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HOUSEHOLD RISKS WITH INORGANIC FIBERS*

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In reviewing the question of mineral fibers, it is well to start historically. The first case of asbestosis was seen in 1898 by Dr. Montague Murray of Charing Cross Hospital in London. He saw a man who was short of breath, and who died. He told Dr. Murray that he had worked in one of the new asbestos factories in London; the industry had started around 1880. When he died, the autopsy showed diffuse interstitial fibrosis. At that time we used very little asbestos in our country, approximately 6,000 tons a year. We now use approximately 600,000 tons a year.

Following that case, we observed the experience of the growing asbestos industry and accumulated knowledge. One of the things we learned was that, unlike the situation with other dusts, the pleura was often involved when asbestos was inhaled. This does not happen with silica, with diatomaceous earth, with aluminum, coal, carbon black. For reasons that we still do not know, with asbestos the pleura will often show fibrotic plaques — sometimes calcified. They are an indication of asbestos inhalation. They need not produce any disability unless they are thick or completely encircle the lung.

In 1935, at about the time we began to learn about the pleura, Kenneth Lynch, then professor of pathology at the Medical University of South Carolina, reported a very unusual case, a man who had carcinoma of the lung and also asbestosis.

The editor of the *American Journal of Cancer* thought this worth publishing.¹ In 1935 — one may not now appreciate it — lung cancer was a rare disease. There was good reason: not many people were smoking

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bands' clothes when they came home from work. Eleven had merely lived within a half mile of one of the asbestos plants in London, at Barking.⁸ This again suggested that one did not necessarily need much asbestos to produce mesothelioma.

FAMILY CONTACT ASBESTOS DISEASE

This, then, addressed a subject we are now discussing, the problem of family contact disease.

We have been tracing the wives and children of workers employed in an asbestos plant in Paterson, N.J., that operated from 1941 to 1954. The workers have been dying of the usual asbestos diseases.⁹ In tracing workers we contacted the wives and children. We have, so far, examined approximately 750. In the first 626, one third had abnormal roentgenograms: pleural plaques, pleural fibrosis, sometimes even interstitial fibrosis of limited extent.¹⁰

B.G. was an example of family contact disease. She was at our hospital in 1978. She had had a normal chest roentgenogram in 1974. It was taken because her mother had died of mesothelioma. Her father had died of lung cancer; he had been a shipyard worker in Massachusetts. A roentgenogram taken in 1978 showed mesothelioma, from which she died. When she was seen she told us that when her father came home from the shipyard, her mother took his clothes and shook them, with the children playing on the floor nearby.

In the factory population that we are tracing, among the 933 workers, of the first 304 deaths, more than 20 years from onset of employment, we have seen 14 mesotheliomas — approximately 5%. In the first 384 deaths studied among the family contacts, more than 20 years from onset, four were due to mesothelioma — approximately 1%. Equally worrisome, it appears that there will be a significant increase in lung cancer rates in the same group. William J. Nicholson and his colleagues calculate that about nine million workers were significantly exposed from 1940 to 1980 and are currently alive.¹¹ There probably are a similar number of wives and children.

There have been other sources of asbestos exposure in the home — among the do-it-yourselfers, for example, the use of spackle compound to tape joints on wall board. No one had told them, over the years, that most spackle compounds contained 12 to 15% asbestos.¹² Some papier maché used by the New York City Board of Education until recently con-

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tained approximately 50% asbestos. When we went to the New York City Board of Education with this information, it was found that they had purchased 50,000 five-pound bags of such papier maché material for kindergarten use. These have been discarded.

There have been asbestos textiles in the home, such as asbestos gloves. There have been do-it-yourself brake repair work and the do-it-yourself carpenter with asbestos cement board. Many furnaces and their pipes have been insulated with asbestos, needing repair and maintenance — sometimes do-it-yourself.

One of the most curious situations we have encountered was a young woman who called me saying, "My name is so and so. I live in Connecticut. I have just bought a coat and the label says it has 7% asbestos." I said, "Quit your kidding; people are seeing asbestos under every bed." She said, "No, I was in Boston and went to a store with a famous basement, and it was a very good buy." She was right; it was a good buy and it did contain 7% asbestos. When we sampled air near the coat, levels were higher than in some factories. When we washed the coat with other clothes in the same tumbling machine, they became contaminated with asbestos.

We then discussed this with the International Ladies Garment Workers Union. Their members were cutting the fabric. They identified the importer. He was very clever. He knew that when a fabric that has a new fiber was imported, the rate of duty reflected the new fiber. Asbestos fiber had a much lower level of import duty. I have been told that a million dollars was saved by putting in 7% asbestos.

As a postscript, one might mention that

Questions and Answers

MR. HARVEY SACHS: (Princeton University) Erionite is a common accessory mineral in the area of Nevada proposed for the MX missile sites, which will require enormous amounts of excavation.

DR. SELIKOFF: You are correct. Erionite is found in many parts of the United States. It has not been used very much commercially until recently. In general—many of them synthetic—are the molecular sieves of chemistry. There are other

colleagues who continued to smoke.¹⁶

This raises ethical and legal questions. Now that we know that people who have been exposed to asbestos should stop smoking, do we have the ethical obligation to identify and to locate these people to advise them about this risk, giving them the chance to stop smoking, to decrease their risk of dying of lung cancer? Is this a legal responsibility as well?

MR. WILDER: May I follow that up? Hasn't everyone who is living and working in midtown Manhattan, where they used to spray asbestos, been exposed?

DR. SELIKOFF: Probably. None of us should smoke cigarettes.

SPEAKER: There is a product that is used in millions of water softeners called sodium zeolite. Is that zeolite of the same type?

DR. SELIKOFF: No. There are many synthetic zeolites. Zeolites are remarkable chemicals, probably some of the most important now being used in industry. We do not know that nonfibrous, granular zeolites will cause the same kinds of lesions as fibrous zeolites such as erionite, which is similar to asbestos in size and shape. We do not know that other zeolites, now widely used in the petrochemical industry, will have the same effect.

You have raised a very important question. I have recommended that research rapidly be done to see whether the zeolites being used now will be hazardous.

REFERENCES

1. Lynch, K. M., and Smith, W. A.: Pulmonary asbestosis. III. Carcinoma of lung in asbesto-silicosis. *Ann. J. Cancer*, 24:56, 1935.
2. Selikoff, I. J., Churg, J., and Hammond, E. C.: Asbestos exposure and neoplasia. *J.A.M.A.* 188:22, 1964.
3. Selikoff, I. J., Hammond, E. C., and Seidman, H.: Mortality experiences of insulation workers in the United States and Canada, 1943-1976. *Ann. N.Y. Acad. Sci.* 330: 91-116, 1979.
4. Selikoff, I. J., Hammond, E. C., and Churg, J.: Asbestos exposure, smoking and neoplasia. *J.A.M.A.* 204: 106-12, 1968.
5. Hammond, E. C.: Smoking in Relation to the Death Rates of One Million Men and Women. In: *Epidemiological Study of Cancer and other Chronic Diseases*. Monograph 19. Bethesda, National Cancer Institute, 1966, pp. 129-204.
6. Selikoff, I. J., and Hammond, E. C.: Asbestos and smoking. *J.A.M.A.* 242: 458, 1979.
7. Wagner, J. C., Sieggs, C. A., and Marchand, P.: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Br. J. Ind. Med.* 17:260, 1960.
8. Newhouse, M.L., and Thompson, H.: Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br. J. Ind. Med.* 22:261, 1965.
9. Selikoff, I. J., Seidman, H., and Hammond, E. C.: Mortality effects of cigarette smoking among amosite asbestos factory workers. *J. Nat. Cancer Inst.* 65:507-13, 1980.
10. Anderson, H. A., Lillis, R., Daum, S. M., and Selikoff, I. J.: Asbestosis among household contacts of asbestos

- factory workers. *Ann. N.Y. Acad. Sci.* 330:11-21, 1979.
11. Nicholson, W. J., Perkel, G., Selikoff, I. J., and Seidman, H.: Cancer from Occupational Asbestos Exposure: Projections 1980-2000. Banbury Report 9. In press, 1981.
 12. Rohl, A. N., Langer, A. M., Selikoff, I. J., and Nicholson, W. J.: Exposure to asbestos in the use of consumer spackling, patching and taping compounds. *Science* 189:551-53, 1975.
 13. Baris, I., Artvinli, M., Sahin, A., et al.: Occurrence of pleural mesothelioma, chronic fibrosing pleurisy and calcified pleural plaques in Turkey in relation with environmental pollution by mineral fibers. *Rev. Mal. Resp.* 7:687-94, 1979.
 14. Sawyer, R. N.: Indoor asbestos pollution: Application of hazard criteria. *Ann. N.Y. Acad. Sci.* 330:579-86, 1979.
 15. Cochrane, J. C. and Webster, I.: Mesothelioma in relation to asbestos fibre exposure. *S.A. Med. J.* 54: 279-81, 1978.
 16. Hammond, E. C., Selikoff, I. J., and Seidman, H.: Asbestos exposure, cigarette smoking and death rates. *Ann. N.Y. Acad. Sci.* 330:473-90, 1979.

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TABLE I
X-RAY CHANGES IN ASBESTOS INSULATION WORKERS

Onset of exposure (yrs.)	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
	1,117	51.5	48.5	366	126	50

TABLE II
ROENTGENOGRAPHIC EVIDENCE OF PLEURAL ABNORMALITY AMONG
1,117 ASBESTOS INSULATION WORKERS

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

done. A list was made of the 632 men in this union on January 1, 1943. Each has been traced and each is still under observation.^{2,3} By 1977 there should have been 329 deaths; instead, there were 478. Rather than 57 deaths of cancer, there were 210; and, instead of 13 deaths of lung cancer, there were 93. One of every five of these men died of lung cancer (Table III). Instead of no deaths of mesothelioma (in general, this has been so rare in the past that it has not been separately coded in the International Classification of Causes of Death), there were 38. They were excess deaths from gastrointestinal cancer, as well as deaths from asbestosis.

This was a small study. Therefore, on January 1, 1967 the entire membership of this union in the United States and Canada was registered. There were 17,800 men on its rolls on that day. By 1977, after some

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- factory workers. *Ann. N.Y. Acad. Sci.* 330:11-21, 1979.
11. Nicholson, W. J., Perkel, G., Selikoff, I. J., and Seidman, H.: Cancer from Occupational Asbestos Exposure: Projections 1980-2000. Banbury Report 9. In press, 1981.
 12. Rohlf, A. N., Langer, A. M., Selikoff, I. J., and Nicholson, W. J.: Exposure to asbestos in the use of consumer spackling, patching and taping compounds. *Science* 189:551-53, 1975.
 13. Baris, I., Arvinli, M., Sahin, A., et al.: Occurrence of pleural mesothelioma, chronic fibrosing pleurisy and calcified pleural plaques in Turkey in relation with environmental pollution by mineral fibers. *Rev. Mul. Resp.* 7:687-94, 1979.
 14. Sawyer, R. N.: Indoor asbestos pollution: Application of hazard criteria. *Ann. N.Y. Acad. Sci.* 330:579-86, 1979.
 15. Cochrane, J. C. and Webster, I.: Mesothelioma in relation to asbestos fibre exposure. *S.A. Med. J.* 54: 279-81, 1978.
 16. Hammond, E. C., Selikoff, I. J., and Seidman, H.: Asbestos exposure, cigarette smoking and death rates. *Ann. N.Y. Acad. Sci.* 330:473-90, 1979.

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