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III. COMMUNITY EFFECTS OF NONOCCUPATIONAL ENVIRONMENTAL ASBESTOS EXPOSURE

Irving J. Selikoff, M.D., F.A.P.H.A., and E. Cuyler Hammond, Sc.D., F.A.P.H.A.

Introduction

THERE has long been curiosity concerning community exposure to pneumoconiosis-producing dusts, partly because the assumed "spill-over" from primary industrial sources might be expected to yield information concerning effects of minimal exposure and partly because community studies were needed to give base-line data against which to evaluate findings in groups of miners living in these communities.[1,2] Nevertheless, it was not until the serious consequences of neighborhood beryllium disease were clarified[3,4] that the important potential of community exposure to pneumoconiotic dusts was widely recognized.

In the recent past, there has been growing interest in this problem, ranging from the health effects on children of cement and similar industrial dusts in Rumania[5] and neighborhood byssinosis in Egypt,[6] to environmental pneumoconiosis adjacent to the Rajasthan desert.[7] We may anticipate that such interest will increase for a number of reasons. First, because of growing concern over air pollution in general and particulate air pollution in particular; second, because of the appreciation that the biological activity of known pneumoconiotic dusts is not limited to heavily exposed workmen, but may affect others indirectly exposed in the community; and, lastly, because study of community effects of pneumoconiotic dusts may suggest methodology and technics suitable for the investigation of other environmental factors, including inhaled dusts largely unidentified at present and of unstudied biological importance.

Environmental Asbestos Disease

It may be profitable to review studies of one pneumoconiotic dust, asbestos, in the light of these suggestions. The possibility of community asbestos disease has been known almost as long as asbestosis itself. In 1927, shortly after republication of the case report which gave the pneumoconiosis its name,[8] Haddow[9] reported finding asbestos bodies in the lungs of a person living close by an asbestos factory. Other neighborhood cases of asbestos disease have been reported since.[10-12] Bobileva, et al.,[13] reported experiences suggesting impaired health in children living downwind of an asbestos plant in the Soviet Union.

The import of such scattered observations was muffled, however, until the reports of three germinal studies, within a short time of each other: Kiviluoto (1960)[14]; Wagner, Sleggs, and Marchand (1960)[15]; and Thomson, Kaschula, and MacDonald (1963).[16] In each, a pathological "marker," considered almost pathognomonic for asbestos exposure, was unexpectedly found in groups in the general population not known to be industrially exposed to asbestos.

Stigmata of Environmental Asbestos Exposure

Pleural Calcification — Scattered instances of calcification of the pleura, particularly the parietal pleura, were observed in asbestos workers during the

1930s and 1940s, but their significance as a roentgenologic sign of asbestos exposure was not appreciated until the report of Jacob and Bohlig in 1955, in which such calcification was described in approximately 5 per cent of 343 Dresden asbestos workers.[17] Investigations since then have demonstrated the extraordinary frequency of this finding among asbestos workers and have emphasized a cardinal feature—that it is rarely seen in less than 20 years from onset of exposure (Table 1).[18]

Kiviluoto discovered 499 instances of such calcification during a community x-ray survey among 6,312 adults in the Kuusjarvi commune in Finland. Struck by this, and the absence of any such cases among 7,101 adults in the neighboring Ilomantsi commune, he astutely observed that the first commune contained an asbestos mine and postulated that the people with calcification (farmers, housewives) had been subject to endemic environmental asbestos exposure.[14] His countrymen, Laamanen, Noro and Raunio, subsequently reported air studies showing asbestos dust at distances up to 50 km from the mines, including deposits ranging from 1.52 gm/100 m³/month 4 km away to 34.6 gm/100 m³/month at 0.5 km.[19]

Pleural Plaques—Pleural fibrosis is another common pleural complication of asbestos exposure but, when diffuse, need not have the same diagnostic significance as calcification. However, such fibrosis may occur in more or less discrete plaques, and Kiviluoto has called attention to these as additional pleural stigmata of asbestos exposure.[20]

Pleural Mesothelioma—Neoplasms of the pleural surface in association with asbestosis have been known for over 30 years. Weiss suggested an etiologic relationship in 1953,[21] but such proposals faced the difficulty of infrequency of reported cases and the absence of epidemiological studies to suggest that these were other than chance associations. Wagner's report changed this. He investigated 33 cases of diffuse pleural mesothelioma in the North Western Cape Province of South Africa (an asbestos-producing area) and in 32 cases demonstrated at least suggestive evidence of potential asbestos contact, usually nonoccupational, as, for example, residence in an asbestos area.[15] Since this report, diagnosis of pleural mesothelioma has been a signal to seek previous asbestos exposure.

Asbestos Bodies in General Population—Asbestos fibers when in the lung, have the capacity of becoming characteristically coated; these fibers form asbestos bodies which, when seen, can hardly fail to attract attention. This was so even before asbestos disease was known (Marchand, 1906).[22] Thomson took advantage of this phenomenon to demonstrate the frequency of inhalation of asbestos by the general public. He examined smears of lung bases in 500 consecutive autopsies in Cape Town, and in 26.4 per cent found asbestos bodies.[16]

While asbestos bodies do not have the same significance as calcification or mesothelioma (disease may be absent and no conclusion concerning time of inhalation can be drawn), Thomson's observations have been very important in demonstrating the frequency of community exposure to asbestos at present in many areas of the world. Simultaneously these observations raise the serious question whether inhalation of presumably small amounts carries a concomitant risk of widespread community asbestos disease.

Table 1—Time of onset of radiologically evident pleural calcification among 1,117 asbestos insulation workers

Years from onset of exposure	No.	Chest roentgenogram		Grade of calcification		
		% normal	% calcification	1	2	3
40+	121	42.1	57.9	37	20	13
30–39	194	65.4	34.5	46	15	6
20–29	77	89.6	10.4	8	0	0
10–19	379	98.9	1.1	5	0	0
0–9	346	100.0	0.0	0	0	0
Total	1,117			96	35	19

Recent Epidemiological Investigations

Stimulated by the above observations, studies of environmental asbestos disease have been extended. First, the relationship of mesothelioma to asbestos has been clarified. Peritoneal mesothelioma has also been shown to be associated; Enticknap and Smither[23] found 9 such tumors in 52 consecutive autopsies in one asbestos factory. Large collections of asbestos-associated mesotheliomas have been published.[24-26] The significant incidence of asbestos-related mesothelioma has now been demonstrated in our laboratory. In a prospective 52-month study of 370 experienced asbestos insulation workers, from January 1, 1963, to April 30, 1967 (survivors of a larger group of 632 followed 1943-1962), there were 94 deaths. Ten were due to mesothelioma, three to pleural and seven to peritoneal[27] (Table 2).

Perhaps more germane have been the studies of Newhouse and Thompson.[28] Investigating 76 mesotheliomas at London Hospital, they found 31 occurred in asbestos workers. Of the remaining 45, 20 occurred in individuals living within half a mile of an asbestos factory or in the household of an asbestos worker. Similar findings were reported by Elmes and Wade in Belfast.[29] Thomson's observations have also been confirmed: 25 per cent to 50 per cent of adults coming to autopsy in Pittsburgh,[30] Finland,[31] Miami,[32] Montreal,[33] New York,[34] and Belfast,[35] are now found to have asbestos bodies in their lungs.

Table 2—Observed and expected number of deaths among 370 asbestos insulation workers during the 52-month period from January 1, 1963, through April 30, 1967. Expected numbers are based upon United States mortality data disregarding the smoking habits of the subjects.

Cause of death	Observed deaths	Expected deaths
Total, all causes	94	47.5
Total cancer (all sites)	49	8.6
Cancer of lung, pleura, bronchus, and trachea	27	2.3
Bronchogenic carcinoma	24	*
Pleural mesothelioma	3	†
Peritoneal mesothelioma	7	†
Cancer of stomach	3	0.6
Cancer of colon and rectum	5	1.2
Cancer of all other sites combined	7	4.5
Asbestosis	15	0
Heart and circulatory disease, including stroke	22	28.5
All other causes of death	8	10.4

* United States data not available, but figure should be only slightly less than 2.3.
† United States data not available, but these are rare causes of death in the general population.

Problems and Perspectives

The foregoing overview indicates that at least one pneumoconiotic dust, asbestos, may be widely disseminated into the general community from industry sources or end-product use; that such dissemination may produce detectable disease; and that methods to study the question can be designed. It would be misleading, however, to suggest that the situation has been clarified, even concerning asbestos, and totally inaccurate concerning other pneumoconiotic dusts. The following questions, for example, require study and explanation:

1. Epidemiologic studies are concerned with the "general public." But insofar as pneumoconiotic dusts are concerned, how "general" is the public? E. L. Collis many years ago described pneumoconiosis in dock workers after inhaling coal dust;[36] their counterparts, 50 years later, were truck drivers who inhaled asbestos at a construction site. When we consider that in some countries 5 per cent of the employed population works in the asbestos-rich construction industry, we may appreciate that indirect occupational exposure may constitute a significant proportion of "community exposure," to be supplemented by considerable unrecognized occupational asbestos exposure. The latter would include the occasional use of asbestos materials by workmen not usually considered prone to asbestos exposure; such uses would include a carpenter sawing asbestos building board, a plumber using asbestos rovings for joints, and an electrician stripping asbestos-covered wire and cable. With over 3,000 uses for asbestos, now currently known,[37] opportunities for indirect and otherwise unrecognized occupational exposures may be frequent.

We are now studying asbestos bodies in the lungs at autopsy in several thousand consecutive deaths in three general hospitals in New York City, and concomitantly we are investigating occupational and other characteristics. Preliminary data suggest that unrecognized occupational sources may add to, and override, general community asbestos exposure (Table 3).

2. The existence of wide potential for direct or indirect occupational exposure has not been considered sufficient, nevertheless, to explain the frequent occurrence of asbestos bodies in the lungs of people coming to post mortem in urban communities. It should immediately be said that neither Thomson in his original communication nor other investigators since undertook to prove that the "asbestos bodies" found by them were actually the result of inhalation of asbestos fibers and were not, indeed, the result of tissue reaction to some other mineral fiber yielding an end-product which had the appearance of those bodies characteristically seen in large numbers in an asbestos worker's lungs. Studies to clarify this aspect of the problem are in progress.

While there is no doubt concerning the frequency of what appear to be asbestos bodies in autopsied lungs, the significance of this observation is not currently clear. Discovery of asbestos bodies in routine post-mortem studies is simply evidence at best that *at some time* asbestos fibers have been inhaled, without indicating when such inhalation occurred. It has long been known that asbestos bodies can be formed in human lungs in two or three months, or perhaps less.[38] Disease could be anticipated only as a result of inhalation that had occurred a considerable time before.[39] On the other hand, it is likely that most asbestos now being discovered post mortem in persons who have not been industrially exposed has been of recent deposition, since there was previously much less asbestos about to pollute the air. For example, annual world production of asbestos was 500 tons in 1890, 200,000 tons in 1920, 500,000 tons in 1930, compared with 4,000,000 tons per year at present.

Table 3—Asbestos bodies in the lungs of a sample of persons in the general population at autopsy in New York City, 1966. Preliminary analysis by occupational categories.

Sex and occupation	No. of subjects	With asbestos bodies	
		No.	%
Men			
Blue collar workers	197	120	60
White collar workers	111	45	41
Women: Housewives	47	14	30
Total	355	179	50

3. *Asbestos air pollution* — General community air pollution by asbestos microparticles may occur from industrial sources or from disintegration or use of asbestos products. Under certain circumstances, as noted, it is probable that asbestos disease may occur. But it is by no means proved that such general asbestos air pollution, presumably with only small numbers of fibers inhaled, results in widespread disease. Cases of pleural calcification among Kiviluoto's farmers may not be evidence of asbestos air pollution from the local mine and mill, a facile explanation. This explanation is not available, for example, for Hromek's finding of 273 instances of similar pleural calcification during a survey of 9,871 persons in an agricultural area of Czechoslovakia with no asbestos mine or other known exposure.[40]

An alternative explanation can be derived from the recent experiences of Zolov, Bourilkov, and Babadjov.[41] These investigators found instances of pleural calcification among farmers (largely tobacco) in the Avren region of Bulgaria (5.6 per cent of males and 2.8 per cent of females examined), in about the same percentage as among the workmen (recruited from the farming population) in a nearby asbestos mine. The mine could hardly have been the only source, since it had been open for less than two decades (latent period). Examination of the soils worked by the affected farmers demonstrated asbestos minerals to be present; even the stone fences were made of outcroppings of anthophyllite mineral. Such pleural disease must be categorized as the result of intimate exposure under unique conditions, among certain agricultural workmen and their families, rather than what may be called general asbestos air pollution.

Much more study of asbestos air pollution is required—its extent, sources, range, persistence—as a background for appropriate epidemiological investigations concerned with its disease potential.

4. *Mesothelioma* — Asbestos workers frequently die of mesothelioma, and other cases are often found to have had close environmental contact.[15,26,35,42,43] But is there any disease potential as the result of general asbestos air pollution? So far, there seem to be few mesothelioma deaths in the general population. A review of 31,652 consecutive deaths, among over 1,048,183 people in the general public (1959-1962), revealed only 3 cases of mesothelioma.[44] Moreover, is asbestos the only pneumoconiotic cause of mesothelioma? Wagner produced experimental mesothelial tumors with silica as well as with asbestos,[45] as did Hueper with polyurethane.[46]

5. The statistical and epidemiological difficulties in evaluating the community effect of an environmental agent, in a disease process with *multiple potential causes*, is seen with lung cancer. This is the most common tumor among asbestos insulation workers at this time; one in five deaths is caused by this neoplasm.[47]

With this lead, asbestos bodies are being sought by a number of investigators among random cases of lung cancer in the general population to find a possible link to asbestos. But how allow for

Table 4—Observed and expected number of deaths due to bronchogenic carcinoma among 370 asbestos insulation workers during the 52-month period from January 1, 1963, through April 30, 1967, by smoking habits

Smoking habits	Workers observed*	Observed deaths	Expected deaths
Never smoked regularly	48	0	0.05
History of pipe, cigar smoking only	39	0	0.13
History of regular cigarette smoking†	283	24	2.98
Total	370	24	3.16

* All with more than 20 years from onset of exposure.
† Includes cigarette smokers who also smoked pipe or cigars.

the effect of cigarette smoking which overwhelms the asbestos effect? Thus, of the 370 asbestos workers referred to above, all with more than 20 years since onset of exposure, followed prospectively for 52 months (January 1, 1963 to April 30, 1967), 87 did not smoke cigarettes. None of these men died of lung cancer. On the other hand, 283 had a history of cigarette smoking; 24 died of lung cancer or eight times the expected number, age and smoking habits taken into account (Table 4).

6. In demonstrating pathogenetic and etiological relationships, chronic disease epidemiology has frequently faced the difficulty of a long period of clinical latency. Asbestos disease is another example. We have recently been able to define the range of such latency for one group of workmen exposed to asbestos—asbestos insulation workers. Table 5

Table 5—Years from onset of asbestos exposure to death of 113 workers who died of bronchogenic carcinoma, mesothelioma, or asbestosis.

Years onset of exposure to death	Total	Bronchogenic carcinoma	Pleural mesothelioma	Peritoneal mesothelioma	Asbestosis
20–24	3	3	–	–	–
25–29	9	8	–	–	1
30–34	25	17	3	2	3
35–39	30	16	3	4	7
40–44	17	11	–	2	4
45–49	20	9	–	2	9
50–54	5	4	–	–	1
55–59	4	1	–	1	2
Total	113	69	6	11	27

Note: 14 of the 17 mesotheliomas were well established as such. In three the histological diagnosis was less firmly established.

Table 6—Age at time of death of 113 asbestos workers who died of bronchogenic carcinoma, mesothelioma or asbestosis.

Age at death	Total	Bronchogenic carcinoma	Pleural mesothelioma	Peritoneal mesothelioma	Asbestosis
35–39	1	1	–	–	–
40–44	1	1	–	–	–
45–49	1	1	–	–	–
50–54	16	9	3	3	1
55–59	24	15	2	2	5
60–64	21	11	1	3	6
65–69	22	16	–	1	5
70–74	10	6	–	–	4
75–79	14	6	–	2	6
80–84	3	3	–	–	–
Total	113	69	6	11	27

shows the number of years from first asbestos exposure to time of death of 113 consecutive deaths due to asbestos-related diseases (bronchogenic carcinoma, pleural mesothelioma, peritoneal mesothelioma and asbestosis). None of the 113 men died in less than 20 years from onset of exposure, and 101 (89 per cent) of them did not die until 30 or more years had elapsed since onset of exposure. Table 6 shows these same 113 men classified by age at time of death. Only three of the men died under the age of 50 while 49 of them died after age 65.

In epidemiological studies designed to ascertain potential asbestos disease, particularly neoplasia, it is obviously necessary to consider such latency and, in the case of joint exposure to two agents, latency of both must be considered. In practical terms, populations studied should contain as many individuals as possible, with more than 20 years from onset of exposure. Asbestos exposure rarely causes disease in less than 20 years from onset; inclusion of youngsters or others with inadequate duration of exposure will teach us little of the potential of asbestos exposure, and will serve only to dilute pertinent observations. Moreover, investigations should aim to follow exposed individuals as long as possible, since risk does not stop at retirement age or with cessation of occupational exposure.

Data from studies which do not encompass such periods of observation may be difficult to interpret.[49] In one recent study, limited to review of social security records of individuals working in asbestos factories between 1948 and 1951, although 21,755 records were examined, a considerable portion represented men who could not have reached 20 years from first employment at the end of the period of observation. Compounding this difficulty was the fact that over 90 per cent were under the age 55 in 1950, and therefore relatively few had reached even age 65 at the end of the study period. For those who had reached age 65, no data were recorded concerning their fate *after* that age. Conclusions drawn from such data can only be guardedly accepted.

The answer to these and other equally intricate problems will require original approaches and diligent efforts. While animal studies outside the laboratory may be interesting [Webster (1963) studied wild baboons and rats to demonstrate asbestos in the environs of an asbestos mine[49] and we have been studying wild rats trapped in urban and rural areas], no reliable conclusions will be available without appropriate epidemiological studies of well-defined cohorts. This especially applies to human populations with graduated degrees of exposure, including groups with indirect occupational and endemic environmental exposure.

As noted, these will have to be investigated with a long latent period in mind. Various disciplines will have to be enlisted—pathologists, clinicians, epidemiologists, basic scientists, and many others—but the results will warrant the efforts. Indeed, they may lead to a new dimension of the word "pneumoconiosis" which, after all, is only 100 years old.[50]

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Dr. Selikoff is Professor of Environmental Medicine, Mount Sinai School of Medicine of the City University of New York (Fifth Avenue and 100th Street), New York, N. Y. 10029. Dr. Hammond is Vice-President for Epidemiology and Statistics, American Cancer Society (219 East 42nd Street), New York, N. Y. 10017

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