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ASBESTOS DISEASE IN THE UNITED STATES 1918-1975 (*)

by

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SUMMARY

The author recalls the important stages of the history of asbestos between 1918 and 1975 in the United States. For approximately 25 years, only the pneumoconiosis due to asbestos was taken into account. The risk of cancer appeared subsequently. The cancers are represented significatively by bronchial and especially pleural cancers. The modes of contamination by asbestos vary. The author defines the different conditions of this contamination. A brief, intense exposure to asbestos can be as dangerous as prolonged contact. Little is known about dose-response relationships. Among the carcinogenic factors, tobacco plays an important role.

Urgent action must be taken in order to reduce the risk in both professional and non-professional areas.

Comparatively few studies were undertaken in the United States concerning asbestos-associated disease until the 1930's, despite the considerable use of asbestos. The first medical reference to disease among asbestos workers may be found in the rich monograph of Frederick L. Hoffman : « Mortality from respiratory diseases in the dusty trades. Inorganic dusts », published by the U.S. Bureau of Labor Statistics in 1918 [1]. He noted that insurance company records showed increased mortality among asbestos workers and commented that these companies were reluctant to insure such workers. In the same year, there was the finding by Pancoast. Miller and Landis of fine fibrosis in the chest X-rays of 15 asbestos workers [2, 3].

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In retrospect, these early scientific reports should perhaps have attracted more attention. There are unpublished data indicating that both compensation awards and insurance claims reflected disability among asbestos workers [4], for example. The first published case report of fatal asbestos disease in the United States appeared in 1930 [5].

The brilliant series of British reports [6, 12] 1927-1929 concerning asbestosis attracted much attention in the United States and stimulated the initiation of important research studies. In 1929, the U.S. asbestos industry commissioned a survey of asbestos health hazards in the United States and Canada, which was conducted from October 1929 to January 1931. The results were reported in 1935 [13]. A serious situation had been found. Of 126 workers employed three years or more, selected at random, 106 had abnormal findings, with only 25 of 121 asymptomatic.

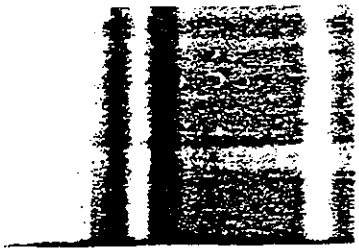
Meanwhile, independent surveys had been undertaken, and had shown significant prevalence of asbestosis in factories [14, 17]. When an extensive investigation by the U.S. Public Health Service confirmed these findings, it became widely known and appreciated that asbestos exposure commonly resulted in a serious pneumoconiosis. Although industrial hygiene data were scant, the U.S. Public Health Service proposed a « tentative standard », to be revised when better information became available [18]. In 1935, Lynch and Smith suggested that lung cancer could also be associated with asbestos exposure [19].

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

With this background, it is difficult to explain the curious quiet hiatus of the next 25 years [20]. Little was done, regulations were few and government inspections and supervision were infrequent. For example, in a clinical survey of 1,117 asbestos insulation workers conducted by us, each worker was asked about dust counts he had seen. Their employment covered the span 1910-1962 [21]. In approximately 3,500,000 working days, seven dust counts had been observed; most, in relation to recent Workmen's Compensation claims.

Towards the end of this « silent era » some disquiet appeared concerning the question of asbestos-induced cancer, and was augmented by an observation reported by O'Donnell and Mann in 1957, from a hospital in a city in Pennsylvania with an asbestos factory, that of 27 cases of asbestosis seen by them, 13 had had lung cancer [22].

It is worthy of note that during these years of inattention, years in which the use of asbestos grew approximately five-fold in a rapidly expanding industry making thousands of products, approximately one million United States men and women began work for shorter or longer periods, unwarned and largely unprotected, a fact for which our scientists can take little pride and little credit. This is especially true because many of the cases of asbestos disease now being seen in the United States had their origin in uncontrolled excessive exposures during this time [23].



Fifteen years ago, asbestos disease in the United States began to attract much more attention. A number of studies appeared which identified three major problems, which we have not solved and with which we are now concerned.

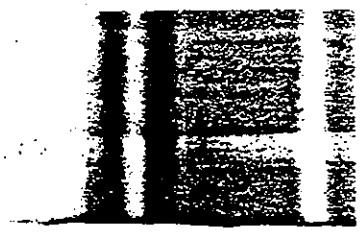
First, although the frequent presence of pulmonary asbestosis among workers in the asbestos trades had been demonstrated 40 years ago, this knowledges has not resulted in an important reduction in its prevalence among such groups of workers nor in the prevention of its more serious consequences, cor pulmonale and respiratory insufficiency. In one study of asbestos insulation workers, for example, of 1,249 men who had been members of the insulation worker's union in the New York metropolitan area at any time from January 1, 1943-December 31, 1962, 1,117 were X-rayed during 1963 [21]. 542 had abnormal X-rays. Notable was the fact that of the 725 with less than 20 years from onset of exposure, only 203 were abnormal. On the other hand, of the 392 men with longer durations from onset of exposure, 399 had abnormal roentgenograms (Table I).

TABLE I. — *X-ray changes in asbestos insulation workers* [21].

Onset of exposure (years)	No.	% normal	% abnormal	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	158	9	0
0- 9	346	89.6	10.4	36	0	0
Total	1,117	51.5	48.5	366	126	50

Moreover, it was found that in many groups of workers from 5 to 7 % of deaths were due to cor pulmonale and respiratory insufficiency [24, 26]. Identification of the problem 1930-1935 had not been sufficient to prevent the occurrence of the disease.

Second, and perhaps more important, although the persistence of asbestosis as an important industrial hazard was rediscovered, it soon became evident that it was not the principal hazard of exposure to the fiber. The principal difficulty was clearly cancer. Two critical studies showed this to be true, both in the factory production of asbestos products [27] and among workers using these materials [24].



In our experience, approximately 40 % of deaths among asbestos insulation workers in recent years have been due to malignancy. In one study, we have followed a cohort of 632 asbestos insulation workers from January 1, 1943 to December 31, 1974; they continue under observation. Altogether, 305 deaths were expected. 451 occurred. The excess was largely due to cancer. Age, year and sex specific analysis indicated that 52 such deaths were to have been expected — 200 occurred. Specifically, analysis of the cancer deaths by site indicated that 12 lung cancer deaths were likely to have occurred; 89 were found. Some two or three times as many cancers of the esophagus, stomach, colon and rectum occurred as expected. There were also 35 deaths of mesothelioma, 10 pleural and 25 peritoneal. Of course, no deaths of this rare disease were expected (Table II).

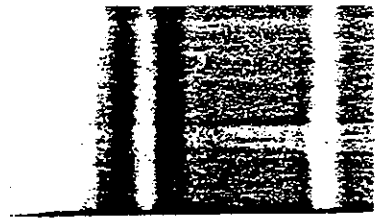
TABLE II. — *Expected and observed deaths among 632 N.Y.-N.J. asbestos insulation workers, January 1, 1943-December 31, 1974.*

	Expected (*)	Observed
Total deaths, all causes	305.20	451
Total cancer, all sites	52.02	200
Lung cancer	12.20	89
Pleural mesothelioma	(**)	10
Peritoneal mesothelioma	(**)	25
Cancer of stomach, esophagus	6.46	20
Cancer of colon	7.64	23
Asbestosis	(**)	37
All other causes	253.18	214

(*) 632 members were on the union's rolls on January 1, 1943. 9 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 deaths are based upon white male age-specific death rate data of the U.S. National Office of Vital Statistics from 1947-1973. Rates were extrapolated 1943-1948 from rates for 1949-1955, and for 1974 from rates for 1969-1973.

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

It was of interest to find that, among the mesothelioma deaths, there were more peritoneal mesotheliomas than pleural. This observation is consistent with that made by Enticknap and Smither [28] among workers in an asbestos factory in London, and is in contrast to the location among patients with mesothelioma derived from environmental contact with asbestos [29]. Perhaps differences in intensity and in nature of exposure are responsible for this variation. Direct occupational exposure, with its exposure to large numbers of fibers including, perhaps, ingestion as well as inhalation, may be more likely to increase the risk of peritoneal mesothelioma while the far fewer fibers in environmental circumstances, tend not to lead to such disease.



Similar findings have been obtained in other studies. In one, we have been following all members of the insulation worker's union in the United States and Canada (International Association of Heat and Frost Insulators and Asbestos Workers, AFL-CIO, CLC). On January 1, 1967 there were 17,800 such men. By the end of 1973, 1,577 deaths had occured. Again there were approximately three times as many cancers as expected (Table III). And again, lung cancer, mesothe-

TABLE III. — *Expected and observed deaths among 17,800 asbestos insulation workers in the U.S. and Canada, January 1, 1967-December 31, 1973.*

	Expected deaths (*)	Observed deaths	Ratio
Total deaths	1,131.11	1,577	1.39
Total cancers, all sites	209.27	658	3.14
Lung cancer	66.99	321	4.79
Pleural mesothelioma	(**)	36	—
Peritoneal mesothelioma	(**)	67	—
Cancer of stomach	9.82	16	1.63
Cancer of colon, rectum	24.55	39	1.59
Cancer of esophagus	4.60	14	3.04
All other cancer	103.31	165	1.60
Asbestosis	(**)	119	
All other causes	921.84	860	0.87

(*) Expected deaths are based upon age-specific white male death rate data of the U.S. National Office of Vital Statistics from 1963-1973. Rates were extrapolated for 1973 from rates for 1968-1972.

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

lioma and gastro-intestinal cancer were the principal categories of such excess neoplastic deaths. 67 deaths of lung cancer were anticipated; 321 occurred. There were 103 deaths of mesothelium (again, with a preponderance of peritoneal sites of origin) and a modest increase of cancer of the esophagus, stomach, colon and rectum. It was of interest that cancers of other sites were also, in the aggregate, increased (103 expected, 165 observed). While increased risk of cancer of a number of other sites exists (larynx [30], oro-pharynx [31] and others) the infrequency of such neoplasms in general makes it advisable to seek further experience before a firm opinion is expressed concerning the extent of such increased risk.

We have considered the possibility that the complex environment in which these insulation workers are employed (industrial construction) might in some way influence their mortality risks. We therefore undertook to investigate the workforce of two factories making insulation materials, exposed to asbestos but not to many of the other materials found at construction worksites. The first was a plant which made amosite asbestos insulation materials (pipe covering, block, asbestos mattresses, etc.). Introduced in the late 1930's, their use increased during World War II. A factory making the products started production in an eastern U.S. city in 1941.

From June, 1941 to the end of 1945, 933 men began work at the plant. Some worked for as little as one day, others for one or more months and a number for somewhat over 13 years, until the plant ceased operations in November 1954. We have traced this group of workers to December 31, 1974. Table IV includes

TABLE IV. — *Death among 933 (*) workers employed in an amosite asbestos factory, starting five years from onset of work 1941-1945 to December 31, 1974.*

Cause of death	Deaths 1946-1974		
	Expected (**)	Observed	Ratio
All causes	285.62	483	1.69
Cancer, all sites	50.10	157	3.13
Lung cancer	12.45	83	6.67
G.I. cancer	12.05	24	1.99
Pleural mesothelioma	(***)	5	....
Peritoneal mesothelioma	(***)	5	....
« Asbestos » cancer	24.50	117	4.78
Other cancer	25.60	40	1.56
Asbestosis	(***)	28	....
All other causes	235.52	298	1.27

(*) 128 workers were omitted from these calculations : 33 had prior asbestos exposure; 28 died in the first five years after onset of employment. 49 were not completely traced; and eight had other asbestos employment after five year from onset point.

(**) Expected deaths are based upon white male age-specific death rate data of the U.S. National Office of Vital Statistics, 1949-1973. Rates were extrapolated for 1946-1948 from rates for 1949-1955 and for 1974 from rates for 1969-1973.

(***) U.S. death rates no available but these are rare causes of death in the general population.

their mortality experience, starting five years from onset of work. It will be noted that their experience was very much like that of the construction workers who used the products made in this factory. Again, there was three times increase of cancer of all sites (50 anticipated, 157 observed). Too, lung cancer, gastrointestinal cancer and mesothelioma were the major categories with excess neoplastic deaths. These experiences confirm earlier observations of neoplasic risk associated with this work [32].

In an investigation of workers in another asbestos factory (the largest in the United States) we had less opportunity of focusing on the production of insulation materials, since many other products were made. Nevertheless, the results are again of interest. On January 1, 1959, there were 689 production employees in this factory (including 611 males) who had begun work before January 1, 1939. We have studied the male mortality experience January 1, 1959-December 31, 1973. There was little difference from our findings in other cohorts. Again, cancer proved to be the major asbestos hazard (although asbestosis, as in the other cohorts, was an important cause of death). Of the cancer categories, bronchogenic carcinoma, mesothelioma and gastrointestinal cancer took pride of place (Table V).

TABLE V. — *Expected (*) and observed number of deaths among 611 New Jersey asbestos factory employées 1 January 1959-31 December 1973 twenty or more years after onset of first exposure to asbestos.*

	1959-1963			1964-1968			1969-1973			Total 1959-1973		
Number of men	611			562			480			611		
Person-years of observ...	2,066			2,646			2,195			7,795		
	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio
Total deaths, all causes	39.87	49	1.32	52.06	82	1.58	59.60	82	1.38	151.53	213	1.41
Cancer, all sites	7.60	19	2.50	10.52	24	2.28	12.75	34	2.67	30.87	77	2.49
Lung cancer	2.31	6	2.60	3.48	10	2.87	4.37	14	3.20	10.16	30	2.95
Pleural mesothelioma	n.a.	1	..	n.a.	8	..	n.a.	6	..	n.a.	10	..
Peritoneal mesothelioma	n.a.	1	..	n.a.	3	..	n.a.	4	..	n.a.	8	..
Stomach, esophagus	0.72	3	4.17	0.87	—	—	0.90	—	—	2.49	3	1.20
Colon, rectum	0.92	4	4.35	1.28	2	1.56	1.56	2	1.28	3.76	8	2.13
Other cancer	3.65	4	1.10	4.89	6	1.23	5.92	8	1.35	14.46	18	1.24
All respiratory disease	2.32	9	3.88	3.34	10	2.99	4.17	10	2.40	9.83	29	2.95
Asbestosis	n.a.	8	..	n.a.	7	..	n.a.	8	..	n.a.	23	..
All other causes	29.95	21	0.70	38.20	48	1.26	42.68	38	0.89	110.83	107	0.97

(*) Expected deaths are based upon white male age-specific death rate data of the U.S. National Office of Vital Statistics, 1969-1973.

n.a. : Specific rates for these causes of death are not available. They are, however, extremely rare in the general population.

A third major difficulty now with us is derived from the appreciation that it may take much less asbestos to cause cancer than to result in asbestosis — sometimes, very little indeed. We learned this first from the work of Wagner and his colleagues [29] and Newhouse [33]. So alerted, we have become concerned not only with asbestos workers, but with widespread dissemination of asbestos from the primary worksite, exposing both other workers nearby as well as individuals not associated with the work at all, but exposed to asbestos as a result of environmental pollution. Examples include household contamination by dust brought home by workers to their households and asbestos air pollution from asbestos-using facilities, contaminating surrounding neighborhoods and, to a lesser extent, the general environment.

Such indirect exposure of workers has been particularly important in the United States in the construction industry and shipyards. In the former, since 1959, a particular hazard has resulted from the use of sprayed asbestos fireproofing [34]. This is now strictly controlled [35].

Anderson and his colleagues [36] have also demonstrated that family contact asbestos disease, among wives and children of asbestos workers, is much more common than originally suspected. 326 household contacts of workers employed in the amosite asbestos factory for shorter or longer periods 1941-1954 were examined in 1974-1975. Approximately one-third had characteristic parenchymal and/or pleural X-ray abnormalities (Tables VI and VII).

TABLE VI. — *Distribution of 326 household contacts of amosite asbestos workers by relationship to worker.*

	Relationship	Number		Percent
	Wives	83		25 %
Children	Daughters	118	188	58 %
	Sons	70		
Sibs	Sisters	20	32	10 %
	Brothers	12		
Others	Mother	7	23	7 %
	Father	3		
	Cousins, etc.	13		

TABLE VII. — *X-ray abnormalities among 326 household members of amosite asbestos workers.*

X-ray findings	Number of household members
Pleural thickening the only abnormality	42 (13 %)
Pleural calcification the only abnormality	7 (2 %)
Pleural thickening and pleural calcification	3 (1 %)
Irregular opacities the only abnormality	35 (11 %)
Irregular opacities, pleural thickening and/or pleural calcification	27 (8 %)
Total	114 (35 %)

At further remove from occupational sources of asbestos contamination, there has also been concern with asbestos pollution of general community air [37]. There are numerous sources for such asbestos air pollution in the United States [38]. In recent months an additional source has been identified : air within buildings fireproofed with asbestos [39]. Concomitant with such environmental contamination has been the frequent finding of chrysotile asbestos fibrils in the lungs or urban dwellers.

CURRENT UNITED STATES APPROACHES TO CONTROL ASBESTOS DISEASE

Perspectives which provide a background to approaches designed to control or eliminate asbestos disease in the United States are largely derived from experiences such as those above and are reflected in proposed regulations, either in occupational [41] or environmental [35] circumstances.

1. *Asbestos-associated cancer of primary concern.* Control measures are now based on the realization that it is necessary to prevent asbestos-associated cancer, as well as asbestosis [41]. In general, this implies that permissable asbestos concentrations need be significantly lower than those calculated to minimize the risk of asbestos alone.

2. *Long period of clinical latency.* In seeking to prevent cancer, it is appreciated that the long period of clinical latency of asbestos cancer must be taken into account. Our studies and those of others have demonstrated that cancers associated with asbestos exposure frequently do not become clinically evident for at least 20 years after onset of exposure. Often, the elapsed period is 30, 40 or more years. We have analyzed deaths of lung cancer in our cohort of 17,800 asbestos insulation workers in the United States and Canada, from January 1, 1967 to December 31, 1972. Table VIII demonstrates that there were no deaths

TABLE VIII. — *Deaths from lung cancer among 17,800 asbestos insulation workers in the U.S. and Canada January 1, 1967-December 31, 1972; relation to elapsed period from onset of work exposure.*

Years from onset	Lung cancer		
	Expected deaths	Observed deaths	Ratio
<10	0.56	0	—
10-14	1.97	5	2.5
15-19	5.87	23	3.9
20-24	9.55	34	3.6
25-29	10.70	56	5.2
30-34	8.20	60	7.3
35-39	4.68	29	6.2
40-44	4.84	27	5.6
45-49	4.51	19	4.2
50 +	4.97	22	4.4
Total	55.87	275	4.9

Expected deaths are based upon age-specific white male death rate data of the U.S. National Office of Vital Statistics. Rates for 1972 were extrapolated from data for 1967-1971.

of lung cancer among individuals with less than 10 years from onset of exposure. A small increased risk was found in the period 10-14 years from onset but the major impact of the increased lung cancer risk was observed 20-40 years from the beginning of work exposure.

It is likely that « duration from onset of exposure » is, in some instances at least, a composite effect and includes both the influence of total exposure and that of the passage of time from first exposure (or, more accurately, from the time sufficient exposure has occurred to result in increased cancer risk — « effective exposure »).

3. *Brief (excessive) exposure may produce later disease.* Even brief exposures — a day, a week, a month — if excessive, can later result in disease. The inhaled fibers remain in the lung. Thus, the men employed in the amosite asbestos products factory mentioned above 1941-1945, worker for varying periods of time. Approximately one-third worked for three months or less, one-third for a year or more. Even less than three months of work resulted, during the next 30 or so years, in significantly increased cancer risk (Table IX). Nevertheless, those with longer periods of work had even greater risk.

4. *Multiple facter interaction — cigarette smoking.* In 1967, it was found that the risk of the most important occupational asbestos cancer, bronchogenic carcinoma, did not depend upon asbestos exposure alone. Rather, if there were absence of concordance of two agents, cigarette smoking and asbestos, there did not seem to be a significantly increased risk [25].

In the first cohort study detailed above (Table II) on January 1, 1963 there were 370 survivors of the group whose observation had begun in 1943. When these men were examined, each was asked of his smoking habits. 87 men had no history of cigarette smoking and 283 had such history. By April 30, 1967, it was anticipated that virtually no deaths would have occurred among the non-smokers. Not one did occur despite their long years of asbestos work. On the other hand, 3 deaths of lung cancer had been anticipated among the 283 smokers. 24 were observed. It was calculated from these data that asbestos workers who smoke cigarettes have approximately 92 times the risk of dying of lung cancer, compared to men of equal age who neither smoke nor work with asbestos (Table X) [25].

The cigarette smoking-asbestos interaction has been confirmed in a larger study [43]. Among 2,066 non-smoking asbestos workers 7.51 deaths of lung cancer were expected from January 1, 1967 through 1972 on the basis of rates derived from the general population of the United States. 2 deaths occurred. On the other hand, among 9,590 men with a recorded history of cigarette smoking, 31.60 deaths of lung cancer were expected, and 179 were observed (Table XI).

No clear association with risk of mesothelioma has been observed, nor of cancer of the stomach, colon or rectum. Asbestos workers who do not smoke may still die more frequently than expected of these diseases, or of asbestosis. Nevertheless, if they do smoke cigarettes, our data indicate that their risk of death of lung cancer is approximately eight times that of other smokers.

TABLE IX. — *Expected and observed deaths subsequent to first year after onset of employment among 870 amosite asbestos factory workers first employed 1941-1945 and observed to December 31, 1973. Distribution by duration of employment*

	3 months work or less			3-11 months work			1 year + work		
	Expected	Observed	Ratio	Expected	Observed	Ratio	Expected	Observed	Ratio
Total deaths, all causes	99.75	112	1.12	94.34	170	1.80	110.55	216	1.95
Cancer, all sites	16.92	28	1.65	16.29	46	2.82	18.90	81	4.27
Lung cancer	4.13	16	3.87	4.00	16	4.00	4.64	49	10.56
Pleural mesothelioma	n.a. (*)	0	—	n.a.	2	—	n.a.	2	—
Peritoneal mesothelioma	n.a.	0	—	n.a.	1	—	n.a.	4	—
Cancer stomach	1.46	1	0.68	1.47	3	2.04	1.73	5	2.89
Cancer colon, rectum	2.38	4	1.68	2.27	7	3.08	2.67	5	1.87
Asbestosis	n.a.	1	—	n.a.	2	—	n.a.	23	—
All other causes	82.83	83	1.00	78.05	122	1.56	91.66	112	1.22
Number of workers		240			294			327	
Person-years of observation		5,747			6,305			7,061	

This table excluded 63 men. Ten died during first year of employment, 34 could not be traced after the first year and 19 had prior occupational exposure to asbestos. Of the 870 men, 18 were partially traced and 16 had subsequent asbestos work. These remained in the calculations until lost to observations of until onset of subsequent asbestos work. Expected deaths are based upon white male age-specific death rate data of the U.S. National Office of Vital Statistics, 1949-1971. Rates were extrapolated for 1941-1948 from rates for 1949-1955 and for 1972-1973 from rates for 1967-1971.

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

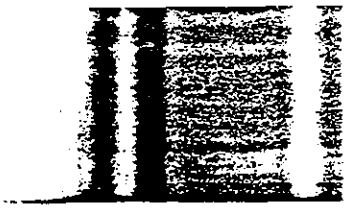


TABLE X. — *Lung cancer among 370 N.Y.-N.J. asbestos insulation workers, January 1, 1963-April 30, 1967.*

	No history of cigarette smoking	History of cigarette smoking
Number of men	87	283
Expected lung cancer (*)	0.15	2.98
Observed lung cancer	0	24

(*) HAMMOND E. C. : Smoking in relation to death rates of 1,000,000 men and women. *In :* NCI Monograph, 1966, *19*, p. 127-204.

TABLE XI. — *Expected and observed deaths of lung cancer among 17,800 U.S. and Canada asbestos insulation workers, January 1, 1967-December 31, 1972. Relation of cigarette smoking.*

	No. of persons	Deaths from lung cancer		
		Expected (*)	Observed	Ratio
Smoking habits not known	6,144	16.76	94	5.6
History of cigarette smoking	9,590	31.60	179	5.7
No history of cigarette smoking	2,066	7.51	2	0.3
Never smoked	1,457	4.40	1	0.2
History of pipe and/or cigar only	609	3.11	1	0.3

(*) 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, pleural, bronchus and trachea, categories 162 and 163 of the International Classification of Diseases and Causes of Deaths, 7th Revision.

5. *Dose response relationship.* There is little doubt that a dose response relationship exists for the several asbestos-associated diseases. This is well demonstrated by the experience of the amosite asbestos factory workers referred to above (Tables IV and IX). Here, workers were exposed to the same fiber during the same period of time, making the same products, in the same city; they varied, however, with regard to their total exposure. Such variations were reflected in differences in the risk of death of both asbestosis and asbestos-associated cancer.

It may be that risk also varies with other factors, such as intensity of exposure, peak exposure, type of fiber, individual susceptibility, etc. Insofar as fiber type is concerned, Wagner [44] has proposed that crocidolite has a greater potential to cause mesothelioma than do other fiber types. Our own studies provide little information concerning this hypothesis. Chrysotile has long been the principal fiber type used in the United States. Comparatively little crocidolite was imported into the United States until World War II [31], and it has not



been used in insulation materials utilized by the asbestos insulation workers investigated in our studies. Our data do not allow comparisons, therefore, between crocidolite and chrysotile risks. Amosite was also imported in the United States in scant quantities until the last part of the 1930's and the 1940's. While 1940 and 1950 deaths among insulation workers [24] are unlikely to have been influenced by amosite exposure, it would be difficult to separate the influence of amosite from chrysotile since that time, since exposure to both fiber types could have occurred and sufficiently long in the past to effect analysis of deaths 1960-1975.

An unresolved question related to the problem of « dose-disease response » relationships is whether or not a « safe » exposure level exists, whether there is, for asbestos, as for any carcinogen, a threshold level. In his animal experiments, Wagner [45] failed to find evidence for a threshold dose of asbestos for mesothelioma. From a practical point of view, one might consider the observations of a continuous reduction in identifiable cancer risk with decrease in levels of exposure suggest that it may be feasible to reach levels at which it will not be possible to statistically define increased risk of cancer of those sites known to be associated with a neoplastic effect of asbestos.

6. *Industry efforts to control exposures.* In the last several years, there has been sharp increase in the scope and intensity of industry efforts to control factory exposures, and the importance of avoidance of cigarette smoking is also now being stressed.

7. *End product use major unresolved problem.* Still unresolved is the control of exposures encountered in the use of asbestos products. This is not only true in the construction industry, shipyards and power plants, but in such uses as brake repair and brake maintenance, as well as in the end product use of some asbestos materials by the general public. Attention has recently been called to one such use, the home utilization of taping and spackle compounds needed for home repairs and rebuilding. Analytical studies showed that many contained asbestos, without warning labels or instructions for the use to special protective precautions [46].

8. *Demolition, waste disposal, maintenance and repair.* Waste disposal, demolition of asbestos-containing structures and the necessity for repair of facilities containing asbestos materials are thorny problems. So far, little research has been devoted to this question, although some notable efforts have recently been made [47], and the U.S. Environmental Protection Agency is seeking to establish regulations aimed at minimizing environmental pollution from these sources [35].

9. *Environmental exposure; conjugal, neighborhood and general community disease.* Although family contact and neighborhood residence have been clearly demonstrated to carry a risk of a asbestos disease, the magnitude of this risk has not been established quantitatively. Indeed, although it is known that mesothelioma may result in such circumstances, there is no information whether other asbestos-associated cancers might also occur in excess among people so exposed.

It is unlikely that extensive asbestosis, sufficient to result in death of cor-pulmonale, will often occur although I have seen cases of moderately extensive pulmonary fibrosis (2/2 in the ILO-U/C classification) among wives of asbestos workers. Studies are currently underway in our Laboratory to provide information on the extent of asbestos-associated disease other than mesothelioma, among family contacts and neighborhood residents. There is no evidence at present that asbestos exposure of the community at large (« general asbestos air pollution ») has been associated with disease risk, although attention has been called to the fact that mesotheliomas not known to be associated with asbestos exposure (approximately 15 % in the series of Greenberg and Lloyd Davies [42] and Webster [48] have also been increasing incidence.

10. *Asbestos contamination of public water supplies.* Asbestos may be found in some food and beverages, presumably as the result of the filtration of these products through asbestos filters or as the result of use of water contamined by asbestos [49]. Water may naturally contain fibers as the result of ground waters coursing through geological formations containing asbestos minerals [38]. In other instances, there is the possibility that water being transported through asbestos cement pipe could, in some circumstances, be contaminated by the asbestos cement material [50, 51]. There are, at present, few data that would allow appropriate evaluation of a potential hazard, especially since the question of asbestos disease resulting from ingestion of the fiber is still unresolved [52].

It has also been fonnd that wide contamination of public water supplies may result from disposal of asbestos-containing waste, as in the contamination of Lake Superior by mining operations [53, 54].

11. *Ineffective therapy.* To the present, little has been achieved in terms of improved survival of asbestos workers suffering from asbestos-induced cancer. (Insofar as asbestosis is concerned, much can be done to lengthen life by appropriate medical surveillance.) In our studies of asbestos insulation workers in the New-York metropolitan area, we observed 1,249 men from January 1, 1963. 370 men had long experience, having been members of the insulation workers' union in 1943. By December 31, 1974, 7 pleural mesotheliomas, 21 peritoneal mesotheliomas and 49 lung cancers were observed. All 28 men with mesothelioma are dead and 48 of the 49 individuals with lung cancer. Among the 879 men who were admitted to the union between 1943 and 1962, 2 pleural mesotheliomas occurred, and 1 peritoneal. All 3 men are dead. Similarly, 10 instances of lung cancer were seen by December 31, 1974; 9 of the 10 are dead.

EPILOGUE

It is sobering to consider that the asbestos disease and asbestos-associated deaths that have recently been seen in the United States are largely related to the inadequately controlled use of asbestos 1930-1940, when some 200,000-300,000 tons per year were used in the United States. At present, approximately 850,000 tons per annum are used (Table XII). To the extent that this use is inadequately controlled, we will see deaths of asbestosis and cancer in the year 2,000, and later [55].

TABLE XII. — *U.S. Consumption of asbestos.*

	Tons
1930	120,000
1940	262,000
1950	727,000
1960	710,000
1973	862,000 (*)

(*) Chrysotile 839,200 tons, amosite 4,273 tons, crocidolite 17,966 tons and anthophyllite 1,162 tons. Source U.S. Bureau of Mines.)

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RÉSUMÉ

L'asbestose aux Etats-Unis, 1918-1975,

par Irving J. SELIKOFF M. D. *(New York)*.

L'auteur résume dans cet article toute son expérience concernant le rôle pathogène de l'amiante.

Il rappelle tout d'abord les grandes étapes historiques. L'asbestose est connue depuis 1918 et, dès 1935, l'asbeste est considérée comme un facteur carcinogène probable. Cependant, pendant environ 25 ans, seules les conséquences mécaniques de l'empoussiérage, insuffisance respiratoire par fibrose interstitielle et cœur pulmonaire chronique, sont prises en compte. Des actions sont menées pour donner droit à réparation à cette nouvelle pneumoconiose professionnelle, mais on continue à utiliser de plus en plus l'amiante (extraction, transformation, applications industrielles diverses). Mais, après cette période de quiétude, il apparaît que le risque majeur du contage asbestosique est l'induction possible d'un cancer. Selikoff observe des cohortes d'ouvriers de

l'amiante pendant et après la seconde guerre mondiale et il met en évidence dans ces groupes une mortalité nettement plus élevée que dans une population ordinaire. D'autre part, cet accroissement de décès est lié à une multiplication des cas de cancer. Enfin, et toujours en comparaison avec une population témoin, il apparait que ces cas de cancers ne correspondent pas à la répartition habituelle des différentes variétés de tumeurs, mais qu'ils sont significativement représentés par des cancers bronchiques et des mésothéliomes pleuraux et péritonéaux; ces derniers sont ordinairement exceptionnels.

D'autres études sont entreprises qui mettent en évidence l'extraordinaire ubiquité du contage asbestosique : industrie de transformation, tissage d'amiante, isolation thermique surtout dans les chantiers navals, flocage des charpentes, peintures incombustibles, etc. Mais les populations urbaines sont aussi soumises à une pollution importante par l'amiante : construction, démolition, proximité des zones industrielles utilisant l'amiante, freins des automobiles, produits courants vendus sans indications ni précautions d'emploi, contages familiaux par l'intermédiaire des vêtements des ouvriers, déchets industriels déversés dans les rivières et les lacs dont l'eau est utilisée pour la consommation.

En fin d'exposé, quelques données sont précisées :

— Il est de règle qu'il existe un long délai (jusqu'à 40 ans) entre le début de l'exposition et l'apparition du cancer. Une exposition intense et brève peut être aussi dangereuse qu'un contage prolongé : les fibres d'amiante inhalées restent dans le poumon.

— Il existe sûrement des co-facteurs carcinogènes, principalement le tabac, qui augmentent le rôle pathogène de l'amiante.

— La notion de relation « dose-effet » n'est pas parfaitement démontrée, mais elle est vraisemblable.

— Les ressources thérapeutiques vis-à-vis des mésothéliomes sont nulles; il est donc nécessaire d'établir une prophylaxie de l'empoussiérage asbestosique. Des contrôles de pollution atmosphérique permettent de faire réduire l'empoussiérage de certaines zones urbaines ou de certains postes de travail. Des doses permises sont déterminées. Une réduction et un contrôle général de l'utilisation de l'amiante s'imposent d'urgence : ceci est plus facilement réalisable en milieu industriel que dans la vie quotidienne, où la présence de l'amiante est beaucoup plus insidieuse.