195

PLAINTIFF'S EXHIBIT 1976c04

HOUSEHOLD-CONTACT ASBESTOS NEOPLASTIC RISK *

Henry A. Anderson, Ruth Lilis, Susan M. Daum, Alf S. Fischbein, and Irving J. Selikoff

Mount Sinai School of Medicine
The City University of New York
New York, New York 10029

The occupational risk associated with asbestos exposure has been well documented. Asbestosis—parenchymal fibrosis (described in the ILO/UC Pneumoconiosis Classification as irregular opacities), pleural fibrosis, and pleural calcification—has afflicted many occupational groups exposed to these mineral fibers. Although optimistic forecasts had predicted a decline in the incidence of asbestos-induced disease, recent experience proves that the risk is still present, involving large numbers of workers in various trades. Epidemiologic studies have shown that such asbestos-exposed individuals have a significantly increased risk of death due to malignant (mesothelioma, lung, and gastrointestinal cancers) and nonmalignant (asbestosis) diseases.

Asbestos pollution from industrial sources was not anticipated to pose any significant health hazard to the general public. However, Wagner's work in South Africa and Newhouse's studies in the United Kingdom suggested that the risk had already spread beyond the factory, mine, or mill gate. In 1960, Wagner et al.[1] reported mesothelioma in nonoccupationally asbestos-exposed individuals. In 1964, Newhouse et al.[2] reported nine cases of mesothelioma in family contacts of asbestos workers and eleven cases among individuals whose only identified asbestos exposure was associated with living within one-half mile of an asbestos factory. These initial case reports were not isolated occurrences, peculiar to only one or two regions (TABLE 1). Additional reports from nine countries have brought the total number of reported cases of household mesothelioma to 37.[1-16] Thus, household asbestos contact has been established as being potentially hazardous.

Such case reports did not assess the actual incidence or extent of risk to household members or the general public, nor did they give any suggestion of the prevalence of asbestos-associated radiologic changes in such populations. We have investigated the problem in order to determine whether the potential for such disease hazard is common or rare, since such evaluation would assist in assessing measures needed for clinical surveillance and for preventive control measures.

METHODS

We have undertaken a study to assess the risks of nonmalignant and malignant disease associated with household exposure to work-derived asbestos

* This research supported by National Institute of Environmental Health Sciences grant ES 00928.

1976 NYAS

TABLE 1

HOUSEHOLD ASBESTOS EXPOSURE MESOTHELIOMA CASE REPORTS

Great Britain	Newhouse & Thompson (1965)[2]	9
	Rusby (1968)[3]	1
	Ashcroft & Heppleston (1970)[4]	1
	Champion (1971)[5]	1
	Greenberg & Davies (1974)[6]	2
United States	Lieben & Pistawka (1967)[7]	1
	Heller et al. (1970)[8]	1
	Lillington et al. (1974)[9]	1
	Anderson (1975)	4
Canada	McDonald et al. (1970)[10]	1
	McDonald & McDonald (1973)[11]	5
Italy	Rubino et al. (1972)[12]	3
South Africa	Wagner et al. (1960)[1]	1
Australia	Milne (1969)[13]	1
East Germany	Bittersohl & Ose (1971)[14]	1
Scotland	McEwen et al. (1971)[15]	1
West Germany	Knappmann (1972)[16]	1
		37

dust. We have investigated one factory in which serious, well-documented asbestos disease has been recorded.[17] From 1941 to 1954 (when the factory closed), 1664 workers were employed in the factory for varying periods of time, producing amosite asbestos products. Their household contacts, living with them at the time they worked in the plant, are currently being traced and examined. When this process is complete, we will examine in detail the morbidity and mortality experience of this cohort.

Among those already traced, two pleural mesothelioma deaths have been identified. Autopsy tumor tissue has been reviewed and confirmed as pleural mesothelioma.

The first, the daughter of a man who had worked with asbestos more than 13 years, died at age 52. Her household exposure had begun in early childhood. The second, the daughter of a man with five years of asbestos work at the plant, died at age 41. These women were first exposed as children, and both had latency periods—first exposure to clinical appearance of mesothelioma—of over 30 years. The worker with more than 13 years of asbestos exposure had also died from a mesothelioma.

In addition, two living cohort members have biopsy-proven pleural mesotheliomas and are currently under therapy. One is a 32-year-old woman, sister-in-law of a worker, and the other is a 40-year-old son of another plant employee.

CLINICAL STUDY

Concurrent with the mortality study, household contacts of the former asbestos workers were invited to participate in medical examinations to assess

their current health status and the prevalence of early asbestos-related health effects such as the radiographic changes of parenchymal fibrosis, pleural fibrosis, and calcification. Each individual provided a comprehensive occupational, medical, smoking, and respiratory history; 14 × 17 inch PA, right and left oblique chest x rays were taken, as were spirometric and flow-volume screening pulmonary function tests. Physical examination was done by a physician, with emphasis on the chest.

All the chest x rays were read "blind" by a panel of five experienced physicians, using the ILO/UC Pneumoconiosis Classification. The consensus reading was used for statistical analysis.

Results

We have so far examined 354 household contacts. After careful questioning, 17 were found to have had prior personal occupational asbestos exposure (5 had abnormal x rays). Eleven did not live in the household at the time of the index worker's active employment at the asbestos factory (all had normal chest x rays). Table 2 represents the 326 household contacts who lived in the index workers' homes during their employment and who themselves had not had prior occupational asbestos exposure.

The actual intensity of household asbestos contamination and length of each family contact's exposure to such fibers is unknown. We do not know, of course, how much asbestos each worker might have brought home on his clothes, shoes, and/or hair. We do, however, know when the worker was first employed, the total duration of his employment, and, in many cases, the extent of his radiologic disease.

The index workers' length of employment ranged from one day to thirteen years. Fifty percent of the household members examined came from households

Table 2

Distribution of 326 Household Members of Amosite Asbestos Workers by Relationship to Worker

	Relationship	Number Examined		Percentage
Children	Wives	83		25%
	Daughters	118		
	Sons	70	188	58%
Siblings	Sisters	20		
	Brothers	12	32	10%
Others	Mother	7		
	Father	3	23	7%
	Cousins, etc.	13		
Total		326		

TABLE 3

DISTRIBUTION OF HOUSEHOLD CONTACTS BY THEIR INDEX WORKERS LENGTH OF EMPLOYMENT

	Total	<1 mo	1.1 mo–1 yr	1.1 yr–5 yr	5.1–10 yr	10.1 +yr
Number of cohort members	326	31 (9.5%)	136 (41.1%)	115 (32.2%)	29 (8.9%)	15 (4.8%)

in which the index workers' length of employment (and thus active contamination of the home) was less than one year. Thirteen percent had employment periods over five years (TABLE 3).

RADIOLOGIC FINDINGS

All examined individuals considered themselves to be healthy. Of the 326, 114 (35%) had chest x-ray abnormalities, (ILO/UC Classification irregular opacities 1/0 or more, and/or calcification and thickening of the pleura) (TABLE 4).

There was a predominance of pleural changes, fibrosis and/or calcification. Pleural changes as the only abnormality were seen in 52 (16%) of those examined; parenchymal fibrosis was found alone in 35 (11%) of those examined (TABLE 5). Combined pleural and parenchymal changes were found in 27 (8%). Only two x rays were read as having parenchymal fibrosis greater than the ILO/UC Classification group 1 (1/0–1/2) (TABLE 6).

FIGURES 1–8 are examples of the radiologic changes seen.

DISCUSSION

Of 326 otherwise healthy household contacts of amosite asbestos workers examined 25–30 years after the onset of presumed household contamination with amosite asbestos, 35% had chest x-ray abnormalities (pleural and/or parenchymal) characteristic of asbestos exposure. We conclude that significant

TABLE 4

X-RAY ABNORMALITIES AMONG 326 HOUSEHOLD MEMBERS OF AMOSITE ASBESTOS WORKERS

X-Ray Findings	Number of Household Members
Pleural thickening present	69 (21%)
Pleural calcification present	19 (6%)
Irregular opacities present	62 (19%)
One or more of above abnormalities	114 (35%)

TABLE 5

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/or pleural calcification	52 (16%)
Irregular opacities the only abnormality	35 (11%)
Irregular opacities, pleural thickening, and/or pleural calcification	27 (8%)
Total	114 (35%)

household contamination with asbestos from industrial sources can and does commonly occur, and may result in characteristic radiologic changes and, less frequently, in asbestos-associated neoplastic disease. Four pleural mesotheliomas have already occurred. When tracing and follow-up of the entire cohort is complete, we will analyze expected and observed deaths by cause to determine whether there is a significant increase in cancer risk with household exposure to asbestos.

Pleural fibrosis and calcification without parenchymal fibrosis were seen more frequently than parenchymal fibrosis alone. The ratio of individuals with parenchymal changes alone to individuals with pleural changes alone was 0.67. Interestingly, this observation differs from the experience of the index amosite asbestos workers, in whom pleural changes as the only abnormality was far less common. The ratio of workers with only parenchymal changes to those with only pleural changes was 1.92. The explanation for this apparent difference in response to these fibers is uncertain. Differences in the intensity and extent of exposure are likely.

The possible relationship of pleural changes to tumor risk is incompletely known. In 1974, Edge [13] reported on the prospective observation of 235 men, former shipyard workers, who had pleural fibrosis and/or calcification on their

TABLE 6

ILO/UC PNEUMOCONIOSIS CLASSIFICATION OF 62 ABNORMAL HOUSEHOLD COHORT X-RAYS

	ILO/UC Classification Irregular opacities			
Total w/Parenchymal Abnormalities	1/0	1/1	1/2	2/2
62	23 (37%)	34 (55%)	3 (4.8%)	2 (3.2%)

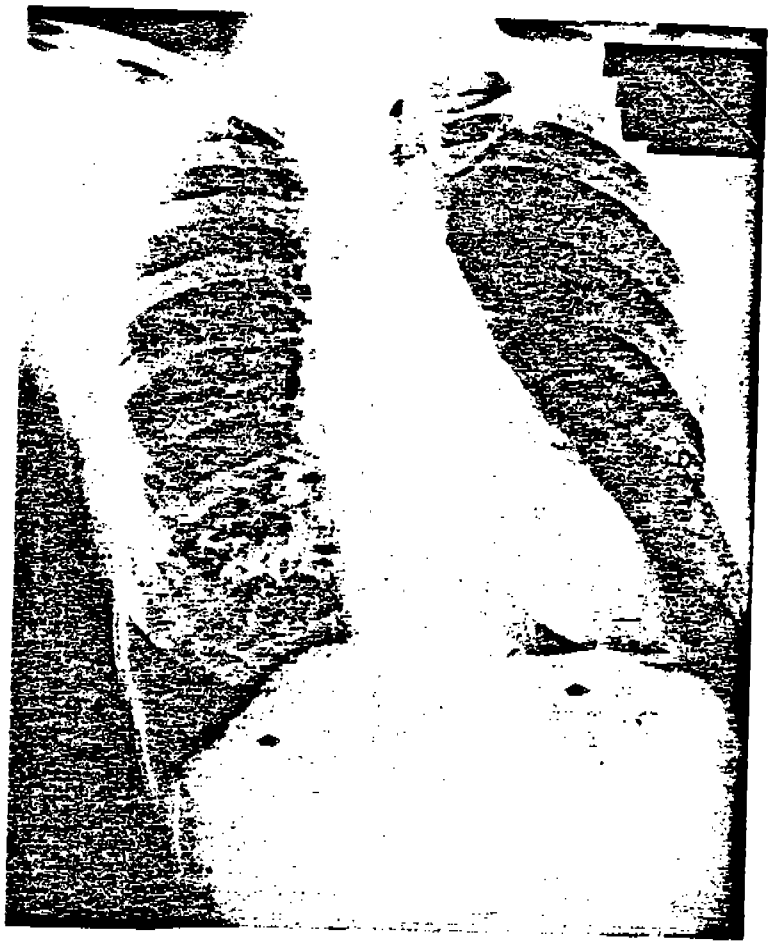


FIGURE 1. Calcified diaphragmatic plaques and lateral-wall pleural thickening in the 61-year-old wife of an asbestos worker employed for 2½ years, 1945–48.

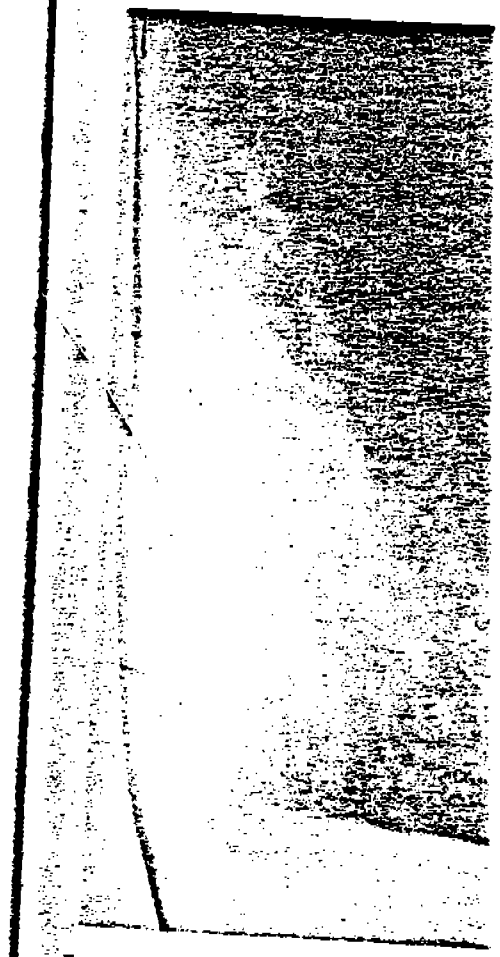


FIGURE 2. Diaphragmatic plaque and lateral
55-year-old wife of an asbestos worker employe

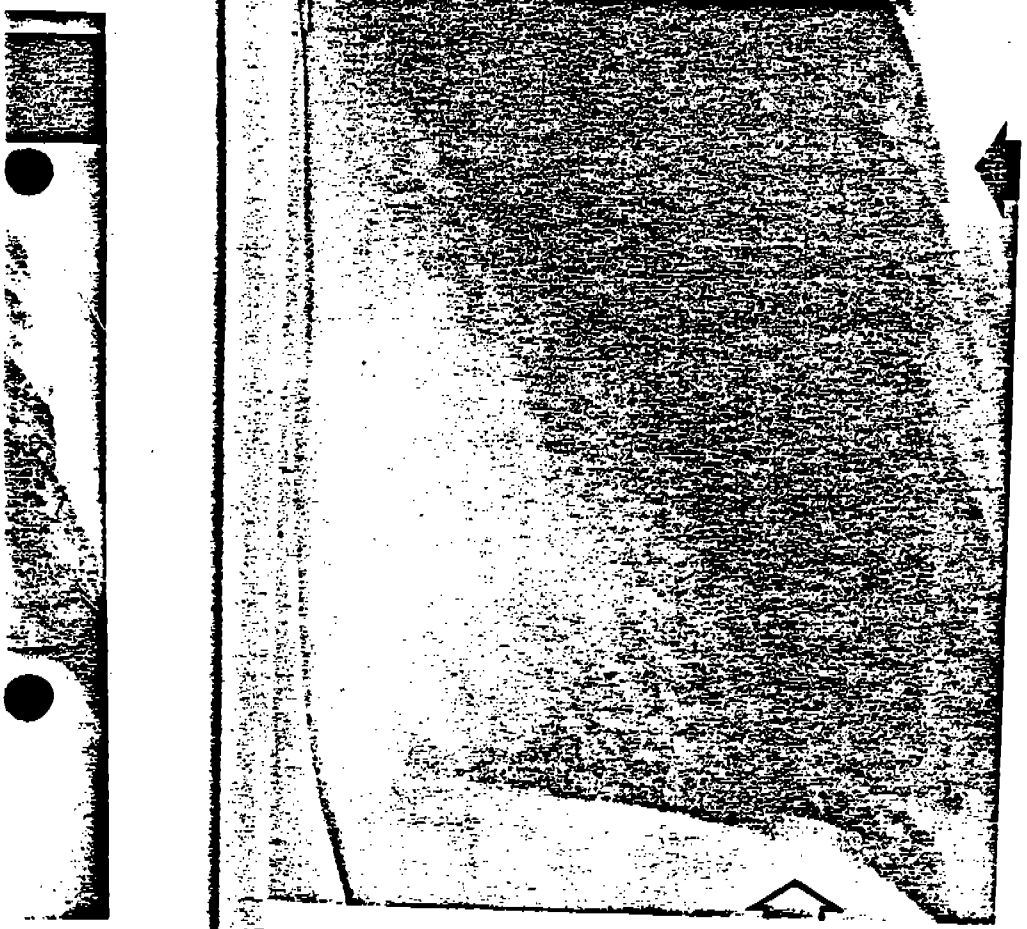


FIGURE 2. Diaphragmatic plaque and lateral chest wall pleural thickening in the 55-year-old wife of an asbestos worker employed for 3 weeks in 1950.

thickening in
45–48.

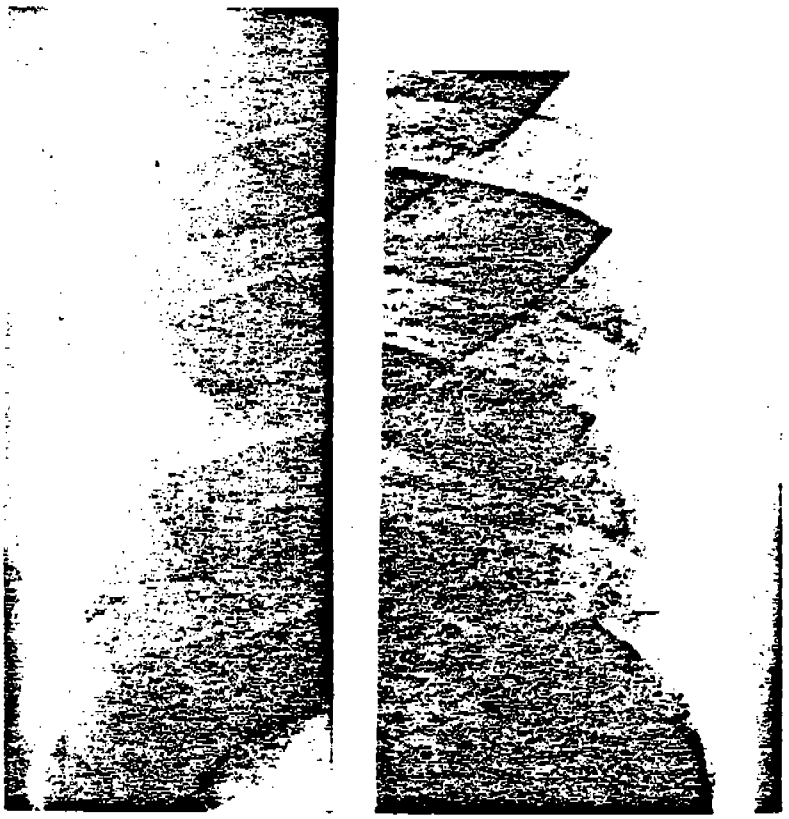


FIGURE 3 (*left*). Lateral-wall pleural thickening in a 43-year-old son of a worker employed for 10 years, 1942–52.

FIGURE 4 (*right*). Lateral-wall pleural thickening in a 31year-old son of an asbestos worker employed for 6 years, 1948–54.



FIGURE 5. Interstitial fibrosis, pleural thickening, and calcification in a 62-year-old wife of an asbestos worker employed for 2 years, 1941–2.



FIGURE 6. Oblique film on case shown in FIGURE 5 more clearly demonstrating the extensive lateral-wall pleural thickening.



FIGURE 7. Pleural mesothelioma in a 52-year-old daughter of an asbestos worker who had been in the trade more than 25 years and in the study plant 1941–54. He also died of a mesothelioma.



FIGURE 8. Pleural mesothelioma in a 41-year-old daughter of an asbestos worker employed for 5 years, 1943–48.

chest x rays in 1970. Of the 70 deaths recorded, 1970–1973, 17 (24%) were from mesothelioma. Further study is needed to investigate the association of pleural fibrosis and calcification and the occurrence of mesothelioma of the pleura. The prospective study of this household-contact cohort should help clarify this relationship, and also the role of such dose-disease response variables as the effect of age at first exposure, possible sex differences in response, the effect of intensity of exposure, and the effect of interrupted versus continuous exposure.

These preliminary data available show significant prevalence of asbestos-

related radiographic changes. Adequate industrial hygiene procedures should not only protect exposed workers, but should prevent environmental pollution as well, including household, neighborhood, and general community contamination.

References

1. Wagner, J. C., C. A. Sleggs & P. Marchand. 1960. Diffuse pleural mesothelioma and asbestos exposure in N.W. Cape Province. Br. J. Ind. Med. 17: 260–271.
2. Newhouse, M. L. & H. Thompson. 1965. Mesothelioma of the pleura and peritoneum following exposure to asbestos in the London area. Br. J. Ind. Med. 22: 261–269.
3. Rusby, N. L. 1968. Pleural manifestations following the inhalation of asbestos in relation to malignant change, J. Royal Naval Med. Serv. LIV(2): 142–148.
4. Ashcroft, T. & A. G. Heppleston. 1970. Mesothelioma and asbestos on Tyneside—a pathological and social study. *In* Pneumoconiosis. Proc. Int. Conf. Johannesburg, So. Africa, 1969. H. A. Shapiro, Ed. : 177–179. Oxford Univ. Press. Capetown, So. Africa.
5. Champion, P. 1971. Two cases of malignant mesothelioma after exposure to asbestos. Am. Rev. Resp. Dis. 103: 821–825.
6. Greenberg, M. & T. A. L. Davies. 1974. Mesothelioma Register 1967–68. J. Ind. Med. 31: 91–104.
7. Lieben, J. & H. Pistawka. 1967. Mesothelioma and asbestos exposure. Arch. Environ. Health. 14: 559–563.
8. Heller, R. M., J. L. Janower & A. L. Weber. 1970. The radiological manifestations of malignant pleural mesothelioma. Am. J. Roent. 108(1): 53–59.
9. Lillington, G. A., R. W. Jamplis & J. R. Differding. 1974. Letter to the Editor. Conjugal malignant mesothelioma. New Engl. J. Med. 291: 583–584.
10. McDonald, A. D., A. Harper, O. A. El Attar & J. C. McDonald. 1970. Epidemiology of primary malignant mesothelial tumors in Canada. Cancer 26: 914–919. October.
11. McDonald, A. D. & J. C. McDonald. 1973. Epidemiologic surveillance of mesothelioma in Canada. Can. Med. Assoc. J. 109: 359–362.
12. Rubino, G. F., G. Scansetti, A. Donna & G. Palestro. 1972. Epidemiology of pleural mesothelioma in North-western Italy (Piedmont). Br. J. Ind. Med. 29: 436–442.
13. Milne, J. 1969. Fifteen cases of pleural mesothelioma associated with occupational exposure to asbestos in Victoria. Med. J. Australia 2: 669–673.
14. Bittersohl, G. & H. Ose. 1971. Zur Epidemiologie des Pleuramesotheliomas. Z. Gesante Hyg. 17: 861–864.
15. McEwen, J., A. Finlayson, A. Mair & A. M. M. Gibson. 1971. Asbestos and mesothelioma in Scotland. Int. Arch. Arbeitsmed. 28: 301–311.
16. Knappmann, J. 1972. Beobachtungen an 251 obuzierten Mesotheliom-Fallen in Hamburg (1958–1968). Pneumonologie 148: 60–65.
17. Selikoff, I. J., E. C. Hammond & J. Churg. 1972. Carcinogenicity of amosite asbestos. Arch. Environ. Health 25: 183–186.
18. Edge, J. R. Asbestos related disease in Barrow-in-Furness. Environ. Res. To be published.