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Carcinogenicity of Amosite Asbestos

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Few data exist concerning the comparative neoplastic potential in man of the several kinds of asbestos. In particular, there has been no evidence concerning whether the amosite variety is carcinogenic. The matter is of practical importance, since amosite use in the United States has sharply increased. The mortality experience of a group of 230 men previously employed in an amosite asbestos factory was studied during the years 1960 through 1971. Total deaths were more than twice the number anticipated: 46.4 were expected, and 105 occurred. Some 14 deaths were due to asbestosis. Both lung cancer and mesothelioma were found in considerable excess. Two or three deaths from lung cancer were expected, and 25 occurred. There were five deaths from mesothelioma. Occupational exposure to amosite asbestos can be associated with serious cancer hazard; its continued industrial use requires rigorous control.

Possible differences in the disease potential of the several common, commercially available varieties of asbestos fiber (chrysotile, amosite, crocidolite, anthophyllite) have been sought, since the use of asbestos might be guided accordingly.¹ Experimental studies were undertaken to seek such information. These have not indicated any critical differences; mesothelioma can be produced easily by intrapleural inoculation of various types of asbestos.² There is less information concerning experimental lung cancer.³

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Few data exist concerning the comparative neoplastic potential of the several kinds of asbestos in man. Some information is available for chrysotile,¹ crocidolite,² and anthophyllite.³ However, there has been no evidence to indicate whether or not the amosite variety is also carcinogenic.

In large part this has been the result of epidemiological difficulties. Since there is often admixture of fiber varieties under industrial circumstances, the experiences of employed populations are not always readily attributable to a single fiber variety. Diligent efforts have been made to investigate the occurrence of disease in the Transvaal in South Africa, the only area in which amosite is mined, and where populations exposed only to amosite could be identified. Environmental studies in this area, reported in 1964, showed no instance of mesothelioma in amosite miners and only isolated instances of carcinoma of the bronchus, although asbestosis was found.⁴ This survey has since been complemented by a study of miners in the area. No attempt was made to study the Bantu workmen who are in the majority, since labor turnover is very high, and "... records kept are such that follow-up of individuals over a period of time is impossible."⁵ The fate of white miners employed between 1954 and 1958 was investigated. Unfortunately, the records turned out to be imperfect and incomplete, since the white work force in these mines was unstable. Only 147 white miners could be followed at all, and only 20 of these were observed for more than 20 years after first exposure to asbestos. Among these 20, there was one instance of lung cancer and none of mesothelioma. Elsewhere, comment is made

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that "The position of amosite is not clear . . ." with only one pleural mesothelioma stated to be known in the Transvaal.⁹

Increasing Use of Amosite in the US

Whether or not amosite is carcinogenic is of some practical importance. Because this variety of asbestos has not been reported to cause cancer, there has been a tendency in Great Britain, for example, to substitute it for other kinds of asbestos, especially crocidolite. Moreover, increasing amounts of amosite have been sent to the United States from South Africa. There is no record of amosite being imported before 1930, and as late as 1940, only small amounts were received. Since the Second World War, the situation has changed. Annual imports have risen from less than 500 tons in 1935 to 4,500 in 1945, 18,000 in 1955, and 21,400 tons in 1965 (Table 1).

The US Department of Commerce has reported imports of amosite and crocidolite into the United States (in tons per year) as follows:

Total Amosite and Crocidolite	
1930	1,907
1931	823
1932	212
1933	233
1934	152
1935	501
1936	1,432
1937	2,928
1938	3,282
1939	6,129

These data should be considered with appreciation of the long-lapsed period between onset of asbestos exposure and appearance of asbestos cancer—usually 20 to 30 years or more.¹⁰ Cancers that may be associated with the amosite now being imported will not become clinically evident until the 1990s or after the year 2000.

Current Investigation

We have investigated the mortality experience of a group of workmen occupationally exposed solely to amosite whose employment started between June 1941 and December 1945. This cohort has been observed through June 30, 1971.

Employed Population.—Nine hundred thirty-

three men were employed for varying periods of time in an asbestos products factory in an eastern city of the United States, starting work some time between June 1941, when the factory began production, and December 1945. The plant continued production until November 1954, when it closed its doors. It manufactured amosite asbestos insulation, primarily for use in shipbuilding and ship repair.

We have sought to trace each of these men and have successfully done so in 868 instances so far (93%). In 65 instances, tracing is still incomplete. Data concerning date of birth, onset of employment, duration of employment, and type of work are known.

For the purpose of this report, we have investigated the mortality experience of those individuals who had at least one year of employment. There were 333 such men who began work sometime between 1941 and 1945: 88 died by Dec 31, 1959; 15 were lost to follow-up; and 230 were alive on Jan 1, 1960, and each of these men has been followed since. Table 2 provides an analysis of man-years of experience for these 230 men, by age category.

We have calculated expected death rates for these men in the Jan 1, 1960 through June 30, 1971 category, using age, year, and sex specific rates for US white men. Expected deaths were then compared with those observed.

Amosite Exposure.—No information is available concerning dust levels in this plant. Although exhaust ventilation was used, discussions with surviving workmen and plant management indicate that dust exposure, at least in some circumstances, could have been high. Respirators were issued to the work force but were inconsistently used.

We have ascertained that only amosite asbestos was used in this factory in several ways:

1. Review with plant management indicated that only this fiber variety was purchased and utilized. This is consistent with the ship insulation specifications under which the products were made.

2. We have obtained samples of the products made; examination of these samples by polarized light microscopy, electron microscopy, electron diffraction, and electron microprobe analysis showed only amosite to be present.

3. Asbestos was still present in the factory building's storeroom. When retrieved and examined, the material was found to be amosite, by employment of the same analytical methods.

4. Several workmen had kept their own respirators, dating back to their period of employment. The filters in these respirators were studied. Fibers retained on the filters were

Table 1.—Estimated US Asbestos Fiber Consumption (Tons)					
Year	Crocidolite	Amosite	Canadian Chrysotile	Other	Total
1920	No data	No data	152,000	4,000	156,000
1925	No data	No data	200,700	4,100	204,800
1930	1,907*		198,200	8,500	208,407
1935	501*		154,200	20,500	175,201
1940	8,068*		225,900	36,300	270,268
1945	8,700	4,500	355,800	9,000	378,000
1950	8,500	5,400	687,400	27,400	728,700
1955	11,700	18,000	699,100	11,600	740,400
1960	19,600	19,500	605,800	24,600	669,500
1965	17,100	21,400	661,100	20,100	719,700

* Amosite and crocidolite combined; not separately recorded.

Table 2.—Man-Years of Experience Among 230 Amosite Factory Workers (1/1/60—6/30/71)		
Age	Jan 1, 1960 Through Dec 31, 1964	Jan 1, 1965 Through June 30, 1971
Under 40	471.7	54.8
40-44	528.8	438.3
45-49	501.9	619.5
50-54	415.2	565.2
55-59	347.3	472.2
60-64	285.2	352.4
65-69	198.4	272.9
70-74	104.5	163.9
75-79	69.7	134.1
80-84	36.0	47.0
85+	8.0	25.0
Total	3088.2	3145.3

Table 3.—Causes of Death Among 230 Amosite Asbestos Factory Workers (1/1/60—6/30/71)*		
Cause of Death	Observed Deaths	Expected Deaths†
Total cancer, all sites	43	8.5
Cancer of lung, pleura, bronchus, trachea	27	2.4
Lung cancer	25	‡
Pleural mesothelioma	2	‡
Peritoneal mesothelioma	3	‡
Cancer of stomach, colon, rectum	5	1.6
Cancer of all other sites	8	4.5
Asbestosis	14	‡
All other causes	48	37.9
Total deaths	105	46.4

* Analysis of lifetime work experience indicates that, for large majority, this was only occupational asbestos exposure.

† Expected deaths 1960-1964 are based on US age specific rates for white men in 1962. For 1965-1971, rates for 1968 were utilized. Smoking habits are disregarded.

‡ US data not available, but these should be only slightly less than 2.4.

§ US data not available, but these are rare causes of death in general population.

removed and analyzed. They were invariably amosite.

5. We have extracted mineral fibers from the lungs of individuals in this cohort who had died. Large numbers of fibers were present. Electron diffraction and electron microprobe analysis showed them to be amosite,¹¹ except for the occasional chrysotile fibril expected to be present in the lungs of urban dwellers in this area.¹²

Results

The mortality experience of this group of workmen, 1960 through 1971, demonstrates that a serious health hazard was associated with this industrial use of amosite. Table 3 records expected and observed deaths.

Total deaths were more than twice the number anticipated: 46.4 were expected and 105 occurred. This excess death rate was limited to two categories: cancers of various sites and asbestosis.

Fourteen deaths were due to asbestosis, when virtually none was expected. That amosite can result in asbestosis has been previously recognized.⁷ In physiological studies of a select group of workmen at this particular factory from 1954, serious pulmonary insufficiency had been demonstrated.¹³

Both lung cancer and mesothelioma were also found in considerable excess. It was

anticipated that 2.4 deaths from lung cancer would eventuate; 25 occurred. The calculation of expected rates disregarded smoking habits, since the smoking habits of individuals not examined by us are not accurately known. In prospective studies from this point on, smoking habits will be taken into account.¹⁴

Mesothelioma caused five deaths: two pleural and three peritoneal. Each has been histologically verified in material obtained during operations in two instances and at autopsies in three. In one case, there had been prior chrysotile exposure; in four, only amosite asbestos exposure had occurred. Parenthetically, additional instances of mesothelioma have occurred among men working in this plant, other than in the cohort reported here.

It may be of interest that more deaths from cancer of the stomach, colon, and rectum have occurred than expected. The increase is only threefold, however. As with similar previous experiences, further observations are required before this association can be regarded as clearly established.¹⁵

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Field investigations were carried out by Dorothy Perron, Shirley Levine, Rayla Margolies, and Charles V. Nolan.

References

1. Whipple HE (ed): Biological effects of asbestos. *Ann NY Acad Sci* 132:706-721, 1965.
2. Wagner JC, Berry G: Mesotheliomas in rats following inoculation with asbestos. *Brit J Cancer* 23:567-581, 1969.
3. Smith WE, Miller I, Churg J: An experimental model for study of co-carcinogenesis in the respiratory tract. Oak Ridge, Tenn, Atomic Energy Commission symposium series 21, 1970, pp 299-316.
4. McDonald JC, McDonald AD, Gibbs GW, et al: Mortality in the chrysotile asbestos mines and mills of Quebec. *Arch Environ Health* 22:677-686, 1971.
5. Wagner JC, Sleggs CA, Marchand P: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Brit J Industr Med* 17:240-271, 1960.
6. Kiviluoto R, Muurman L: Results of asbestos exposure in Finland, in Shapiro HA (ed): *Proceedings of International Conference on Pneumoconiosis, Johannesburg 1969*, Capetown, South Africa, Oxford University Press, Inc, 1970, pp 190-191.
7. Sluis-Cremer GK: Asbestosis in South Africa: Certain geographical and environmental considerations. *Ann NY Acad Sci* 132:215-234, 1965.
8. Sluis-Cremer GK: Asbestosis in South African asbestos miners. *Environ Res* 3:310-319, 1970.
9. Wagner JC: Asbestos cancers, editorial. *J Nat Cancer Inst* 46:v-ix, 1971.
10. Selikoff IJ, Bader RA, Bader ME, et al: Asbestosis and neoplasia, editorial. *Amer J Med* 42:487-496, 1967.
11. Langer AM, Rubin I, Selikoff IJ: Electron microprobe analysis of asbestos bodies, in Shapiro HA (ed): *Proceedings of International Conference on Pneumoconiosis, Johannesburg 1969*, Capetown, South Africa, Oxford University Press, Inc, 1970, pp 57-69.
12. Langer AM, Selikoff IJ, Sastre A: Chrysotile asbestos in the lungs of persons in New York City. *Arch Environ Health* 22:348-361, 1971.
13. Bader ME, Bader RA, Selikoff IJ: Pulmonary function in asbestosis of the lung: An alveolar-capillary block syndrome. *Amer J Med* 30:235-242, 1961.
14. Selikoff IJ, Hammond EC, Churg J: Asbestos exposure, smoking, and neoplasia. *JAMA* 204:106-112, 1969.
15. Selikoff IJ, Churg J, Hammond EC: Asbestos exposure and neoplasia. *JAMA* 193:22-26, 1964.

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