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HOUSEHOLD EXPOSURE TO ASBESTOS AND RISK OF SUBSEQUENT DISEASE[1]

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INTRODUCTION

Today, everyone living in an industrialized country is exposed to dusts, fumes and chemicals inadvertently released into the environment during manufacturing, processing or use of finished products. Initially, public awareness was focused on the most obvious pollution types and sources, plumes of malodorous smoke and discharge of pollutants into rivers and lakes. A more recent phenomenon is concern that unrecognized and less obvious environmental pollution might pose a significant health hazard to the general public.

Largely unrecognized was the contamination of workers' homes, and the resulting exposure of wives and children to toxic materials brought from the work place via contaminated clothing. Insidiously, household residents were exposed to materials used and manufactured by the worker living in the household.

It has been known that disease related to toxic industrial substances can occasionally be found among household contacts of exposed workers, as well as among individuals living in the neighborhood of factories producing or using these toxic substances. Nearly 40 years ago, chloracne was described among family contacts of a chemical worker employed in a polychlorinated biphenyl (PCB) manufacturing plant (Commonwealth of Pennsylvania, 1935). The PCB responsible for the chloracne came from the worker's contaminated coveralls. His clothes, shoes and even his hands and hair contained enough PCB to cause death among his unsuspecting family.

That significant household contamination with asbestos dust brought home on the clothes of workmen can occur has been assumed on the basis of reported cases of pleural mesothelioma in individuals whose only asbestos contact would have been from residence in an asbestos worker's household (Newhouse and Thompson, 1965). The growing incidence of such mesothelioma cases has focused new attention on the potential health risk, especially neoplastic, associated with environmental exposure to asbestos.

Besides mortality experience and the appearance of rare "signal tumors" such as mesothelioma, other more readily obtainable means exist to assess whether changes have resulted from environmental asbestos exposure. Thus, few attempts have been made to assess the prevalence of asbestos-associated radiographic abnormalities among household contact of asbestos workmen.

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POPULATIONS STUDIED

We are investigating the status of household contacts of 1,884 asbestos workers employed in a factory which produced amosite asbestos products from 1941 to 1954.

The worker population has a well-characterized clinical and mortality experience since they have been under clinical observation by one of us (Irving J. Selikoff) for more than 20 years (Selikoff *et al.*, 1964, 1972). Table 1 summarizes the mortality experience of the sub-group (933) first employed from 1941 to 1945. The ratios of observed to expected deaths due to lung and gastrointestinal cancer, malignant pleural and peritoneal mesothelioma and asbestosis show the extraordinary experience of this population. Additional analysis has revealed that workers from this group who had as little as one month of employment began to experience excess deaths 30 years later (Seidman *et al.*, 1976).

TABLE 1. Expected and Observed Deaths Among 933 Amosite Asbestos Factory Workers Employed 1941–1945, Observed to December 31, 1977

	Deaths 1941–1977		
	Expected[1]	Observed	Ratio
All Deaths	366.62	594	1.62
Cancer, all sites	73.35	185	2.52
Lung cancer	19.16	100	5.22
Pleural mesothelioma[2]		6	-
GI cancer	21.55	31	1.44
All other cancer	32.64	47	1.44
Asbestosis[2]		30	
Other non-infectious respiratory disease	8.47	19	2.24
All other causes	286.80	350	1.22

[1] Expected deaths based upon age-specific death rate data for New Jersey white males in corresponding years.

[2] Death rates not available, but these have been rare causes of death in the general population.

CONTROL POPULATION

For comparison purposes in the chest x-ray interpretation, a control population of urban New Jersey residents who lived in the same community as the study population was identified from the general medical clinic at the facility used to examine the workers and household members. Controls were matched for age and sex. Clinical records were reviewed and only those with no known personal occupational contact with asbestos were included.

METHODS

In 1973, we began tracing all the household contacts who could be identified as being resident in the workers' households during the period of their active asbestos employment. All individuals were invited to participate in periodic special clinics established previously for prospective observation of the workers.

Examinations included comprehensive occupational and residential histories, past and current medical history, history of current symptoms, smoking and Medical Research Council of Great Britain respiratory questionnaires, physical examination, 14" ×17" postero-anterior, and right and left oblique chest x-rays, and spirometry.

The chest x-rays of household contacts, factory workers and controls were intermixed and read without knowledge of exposure category. The ILO U/C Pneumoconiosis Classification of 1971 was utilized by the authors and a consensus reading reported for statistical analyses. A film was classified as abnormal if one or more of the following abnormalities was recorded: irregular opacities 1/0 or more; any lateral wall pleural thickening; any pleural calcification; pleural plaques on the diaphragm.

RESULTS

Table 2 gives the distribution of 704 household contacts who have so far undergone clinical examinations by relationship to the index worker. Fifty-six percent of those examined were children. Occupational and residence histories revealed that, in addition to household asbestos exposure, 46 individuals had had occupational exposure to asbestos or other fibrogenic dusts. Thirty-two did not live

TABLE 2. Distribution Of 704 Household Contacts Of Amosite Asbestos Workers By Relationship To Index Worker

Relationship	Number	Percentage
Wives	155	22.0%
Daughters	222	31.5%
Sons	178	25.2%
Siblings	81	11.5%
Parents	12	1.7%
Other	56	8.1%
Total	704	

in the index household during the workers' asbestos handling employment. The 78 individuals listed in Table 3 did not fit the criteria for inclusion in the study and have been excluded from the following analyses. This report is concerned with the 626 household contacts who had lived in the household of an asbestos worker while he was actively employed in the amosite asbestos factory, and who themselves had not had an occupational exposure to asbestos.

326 control subjects were identified. The age and sex distribution of the subjects and controls were not statistically significantly different.

TABLE 3. Occupational Exposures Leading To Exclusion From Household Asbestos Contact Study

Direct asbestos trade	18	23.1%
Automobile brake repair	7	9.0%
Drywall construction taper	1	1.3%
Shipyard work	5	6.4%
Other indirect asbestos exposure	13	16.7%
Other fibrogenic dust exposure	2	2.6%
Resident in index household after active asbestos employment period	32	41.0%
Total excluded	78	

Table 4 shows the age distribution of the household contacts. One-third of those examined were under the age of 40.

TABLE 4. Age Distribution Of 626 Household Asbestos Contacts

Age	Number	Percentage
20-29	43	6.9%
30-39	161	25.7%
40-49	182	29.1%
50-59	136	21.7%
60-69	69	11.0%
70 +	35	5.6%
Total	626	

Table 5 shows the distribution of examined household members by the length of time their index worker was actively employed in the asbestos factory and by year that employment began. Duration of active employment, and presumably concomi-

TABLE 5. Distribution of 626 Household Asbestos Contacts by Duration of Index Workers' Employment

Year first exposed	Total # examined	1 yr.	1-5yrs.	5-10yrs.	10+yrs.
1941-45	413 (66.0%)	166 (40.2%)	196 (47.5%)	30 (7.3%)	21 (5.1%)
1946-54	213 (34.0%)	97 (45.5%)	103 (48.4%)	13 (6.1%)	0
Total	626	263 (42.0%)	299 (47.8%)	43 (6.9%)	21 (3.4%)

tant active daily household contamination was less than one year for 42% of those examined. In only 10% was employment, with increasing household contamination, of more than five years duration. We do not know the dimensions of the asbestos contamination which remained in the house after the worker changed employment. However, amphibole asbestos has been found in settled dust of these workers' homes even 30 years after they ceased their asbestos work (Nicholson, 1979) thus attesting to the persistence of asbestos in the home environment.

At the time of their examination all study participants were more than 20 years from onset of their first asbestos exposure. Two-thirds had already exceeded 30 years from onset of exposure.

Overall, 225 (35.9%) of the 626 had chest x-ray abnormalities (most commonly) associated with past asbestos exposure (Table 6). In the control population, only 15 (4.6%) of the 326 films reviewed had similar abnormalities. The prevalence of abnormalities is statistically significantly different at the $p < .001$ level.

TABLE 6. Chest X-ray Abnormalities[1] (1973-1976) Among 626 Household Contacts Of Amosite Asbestos Workers And 326 Comparison Individuals

	Total number examined	Number with abnormal x-rays
Household contacts	626	225 (35.9%)
Comparison group	326	15 (4.6%)

[1]By ILO U/C Pneumoconiosis Classification. Irregular opacities 1/0 or greater. All grades of pleural thickening, calcification and solitary plaques. Blunting of costophrenic angles, not included.

Table 7 summarizes the types of radiologic abnormalities. Pleural abnormalities were the most common. No pleural calcifications were seen in the control group while 8.1% of the study participants had calcification present. Pleural thickening (fibrosis) was seen on 19% of the study films compared to 1.2% of control films. The most common abnormality among controls was that of small irregular opacities which were seen in 3.4%. Nearly 17% of the study population had small irregular opacities. For all types of abnormalities, the prevalences were statistically significantly different.

TABLE 7. Chest X-ray Abnormalities Among 626 Household Contacts of Amosite Asbestos Workers and 326 Individuals in a Comparison Group

	Total examined	Pleural thickening present	Pleural calcification present	Pleural plaques present	Irregular opacities present
Household contacts	626	119 (19%)	51 (8.1%)	50 (8%)	106 (16.9%)
Comparison group	326	4 (1.2%)	0	2 (0.6%)	11 (3.4%)

Table 8 further examines the prevalence of abnormalities. The predominance of pleural disease is striking. Of the abnormal films, 54% had only pleural changes present. In 23% of cases, both pleural and parenchymal abnormalities were recorded. Tables 9 and 10 demonstrate that not only was pleural disease more prevalent, but when present was more extensive. Of the 106 films showing small irregular opacities, only 2 (1.9%) were classified as having profusion category 2. Pleural thickening was more extensive. Of the 119 films showing pleural thickening, 47 (39.5%) had a "b" width of 5-10 mm and 12 (10.1%) had very thick pleura "c" width — greater than 10 mm.

TABLE 8. Classification of 225 Abnormal Chest X-rays Of Household Contacts Of Amosite Asbestos Workers

Type of abnormality present	Number	Percent
Pleural thickening only	64	(28.4%)
Pleural calcification only	16	(7.1%)
Pleural plaques only	18	(8.0%)
More than one pleural change	22	(9.8%)
Total with pleural abnormalities only	120	(53.3%)
Irregular opacities only	53	(23.6%)
Irregular opacities and some pleural change	52	(23.1%)
Total with abnormal findings	225	

TABLE 9. ILO U/C Pneumoconiosis Classification of 106 Household Cohort X-rays with parenchymal abnormalities

Total with irregular opacities	1/0	1/1	1/2	2/2
106	43 (40.6%)	58 (54.7%)	3 (2.8%)	2 (1.9%)

Among occupational groups exposed to asbestos, two factors, duration from onset of exposure and duration of exposure, have been associated with an increase in prevalence of x-ray abnormality. The same relationships were seen in the study participants. Tables 10 and 11 show the distribution of abnormalities by duration from onset of exposure (more than 30 years and less than 30 years). For all the abnormalities described, the prevalence was higher among those with more than 30 years since first exposure. The greatest difference is seen for pleural calcification. The group with longer duration from onset had a three times higher prevalence of pleural calcification than that of the group with less than 30 years from onset.

TABLE 12. Pleural Thickening and Duration of Index Worker Exposure in 626 Household Asbestos Contacts

	1 yr.	1-5yrs.	5-10yrs.	10+yrs.	Total
Number examined	263	299	41	23	626
Pleural thickening	32 (12.2%)	65 (21.7%)	14 (34.1%)	8 (34.8%)	119 (19.0%)

TABLE 13. Distribution Of X-ray Abnormalities Among Household Contacts By Their Relationship To The Index Asbestos Worker

Relationship	Total number examined	Number with abnormal x-ray findings
Wives	151	70 (46 4°o)
Daughters	195	46 (23.6°o)
Sons	148	59 (40°o)
Siblings	76	27 (35.5°o)
Others	56	23 (41.1°o)
Total	626	225 (35.9°o)

TABLE 14. Distribution of Physical Findings In Relation To X-Ray Abnormalities

	Total	Rales[1]	Cyanosis[2]	Clubbing[3]
Normal x-rays	401	13 (3.2%)	0	1 (0.2%)
Abnormal x-rays	225	17 (7.6%)	4 (1.8%)	4 (1.8%)
Total	626	27 (4.3%)	4 (0.6%)	5 (0.8%)

[1] $x^2 = 5.88$ $p < .02$
[2] $x^2 = 7.17$ $p < .01$
[3] $x^2 = 4.25$ $p < .05$

The prevalence of abnormal physical findings among individuals with abnormal chest x-ray findings was statistically significantly different from such findings among those with normal chest x-rays.

DISCUSSION

Inorganic dusts emitted from industrial sources became a recognized hazard when Kiviluoto described an abnormally high prevalence of pleural calcification among 6,312 residents of a district in Finland in which an anthophyllite asbestos mine and mill had been operating for many years (Kiviluoto, 1960). In a contiguous district without an asbestos mine, no such instances of calcification were observed among 7,101 surveyed residents. Subsequently, similar observations were made in other countries (Zolov *et al.*, 1967; Yazicioglu, 1976; Navratil and Trippe, 1972).

Navratil (1972) studied factory workers, neighborhood residents around the workers' factory, household contacts of the workers and an unexposed control population. Table 15 summarizes the results of these surveys. All three exposed populations had an increased prevalence of pleural calcifications.

TABLE 15. Prevalence Of Pleural Calcifications In Populations With Different Sources Of Exposure To Asbestos In Czechoslovakia[1]

	Number examined	Number with pleural calcification
General non-exposed population	8,133	28 (0.34%)
Blood relatives of factory workers	114	4 (3.5%)
Neighborhood residents of asbestos factory	155	9 (5.8%)
Factory workers	755	42 (5.3%)

[1] Navratil *et al.* 1972. Categorization by duration from onset of exposure not available.

The results of our study extend the observations of these and other investigators. Pleural and/or pulmonary disease occurring under the conditions of non-occupational exposure experienced in the home of asbestos workers is more common than previously appreciated. Radiographic examinations 1973-1976 of 626 household contacts of amosite asbestos workers found that 225 (36%) individuals had pleuropulmonary abnormalities characteristic of asbestosis. Consistent with occupationally exposed populations, the prevalence of abnormalities increased among those with a longer period of active household exposure as estimated by duration of the index workers' length of asbestos employment. The importance of sufficient elapsed latency time, duration from first exposure, was also demonstrated. This was most apparent for pleural calcification, which was three times more prevalent among those with more than 30 years from onset compared to those with less than 30 years (10.4% vs. 3.8%). The study begins to demonstrate the time dynamics for the appearance of radiographic abnormalities after this type of environmental exposure. Serial observations of this cohort will provide additional information concerning the interaction of length of exposure and lapsed time from onset of first exposure. For environmental asbestos exposure, it appears that a longer elapsed time is required before disease becomes apparent, compared with direct occupational exposure.

It is apparent from this study and others that for environmental and other low-level asbestos exposures, the radiographic appearance of disease is predominantly pleural. Therefore, the presence of pleural changes when seen in the general population is of considerable significance and, when present in the absence of other medical causes, possible past asbestos exposure should be investigated. In environmental asbestosis, the "classical" radiographic appearance of combined parenchymal and pleural abnormalities is the exception rather than the rule.

In addition to the now recognized increased prevalence of radiographic abnormalities, household asbestos contact carries a risk of developing mesothelioma. Such case reports now total over 50. In a previous report we described four instances of pleural mesothelioma from among the household contacts of the 1,664 index amosite asbestos workers (Anderson *et al.*, 1976). In a recent study by Vianna and Polan (1978), of 52 cases of mesothelioma among women in New York State 1967-1973, nine additional household contact mesotheliomas were reported and they were able to calculate a relative risk factor of 10 for residence in the homes of a worker in an asbestos-related industry. Prospective observation of our cohort will help determine the extent of the malignancy risk which attends such exposure.

Household environmental contamination from industrial sources may be more widespread than hitherto suspected. Asbestos is not unique in its ability to escape from the industrial setting in quantities sufficient to cause disease in the community. PCBs, lead and beryllium have been demonstrated to cause such disease (Baker *et al.*, 1976; Rice *et al.*, 1978; Eisenbud *et al.*, 1949). It must be recognized that asbestos will not remain as the only occupational carcinogen to cause malignancies in individuals environmentally exposed. As more industrial substances are linked to neoplastic and other diseases after 20 to 30 year latency periods, the risk of associated household and environmental exposure must be considered. This study demonstrates that environmental contamination from one industrial source of amosite asbestos commonly occurred and resulted in the appearance of characteristic radiologic changes as well as neoplastic disease.

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