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BIOLOGICAL EFFECTS OF ASBESTOS

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Asbestosis in asbestos cement workers

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The bulk of the current world output of asbestos is consumed in the production of asbestos cement and asbestos cement products. No other single use of asbestos has grown so rapidly in recent years. Both chrysotile and crocidolite asbestos are used, the former where flexibility in the finished product is of primary importance, and the latter where strength and resistance to acid corrosion are required.

Exposure of workers occurs during the unpacking and mixing of asbestos fibre, and again when the dried product undergoes cutting, punching and grinding, at which time there is also exposure to silica. Dust exposure is minimal during intermediate stages of the process, when the product is wet.

There are a number of reports concerning pneumoconiosis among asbestos cement workers. El-Sowefy (1969) reported on the chest X-ray examinations of 347 unskilled labourers in a cement asbestos pipe factory in Egypt. These workers were mobile, being exposed to asbestos dust on one shift and to silica and/or cement dust on another. Dust concentrations in the factory were high, with hourly suspended dust counts ranging up to 640 mg/m³. The proportion with positive chest X-rays increased with duration of exposure, ranging from 50% for workers with under 10 years' exposure to 81% for workers with 20 years' or more exposure. Radiological appearances were not all consistent with asbestosis and often showed mixed dust pneumoconiosis and silicosis.

A similar study of 49 workers in an asbestos cement factory in India, with airborne dust concentrations seven times the maximum allowable concentration of 5 million particles per cubic foot (mppcf) of air, showed 27% to have positive chest X-ray findings (Barnejy, 1968). Ninety per cent of the workers had been continuously exposed to asbestos dust for five years or more. Twenty-eight per cent of the workers showed vital capacities of less than 74% of the standard value for Indian males, while 41% complained of dyspnoea. Four cases showed some tendency to clubbing, and there was cyanosis in one case.

One of us (HW) has completed the first phase of an investigation of health effects related to dust exposure in the manufacture of asbestos cement products in the United States (Weill *et al.*, 1973). Clinical information was obtained from 908 currently employed, recently retired or separated workers from two plants by means of a personal interview and pulmonary function studies, including measurement of lung volumes and its components, maximum respiratory flow rates, pulmonary diffusing capacity and gas exchange at two submaximal levels of exercise. Chest X-ray films were read according to the UICC Cincinnati classification. Each worker's job history was obtained from plant records; and exposure assessments were based on dust measurements made over the past 20 years by governmental or company industrial hygienists. Indices for total dust and its constituents, including chrysotile, crocidolite and silica contents were calculated for each job each year and ultimately derived cumulatively for each worker. Initial analysis of data has been concerned with the correlation of dust exposure, pulmonary function and radiographic patterns.

It was found that the mixed dust exposure to asbestos and silica produced both small rounded and irregular opacities radiographically. Table I shows that radiographic changes of 2/1 category or above begin to appear after a total cumulative dust exposure of 100 mppcf-months, with a steady increase

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Table 1. Rates [illegible] of rounded or irregular small opacities for total dust categories

Maximum profusion UICC Cin [illegible]	Total dust exposure (million particles per cubic foot-months)					
	< 250	251–500	501–1000	1001–2000	2001–4000	4001 +
0.0 and 0.1	95	33	94	81	70	60
0.1	4	6	4	11	16	17
1.0, 1.1 and 1.2	0	0	2	3	6	13
2.1 +	1	1	0	5	8	10
	(161)	(37)	(105)	(122)	(209)	(231)

in the rates of abnormal radiographic patterns as exposure becomes higher. There was no clear association between silica exposure and rounded opacities or asbestos exposure and irregular opacities, probably because a limited range of exposures to these two materials was encountered in the plants. Profusion of rounded opacities was better correlated with total dust exposure than was profusion of irregular opacities, and cumulative dust exposure was better correlated with the presence of diffuse changes on the chest X-ray than was number of years in the industry.

In order to determine whether the pulmonary physiologic pattern encountered in individuals with well-established irregular opacities (2.1 +) differs from that in those with rounded opacities (2.1 +), mean values for lung volumes, gas transfer and expiratory flow rates were expressed in per cent of predicted, and these were compared in the two groups. Figure 1 shows that in those persons with either type of small opacity pattern, total lung capacity and vital capacity are significantly lower than in the group without radiographic evidence of diffuse pulmonary infiltrations; and mean values for both of these volumes are considerably lower in the group with irregular opacities than in the group with rounded change. The mean residual volume:total lung capacity ratio was significantly higher in the rounded opacity group than in the negative X-ray group, and also higher than in those with irregular opacities. Pulmonary diffusing capacity was significantly lower with both X-ray patterns than when these changes were absent, being lowest in the group with irregular opacities. The change in diffusing capacity was related primarily to reduction in alveolar volume rather than in diffusion constant. Mean maximum expiratory flow rates were significantly lower in those with definite small opacities than in those without parenchymal change, but the type of opacity could not be distinguished on the basis of

Fig. 1. Pulmonary function mean values in groups with small irregular and rounded opacities

[illegible]	TCL I	TCL R	VC I	VC R	RV/TLC I	RV/TLC R	% Predicted	D^{1} I	D^{1} R
	[illegible]							(800)	
120									
[illegible]	15	28					110	(15)	(27)
	[illegible]	[illegible]				2 +		(21)	(23)
								-0-	
[illegible]							100		
					-1-			-1-	-1-
					2				
	[illegible]					-1			
[illegible]			0			2	90		-2-
		2 +							
								-2-	
							80		
	2 +			2 +					
							70		

[illegible] 2 [illegible] of opacities 0 0 [illegible] opacities 1 irregular only [illegible]

this measureme noted radiograp were affected ad of opacities, w reduced only in

It appears [illegible] advanced profu have lower lung capacity than the inflation is asso change, while n tinguish the type patterns are co small rounded silica exposure asbestos effect

Some studies parisons of the cement workers asbestos produc workmen's co asbestosis with years 1958–66 asbestosis was than in asbesto the ratio of case to those from estimated at cancer it was

Somewhat earlier by Ent of mortality at working in the they found t very much lo than in asbesto tile plants. T respiratory can for age-time di rates were sh workers than f

Vigliani (19 tosis compens the occupations Of a total of 5 kers making terial. Unfort not given, s asbestos cem be assessed

Currently

this measurement. When pleural changes were noted radiographically, lung volumes and flow rates were affected adversely in the presence of both types of opacities, while diffusing capacity was further reduced only in those with rounded change.

It appears that individuals with a moderately advanced profusion of irregular opacities tend to have lower lung volumes and pulmonary diffusing capacities than those with rounded opacities. Hyperinflation is associated with the rounded nodular change, while measurements of flow rates do not distinguish the type of opacities. These physiological patterns are consistent with the hypothesis that the small rounded opacities are primarily the effect of asbestos exposure and that irregular opacities indicate asbestosis effect.

Some studies have been made which permit comparisons of the occurrence of asbestosis in asbestos cement workers with that in other workers in the asbestos products industry. Bohlig (1970) studied workers' compensation cases for asbestosis and asbestosis with lung cancer in West Germany for the years 1958–66. He concluded that the incidence of asbestosis was lower in asbestos cement workers than in asbestos textile workers. For asbestosis, the ratio of cases from the asbestos cement industry to those from the asbestos textile industry was estimated at 1:13, while for asbestosis with lung cancer it was 1:5.

Somewhat similar findings had been reported earlier by Enterline & Kendrick (1967). In a study of mortality at ages 15–64 in a cohort of 21,755 men working in the American asbestos products industry, they found that the asbestosis mortality rate was very much lower in the building products industry than in asbestos friction materials and asbestos textile plants. The contrast was not so striking for respiratory cancer, however, and after adjustment for cigarette differences, respiratory cancer mortality rates were slightly higher for building products workers than for friction materials workers.

Wagner (1969) has reported on new cases of asbestosis diagnosed at Penn during 1964–66, giving the age, sex and occupation of the patients involved. Of the 47 cases compensated, 342 were workers in asbestos textiles and asbestos cement manufacture. Evidently, the populations at risk are [illegible] risk to workers exposed to [illegible] effects of asbestos cement [illegible]

[illegible] PHS conducting a study of the mortality experience of ages 65 and over of a retired population of asbestos workers in the United States. These are workers who spent most of their working lifetime mining or milling asbestos or as production or maintenance employees in the manufacture of asbestos cement products, insulation materials, packing and gaskets, friction materials, textiles, paper and flooring. For each worker, data compiled include a time-weighted measure of total asbestos dust exposure, type of work performed and type of asbestos used. For 1348 workers retired during the period 1941–67 on reaching age 65 and followed for deaths until 1969, a linear relationship was found between the time-weighted measure of asbestos dust exposure (as measured by the midget impinger) and deaths from asbestosis, with annual mortality rates for asbestosis ranging from 35 per 100,000 for men with exposures of less than 125 mppcf-years to 1139 deaths per 100,000 for men with exposures of 750 mppcf-years or more. Asbestos dust exposure was also found to be highly related to respiratory cancer mortality, with the excess

Table 2. Observed and expected deaths and standardised mortality ratios (SMR's) for selected causes of death by type of product: males retiring 1941–67 from the asbestos industry and followed through 1969

Cause of death	Asbestos cement		
	Observed	Expected[a]	SMR
All causes	202	176.4	114.5
Cancer (140–205)[b]	52	30.4	171.0
Digestive (150–159)	16	11.1	141.6
Respiratory (162, 163)	17	6.6	257.6
All other cancers	19	12.5	152.0
Non-cancer diseases (420–527)	18	10.0	180.0
Pneumoconioses (523, 525)	10	—	—
Asbestosis (523.2)	6	—	—
Other respiratory diseases	8	10.0	80.0
All other causes	132	136.0	97.0

Cause of death	Other asbestos		
	Observed	Expected[a]	SMR
All causes	552	490.9	114.8
Cancer (140–205)	115	79.0	145.6
Digestive (150–159)	37	30.5	121.3
Respiratory (162, 163)	41	15.1	271.5
All other cancers	37	33.4	110.8
Non-cancer diseases (420–527)	40	26.8	182.8
Pneumoconioses (523, 525)	19	—	—
Asbestosis (523.2)	12	—	—
Other respiratory diseases	20	26.8	111.9
All other causes	384	375.0	103.5

[illegible] rates for US white males [illegible] to ratios of the International [illegible]

[illegible] Causes of Death, 7th revision. World [illegible] 1967

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ranging from 1.7 times expected in the lowest exposure category to 5.6 times expected in the highest (Enterline *et al.*, 1973a).

For this review, special tabulations were prepared of data from 422 men who had at one time worked with asbestos cement products. Table 2 compares the mortality experience at ages 65 and over for the 422 men who worked in asbestos cement products with that of 926 other workers in the study. For both groups the mean time-weighted dust exposure at time of retirement was about the same, 249 mppcf-years versus 247 mppcf-years. Also, mean age at first exposure was about the same for each group (39.8 versus 38.6). Table 2 shows that mortality rates among asbestos cement and other asbestos workers were very similar. This was true for all causes of death and for the selected causes shown.

Of the 422 retired workers from asbestos cement products industries, 316 had worked in the manufacture of rigid shingles and sheets while 106 had worked in the manufacture of asbestos cement pipe. Table 3 compares mortality in these two groups of workers. While mortality for all causes was about the same, death rates for cancer and respiratory disease were quite different. Men who had manufactured asbestos pipe had a very high death rate for respiratory cancer and no deaths from asbestosis; on the other hand, the excess in respiratory cancer mortality was not remarkable for men making rigid shingles, but this group contained all the deaths from asbestosis.

Although the numbers of asbestos cement workers used in Table 3 are small, one might speculate as to possible reasons for a real difference in mortality between workers in asbestos cement shingles and asbestos cement pipe. The manufacture of asbestos cement pipe was introduced into the United States only after 1928, with the result that retired men in this study started work somewhat later than did other asbestos cement workers; for pipe workers in this study the mean year of hire was 1937, as compared with 1931 for other cement workers. Thus, time since first exposure and duration of exposure were somewhat shorter for pipe workers, and this resulted in lower time-weighted exposure for this group (225 mppcf-years versus 256 mppcf-years), which might [illegible] for the absence of asbestosis [illegible] not account, however, [illegible] cancer mortality.

Most [illegible] manufactured in the United States contains crocidolite asbestos (currently 3[illegible]) but more in the past—and all asbestos cement pipe workers had exposure to crocidolite asbestos. Rigid shingles and sheets, on the other hand, rarely contained crocidolite asbestos, so hardly any of the workers making these products were exposed to it. This disparity may account for the difference in incidence of respiratory cancer between these two groups. Other factors not considered in Tables 2 and 3, such as time of retirement and type of work, do not account for the difference shown. In an earlier report it was pointed out that maintenance-service workers exposed to asbestos had very high respiratory cancer death rates and low asbestosis death rates (Enterline *et al.*, 1973b). Adjustment of the data to take account of this factor slightly increases respiratory cancer mortality rates for asbestos cement workers in comparison to other workers (Table 2) but does not change comparisons between rigid shingle and cement pipe workers (Table 3).

Table 3. Observed and expected deaths and standardised mortality ratios (SMR/S) for selected causes of death by type of product, males retiring 1941–67 from the asbestos industry and followed through 1969

Cause of death	Rigid shingles and sheets		
	Observed	Expected[a]	SMR
All causes	156	135.2	115.4
Cancer (140–205)[b]	35	23.0	152.1
Digestive (150–159)	12	8.6	139.5
Respiratory (162–163)	7	4.9	142.9
All other cancers	16	9.5	168.4
Respiratory diseases (470–527)	16	7.6	210.5
Pneumoconiosis (523–525)	9	—	—
Asbestosis (523.2)	6	—	—
Other respiratory diseases	7	7.6	92.1
All other causes	106	104.6	101.3

Cause of death	Cement pipe		
	Observed	Expected[a]	SMR
All causes	46	41.2	111.6
Cancer (140–205)	17	7.4	229.7
Digestive (150–159)	4	2.7	148.2
Respiratory (162–163)	10	1.7	588.2
All other cancers	3	3.0	100.0
Respiratory diseases (470–527)	2	2.4	83.3
Pneumoconiosis (523–525)	1	—	—
Asbestosis (523.2)	0	—	—
Other respiratory diseases	1	2.4	41.7
All other causes	27	31.4	86.0

[a] Based on time-age-cause specific rates for US white males.
[b] Figures in parentheses refer to rubrics of the International Classification of Diseases and Causes of Death, 7th revision, World Health Organization, Geneva, 1957.

CONCLUSIONS

Exposure to asbestos and silica dust in the asbestos cement industry produces radiographic and physiologic evidence of asbestosis and/or silicosis, and these fibrogenic effects appear to be related to the cumulative level of dust to which the worker is exposed. It is probable that a level of total dust exposure can be attained which will be unlikely to produce the pneumoconioses which have resulted from past higher levels of dust. Although to date it has been impossible to distinguish between exposures to asbestos and silica in these workers, the pulmonary function patterns suggest that the rounded nodules seen on the chest X-ray may represent an exposure predominantly to silica, and that irregular changes are associated with the effect of asbestos. It is not clear why individuals with seemingly similar exposures in this industry develop differing radiographic and physiologic patterns.

Some comparisons between asbestos cement workers and other asbestos workers are possible from studies of worker compensation records and from mortality studies. Comparisons of working populations suggest that asbestosis is less of a problem in the asbestos cement products industry than in other applications of asbestos. On the other hand, a study of a retired population shows little difference. One problem in comparing working populations is that since the asbestos cement products industry has grown rapidly, its work force is younger and has had less exposure than have other asbestos workers; this may account for their relatively lower asbestosis rate.

Regarding the effects of exposure to various types of asbestos, a mortality study of retired asbestos cement workers showed that workers exposed to crocidolite asbestos showed no asbestosis but a high respiratory cancer mortality, whereas workers exposed mainly to chrysotile asbestos showed considerable asbestosis but moderate respiratory cancer mortality. While based on small numbers, this finding supports other observations regarding the carcinogenic properties of crocidolite asbestos.

SUMMARY

The asbestos cement industry consumes the bulk of the world's output of asbestos. Workers in this industry are exposed to silica and asbestos, and they develop both asbestosis and silicosis in numbers related to their total dust exposure. It is estimated that radiographic changes of 2/1 category or above appear after a total dust exposure of 1000 mppcf months. Asbestosis appears to be less of a problem in the asbestos cement industry than in other asbestos products industries, possibly because the former is growing more rapidly and its work force has had less dust exposure. Both chrysotile and crocidolite asbestos are used, with the latter confined almost entirely to asbestos cement pipe. Cement pipe workers appear to have a great excess in respiratory cancer but little asbestosis, whereas other cement workers have a modest excess in respiratory cancer but a considerable amount of asbestosis.

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ASBESTOSIS

Asbestosis in Asbestos Cement Workers

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