

**ENVIRONMENTAL
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FUND**



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June 27, 1973

Honorable Sherman Gardner
Acting Commissioner of Food and Drugs
Department of Health, Education and Welfare
Washington, D. C. 20204

Dear Sir:

The undersigned Petitioners, Center for Science in the Public Interest and Environmental Defense Fund, submit the attached petition pursuant to sections 409, 701(a) and other provisions of the Federal Food, Drug and Cosmetic Act with respect to the issuance of regulations necessary to protect the public health from adulteration of foods and drugs with asbestos particles. The Commissioner's authority to issue the requested regulations is found in sections 201(s), 301(a), 402(a), 409, 501(a) and 701(a) of the Act.

Attached hereto in quintuplicate and constituting a part of this petition are the proposed regulations in the form proposed by Petitioners and a statement of the grounds upon which the Petitioners rely for the issuance of the regulations.

Very truly yours,

Scott H. Lang

Scott H. Lang

Attorney for Petitioners

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ASBESTOS AND YOU

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**Center for Science in the Public Interest
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1973

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TABLE OF CONTENTS

<u>Abstract</u>	1
<u>Asbestos, Friend or Foe?</u>	3
I. <u>Health Effects:</u>	8
Asbestosis, the First Asbestos Disease	8
Lung Cancer from Asbestos	10
Mesothelioma from Neighbors and Relatives	16
Other Cancers	26
Asbestos Bodies in the Streets	27
II. <u>Environmental Aspects:</u>	30
Urban Air	30
Asbestos in Buildings	31
Asbestos in Personal Items	32
Asbestos in Food, Drinks, and Drugs	35
<u>Conclusion and Recommendations</u>	38
<u>Appendix 1</u> Letters to Environmental Protection Agency on Asbestos in Air Ducts in Buildings	44
<u>Appendix 2</u> Letter to Food and Drug Administration on Talc Use	47
<u>Appendix 3</u> Letter to the Environmental Protection Agency on Air Pollution Standards	48
<u>Appendix 4</u> Letter to Labor Department on Occupational Standards	49
<u>Table I</u> Uses of Asbestos	50
<u>Table II</u> Consumption and Production of Asbestos in the U.S.	51
<u>Table III</u> Population Groups with Occupational and Environmental Exposure to Asbestos	52
<u>Table IV</u> Uses of Talc	53
<u>References</u>	54

ABSTRACT

Asbestos has become a widely used material in our industrial society over the last 100 years. Unfortunately, we have found that there are grave hazards attendant upon its use. Severe lung scarring, which can lead to death from heart failure, results from long-term, heavy exposure (asbestosis). With the implementation of basic dust controls in asbestos processing plants, it was hoped that the workers' hazard would be eliminated. However, as asbestosis became a less severe problem, a startling incidence of lung cancer and stomach cancer appeared among asbestos workers. In the late 1950's an epidemic of an extremely rare cancer was discovered and traced to environmental asbestos exposure (malignant mesothelioma). As these diseases were associated with successively lower exposures, the populations known to be at risk have increased. Even while this information was being published, new and dangerous applications of asbestos were developed, the worst of which was sprayed fireproofing insulation. This alone exposed millions of American construction workers to high concentrations of asbestos during the 1960's, as well as neighbors and passers-by to a lesser extent. It is now established that urban dwellers with no history of occupational exposure to asbestos have asbestos fibers and scarring in their lungs. Whether this significantly increases their cancer risk will not likely be known for decades, due to the long latent periods of these diseases and the inability of past studies to define the dose-response relationship.

The purpose of this report is to stimulate a greater awareness of asbestos hazards, and generate actions which will reduce and eliminate these hazards. At this time, the many government agencies which should regulate uses of asbestos-- The Labor Department's Occupational Safety and Health

Administration, HEW's Environmental Protection Agency and Food and Drug Administration, Interior's Bureau of Mines, and the Federal Trade Commission are all failing to take appropriately strong measures. In many cases, the hazards can be eliminated by substitution of less dangerous materials or reduced by implementing safer work practices.

ACKNOWLEDGMENT

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Asbestos, Friend or Foe?

This report deals with the health and environmental effects of asbestos. We Americans have shown far more skill and ingenuity in using asbestos in our consumer products than we have in safeguarding ourselves from this toxic material. We know that asbestos is both useful and toxic, but we don't have dose-response information on low levels of environmental asbestos exposure. There is evidence that asbestos exposure in combination with other hazardous substances carries severe risk of cancer. Since surveillance techniques are extremely tedious or primitive, no one has yet defined the concentrations and distribution of asbestos particles within the environment.

We show great ingenuity in using asbestos in homes, trains, airplanes, ships, farms and factories. Fortunately, a part of the asbestos used is more or less harmless due to the lower possibility of erosion of the fibers from such items as ceramic products and plastic and tile products. However, other uses are extremely dangerous due to the fact that asbestos is not held tightly in the product or on the applied surface. Examples are asbestos cloth, rope, paper and insulation materials.

For ten years we have known that brief and low level exposures to asbestos can cause cancer. Yet this material continues to be incorporated into hundreds of new products, with a blessing and a hope that it is safe. Until recently hazard labeling of asbestos products, such as wallboard sold in hardware stores, was not required. The present situation is not much of an improvement, due to the foot-dragging by enforcement agencies, weak regulations and actual loopholes in the law.

What is this mysterious substance? Asbestos is an economic term, not a scientific one. It refers to a group of hydrated silicate minerals that separate readily into fibers. These are divided into two exotic groups:

serpentines (chrysotile) and amphiboles (crocidolite, amosite, tremolite, actinolite and anth_ophyllite). About 95% of the asbestos that we use is in the form of chrysotile.

The major producers of asbestos are the Soviet Union and Canada, which account for 44% and 34% of the world output respectively (all chrysotile); and the Republic of South Africa, which produces 6% (mostly crocidolite and amosite). The United States produces only 3% of the asbestos but is the largest consumer using about one-fourth (1,2).

Asbestos has been known to man for thousands of years. Archeologists have found that asbestos was in general use as much as 4,500 years ago in Finland, as a cementing agent in the preparation of clay pottery (3). Herodotus (450 B.C.) described the Romans mining asbestos in the Italian Alps. He also described a cremation cloth made from it.

Pausanias, when speaking about the golden lamps made around 430 B.C. by Callimachus mentioned the wicks of Carpasian flax which were incombustible by fire. Strabo, Solinus and Plutarch referred to woven cloths that did not burn and the "ever-burning lamps" in the Grecian temples. Asbestos is derived from the Greek word meaning inextinguishable (3a).

Pliny (A.D. 50) referred to difficulties weaving asbestos and the use of respirators to avoid inhalation of its dust. Charlemagne (800 A.D.) had a tablecloth made from asbestos (4); he would impress his enemy warriors by cleaning it by passing it through a fire. Maybe an investigation of his tomb would reveal that asbestos particles were implanted in his lungs. Marco Polo (about 1250 A.D.) wrote that he had seen Tartars using cloth that withstood fire (5).

In spite of its long history, it has only been in the last century that asbestos has become more than a mere curiosity. Gradually the heat resistance

characteristics have made this substance useful. World production has expanded a thousandfold since 1900, with a steep growth rate since 1940. People learned that asbestos had other characteristics such as a reinforcing binder in such products as portland cement, rubber and plastics. These expanded uses have resulted in the following consumption figures: 500 metric tons in 1880; 330,000 tons in 1925; 446,000 tons in 1938; over two million tons in 1958; 3.9 million tons in 1966 (1,6).

Both because of its heat resistance and spinnability, asbestos has a use in textiles. Some better-known products of the asbestos textile industry are safety clothing, lagging cloth, woven brake linings, clutch facings and electrical insulation. (See Table I and II on uses)

Cement products account for the major use of asbestos (some estimate that 70% of the world's production of all types of asbestos is used in cement building materials). The cement product is rigid, strong, weather-resistant and heat-insulating. The sheet products are such items as shingles, electrical switchboard panels, and laboratory table covers. Asbestos cement is used also in making drain pipes and pressure pipes, water and gas mains, sewage pipes, venting flues, and for electrical cables and mining operations. It is estimated that in some countries 50% of the drinking water is supplied in asbestos cement pressure pipe (5a).

The second highest use of asbestos is in vinyl and asphalt floor tile. Likewise, it is used as an inexpensive filler in paints, roof coatings and caulks. Miscellaneous uses include gaskets, sprayed insulation for building construction, undercoating for automobiles, asphalt paving and curbing, welding rod coatings and filter media (6,7).

It is estimated that about 85% of the asbestos consumed currently is used in applications in which the fibers are "locked in" or tightly bound

and not able to become airborne to any significant extent (8). In 1959 there were over 3,000 industrial applications of asbestos, and the list is rapidly growing (9).

Differences among the types of asbestiform materials are important in terms of their applications as well as in their biological effects. Chrysotile is flexible, has relatively high tensile strength and has far better spinnability than all other types. On the other hand, chrysotile is chemically unstable in the presence of dilute acids and even distilled water. An aqueous solution with pH under 10.8 will leach magnesium from the fiber (10). Acid resistance of the other major types, crocidolite and amosite, is good. Crocidolite has the greatest tensile strength of the three, but has poor heat resistance (11).

Chrysotile fibers may be curved and can break down into elementary fibrils as small as 200-400 $\text{\AA}$ in diameter. The unit fibrils are susceptible to attack by body fluids. Fibers of the amphibole types are straight, and do not break down in similar fashion (12). The smallest amphibole fibers are 800-1,000 $\text{\AA}$ in diameter (13).

There is a wide variability in trace metal content of the asbestiform minerals. It is noteworthy that several metal complexes, especially those containing iron, have been shown to induce cancer; and several metals, including chromium, nickel and lead have been proven to be carcinogenic. Special attention has been called to high nickel and chromium content of chrysotile, and the manganese content in crocidolite and amosite (14,15). Iron is a major constituent in crocidolite and amosite.

Traces of primary oils occur in crocidolite and amosite (chrysotile has none). Commercial fibers of these amphibole types (i.e., after possible contamination by secondary oils) have low levels of polycyclic hydrocarbons in their oils. Jute bags used to transport and store asbestos fiber are a

Section I Health Effects

There are a number of diseases which have been shown to have been caused by asbestos exposure. It is noteworthy that by 1918, American life insurance companies routinely declined to insure asbestos workers (3b). This was six years before Cooke reported the first case of asbestos disease to appear in the general medical literature.

Asbestosis, The First Asbestos Disease

The first recorded case of asbestos disease was reported by Murray in 1906 (4). His patient was the last of a group of ten asbestos cardroom workers to die before the age of 35 of pulmonary disease, presumed to have been tuberculosis. On the postmortem examination diffuse fibrosis of both lungs was discovered and attributed to asbestos exposure. It was not until 1924 that a second case of asbestosis was reported by Cooke (16). In 1927, Cooke cited "curious bodies" in the lungs of his subject, a female asbestos textile worker who died at age 33 (5). The unique in situ reaction to asbestos fiber was called the "asbestosis body" by Stewart and Haddow in 1929 (17). That year Cooke wrote that he considered the curious bodies to be pathognomic of pulmonary asbestosis if found in any numbers (18). In 1930, Merewether and Price published a report following a thorough investigation of conditions in England's asbestos manufacturing industry. On the basis of 363 workers examined, they concluded that one in eight asbestos workers would have evidence of fibrosis. They made a number of recommendations concerning improved ventilation and dust suppression measures (19). Regulations were drawn up shortly thereafter and became effective March 1, 1932, markedly reducing dust levels in asbestos plants (20).

In the United States, Dreessen and co-workers made a Public Health

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Service study (21,22) of asbestos textile mill workers. A high incidence of definite clinical and roentgenographic evidence of asbestosis was found in persons who had had 5-10 years of exposure to dust levels in excess of 5 million particles per cubic foot (mppcf). The authors pointed out that methods for controlling dust levels below that threshold existed for most processes. Since only three doubtful cases of fibrosis were found among those with milder exposure, the authors concluded that "5 million particles per cubic foot may be regarded tentatively as the threshold value for asbestos dust exposure until better data are available." A serious flaw inherent in this study was that the average age of the workers was only 32.1, and more than one half were under 30 years old.

The British Occupational Hygiene Society, in setting its Standards for Chrysotile Asbestos Dust in 1968, spoke in terms of the workers cumulative lifetime dose. The recommended maximum accumulated exposure (100 fibers/ml-years, fibers longer than 5μ) corresponds to about 15 mppcf-years for asbestos textile mills (23,24). This means a maximum of 2f/ml-years for 50 years, 10 f/ml-years for 10 years, etc. This cumulative level of exposure is said to reduce the risk of contracting asbestosis to one percent among those who have a lifetime's exposure. "By 'asbestosis' this committee means the earliest demonstrable effects on the lung due to asbestos." In the final paragraph, the Society admitted that it is not possible at present to specify a safe threshold for asbestos-associated cancers.

The threshold limit value (TLV) for time-weighted average 8-hour exposure officially recommended by the American Conference of Governmental Industrial Hygienists in 1970 was 5 mppcf (25). In 1968, ACGIH published notice of intended change to lower TLV to 12 fibers/milliliter > 5 microns

in length or 2 mppcf; and in 1970 they published a limit of 5 f/ml $> 5\mu$ as a notice of proposed intended change. On December 7, 1971, the U.S. Department of Labor adopted an emergency standard of 5 f/ml $> 5\mu$ effective immediately. The Public Health Service recommended in January of 1972 that a standard of 2 f/ml $> 5\mu$ be effected in mid-1974 (25a). In June of 1972, the Department of Labor announced that the 5 f/ml standard would remain in effect until July 1, 1976, at which time a 2 f/ml limit would be in force (25b).

Pulmonary asbestosis is a slowly progressive disease. It may not be readily apparent on roentgenograms in early stages, and sometimes not even when extensive. Its classical symptom is dyspnea. In addition, finger clubbing, cyanosis, and extensive fine basal rales tend to develop after a long period from onset of asbestos exposure (26). Severe fibrosis can ultimately lead to death from cardiac failure. The pathogenesis of the fibrotic lung lesions is still not quantitatively understood (27).

It is known that the disease progresses long after cessation of exposure, once established. It has occurred in workers with short, heavy exposures (20,26,28,29). In general, changes indicative of clinical disease are rarely manifested less than 10 to 20 years after onset of asbestos exposure (26,28).

Improvements in dust control have resulted in decreased mortality from asbestosis and longer life expectancy for workers in asbestos processing plants.

Lung Cancer from Asbestos

In 1935, attention was first called to the coincidence of asbestosis and lung cancer (also a rare disease at that time) in a single case (30). There followed a number of clinical reports, but the possibility that asbestos exposure carried an elevated cancer risk was not generally taken seriously

before 1947. Merewether found that in Great Britain, of 235 deaths with asbestosis then on record, 31 (13.2%) also had lung cancer. Merewether contrasted this with a frequency of 1.32% lung cancer in 6884 cases certified at death as having silicosis (31). In 1951, Gloyne (32) published a review of 1205 necropsies on persons who had worked in dusty occupations. He found lung cancer in 14.1% of necropsies on subjects with asbestosis (17 out of 121), against 6.9% in silicotics (55 out of 796) and 8.3% in 169 with no pneumoconioses.

Doll (33) found that among 105 asbestos factory workers examined at necropsy from 1935-53, 18 had lung cancer. A follow-up study of 113 men with at least 20 years' exposure showed that 39 deaths had occurred (against 15.4 expected in the general population). The excess was entirely due to lung cancer (11 against 0.8 expected) and other respiratory and cardiovascular diseases (22 against 7.6 expected). Doll concluded that the average lung cancer risk among men employed 20 years or more was of the order of 10 times that experienced by the general population. He noted that these men had had years of exposure prior to implementation of dust controls in 1932. Including Doll's cases, Hueper (34) listed a total of 99 cases of asbestosis lung cancer reported in the literature (1955).

In 1958 Braun and Truan (35) published a study of lung cancer among Canadian chrysotile miners. In their opening remarks they reviewed critically the case for a casual relation between asbestos and lung cancer. They first cited studies by Saupe (no lung cancer among 620 cases of asbestosis) and Wegelius (no lung cancer in 126 radiologically diagnosed cases of asbestosis in Finland, among 476 workers examined) (36,37). They claimed that a series of clinical (not epidemiological), reports had led to the belief that the relationship was proved. They cited the frequent absence of information on smoking habits, length of time in the industry,

and dust exposure levels. Gloyne's cases were criticized as coming from a select group of cases which were brought to his attention for their unusual or complicated pathology. They suggested that Hueper's 99 cases contained some duplication among British authors; and finally, only 17 were in the United States and Canada (the other 82 were in Great Britain).

The cohort studied consisted of nearly 6,000 miners with at least 5 years employment as of the start of the study (1950). They were followed for the next six years and a careful search was made for possible lung cancer deaths. Only 9 proven and 3 questionable cases of primary lung cancer were found. All 12 were known to have smoked at least 5 cigarettes per day. This rate is not significantly higher than the age-standardized male mortality for the province of Quebec in those same years.

Major weaknesses of the Canadian study were the short period it covered, and the large dilution factor (66%) of men aged 20-44 in the original cohort. It should be borne in mind that the miners in the original cohort were healthy in 1950 and themselves represented a "survivor group". Their chances of living another six years were undoubtedly greater than those of age-standardized counterparts in the general population.

The only epidemiological study of lung cancer in asbestos factory workers to result in negative findings was done in Dresden in the late 1950's.

(38) In subsequent years, a sharp rise in lung cancer among these same workers was found, however. The later report (39) concluded that lung cancer had replaced right heart failure (cor pulmonale, usually the result of severe asbestosis) as the most common cause of death among Dresden asbestos workers. Jacob and Anspach noted that the average lapsed period from onset of exposure to death from cor pulmonale (25.7 years) was considerably shorter than the corresponding lapsed period for lung cancer associ-

ated with asbestos exposure (30.7 years). The implementation of improved industrial hygiene methods and the great reduction of tuberculosis since the advent of antibiotics (1942) were given as the reasons for asbestos workers surviving long enough to die of lung cancer (39,40).

In a study of workers employed in an asbestos products plant as of 1938 followed through 1960, Mancuso and Coulter found an excess of deaths from lung cancer (19 vs. 5.6 expected). The authors considered this a conservative figure because: the whole plant population was used, not just production line employees at risk; and events of the war affected employment patterns of 2/3 of the cohort aged 15 to 34 in 1938 (41).

By the end of 1963, the British Ministry of Labour had knowledge of 584 persons in whose death certification asbestosis had been cited. The proportion with lung cancer as a complication had risen steadily from 13.2% in Merewether's 1947 report to 25% in 1963 (42).

Building trades insulation workers in the New York area who had entered the trade before 1943 were traced by Selikoff and co-workers through 1962 (43). Of 255 deaths, 45 died of cancer of the lung and pleura, where only 6.6 such deaths were expected. These men have relatively light, intermittent exposure to asbestos. Their practices have changed little over the years. Recent measurements have shown that their 8-hour time-weighted average exposure to asbestos is 3 f/ml, with occasional peaks about ten times that level (44, 44a). This study has been extended through December 31, 1971 (45, 46), by which time 423 men who had lived for 20 years after their first exposure had died (against 288.5 expected). Of these, 84 (one in five) died of lung cancer (11.4 expected); the average lapsed period was 30-40 years; of 42 cases of lung cancer found through 1971 and for which smoking histories were known, 41 had histories of cigarette smoking. This indicates that an asbestos worker who smokes cigarettes has 92 times

the risk of dying of lung cancer compared with a man who neither works with asbestos nor smokes cigarettes. This is an order of magnitude above the risk attributed to cigarette smoking alone; in other words, 9 out of 10 cases of lung cancer among cigarette-smoking insulation workers are attributable to occupational hazard. On the other hand, no increased lung cancer incidence was found among the non-smoking workers. It is striking that the separate effects of cigarette smoking and the asbestos work are negligible compared to their combined effect in causing lung cancer. Tragically, most lung cancers detected among insulation workers are inoperable because the men have some degree of asbestosis and cannot support any further loss of lung function.

Newhouse (47) found no excess mortality from lung cancer in asbestos factory workers first exposed less than 17 years before her study (1969). There was significant excess among those with longer lapsed periods. There was little difference in lung cancer mortality between those with under two years on the payroll (8 against 1.6 expected) and those with over two years on payroll (11 against 1.5 expected) among men working heavily exposed areas. In areas with low to moderate dust exposure, no excess mortality occurred (3 lung cancer deaths in 464 men with over 16 years lapsed time, 2.4 expected.)

Kiviluoto and Meurman (48) found threefold excess of lung cancer deaths (8 cases) among anthophyllite mine and mill workers in Finland, all of whom had asbestosis. This was a small study and the exposure times observed were short; but it indicates that occupational exposure to this type of asbestos carries a lung cancer hazard also.

Isselbacher and co-workers (49), in reviewing published autopsy reports, noted that in 71 cases of asbestosis lung cancer in which the patients' sex was given, 21 (29.6%) were females, in contrast to 4.0 to 10.3% in the

general population. In Dresden (39), where females made up less than half of the work force studied, they incurred 21% of the lung cancer against 9% in the general population. Keal (50), reporting 15 deaths of females with asbestosis, listed 4 as having lung cancer, all of whom were non-smokers. Of 15 males with asbestosis, 10 died of lung cancer.

In a recent study of mortality in chrysotile mines and mills of Quebec (51) McDonald and co-workers found only a small excess lung cancer mortality (97 against about 82 expected). However, the "heavily exposed" workers had a rate of lung cancer 5 times as great as that of the "lightly exposed" workers. The study group was composed of all the individuals employed between the years 1891 and 1920 (11,788). Of the 9981 (88%) who were traced 2457 had died. This provides evidence that miners and mill workers incurred a lower excess of lung cancer than other types of asbestos workers.

The reason for the carcinogenicity of asbestos is not known, and it has not been determined whether more than one fiber is necessary to induce a malignant tumor. Cox has suggested that the probability of cancer induction is proportional to the number of asbestos fibers, the number of susceptible cells, the concentration of carcinogens on the fibers, and the time from exposure (52).

In conclusion, it is now established that excess lung cancer mortality occurs among asbestos workers. Asbestos workers who smoke cigarettes have a much higher incidence of lung cancer than groups of workers whose smoking histories were not obtained, indicating a strong cocarcinogenic effect. There are no epidemiologic data, such as available for cigarette smokers, which indicate that cessation of exposure to asbestos is associated with subsequent decreased risk of lung cancer (26). The cases reported in recent years are a reflection of conditions decades earlier. Continuous and substantial improvements have subsequently been made in asbestos processing plants. The

hazards have not been long realized in trades where asbestos-containing materials are handled (and exposures are milder). Newhouse's recent study indicates that lung cancer risk may be normal among those with light exposure, even in cases of long exposure. Selikoff has found that virtually all recent lung cancer deaths in a group of insulators were among those who smoked cigarettes. It remains for more quantitative studies of lightly exposed workers to define the dose-response relationship, both for workers who smoke cigarettes and those who do not.

Mesothelioma for Neighbors and Relatives

The existence of primary mesothelial tumors of the pleura, peritoneum, and pericardium has been a matter of dispute until very recent times. These tumors are rare, and in almost all cases death follows within months of development of symptoms. Campbell (53) reported finding 4 pleural mesotheliomas in 3533 consecutive autopsies. The possibility that mesothelial tumors were metastatic from undetected primary sites was suggested by Willis (54) and by Hinson (55) and maintained as recently as the 1960 edition of Willis' pathology text (56). Others, however, observed cases of mesothelial tumors with no detectable primary tumors in other organs (53,57-61).

The first report of pleural mesothelioma with asbestosis was made in 1943 by Wedler in Germany (62). In 1952 Cartier (63) reported 2 cases of pleural mesothelioma among Canadian chrysotile miners. Two years later Leicher (64) reported finding a primary peritoneal tumor in an asbestos worker. By use of X-ray diffraction techniques he detected the presence of asbestos within the tumor. Bonser and co-workers (65), reporting 72 cases of asbestosis (in factory workers examined post-mortem), found 2 pleural and 4 peritoneal cancers, "the primary site not being found in any organ".

Nearly 20 years after the British asbestos industry was ordered to control

dust exposures in domestic processing plants, there were still no dust controls in South Africa where the asbestos was mined.

Schepers gave the following account of a 1949 survey of the Transvaal asbestos mining district (reportedly less dusty than the Cape District) (65a). "At that time industrial hygiene in one of those mines and asbestos works was simply deplorable. Exposures were crude and unchecked. I found young children completely enclosed within large shipping bags, trampling down fluffy amosite asbestos, which all day long came cascading down over their heads. They were kept stepping lively by a burly supervisor with a hefty whip. I believe these children to have had the ultimate of asbestos dust exposure. X-ray revealed several to have radiologic asbestosis with cor pulmonale (right heart failure) before the age of 12....

"In the valley where the mill was located, asbestos dust rolled through like the morning dust, and I had a hard time keeping my staff in working trim because of itching skins caused by asbestos adhering to our clothes. Even food at the local hotel was gritty with dust."

Progress in dust control has been substantial but production has also increased tenfold in the Cape District in the last 20 years (65a,65b)

In 1956, the first South African case of pleural mesothelioma was diagnosed. Within a year, Sleggs and Marchand had 16 cases in Kimberly and Johannesburg with histologically proven pleural mesotheliomas. They suggested that asbestos might play some part in the etiology, since asbestos bodies had been found in the lungs of the first case and large crocidolite asbestos deposits were located 200 miles west of Kimberly. None of the original cases admitted to mining asbestos. Detailed histories were obtained from patients and surviving relatives. By 1959, an association with the Cape asbestos fields or the use of asbestos was established in 32 out of 33 cases with histologically proven pleural mesothelioma (66).

As an example of environmental exposure, Warner mentioned a woman born on the asbestos fields in 1900. She left there at the age of 5, and was in good health until she developed a pleural effusion at 55. She died 18 months later of pleural mesothelioma. Detailed questioning revealed that she had attended an infants' school near an asbestos dump. The children used to slide down the dump on their way home. Two of her playmates at this school have subsequently died of the same condition. (67)

The only type of asbestos mined in the Cape province is crocidolite. In Transvaal province (330 miles away), where amosite is the principal type mined (with some crocidolite), no mesotheliomas were found as late as 1964, despite intensive search. As of the end of 1968, 179 cases of "definite" mesothelioma had been found in South Africa. (68) Of the 148 in which the history was known, 3 were listed as having exposure in the Transvaal amosite mines, against 51 who worked in the mines of the Cape. In 24 there was no evidence of environmental or occupational exposure to asbestos. It is still unexplained how such a disproportionate fraction of cases had exposure in the Cape fields as compared with exposure in the Transvaal amosite and crocidolite areas. Levels of dustiness and numbers of persons-at-risk indicate that more cases should have been found in the Cape area, but not as many more as were observed (69). Recently, however, it has been reported that analysis of samples of airborne dust from Transvaal mines show that the average fiber diameter for amosite and crocidolite is three times that found in samples from Cape mines (70). If the very small fibers cause mesothelioma, as many observers feel, this alone could explain the discrepancy.

In 1962, McNulty reported a case of pleural mesothelioma in an asbestos miner in Australia, one of the few other places in the world where crocido-

lite is mined (71).

In their study of asbestos workers in the United States (41) Mancuso and Coulter found 5 cases of peritoneal mesothelioma, and suspected others.

Enticknap and Snither (72) reported 9 primary peritoneal tumors among 52 cases of asbestosis seen at necropsy. All 52 worked in an asbestos factory where all three major types of asbestos were handled (Essex, England). Outside of these cases, the writers had not encountered a mesothelioma in several thousand necropsies. "A remarkable feature of the cases is the minimal fibrosis of the lungs."

Hourihane (73) reviewed all cases of primary diffuse tumors of the pleura and peritoneum in the necropsy files at London Hospital from 1917-1962. He found asbestos bodies or fibers in the lungs, usually accompanied by fibrosis of the lungs, in 15 of 30 ("certain" and "probable") mesothelioma cases. He found no cases with fibrosis and only 3 with asbestos bodies or fibers in 100 controls at necropsy. More recently, Hourihane detected amphibole asbestos within mesothelial tumors of one quarter of the cases (74).

Newhouse and Thompson made a careful study of London Hospital mesothelioma cases for histories of exposure to asbestos (75). Eighty-three cases were confirmed by necropsy or biopsy through 1964; in 76 of these full occupational and residential histories were obtained (73 died in 1950 or later). Forty (52.6%) had a history of occupational or domestic (living in the house with an asbestos worker) exposure. In comparison, only 9 out of 76 (11.8%) control patients from the same hospital suffering from other diseases had such exposure ($P < 0.001$). Among those with no evidence of occupational or domestic exposures, 11 of the mesothelioma patients lived within half a mile of an asbestos factory, compared with 5 of the control series ($P < 0.01$). Mean interval between first exposure and death varied from 29.4 years among 23 factory workers to 48.6 years among the 11 neigh-

borhood cases.

In Belfast, McCaughey and co-workers reported finding asbestos bodies in the lungs of 12 out of 15 patients who died of pleural mesothelioma (76). Three years later there was a total of 45 cases (77). A highly significant difference in histories of exposure to asbestos was found between the 42 most recent cases and matched controls (32 against 9: $P < 0.001$). Asbestos bodies were observed in 21 (88%) of the 24 mesothelioma cases where lung tissue was available. In 24 matched controls and 5 other comparison groups (totaling 400 cases) the prevalence of asbestos bodies varied from 14% to 27%. Most of the mesothelioma patients had "light" exposures, such as indirect occupational exposure in the shipyards. No attempt was made to classify exposure by asbestos type since all three major types are used in shipbuilding and repair. Similar reports of mesothelioma in shipyards in the Netherlands (78) and Great Britain (79,80) followed.

In 2500 consecutive autopsies in New York (1953-1964) Selikoff and co-workers found 4 pleural and 3 peritoneal mesotheliomas (81). All were associated with asbestosis (found only in 26 of the autopsies). One of the men with mesothelioma was a carpenter, who had denied ever having seen, used, or handled any asbestos product. This may have been a case of indirect occupational exposure in the construction industry.

In a survey of 152 hospitals (Pennsylvania), Lieben and Pistawka (82) found 42 mesothelioma cases in the years 1958-1963. Ten worked in asbestos plants, 8 lived or worked close to an asbestos plant, 3 were family members of asbestos workers. In 10 others a history of questionable exposure to asbestos was obtained after prolonged questioning. In the other 11, no history of exposure could be found. The "most striking finding" was the clustering of 6 of the 42 cases around one insulation plant (only 2 worked there) in the geographical area of approximately 30,000 square miles

covered by this study.

Borow and co-workers (83) reported a concentration of mesothelioma cases (17 in 3 years) at a hospital 2 miles from a huge asbestos plant in Manville, New Jersey in which 75% of all asbestos mined in North America is converted to commercial use. Two patients there had only environmental exposure; i.e. they lived near the plant. More recently, the total reported number of cases from this town of 15,000 reached 52 (83a).

Hamburg, Germany is a center for asbestos factories, as well as a major port. Between 1958 and 1968, 319 cases of pleural mesothelioma were found in 4 medical institutions there (84). "There may be many more in other hospitals." The prevailing wind in Hamburg is from the west, and most of the cases resided to the east of the shipyards and in a residential district around the largest asbestos plant. In this district alone there were 51 cases of mesothelioma among 53,000 inhabitants. Of the 119 of these cases which could be fully investigated, "definite" or "likely" histories of asbestos exposure were established in 55 (46%). Average lapsed time from onset of exposure to tumor development was 35.2 years (85).

The first use of dust suppression techniques within the Hamburg asbestos plants was in the mid-1930's, but only after World War II did the asbestos factories, for economic reasons, begin to filter the asbestos dust from air exhausted from the factories. The fact that over 50% of these cases had had no other known asbestos exposure led the authors to conclude that neighborhood exposure to pollution from these plants was a major cause of death in the Hamburg cases (85).

At first, it was hoped that mesotheliomas were caused only by exposure to crocidolite asbestos, the type mined in South Africa's Cape Province. The insulation workers studied by Selikoff and co-workers had extremely high incidence of mesothelioma: 6 pleural and 16 peritoneal tumors in 380

workers who had died by the end of 1968 (45). The mean lapsed period from onset of exposure was 34.8 years for pleural mesothelioma and 43.0 for peritoneal. The only types of asbestos known to have been used in insulation materials are chrysotile and amosite. The largest asbestos manufacturer in the United States has never used crocidolite for the manufacture of insulation. Only small amounts of crocidolite were imported into the U.S. before 1940 (crocidolite is not mined in the U.S.). In other words, even if crocidolite exposure occurred, it could not account for the pattern of mesotheliomas seen among these insulation workers.

Selikoff and co-workers did a prospective study of local units of the International Association of Heat and Frost Insulators and Asbestos Workers where all insulation work is done in shipyards. The purpose of their study, which included two comparison groups of workers with little and no shipyard employment, was to compare the health hazards of insulation work which included amosite asbestos exposure (shipyard insulation) with insulation work much more limited to chrysotile (general construction work). All three groups were found to suffer similar, severe mortality primarily from lung cancer, mesothelioma, and asbestosis (84a).

These researchers also followed 230 men who began work in an amosite insulation factory between 1941 and 1945, worked there for a year or more, and were alive on January 1, 1960. Total deaths in this group through June 30, 1971 was 105, against 46.4 expected. The excess was limited to the categories of cancer of various sites and asbestosis, including 5 cases of mesothelioma (84b).

Gilson attempted to register all mesothelioma cases in the United Kingdom (86). In 1969, he reported that 550 cases had been found. From the distribution of cases by year when known (8/year from 1955-1959, 27/year from 1960-1964, 48/year from 1965-1968) he concluded that, "Although some of the increase is undoubtedly due to more widespread recognition of

the tumour, it seems highly probable that there has been a real increase in incidence as well." He went on to say that it is likely that the totals are under- rather than over-estimates.

In checking the validity of death certificates of 301 asbestos workers, Howhouse and Wagner examined necropsy reports for 158 and histological material for 81 of those. The additional information gathered led to a number of revisions in the stated causes of death. Mesothelioma was the certified cause of death of 4; another 15 mesotheliomas were identified upon review of histological material. Most of these workers died between 1955-1964, a period of ascending prominence of mesothelial tumors. In contrast, the revised number of lung cancer deaths was 28 against 29 certified (87).

Thomson (88), writing in 1969, pointed out that the criteria for diagnosing mesothelioma put forth by Churg and co-workers in 1965 are too restrictive for diagnosing pleural tumors. The criteria are (89):

- (a) there is no other primary tumor that can conceivably cause serosal spread;
- (b) the gross appearance of the tumor is that of a superficial growth with only shallow invasion of the underlying organs;
and
- (c) metastases, if any, are by and large limited to regional lymph nodes.

Thomson remarked that while most cases have no secondary tumors, there are examples of extensive spread. He based his conclusions on a series of 17 recently autopsied cases and more than 50 positive biopsies. Included were examples of direct spread as well as blood and lymphatic spread to a wide variety of organs. Referring to the confusion in the standard texts and the medical literature, he noted that the histopathologist who is not familiar

with mesotheliomas and wishes to diagnose them is presented with severe difficulties.

All deaths from mesothelial tumors known to pathologists in Canada between 1959 and mid-1968 were registered. They numbered 165 (2/3 in males), or about one per million persons per annum. Occupational and residential histories were obtained from survivors of 91% of the cases and two matched control series. An association with definite or probable contact with asbestos was clearly demonstrated ($P < 0.001$), but only 20% of the male cases and one female case had any such contact. There was increase in rate of occurrence in the final few years of the period, but the authors attributed this to record-keeping differences and to greater interest in these tumors. The mortality was appreciably higher in Quebec (1.5/million) than in Ontario (0.8/million) or the rest of Canada (0.9/million). The "most noteworthy finding" was that all excess deaths occurred in workers engaged in manufacture and industrial applications rather than in mining or milling (51,90).

Two reasons for the relatively low cancer risk of mining chrysotile were offered: either chrysotile is less likely to cause mesothelioma and lung cancer than other types of asbestos, or something in addition to asbestos is necessary (90). It is certain that people living along the serpentine belt in eastern Quebec have a lower incidence of mesothelioma than those living in the Cape province of South Africa. Likewise, there has been no excess incidence of mesothelioma in the anthophyllite mining region of east Finland (91).

Wagner and co-workers (92) have produced mesotheliomas in 1/3 to 2/3 of the rats they injected intrapleurally with the three major types of asbestos. There was no difference in effect between the natural and oil-extracted forms of crocidolite. The risk of developing a mesothelioma was proportional to the dose applied. Wagner suggested that although chrysotile

is biologically active, the spiral shape of many of its fibers may inhibit their inhalation, rendering them less hazardous. This may be one reason for the comparatively low mesothelioma mortality of chrysotile miners and mill workers.

Earlier, Wagner reported induction of mesotheliomas in rats injected with silica (93). Hueper reported producing mesotheliomas in rats by peritoneal injection of polyurethane foams (raising concern over the use of polymeric foams in surgery) (94). This leads to the question: what normally encountered agents besides asbestos fibers can lead to development of mesothelial tumors in man?

Cocarcinogenic agents have been sought for mesothelioma as well as for lung cancer. Iron and manganese are mined in the Cape province but not in Transvaal province. Welders and other men employed in shipyards are exposed to iron oxide dust as well as asbestos. Several authors have suggested that there may be a combined effect which enhances the likelihood of development of mesothelioma (68, 82). So far, the association between mesothelioma and the suspected cocarcinogens named is only casual, however.

Assertions of family or neighborhood exposure should be viewed with caution, according to Selikoff (95), as often there is a short but forgotten period of employment in an asbestos plant. Relatives of workers and those who live near asbestos plants would know when openings existed and may have worked a month or so when their own trade was slow. Selikoff and Hammond (96) concluded that no quantitative conclusions regarding the dose-response relationship will be available without epidemiological studies with indirect occupational exposure and environmental exposure.

Mesothelioma is still a rare tumor in the United States. In a prospective study of cancer in the general population, Hammond (97) found that only 3 out of 31,652 deaths (in over a million people between 30 and 89 years of

age) were caused by mesothelioma.

The previous studies lead to the following conclusions. Mesothelioma is caused by exposure to airborne asbestos fibers. Though there is probably a difference in the mesothelioma hazard posed by different types of fibers in different applications, it is prudent to regard all asbestiform materials as hazardous. There is indication that the lapsed time from exposure is longer for low levels of exposure (76,86). At low levels (where there is little or no competing risk of asbestosis or lung cancer) the likelihood of contracting mesothelioma must be a monotone increasing function of dosage. The large proportion of cases of mesothelioma with light exposure to asbestos (occupational, indirect occupational, neighborhood, and domestic) indicates that the safe exposure threshold level is substantially lower for mesothelioma than for asbestosis or lung cancer.

Other Cancers

There is evidence that gastrointestinal cancer occurs in excess among those exposed to asbestos: the insulation workers studied by Selikoff and co-workers (46) have had more than 3 times the expected number of deaths from this cause, 41, against 12.5 expected. The authors considered the number sufficient to be conclusive proof of relationship, taken together with similar findings among insulation workers in Belfast (100).

Asbestos has also been suspected in increased incidence of ovarian cancer. Graham and Graham (98) reported that 6 out of 12 patients with early ovarian malignant changes had birefringent crystalline material in the ovaries. The main point of the authors is that there was a similarity in the histological appearances of ovarian carcinoma and mesothelioma. To date, epidemiological evidence to support the hypothesis that asbestos exposure carries an excess risk of ovarian cancer does not exist.

Asbestos Bodies in the Streets

As mentioned earlier, Cooke first referred to "curious bodies" in asbestosis, noting that the lung characteristically reacted by coating entrapped asbestos fibers (5). These were first called "asbestosis bodies" in 1929 (17). The designation was shortened to "asbestos bodies" when it was realized that people without asbestosis were found to have them in their lungs.

The first report of non-occupational asbestos bodies was made in 1929 by Stewart and Hadow (18), who examined lung tissue of a person who lived across the road from an asbestos plant. Shortly afterward identical "bodies" were reported in the lungs of coal miners (101). Subsequently, they have been reported in graphite workers and diatomaceous earth workers (102). Until recently, however, these "bodies" have generally been associated with occupational exposure to asbestos.

In 1963, Thomson and co-workers (103) reported what appeared to be asbestos bodies in the lungs of a large proportion of urban dwellers who had no recorded occupational exposure to asbestos. Analysis of lung tissue sections and expressed lung fluid all over the world (99, 104-112) substantiated Thomson's findings. In most studies where sex was noted, prevalence of "asbestos bodies" was higher in males than in females. The percentage of cases in which "bodies" were found varied with the technique used in the investigation, the amount of lung tissue examined, and the diligence and persistence with which they were sought. The percentage of positive findings has been reported as high as 97% (99) and 100% (112).

A question arose whether the "bodies" found in the lungs of urban dwellers with no occupational exposure to asbestos were asbestos bodies or pseudo-asbestos bodies; that is, did they have asbestos fiber cores or not? Gross and co-workers produced what they called "ferruginous bodies" (114) by

injecting intratracheally aluminum silicate fibers, silicon carbide whiskers, glass fibers, and cosmetic talc (commercial talc may contain as much as 82% tremolite asbestos) (115) into the lungs of hamsters. These were identical to asbestos bodies. Still the question remained, is asbestos present in the lungs of urban dwellers, either enclosed in ferritin or as free fiber?

Numerous technical difficulties impeded the quantitative analyses of cores of "bodies" found in lung samples. The main problem was that chrysotile, the only asbestos type with an unmistakable morphology (and by far the most abundant type), tends to split into unit fibrils 200 to 400 Angstroms in diameter. It is also altered chemically and physically in vivo (12). Langer and co-workers (116) finally overcame these obstacles, employing electron-microprobe and high magnification (up to 217,000 X) electron microscopic techniques. They found that 15 of 16 electron microprobe analyses of asbestos bodies found in lungs of urban dwellers were consistent with magnesium-leached chrysotile, and one was consistent with amosite. Furthermore, analyses of samples (conservatively estimated at one millionth of the total lung burden) of lung unequivocally showed chrysotile in 24 of 28 consecutive New York City autopsied cases (117). Similar observations have been made in London; Pooley and co-workers (117) not only found chrysotile asbestos in almost 80% of their cases, but also noted it to be the most common and abundant of all fibers detected.

Eight hundred fifty-six New York City autopsy cases were examined for asbestos bodies in lungs and lifetime occupational histories were obtained (118). The results suggested that asbestos bodies are not randomly distributed among the general population. Forty percent of housewives showed asbestos bodies, 50% of males in clerical or professional work, 50% of males

in manual work exclusive of those who had a history, however brief, of shipyard or construction work. The last mentioned group had asbestos bodies in 90 of 129, or 70% of the cases.

One hundred consecutive New York City autopsy cases in each of two years, 1934 and 1967, were examined for asbestos bodies in ashed 175 μ lung sections by Selikoff and Hammond (118). No significant difference in the percentage of positives occurred in the two years (53% and 60%, respectively).

A similar study of secular changes in asbestos body prevalence was carried out by Pooley (119) in London. He examined 30 μ lung sections (not ashed) in 123 cases autopsied in 1936, and 100 each in 1946, 1956, and 1966. He found no asbestos bodies in those who died in 1936 or in females who died in 1946. The overall percentages of positive cases rose steadily to 20% in 1966, a figure which Pooley noted is in accord with that found in London in a recent multicenter survey using the same technique of analysis.

Do such preliminary studies reflect real differences in prevalence trends between New York and London? If further study can establish that the prevalence of asbestos bodies in the general population was the same 30 years ago as it is today, and if pathological studies can demonstrate that mesothelioma was indeed uncommon until recently, there will be less reason to suspect that the finding of asbestos bodies in the lungs of the general population is a harbinger of an incipient epidemic of mesothelioma.

Section II Environmental Aspects

Having considered in detail the various diseases which can be caused by breathing and coming in contact with asbestos fibers, let us now look at the various parts of the human environment where the danger of asbestos contamination exists.

Urban Air

Lynch and Ayer (121), compared light microscopic samples with the same material studied by electron microscopy; they reported that only 1 in 100 fibers present in air could be seen by light microscopic methods. This meant that dust-counting instruments used in making measurements in asbestos factories could not be used for environmental asbestos measurement.

Preliminary electron microscopic analysis (122) suggested that chrysotile levels were greater in Manhattan than elsewhere in New York. Nicholson and his co-workers attributed this to the extensive use of asbestos in construction in Manhattan. While values ranging from approximately 10 to 200×10^{-9} grams per cubic meter of sampled air is a small quantity of asbestos (total urban particulate loading is on the order of 100×10^{-6} grams per cubic meter (123)), 10^{-9} grams of chrysotile could represent a million fibrils.

"For the past 10 years, during which spray fire-proofing came to be extensively used, it was not uncommon to see extensive snowfalls of asbestos-containing material over widespread areas of New York and other metropolitan cities. In some cases, the fireproofing was even done with no attempt to enclose the spray area by tarpaulins or other means." (122) Sampling around construction sites where extensive spraying of asbestos fireproofing was in progress demonstrated a significant contribution of this material to air pollution. In some instances chrysotile asbestos levels approximately

10 times "background" were observed. A few months after this report was made, the use of sprayed asbestos insulation was totally banned in Boston and Chicago and severely restricted in New York, Los Angeles, Detroit, and Philadelphia (124). The U.S. Environmental Protection Agency has proposed to ban it nation-wide (124a).

Another suspected significant source of asbestos pollution is abrasion and erosion of brake lining and clutch facings in motor vehicles. The brake linings are generally fabrics woven from asbestos yarn only or from asbestos yarns and metal threads. The linings are made to adhere to drums or plates by means of thermosetting resins. Lynch examined the emissions from automobile brake wear and found that most, but not all the fiber was destroyed by heat of friction (125). Mechanics typically use air hoses to blow the dust from brake parts and thus contaminate garage areas with asbestos dust. Vacuuming or wet washing with a paintbrush and kerosene would be much safer.

Asbestos in Buildings

It is evident from Table 1 that the major use of asbestos is in materials used in building construction. These include cement products of which 70% of the world's production of asbestos is used. The cement product is rigid, strong, weather resistant and heat insulating. Cement sheet products include fireproof shingles, electrical switchboard panels and laboratory table covers. Asbestos cement is also used in making drain pipes, water and gas mains, sewage venting flues and for coverings to electrical cables. Though this use is not dangerous in itself, in the process of installation of cement products, the fitter must often cut, smooth, and work the materials, often allowing considerable dust to accumulate in the working areas.

One building practice may result in serious contamination of asbestos in interiors of public structures. This is the practice of designing ceiling

spaces as return air plenums. Instead of running metal ducting across the ceiling and connecting exhaust with return ducts, air is allowed to move through ceiling spaces sprayed with asbestos for fire protection and thermal control. The asbestos coating can easily erode through the steady current of air passing over its surface, and the particles of asbestos can find themselves in the reheated or cooled air.

The degree of this contamination is dependent upon a number of factors: rate of air flow, type of asbestos used, method of application to building surface, length of space traversed by returned air. No one knows the degree of asbestos contamination by this source, but Mr. William Ruckelshaus of the Environmental Protection Agency promised that the Agency would look into this problem of potential contamination of indoor air (126). However, after a 20 month lapse the Environmental Protection Agency still has not initiated a monitoring program. (See Appendix 1)

A school built in Lander, Wyoming in 1961 had sprayed on asbestos ceilings in classrooms. Public Health Service investigators found that "asbestos falls intermittently in both large and small quantities, and one is able to wipe up asbestos dust at random from the surfaces of the schoolroom furniture. The ventilation system recirculates room air, and the intake filters are clogged with asbestos fibers." Though airborne asbestos levels were below 5 f/ml, these investigators declared the condition a hazard to health, and renovation to replace the asbestos material began in June, 1972 (127). A similar situation is being repaired in dormitories at UCIA.

Asbestos in Personal Items

With over 3,000 uses of asbestos, little wonder each and every American should become concerned. From ironing board covers to drapes, asbestos items find their way into the homes of our people. It is thought that one of the

most dangerous areas where asbestos contamination would cause untold harm is when used as additives in tobacco products. Many patents exist for such practices. Investigation has revealed that none are being used today, but one of the first American cigarette filters was almost pure asbestos. An investigation of European cigarette filters is being conducted at this moment.

Those of us who use gas masks should be pleased to know that the Naval Research Laboratory has developed a fiberglass filter said to be "superior" to Bolivian crocidolite asbestos, the Bureau of Mines reported in 1970 (128). Attempts are being made to find out whether the Bureau has taken steps to assure that this hazardous use of crocidolite has been discontinued.

One can buy asbestos dust in many hardware stores. One of the uses made of this dangerous commodity was a practice among art teachers until recently. They would mix asbestos dust with modeling clay for consistency and ease at working the material. However, the presence of the dust and use by children was declared a major hazard, and warnings were given by the Food and Drug Administration under pressure from public interest groups.

One use of asbestos can be a source of danger for persons of special occupations: protective clothing. Firemen use suits (129) and other equipment made of asbestos; asbestos gloves are found in places where ovens are operated and around scientific laboratories. Continual use over a period of time may be a source of asbestos insult. In laboratories are also asbestos heating tapes, asbestos insulation pads, filtering materials and asbestos paper for making insulated coatings around heated glassware. Many of these items are subject to wear and flaking. The atmosphere of the working space can easily be contaminated by asbestos. For other groups, see Table III.

One unsuspected source of asbestos is in talcs which contain varying amounts of tremolite asbestos. Talcs are soft and can be reduced to small

particle size; they are cheap and are good thermal and electrical conductors and, like asbestos, are resistant to attack by acids and bases. These properties allow for a multiplicity of uses (see Table IV) ranging from fillers in scouring soaps to solid lubricants.

Elejer (130) has called attention to the potential cancer hazard posed by the strong chemical similarity between talc and asbestos, and has also written to one of the authors that many mineralogists doubt if such a thing as an asbestos-free talc deposit exists. This latter complication has so far prevented the appearance of a study of cancer incidence of workers exposed to pure talc. Talc miners who work with a talc containing about 30% tremolite risk developing a talc fibrosis similar to asbestosis and incur four times as much cancer of the lung and pleura as their counterparts in the general population (131).

A few mineralogical analyses have been made on talcs used in these many items. A study by Schulz and Williams on 51 commercial talcs indicated a range of 0 to 82% tremolite with greater than trace amounts in 17, or one third (115). This alarming report beckons a thorough analysis of all uses of talcs. Some of the area which requires immediate investigation should be in talcum powders and vaginal deodorants. Here large quantities of talc which could contain different amounts of asbestos can be easily administered to prime cancer sites.

Among talc uses which should be strictly controlled include the use as lubricants in the interior walls of balloons, where children naturally inhale and exhale the air used to inflate the item. Recent analyses of a limited number of brand by the Food and Drug Administration revealed no asbestos in the talc. However, strict regulations on talc use are still needed with an active program of surveillance.

A recently discovered serious contamination problem is in the area of

coats imported from Italy which contain asbestos mixed with wool. This was detected because the coats were declared to contain asbestos in order to escape the wool duty. Before the public was alerted in 1971, there was an estimated 100,000 coats sold to women in this country. A measurement of air concentrations near lightly brushed coats were 10,000 times more asbestos than normal background (132).

Asbestos in Food, Drinks, and Drugs

Though asbestos is not directly added to human foods, still it is able to contaminate them through careless and thoughtless practices. One such practice is the polishing of rice by the use of talcs. Talc is also used as a dusting powder for salami and a polishing medium for peanuts (128). Merliss believes that since much of the polished rice eventually is exported to Japan, this might be a reason why there is a very high incidence of stomach cancer in that country (133). While this inference was highly questioned in recent months, Merliss' report eventually led the Food and Drug Administration to propose a ban on asbestos-containing talc as a polishing agent (see Appendix 2).

A use of asbestos which causes considerable worry is the use of asbestos filters for beer, wine, and hard liquors. This allows for small fibers to be washed into the final product and find their way into the stomachs of drinkers. One of the authors spoke with the chief engineer at a Carling brewery and informed him of the hazardous potential of asbestos to workers and the public. The company immediately procured a cellulose substitute from Grefco, Incorporated, which had also furnished the asbestos-containing filter material. Asbestos filter media are widely used in United States breweries, and at this time a number of companies are switching to substitutes voluntarily in order to preempt government regulation and adverse publicity.

The above experience supports the claim that the resistant properties and low cost of asbestos lead to widespread, nonessential uses which can be readily substituted and eliminated.

Asbestos-containing filters are also used by some manufacturers of gin. Representatives of Seagrams, one of whose products is Calvert gin, maintain that asbestos has unique filtration properties and the company is seeking other materials which can produce satisfactory clarity in their product. Electron microscopist Henry Wehman found greater concentrations of asbestos fibers in Calvert gin than were present as background levels in the water from which it was made (133a).

Asbestos filters are also used in processing fruit juices, sugar, lard, and vegetable oils, and the listing here is by no means all-inclusive.

Nicholson and co-workers found that asbestos filters contaminate drug solutions used to intravenous, intramuscular, and intraperitoneal injections (133b). This last form of "therapy" is particularly alarming in view of the fact that the peritoneum is a prime site for development of mesothelioma.

The Food and Drug Administration has said it is conducting analyses to determine if asbestos is entrained in certain products filtered with asbestos. However, the FDA spokesman claims they are having trouble adapting their chosen techniques, optical microscopy, and X-ray diffraction, to perform the analyses. This is because optical microscopy is incapable of detecting most of the tiny fibers, and X-ray diffraction depends upon the crystal structure remaining intact (as mentioned earlier, even water will leach magnesium from chrysotile). A leading New York asbestos researcher has not only told FDA officials that their methods won't work, but has also demonstrated this claim for them. This deliberate subterfuge of studying a problem to death shows the FDA's contempt for its stated mission of protecting the public health.

Asbestos cement pipe carrying potable water requires periodic drilling

and tapping for maintenance purposes. In addition, asbestos may be slowly eroded by water flowing through these pipes. Analyses are being undertaken to determine the degree of contamination of water from these causes, where this pipe is used. In Baltimore, asbestos cement pipe is prohibited for metropolitan water because cast iron and prestressed non-asbestos cement pipe have better physical properties (133c). However, word did not travel far. In neighboring Harford County, 80 percent of the water supply is carried in asbestos cement pipe.

The Environmental Protection Agency has proposed no regulations for the discharge of asbestos into waterways.

Conclusion and Recommendations

The widespread contamination of asbestos throughout the United States is known but has not been publicized. The public is simply unaware of how dangerous this toxic material is. The work of good research scientists over the past few decades which shows the cancer-causing potential of this material has not been coupled by a socially responsible education of the great mass of citizens who come in contact with asbestos. It is time that public interest scientists broadcast the simple proven truth and demand that unanswered questions as to extent of contamination and types of use be addressed.

The leading cause of death among asbestos workers today is lung cancer. Asbestosis, formerly the leading cause of death of these workers, is declining. The reason is that asbestosis is caused by relatively severe occupational exposure, characteristic of pre-1940 asbestos plant conditions. Continuous improvements made since then and implementation of recently adopted standards should virtually eliminate asbestosis as an occupational hazard. In the meantime, incidence of lung cancer in asbestos workers has increased dramatically.

The recent discovery that insulators who smoke cigarettes have 92 times as great a chance of developing lung cancer as men who neither handle asbestos nor smoke is particularly alarming. Lung cancer in urban areas is twice as high as in rural areas, and the National Academy of Sciences attributes the difference largely to air pollution (134). It is also known that urban dwellers with no history of occupational exposure to asbestos commonly have numerous asbestos fibers in their lungs. What role do low levels of asbestos exposure play in urban lung cancer, particularly among the millions of cigarette smokers?

Mesothelioma is another major cause of death among asbestos workers. Others who receive neighborhood and indirect occupational exposure are also

subjected to increased mortality from mesothelioma, which occurs mostly in workers with light exposure. Because it is caused by lower level exposure, mesothelioma has become the disease of greatest concern to those who study the health hazards of asbestos exposure.

Gastrointestinal cancer is two to three times as prevalent among asbestos workers than in the general population. It might be considerably more common if not for the competing risks of other asbestos cancers and asbestosis. This finding has grave implications for the presence of asbestos fibers in foods and drinks consumed by the public.

That excess mortality in the above categories claims 40% of insulators and asbestos processing plant workers has led some to call asbestos work the worst occupational health problem in the U.S. Recent Labor Department standards are demonstrably insufficient to protect workers against asbestos cancer.

Selikoff and co-workers (135) estimated that there are fewer than 100,000 workers regularly using asbestos in construction trades in the United States. Yet more than 3,000,000 other building trade employees work in the same structures. A large number of workmen have similar indirect occupational exposure in shipyards (136).

In addition to the plight of these workers, there are many Americans subject to family and neighborhood exposures to asbestos. Everything from talcum powder to playing and living near construction and demolition sites open about half of all Americans to serious confrontation with asbestos.

The knowledge we now have about the carcinogenic effects of asbestos came as a result of retrospective epidemiological studies. Because of the inherent incompleteness of these studies and the difficulty in compiling accurate information on causes of deaths, we have obtained an inconclusive picture about dose-response relationships for mesothelioma and lung cancer. Studies now undertaken will not yield results for many years owing to the

long latent periods of those diseases.

Since the threshold concentration that humans can safely tolerate for continuous exposure has not been determined, it is impossible to evaluate the health hazards posed by present ambient concentrations. Likewise, it is not possible to determine the hazard posed by nonoccupational use of asbestos-containing items at this moment. The effect of asbestos as an environmental carcinogen will be more fully determined in future studies on mesothelioma mortality trends.

Asbestos is one of the major environmental contaminants; it was declared so by the Environmental Protection Agency in the spring of 1971. However, this declaration has not been fortified as of this moment by Standards. The original proposed regulations (Federal Register, Dec. 7, 1971) have not yet been implemented. In fact, an intermediate draft document was found to be weakened (see Appendix 3).

While more scientific research is necessary, the need should not retard us from demanding stricter regulations on the manufacture, advertisement, distribution and use of asbestos products. Both collective and individual action is necessary. Among collective citizen actions should be the following:

- a) demand a zero fiber time-weighted average for all occupational exposure to asbestos by July, 1976 (see Appendix 4);
- b) provide that all hand-operated and power-operated tools for asbestos have local exhaust ventilation systems including fabric filter dust dust collections;
- c) require personal protective equipment for work places where asbestos concentrations exceed the level of 2 f/ml;
- d) ban the use of friable asbestos materials in building construction, except where no substitutes can be found;
- e) implement strict control of asbestos use in shipyards in order to eliminate indirect occupational exposure;

- f) inform all brake servicing mechanics, people who wear asbestos protective clothing and others who are exposed to asbestos in their work of the hazards, and recommend safe practices;
- g) affix caution labels to all raw materials, mixtures, scrap, waste, debris and other products containing asbestos fibers;
- h) halt the use of asbestos in the making of any friable consumer product and remove these materials from the store shelves;
- i) discontinue the use of asbestos filter materials in the preparation of foods, drugs, and alcoholic drinks;
- j) stop the use of talc containing even trace amounts of asbestos in any product where fibers could become airborne;
- k) forbid the use of talc in foods and some consumer products including vaginal deodorant sprays;
- l) require public buildings, which allow return air to cross sprayed asbestos areas, to have air plenums surfaced with metal ductwork;
- m) require all buildings with exposed asbestos ceiling surfaces to undergo renovation for removing or covering the asbestos;
- n) require adequate wetting to lay dust in demolition of buildings containing asbestos;
- o) prohibit the use of asbestos cement pipe in metropolitan water supply systems.

What can you do as an individual concerned citizen? Now that you know that asbestos is used in a multitude of products, many of which are not labeled, beware of any product which could conceivably contain friable asbestos fibers. If you suspect that a product in a hardware store contains asbestos, read the label, ask the salesman, and (if necessary) write to the manufacturer. Ask your local hardware dealer to remove asbestos dust from the shelf.

Check pipe and boiler insulation in your home and see that the surface is not broken. You can buy resin-impregnated cloth to wrap the pipe insulation. Replace torn ironingboard covers and bury them (asbestos waste can otherwise find its way into the air via a municipal incinerator). Likewise, bury any imported coats purchased in 1970-71 which contains asbestos as indicated on the label.

Make sure that your neighborhood elementary school no longer has asbestos powder for making puppets and masks. Find out whether the building you work in has uncovered asbestos fireproofing in the recirculation air plenum or on ceilings. Ask local breweries and distillers if they use asbestos filters.

If there is an asbestos mine, mill or manufacturing plant near you, find out what it discharges into the air and water. Are ore-carrying trucks covered with tarpaulins?

Before 1895, when Johnson and Johnson began talc manufacture, babies were commonly dusted with corn starch; this safe substitute is still available, and at one-fourth the cost of talc. We recommend it. Don't use feminine hygiene sprays which contain talc. Avoid talc dusted balloons, rice, prophylactics and chewing gum. If you suspect a product, write to the manufacturer—and let us know if you discover another use of asbestos.

While many of the applications of asbestos products cannot be judged at this moment due to lack of sufficient evidence of hazardous use, still we should err on the side of prudence; we should remove a suspected product until the manufacturer proves that the item is not harmful. It is not the duty of the public interest scientist to prove whether each and every item is harmful or safe. The public has the right to demand this scientific evidence from the manufacturers of the consumer products which will otherwise simply be means to further profits at the expense of the health and safety of the American people.

Economics should not be the major determinant as to whether to remove an asbestos-containing product. Building plans may have to be modified and construction and demolition costs increased, but these are small when compared to the risk of health involved. The application of insulation in sheet or other solid form, although less convenient than spray application, still provides adequate insulation and fireproofing. Furthermore, asbestos-free sprays have been developed.

It is time for the American people to view asbestos as neither friend nor foe. The far wiser position is to treat this potentially toxic material with utmost respect, and only use it where absolutely necessary. If we did so, the volume of asbestos mined and processed would undoubtedly decrease, but the life and health of many people would be prolonged and improved. It is time the citizen makes his views known on this environmental pollutant.

March 29, 1971

Mr. William D. Ruckelshaus, Administrator
Environmental Protection Agency
1626 K Street, N.W.
Washington, DC. 20460

Dear Mr. Ruckelshaus:

Some time ago our attention was called to the new building practice of designing ceiling spaces, especially in large public buildings, as return air plenums. Many plenum areas have been sprayed with asbestos fiber for fire protection and thermal control. The connection of these closed ceilings with the duct system must result in some erosion of the asbestos coating and varying amounts of asbestos contamination of the air most surely occur. The degree of this dreaded contamination is dependent upon many factors including the rate of air flow and the type of asbestos fiber used. This letter is a request that you initiate federal monitoring of the asbestos content of indoor air.

We have brought this potential asbestos pollution problem to the attention of sheet metal contractors, unions and various interested parties. Presidents A.E. Hutchinson of the International Association of Heat and Frost Insulators and Asbestos Workers Union and E.J. Carlough of the Sheet Metal Workers International Association have expressed concern and encourage us in this request for government monitoring of indoor ambient air.

Further investigation by a staff member of the Center for Study of Responsive Law showed that no government agency was doing systematic monitoring of interior air systems of residential or business establishments, although several agencies, including the Bureau of Mines and the National Bureau of Standards, have the sophisticated equipment necessary for such studies. The National Bureau of Standards is presently conducting a study on indoor pollution, but does not intend to look

JUN 1 1971

Mr. Ralph Nader
Dr. Albert Fritsch
Box 19367
Washington, D.C. 20036

Gentlemen:

This is in reply to your March 29 letter calling attention to the practice of using ceiling spaces in new buildings as return air plenums and the consequent potential hazard of introducing asbestos fire-protection fibers into the air.

This Agency is concerned about airborne asbestos fibers. On March 31 of this year we included asbestos in the initial list of hazardous air pollutants. Under section 112 of the Clean Air Act, as amended, Federal regulations controlling emissions into the ambient air of any hazardous pollutant so listed must be promulgated by March 31, 1972. During the past year we have carried out a field sampling program for asbestos in some fifty urban and nonurban areas. These samples are being analyzed at a rate of about 20 to 30 samples per month.

I note that your letter questions whether the Environmental Protection Agency has authority to require modifications in building design in order to control potential contamination of indoor air. Nevertheless, exploratory measurements have already been made inside buildings in Columbus, Ohio, in New York City, and in Berkeley, San Diego and Santa Clara, California. Our present plans are to initiate a pilot study to measure asbestos concentrations inside and outside public buildings as soon as a competent contractor can be found.

Let me again say that we welcome the continued efforts of the Center for Study of Responsive Law to help us identify problems of the environment and develop effective programs to deal with such problems.

Sincerely yours,



William D. Ruckelshaus
Administrator

Appendix 1a

44a

Mr. William D. Ruckelshaus

page two

March 29, 1971

Protection Agency is charged with protecting our people from different types of air contaminants, this problem seems to enter under its jurisdiction. EPA may not be able to require modifications in building design, but it has the authority to take air samples, analyze them and fine known polluters.

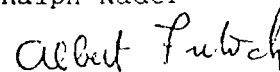
The hazards of continual breathing of asbestos particles over a period of time are well known. Prolonged inhalation of asbestos dust is known to cause cancer of the lung, pleura and peritoneum. The dangers of asbestos pollution are well documented by medical authorities. These experts tell us that asbestos contamination can cause an epidemic when growing numbers of people spend large amounts of their work, study, recreational and shopping time in public buildings with such duct systems. If the clear warning of these men is valid, then some monitoring of the dust is necessary. Generally several decades elapse between initial asbestos exposure and noticeable detrimental health effects. Americans can not afford to wait until these effects are observed; we must clearly see how serious are the asbestos and inorganic particulate levels of our buildings, so that steps be taken to correct faulty designs in ventilation and heating systems.

It is incredible that such a large number of people can be subjected to such pollution dangers all for construction savings of a few hundred feet of sheet metal duct work.

We trust you will give this matter your urgent consideration.

Sincerely,


Ralph Nader



Albert Fritsch
[Refer questions]

/cjs

833-3404

CENTER FOR SCIENCE IN THE PUBLIC INTEREST

1779 Church Street, N.W.
Washington, D.C. 20036

(202) 332-6000

December 15, 1972

Mr. William Ruckelshaus
Administrator
Environmental Protection Agency
401 M Street, S.W.
Washington, DC 20460

Dear Mr. Ruckelshaus:

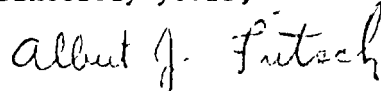
I sent a letter to you on March 24, 1971 to call your attention to asbestos hazards arising from circulation of air in buildings through areas with exposed asbestos on interior surfaces. It was requested that you embark upon a program to determine to what extent these plenums are contaminated by asbestos. In your reply of June 1, 1971 you mentioned having plans to initiate a pilot study to measure asbestos concentrations inside and outside public buildings. After an 18 month lapse we are seriously concerned about your findings.

It is evident from your letter that your agency has the authority to require modifications in building design in order to control potential contamination in indoor air. I would like to know what corrective actions have been initiated as the result of the above study.

One final request. May I have permission to use our correspondence in an appendix to a report on asbestos which is to be published in January, 1973. A prompt reply would be deeply appreciated.

Thank you for considering these requests.

Sincerely yours,



Albert J. Fritsch, Ph.D.

cc American Institute of Architects
Environmental Defense Fund
Attorney Scott Lang; Dr. Lucile Adamson

ENVIRONMENTAL PROTECTION AGENCY
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

January 17, 1973

Dr. Albert J. Fritsch
Center for Science in the Public Interest
1779 Church Street, N.W.
Washington, D.C. 20036

Dear Dr. Fritsch:

This is to confirm our telephone conversation today on the problem of asbestos concentrations in indoor air of selected commercial buildings. It will also respond to your letter of December 15 to Mr. Ruckelshaus. In our discussion, I believe I covered fully the points raised in your letter. In summary:

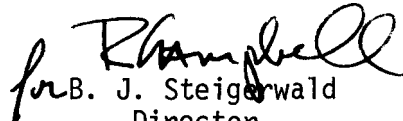
1. Our emission standards for hazardous pollutants, soon to be promulgated, will contain a ban on spraying of materials containing over 1% asbestos during construction of buildings. This should eliminate future situations.
2. The existence or magnitude of the problem of asbestos in indoor air is unknown; the measurements just don't exist. Contrary to our earlier indications to you, EPA's sampling program has been limited to outdoor air.
3. Erosion from asbestos-lined plenum chambers in buildings is a potential problem. With the successful promulgation of the regulations to reduce emissions of asbestos into the outdoor air, it seems appropriate at this time to shift some of our resources into programs to define the magnitude of indoor exposures. This is also in keeping with a recent formalization of concern in my group for indoor pollutants. We will initiate a modest sampling program to develop a data base on asbestos in indoor air and attempt to relate it to past construction practices.
4. The question of the need for and advisability of control, regulations, and legal authority are all ambiguous at this time. However, such considerations are premature until we determine whether we have a problem and we will postpone further discussion on them until a later date.

2

We will begin to develop a monitoring study and keep you informed of progress and schedules. Mr. Robert Neligan, Director, Monitoring and Data Analysis Division, Office of Air Quality Planning and Standards, will be responsible for the program. I have presumed on your knowledge and interest and assured Bob that you would be willing to make recommendations to him and our contractor on selection of sampling sites and be available for additional discussions on the problem.

I enjoyed talking with you and hope we can continue the exchange.

Sincerely yours,


for B. J. Steigerwald
Director
Air Quality Planning
and Standards

cc: W. D. Ruckelshaus
R. L. Sansom
R. Neligan

CENTER FOR SCIENCE IN THE PUBLIC INTEREST

1779 Church Street, N.W.
Washington, D.C. 20036

(202) 332-6000

October 4, 1972

Hearing Clerk
Department of Health, Education and Welfare
Room 6-88
5600 Fishers Lane
Rockville, Maryland 20852

Dear Sir or Madam:

We are writing to comment on the proposed new regulations (Federal Register, August 12, 1972) relating to prior-sanctioned food ingredients and a change in the prior sanction for talc.

It has long been known that many talcs contain substantial fractions of tremolite asbestos. Schulz and Williards¹ analyzed 51 different samples of commercial talc and found tremolite asbestos present in greater than trace amounts of 17. Kleinfeld et al² performed a mortality study on talc miners in New York and found a substantial excess of lung cancer. The talc these workers handled contained 25 to 35% tremolite.³

Among asbestos workers, even more severe hazards were exposed in Selikoff's investigations⁴ of mortality of insulation workers. Among these men, who handled chrysotile and amosite containing materials, 40% of the observed deaths were attributable to occupational asbestos exposure. Gastrointestinal cancer was the cause of death in 37 cases (versus 11.2 expected based on U.S. Mortality data), and malignant mesothelioma of the peritoneum was the cause of death in an additional 14. This latter is an extremely rare cause of death in the general population. There certainly would have been larger numbers of cases in the above categories were it not for competing risks of lung cancer (66 versus 8.9 expected), pleural mesothelioma (6 versus none expected), and asbestosis (27 versus none expected).

In a review of the literature for the National Air Pollution Control Administration, it was stated by the authors that, "it has not been determined whether more than one [asbestos] fiber is necessary to induce a malignant tumor."⁵

We are concerned that talc will continue to enjoy the status, "generally recognized as safe." There is a strong similarity in the compositions of the asbestiform silicates and talc. Talc particles have been found deeply embedded within tumor tissue taken from a number of patients

with ovarian and cervical cancer.⁶ No asbestos particles were found in these tumors.

We know of no epidemiological studies of workers with pure talc exposure. However, at this time there is plenty of reason to suspect that talc is not "safe" for use in foods, perfumed vaginal sprays, etc.

In view of the above reports, we conclude that a ban of asbestiform minerals in foods is long overdue. The presently proposed ban on asbestos-containing talcs is a beginning, and we favor its adoption. We urge the Food and Drug Administration to extend its activities to assure that asbestos is not used in filtering of such items as beer, whiskey and parenteral drugs.

Sincerely yours,

Albert J. Fritsch
Albert J. Fritsch, Ph.D.

Michael F. Jacobson
Michael F. Jacobson, Ph.D.

Barry Castleman
Barry Castleman

Appendix 2

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CENTER FOR SCIENCE IN THE PUBLIC INTEREST

1779 Church Street, N.W.
Washington, D.C. 20036

(202) 332-6000

November 9, 1972

Mr. Wm. D. Ruckelshaus
Administrator
Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Dear Mr. Ruckelshaus:

We have been following for some time your agency's progress toward National Emission Standards for hazardous air pollutants. We are particularly concerned about proposed standards for asbestos, which is a major contaminant in our environment. Our center has received copies of the proposed "Standard for Hazardous Air Pollutants: Asbestos, Beryllium and Mercury" as sent to you for final approval. Further, we have seen the comments of the Baltimore County Health Department on this document. There is no need to repeat the deficiencies referred to in their letter of November 6th. We concur with all the contents of that letter and think it should be given immediate consideration.

Overall, our impression is that the watered-down version of the Standard sent to you for approval is not really a regulation--it is a permit to operate "business as usual." All six sections of the Standard, as originally proposed in the Federal Register of December 7, 1971, have been weakened or discarded completely. We remind you that under the law, the emission of hazardous air pollutants must be regulated by the Environmental Protection Agency so as to provide "an ample margin of safety to protect the public health." As the Baltimore County letter makes clear, you will be put in an embarrassing position if called upon to explain how the sanctioning of visible emissions of asbestos dust from demolition (or any source) is protective of the public health.

There is little need to remind you of the voluminous medical literature on asbestos. We are all familiar with many reported cases of malignant mesothelioma of the pleura and peritoneum, a disease extremely rare in the general population, but quite common among asbestos workers. Also a number of persons have this disease whose only known exposure to asbestos was environmental, that is, they have lived or worked near a mine, mill, manufacturing plant, or fabrication area where asbestos was handled. Lung cancer among men who are occupationally exposed to asbestos and smoke cigarettes is nearly 100 times as common as among men who neither smoke nor handle asbestos, and is ten times as common as among men who smoke but do not work with asbestos. These figures would undoubtedly be considered higher if asbestos workers were not simultaneously subjected to competing risks of mesothelioma, gastrointestinal cancer, and asbestosis.

It is nearly impossible to estimate the level of asbestos exposure to which the general population can be continuously exposed with no increased risk of mesothelioma, or to which the cigarette-smoking population can be exposed with no increased risk of lung cancer. The National Institute of Occupational Safety and Health, in its recently published criteria document on "Occupational Exposure to

Asbestos Standard

Page 2 :

Asbestos" says: "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." In the Preliminary Air Pollution Survey of Asbestos, it is stated that "...it has not been determined whether more than one fiber is necessary to induce a malignant tumor."

Obviously, it was appropriate to designate asbestos as a hazardous air pollutant, and make it the subject of a regulation whose only criterion for acceptance is the protection of the public health. The Labor Department has gone through the ritual of promulgating a standard for occupational exposure which is insufficient to protect the health of asbestos workers and many others working in the vicinity of asbestos construction materials. We strongly urge the Environmental Protection Agency to adopt a much tougher position on asbestos contamination, and to do so at the earliest possible date.

Sincerely yours,

Albert J. Fritsch

Albert J. Fritsch, Ph.D.
Director, Center for Science
in the Public Interest

Barry Castleman

Barry Castleman, M.S.E.

Appendix: Letter to Mr. E.C. Guenther, Occupational Safety and Health Administration.

Appendix
3

CENTER FOR SCIENCE IN THE PUBLIC INTEREST

1346 Connecticut Avenue, N.W., Room 812

Washington, D.C. 20036

(202) 833-3721

July 17, 1972

Mr. E.C. Guenther,
Assistant Secretary of Labor
Occupational Safety and Health Administration
Constitution Avenue and 14th Street
Washington, D.C. 20004

Dear Sir:

We deplore your recently promulgated Standard for Exposure to Asbestos Dust (Federal Register, Vol. 37, No. 110--June 7, 1972). It is well established in the medical literature that workers exposed to the conditions prescribed face a greatly enhanced risk of developing fatal asbestos diseases. Your action followed pleas by the AFL-CIO for a one fiber per cubic centimeter (1 f/cc) limit and by the Oil, Chemical and Atomic Workers Union for a zero exposure limit, as well as statements by recognized authorities of the Mount Sinai Environmental Sciences Laboratory to the effect that 40% of deaths of insulation workers in this country, whose time-weighted exposure to asbestos is 3 f/cc, are attributable to occupational asbestos exposure. This fact was revealed at the March 1972 Hearings.

The Criteria document prepared by the National Institute for Occupational Safety and Health, "Occupational Exposure to Asbestos," makes it clear that while a 2 f/cc level cannot be declared safe, it is undoubtedly less hazardous than 5 f/cc and all haste should be made to reach that level industrywide.

Tabershaw, reviewing the literature on asbestos in 1967, compared asbestos to radiation. If there is a safe threshold above natural background to which large human populations can be exposed with no enhanced risk to developing certain diseases, this threshold is not known, it will not soon be known, and it appears to be quite low. We are perplexed and alarmed by the government's inconsistency in imposing severe restrictions on occupational exposure to radiation while historically treating asbestos as a typical workroom air contaminant.

The following is the standard we seek:

a) Permissible exposure to airborne asbestos fibers-- a 2 f/cc maximum time-weighted average (TWA) to become effective July 1, 1973, followed by a 1 f/cc (TWA) to become effective on July 1, 1975 and a zero fiber TWA effective on July 1, 1976.

Ceiling concentrations-- 5 f/cc effective January 1, 1974, to be reduced to 2 f/cc by July 1, 1976.

2.

b) Methods of Compliance: all hand-operated and power operated tools which may produce or free asbestos fibers, shall be provided with local exhaust ventilation systems in accordance with the American National Standard Fundamentals Governing the Design and Operation of Local Exhaust Systems, ANSI Z9.2-1971.

c) Personal Protective Equipment: for exposures above the permissible levels but less than 10 f/cc, a reusable or single-use respirator must be used; between 10 and 50 f/cc a full facepiece powered air purifying respirator must be used; above 50 f/cc a type "C" supplied air respirator must be used. The use of respirators would not be an acceptable substitute for application of the best available technology.

d) Caution Labels: these shall be affixed to all raw materials, mixtures, scrap, waste, debris and other products containing asbestos fibers, or to their containers where, during any foreseeable use, handling, storage, disposal, processing or transportation, airborne asbestos fibers will be released. "Foreseeable use" will not be limited to directions printed on product labels (for instance, recently reported cases of mesothelioma from floor tile sanding, an operation not specified by the manufacturer).

We are in general agreement with other aspects of your recently promulgated standard not alluded to above (monitoring, housekeeping practices, etc.)

The asbestos industry has stated that it would be hard pressed to comply with such strict standards as those mentioned here. We believe that what is needed is a complete review of all possible exposure of the labor force to airborne asbestos, as well as a careful examination of the many unnecessary exposure of the general population to asbestos fibers in consumer products foods and beverages, and drugs. After comprehensive review, it must be decided which are necessary and essential uses and which are not. Exceptions might be granted but the burden of proof would be on the manufacturer to show that a product could give rise to no hazard before marketing.

We believe that this action is necessary for the health and safety of Americans, both workers and the general public. We can hardly afford another decade of asbestos exposure while academicians and governmental agencies quibble over how much cancer the market can bear. Please give this matter your urgent consideration.

Sincerely yours,

Albert J. Fritsch
Albert J. Fritsch, Ph.D.
Center for Science in the
Public Interest

Appendix 4

Table IUses of Asbestos

(1) aprons	(50) lining
(2) arm protectors	(51) lumber
(3) bags	(52) mats
(4) belting	(53) metallic cloth
(5) blankets	(54) millboards
(6) blocks	(55) mittens
(7) boards & shingles	(56) overgaiters
(8) bonded pipe	(57) packed corrugated metal gaskets
(9) braid	(58) packing
(10) brake lining	(59) pads and covers
(11) caps	(60) paints
(12) cards	(61) panels
(13) cement boards	(62) paper paste
(14) " clay	(63) pipe & boiler coverings
(15) " machy	(64) plastic shingles
(16) " partitions	(65) pot & pan holders
(17) " roofing	(66) powder
(18) " tile	(67) protected metals
(19) " sewer pipe	(68) protecting roofing
(20) cements	(69) ribbons
(21) cloth)	(70) rings
(22) clothing	(71) roof coatings
(23) coating	(72) roof ventilators
(24) cords	(73) rope
(25) corrugated roofing	(74) scrap
(26) " sheets	(75) sheeting
(27) covered cables	(76) sheathing
(28) " hose	(77) sheets
(29) " wire	(78) siding
(30) covering	(79) sleeves
(31) curtains (theatre)	(80) sleeving
(32) covers	(81) stove linings
(33) discs	(82) stove mats
(34) fabrics	(83) suits
(35) felt	(84) table padding
(36) fibre & cement pipe	(85) tapes
(37) fillers	(86) textiles
(38) filtering materials	(87) thread
(39) filters	(88) tubing and tubes
(40) flooring	(89) twine
(41) gaskets	(90) wallboard
(42) gloves	(91) washes
(43) hats	(92) wicking
(44) helmets	(93) wire
(45) hoods	(94) wood
(46) insulated cables	(95) wool
(47) insulation	(96) working machy
(48) lagging	(97) yarns
(49) legging	

Reference: "Thomas Register of American Manufacturers", 1971, Vol. 8

Table I
(Continued)

Uses of Asbestos

	U.S. Consumption in short tons (1968)
a) Cement products:	
siding shingles, roofing shingles, interior wall siding, pipe (flat and corrugated)	410,000
b) Floor tiles:	
lineoleum, epoxy, asphalt and vinyl asbestos	200,000
c) Millboard products and paper	30,000
d) Friction products:	
brake linings, clutch facings, packings	104,000
e) Gaskets	-
f) Electrical and heat insulation	-
g) Textiles	18,000
h) Felts and home insulation	-
i) Miscellaneous	55,363
	817,363

Table II

<u>Consumption & Production of Asbestos in U.S. in Recent Years</u>		
1964	813,306 (short tons)	Production
1965	794,708	118,275
1966	805,391	125,928
1967	720,583	123,189
1968	817,363	120,690
1969	784,321	125,936

Reference: "Minerals Handbook", 1968, Vol. 2, p. 179 ff.

TABLE III

POPULATION GROUPS WITH OCCUPATIONAL AND ENVIRONMENTAL EXPOSURE TO ASBESTOS (6)

Occupational Groups

asbestos rock miners	asbestos loaders
asbestos truckers	asbestos crushers
asbestos millers	asbestos spinners
asbestos weavers	electrical appliance and wire manufacturers
masons	carpenters
heating equipment workers	rubber workers
shingle and tile manufacturers	filtering material manufacturers
molders of asbestos products	asbestos-asphalt makers
putty manufacturers	asbestos cement makers
asbestos paper workers	cardboard and brake-lining producers
asbestos felt insulation workers	asbestos sound insulation workers
asbestos insulators	pipe coverers
asbestos tube wrappers	asbestos cork insulation workers
construction workers	automobile makers
garage attendants	

Nonoccupational Groups

Residents in vicinity of asbestos processing and textile mills inhaling plant effluents polluted with asbestos dust, and individuals living and working along roads on which asbestos is trucked; residents in the vicinity of building construction and demolition, inhabitants of homes or offices with asbestos acoustical tile.

Table IV
Uses of Talc

1. Asphalt filler
2. Carving
3. Ceramics
4. Chewing gum
5. Dressing textiles
6. Dusting foundry facings
7. Dusting rubber to prevent sticking
8. Fillers for asbestos paper
9. Fillers for scouring soaps
10. Fillers for rubber products
11. Gasburner tips
12. Glazes
13. Insecticide carrier
14. Non-transparent glass
15. Plaster Products
16. Polish
17. Porcelain
18. Refractory Brick
19. Roofing
20. Solid lubricants
21. Tailor's chalk
22. Toilet preparations: talcum powder, vaginal deodorants.
23. Toy balloons
24. Surgical gloves (discontinued in most hospitals).

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BIOLOGICAL EFFECTS OF ASBESTOS

National Institutes of Health
February 1, 1973

Chairman: David Bell, M.D.
National Institute of Environmental Health Sciences

Environmental Sciences Laboratory
Mount Sinai School of Medicine of
the City University of New York

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E. C. Hartman, Ph.D.
A. M. Langer, Ph.D.
W. J. Nicholson, Ph.D.
I. I. Selikoff, M.D.
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Program

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|--|---|
| 1. <u>Introduction</u> | I. J. Selikoff, M.D. |
| 2. <u>Introduction</u> | A. M. Langer, Ph.D. |
| 3. <u>Asbestos</u>
Lung cancer
Pleural and peritoneal mesotheliomas
Gastro-intestinal neoplasms
Other neoplasms
Pneumosis and pleural changes | I. J. Selikoff, M.D. |
| 4. <u>Genetic factor interactions</u> | E. C. Hammond, Sc.D. |
| 5. <u>Pathology</u> | J. Churg, M.D.,
Y. Suzuki, M.D. |
| 6. <u>Environmental contamination</u>
Dose-response relationships
Sources of environmental contamination
Air pollution
Adverse lung burdens | W. J. Nicholson, Ph.D.
A. M. Langer, Ph.D. |
| 7. <u>Summary 1873-2000</u>
Major unresolved problems | I. J. Selikoff, M.D.
E. C. Hammond, Sc.D. |
| 8. <u>Concluding and discussions</u> | |

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Outline of Asbestos Mineralogy

1. Asbestos defined: Asbestos is a generic term for 5 varieties of fibrous, hydrated, silicate minerals which possess the following common properties: are separable into thin fibers; are electrical and thermal insulators; may be woven and fabricated into forms easily; are chemically resistant.

<u>Mineral Group</u>	<u>Mineral Species</u>
Serpentine asbestos (1)	Chrysotile
Amphibole asbestos (4)	Amosite Crocidolite Anthophyllite Tremolite

2. Structure:

(A) Chrysotile: (sheet silicate)	Sheets: silica (tridymite), brucite Curvature Spiral; concentric growth Fibril unit Bundle
(B) Amphiboles (chain silicate)	Silica tetrahedra (units) Linking of staggered double chains Cation sites (0.40 - 1.40 Å accommodation) Anion configuration Chemical variation Natural occurrence Order-Disorder of cations

3. Chemistry: (structural formula)

(A) Chrysotile	$(X)_6(Z_4O_{10}) (O, OH, F)_8 (M_3Si_4O_{10}(OH)_8)$
(B) Amphiboles	$(W, X, Y)_{7-8}(Z_1O_{11})_2 (O, OH, F)_2$ $(W_0-1X_2Y_5)(Z_2O_{22}) (O, OH, F)_2$ for $W=0$ $O_2X_2Y_5$.

Range of Superior Chemical Analyses for Asbestos Minerals

	<u>Chrysotile</u>	<u>Amosite</u>	<u>Crocidolite</u>	<u>Anthophyllite</u>	<u>Tremolite</u>
SiO ₂	41.8-42.0	49.47	47.6-50.1	50.1-58.9	54.9-59.5
TiO ₂	0- 0.1	0.25	0- 1.9	0- 1.2	0- 0.3
Al ₂ O ₃	0.1- 0.5	0.63	0.2- 3.5	0.6- 8.1	0- 4.6
Fe ₂ O ₃	0.2- 1.3	4.15	9.9-20.1	0- 4.5	0- 0.5
Cr ₂ O ₃	0- Tr	0.	0-	Tr-	0-
FeO	0.1- 1.6	35.63	2.3-24.4	0-20.5	0- 0.4
NiO	0- Tr	0.	0-	Tr-	0-
MnO	0- Tr	0.61	0- 1.5	0- 2.5	0- 0.4
MgO	41.4- 42.8	6.57	0-15.8	17.6-30.8	21.7-25.5
CaO	0- 0.1	0.52	0- 4.9	0.1- 3.5	11.9-13.1
K ₂ O	0- Tr	0.02	4.5- 8.8	0- 0.8	0- 1.3
K ₂ O	0- 0.1	0.20	0- 2.1	0- 0.2	0- 0.6
H ₂ O	13.6-14.0	2.33	0- 3.3	1.6- 4.8	0- 2.3

Outline of Asbestos Mineralogy - 2

4. Associated Trace Mineral Phases:

- (A) Chrysotile: Serpentine phases (lizardite; antigorite)
Magnetite
Chromite
Bacite (fibrous-nemalite)
Talc
Magnesite
Metallic phases (awaruite)
Calcite
Olivine, Pyroxenes, amphiboles
- (B) Amphiboles: Amosite (quartz, Fe oxides ± hydrated)
Crocidolite (as amosite + amphiboles)
Anthophyllite (talc, chrysotile, ± amphiboles)
Tremolite (talc, "serpentine," ± amphiboles)

5. Associated Trace Elements:

- (A) Chrysotile: Ni, Cr, Co, Cu, Mn, Ca
- (B) Amphiboles: (eg. anthophyllite: Ag, Ba, Co, Cr, Cu, Li, Mo, Ni, Sr, V, Zr present in > 10 ppm)
(eg. crocidolite: Zr, Nb, Th, Ce, Y, La, Ba, V, Mo)

6. Associated Organic Contaminants:

- (A) Chrysotile: Small quantities of oils and waxes
- (B) Amphiboles: Up to 0.3% oils, waxes, amino acids in amosite and crocidolite from So. Africa

7. Surface Properties:

- (A) Chrysotile: Slightly (+) H⁺ from surface hydroxyl group; changes with pH and sorbed material.
- (B) Amphiboles: Moderately (-), changes with species, substitution of cations and sorbed surface material. Also different charge at different sites.

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Asbestos Disease

1. Asbestosis

.This is confined to occupational exposure.

.There is considerable individual variation in response.

Some workers may be employed in asbestos trades for more than 40 years and still have normal chest X-rays. In others, as little as 1 day of factory exposure (inadequately controlled) will result in parenchymal or pleural disease decades later.

.Asbestosis sufficiently severe to be fatal has been seen in our studies in as little as eight years after onset of exposure, but this is most unusual. Death of asbestosis, when it occurs, usually is observed twenty, thirty, forty or more years after onset of work. Three factors generally must be considered in any analysis:

Intensity of exposure
Duration of exposure
Duration from onset of exposure
(Residence time of dust in lungs)

.By and large, significant asbestosis is not seen until at least 20 years from onset have elapsed -- the "20 year rule."

.All varieties of asbestos can produce disease; whether one type is more fibrogenic than another is uncertain.

.Clinical features of note are:

Monosymptomatic disease -- dyspnea
Restrictive lung function pattern; diffusion defects
Fine rales
Finger clubbing
Cough and sputum not prominent, except with chronic
tendency to serum rheumatoid factor
Cox pulmonary in late stages
Incomplete clinical-X-ray-function correlation

Symptoms or signs are not useful for "early" diagnosis. X-ray and pulmonary function changes vie for early detection of disease, but years may go by before abnormalities become evident. At present, we cannot predict which workers will suffer early and/or severe asbestosis. Appropriate predictive diagnostic techniques do not exist.

.Pleural involvement is an important feature of asbestos disease.

Both localized "plaques" or diffuse fibrosis may occur, and calcified plaques are often seen radiologically. When bilateral, they are almost pathognomonic of asbestos exposure even unilateral calcification is usually reliable for diagnosis, especially when there is no history of tuberculosis, leukemia, or lymphoma.

ANNEX 2. CHANGES IN X-RAY

X-ray changes tend to be extensive after more than 20 years from onset of work. Findings in 1,117 asbestos insulation workers.

Onset of exposure (yrs.)	No.	%	%	Asbestosis (grade)		
				1	2	3
40+	121	5.8	94.2	35	51	28
30-39	194	12.9	87.1	102	49	18
20-29	77	27.2	72.8	35	17	4
10-19	379	55.9	44.1	138	9	0
0-9	346	89.6	10.4	36	0	0
	1,117	51.5	48.5	366	126	50

Pleural changes on X-ray also are notable more than 20 years from onset of work

Years from onset of exposure	Number examined	Normal pleura	Abnormal pleura	
			Fibrosis	Calcification
40+	121	28	65	70
30-39	194	96	62	67
20-29	77	47	25	8
10-19	379	340	36	5
0-9	346	342	4	0

The cardinal clinical symptom -- dyspnea -- generally obeys the "20 year rule."

Onset of exposure (yrs.)	No.	%	%	Dyspnea (degree)		
				1	2	3
40+	121	52.1	47.9	26	17	16
30-39	194	70.6	29.4	35	14	8
20-29	77	76.6	23.4	12	3	3
10-19	379	92.6	7.4	27	1	0
0-9	346	98.6	1.4	5	0	0

2. Lung cancer

Important risk begins as early as 10-14 years from onset of exposure, but results in significant number of excess deaths only after 20 years from onset of exposure. Depending upon the nature of the cohort under study, we have found a 5X - 7X increase in lung cancer.

This is the most important cause of death among asbestos workers -- 20% of all deaths are caused by the tumor. All cell types are seen (see Pathology). However, the cancer has some unusual features in asbestos exposure. 2/3 are lower lobe rather than upper lobe; they tend to be peripheral (main bronchus tumors are infrequent; so bronchoscopy tends to have less utility); the pleura is often involved early. Diminished respiratory reserve makes operation less available, and concomitant asbestosis may make radiological diagnosis difficult. The clinical course and prognosis are very much like those in non-asbestos cases.

Lung cancer as a result of environmental asbestos exposure has not yet been explored; it may turn out to be even more important, in terms of number of cases, than environmental mesothelioma. It should be noted that adults in urban areas tend to have asbestos fibers in their lungs (albeit many fewer in number than in lungs of asbestos workers). The significance of the presence of these fibers among cigarette smokers in the general population is not now known. Studies are under way to determine if lung cancer rates are increased among family contacts of asbestos workers, among residents of neighborhoods about asbestos factories, and among workers indirectly exposed to asbestos in the construction industry. Rates will be correlated with asbestos lung burden of individuals in these populations. If rates in these groups are elevated, additional approaches will be necessary to evaluate whether asbestos lung contamination in the general public contributes to its lung cancer risk.

4. Gastro-intestinal cancer (stomach, colon, rectum, esophagus)

There is a modest increase of gastro-intestinal tract cancer among asbestos workers (2X - 3X). Whether it also occurs as the result of environmental asbestos exposure has not been studied.

Two observations may be relevant. 1) Among asbestos workers, asbestos fibers are present in the bowel wall long after occupational exposure has ceased. 2) There may be opportunity for asbestos contamination of food and fluids (filtration through asbestos filters; flow through asbestos cement pipes; talc as food additive). The significance of these observations is not known.

Mortality Data

In general, deaths in the asbestos worker cohorts studied may be broadly categorized as follows:

Total cancer.....	40%
lung cancer.....	20%
mesothelioma.....	7%
G.I. cancer.....	8%
Asbestosis.....	7%

The direct occupational risk is associated with current or prior regular work with asbestos. It has been estimated that 1,000,000 in the United States have or have had such experience.

While the number of individuals presumed to have indirect occupational exposure (or family contact, or neighborhood exposure) is very much larger, the magnitude of their risk of asbestos-associated disease has not yet been determined quantitatively.

The data in the first six tables which follow are derived from the experience of cohorts of workers with direct occupational exposure and refer only to such groups. Tables 7 and 8 provide some information from 2 series concerning the proportion of environmentally-induced mesothelioma, relative to those occupationally derived.

Table 1

Expected and observed number of deaths among 623 New York-New Jersey
asbestos insulation workers, Jan. 1, 1943-Dec. 31, 1971, twenty or more
years after onset of first exposure to asbestos.

	<u>1943-1951</u>		<u>1952-1961</u>		<u>1962-1971</u>		<u>Total 1943-1971</u>	
	Exp.	Observ.	Exp.	Observ.	Exp.	Observ.	Exp.	Observ.
<u>Total Cancer: all sites</u>	11.3	29	18.3	57	17.6	194	47.2	189
Lung cancer	1.5	13	3.5	23	5.1	48	10.1	84
Pleural mesothelioma	**	1	**	2	**	5	**	8
Peritoneal mesothelioma	**	1	**	3	**	20	**	24
Cancer of stomach, colon, rectum, esophagus	4.1	7	5.0	18	3.9	16	13.0	41
Cancer all other sites	5.7	6	9.8	11	8.6	15	24.1	32
<u>Asbestosis</u>	**	1	**	10	**	22	**	33
<u>All other causes</u>	65.1	44	69.4	94	78.3	87	212.8	221
<u>Total all causes</u>	76.4	73	107.7	161	95.9	187	280.0	444.3

Person-years of
 observation:

3,726

4,406

2,898

11,030

632 members were on the Union's rolls on January 1, 1943. Seven died before reaching 20 years from first employment. All others entered these calculations upon reaching the 20-year-from-Onset of first exposure point.

Expected rates are based upon age-specific death rate data of U.S. National Office of Vital Statistics from 1949-1967. Rates were extrapolated 1943-1948 from rates for 1949-1955 and for 1968-1971 from rates for 1961-1967.

**U.S. death rates not available but these are rare causes of death in the general population.

Table 2

Expected* and observed deaths among
17,800 asbestos population workers
in the United States, January 1, 1967-December 31, 1971

Distribution by duration
from onset of exposure

	<u>Total</u>		<u>Less than 20 years</u>		<u>20 years and more</u>	
	<u>Expected</u>	<u>Observed</u>	<u>Expected</u>	<u>Observed</u>	<u>Expected</u>	<u>Observed</u>
<u>Total deaths</u>	863.63	1,092	178.31	211	326.69	881
<u>Cancer: all sites</u>	144.09	159	26.31	51	117.78	108
Lung cancer	44.12	210	7.93	22	37.29	191
Pleural mesothelioma	**	23	*	2	**	24
Peritoneal mesothelioma	**	5	**	3	**	48
Cancer of stomach	6.62	13	0.97	1	5.65	15
Cancer of colon, rectum	17.51	26	2.51	3	15.00	23
Cancer of esophagus	3.21	13	0.14	1	2.77	12
All other cancers	72.23	114	15.36	19	56.97	95
<u>Asbestosis</u>	**	78	**	5	**	73
<u>All other causes</u>	661.54	535	152.63	155	308.91	400
Number of men	17,800		12,681		5,119	
Person-years of observation	80,000		62,673		23,627	

*Expected deaths are based upon age specific death rate data of the U.S. National Office of Vital Statistics. Rates for 1968-1971 were extrapolated from rates for 1961-1967.

**U.S. death rates not available, but these are rare causes of death in the general population.

Table 4

Expected and observed deaths among 689 asbestos
production and textile workers, Jan. 1, 1959 - Dec. 31, 1971

	<u>Observed deaths</u>	<u>Expected deaths</u>
Total cancer (all sites)	72	27.8
Cancer of lung, pleura, trachea, bronchus	35	8.4
Lung cancer	27	+
Pleural mesothelioma	8	+
Peritoneal mesothelioma	7	+
Cancer of stomach, colon and rectum	13	5.0
Cancer all other sites	17	14.4
Asbestosis	24	+
All other causes	103	106.5
Total deaths	199	134.3

†United States data not available but figure should be only slightly less than 8.1

‡United States data not available but these are rare causes of death in the general population.

Table 2

Expected* and observed deaths among 933*
amosite asbestos factory workers first
employed 1941-1945, and observed to Dec. 31, 1971

	<u>Before 1952</u>		<u>1952-1961</u>		<u>1962-1971</u>		<u>Total, 1941-1971</u>	
	<u>Expected</u>	<u>Observed</u>	<u>Expected</u>	<u>Observed</u>	<u>Expected</u>	<u>Observed</u>	<u>Expected</u>	<u>Observed</u>
<u>Total deaths</u>	69.98	88	102.27	146	121.24	250	299.49	484
<u>Total cancer: all sites</u>	9.81	15	18.72	44	21.63	84	50.16	143
Lung cancer	1.49	3	3.71	23	6.21	47	11.41	73
Pleural mesothelioma	***	1	***	0	***	2	***	3
Peritoneal mesothelioma	***	0	***	0	***	4	***	4
Cancer of stomach	1.45	3	1.84	4	1.29	4	4.58	11
Cancer of colon, rectum	1.56	2	2.31	5	2.88	3	7.05	15
Cancer of esophagus	0.27	0	0.45	0	0.51	0	1.23	0
All other cancers	5.04	6	10.11	12	10.74	19	25.89	37
<u>Asbestosis</u>	***	3	***	7	***	17	***	27
<u>All other causes</u>	60.17	70	89.55	95	99.61	149	249.33	314

*Expected rates are based upon age-specific death rate data of U.S. National Office of Vital Statistics from 1949-1967. Rates were extrapolated 1941-1948 from rates for 1949-1955 and for 1968-1971 from rates for 1961-1967.

**933 men were employed. In 5 cases, ages were not known and these men have been excluded and from these calculations. 877 men were traced to death or to Dec. 31, 1971. 51 men were partially traced and remain in the calculations until being lost to observation.

***U.S. death rates are not available, but these are rare causes of death in the general population.

Table 5

Deaths of lung cancer and pleural mesothelioma
among 17,800 asbestos insulation workers
in the U.S. and Canada, Jan. 1, 1967-Dec. 31, 1971:
relation to elapsed period from onset of work exposure.

<u>Years from onset</u>	<u>Expected deaths*</u>	<u>Lung cancer</u>		<u>Pleural Mesothelioma</u>
		<u>Observed deaths</u>	<u>Ratio</u>	<u>Observed deaths</u>
< 10	0.48	0	--	0
10-14	1.69	4	2.4	0
15-19	4.86	18	3.7	2
20-24	7.55	25	3.3	4
25-29	8.50	41	4.8	7
30-34	6.24	44	7.1	4
35-39	3.53	23	6.5	1
40-44	4.04	24	5.9	3
45-49	3.72	17	4.6	4
50+	3.81	17	4.5	1
Total	44.42	213	4.8	26

*Expected deaths are based upon age specific death rate data of the U.S. National Office of Vital Statistics. Rates for 1968-1971 were extrapolated from data for 1961-1967.

Table 6

Expected and observed deaths of lung cancer among 876
amosite asbestos factory workers, first employed
1941-1945, and observed to Dec. 31, 1971.*
Distribution by duration of employment.

Duration of employment	Number of Men	Person-years of observation	Deaths of lung cancer		
			Exp.**	Observ.	Ratio
< 3 months	256	5,869	3.55	13	3.66
3-11 months	291	6,158	3.58	15	4.19
1+ years	<u>326</u>	<u>6,912</u>	<u>4.09</u>	<u>45</u>	<u>11.00</u>
Total	876	18,939	11.22	73	6.51

*This table excludes 57 men. 10 died during first year of employment, 39 could not be traced after the first year, 7 had prior occupational exposure to asbestos and 1 had employment of uncertain duration. 17 men of the 876 were partially traced and remained in the calculations only until lost to observation.

**Expected rates are based upon age-specific rate data of U.S. National Office of Vital Statistics, 1919-1967. Rates were extrapolated 1911-1918 from rates for 1919-1955 and for 1968-1971 from rates for 1961-1967.

Asbestos Disease - 12Table 7

Mesothelioma in London Hospital
(Newhouse and Thompson, 1965)

Total patients.....	76
Occupational asbestos exposure....	31
Family contact.....	9
Neighborhood residence.....	11
No known contact.....	25

Table 8

Pleural Mesothelioma: 232 cases in
South Africa Mesothelioma Registry*

Exposure history not available.....	22
Exposure ascertained.....	210
Occupational asbestos exposure....	102 (48.6%)
Environmental asbestos exposure....	76 (36.2%)
No asbestos exposure.....	32 (15.2%)

*Annual Report 1971. National Research Institute for
Occupational Diseases of the South African
Medical Research Council, Johannesburg, 1972.

Bibliographical Landmarks in Asbestos Disease

- 1924 First case report of pulmonary asbestosis (Cooke, 1924).
 Not fully accepted at first, reported again in 1927 (Cooke, 1927) when case reports elsewhere confirmed the existence of the disease. In retrospect, Parliament had been notified of the hazard in 1906, when Montague Murray testified concerning fatal pulmonary fibrosis among asbestos textile workers. (Departmental Committee on Compensation for Industrial Disease, 1907).
- 1931 Industrial survey showed high prevalence of asbestosis in asbestos textile factories in Great Britain (Merewether, 1931).
 Similar prevalence in U.S. asbestos textile industry was found in PHS study (Dreessen, et al, 1938).
- 1935 A case of lung cancer with asbestosis was reported and etiological association suggested (Lynch and Smith, 1935).
 Despite numerous similar case reports, the possibility of a chance association could not be avoided and the question remained open.
- 1953 An instance of pleural mesothelioma, and one of peritoneal mesothelioma, in association with asbestosis, were reported (Weiss, 1953;
1954 Leichner, 1954).
 Again, these random cases, and others reported elsewhere in the next several years, did not prove an etiological relationship; the possibility of a chance association remained, although the frequent linking with asbestos of this otherwise rare tumor (see Wagner, et al, 1960) was striking.
- 1955 Lung cancer risk of asbestos factory workers clearly established by epidemiological study (Doll, 1955).
 Careful epidemiological study, going beyond lung cancer, showed U.S. factory hazard as well (Mancuso and Coulter, 1963).
- 1955 Pleural calcification emphasized as radiological accompaniment of asbestosis (Jacob and Bohlig, 1955).
 Extraordinary incidence in asbestos workers later demonstrated (Selikoff, 1965). Pleural calcification may occur in 50% of experienced asbestos workers against 1 in 1,000 general hospital admissions.
- 1960 Environmental asbestos disease suggested by two brilliant studies. Each took advantage of clinical asbestos "marker."
 Pleural calcification was found to be common in neighborhood about an asbestos mine and mill (499 of 6,312 x-rayed).
 (Kiviluoto, 1960.)
 Numerous cases of pleural mesothelioma reported in asbestos-rich area of South Africa. On inquiry, many had had no occupational exposure but, rather, potential environmental contact (Wagner, Sleggs and Marchand, 1960). This study also did much to establish asbestos-mesothelioma relationship.

- 1961 Physiological defect in asbestosis clarified as restrictive disease without important obstructive airway component (Bader, Bader and Selikoff, 1961).
- 1962 Experimental mesothelioma produced by intrapleural instillation of asbestos (Wagner, 1962).
 More recently, this model has been well used to investigate important variables (Stanton and Wrench, 1972). Lung cancer has also been induced (Gross, 1967).
- 1963 Wide environmental contamination suggested by frequent presence of "asbestos bodies" in lungs of routine autopsies (Thomson, Kaschula and MacDonald, 1963).
 This was soon confirmed in many cities of the world. The concept derived from these observations was that lungs of urban dwellers are regularly contaminated by asbestos. This was disputed. It had long been known that a number of fibers other than asbestos could also be coated to give similar appearance. It was proposed that, unless one was certain of the nature of the core, non-specificity be signaled by calling these structures "ferruginous bodies" (Gross, Cralley and de Treville, 1967). The matter was not resolved until 1971 (See Langer, et al).
- 1964 Asbestos disease described as common among insulation workers, drawing attention to the construction industry, which uses 2/3 of asbestos in U.S., and the potential for much wider environmental contamination than that derived from factories (Selikoff, Churg and Hammond, 1964).
- 1964 Gastro-intestinal cancer found three times as frequent as expected in cohort of asbestos workers (Selikoff, Churg and Hammond, 1964). Confirmed in later studies.
- 1965 Epidemiological investigations established mesothelioma as a common occupational risk (Selikoff, Hammond and Churg, 1965).
 Peritoneal mesothelioma was now emphasized in these and other studies (Entlichap and Suther, 1966).
- 1965 Potential significance of environmental asbestos disease highlighted by report of mesothelioma among family contacts and residents about an asbestos plant (Newhouse and Thompson, 1965).
- 1968 Spectrum of asbestos disease further widened by description of mesothelioma among shipyard construction workers with a history of only indirect exposure to asbestos, decades before (Harries, 1968).
 Again, mesothelioma was used as a "marker." The problem of cancer as a consequence of asbestos contamination of construction work sites and shipyards (carpenters, plumbers, electricians, masons, laborers, etc.) may turn out to be more important than direct asbestos work (many millions versus hundreds of thousands). Lung cancer as well as mesothelioma may be involved (Fletcher, 1972).

- 1968 Multiple factor effect demonstrated. In addition to direct carcinogenic and fibrogenic action, asbestos multiplied the lung cancer potential of cigarette smoking (Selikoff, Hammond and Churg, 1968).
Asbestos workers who smoke cigarettes have eight times greater risk of dying of lung cancer, as cigarette smokers who do not work with asbestos, and ninety times the risk of men who neither smoke nor work with asbestos.
- 1970 Soils naturally contaminated with asbestos-bearing geological formations may expose agricultural populations (Burlilov and Michaelova, 1970) and provide some measure of "natural background" of environmental asbestos contamination.
- 1971 Chrysotile asbestos in lungs of New Yorkers demonstrated by electron microscopy (Ganger, Selikoff and Sastre, 1971).
Biological potential of such contamination not now known.
- 1972 Asbestos air pollution may be responsible, at least in part, for lung contamination of urban dwellers (Nicholson, Roul and Ferrand, 1972; Selikoff, Nicholson and Langer, 1972).
Air samples, studied by electron microscopy, uniformly showed asbestos present in each of 47 U.S. cities. Levels were well below those of asbestos work sites, however.
- 1972 Environmental causes of mesothelioma stressed by analysis of cases in South Africa (South African Medical Research Council, 1972).
36% of 240 cases were considered the result of environmental exposure to asbestos, 49% were occupational in origin and 15% had no history of asbestos exposure.

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Multiple Factor Effect

In 1963, a study was initiated of a cohort of 370 asbestos insulation workers, each of whom had begun work at least twenty years before (indeed, 333 had reached thirty years from onset). Previous analysis of the experience of members of this union 1913-1962 had shown lung cancer deaths to be 6.8 times as frequent as expected. It was assumed, with good reason, that these 370 men would have an unhappy experience with lung cancer.

It was soon found, however, that there are asbestos workers and asbestos workers. Thus, among the 370 men, there were 87 who had no history of cigarette smoking. By April 30, 1967, no death of lung cancer had occurred among them. On the other hand, among the 283 with a history of regular cigarette smoking 24 died of lung cancer, although only 2.98 such deaths had been expected, given their smoking habits. It was not the smoking alone; only three deaths or so would have occurred. It was not the asbestos alone -- the 87 non-smokers had no lung cancer deaths. The combination of the two had a sharp, multiplying effect. It has been calculated that an asbestos worker who smokes cigarettes has eight times the risk of dying of lung cancer compared to similar smokers of the same age who do not work with asbestos, and 92 times the risk compared to a similar man who neither works with asbestos nor smokes cigarettes.

This cohort has been followed an additional 56 months; the findings remain very much the same. By December 31, 1971, of the 283 men who had smoked cigarettes regularly 41 died of lung cancer while of 87 men who never smoked cigarettes regularly, only one died of lung cancer.

We have obtained data in a second far larger study. On January 1, 1967, we registered all members of the insulation workers union in the United States and Canada (17,800 men). 11,656 completed a questionnaire providing, among other details, information concerning their smoking habits. Observation of these men through December 31, 1971, confirms and extends our original observations. Of 2,036 men with no history of cigarette smoking, only two deaths (of 73) were due to lung cancer. Among the 9,590 cigarette smokers, 154 deaths (of 596) were the result of lung cancer.

Lung cancer is uncommon among asbestos workers who have no history of cigarette smoking; if their risk is increased, such increase is not great. On the other hand, if these exposed individuals do smoke, the risk associated with smoking in general is multiplied many times.

Table 1

Expected* and observed deaths among 370 New York-
New Jersey asbestos insulation workers,
Jan. 1, 1962-Dec. 31, 1971

	Total	No history of cigarette smoking**		History of cigarette smoking		
Number of men Jan. 1, 1963	370		37		283	
Person-years of observation	2,520		608		1,912	
	<u>Expected deaths</u>	<u>Observed deaths</u>	<u>Expected deaths</u>	<u>Observed deaths</u>	<u>Expected deaths</u>	<u>Observed deaths</u>
<u>Cancer all sites</u>	15.74	94	4.75	15	10.99	79
Lung cancer	4.57	42	1.26	1	3.31	41
Pleural mesothelioma	***	5	***	-	***	5
Peritoneal mesothelioma	***	20	***	7	***	13
Cancer of stomach	0.94	6	0.30	2	0.64	4
Cancer of colon, rectum	2.15	6	0.69	2	1.46	4
Cancer of esophagus	0.37	-	0.11	-	0.23	-
<u>Asbestosis</u>	***	21	***	5	***	16
<u>All other causes</u>	69.22	53	22.28	15	46.94	38
<u>Total deaths</u>	84.96	168	27.03	25	57.93	133

*Expected deaths based upon age specific U.S. mortality for white males, disregarding smoking habits. Lung cancer estimates based upon U.S. rates for cancer of lung, pleura, bronchus and trachea, categories 162 and 163.

**Including 39 men who smoked pipe or cigars.

***United States data not available, but these are rare causes of death in the general population.

Table 2

Expected and observed deaths among 17,800
U.S. and Canada asbestos insulation workers,
Jan. 1, 1967-Dec. 31, 1971*

	Total	No history of cigarette smoking**	History of cigarette smoking	Smoking habits not known
Number of men Jan. 1, 1967	17,800	2,066	9,590	6,144
Person-years of observation	86,300	10,163	46,615	29,522

	Expected deaths	Observed deaths	Expected deaths	Observed deaths	Expected deaths	Observed deaths	Expected deaths	Observed deaths
<u>Cancer all sites</u>	146.09	459	19.92	33	79.58	265	44.59	161
Lung cancer	44.42	213	5.98	2	25.09	134	13.35	77
Pleural mesothelioma	***	26	***	2	***	17	***	7
Peritoneal mesothelioma	***	51	***	9	***	29	***	13
Cancer of stomach	6.62	16	0.95	1	3.60	8	2.07	7
Cancer of colon, rectum	17.51	26	2.52	4	9.53	14	5.46	8
Cancer of esophagus	3.21	13	0.44	0	1.80	7	0.97	6
<u>Asbestosis</u>	***	78	***	4	***	45	***	29
<u>All other causes</u>	661.54	553	92.67	36	356.67	286	212.20	233
<u>Total deaths</u>	805.63	1,092	112.59	73	436.25	596	256.79	423

*Expected deaths based upon age specific U.S. mortality rates for white males disregarding smoking.
Lung cancer estimates based upon U.S. rates for cancer of lung, pleura, bronchus and trachea, categories 162 and 163.

**Included 609 men who smoked pipes or cigarettes.

***United States data not available, but these are rare causes of death in the general population.

Pathology of Asbestos-Associated Disease

Asbestos enters the human body via two principal routes: the respiratory tract and the gastrointestinal tract.

Respiratory tract

Asbestos dust consists mainly of fine, needle-like fibers, ranging in length from a few hundred microns to considerably less than one micron. The dust is easily inhaled and smaller fibers are carried to the most distant segments of the lung -- respiratory bronchioles and alveoli -- where they become trapped. Some of the fibers remain in situ; others are taken up by macrophages and carried into the alveolar septa and the lymphatic vessels, and from there to the regional lymph nodes and to the subpleural lymphatics. Asbestos fibers are able to penetrate into the pleural space; either because they have sharp ends and are quite rigid, or because they are carried by macrophages.

Fate of asbestos fibers:

Some fibers slowly dissolve at a rate dependent upon the type of asbestos. Some are coated by iron-protein compound and thus probably neutralized. Both of these are very slow processes. In other instances, fibers remain in situ apparently little altered. In either case, asbestos has time to exert injurious effect on the cells of the lung. Its toxicity probably depends not only upon its chemical composition but also upon its physical properties (e.g. size and shape of fibers, piezoelectric properties) and its ability to concentrate various, often toxic, substances on its highly adsorbent surfaces.

Pulmonary fibrosis (asbestosis):

Minor degrees of injury can be repaired. Considerable concentration of asbestos is needed to produce widespread irreversible damage. Injury and death of cells and their replacement by connective tissue leads to pulmonary fibrosis. This at first is limited to the septa, but eventually destroys the alveoli and bronchioles while some of the remaining air spaces become dilated. Within the fibrosed areas variable numbers of asbestos fibers and bodies can be found. The function of the lung, gas exchange, is much reduced. Pneumonia is a frequent and sometimes fatal complication, because of poor ventilation and poor clearance of the affected lung tissue.

Plaques represent localized areas of pleural thickening. Though they can occur in various pulmonary diseases, they are particularly frequent in asbestosis and are more often found on the parietal than on the visceral pleura.

Right sided heart failure is another frequent complication of severe asbestosis. It is caused by obliteration of a large part of the pulmonary vascular tree, leading to pulmonary hypertension and to cardiac overstrain.

Malignant tumors:

Pulmonary asbestosis is relatively less frequent now than it was 30 years ago, when the danger of inhaling asbestos was less widely appreciated. However, another serious complication appears many years after asbestos exposure, namely, malignant tumors. The exposure is very often light and limited in time causing only slight or moderate pulmonary fibrosis. However, 20 or 30 years later a large proportion of exposed people develop either pulmonary carcinoma or pleural or peritoneal mesothelioma.

Pulmonary carcinoma associated with asbestos exposure has a predilection for the lower lobes (in contradistinction to the general population where it is more common in the upper lobes), but otherwise it is similar in its rate of growth, metastases and histologic structure. Pulmonary fibrosis, if present, may mask early lesions and make recognition difficult.

It can be demonstrated experimentally that asbestos is a carcinogen, though in the lung its carcinogenic potential is very weak. Of the people exposed to asbestos, only those tend to develop carcinoma who also smoke cigarettes, but the combination of asbestos and smoking produces strikingly high incidence of malignancy. (See multiple factor effect data.)

Pleural mesothelioma arises from the lining cells of the pleura, the mesothelial cells. It is less common than carcinoma, but in those exposed to asbestos it is at least 100 times as common as in the general population. Smoking apparently has no effect upon its incidence. Mesothelioma tends to grow along the pleural surfaces encasing the lung in a thick layer of tumor tissue, but it may also invade the lung and produce distant metastases. It comes in a variety of histologic patterns, the most characteristic of which combines features of carcinoma and sarcoma (biphasic tumor). Cells of mesothelioma, as well as the normal mesothelial cells, secrete material rich in acid mucopolysaccharides. Experimentally mesothelioma can be produced by intrapleural injection of asbestos.

Gastrointestinal tract

Part of inhaled asbestos may be expectorated and swallowed, or asbestos may be carried into the mouth by contaminated food or fingers. It can be demonstrated experimentally that asbestos fibers lodge in the walls of the stomach and intestine and may penetrate into the peritoneal cavity. In limited studies to date, no excess of tumors has been observed in the gastrointestinal tract of animals fed asbestos, but in man carcinoma of stomach and large intestine is more frequent in those exposed to asbestos than in the general population. The incidence of mesothelioma of the peritoneum is strikingly increased after asbestos exposure. Mesothelioma can be produced in animals by intraperitoneal injection of asbestos. Peritoneal mesothelioma tends to grow along the peritoneal surfaces, but it also produces large solid or nodular masses. Histologically, it is very similar to pleural mesothelioma.

Asbestos Dose-Disease Relationships

Only limited data are available on asbestos dose-disease relationships. Few dust counts were taken 20, 30, or 40 years ago and those done usually reflected the presence of other dusts, silica, talc, mica, etc., along with asbestos. Materials, production techniques, and control equipment have changed significantly and current conditions cannot be used to evaluate past exposures.

Some data, albeit scanty, are available on the asbestos exposure of insulation workmen and a group of workpeople in an integrated asbestos manufacturing complex. These exposure data, which are only semiquantitative, can be related to excellent mortality information (Table 1 and Table 4 of Asbestos Disease section). As a result of past occupational exposures of about 10 to 20 fibers (longer than 5 μ) per milliliter of air nearly 40% of the deaths of these workpeople can be attributed to their work environment.

No data are available on the asbestos dose-disease relationship at lower exposure levels. While any asbestos disease present in other than occupational circumstances will be at a significantly reduced intensity, the large number of people at risk give rise to serious concern. In addition to the 250,000 workpeople directly exposed to asbestos by virtue of their job, 5,000,000 are exposed indirectly in their occupations. Their asbestos exposure may be 10 to 1000 times less than the exposures which produced the current catastrophic occupational asbestos disease experience. At these lower exposures, if the percentage of deaths related to asbestos is, hypothetically, even 100 times less than direct occupational, 40,000 individuals may be affected. In environmental circumstances with 200,000,000 people potentially exposed, an additional tenfold reduction in asbestos associated disease would still leave 80,000 individuals affected.

Dose-response data are urgently required for other than direct occupational asbestos exposures. Analysis of the mortality experience and asbestos exposure of workpeople indirectly exposed is important. Indirect methods of assessing past air levels, such as the analysis of settled dust, can be helpful in the study of populations environmentally exposed. Autopsy lung tissue burdens of asbestos provide an especially useful method of assessing past exposures.

Analytic methods will require electron microscopic techniques. Most asbestos fibers present in the air, even in occupational circumstances, are not visible by light microscopy. In many circumstances, positive identification of single asbestos fibers can only be accomplished by electron microprobe techniques.

Sources of Environmental Contamination

Indirect occupational exposures to asbestos have been documented as causative of disease in a wide variety of trades in the shipbuilding and ship repair industry. Mesothelioma is now observed, at an increasing rate, in other than asbestos workers -- in plumbers, welders, electricians, carpenters, pipe fitters, etc. The exposures of these other workmen may come from simply working in the vicinity of asbestos application or from brief periods when asbestos must be removed from pipes or fittings before their own work can be done.

Similar exposures are common throughout the construction industry, especially during the past ten years. Since 1960 spray fireproofing of steelwork with asbestos containing materials has produced extensive contamination throughout construction sites with men of all trades exposed. Over 4,000,000 workmen are employed in the building industry. Their numbers alone create serious concern for their potential risk.

A variety of indirect occupational exposures can occur whenever asbestos is widely used. Many electric utility employees are exposed during repairs of boilers or turbines. Chemical plants make extensive use of asbestos insulation material on high temperature pipes and vessels as well as incorporating the fiber into some of their products. For example, in one chemical plant we have seen, the transport of asbestos from a warehouse area, through the plant, to the point of use exposed unnecessarily every production employee.

Family exposures constitute an insidious form of asbestos exposure. The dust on workmen's clothing (see Figure 1c) serves as a ready source of contamination of the employee's home. We readily find fibers of asbestos and other insulation material in the settled dust of asbestos clothes have been implicated in the deaths of wives of industry workmen. Moreover, children's play clothes washed with a father's asbestos laden overalls are likely to become contaminated with fiber and provide a continuing asbestos exposure to the child. (This cross contamination of garments has been found to occur during the dry cleaning of a woman's coat containing asbestos fiber.)

The magnitude of average asbestos air levels in these family exposures is not known. In fact the variability of the exposure precludes accurate definition. However, these family exposures can be virtually eliminated by proper use of change rooms, shower facilities, and laundry facilities which should be available to all asbestos workpeople. At the present time only a limited number of work sites have adequate change rooms and few, if any, special laundry facilities exist in the asbestos industry. Such laundry facilities must, of course, be well controlled as mesothelioma has been found among dry cleaning and laundry employees.

Environmental exposures range from ubiquitous, virtually continuous, low level concentrations derived from a wide variety of commonly used products to those from short term "pathological" uses of asbestos with extremely high concentrations of limited duration. Examples of the former are airborne asbestos from the erosion products of brake linings, residue near a factory with inadequate emission controls, asbestos in water systems, and asbestos in beverages and food products.

Environmental Contamination - 2

It is estimated that 40,000,000 pounds of asbestos is incorporated into brake linings each year. While high temperatures may alter much of the fiber during use, hundreds of thousands of pounds of asbestos can enter the environment annually from this source. Definitive data on the asbestos air concentrations from motor vehicle brake usage are lacking. However, in some very limited studies, asbestos levels two or three times background were observed at sites of extensive braking.

Water systems have been found to contain concentrations of asbestos in excess of 10 micrograms per gallon. The source of this water contamination is ill defined but it could arise from geological erosion, rain cleansing of the air, or pollution from human use of asbestos.

Asbestos filters have often been used in the food, beverage, and drug industry. Studies in our laboratory have demonstrated that erosion of the fibers from the filters occurs and the products can become contaminated.

Some very questionable uses of asbestos that have given rise to significant environmental exposures include the open transport and dumping of asbestos waste, the use of asbestos in paper mache and cement art material which is mixed dry by children, the incorporation of asbestos into consumer fabrics, and any procedure which can generate uncontrolled asbestos aerosols (such as spray fireproofing).

Often, asbestos exposures occur as a result of fiber contamination of other products. Talc often coexists with varieties of asbestos and the use of cosmetic talcum powders can produce a significant asbestos exposure. In some foreign "talcum powders" the material may contain substantial amounts of anthophyllite asbestos. Industrial talcs, especially, are liable to be contaminated as a major source of such talc coexists with extensive deposits of asbestos.

Individuals doing home repairs may occasionally have short term exposures, often unsuspected. Gypsum spackle compounds used to seal wallboard joints or patch plaster often contain asbestos in amounts up to a few percent by weight. The sanding of such material, when dried, can generate high concentrations of asbestos. An insidious such exposure is that to individuals in urban areas whose apartments have been rehabilitated under lead control programs. Here, wallboard is typically installed over the lead contaminated paint. Spackle is applied to the joints and sanded with the result that asbestos dust rather than lead may contaminate the household.

Over 3000 uses of asbestos have been documented. Many of these products are produced safely and can be used safely. The production and use of others, however, can easily give rise to significant human exposures. Only constant vigilance by industry, by government, by research scientists, and by consumers will assure their eventual safe use.

Asbestos Air Pollution

Ambient air levels of asbestos range from approximately 10^{-10} gm/m³ to over 10^{-7} gm/m³. Thus, asbestos may constitute only 0.0001 percent to 0.1 percent of the particulate matter present in a given air sample. Moreover, the asbestos found in the ambient air includes both micron-size fibers and numerous individual fibrils which may be agglomerated with a variety of other material present in the air sample.

These considerations preclude the possibility of quantitative analysis of such ambient air samples by light microscopy, bulk spectroscopic techniques, or X-ray diffraction. The agglomeration of the asbestos with other materials and the presence of many sub-light microscopic fibers render light microscopy ineffective. Moreover, the unique identification of small optically visible fibers is not always possible, even using a petrographic microscope. Any bulk analysis method attempted to date has failed because of the presence of the much greater quantity of other inorganic and mineral material.

The only effective analysis method has required the use of electron microscopic techniques and involves the following steps:

1. collection of the air samples on membrane filter paper,
2. low temperature ashing of the collected material to remove the filter material and other organic matter,
3. dispersion of the residue by grinding or use of ultrasonic energy,
4. fixation of the dispersed residue in a nitrocellulose film which is mounted on an electron microscope grid, and
5. scanning at 40,000 X magnification using electron microscopy to determine the mass of asbestos originating from a prescribed fraction of the initial sample.

The dispersion procedure has been found necessary as large fragments of other inorganic material obscure the presence of asbestos which often exists in fibril form attached to other particles. Unfortunately, this procedure allows only the mass concentration to be determined and information on the size distribution of the fibers is lost.

One hundred eighty seven samples from 49 United States cities, collected by the National Air Pollution Control Administration during 1969 and 1970, were analyzed at the Environmental Sciences