbestosis during the few years which have lapsed since its first recognition (Cooke, 1924, 1927; Stuart McDonald, 1927; Merewether, 1930; Lynch and Smith, 1930; Kruger et al., 1931; Ellman, 1933) that only a brief introductory Statement is necessary here.... The length of exposure to dust has varied within wide limits, but it is clear that gross disease may follow even as short an exposure as 18 months, provided the dust concentration has been sufficiently high.”


Part 1, p. 2: “An effort has been made to group under the heading ‘inert’ a certain number of dusts; the number, however, is becoming more and more reduced since certain dusts such as talc and asbestos, until recently considered inert nevertheless have caused serious organic lesions among certain classes of workers.”

Part 1, p. 38: “Collis defines asbestosis as a pneumoconiosis that advances to a fatal end without the subvention of any characteristic infection. It is a simple dust condition, just as is simple silicosis; but it is more distressing in life and more
rapid in this progress than silicosis. It contrasts even more strongly with pulmonary mycosis, which is due to a living infection upon otherwise healthy lungs.”

1935

“Memorandum on the Industrial Diseases of Silicosis and Asbestosis,”
London, His Majesty’s Stationary Office, 1935.

P. 12: “Asbestosis is a disease of the lungs produced as a result of the inhalation of asbestos dust usually over a period of several years.” resembles and differs from silicosis. ...”

“It occurs in workers in occupations associated with production of asbestos dust in a fine state of division;

In range of industries “and the sawing, grinding and turning in the dry state of articles composed wholly or partly of asbestos such as motor car brake and clutch linings, jointings, electric insulating materials and some types of electrodes.” need suppress dust.

1935


Asbestos industry in PA “consists mainly of several fabricating plants engaged in the making of asbestos cloth, brake lining, insulating tape, asbestos rope and wick, and other miscellaneous products.”

3 types of asbestos – chrysotile, crocidolite, and amosite, the first is the most predominant used.

Used in textiles, weaving, gasket making; also used in range of products – insulation in steam pipes, asbestos cements, shingles, lumber, molded fittings, and household appliances.”

Study effect of exposure to dust in asbestos fabricating plant – found that 14 workers, 25% of exposed group were found with asbestosis. – had both clinical and x-ray evidence of disease

1935


P. 17: “a second group of dusts produces somewhat the same clinical picture as silicosis in its early stages, yet the x-ray of the chest shows the shadows pretty much confined to the lymph nodes. The damage appears not to be so serious as silicosis in many ways,.... Asbestosis falls in this group ....”

1935

“There is a sinister ghost that stalks every industry – a ghost whose name itself is forbidding. It is called ‘pneumoconiosis’ and it is quite as dangerous as it sounds.”

“Asbestosis is caused, as the name implies, by the inhalation of microscopic particles of asbestos dust. This type of pneumoconiosis produces a definite and general fibrosis of the lungs.” Issue includes an ad for “The Pneumonokonioses (Silicosis): Bibliogrpahy and Laws” covers world literature from 1556 through 1933 – “Its purpose is to put between two covers all references to the world’s literature on the Pneumonokonioses, that that anyone interested in the subject can quickly find out: who said what – and when – and where.”

1936 “Occupational Diseases, Health, Comfort and Safety and Changes in Blower Act,” Industrial Review 9 (February 1936) 3: (Published by the Illinois Manufacturers Association)

p. 2: Describes Workmen’s Occupational Diseases Act: This is a Bill covering the subject of liability of employers for injuries or deaths resulting from diseases.”

p. 3: “the Bill further provides that no compensation shall be payable for occupational diseases unless disablement occurs within one year after the last day of the last exposure to the hazards of the disease, except in cases of occupational diseases caused by the inhalation of silica dust or asbestos dust, and in such cases within three years after the last day of the last exposure.”

“Illinois Occupational Disease Law: Summaries of the 1936 Enactments,” Industrial Medicine, June 1936

Same as above.


“Although the actions of various dusts differ within the body, the exposure to asbestos fibres, present in the weaving and grinding of dry asbestos material, offers another type of dust which may cause fatalities among workers.”

1936 Dr. J. Newton Shirley, “Do We Have Occupational Disease Hazards in the Rubber Industry,” Transactions of the 25th National Safety Congress, Atlantic City, NJ October 5 - 9, 1936

“In a comparatively small field we find asbestos used in the manufacturing of heat resisting products, such as gaskets. Asbestos is a complex silicate which is capable of producing asbestosis, a form of pneumonoconiosis. Workmen who are required to handle this material during the compounding stages, when it is dusty, should be protected with respirators.”
1936  **Hunt, Robert B.,** “Lesser Known Facts about Occupational Diseases,”  

P. 35: “Although the actions of various dusts differ within the body, the exposure to asbestos fibres, present in the weaving and grinding of dry asbestos material, offers another type of dust which may cause fatalities among workers.”

1936  **Lanza, A.J.,** “Asbestosis,”  *JAMA*, 106 (February 1, 1936), 368-69.

P. 368: “While Hoffman called attention to the possible harmfulness of asbestos dust in 1918, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen’s compensation in Massachusetts.” Describes cases and Reports in Britain.

In 1927 a fatal case of uncomplicated asbestosis was reported to the Medical Society of South Carolina, and since then, including this case, there have been eleven fatal cases reported in the United States.... These Reports, together with the fact that asbestosis figured int eh extraordinary occupational disease litigation that has spread over this country, resulted in both laboratory and field studies of this new hazard.”

“A search of all the death records on file in the Metropolitan Life Insurance Company revealed that asbestosis had been given as a cause or contributing cause of death in only nineteen cases. The first case noted was in 1924, there were two in 1927, one occurred in 1931, and the rest have occurred since 1933. The diagnosis was supported by autopsy in only six. In one of the six the primary cause of death was carcinoma, in another glioma of the brain ....”

“Recently Miller described the effects of the injection of three varieties of asbestos; namely, chrysotile, crocidolite and amosite. Chrysotile is Canadian asbestos, and as previously stated, is largely magnesium silicate. Crocidolite and amosite contain only a small quantity of magnesium, containing instead iron silicate in approximately the same quantity. The three types of asbestos produced the same type of reaction.”

1936  **Donnelly, J.** “Pulmonary Asbestosis: Incidence and Prognosis,”  *Journal of Industrial Hygiene and Toxicology*, April 1936, 222-228.

P. 222: “Within a comparatively short time the occupational disease, pulmonary asbestosis, has become a matter of considerable importance, not only to manufacturers of asbestos products, but also to the workmen engaged in the industry.... 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.”

“...workers must be protected against the hazard as effectively as possible.”

Current protections not adequate. Cases from an asbestos plant.


1936 “Relationship of Asbestosis and Silicosis to Disability,” Industrial Medicine, August-September 1936, 456-458.

p. 456: “It has been suggested that the excessive absorption of silica or asbestos over a long period of time may exert some toxic influence upon the various body tissues, but to date sufficient data are not available regarding such action to measure the specific effect produced.”


Section 11: “provision for autopsy, by competent pathologist, where death may have been due to silica or asbestos dust.”

Section 23: “Employees with silicosis or asbestosis but not disabled prior to October 1, 1936, may file with industrial commission within 60 days after effective date of this act, a request to waive full compensation for disability or death from silicosis or asbestosis, or any direct result thereof, supported by satisfactory medical evidence.” If the industrial commission approves such waiver the compensation payable after further exposure and resulting death or disability shall be 50% of what it otherwise would have been.”

Section 25; if claiming silicosis or asbestosis must have exposure for 60 days or more.

1936 General Manager, National Council on Compensation Insurance to R. Campbell Starr, Asst. Safety Engineer, U.S. Dept of Labor, October 14, 1936, National Archives.

encloses classifications of their Occupational Disease Committee felt had a sufficient dust hazard to warrant a special premium charge. Includes Asbestos Good Mfg. As producing asbestosis – for “relative Expectancy of Occupational disease – from 1 to 10 gets a 10.; Also Building and Roofing Paper Mfg.

1936 McPheeters, S.B., “A Survey of a Group of Employees Exposed to Asbestos Dust,” Journal of Industrial Hygiene and Toxicology, 18 (April 1936), 229 - 239.
P. 229: “The dangerous dust diseases, silicosis and asbestosis, are being widely studied at present... 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 this 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.”

Survey of 210 persons exposed to asbestos dust.

P. 238: individual response important; “The evidence presented in this study warrants the opinion that with the reduction of dust concentration now feasible by available dust eliminating devices, the incidence of significant asbestosis would in time approach the vanishing point.”

1936 Middletown, E.L. “Industrial Pulmonary Disease Due to the Inhalation of Dust: With Special Reference to Silicosis,” The Lancet, 2 (July 11, 1936), 59-64.

P. 59: “From the point of view of health by far the most important of the silicates is asbestos.”

“During the last ten years an extensive literature has been produced on this subject, and it will suffice for the present to mention briefly some of the features in which this disease differs from silicosis.”

P. 64: “When inhaled dust consists of silica combined with bases, as silicates, some degree of change in the pulmonary tissue appears to result. In this respect asbestos dust is unique amongst silicates int eh prevalence and severity of the disease which it causes. The physical form of asbestos differs from that of all other industrial dusts. The fibrosis produced in the lungs by the action of silicates differs from that produced by free silica, and the types can be distinguished by radiological and histological means.”


P. 46: case of person who was “employed in a chemical works handling asbestos.”


P. 143; “the following report was deemed especially interesting since it is apparently the first to describe primary carcinoma of the lung as an associated condition.”
Hungarian immigrant was a weaver in an asbestos factory. P. 149: 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.”

1936 “Relationship of Asbestosis and Silicosis to Disability,” Industrial Medicine, August-September, 1936, 456 – 458.

P. 456 – defines asbestosis. “Attention is called to the fact that the definitions stated do not specify that a diagnosis necessarily means disability. Both are slowly developing conditions and clinical and roentgenological evidence of pulmonary fibrosis may in some cases exist for several years prior to the demonstration of any decreased capacity for work.”


“A year ago the writer published a short note on the occurrence of squamous carcinoma in two cases of pulmonary asbestosis. The following case in which the asbestosis was accompanied by an oat cell carcinoma of the lung appears to be worth recording in view of the pathological differences between the two types of cases”

Packer in an asbestos works; had extensive asbestosis.


P. 1678: “The dusts investigated produced distinct types of reaction, which made it possible to classify them into the following three groups: 1) A group in which the dust was absorbed or disappeared without visible gross damage; 2) a group in which the dust initiated cellular proliferation followed by fibrosis and retrograde changes; 30 a group in which the dust remained inert in the tissues, neither being absorbed nor causing gross proliferation.”

P. 1688: quartz produced “a proliferative reaction”; asbestos was “inert in reaction”


P. 36: “Clinical experience has shown that the inhalation of a sufficient amount of asbestos dust causes a serious type of fibrosis....”

P. 279: “Probably the first case in English medical literature to be definitely proved as asbestosis, was reported by Cooke in 1924 ,,, Little attention has been paid to this form of pneumonoconiosis in the American literature until 1928, which the ‘Journal of the American Medical Association’ commented editorially on Cooke’s report and suggested that this condition deserved more consideration than it has been given. The first case reported in America was by Mills, in 1930. Since then many individual case Reports, or small groups of cases, have been brought to our attention; but no large series from which a comprehensive study can be made has appeared.”


Cites Leroy Gardner for view “At the present time two kinds of dust are generally recognized as possessing the most irritating properties. ....asbestos, a silicate magnesium, is as bad if not worse [than silica]. Can take” from 7 to 11 years in asbestos to produce asbestosis.” pushing instrument for determining dust concentrations.


A book of “International Abstracts, Extracts and Reviews of the Pneumonokoioses and their Associated Diseases and Subjects for the year 1935-1936 in the United States and internationally. Over 1000 pages, including an index that lists numerous references to asbestosis.


P. 59: “‘For the past several years industry generally has become ‘dust-conscious’ and many lines of business have had to concern themselves with the legal responsibility created by the effect of dust upon the health of employees exposed to this hazard. We have heard and used many terms that are relatively new to most of us, such as ‘silicosis,’ ‘asbestosis’ ....”


On Illinois new Occupational Disease Act. Before the Act: “Throughout the state the pattern of procedure was almost unvarying. Employees were given X-
ray examinations of the chest, and if the results showed even a trace of silicosis or asbestosis they were discharged.”

1937 Auerbach, Oscar, “Pathology of the Pneumoconioses,” Industrial Medicine, 6 (January, 1937), 38 – 48.

P. 46: length of exposure shorter for asbestosis than silicosis; “Lanza has found that in the United States the course of asbestosis is a much milder one than that reported by the English ....”


p.27: “It is generally agreed that the inhalation of silica dust or asbestos dust over a long period of time will produce a fibrotic condition of the lungs known respectively as silicosis or asbestosis. Silicosis and asbestosis are not new diseases.... These diseases however have been recognized in this country only in recent years as presenting a major social and industrial problem.” Concerned about liability and need for workers compensation legislation.

p.29: “Compensation for occupational diseases of all sorts and particularly silicosis and asbestosis present a variety of problems which are not involved in providing compensation for accidental injuries.”

1937 “Dust Diseases and Workmen’s Compensation Legislation,” Industrial Medicine, 6 (February 1937), 90 - 96.

P. 90: “Pneumonoconiosis, or dust disease, one of the most extensive of the graver occupational diseases, endangers approximately 500,000 workers in the United States and annually leaves in its wake a train of several thousand disabled victims in addition to several hundred thousand deaths.... Despite this growing notoriety, insufficient effort has been expended to prevent the disease, and clear provision is today made for its victims in the workmen’s compensation acts of only 12 states.”


p. 121: “Asbestos dust and the reaction that it provokes is for the present best considered as a special case. Its fibrous particles do not seem to be readily handled by the protective mechanisms of the lung. They are not removed to the lymphoid tissue but remain in contact with the delicate walls of the air spaces. They become surrounded by an iron-containing coating that fractures and gives rise to the peculiar structures known as asbestosis bodies. The fibers are irritating,
perhaps because of their form and they excite a fibrosis which begins about the terminal bronchioles and spreads to form diffuse patches in the parenchyma. Often the distribution is subpleural.

1937

“P. 1713: One of its important industrial uses is in the manufacture of fire-resistant textiles.
P. 1714: 300 people employed in plant – focus on preparation, carding and spinning.
P. 1727: not have enough data for threshold info. “Nevertheless any appreciable decrease in the amount of asbestos dust will cause a decrease in the incidence and severity of the resulting asbestosis.”

1937

1938
P. 49: “Some [industrial dusts], such as asbestos dust, are known to produce permanent injury, but the limits of tolerance have not been established. Where such dusts are known to be present, they should either be so exhausted as not to contaminate the atmosphere, or the worker should be provided with a suitable respirator or mask.”

1938
P. 187 – cites several cases in the literature of cancer and asbestos. Includes Gloyne, 1935; Lynch and Smith, 1935.

1938

1938
p. 59: cites Lanza, McConnell and Fehnel; Cressen, et al; Page and Bloomfield

p. 60: “As with silicosis, it was found that the incidence of asbestosis increased rapidly with increased dust exposure.”

P. 419: “Recent years have seen an immense increase in the use of asbestos, both for brake linings of automobiles and for heat resisting and insulting materials. With relation to public health, asbestosis is not nearly as serious a problem as silicosis. The number of persons in the industries using asbestos is very much less than for silica and in the United States amounts to about 12,000 factory workers, of whom approximately 25 per cent are women.”


P. 55: “Postmortem examination of 3739 individuals exposed to dust, as compiled from the literature and from our own series, revealed 30 individuals with pulmonary carcinoma, or an incidence of 0.8 per cent. This incidence is lower than that reported in routine autopsy examinations of the general populations. Experimentally, of 3338 animals exposed to many different kinds of dust for long periods of time, only 2 guinea pigs revealed the presence of a pulmonary neoplasm. The tumors in both cases were similar and were interpreted as benign adenomas. All other [p. 56] animals failed to show evident irritation, hyperplasia or tumor transformation of the epithelium lining the respiratory passages. This was observed irrespective of the activity of the dust, whether it was inert or had caused marked fibrosis of the pulmonary connective tissue. Inhaled dusts, therefore, except those containing recognized carcinogenic substances such as radium and tar, cannot in general be considered as etiological factors in the development of primary pulmonary carcinoma.”

1938 “Pneumoconiosis and Pulmonary Carcinoma,” JAMA, 110 (June 18, 1938), 2068.

Cites Vorwald and Karr of Trudeau Foundation study and their conclusions.


P. ix: “Asbestosis, a lung disease caused by long-continued inhalation of asbestos dust, was the principal physical defect found on medical examination of 541 men and women employed (or recently employed) in three asbestos textile factories.” Recommends limit of 5 mppcf. Effect – “initiation of fine, interstitial, pulmonary fibrosis”

Symptoms – dyspnea, variable cough which sometimes raises blood
streaked sputum and loss of weight.

1938  

P. 49 – Lists asbestos dust as “known to produce permanent injury”

1938  

P. 2 – “All manipulation of asbestos fibre, by hand ... or mechanically, produces dust which, if uncontrolled, is often in dangerous concentration. The inhalation of this dust over a period of time results in the development of a fibrosis of the lung.... The disease produced, asbestosis, is therefore in the same category as silicosis ....”

P. 9: “In addition to these four main complications [bronchitis, pneumonia, TB, emphysema] may be noted two which are less common ... Carcinoma. The number of cases of the latter so far described is small and it is not yet established that there is any aetiological connection between the two diseases.” for a cancer to be classified as of industrial origin needs: 1) incidence rate that exceeds that of general population; and “should be sufficient association of a worker with a substance proved experimentally to have carcinogenic properties.

P. 10: “These two postulates cannot yet be regarded as having been fulfilled in the case of the asbestos industry, but there is sufficient evidence to warrant careful observation in the future.”

“Exposure to asbestos dust for less than five years can result in the development of a degree of asbestosis sufficient to cause death.”

P. 14: “A practical maxim of the greatest value is that every translation of fiberised asbestos in the factory produces dust which, if not controlled, is dangerous.”

Great Britain passed compensation for asbestosis for all workmen employed after May 1931; Germany December 1936; North Carolina passed law in 1935.

1938  

P. 82: “According to Drinker, there are four different types of reaction produced in man by the inhalation of dust. The first and most important are the pneumoconioses, such as silicosis and asbestosis, which cause specific lung pathology and often are followed by pulmonary tuberculosis.”

P. 53: “When the inhaled dusts consists of silica combined with bases, silicates, some degree of change in the pulmonary tissue may result. In this respect asbestos dust seems to be unique among silicates in the prevalence and severity of
the disease it causes.”

1938  

P. 371: Pulmonary Asbestosis differs from the other pneumoconiose under discussion by reason of the nature of the particles inhaled, and by reason of their different chemical composition. Further, the shape of the particles causes them to be arrested more often in the bronchioles than in the alveoli, and their size and shape render them incapable of being readily transported into the lymphatic system.”

P. 373: The symptoms of the disease are very much more pronounced than the physical signs, or the radiological appearance, ....”

Prevention

P. 377: Complications: “Carcinoma has been found in 6 cases, by Gloyne, at autopsy.”

(From Report in Journal of Industrial Hygiene; “chief complications ... (4) Carcinoma.”

1938  
Abstract (by L. Teleky) of article by Nordmann, “Abstract of the Literature of Industrial Hygiene,” Supplementary to Journal of Industrial Hygiene and Toxicology, 20 (1938), 184.

Abstract, “Occupational Cancer in Asbestos Workers,” M. Nordmann, Ztschr. F. Krebsforsch, 47 (1938), 288-302: reports on 2 cases of lung cancer. “The author mentions 4 additional cases of cancer and asbestosis in the literature and one must agree with him that, in view of the small number observed and the very few cases of asbestosis autopsied (12 cases in German) the number of cancers found is a very high one. ... After all, the author seems quite right in seeing a causal relation between asbestosis and lung cancer.”

1938  

P. 49: “Some [industrial dusts] such as asbestos dust, are known to produce permanent injury, but limits of tolerance have not been established. Where such dusts are known to be present, they should either be so exhausted as not to contaminate the atmosphere, or the worker should be provided with a suitable respirator or mask.”

1938  
P. 6: “Mechanical-filter Dust, Fume, and Mist Respirators, Approved Under Following types approved by Bureau of Mines: Schedule 21; Type A Mechanical Filter Respirators: For Protection Against Pneumoconiosis-Producing or Nuisance Dusts, as Quartz, Asbestos, ....”


“Physiological Effects of Dust,” “a. Pneumoconioses (silicosis and asbestosis) which result in specific lung pathology.”

“Asbestos is the only silicate at present recognized as causing pathology, and even its reaction is definitely different from that due to silica alone (Drinker). .... Asbestos, when inhaled, produces fibrous tissues in the lungs of both men and animals.”

Threshold limits – asbestos – five mppcf


P. 24: Hazards of Asbestosis. Asbestosis is an Industrial disease of the lungs incident to the inhalation of asbestos dust for prolonged periods and is distinct from silicosis..... The workers in the Pipe Covering and Insulation Shop are exposed to the inhalation of asbestos dust incident to the cutting of asbestos insulating felt in the fabrication of covers for flanges, valve bonnets, and high temperature steam turbines...” Did medical survey of 11 employees in this shop. 6 had more than 10 years exposure all the men denied disability due to asbestosis and x-rays “essentially negative.”. However it was not considered that the negative findings precluded the future development of asbestosis by continued exposure to present occupational conditions.” recommends for reducing dust.


P. 40: cites Lynch and Smith, Egbert and Geiger, Gloyne;

“Middletown in a review pertaining largely to silicosis refers to 3 cases of lung cancer in 54 cases of asbestosis.... It is difficult to evaluate these findings and to determine their actual significance. Though the coexistence of the conditions has been observed with amazing infrequency by some, others feel that this may be due to the lack of suitable material. In the main the tendency is to support the
contention that silicosis or other forms of pneumoconiosis may play a part in the production of primary carcinoma of the lung.

1939


P. 196: “in 1928 we began the intensive study of those exposed to the asbestos hazard in one of the large industrial plants in Massachusetts, where brake lining was manufactured.”

P. 199: “We are of the opinion that in x-ray examination we have the best and perhaps the only method of diagnosis of asbestosis at the present time.”

Pp. 199-200: In the first place, there is an increased density in the lung-fields,. This increased density is characterized first by bilateral distribution, and secondly, by its being limited to the lower lobes of both lungs.”

P. 200 – “in the second place, asbestos dust produces changes int eh pleura, the most constant finding being an accentuation of one or more inter lobar pleural septa.”

“In the third place the x-ray shows the usual signs of emphysema.’

“In the fourth place, we have noted in the cases of asbestosis certain changes in the contour of the heart shadow which we feel is somewhat causally related to the process in the lungs.”

1939

V.A. Zimmer to “The Secretary [Perkins], May 23, 1939, National Archives.

“Re: Occupational disease bill now before Governor O’Conor of Maryland.” “The bill provides that a worker must, in order to have a valid claim for silicosis or asbestosis, give written notice to his employer within ten days after the first ‘manifestation’ and not first diagnosis of the disease.” Uses as an example of the meager benefits of the bill.

1939


P. 567: “following out report in 1935, some further interest in the occurrence of lung cancer in asbestosis has been manifested. Six cases have been recorded, by Gloyne, Egbert and Geiger, and Nordmann. The present report brings the total to 8. Nordmann believes that these cases should be classified as occupational cancers.”

Case of worker in an asbestos mill.

P. 573: Including all necropsies and all lung cancer cases, the incidence of primary carcinoma in our necropsy service over this period is 0.3 per cent.
Omitting the 35 cases showing asbestos deposits and the 2 cases of lung cancer in asbestosis, the incidence rate is 0.21 percent. Among the asbestosis cases (35) the incidence of lung carcinoma (2 cases) is approximately 6 per cent. Whether this is to be taken as of significance, especially in comparison to the general rate, is questionable. The series of asbestosis cases is small and the possible statistical error great. It has seemed desirable, in addition, to record the observation that advanced asbestosis may lead to bronchial epithelial metaplasia of a type encountered in other locations where cylindrical epithelium may give rise to squamous-cell carcinoma."


On asbestos textile industry – study by Public Health Service.

P. 213: findings – “In a study of 541 employees of North Carolina textile mills, pulmonary asbestosis was the principal physical defect found.” dust particulate and fibrous

Pp. 213-4 – “Definite clinical and roentgenographic evidence of asbestosis is observed in exposed persons after 5 to 10 years of work in exposures exceeding 5 mppcf.”

P. 214: Methods for controlling the dust below this tentative threshold are already available for most of the processes in the industry.”

1939  **Roche, John M. “Settling the Dust Problem,” National Safety News, May 1939, pp. 18 -19; 72 -74.**

case of asbestos particle size and concentration important

1939  **Zimmer, V.A. to Harold D. Smith, Director, Bureau of the Budget, June 8, 1939, National Archives.**

Writes about concerns of employers and insurance carriers about “the assumption of liability for this disease [silicosis] under a newly adopted State act [that] develops an immediate or imminent liability for a condition acquired over a period of years. Hence it is that under several of the recently adopted State workmen’s compensation acts full compensation is given for all other occupational diseases with the exception of silicosis and asbestosis, while in respect of these the worker is permitted to wave wholly or in part his benefits, or the benefits themselves have been drastically reduced, or both.”

1939  **Laws of Pennsylvania, “Session of 1939,” No. 284, An Act Defining the liability of an employer to pay damages for occupational disease contracted by an employee arising out of and in the course of employment.**
Specifically mentions asbestosis

1939  Laws of Maryland” Chapter 465. House Bill 484.
      P. 991: “Occupational Diseases. 32A. Every employee who, in the regular course of his employment, is injuriously subjected to an exposure to any of the occupational diseases hereinafter named, in an occupation or process hereinafter set down opposite the name of such disease, shall be deemed to be engaged in an extra-hazardous employment within the provisions of Section 32 of this Article.”

      P. 995: “34. Asbestosis. Any process or occupation involving an exposure to or direct contact with asbestos dust.”


          p.1: “How to examine the thousands of workers scattered all over New York State who were exposed to silica, asbestos and other dusts? And to do it cheaply – without interrupting the flow of work more than necessary? Obviously these workers could not be brought to a central clinic. So Dr. Leonard Greenberg, director of the Division, set about designing a clinic on wheels. He developed a truck with a special body and chassis and equipped it for conducting x-ray and medical examinations of workers.”


          P. 12: “Among the newer disease, which are a byproduct of our industrial age, asbestosis has come to occupy a fairly prominent position. .... Only within the last decade has the subject of asbestosis really received the attention of the medical world.”

          P. 13: Report based on study of 180 patients [p. 15] “employed for three years or more in a factory manufacturing asbestos brake lining for automobiles and worked in the carding, spinning or weaving rooms.”

          P. 20: “Conclusions. 1. Asbestosis, like silicosis, constitutes an occupational hazard, arising from exposure to asbestos dust. 2. Pathological findings are those of pulmonary fibrosis and thickened pleura, most marked at both bases. 3. Dyspnea is an early symptom and is the chief cause of early disability.”

P. 535; “The two clinically important forms of pneumoconiosis that are known as silicosis and asbestosis are respectively due to inhaled free silica and asbestos dusts.”

1940 Gardner, Leroy U. “Recent Developments in Relation to Silicosis,” Industrial Medicine, 9 (February 1940), 46 - 48.

Not suggest a chemical injury
P. 47L “For the last year or two we have been coming to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are still fibres which mechanically irritate the lungs.”

1940 Koontz, E. Ransom, “Compensation for Industrial Injuries and Occupational Diseases,” JAMA, 114 (February 17, 1940), 563 - 569.

P. 564: Lists states which have compensation for Occupational Diseases – 25 – and lists those where silicosis is “specifically covered” – 13. Footnote says “Many of the laws which are designated as specifically providing compensation for silicosis also provide compensation for other occupational diseases, such as asbestosis and pneumoconiosis.

1940 Macklin, Madge Thurlow and Charles C. Macklin, “Does Chronic Irritation Cause Primary Carcinoma of the Human Lung,” Archives of Pathology, 30 (1940), 924 - 955.

Article deals with the difficulty of establishing any specific irritant as a cause of cancer in any individual case.
P. 942: “Few cases of asbestosis associated with cancer of the lung have been reported.... Here again we should expect to find the two conditions associated in the same person in some cases, on chance alone.” Criticizes Lynch and Smith for their small sample.

1940 Sappington, C.O., review of Lanza’s Silicosis and Asbestosis, Industrial Medicine, (April 1940), 210.

“Considered first American textbook on the subject.” “very great assistance ... to those who must know and appreciate the difficulties which have heretofore been experienced with these diseases in their many complicated inter-relationships.”

1941 Hannesson, Hannes, “A Case of Pulmonary Asbestosis Accompanied by
1942


P. 763: Focus on chrysotile asbestos – Canadian – but all the asbestos minerals “are silicates consisting of combinations of various bases with hypothetical silicic acids. All are characterized by a fibrous structure due to peculiarities of molecular arrangement.” 5 different types

P. 766: “In the course of experiments upon the action of asbestos fibres upon living tissue it has been observed that this irritant is not like finely divided quartz, which provokes specific cellular responses in any organ of all species of animals investigated. Asbestos fibres are acted upon to produce the ‘bodies,’ described by Cooke, only in the lungs of a few species like man, guinea pigs and white mice.”

1942


P. 123: “Following the report of a case of carcinoma of the lung in the presence of pulmonary asbestosis by Lynch and Smith in 1935, several similar papers appeared in rapid succession.” Gloyne, 1935; Egbert and Geiger, 1936; Nordmann, 1938; Lynch and Smith. “Two cases here reported bring the total number recorded in the literature to ten.”

Case 1 – employed “by various manufacturers of asbestos products.”

P. 129: “… it is known that inhaled asbestos fibers undergo dissolution and phagocytosis, and may eventually disappear entirely, leaving behind irreparable damage. Both of these individuals were employed for unusually long periods in occupations in which the exposure to asbestos dust was relatively low.”

1942


P. 403: “Since 1935 an appreciable number of cases of asbestosis associated with carcinoma of the lung have been reported from England, Germany and the United States.”

P. 404: “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. 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 in the direction of the predominance of cancroids, and in the multicentric development of two and possible even three of these pulmonary neoplasms.

1942  

P. 194: “The present toxic limit for asbestos is five million particles of dust per cubic foot of air. This is a very small concentration, so small in fact that the condition may look good even to a critical eye and still present an exposure greater than this low limit.”

“In the case of asbestos dust, however and this holds with even more certainty for dusts high in free silica content, the toxic limit is so low that the only safe procedure is to have recourse to actual dust determinations. This is especially important where the injurious condition is not immediately evident but requires years to develop as in the case of asbestosis and silicosis.

1942  
**Francis R. Holden, “What the Foundation Plant Surveys are Disclosing,”** *IHF, Seventh Annual Meeting of Members, Pittsburgh, PA, November 10-11, 1942*

“Every new chemical or product should be investigated as to its toxicity before it is prepared in large amounts and released to the public. This practical common-sense procedure is followed by several larger producers of synthetic chemicals. At least two of the companies are members of the Foundation and can furnish details of their experience to other interested members.”

1943  

P. 14: “It is my purpose to direct attention to the amazing number and variety of proven chemical carcinogenic agents ....”

P. 16 Cites voluminous literature “and other miscellaneous substances, proven or suspected including ... asbestos....”

1943  

P. 64: “Occupational cancers are elicited by a great variety of chemical and physical agents such as arsenic, chromates, nickel carbonyl, radium, mesothorium,
asbestos, crude mineral oil, pitch, tar, soot, ...” and many more.

P. 66: “Asbestosis cancer of the lung is the most recent newcomer among the 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 five 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.”

1943


- Adds 3 more cases to bring total to 19.
- P. 804-5: “A review of the literature on the association of pulmonary asbestosis and carcinoma revealed that there are at least 19 known cases (including the 3 herein reported) of asbestosis associated with primary pulmonary carcinoma. In this laboratory the association of the two conditions is remarkably high. In 8 cases of asbestosis there were 4 instances of primary pulmonary carcinoma.”

1943


1943


- 11 - Jobs Requiring Respiratory Protective Equipment.
- P. 5: 11.1 Dust. “Asbestos (as in covering pipes)”

1943

Importance of respirators and ventilation

P. 402: “the use of water repellent asbestos insulation has recently replaced some types of material formerly used in ship work. For protection against dust or possible asbestosis from such material, it is recommended that both on sups and in shops, or where the material is prepared, it be dampened and that dust respirators be worn, also that special ventilation be provided. Periodic medical examination of those exposed to such hazards is also necessary.”

1943


P. 10: “Increased demands for war materials and for men to use those materials on the fighting fronts make it imperative that workers on the production front be protected from industrial hazards. Although the number of workers exposed to asbestos dust is not very great, the potential harm ranks it with the most hazardous industries, because ‘asbestosis’ is one of the most insidious and disabling industrial diseases.”

“Because of the danger and insidious effects of exposure to high concentrations of asbestos dust, effective control measures must be adopted by this industry now in order that accelerated operations will not be accompanied by a corresponding increase in cases of asbestosis. Asbestos has been incorporated into a number of products, such as protective clothing, brake linings, cements, roofing shingles, plastics, pipe coverings, and other forms of insulation, etc. This results in a variety of hazards starting with the breakdown of the native mineral.”

1943


“This article is a record of two cases of asbestosis complicated by cancer of the lung. The author suggests there is a causal relationship between asbestosis and carcinoma.” [asbestos factory]

1943


“In the last 12-15 years, such extensive experience in the field of asbestosis has been reported by both German and foreign literature that we are now quite familiar with the dangers to which asbestos workers are subjected, and the clinical picture as well as the development of this occupational disease.... One question which is as important as it is conclusive is that concerning the correlation to lung cancer, which incidence in asbestosis patients has been repeatedly observed over the last few years. The interest that this creates ... extends far into the field of
general medicine, since this would produce a new example of the exogenetic appearance of cancer. The number of such observations reported in itself is very small, but is nevertheless so significant in relation to the rare occurrence of this disease that we feel it warrants the publication of a detailed report. This is also justified by the fact that, as yet, from a clinical side, no such work has ever been published.”

Cites previous literature: Gloyne (1935); Lynch and Smith (USA)(1935); Egbert and Geiger (USA)(1936); Gloyne (1936); Sparks (1938); Nordmann (1938); Wedler (1939); Wedler and Linzbach (1941) (detailed clinical report of one patient); Animal studies: Nordmann and Sorge

p. 11: He examines 30 autopsies of German asbestos workers: “By performing the necessary calculations we get a round percentage number of 20% for malignant tumors in the lungs of cases of asbestosis autopsied in Germany” Average age of death lower than other cancer victims. “All cancer patients were suffering from chronic to severe asbestosis.” “These figures clearly show that lung cancer is the most common complication encountered in cases of asbestosis, which the exception of agonal pneumonia and cardiac insufficiency. Even tuberculosis, whose rate of incidence is generally far higher than that of cancer, is considerably lower than for lung cancer in these cases. This constitutes conclusive evidence of the close4 correlation between asbestosis and lung cancer.

P. 12 – In Studies of English patients there is “a lung cancer incidence of 12% in asbestosis patients.” In us it is “about 15%.”

P. 17 – Not have animal experiments to confi...
Asbestos (as in covering pipes.)"


1944 New Jersey, Chapter 88, Laws of 1944.

P. 186: An act establishing an elective system of compensation for the occupational diseases known as silicosis and asbestosis, .....”

“‘Asbestosis’ means a disease of the lungs, due to breathing air containing asbestos dust, characterized anatomically by generalized fibrotic changes in the lung, demonstrable by X-ray examination or by autopsy, resulting from any process or occupation involving the inhalation of asbestos dust.”


“The agents known or suspected to cause occupational cancer are arsenic, chromates, nickel carbonyl, radium, mesothorium, asbestos, ....[many others]


p.2: “all manipulation of asbestos fibre, by hand (as in sack filling or emptying, blending, seeping and shoveling) or mechanically, produces dust which, if uncontrolled, is often in dangerous concentration.”

P. 9: lists 4 main complications – bronchitis, chronopneumonia; pulmonary TB; emphysema; 22 are less common – 2nd is carcinoma. p. 10 : there is sufficient evidence to warrant careful observation in the future.”

P. 13: need “extensive and stringent” preventive measures. – dust suppression


Pp. 17, 90:“The use of water-repellant asbestos insulation has recently replaced some types of material formerly used in ship work. For protection against dust or possible asbestosis, it is recommended that such material be dampened wherever possible and that dust respirators be worn in addition to the provision of special ventilation.” also periodic medical examination

P. 611 – 4 general headings of occurrence of cancer – 1) Environmental or exogenous agents causing cancer.

P. 612: The substances or agents now known to cause cancer in man include ..., possibly asbestos,...”


p. 57: “The health problem arising from dust in industry is one of increasing seriousness. Socially, economically and legally it has become evident that immediate steps must be taken to combat the hazard represented by wholesale industrial exposure to the pneumoconiosis-producing dusts.”

P. 60: “Asbestos dust is second only to free silica in the magnitude of health hazard which it represents.”

P. 61: “Like silicosis, it is incurable and progressive; a case is reported of a patient who was exposed to asbestos dust for one year and whose sputum showed the presence of asbestosis bodies fourteen years later.”


P. 707: “Although it is true that the series is limited, the finding of an increasing incidence of pulmonary carcinoma in persons suffering with asbestosis seems too significant to be overlooked.” cites Homburger and Welz.

1944  “Abstract of the Literature of Industrial Hygiene,” Supplementary to Journal of Industrial Hygiene and Toxicology, 26 (November 1944), 183.

Abstract of H.W. Wedler, “Asbestosis and Pulmonary Carcinoma,” Duet. Med. Woch., 69 (August 6, 1943), 575-76. Author found 14 cases of malignant disease of the lungs and pleura in 92 post-mortem examinations. (16%) In excess of proportion of lung cancers generally – 2-6%)Cancers “generally in the part of the lung most affected with asbestosis.” Author thinks “must now be regarded as an occupational cancer.”

1945  Abstract in Industrial Hygiene Foundation, Industrial Hygiene Digest, 9 (January 1945), 19 – Same as previous Abstract.


From JAMA of March 24, 1945"Asbestosis, when extensive, redcues pulmonary ventilation. This may embarrass the heart and induce cardiac failure.
Distinguished between silicosis and asbestosis on the one hand and “benign pneumoconiosis on the other.”

**1946**


p.65 Table of “Workers in selected industry groups in the United States exposed to specified materials, 1936-1939,” asbestos dust: 34,949.

**1946**


“In recent years, however, cancers of the lung have been noted among workers in chromate plants, nickel refineries, and asbestos-manufacturing establishments.”

**1946**


P. 192: 3 groups of occupational carcinogens. “The second group of exogenous carcinogens consists of agents which do not possess any direct carcinogenic properties but which exert such action by changing some normal chemical constituent of the cells or tissue fluids in such a way that it becomes endowed with carcinogenic properties..... An indirect or secondary mechanism of cancerization may also be responsible for the occupational cancers observed in workers employed in chromate pants, nickel refineries, asbestos factories and arsenic operations.”

P. 200: “The continued occurrence of occupational cancers,... represents a challenge not only to the intelligence but also to the social conscience of human society, because industrial cancers may be compared with a biologic bomb having a delayed time fuse which may be placed in the body of the victim without his knowledge and realization and which may display its deadly effect many years later when the conditions connected with its introduction are often forgotten.”

**1946**


P. 195 experiments with rabbits “these experiments suggest that when asbestos is retained in the lung it will excite a connective-tissue response ultimately leading to fibrosis. The localization of the lesion depends, however, on the size of the asbestos fibre. In the case of a long fibre, which is too big for removal by phagocytosis and is not expectorated, the reaction is an intra-alveola deposition of connective tissue. This results in a nodular distribution of fibroid
tissue. With shorter fibres [196] capable of being mobilized into the alveolar walls and lymphatics the fibrous tissue is laid down in the alveolar walls, which ultimately leads to diffuse interstitial fibrosis.”

1946  

P. 493: “It is the purpose of this report to present the findings in such a unique case, the only example of either pulmonary asbestosis or sarcoidosis in a series of 1870 necropsies done at this hospital.” sawed dried asbestos pipe.

1946  
Bamforth, J., “Examination of the Sputum and Pleural Fluid in the Diagnosis of Malignant Disease of the Lungs,” *Thorax*, 1 (June 1946), 118-127.

Different cells from malignancies of the lung: Oat Cell carcinoma; columnar cell carcinoma; squamous cell carcinoma.

1946  

Used amosite. Incidence of asbestosis in shipyards of pipe coverers was low. Asbestosis is “a well known industrial disease caused by only one thing – prolonged breathing of asbestos dust.”

July 1946  
Henry F. Smyth, Mellon Institute, “Solving the Problem of the Toxicity of New Chemicals in Industry,” West Virginia Medical Journal pp. 177-8

“It is clearly the duty of a manufacturer to delay production of a chemical until the health hazards are well enough defined so that protection of his workmen is possible. It is also his duty not to sell a chemical for an application in which it would endanger the health of the public and to inform customers, by proper labeling and otherwise of the hazards of the compounds they buy.”

It is the “responsibility of industry” not state and federal governments

First do quick test; but if material is going to be produced in large quantities, need “Perform more detailed studies.”

1946  

Part 1, p. 1”need for furnishing appropriate information in those cases
where special precautions are necessary.” “The education of employees regarding chemical hazards is, and must remain, the direct responsibility of their employers. However, such hazards are not confined to employees alone, and information concerning them should, so far as practical, reach every person using, transporting, or storing chemicals. The most practical means for the seller to disseminate this information appears to be by labels affixed to containers of hazardous chemicals, bearing appropriate precautionary statements and instructions stated as simply and briefly as circumstances permit.”

P. 2: “Precautionary statements must be accurate, selectively chosen and expressed in clearly defined terms.... In all other cases [than poisons] the relative degree of the hazard can be better indicated by the use of signal words such as: ‘DANGER’ to cover extremely serious hazards, ‘WARNING’ to cover less serious hazards; and ‘CAUTION’ to cover minor hazards, together with a descriptive Statement of the hazard.”

P. 5 “Dusts – solid particles generated by handling, crushing, grinding, rapid impact, detonation and decrepitaion of organic or inorganic materials such as rock, ore, metal, coal, wood, grain, etc.”

P. 17: Sample labels: “VI. Harmful Dusts. Caution: Harmful dust. Avoid repeated breathing or skin contact....”


p.5: lists asbestos as among industrial atmospheric contaminants with a Maximum Allowable Concentration of 5 mppcf.


P. 481: “From time to time the attention of the Division of Industrial hygiene has been drawn, through compensation claims, to incapacitating fibrosis in talc workers. Since silicate dusts, of which talc is one, have been considered relatively innocuous in the opinion of most authorities (the single exception being asbestos), the occurrence of these cases has naturally raised several questions – the obvious question as to whether this incapacity has resulted from talc exposure alone, and the second and more interesting question as to whether industrial hygienists, with few exceptions, have erred in considering asbestos to be the only injurious silicate dust.”

P. 500: “this study identified tremolite talc as a silicate dust capable, like
asbestos, of causing a disabling pneumoconiosis. It reveals distinctive pathological findings associated with exposure to tremolite talc dust which postulate a special etiology for the resulting disease.” some similarities with asbestos disease (p. 498-9)

1947


P. 407: 37 year old asbestos worker from Sweden. Work was cutting asbestos insulating board. “He denied exposure to undue amounts of dust.” tumor. What kind?

P. 411; Clinical diagnosis: Carcinoma of the lung, probably metastatic.... anatomical diagnosis: Mesothelioma of pleura and pericardium, with metastases to right lung and retroperitoneal lymph nodes.”

P. 412: “A number of papers have been written to the effect that there is no such tumor as mesothelioma of the pleura, that the cells lining the pleura do not form tumors and that these tumors really arise from a small focus in the lung. We have held a similar opinion for a long time. This is perhaps the first case in which we believed that there was actually such a tumor. It certainly fits in which most of the cases of mesothelioma of the pleura that have been reported.”

1947


P. 2: “the 'maximum permissible dustiness' for asbestos is commonly taken to be five million particles per cubic foot. This represents good attainment in the dust control program. It is emphasized, however, that dust elimination to this extent does not positively insure that no asbestosis will develop in some workers after a long working life (greater than 20-25 years.) Scientific evidence is obscure on this point.”

p. 15: marked variation in asbestosis in plants: “Plant A has recorded cases diagnosed as asbestosis to the extent of about 20 per cent, and in the past nine years has compensated, on the average, two cases per annum” also true of Plant J; 3 other plants have markedly fewer cases

p. 17 – need for periodic medical examinations

p. 18: should also form a Medical Asbestosis Panel – outstanding medical men to review x-ray files and clinical data

aim to eliminate “asbestos cases from the plants of the Asbestos Textile Institute members.”

P. 22: “the information available does not permit complete assurance that five million is thoroughly safe nor has information been developed permitting a better estimate of safe dustiness.”

P. 110: “Most silicates will produce dust; and if workers are exposed to these dusts they are likely to show radiographic changes in the lungs, and in certain instances they will develop serious diseases.... and asbestos, the last named producing by far the most serious illness.”

P. 116: “Incidence of carcinoma of lung is abnormally high in those exposed to arsenic dust and to asbestos.”


p. 70: “Asbestosis and Carcinoma of the Lung. During the 23 years 1924 to 1946 inclusive, 235 deaths, either caused by Asbestosis or in which Asbestosis has been proved at autopsy have come to our notice. Cancer of the lungs or pleura was found to be present either as a cause of death or an a concomitant in 31 (13.2%) of these 235 cases....” p. 80: average duration of exposure to asbestos dust was 16.5 years for those cases complicated by carcinoma of lung, compared with 13.4 years for those not so complicated.


P. 260; Object – “To ascertain whether cancer of the lung showed any special incidence upon particular occupations.”

P. 296: “Cases of cancer of the lung have occurred in some occupations involving exposure to asbestos. In the death certificates examined, and in the Reports of the Chief Inspector of Factories, no occupations involving exposure to any kind of dust, except those concerned with asbestos, arsenic and nickel, which employ very small numbers, have been found in which there might be an increased incidence of cancer of the lung.”


P. 249; “recent reviews of the literature on the association between asbestos and lung cancer reveal different opinions as to whether asbestosis has been an aetiological factor in these cases. Homburger, in 1943, reviewing nineteen known cases found no reliable answer to the problem, whereas Wedler [H.W., “Lung Cancer in Asbestosis Patients,” Über den Lungenkrebs bei Asbestose,
March, 1943; “Asbestosis and Lung Cancer,” Deutsche Medizinische Wochenschrift, August 1943] in the same year considered there was a significantly high incidence of lung cancers in autopsied cases of asbestosis. Kennaway and Kennaway (1947), who examined this problem as part of their broader statistical study of lung cancer, also leave the question unanswered. The statistical difficulties are due to the small numbers of asbestos workers, and to the paucity of published autopsy records of such cases.”

Presents a case – making asbestos pipe covers from age 15-22.

P. 252: long latent period (also noted by Lynch and Smith, Gloyne and Egbert and Geiger.


P. 882: Literature divided on relationship of carcinoma of lung to asbestosis and silicosis; “In our necropsy service has been encountered an additional case, making a total of three in forty cases of asbestosis during a period of eighteen years. This incidence of 7.5 per cent is to be compared with a general incidence of 1 percent of carcinoma of the lung in 2,683 necropies in the last ten years. Of further interest in the question is the fact that each of our three cases had medium to advanced grades of asbestosis.”

“Apparently to the present there are at least sixteen necropsied cases of carcinoma of the lungs in asbestosis, which, considering the comparatively small necropsy experience in the disease, seems excessive.”

“Discussion,” Leopold Brahdy, P. 886 “Although we must keep an open mind, the evidence warrants the tentative conclusion that asbestos workers do have a higher incidence of lung cancer.”

P. 887notes failure to recognize this occupational disease during workers’ lives.

Saita, G “Occupational Tumours of the Lung, Med. D. Lavoro, 39 (1948), 105-116 English summary at end of article reprinted in Industrial Hygiene and Toxicology, 23 (1948), 865.

“The number of tumors of the lungs has greatly increased during the last years and many doctors think that this fact is partly due to the increased industrial use of irritator toxic carcinogenic matter.... The conclusion is that only asbestos dust and coal tar derivatives possess an undoubted carcinogenic power on the lungs.”

Castrop, V.J., “Fume and Dust Exposure,” National Safety News, 57
P. 79: “Asbestos used in the formulation of brake lining is a potentially harmful compound.” produces asbestosis. Permissible limit of 5 mppcf.


P. 1219; “Until recently the coexistence of asbestosis and cancer of the lung was considered by many investigators a coincidence. Since 1935, 23 such cases were recorded by American, English and German physicians. Wedler [German study] noted 14 cases of asbestosis cancer in a series of 92 necropsies on patients with asbestosis, or about 15 per cent of cancer of the lung in persons who died from this industrial disease.”

“A causal relation between asbestosis and cancer of the lung is supproted by the following observations: the incidence rate of cancer of the lung in this group is excessive, since the normal death rate from cancer of the lung among adults examined at necropsy at present is about 1 per cent of all necropsies. Moreover, there is a distinct shift in the sex distribution of cancer of the lung in the series of asbestosis cancers reported from England. The male-female sex ration is 2.4:1, while it is 5:1 for cancers of the lung in general. This shift indicates that an environmental and evidently occupational carcinogen was active in the asbestosis group, tending to equalize the incidence rate of cancer of the long for both sexes.”

Also experimental work supports this. “The histologic character of the cancers (squamous cell cancer instead of adenocarcinoma seen in the spontaneous cancer of the lung of mice) and the histogenic derivation of the tumors (bronchial mucosa instead of alveolar epithelium of the spontaneous type) indicate that a specific factor of exogenous origin, represented by the inhaled asbestos dust, was responsible for the bronchial cancers observed.”

P. 1220: “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.”


P. 11: On first page lists asbestos among “Carcinogens known to be present in human environment”

P. 12: and again on 2nd page under lungs as “original sites of malignant growth due to carcinogens in the environment.”

1949 Meiklejohn, A. “Pneumoconiosis,” *Postgraduate Medical Journal*, December 61
1949, 599 - 605.

P. 600 “‘Pneumoconiosis’ means fibrosis of the lungs due to silica dust, asbestos dust or other dust, and includes the condition of the lungs known as dust reticulation.”


P. 632: “Whatever doubts there may be as to the fibrogenic potentialities of many silicates, the processing of asbestos unquestionably exposes the worker to the risk of lung disease.”

“Today asbestos is used for a wide variety of purposes. Textiles enter into the manufacture of fire-fighting suits, safety curtains, and boiler mattresses. Asbestos enters into the composition of lagging material for steam pipes, jointing for steam pipes, panelling [sic] of rooms, tiles, the lining of chemical pans, the coating of bulkheads of ships and marine piers and perhaps most significantly into the brake linings and clutch rings of motor cars.”


P. 639: “Although silica, asbestos and coal are the most important dusts which cause pulmonary fibrosis,.....”

P. 645: “it is now known that the incidence of cancer of the lung in certain occupations is sufficiently high to suggest a significant etiological relationship between the disease and the occupation.”

“...evidence is accumulating that cancer of the lung is unduly associated with asbestosis.”


P. 5: “Of all forms of pneumoconiosis which have been reported as possible etiologic factors in the development of bronchiogenic carcinoma, none has been the subject of greater argument and uncertainty than asbestosis.... It should be pointed out, however, that more and more evidence appears to be accumulating to incriminate asbestosis in this connection.” cites Lynch and Smith (1939), Holleb and Angrist (1941), Nordmann (1938), Annual Report of chief Inspectors of Factories, 1947, Wedler (1943), JAMA (1949).
“Annual Report of the Chief Inspector of Factories for the Year 1949,”
London, His Majesty’s Stationary Office, 1951.

P. 144: code of Regulations for handling asbestos in force since 1931
Need constant vigilance. “... it is very necessary to keep an ever watchful
eye for the new use of asbestos in some manufacturing or other process, for
examples, on ships or buildings where the work may be undertaken by someone
not fully realizing the necessity of preventing as far as possible the inhalation of
asbestos fiber and dust.”
P. 145 – well known where there is a dust hazard

Brown, Vandiver, “Discussion: The Management Viewpoint,” Sixth Saranac
Symposium, Proceedings, Pneumoconiosis, (New York: Paul B. Hoeber,
1950), 567-573.

p.569: [Regarding the TLVs for asbestos]”The allowable limit for
industries using this material is a ‘mandatory’ requirement of not more than five
million particles per cubic foot, 10 microns or less in longest dimension. ... So far
as I have ever been able to ascertain, no one can state with certainty what is the
maximum allowable limit for asbestos dust. ... I question whether there exists
sufficient data correlating the disease to the degree of exposure to warrant any
determination that will even approximate accuracy. It is my guess that the figure
was allocated to asbestos either because it had already been prescribed for
silicosis” or because of some other arbitrary decision. Says that Gardner found that
fine particles of asbestos not dangerous in experimental animals, just those 20 to
50 microns in length and 1 to 3 microns of thickness.

Hueper, W.C., “Environmental Cancer Hazards CAUSED BY Industrial Air
Pollution,” Archives of Industrial Hygiene and Occupational Medicine, 2
(1950), 325 -328.

P. 327: Table of Environmental Cancerogenic Agents and Sites of Cancer:
Lists asbestos – type of contact – respiratory; Site of cancer,
carcinoma of lung.

Meiklejohn, A. “Advice to Pneumoconiosis Patients,” British Journal of
Tuberculosis, 44 (1950), 104-108.

Merewether, E. R. A., “The Pneumokonioses: Developments, Doubts,
Difficulties,” Canadian Medical Association Journal, 62 (February 1950), 169-
173.

P. 172: “A point of considerable interest which comes to light from
our records is the possible association of asbestosis and cancer of the lungs. In the
same series, 13.2% of cases of asbestosis were found on autopsy to have cancer of
the lungs. This is in contrast to silicosis in which our figure is 1.32%.”


P. 49; “In the opinion of Stewart (1947) asbestosis is a more serious disease than silicosis. It is now recognized that the fibrous form of asbestos determines many of the characteristics of the disease: the particles, which may reach 100 u in length, are caught in the bronchi and bronchioles; phagocytosis is imperfect; the bases of the lungs are chiefly involved; the essential lesion is diffuse, never nodular, fibrosis;”
“The association of asbestosis with cancer has suggested a causal relationship.”


“Studies conducted by the United States Public Health Service now point to airborne particles as the cause of some forms of cancer and respiratory ailments. Dr. W.C. Hueper of the National Cancer Institute, Bethesda, Md., referred specifically to air pollution with asbestos, selenium, beryllium, arsenic, and chromates as the probable cause of increased lung and respiratory tract cancers.”


P. 567: “Both silica and asbestos have on many occasions been indicated as predisposing to lung cancer; but in the case of silica, it has never been demonstrated statistically, while accumulating evidence points more and more strongly to asbestos as a carcinogenic agent.”


P. 778: “Asbestos, which contains no free silica, produces a severe pulmonary disease, but the mode of action of the asbestos fibre differs from that of the silica particle.” “…in regard to asbestos, it appears that the larger particles are more harmful than the smaller and that in the lungs they act as mechanical rather than as chemical irritants.”
P. 781: prevention – total enclosure of processes in mechanically ventilated structures; isolation; substitution; local mechanical exhaust ventilation; wet processes; good plant ventilation; personal respiratory protection.

p. 2: Although free silica dust ... has been considered the most harmful, silicates (for example, asbestos) have been found almost as harmful, and other dusts certainly have a detrimental effect upon health under certain conditions.


P. 812; “there were 80 cases of primary neoplasm of the lung distributed as follows: 55 in the silicosis group (i.e., 6.9%), 17 in the asbestos group (i.e., 14.1%) and 8 in the other forms of pneumoconiosis (i.e., 6.7%).


Presents case where “shortness of the exposure to asbestos dust, the long length of time which elapsed before the growth developed, and the unusual histological pattern of the growth.”


Worked for WPA as a pipe coverer and “worked with asbestos exclusively for one and a half years.” then worked with asbestos in a shipyard

P. 834: “a case of bronchogenic carcinoma in association with pulmonary asbestosis is presented .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 the asbestosis, and the need of careful preventive measures.”


Report on 7 cases

P. 150: “The term most often applied to the tumors (endothelioma) implies that they arise from the endothelial tissues of the lymphatics, blood vessels, or lining cells of the pleura. Those who stress the point that the pleural endothelium is embryologically derived from the epithelial component of the mesoderm have generally preferred to employ the more inclusive term ‘mesothelioma.’ In common with Robertson, a number of writers assume that there is no such entity as an endothelioma or mesothelioma of the pleura, that all primary pleural tumors should be classified as sarcomas, and that all other tumors are secondary to neoplasms of the lung or other unrecognized malignant tumors.”
P. 57: “A fundamentally different situation, ... [from silica] seems to prevail in regard to the causal relation between asbestosis and cancer of the lung. ... since 1935 ... an appreciable number (55) of cases of asbestosis complicated with cancer of the lung have been recorded from England, Germany, Canada, and the United States .... The proportion rate of lung cancer in persons with asbestosis who came to necropsy stands between 13 and 15%. The exposure time ranged from three to 27 years (average 15 years. The latent period after cessation of exposure was from 10 to 12 years..... Although Warren rather recently maintained that the connection [p. 58] between asbestosis and lung cancer is of coincidental nature, the actual existence of a causal relation appears to be very likely.”

P. 58: while the experiments of Vorwald and Karr performed on guinea pigs that were exposed to asbestos dust gave negative results, those conducted with mice by Nordmann and Sorge apparently yielded positive results....”

P. 312: Appendix 1 “List of Occupational Diseases Which in Social Insurance are Considered Equivalent with Occupational Accidents,” 29 “Asbestosis with diminished capacity to breathe and impaired circulation or associated with cancer of the lungs.”

P. 1: Asbestos mean literally “unburnable.”

P. 2 – Many years Saranac Laboratory carried on investigation of “the cause, nature and development of asbestosis . – present paper on experiments with animals.

P. 41: Summary; A. “Various species of animals, including the guinea pig, the rat and the rabbit, but not the mouse and the dog, develop peribronchiolar fibrosis of the lung similar to human asbestosis after being exposed by inhalation or intratrachial injection to long chrysotile asbestos fibers.”

B. “Long asbestos fibers are essential in the production of the peribronchiolar fibrosis; short fibers are incapable of producing this reaction.”

P. 42: C. Mode of action mechanical rather than chemical.

D. As the concentration of dust increases, the reaction “develops in shorter time.”

Supported by “a group of companies of the asbestos industry.”

   P. 56: “At the present time it is generally agreed that the most important sources of industrial disease of the lungs are free or uncombined silica and asbestos.”


   P. 475: “Asbestos as a cause of respiratory-tract carcinoma is disputed by Vorwald [personal communication] in this country. English Reports seems convincing....” [cites variety of evidence.]
   P. 480 cites Vorwald’s work on Experimental asbestosis


   P. 2382: “...asbestosis and the inhalation of certain of the chromate dusts appear to play etiologic roles in the production of lung cancer whereas silica does not.”


   “Cancers of the respiratory tract involving the mucosa of the nose, the antral cavities, the larynx, the bronchi and lungs occur in workers who are exposed to the inhalation of chrome salts, asbestos dust and nickel carbonyl.”


   P. 12: “Silicosis and asbestos are of special important among the dust which fall into this category [pneumoconioses]... silicosis is not, apparently associated with, or productive of lung cancer, whereas asbestosis very probably is.”
   P. 14: “The relation of asbestos to lung cancer, long suspected, has received new confirmation from the 1949 Annual Report of the Chief Medical Inspector of Factories in England. During the period from 1924 to 1946 inclusive, 235 deaths were reported either caused by asbestosis or in which asbestosis had been established at necropsy. Cancer of the lungs or pleura was found in 31 of these cases or 13.2 per cent. This is very high compared with the general incidence of 1 per cent for cancer of the lungs among all adults examined at necropsy in
England.” seen as more related to asbestosis than the asbestos dust itself.

1952


P. 212: Disputes Nordmann and Sorge’s study and evidence that asbestos dust is capable of provoking tumors. “On the contrary, it would appear to demonstrate that asbestos dust did not provoke lung tumors under the conditions of test employed.”

1952


P. 253: found info on 21 persons in Britain on lung cancer and asbestosis “of whom only one was a man working under conditions now prevailing in the industry.... It was the consensus of Dr. Gloyne, Dr. Wyers, and Dr. Merewether that the nature of the disease, asbestosis, as seen in England has changed so that it is less common and less severe in individuals whose employment in the industry has taken place only since 1932. It was the consensus that a lung tumor hazard formerly existed in this industry in Great Britain but that there is no evidence to show that such a hazard continues to exist under the working conditions now prevailing....Individuals beginning employment in the 1930s are therefore only now reaching the mean age at which tumors might be expected to appear. The size of this group is not known, but the fact that it has thus far provided only a single case of coexistent lung cancer and asbestosis argues that any lung tumor hazard has been greatly diminished, at least in point of time. Whether there will be any significant number of cases with delayed appearance remains to be seen.”

1953


P. 49: “But it can be said that the diseases of the lungs caused by dust are more dangerous than the industrial dermatoses.... the pneumoconioses come on like a thief in the night, or rather, like a thief who plies his trade for days, nights, and years before he is finally caught. And by that time the damage is done.”

Chronic fibrosis – asbestosis

1950

p. 72: Harry Green, Yale University School of Medicine, “Fragments of embryonic mouse lung were impregnated with asbestos fibers and transplanted to the subcutaneous space of more than 150 adult animals. ... At the end of 9 months] ... all showed asbestosis but in no instance of cancer found.”

p. 115: Kenneth Lynch, “Discussion – Potential Occupational Factors in Lung Cancer – Asbestos.” “In an autopsy series [of 49 cases of workers in asbestos manufacturing] who were shown to have demonstrable deposits of asbestos in the lungs, there was associated carcinoma of the lung in four instances.” [despite statistical limitations] “this gives an incidence of 8.2%.”

p. 117: Questions validity of Nordmann and Sorge.
   Notes their own study supported by National Cancer Institute. Not been concluded. “From an analysis of the problem … it appears that the available evidence on whether asbestos may play an etiological role in the occurrence of carcinoma of the lung is not conclusive but that it does support the suspicion sufficiently to require continued investigation.”


“...a vast amount of information on pneumoconiosis, which has come, during this century, to be recognized as the outstanding occupational disease. It came to notoriety with the recognition of silicosis with tuberculosis superimposed. At first the tendency was to regard silica dust as the one toxic dust, next asbestos demonstrated its power for evil, and other followed....”

Protection of worker: abolition of processes creating dusts; substitution; suppression of dust at source; removal of dust at point of origin; ventilation; wetting; electrostatic precipitation; and last result personal protection equipment.


P. 526; “With lung cancer, exceptional risks have been recognized in several unrelated industries.... asbestos manufacture....”

“In the manufacture of asbestos and arsenical sheep dip the risk more like to have been of the same order as that involved in the production of gas [double that expected].


P. 728: “the association of asbestosis and carcinoma of the lung has been mentioned frequently in the literature. Heretofore some authors have believed that
the cases were too few in number to be of significance; others, especially Vorwald and Karr, have stated that ‘inhaled dusts, except those containing recognized carcinogenic substances (as radium and tar) cannot in general be considered as etiologic factors in the development of primary pulmonary carcinoma.’ [1938] Our conclusion at present is in favor of the concept that the association of bronchogenic carcinoma with asbestosis is more than coincidence.” Cites table that shows incidence rate based on 5 studies of 13.8%. “This is considerable higher than the incidence of lung carcinoma in routine necropsies, which in a comparable period (1935-1948) ranged from 0.8 to 2.4 percent.”

P. 730: “since there are approximately 10,000 workers engaged in potentially hazardous asbestos operations in this country, it is reasonable to assume that there are many unrecognized cases of asbestosis. From the evidence presented a higher incidence of bronchogenic carcinoma should be expected in this group.”


P.45: “The pathologic changes produced by asbestos are not like those of silicosis. The asbestos fibers group about the neck of an alveolus and stimulate the formation of a diffuse fibrosis. There is no definite migration or transportation of the dust particles to the lymph nodes... as the fibrosis increases, reduction in lung area causes serious dyspnea.”

p.45: “In the lungs of patients who have died after prolonged exposure to asbestos dust and in the sputum of men with considerable asbestos-dust exposure are found what first were called curious bodies and later asbestos bodies.”

p.46: “Asbestosis and cancer” cites 1947 report of British chief inspector of factories that “13.2 per cent [of cases of asbestosis autopsied between 1924 and 1946] were complicated by carcinoma of the lungs or pleura.”[Admires the British data and questions whether US data – specifically he cites Vorwald and Karr of Saranac in 1938 -- as possibly missing this relationship because its data may not be as good.] “For example Vorwald and Karr at the Saranac laboratories reviewed such data from their own experience and concluded that ‘inhaled dusts,’ except those containing recognized carcinogenic substances, ‘cannot in general be considered as etiological factors in the development of pulmonary carcinoma.’ But we still are a bit envious of the tidy way in which the British assemble their industrial data on morbidity and mortality.”


Cites previous studies that implicated asbestos “in the causation of cancer.”


P. 122: “Rats injected into the marrow cavity of the femur, pleural cavity, or paranasal sinuses with powdered arsenic, beryllium, or asbestos suspended in lanolin did not develop any significant number of cancers – either at the site of deposition or in any remote organ – causally related to the chemicals introduced.”


P. 28: “Our findings, therefore, lead us to the conclusion that in man the onset of asbestosis is not determined by the absolute amount of mineral material present in the lung parenchyma but is related to changes occurring in the inhaled material after a time interval from first exposure, which may be as long as 20 to 30 years. As a result of these changes, a fibrogenic agent is released which is capable of exerting its effect under favorable circumstances. The nature of this agent is unknown, and the Environmental factors are not yet clarified.”


P. 35: “It is considered that the mechanism which is concerned with the production of human asbestosis is not the same as that which is responsible for the production of asbestotic changes in the experimental animals exposed to high concentrations of asbestos dust. The appearance of such changes appears to be related to the breakdown of asbestosis bodies which may liberate some fibrogenic agent.”


p. 204: “To be fully effective to the industrial hygiene team, including the responsible physician, toxicological data on a chemical should include answers to the following questions: 1. What uniform concentration is tolerable 8 hours a day for a working life time? 2. What correction in the average must be made for brief
peak concentrations? 3. What single brief exposure to a high concentration is tolerable each day when there is no exposure the rest of the day?

Refers to TLVs of ACGIH: “The values are well considered and they usually represent the consensus of several years widespread experience with each material. Unfortunately, since the publication of Cook’s article in 1945 no one has pointed out the scientific basis for each tabulated value to allow assessment of its relative validity. Some are simply extrapolations from animal experiment which are subject to correction as experience accumulates and others are based on many published reports of human experience. … For newer and less widely used materials there is not one place to look for hygienic standards.”


P. 131: “Cancer arising primarily in pleurae, lungs or bronchi was present in 26.1 per cent of males and 8.7 per cent of females [in asbestos textile workers]. This significantly higher incidence in males may be accounted for by the fact that 7 women died before the cancer age, ....”

P. 132: “In connection with this particular form of industrial hazard, it must be emphasized that since the introduction of regulations for the control of asbestos dust in 1931, the amount of exposure of workers to dust has been enormously reduced and we shall therefore expect to see a disappearance of those severe degrees of pulmonary fibrosis that have been described heretofore and possibly a disappearance of associated cancer. All the present cases that had asbestosis and cancer had been exposed prior to 1931.”


[gives TLV for asbestos at 5 mppcf]


P. 427: “Thus, several types of employment may constitute etiologic factors in lung cancer.... Other studies, including our own, strongly implicate the occupations shown in table VIII: painting, welding and other work involving hot metals, commercial cooking, metal mining, steam fitting and other asbestos work.”


“Not all cases of asbestosis have a breathing problem .... Unlike silicosis,
we found some persons with slight x-ray changes indicating asbestosis with
definite functional impairment present.”

1955

Doll, Richard, “Mortality from Lung Cancer in Asbestos Workers,” British
Journal of Industrial Medicine, 12 (1955), 81-86.

P. 81: “Sixty-one cases of lung cancer have been recorded in persons with
asbestosis since Lynch and Smith (1935) reported the first case. In view of the
infrequency of asbestosis, this large number of cases suggests – but does not prove
– that lung cancer is an occupational hazard of asbestos workers.”

P. 83: “It appears that the men who had been exposed to asbestos dust
suffered [p. 83] an increased mortality from lung cancer, other respiratory diseases
and cardiovascular diseases, in association with asbestosis, but that mortality from
other diseases was close to that expected.”

P. 86 “From the data it can be concluded that lung cancer was a specific
industrial hazard of certain asbestos workers and that the average risk among men
employed for 20 or more years has been of the order of 10 times that experienced
by the general population. The risk has become progressively less as the duration
of employment under the old dusty conditions has decreased.” (11 deaths with
only 0.8 expected)

1955

(September 24, 1955), 780-781.

Cites Doll study for evidence of link between asbestos and cancer.

1955

Cartier, Paul, “Some Clinical Observations of Asbestosis in Mine and Mill
Workers,” Archives of Industrial Health, 11 (19555), 204-207.

P. 207 – data from Thetford Mines by employee
“To summarize, I believe that asbestosis is a serious disease in some
instances, but more frequently it remains a disease which can be tolerated quite
well for many years, even without appreciable symptoms, as long as another
serious disease does not supervene to cause death. On the other hand, in practice,
this disease may look more serious and cause important medicolegal problems if a
too scientific medical concept or a too literal social interpretation is accepted by
the medicolegal professions, labor and compensation bodies.”

1955

Gardiner, John F. “Occupational Diseases of the Lungs,” Nebraska State

Repeats information that asbestos “produce[s] disabling pneumoconiosis.”

P. 1388: “the epidemiologic and pathologic evidence supporting a causal relationship between asbestosis and cancer of the lung ... is quite substantial.”
Diversify of histologic types – squamous cell; round cell-anaplastic variety; adeno-carcinomas; combination. Exposure period from 6 months to 42 years.
P. 1389: “the available evidence is adequate for recognizing asbestosis cancer of the lung for medicolegal reasons as an occupational disease.”


Case of 53 year old man employed for 26 years spinning course yarn and asbestos.

Lynch, Kenneth M. “Pathology of Asbestosis,” Archives of Industrial Health, 11 (1955), 185 - 188.

P. 185: up to 30 years ago, “any thought of ill effects of occupational exposure to asbestos dust tended to confuse the resulting disease with silicosis.”
Even though conditions have improved “it should not be assumed that the hazard has been eliminated. Harmful asbestosis is still occurring.”


P. 222: Table 1 – Dust Diseases of the Lung – Cancer – Asbestos.”
P. 224: Section on asbestosis; Section on prevention


“Years ago industrial research was unconcerned about problems of health and safety that might be involved in a product. When the product was developed it was put on the market. Today our industrial research organizations probe into
every health hazard. And every safeguard is insisted upon before the product is marketed to the consumer. A number of companies have established research organizations and laboratories for the sole purpose of eliminating health hazards that might injure the consumer…. Some companies have granted fellowships for this specific purpose. And industry has inspired, to a very great extent, the movement to label properly certain types of products that might harm the consumer if he were not forewarned.”

1956  

Asbestos – p. 667: This dust produces a diffuse interstitial pulmonary fibrosis known as asbestosis.”

P. 668 “It has also been surmised that asbestosis may predispose to bronchogenic carcinoma – a hypothesis which has not been fully confirmed.”

1956  

“31 workers engaged for 20 years or more in different kinds of insulation works were examined for occupational lung disease. They had been exposed to dust containing asbestos, kieselguhr, magnesia, glass-wool and rock-wool. 9 were found to have definite signs of pulmonary asbestosis. 19 showed abnormalities of the pleura, 12 of these being bilateral. In 11 cases calcification of the pleura was found, being bilateral in 8 of them.”

1956  
Stokinger, Herbert E. “In the Field of Toxicology,” American Industrial Hygiene Association Quarterly, 17 (September 1956), 340 - 344.

P. 342: Reviews Doll, Cartier studies.”With such relatively small numbers of cases one must be extremely cautious in drawing conclusion of a causal relationship between exposure and disease.... it would seem well to wait until a more impressive number of cases has been documented.”

1956  

P. 254, Theodore Hatch: “It should be the industrial hygienist’s responsibility to accumulate all the information that is available, and it should be his responsibility to digest this information and determine to what extent more information is needed, because he can’t limit his responsibility by simply saying that only so much is known about the substance and therefore, decisions have to be based upon this limited data. He must see to it that
the necessary additional information is obtained.”

P. 257: one large chemical company “set up an elaborate program of education of the ultimate consumer of the material, in recognition of the very important fact that, without adequate safety measures, serious difficulties were bound to result.”

P. 267, Zapp of DuPont – on importance of animal experimentation.

P. 270 Moderator asks, “Are present labeling laws adequate with respect to requiring warning and preventive measure as to toxic qualities.”

H.S, Baile, General Accident, Fire and Life Assurance Corp: a) adequate in terms of protection from liability of company: “The common law itself imposes a duty upon the manufacturer to bring to the attention of the users of his product and people who are in the vicinity of its use, the dangers which are inherent in its use or may flow from it.” b) adequacy of the laws to protect the public from injury. “I think we have to say that the laws as such are inadequate.’

P. 272: L.C. McGee, Medical Director, Hercules Powder Co.: responds to question “whether industries should tell their employees that the material they are handling is toxic or carcinogenic.” Answer “these people are entitled to it…. I would say that we must give the employee that knowledge so that he can understand and use it in coming to a conclusion as to why he must do certain things to protect himself.”

P. 273: hatch agrees

P. 276, P.W. Bachman, VP and Director of Research and Development, Koppers Co.”To maintain safety awareness you must say that a company has to tell its employees what the nature of the materials is which they are handling, whether they are toxic or carcinogenic, because you can never have a safety program unless the give the people working with toxic or carcinogenic materials such information.”


p.132: “In the introduction to its 1956 list (ACGIH 1956) the Committee on Threshold Limits says, ‘values are given … for the maximum average atmospheric concentrations of contaminants to which workers may be exposed for an 8-hour working day without injury to health.’ Careful study of the data which support the currently accepted values suggests that no such description can be truthfully attached to most of them. Industrial hygienists recognize this. They are accustomed to emphasize that the values should be regarded simply as benchmarks, guides to good practice. Indeed, the threshold limits committee itself confusingly warns ‘threshold limits… should not be regarded as fine lines between safe and dangerous concentrations.’”

p.134: “At this time it is prudent to set the standard for a carcinogenic substance substantially at zero, as has already been done for nickel carbonyl, and no considerations can justify allowing the inhalation of any concentration which is avoidable.”


Warren Cook, Introduction, p.274: refers to the “Known Carcinogenicity of …
Henry F. Smyth, “Prepared Discussion,” p.284: “It is obvious that there is no single method or pattern of methods which can satisfy the varied requirements for establishing threshold limits. It is equally obvious that although rigid standards were described that would satisfy a discriminating jury of scientists, the available data for stabilizing threshold limits for all but a very few substances would fail to satisfy such limits. The real use for threshold limits, as a guide for industry in the control of exposures and as a measure for action by governmental agencies, demands a continuing improvement in the quality and quantity of the methods by which truly valid criteria may be achieved.”

Herbert E. Stokinger, “Prepared Discussion,” p.285: “There is a real need for more data based on industrial experience. The often-heard statement ‘the threshold limits are nothing but educated guesses’ unquestionably reflects the wish at least that more data be firmly based on industrial experience to substantiate the choice of limits…. Although the table shows that the educated guesses account for a relatively small number [9 percent], it does confirm the often expressed feeling of the need for more solidly based levels.”

p.286: “…with one exception, nickel carbonyl, limits taking into consideration potential carcinogenicity have not been assigned…. As a suggested method of approach, the following is offered: to the level judged safe for other types of systemic injury add a safety factor for carcinogenicity. The magnitude of the safety factor is suggested from 100 to 500.”

1957 Abstracts of World Medicine, 21 (January to June 1957). 359.


Studied 4 main groups – from “Unaffected Cancer incidence,” to “Increased Cancer Incidence, e.g. asbestos workers (Doll, 1955).”

P. 33: “There is general agreement in this country that asbestosis is associated with an increased cancer incidence. Gloyne (1951) found 14 per cent; the Annual Report of the Chief Inspector of Factories (1955) fives it as 16 percent; and Doll (1955) as 14.2 percent.”


P. 333: “Asbestos shares with quartz the capacity for producing rapidly, in a few years, a high degree of fibrosis of the lungs when inhaled as airborne dusts…. Persons affected with asbestosis have an increased risk of contracting cancer of the lung, a tendency which has not been observed with other pneumoconioses.”

P. 334: “characteristic asbestos bodies are found in the fibrous tissue, air
spaces and sputum and within macrophages; they consist of asbestos fibres sheathed with iron-containing protein derived from the capillary exudate. They were first observed in the lungs of an asbestos worker in 1927....”

P. 343:”the only other dusts [than quartz] which cause rapidly developing collagenous growth are fibrous silicates, loosely termed asbestos. The pathology of asbestosis contrasts with silicosis because the physical nature of the fibres renders them liable to become caught in the fine airways of the primary lobule. It is possible that chemical factors are concerned in the ensuing fibrosis which seems to lack the active progressive of silicosis.”


P. 213:“Primary lung tumors were demonstrated microscopically in 46% of the dusted mice and in 36% of the control animals. Multiple pulmonary tumors were demonstrated in 18% of the dusted animals and in 8% of the control animals.... No clear-cut proof of malignancy was found in any of the lung tumors studied.... We have been unable to demonstrate any proof of the carcinogenicity of asbestos under the conditions of this experiment, but the increased incidence of multiple lung tumors in dusted animals must be regarded as a possible accentuation by the inhalation of asbestos dust of an existing tendency to develop lung tumors ... The equivocal nature of this Reports does not answer the questions raised by accumulating clinical evidence that asbestosis is strongly associated with, and therefore a possible cause of, pulmonary carcinoma in man.”


“Although some of the patients were still employed in the asbestos textile mill when evidence of neoplasm appeared, there were several in whom carcinomas developed 20 to 30 years after cessation of exposure, while they were no longer workers in this industry. The primary anatomical site of the carcinoma was in the lower lobes of the lungs in all cases; 7 involved the lower lobe of the right lung and 6 began in the lower lobe of the left lung. In the morphologic classifications of the neoplasms, 8 were found to be of the squamous cell type, 2 were anaplastic, and 3 were adenocarcinomas. This frequent association of pulmonary asbestosis and bronchogenic carcinoma (almost 50 percent) as seen in this study lends further support to the proposition that asbestosis is a carcinogen in susceptible individuals after critical exposure in the textile phase of the industry where asbestos dust is in high concentration.”

p.269: “Unlike other toxicology laboratories, those working for industry have not discharged their obligation when they have published their results in the scientific literature. In industry, toxicological investigation is a staff function, furnishing information which guides the actions of many specialists. The laboratory is a member of a team. The purpose of the entire team is to make a profit by satisfying human needs. The purpose of the toxicology laboratory itself is to see that the commerce and technological advance which the team produces shall not be at the expense of human health.”

p.271: “probably every compound planned or made by research or development laboratories should be considered by the toxicology laboratory, at least to the extent of making predictions about handling hazards. If the compound is of sufficient interest for the firm to offer samples to customers, then its acute toxicology should be estimated by range finding methods. This should be completed and predictions should be made about chronic toxicity before any of the compound is sold. …. The amount of more detailed toxicological study which a product receives should be proportioned to its probable uses, to the degree of human exposure in its use, to the degree to which it reaches the consuming public, and top the magnitude and subtlety of its toxic effects…. Toxicological information should be furnished to every customer, because the supplier is responsible for seeing that the customer has the facts to allow safe use. The more direct the contact between customer and toxicologist can be, the less chance for distortion of information. Before a chemical is in general use, the toxicological facts to safe handling should be published in the medical literature.

p.272: Despite the potential hazards of hundreds of new chemicals each year, most injuries from chemicals are due to those which have been familiar for a generation or more. It is important for the perspective of the toxicologist that he keep this fact well to the forefront of his mind. He must not neglect taking about the hazards of the old standbys – lead, benzene, and chlorinated hydrocarbons, just because this week he discovered the horrifying action of something brand new.”

1957  Military Standard, 129B, April 10, 1957, p. 6; paragraph 2.2.10.4.3

“Hazardous Chemicals:” “all package units of hazardous chemicals to be ultimately issued to the consumer who may be exposed to such chemicals under conditions of ordinary use shall have affixed thereto such warning labels as may be required in accordance with the Manufacturing Chemists Association Manual L-1…..”


p. 6: The United States Public Health Service has approved the principles of precautionary labeling developed by the Manufacturing Chemists Association…..”

p. 6: “The warning labels suggested in this Manual should be used in addition to, or in combination with, any label required by law.”
Secretary of the Navy to Chiefs of various Bureaus and other organizations in Navy, September 24, 1956, “Uniform labeling program for hazardous industrial chemicals and materials,”

Enclosure 3: “Elements of a Labeling Program,” Hazardous chemicals and materials within the scope of this Instruction have been groups into six general classifications defined as follows: …, Class III. Toxic. Any industrial or military material which may give off a harmful vapor, dust, fume or mist during handling or operations. The injurious effect may arise from one exposure (acute) or from repeated exposures over a prolonged period (chronic). The mode of entry into the body may be by ingestion, inhalation, or absorption through the skin.”


From American Industrial Hygiene Association, one of many guides “Long continued inhalation of asbestos dust results in a form of pneumoconiosis known as asbestosis. The primary effect of inhalation is an interstitial pulmonary fibrosis. The disease is characterized by asbestos bodies in the lungs and sputum…..There have been reports of an increased incidence of lung cancer in persons with asbestosis.’

Need prevent exposure to dust.


Study made possible by grant from Quebec Asbestos Mining Association. P. 634: “Since 1951, additional cases of cancer of the lung coexisting with asbestosis have been reported, and, according to Hueper about 100 such cases had been reported up to 1955. As a result, an association between the two diseases appears to have been accepted by many authors, and several writers were using the term ‘asbestos cancer’ of the lung…. On the other hand, not all authors accepted this alleged association without reservation.”

P. 635 – many studies lack epi techniques] P. 650: “According to the findings in this study, the mortality rate from lung cancer does not appear to increase with the length of exposure or with degree of exposure, a fact which presents strong evidence against the carcinogenicity of asbestos.”


p. 2154: smattering of federal and state actions before 1950: “ Despite these early expressions of organized sentiment for the labeling of aitches with poisonous
ingredients, many chemical products have remained essentially free of federal or state labeling regulations.” AMA has called for study of the problem

p. 2157: “Many users of hazardous substances are not likely to draw a practical distinction between ‘danger’ and ‘poison’ and even fewer are likely to appreciate the difference between ‘warning’ and ‘caution on the label. However, the use of familiar symbols such as a flame for inflammable substances or a skull for a poisonous substance are readily recognized and meaningful even to the illiterate.”

“...warning labels and the declaration of hazardous ingredients form the keystone on which to base social remedies for increasing public awareness of hazards and encouraging careful handling and storage of the many possibly harmful chemical products now available.”

AMA Committee on Toxicology has drafted a bill to serve “a model for uniform laws to require the declaration of hazardous ingredients and warning statements on the label and in the accompanying literature of chemical products.”


P. 24: “the roentgen findings in asbestosis are seen first in the lower half of the thorax. This form of pneumoconiosis is notoriously difficult to diagnose during its so-called early stages.”

P. 35: “Lynch and Pratt-Thomas indicate that the statistical evidence for a causal relation between lung carcinoma and asbestosis is strong. An opposite opinion is expressed by Jacob and Bohlig.”

P. 37 “Doll, in a study of 105 autopsies of asbestos workers, found 15 carcinomas of the long associated with asbestosis. He concluded that lung carcinoma is a specific industrial hazard of certain asbestos workers and that the average risk among men employed twenty years or longer is 10 times that experienced by the general population.”


P. 162: “It may be used by itself or combined with other materials for valve packings, gaskets, boiler lagging and pipe covering, protective clothing, shielding materials, and as automotive brake linings. In the building industry it is used in the manufacture of asbestos cement products, heat insulating, and fireproofing materials.”

Occupational Hygiene, (1959), 54-74 (Bamblin is with Turner Brothers Asbestos Co.)

1959


p. 97: “The most widely known examples of [pneumoconioses] are silicosis and asbestosis because of their serious nature and the irreversibility of the lung lesions produced.”

p. 100: Lists asbestos as a cause of asbestosis and cancer of the lung.

p. 101: “Asbestos is the only silicate which has been demonstrated to produce pulmonary fibrosis.”

p. 105: “The frequent development of lung cancers in asbestos workers is discussed later in this chapter.”

p. 106: “There is not specific chemical prophylaxis against or treatment for asbestosis. The treatment is symptomatic. Prevention depends upon dust control and the reduction of the concentration of asbestos in the atmosphere to 5 million particles per cubic foot or less.

p. 110: “Only recently has asbestos been recognized as a cause of lung cancer in workers exposed to the dust of this material. [based on 92 autopsies] 14 cases or 15 percent had cancer of the lung. The exposure time ranged from 3 to 27 years and the ages of the patients ranged from 35 to 75 years. Similar incidents of cancer has been reported in several other studies. The experimental production of lung cancer in mice by asbestos dust has also been conclusive. Twenty per cent of the animals developed squamous cell cancer originating from the bronchial mucosa.”

1959


p. 993: “dyspnea is again the dominant symptom and severity is often out of all proportion to the radiological findings…. An unusual and interesting feature of this form of pneumoconiosis is the incidence of carcinoma of the lung which has been recorded – as high as 13 per cent.”

1959


p. 4: Cites Lynch and Smith, 1935. “Although there are still those who do not agree thoroughly that asbestos was the cause of these cases, most authorities believe that the incidence of lung cancer among asbestosis cases is higher than would be anticipated.”

p. 99: summarizes 1954 Annual Report of Chief Inspector of Factories, England: “The most complete analysis of a large series of these cases is contained in the
In this report a total of 344 asbestos deaths were analyzed of which 205 were men and 139 were women. Cancer of the lung was observed in 55 or 16%. Among the men 41 lung cancers, or 20% were observed while 14 were seen in women or 10.1%.”

pp. 132-33: “the solution of the problem of lung cancer resulting from asbestos lies in the control of the problem of asbestosis. The disease is the result of prolonged inhalation of asbestos dust, so that its control lies in the control of the dustiness of the operation.” If not use respirators.

p. 133: “It should be remembered that asbestos is widely used in a wide variety of other occupations and industries. Whenever it is used in such a way that dust can be produced, and the work is of such a nature as to permit prolonged, repeated inhalation of the dust, a potential asbestos hazard exists.”


P.77: changes classifications to 3 major classifications of mineral dusts: silica, silicates, and miscellaneous. “A value of 5 million particles per cubic foot for udst containing more than 5% crystabolite ....”


P. 109: “Asbestos is the name given to a series of minerals composed of fibrous silicates of magnesium and iron which find widespread use in industry. Inhalation of the dust may cause asbestosis, a crippling form of pulmonary fibrosis, which usually does not appear until many years after the initial exposure. There is also an increased incidence of carcinoma of the lung in patients with asbestosis, and Doll (1955) estimated that the risk ... “10 times “The diagnosis of asbestosis at present depends on the history of exposure to the dust together with certain physical signs and radiological changes. The most characteristic features of the latter are a ‘ground-glass’ mottling of the lower zones of the lung fields with a shaggy border to the heart. The physical signs are clubbing of the fingers and fine metallic crepitations on auscultation of the lung bases.”

P. 119: The functional changes of asbestosis were those common to other interstitial fibroses, and consisted of a lowered diffusing capacity together with a
reduced inspiratory capacity, hyperventilation (often accompanied by arterial desaturation) on exertion with no evidence of airflow obstruction except in cases complicated by asthma or emphysema.”


Prospective Study – studied Asbestos workers and 9 other occupations. P. 1478 - asbestos workers had 2nd highest observed lung cancers to expected of the 10 occupations. – and about average for cancers other than lung. Suggests possible impact of “occupational exposure enhancing the effect of smoking.


P. 217: “These observations confirm that by the time that radiological abnormalities have developed the disease has already entered a progressive phase. Though deterioration may not be noticed if the follow-up is short it appears to be inevitable, despite removal from further exposure to the dust. This emphasizes the importance of taking every possible step to prevent the disease from occurring.”

P. 225 - “It has been shown that a low vital capacity accompanies fibrosis of the lungs of asbestos workers.”


P. 1350 Comment by Dr. Hugh-Jones: Asbestosis is a pneumoconiosis arising from the inhalation of asbestos dust during the manufacture of asbestos goods, such as asbestos sheeting, fireproof clothing, brake linings, lagging for boilers and pipes, etc.”

P. 1351: “Carcinoma of the lung is a serious and well-recognized complication in asbestosis. Its frequency in asbestosis is difficult to determine, for it is now a common condition in the general population. Moreover, exposure to asbestos may have occurred many years before the cancer develops. [cites Doll] ... Another hazard is mesothelioma of the pleura. This rather rare tumour may draw attention to the fact that a patient has worked in asbestos dust. Finally, in women who work with asbestos there is a high incidence of cancer of the ovary.”

P. 260: Mesothelioma of the pleura is regarded as an uncommon tumour. In the last four years we have seen 33 histologically proven cases; 28 of these had some association with the Cape asbestos field and four cases had been exposed to asbestos in industry.”

P. 267: “In 1924 Robertson denied the existence of primary malignant tumours of the pleura and considered them to be secondary in origin. Since then, on the one hand, Willis (1948, 1953) and Smart and Hinson (1957) have supported Robertson’s views, while on the other hand, primary neoplasms of this nature have been described by many authors in recent years. These include Tobiassen (1955) in Sweden, Belloni and Bovo (1957) in Italy, Godwin (1957) in the United States, and McCaughey (1958) in Britain. Evidence of the mesothelial origin of these tumours can be found in the tissue culture experiments of Stout and Murray (1942) and Sano, Weiss, and Gault (1950).

P. 268 – included in Dolls cases in 1955 was one mesothelioma; Cartier (1952) mentioned 2 cases from a Canadian chrysotile mine; 3 cases – 1958 – described by van der Schoot

P. 269: “Our findings suggest that mesothelioma occurs 20 to 40 years or more after exposure to dust.”


p. 511 – distinguishes between 2 types of mesothelioma – diffuse and localized.

p. 514: need correct pathologic interpretation. 2nd case had “history of long-time exposure to asbestos and the discovery of asbestos bodies in the long biopsy specimen. The etiologic association of asbestos and malignant mesothelioma has been repeatedly discussed in the literature. Not all investigators agree that exposure to asbestos predisposes to malignancy of the pleura. However, such history alerted the suspicious of the authors in the second case.” Pathologist doubted that tumor related to asbestos. “However, asbestos material could have reached the pleural tissues without being demonstrable microscopically. In addition, along the diaphragmatic surface of the pleural space there was a thick gray rind which was partially calcified at autopsy.”


Case report of a man with asbestosis with carcinoma of the right lower lobe
bronchus.

1961  

1961  

Examination of six cases. P. 279: “It is to be noted that three of the patients in this series died of lung cancer. Although great care has to be taken in interpreting isolated cases of carcinoma or small series occurring in patients with asbestosis, there seems little doubt now that there is an increased incidence of carcinoma of the lung in this disease.”

1961  

1961  

P. 19: “An abnormal relationship has been found between the permeability to carbon monoxide of the alveolar-capillary membrane and lung volume in patients with asbestosis and chronic bronchitis with emphysema.”

1961  

P. 254: study group 402 white males “Of the 63 who died of lung cancer 1 was a forty-one year old occasional cigar smoker who had worked in an asbestos factory for twenty-three years. None of the others had worked in occupations that are highly suspect in relation to lung cancer.”

1961  
**Rutherford T. Johnstone,** Letter to Editor of *JAMA,* 176 (1961), 81.

“Of the several silicates, asbestos is held in suspicion, especially in Great Britain. In the American literature there is no evidence that there is a relationship between asbestosis and lung cancer. The reason for the difference between the English and American experience is not apparent. It may be due to a difference in the type of asbestos fiber or to the total dosage.”

Response October 21, 1961, pp. 352-3: Contests Johnstone’s view: Some publications seem to indicate otherwise – cites Stoll, 1951; Isselbacher, 1953;

Johnstone Answers, p. 353: “It may be that there is a relationship between lung cancer and exposure to asbestos. However, as implied in our original answer, there is no epidemiological evidence of such among American workers.”


P. 379: “In addition to his routine duties for the last six years he had been engaged on a contract basis in applying an asbestos-base undercoating on new cars.”


P. 28: Supplements 1960 paper of 33 cases with “a description of the clinical and radiological findings in the 34 cases which we ourselves investigated.”

P. 29 Cites Lynch and Smith (1935), Doll (1955), Merewether on relationship between asbestos and carcinoma of the lung. Six cases of mesothelioma of the pleura associated with asbestosis have been described. One was mentioned by doll, 2 by Cartier, and 3 by Van der Schoot. A further 33 cases with possible asbestos exposure were described by Wagner, et al.” (1960.) Latency of between 20 and 60 years

p. 34 – history of exposure to crocidolite asbestos – asbestos bodies found in lung tissue of 30% of cases.”The findings of this and our other paper are sufficiently striking to justify further epidemiological and laboratory investigations. These investigations will attempt to determine the causative and correlative factors, including a possible relationship with asbestos or other elements....”


P. 795: “Since the first discovery reported in 1935 on the coexistence of
asbestosis and carcinoma of the lung in American and English textile workers...it has become increasingly evident that the inhalation of asbestos fibers, especially when it results in the development of asbestosis (Murray), creates an excessive liability to the subsequent appearance of a carcinoma of the lung, which not infrequently may be of multicentric origin, being derived from the multifocal adenomatoid and metaplastic bronchiolar and bronchial epithelial lesions commonly found in such lungs. The first and still most frequent data indicating excessive liability of asbestotics to develop cancer of the lung were obtained from postmortem observations. Although this material is selective, the significance of the evidence obtained from it is attested to by the consistency of its character, as shown by studies from different countries and occupational groups.”

Raises objection to methodology and analysis of Craun and Truan.

P. 797: “The causal relation between asbestosis and cancer of the lung is demonstrated by the fact that asbestosis cancer affects more often the lower lobes than the upper lobes in contrast to the general type of lung cancer which involves more frequently the upper lobes. The coexistence of asbestosis and lung cancer has been reported from the United States, Canada, England, France, Germany, Italy, Switzerland, and Finland in asbestos miners, millers, crushers, loaders, sorters, grinders, cleaners, pipe laggers, cement mixers, weavers, spinners and cement workers. Recent observations in Germany, Great Britain, the United States, Canada, Italy, and South Africa strongly suggest that asbestosis is also causally related to mesothelioma of the pleura and peritoneum (Cartier, Mancuso, (personal communication) Bohlig, Jacob and Muller, Leicher, Wedler, Koenig, Doll, Keal, Wagner, Sleggs and Marchand, Sleggs, Marchand and Wagner).”

Victims include children who had played on asbestos dumps, and who had visited mills and mines or lived near such establishments. “The evidence on hand indicates that both carcinoma of the lung and mesothelioma of the pleura and peritoneum may develop subsequent to and as the result of inhalation of asbestos dust and that such cancerous sequelae may appear after an exposure to asbestos mined in different parts of the world and differing in chemical composition. Asbestosis of the lung does not have to be well developed, according to more recently obtained evidence, in order to be a prerequisite for the development of cancerous sequelae. In fact, pleural and peritoneal mesothelioma has been observed in patients showing only a minimal amount of usually locally restricted asbestosis.”


Still 5 mppcf

p.355: “Asbestos and asbestos-rockwool cement of fiberglass mixtures may be applied as interior finishes on solid surfaces to achieve a high-fire resistant rating and to deaden sound. … Limited to use in areas where they may be protected by either their inaccessibility or additional coverings. … Applied either manually or pneumatically.” New York State Division of Industrial Hygiene not yet completed studies “have shown the presence of extremely dusty conditions” when this is applied. “High percentage of asbestos [in the air]” “the hazards produced by the pneumatic application of asbestos cement plaster are not limited to the spray operator, but may affect anyone working within fifty feet.”


“In view of the possible association between the development of mesotheliomas of the pleura and exposure to asbestos dust in people living in the Cape asbestos fields, a preliminary experiment was undertaken to see if it was possible to produce mesotheliomas in animals, by injecting various forms of asbestos into the pleural cavities.” Preliminary report of positive findings.


P. 573: asbestos used in wide variety of products – blankets, clothing, threats, rope, tape, brake linings, wall appaer, wallboard, shingles, floor covering, plastics, insulation for pipes, boilers, wire, heating pads. “Due to the rapid expansion of its use, an ever-increasing number of craftsmen and laborers are exposed to the mineral.” cites case of auto mechanic who acquired asbestosis “while undercoating vehicles.” Others may be exposed through air pollution.

P. 574 - all agree that pleural changes occur; not so often recognized. “It may be a mild self-limited disease; it may appear as a prolonged or recurrent disorder; or it may be a primary malignancy of the pleura.”

P. 577: The widespread use of asbestos makes asbestosis an important occupational health problem. This type of pneumonoconiosis is not easily recognized. A history of exposure to such mineral dust is a significant clue. …”


P. 759: “mesothelioma is now accepted by the majority of pathologists as an entity and in recent years diffuse or malignant mesothelioma has been shown to be associated with pulmonary asbestosis in Canada, Holland, South Africa, and
Germany. All these cases were primary in the pleura and in subjects who had worked with asbestosis in mines or asbestos factories, or who lived in areas where asbestos was mined or treated.”

P. 160 – 6 cases of mesothelioma in autopsy of one Hospital in 3 years.

“The enormous increase of the world consumption of asbestos, and its use in a wide variety of industrial products makes it possible for an increasing number of people manufacturing, handling, or using these products to inhale enough asbestos fibres to produce this limited basal asbestosis, without having any clinical or radiological evidence of it. In modern home asbestos may be present, from the roof, asbestos tiles or roofing, in ceilings, in floor tiles, .... etc] It does indicate however, how widespread is the use of asbestos today, and while the number of cases of classical asbestos today, and while the number of cases of classical asbestos with pulmonary signs and symptoms may not significantly increase, we may have a marked increase in those with the limited basal asbestosis described, and of mesothelioma of the pleura and peritoneum in people who have occupations in which association with asbestos is not suspected.”


“Investigations in South Africa and recent inquiries in Great Britain and elsewhere have indicated that there may be an association between diffuse mesotheliomas of the pleura and peritoneal cavities and exposure to asbestos dust. There appears to be no correlation between the severity of any pulmonary asbestosis and the occurrence of these tumours. In a number of cases the exposure to asbestos dust appears to have been minimal.... However, a detailed occupational history has, in nearly all cases, revealed some contact with asbestos fibre.” Wants more information.


Response to previous letter: describes a number of cases of mesotheliomas. “In most of these cases exposure to asbestos was not continuous or prolonged and in some instances there was a considerable gap between the last exposure and the onset of symptoms due to the tumour.”

1962 McNykty, J.C., “Malignant Pleural Mesothelioma in an Asbestos Worker,”

Worked in an asbestos mine first in 1948 – died of malignant pleural mesothelioma in 1961 – 13 years later. “There appears to be a relationship between exposure to blue asbestos and the development of the pleural mesothelioma in this case.” short exposure period “confirms an impression received from Wagner, et al (1960, 1961) that these tumours may arise after transitory exposure to crocidolite in susceptible persons.”


P. 1131; “In view of the fact that the role of asbestosis in the pathogenesis of carcinoma of the lung remains unproved, we felt it would be worthwhile to review the existing evidence and present clinical and pathological findings on the cases we have seen. Reviewed 11 cases from cases the authors have seen as well as cases from the Armed Forces Institute of Pathology.

P. 1185 – 4 of individuals “had no known history of direct exposure to asbestos dust.” asbestos used in manufacture of many products. “the fact that no known exposure existed in 4 cases indicates that the disease can occur without prolonged exposure to known sources.” Average time from initial exposure to development of asbestosis is 9 years; for cancer it is 15 years “with latent periods of from 15 to 22 years between the initial exposure to the asbestos dust and the development of a malignant tumor.”


p.38: contrasts the TLVs which is a time weighted average with the American Standards Association Z-37 Committee, Maximum Allowable Concentrations which are peak concentrations, not averages.

p.39: “We know so little about the causation of cancer by most substances that it may be prudent to limit the concentration to a form of zero, the smallest amount which can be analytically estimated.”

1962 J. G. Thomson, Letter, “Exposure to Asbestos Dust and Diffuse Pleural Mesotheliomas,” The Lancet,

Found several cases of mesotheliomas in US.

“In very few of these cases was an industrial or occupational exposure to
asbestos mentioned, but this is of no significance unless direct questions are put to patients or their relatives. What is more important is that the world’s annual consumption of asbestos has increased from 500,000 tons in 1942 to 2,400,000 tons in 1961, and that asbestos is today used in such a wide variety of manufactured products that, theoretically at least, the number of people who may be exposed to asbestos is enormous, and few of those thus exposed work in factories which are designated asbestos factories.”


P. 210: Takes a cohort approach (cohort of workers who were employed in this company at any time in 1938 or 1939) with a company in Ohio “in which the principal exposure was to asbestos, in the manufacture of asbestos products....” Study deaths from 1940 to mid-1960

P. 213: The data on underlying cause of death as recorded on the death certificate showed notable excesses of observed over expected deaths at ages 25-64 years for 2 major chronic illnesses, neoplasms and asbestosis.”

P. 216: the greatest contribution to the excess mortality due to neoplasms as an underlying cause in the period 1940 to mid-1960 was for cancer of the lung, bronchus, and trachea....” Also true for malignant neoplasms of the digestive system and peritoneum.

Conservative estimates.


P.65 – “asbestosis, which is a fatal occupational disease is still occurring 30 years after steps were taken to eliminate the disease. This is because of expansion of the industry and especially of the insulating uses of asbestos.” Need find a substitute for asbestos. “Until such steps are taken this essentially preventable disease will continue to cause unnecessary death and suffering.”


P. 91: Physiological effects; Reactions ”a. The cardiopulmonary reaction which consists of the pneumoconioses, such as silicosis and asbestosis.”

Wagner (1960 [Proceedings of the Pneumoconiosis Conference, Johannesburg, 1959, p. 353]) first suggested an association between mesotheliomata and exposure to asbestos, and this study confirms this association.” used control group.

P. 278: “Summary” “The association of these tumours [mesos] with asbestos bodies in lung tissue is confirmed. Many of the cases gave no history of industrial exposure, and it is possible that temporary or relatively trivial exposure may have occurred. Once symptoms from the tumours have arisen, the prognosis appears to be measure in months rather than years.”


P. 22; Abstract” “Building trades insulation workers have relatively light, intermittent, exposure to asbestos. 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; ...[high incidence]

P. 25 "The results with regard to carcinoma of the lung are clear. Industrial exposure to asbestos by insulation workers, as studied here, results in a marked increase in the incidence of cancer of the lung, approximately six to seven times the expected incidence.” Not have figure on incidence of mesos, “Nevertheless, the growing number of reports of individual cases suggest that these tumors are perhaps becoming relatively frequent complications of asbestos exposure.”

P. 25: “the incidence of more than 1% of deaths from pleural mesothelioma is strikingly high for a tumor which is generally considered to be extremely rare.’

Question of Environmental exposure and “also other workers at job site.” Thus for example, insulation workers undoubtedly share exposure with their workmates in other trades; intimate contact with asbestos is possible for electricians, plumbers, sheet-metal workers, steamfitters, laborers, carpenters, boiler makers, and foremen.”


P. 318: “... cases both of asbestosis and neoplasia associated with asbestosis
have been recorded following the minimal exposure of community Environment.”

Questions adequacy of 5 ppmcf standard.

1964


P. 107 –“3,000 specific conditions in which asbestos serves uniquely as a protectant against excessive heat, cold and corrosion. There are, therefore, a multiplicity of circumstances under which asbestosis can be encountered.”

No agreed on safe limits – some 5 million; some 1 million; some 10 million.

1965


“The main types of asbestos of commercial interest are amosite, anthophyllite, chrysotile, crocidolite, and tremolite. There is evidence of an association between exposure to asbestos and malignant neoplasia. This has been established mainly on information from Germany, Italy, South Africa, the United Kingdom, and the United States of America, the types of tumours which have been shown to be associated with exposure to asbestos dust are (1) carcinoma of the lung, and (2) diffuse mesothelioma of the pleura and peritoneum. There is some suggestion of an association also with gastro-intestinal carcinoma, and possibly ovarian tumours. The latent period between first exposure to the dust and detection of the related tumours is many years, usually 20 or more. Instances up to 60 years have been reported. For this reason, further cases of these associated tumours are expected to occur for many years to come, even if dust exposures are now greatly reduced. Present evidence indicates that the associated carcinoma is not limited to exposure to any one type of asbestos fibre. However further investigations are urgently needed to establish whether the degree of risk is importantly related to the type of fibre inhaled. In the case of mesotheliomata, evidence from several countries suggests that exposure to crocidolite may be of particular importance, but it cannot be concluded that only this type of fibre is concerned with these tumours, and further investigation of this problem is needed.”

1965

Keynote for conference:

p. 9: “Asbestos can truly be called the 20th century mineral. Its output has increased over a 1000-fold in 60 years, compared with a mere 50 fold for oil – an industry often regarded as the symbol of industrial growth.”

P. 11: Early research shows “that a disease very like asbestosis can be produced in animals and that some of the tumors that occur in man can also be produced in animals.”


Chrysotile – 90% of total asbestos output

p. 14: Good chart of characteristics of 6 different types of asbestos.

P. 18 – major uses of asbestos – textile; cement products (2/3 of total); friction materials and gaskets; paper; floor tile; paints, roof, caulsks, plastics.

G.W.H. Schepers comment p. 21: “I believe, however that it should be stressed that the medical and Health problem originating through exposure to asbestos dust may come about equally through minor uses of asbestos.”


P. 184: Numerous reports by many investigators from various countries establish the fact that an occupational or nonoccupational inhalation of dust of different chemical and physical types of asbestos results in the development of asbestosis of the lungs which, depending upon the type of exposure may be designated as an occupational, neighborhood or household pneumoconiosis. Since 1935 an increasing amount of epidemiologic, clinical and pathologic evidence, moreover, incriminates this health hazard as one of the Environmental sources of cancers of the lung, and more recently also of mesotheliomas of the pleura and peritoneum, although some commercially interested parties and their medical guardians and protectors still prefer for their own reasons and motives, to deny the existence of these dangerous and usually fatal sequelae of a respiratory contact with asbestos dust. The obvious fallacy of such allegations is well demonstrated by the remarkable and a progressive rise in the number of cases of asbestosis and cancers of the lung, pleura, and peritoneum reported from various countries during the past two decades.”

P. 186: world production of asbestos increased form 500 tons in 1880;
330,000 tons in 1925; 446,000 metric tons in 1938; over 2,000,000 in 1958. “Main producing country is Canada, while the principal consuming country is the United States of America. Chrysotile represents 95% of total asbestos produced.

P. 187: “asbestosis and asbestos cancer hazards related to an inhalatory exposure to asbestos exist not only for asbestos workers proper ... but also for the large number of individuals who may sustain such contacts on an incidental basis. Such persons may be employed permanently or temporarily in or near operations where asbestos and asbestos products are produced or handled and where they inhale...”

P. 189 – total number between 50,000 and 100,000.

P. 188 – Table 2 – “Operations and Products with contact to asbestos for producers, processors, users, consumers, residents” – wide variety; Occupational groups; nonoccupational groups

p. 191 – “enormous rise in the production and industrial use of asbestos during the past 50 years; harmful effects ...reflected not only in a growing number of Reports on the occurrence of asbestosis and asbestos cancer in a variety of asbestos workers but also in the fact that such Reports originate from a rising number of countries....”


p. 364:”Roentgenographic study of 1,117 asbestos insulation workers showed pleural calcification to be a common finding. Altogether, 150 instances of such calcification were present..... rarely occurs in less than 20 years from onset of exposure. ... the extent of calcification also increased with duration of exposure.”

P. 365: The pleural calcification is primarily found in the parietal pleura, more heavily in the lower portions of the chest. ... Pleural calcification not infrequently is present in the absence of visible pleural fibrosis and occupationally may be present in the absence of radiological visible parenchymal fibrosis.”


Work on asbestos insulation workers in US work with union of insulation workers in New York and New Jersey. (International Association of Heat and
Frost Insulators and Asbestos Workers.)

P. 152: Investigation of 1522 asbestos insulation workers in NY-NJ metro area.” of 392 with 20+years from onset of exposure, “radiological evidence of asbestosis was found in 339…. [among deaths.] Lung cancer was found to be at least seven times as common as expected and cancer of the gastrointestinal tract three times as common as expected. There were 10 instances of mesothelioma of the pleura or peritoneum.”

“We may conclude that asbestosis and its complications are significant hazards among insulation workers in the United States at this time.”


Examine “whether mesothelioma of pleura and peritoneum had an important relation with asbestos exposure in the United States.” “If mesothelioma could be found with increased frequency in association with asbestos in this country it would demonstrate that this tumor was another neoplastic hazard of asbestos exposure in general and not limited to one area or to one type of fiber.”

P. 563: “Lung cancer has previously been demonstrated to be a frequent complication of asbestos exposure among insulation workers in the United States…. Ten deaths from mesothelioma among 307 consecutive deaths from mesothelioma among 307 consecutive deaths is an extraordinarily high rate and permits the conclusion that this disease is an important complication of asbestos exposure. This conclusion, moreover, refers to such exposure in the United States, under working conditions of the recent past, with relatively light and intermittent asbestos exposure. [p. 564] it indicates, too, that mesothelioma may not necessarily be entirely a problem of only one kind of asbestos (crocidolite) and that it is surely not limited to South Africa.”

P. 565: “These examples of indirect occupational exposure and community exposure raise the important question whether mesothelioma will be found to be a community problem in addition to that existing with industrial exposure to asbestos.”

“Our study of 307 consecutive deaths (1943 - 1964) among asbestos insulation workers in New York and New Jersey disclosed 10 deaths caused by mesothelioma of the pleura (4 cases) or peritoneum (6 cases). This is an extraordinarily high incidence for a tumor generally so rare that it is not separately
coded among causes of death.”

“It appears that mesothelioma must be added to the neoplastic risks of asbestos inhalation, and joins lung cancer (53 of 307 deaths) and probably cancer of the stomach and color (34 of 307 deaths) as a significant complication of such industrial exposure in the United States.”


This data extension of previous data – experience of 632 men from NY and NJ. P. 524: “Taken at face value, it would appear that asbestos workers have an abnormally high risk of dying of gastrointestinal cancer. However, we will refrain from drawing conclusions on this matter at the present time.”

P. 522: Altogether there were 45 deaths from cancer of the lung and pleura compared with 6.6 expected; a mortality ratio of nearly 7 to 1.”

P. 524 - 5: “Obviously, light exposure to asbestos dust does not lead rapidly to pulmonary neoplasia. Equally obviously, this disease is unlikely to appear if workers die at a young age due to some other effect of asbestos exposure. However, with light exposure and the availability of antibiotics for the treatment of infectious disease, neoplasia is a major cause of death of such workers.”


P. 382: “Clinical experience in Boston is used here to report to the Association that all three of these harmful effects of asbestos are seen in the United States.” fibrosis, bronchogenic carcinoma, and “malignant mesothelioma among workers and neighbors.” uses 4 cases

P. 388: “As total exposure to asbestos was lowered, bronchogenic carcinoma appeared as a complication of less severe asbestosis. More recently, with greatly increased us of asbestos, risk of pleural mesothelioma (and perhaps intra-abdominal tumor) in worker snad neighbors appears at exposures lower than
those productive of fibrosis.’

1965


P. 130: “This Article describes a patient with pleural calcification followed by mesothelioma of the pleura. Ten years previously, after twenty years in charge of the stores of a firm engaged in distributing a variety of asbestos insulating materials for industry and the building trade, he was transferred to the office and had no further contact with asbestos.”

P. 131: “More recently attention has been drawn to the development of other types of neoplasm in patients exposed to asbestos .... [cites Hourhane] Owen (1964 studied 17 cases of diffuse mesothelioma in the Merseyside area, of which 16 were pleural, and found a history of exposure to asbestos in 14. Fowler, Sloper and Warner (1964f) described 2 cases of mesothelioma of the pleura in whom the lungs contained asbestos bodies and there was an occupational history of exposure to asbestos....[cites Selikoff, Churg and Hammond] Elmes, McCaughey, and Wade (1965) obtained a history of occupational exposure to asbestos in 32 out of 42 patients with mesothelioma of the pleura in Belfast. In 42 matched control patients of the same age and sex a history of exposure was obtained in only 9. As originally observed by Wagner, et al (1960) an interesting feature of many of the reported cases, in common with the patient described above, was that the exposure to asbestos was slight in degree and remote in time.”

1965


1965


P. 266: There seems little doubt that the risk of mesothelioma may arise from both occupational and domestic exposures to asbestos.”

1965


Cites cases of asbestosis in variety of occupations, including insulating, and brake lining:

Also of the 247 cases, 59 had died with 21 dying from cancer of the lung
and 3 from mesothelioma – 2 of the pleura and 1 of the peritoneum.

“In addition to the figures of death from lung cancer, four of the survivors in the series are at present permanently incapacitated by asbestos and pleural effusion. In two, lung cancer has been diagnosed, and in the other two mesothelioma of the pleura is suspected.”


P. 3 – “A four-year investigation on the effects of the inhalation of chrysotile asbestos dust on the lungs of hamsters, rats and guinea pigs is nearing completion. In addition to the determination that the fibrogenicity resulting from chrysotile dust approximates that of quartz dust, it was found that asbestosis in hamsters is a progressive, nonhealing, and fatal disease; whereas in rats, the disease is nonprogressive and heals. Also, it was found that 2 percent of the asbestotic rats developed primary lung cancers. One of these tumors was a malignant primary pleural mesothelioma. (Sponsor: U.S. Public Health Service.)


P. 563; “the most striking finding of this study was that of the 42 mesotheliomas which came from a geographical area of approximately 30,000 sq. mi. six were clustered in and around an insulation plant (figure [which shows “Occupational, neighborhood, and family contact patients with mesothelioma associated with one insulation plant.” - p. 562]). These included two insulation manufacturing workers, three neighborhood cases and one family contact – the daughter of an insulation worker.”

Studies 52 cases of mesos reported from 152 hospitals over a five year period.


p.307: “Another 13 inorganic substances comprise the insoluble dusts, which are largely retained in the lungs and whose affects are mainly confined to that organ. They include silica, asbestos, mica and other minerals.”


p. 248: “The Ministry of Defense (Navy) took steps to reduce the hazards associated with asbestos. These steps included a review of the use of asbestos, and where possible, the introduction of substitute materials.”

p. 259: “High-temperature jointing and packing materials. Asbestos fiber and compressed asbestos fiber. No substitute heat-resistant material is available. No health hazard in forms used in shipyard applications.” [no evidence provided for this]

p.250: “Precautions to be observed by all dockyard employees working with asbestos…. To isolate asbestos work… to reduce the amount of dust created by asbestos work by improving work methods and the materials themselves. To protect all workers whether they work directly with asbestos or not….”

p.253: “From the present survey it is clear that all processes involving work with asbestos insulating materials in Naval Dockyards give rise to asbestos dust concentrations of more than 2 fibres/cm³. Many processes have dust concentrations of 50 fibres/cm³ or more. Despite the improvements which have been made towards reducing the asbestos dust concentrations in ship repairing it is obvious that personal protection for the [p254] workers will have to be provided for many years….”

1972


Recommends standard of 8 hour TWA airborne concentrations of asbestos dust not exceeding five fibers longer than five micrometers per millimeter.”

“It is concluded that there should be one minimum standard to asbestos applicable to all workplaces exposed to any kind, or mixture of kinds, of asbestos.”

1972


P: II-2: “The recommended standard is designed primarily to prevent asbestosis. For other diseases associated with asbestos, there is insufficient information to
establish a standard to prevent such diseases including asbestos-induced neoplasms by any all-inclusive limit other than one of zero.”


Register of meso cases maintained by the Department of Employment, Medical Services Division. Paper describes investigation of 413 notifications to the Register in 1967-68 from England, Wales and Scotland.

P. 103: The observed incidence of definite mesothelioma in this series was approximately 120 per year. For the reasons stated above this figure may considerably understate the true incidence. In this study the briefest occupational exposure to asbestos associated with mesothelial tumour was three weeks, but if asbestos was a cause of mesothelioma it cannot be assumed that lesser exposures are safe:


P. 8: “Thus, by 1935, the main directions of the problem were known. Chrysotile asbestos, virtually the only fiber then used, could produce widespread disease, this disease could be fatal and malignancy might be a result of exposure.” But then inattention and use of asbestos grew fold. Became clear that principal problem was cancer – “Two critical studies showed this to be true, both in the factory production of asbestos products [Mancuso, 1963] and among workers using these materials [Selikoff, 1964].” p. 10 “In our experience, approximately 40% of deaths among asbestos insulation workers in recent years have been due to malignancy.”

P. 11: “lung cancer, mesothelioma, and gastro-intestinal cancer were the principal categories of such excess neoplastic deaths.” p. 14: “it may take much less asbestos to cause cancer than to result in asbestosis – sometimes, very little indeed.” P. 15 – long latency – frequently cancers “do not become clinically evident for at least 20 years after onset of exposure.” p. 16: “Even brief exposures – a day, a week, a month – if excessive can later result in disease.” If smoke risk of death of lung cancer is approximately eight times that of other smokers.”


p.92: “Available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have occurred following exposure to chrysotile, crocidolite, amosite, and anthophyllite. Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have developed also in individuals living in the neighborhood of asbestos factories and near crocidolite deposits, and in persons living with asbestos workers…. 

“Likewise, all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or IP injection. Mesotheliomas and lung cancers were induced following even 1 days exposure by inhalation.

“The size and shape of the fibers are important factors; fibers less than 0.5 µm in diameter are most active in producing tumors…. 

“There are data that show that the lower the exposure, the lower the risk of developing cancer. Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a ‘safe’ level of asbestos exposure.”

p.93: “This recommended standard of 100,000 fibers ›5µm in length/m³ is intended to (1) protect against the noncarcinogenic effects of asbestos, (2) materially reduce the risk of asbestos-induced cancer (only a ban can assure protection against effects of asbestos) …. ”


Section 1304.2, “Purpose… These products present an unreasonable risk of injury due to inhalation of fibers which increase the risk of developing cancer, including lung cancer and mesothelioma, diseases which have been demonstrated to be caused by exposure to asbestos fibers.”

1304.5 Findings. “The Commission noted that in the scientific literature, there is general agreement that there is no known threshold level bellow which exposure to respirable free-form asbestos would be considered safe…. For purposes of this assessment, the Commission considered the use of patching compounds by the consumer, for six hours a day four times a year, to be a high yet reasonably foreseeable exposure.”

P. 1061: Article assesses “the possibility that household exposure might be a major risk factor for females with malignant mesotheliomas.”

P. 1063: “Our results suggest that women run the risk of malignant mesothelioma not only if they work in asbestos-related industries but also if they are indirectly exposed to Environmental asbestos. The husband’s occupation was clearly the most important risk factor, but 6 of our patients also lived in environments where more than one source of exposure was likely. The importance of other non-occupational exposures varies with types of industry (e.g. asbestos mines and factories, shipyards, etc) present in an area and their proximity to homes.”