

Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture

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ABSTRACT This report describes the second in a series of three parallel cohort studies of asbestos factories in South Carolina, Pennsylvania, and Connecticut to assess the effects of mineral fibre type and industrial process on mortality from malignant mesothelioma, respiratory cancer, and asbestosis. In the present plant (in Pennsylvania) mainly chrysotile, with some amosite and a small amount of crocidolite, were used primarily in textile manufacture. Of a cohort of 4137 men comprising all those employed 1938-59 for at least a month, 97% were traced. By the end of 1974, 1400 (35%) had died, 74 from asbestosis and 70 from lung cancer. Mesothelioma was mentioned on the certificate in 14 deaths mostly coded to other causes. All these deaths occurred after 1959, and there were indications that additional cases of mesothelioma may have gone unrecognised, especially before that date. The exposure for each man was estimated in terms of duration and dust concentration in millions of dust particles per cubic foot (mpcf) from available measurements. Analyses were made both by life table and case referent methods. The standardised mortality ratio for respiratory cancer for the whole cohort was 105.0, but the risk rose linearly from 66.9 for men with less than 10 mpcf.y to 416.1 for those with 80 mpcf.y or more. Lines fitted to relative risks derived from SMRs in this and the textile plant studied in South Carolina were almost identical in slope. This was confirmed by case referent analysis. These findings support the conclusion from the South Carolina study that the risk of lung cancer in textile processing is very much greater than in chrysotile production and probably than in the friction products industry. The much greater risk of mesothelioma from exposure to processes in which even quite small quantities of amphiboles were used was also confirmed.

Cohort mortality studies were conducted simultaneously in three American plants to clarify the effect of mineral type of asbestos and of processing, as distinct from production, on mortality from malignant mesothelioma, lung cancer, and asbestosis. Preliminary reports describing results by duration of employment in all three plants¹ and detailed exposure-response findings in one of them—a textile plant that used only chrysotile²—have been published. This report concerns a second plant which manufactured mainly textiles but also friction products and packings, many of which were made from the textile products. In addition to chrysotile, some amphibole asbestos (amosite and crocidolite) was also used. In

the third plant friction materials and packings were made from chrysotile only and no textiles were manufactured.³ The plant dealt with in the present report has been the subject of two previous studies^{4,5}; in both, excess mortality was reported from chronic respiratory disease, lung cancer, and malignant mesothelioma. In neither study was exposure estimated other than by duration of employment. In the present study the cohort was much larger, and individual dust exposures were estimated from environmental measurements made in the work place.

Materials and methods

The workforce at the plant averaged 1200 men and women between the late 1930s and 1975; before then it was rather smaller. Social security numbers

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were allocated in 1937. Details of all those employed before 1 January 1959 for at least one calendar month, with a recorded social security number, were submitted to the United States Social Security Administration. Those whose numbers and names were consistent with the administration's records comprised the cohort. The cohort therefore fell into two groups: (a) all those first employed after 1937 and (b) those first employed before that date. Because the latter group was selected in that it excluded employees who had left before 1938, the two groups were analysed separately. In fact, the findings were similar for those two groups and are reported together in this paper.

In all, 4137 men and 998 women met the cohort criteria. We ascertained survival status as of 31 December 1977 through local inquiries and from information on deaths provided by Social Security Administration. All but 113 men (3%) and 61 women (6%) were traced; of those traced, 1400 men (35%) and 170 women (18%) had died. Copies of death certificates were obtained from state departments of vital statistics or from the country where death had occurred for 1354 (97%) of the men and 165 (97%) of the women. In two male deaths without certificate the cause was ascertained from other sources. Causes of death were coded according to the seventh revision of the International Classification of Disease (ICD) by a single qualified nosologist. This paper deals only with the 1392 male deaths, shown by age and cause in table 1. Eight deaths are omitted from this table because of insufficient information on age. Female mortality in this and the other two plants will be reported separately.

THE PROCESS

The plant was opened in a rural area of central Pennsylvania in the early 1900s. From the beginning it has manufactured a variety of products, almost all containing asbestos. The factory had a large asbestos textile division of traditional type. Crude fibre was cleaned, opened, carded on modified wool carding machines, spun using mule and later ring-type frames, and wound and woven into cloth or plyed to form tape. Much of the cloth or tape was treated with resin, dried, and then finished into brake linings, clutch facings, and transmission strips. Since the early days the plant has also manufactured friction materials by blending raw asbestos with resin-binding ingredients followed by extrusion, rolling, or bricketting.

Chrysotile, obtained mostly from Canada and Rhodesia, was the main type of asbestos processed, with some 3000 to 6000 tons of varying quality used annually. Crocidolite and amosite were used from 1924 onwards for making insulation blankets for locomotives and turbines and equipment for chemical factories, and paper mills. Amosite insulation blankets were produced in large quantities during 1942-5 to meet US naval specifications. Crocidolite packings were also produced, as were crocidolite and amosite filter materials. Much of the crocidolite was imported as yarn and only about three to five tons of raw fibre were used annually whereas, in 1943, the use of amosite reached a peak of 600 tons. From 1925 to 1931 manufacture of insulation mattresses and filter cloth was transferred to a disused opera house in the town. The mattresses were packed with crocidolite or amosite, the crocidolite yarn being imported from England. After 1931, the opera house was used as a warehouse and the manu-

Table 1 Male deaths by age and certified cause

Cause of death (ICD code)	Age at death			Total
	<45	45-64	≥65	
All causes	191	667	534	1392
Malignant neoplasms:				
Lung* (162-164)	3	49	18	70
Oesophagus and stomach (150-151)	1	7	6	14
Colon and rectum (152-154)	2	21	12	35
Other abdominal* (155-159)	3	16	5	24
Larynx (161)	0	0	0	0
Other* (140-148, 160, 165-205)	16	57	40	113
Heart disease (400-443)	43	285	245	573
Respiratory tuberculosis (001-008)	5	4	2	11
Other respiratory (470-522, 525-527)	6	17	25	48
Pneumoconiosis (523-524)	2	48	24	74
Cerebrovascular (330-334)	3	33	44	80
Accidents (800-999)	74	44	20	138
Other known causes*	23	73	80	176
Cause not known	10	13	13	36

*In 13 cases in these categories, mesothelioma was given as the cause of death; in one death ascribed to asbestosis, mesothelioma was also mentioned.

Table 2 *Estimated average prevailing dust concentrations (MPCF) in main departments 1930-70*

	1930	1940	1950	1960	1970
Textile:					
Preparation	15.0 *	14.6 *	3.5 *	2.0 *	1.5 *
Carding	12.0 *	9.3 *	3.2 *	2.0 *	1.6 *
Spinning	7.0		2.5	1.8	1.5
Twisting	7.2		4.0	0.5 *	1.1
Winding	3.0		1.5		0.8
Cloth weaving	7.9	*3.4		0.9	
Tape weaving	7.3	*3.1	1.4		1.2
Felted tape	2.0			0.5	
Rope	4.0			1.2	0.6
Friction:					
Woven brakes		2.0	1.5	1.0	0.7
Extruded brakes		2.0	1.5	1.0	0.8
Dry mix brakes		10.0	6.0	4.0	3.8
Clutch		2.0	1.5	1.5	1.5
Brake finishing		2.0	1.5	1.0	0.7
Sanding and finishing		2.0	1.5	1.0	0.7
Finishing and shipping		0.5		0.2	0.2
Packings, gaskets		1.3	1.3	0.6	0.7
Maintenance, etc		0.5	0.5	0.2	0.2

*Asterisks shown against textile processes indicate approximate date of improvements usually associated with technical change. Figures for friction and other departments are estimates for each decade.

Table 3 *Age at start, duration of employment, and dust exposure (male only)*

	Length of gross service				Total
	<1	1, <5	5, <20	≥20	
No	1248	906	855	1013	4022*
Average age at start (years)	28.80	29.30	30.77	27.22	28.92
Gross service (years)	0.40	2.39	11.01	30.63	10.71
Net service (years)	0.38	1.87	8.06	27.51	9.18
Average dust concentration (mpcf)	2.60	2.40	2.73	1.58	2.32

*Excluding two whose employment histories were incomplete.

facture of cloth and mattresses was returned to the main site.

EXPOSURE

Conditions are reported to have been very dusty in the 1920 and 1930s, although oil spraying introduced to facilitate handling probably had some damping effect. The first steps to reduce exposure were taken in 1930; these included wetting, improved handling methods, and ventilation hoods. By about 1939 exhaust ventilation had been installed in the textile mill with substantial reduction in dust levels; thereafter improvement was gradual.

Reports of surveys made by the Metropolitan Life Insurance Company in the period 1930-9 were available, as were surveys by the United States Public Health Service made in 1967 and in 1970. Measurements were made routinely by the company from 1956 onwards. Until 1967, all measurements were made by impinger and recorded in millions of particles per cubic foot (mpcf). Dust level estimates for each department were made by the hygienist of our group (AJW) and are summarised in table 2. These estimates do not take into account certain

additional exposures which may have been quite short but which were certainly very heavy. These were associated with daily "blowing down" and "whipping the burlap" in the dust house. At weekends waste asbestos thus recovered in the dust house was swept up and bagged for recycling. A new dust collection system introduced in the early 1950s virtually removed this hazard.

The data available on exposure are summarised in table 3, grouped by duration of employment. The average age at start of work and the average dust concentration differed little in the four duration groups.

STATISTICAL METHODS

Mortality in the cohort was analysed (a) by the man-years life table method of Hill,⁶ using Pennsylvania death rates for reference, and (b) by the log rank procedure of Mantel and Heanszel,⁷ exactly as in the South Carolina study.²

Results

The standardised mortality ratio (SMR) for all

Table 4 Male deaths 20 years after first employment, by cause, in relation to length of service

Cause of death*	Length of gross service years									
	<1		1, <5		5, <20		≥20		Complete cohort	
	0	SMR	0	SMR	0	SMR	0	SMR	0	SMR
All causes	171	87.2	154	106.2	187	104.5	383	127.2	895	109.0
Malignant neoplasms:										
Respiratory	9	69.6	3	32.9	14	128.8	27	158.9	53	105.0
Abdominal	8	72.9	11	133.7	11	105.9	24	131.3	54	112.7
Other	19	132.4	16	152.0	15	118.5	32	155.3	82	141.1
Heart disease	77	92.7	77	125.1	78	100.2	153	115.7	385	108.5
Respiratory tuberculosis	0	—	1	133.4	0	—	2	67.3	3	51.7
Other respiratory:	4	54.2	2	38.1	11	161.0	50	442.4	67	215.0
Pneumoconiosis	(2)	—	(1)	—	(10)	—	(46)	—	(59)	—
Cerebrovascular	7	54.9	10	106.5	10	77.7	20	87.6	47	81.2
Accidents	13	117.5	15	181.1	8	87.2	9	60.1	45	103.5
Other known	30	75.2	15	52.4	37	103.0	62	103.1	144	87.2
Not known	4	—	4	—	3	—	4	—	15	—

*As in table 1 except that ICD codes 160–164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490–502, 523–4).

Table 5 Male deaths 20 years after first employment, by cause, in relation to dust exposure (mpcf.y) accumulated to 10 years before death

Cause of death* (See table 4)	Dust exposure (mpcf.y)									
	<10		10 < 20		20 < 40		40 < 80		≥80	
	0	SMR	0	SMR	0	SMR	0	SMR	0	SMR
All causes	470	93.1	86	82.1	130	125.6	105	174.9	104	215.2
Malignant neoplasms:										
Respiratory	21	66.9	5	83.6	10	156.0	6	160.0	11	416.1
Abdominal	26	90.2	8	130.5	5	79.7	8	218.8	7	237.2
Other	47	130.4	5	68.5	11	148.6	7	164.7	12	372.8
Heart disease	221	102.7	41	89.2	60	130.6	34	130.5	29	108.5
Respiratory tuberculosis	1	34.3	0	—	—	—	1	169.7	1	163.6
Other respiratory:	8	43.6	5	122.0	10	263.0	14	623.3	30	1689.2
Pneumoconiosis	(4)	—	(1)	—	(9)	—	(9)	—	(36)	—
Cerebrovascular	27	78.3	1	13.3	10	133.5	8	187.2	1	29.3
Accidents	33	120.1	3	56.2	1	18.6	6	193.9	2	91.0
Other known	74	73.3	17	80.0	23	109.7	21	172.2	9	97.8
Not known	12	—	1	—	0	—	0	—	2	—

Table 6 Dust exposure in male deaths from selected causes and controls (Mantel-Haenszel analysis⁸)

	Dust exposure (mpcf.y) accumulated up to 10 years before death of case					Chi square Difference	Linearity
	<10	10 < 20	20 < 40	40 < 80	≥80		
Pneumoconiosis (ICD 523):							
Deaths	3	4	10	11	28	39.56	39.17
Expected	14.6	8.1	10.9	8.1	14.3		
Relative risk	1	4.04	13.72	14.93	37.90		
Lung cancer (ICD 162–4)							
Deaths	20	4	10	6	11	5.77	4.98
Expected	24.4	5.2	8.0	5.6	7.7		
Relative risk	1	0.83	1.54	2.90	6.82		
Abdominal cancer (ICD 150–9):							
Deaths	26	8	5	8	7	3.22	1.09
Expected	28.8	6.8	7.0	5.3	6.1		
Relative risk	1	1.15	0.66	2.45	2.85		
All causes							
Deaths	451	81	121	100	99	34.66	26.12
Expected	476.6	104.5	118.6	80.4	72.0		
Relative risk	1	0.82	1.20	1.6	2.12		

causes of death was 109.0. Those employed for under one year had an SMR of 87.2, and those who had worked 20 or more years, 127.2 (table 4). Malignant neoplasms, heart disease, and "other respiratory" disease were mainly responsible for the higher SMR in these long term workers. The other respiratory category included bronchitis and pneumonia (ICD 470-502) and pneumoconiosis (ICD 523-4) and was chosen for study because expected figures for pneumoconiosis alone were not available. Table 5 shows SMRs by cause and by accumulated dust exposure. The SMR for all causes rose steadily from 93.1 for men with an exposure at under 10 mpcf.y to 215.2 in the highest category (≥ 80 mpcf.y). Respiratory, abdominal, and other malignant disease and the non-malignant other respiratory group all contributed to this rising trend. On 14 death certificates a diagnosis of mesothelioma was specified: 10 were pleural tumours and four peritoneal. These deaths occurred in the period 1960-75. One (in 1960) was 16 years after first employment; the remaining 13 occurred 25-53 years after first employment. Two of the deaths from mesothelioma had been given the ICD code 199 (malignant neoplasms of other and unspecified sites); another 30 deaths 15 more years after first employment were given the code 199. Seventeen of these 30 deaths occurred before 1965, the year after which most of the deaths from mesothelioma occurred. The diagnosis given in many of these cases was consistent with an unrecognised peritoneal mesothelioma.

The Mantel-Haenszel (log rank) analysis (table 6) bore out the exposure-response relationships observed in table 5. There is a small shortfall (5% overall) between the numbers of cases used in this analysis and in the man-years analyses presented in tables 4 and 5. The deficiency is explained by failure to find matching controls for every selected case.

Discussion

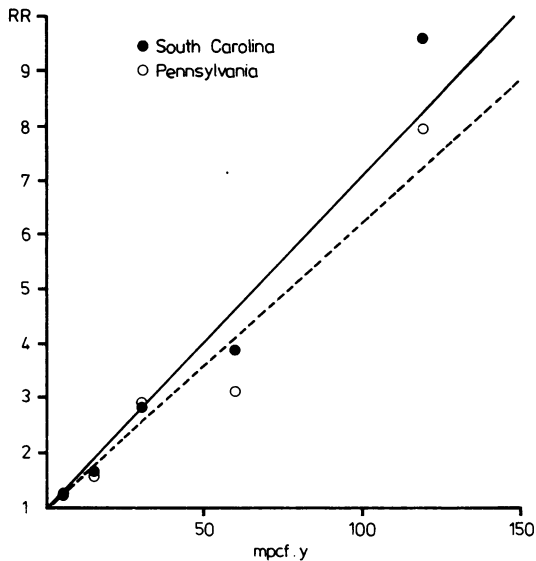
The present cohort in the Pennsylvania plant was constituted in exactly the same way as that in the South Carolina chrysotile textile plant described elsewhere.² Compared with the South Carolina employees (see table 3), those in Pennsylvania had worked for similar periods but were on average some four years older when they started work (28.93 years compared with 25.08 years). The Pennsylvania cohort was exposed to a somewhat higher average dust concentration: 2.32 mpcf compared with 1.80 mpcf in South Carolina. The mortality pattern in Pennsylvania resembled that in South Carolina in showing a rising SMR with increasing dust exposure for all causes of death, for respiratory cancer, and for pneumoconiosis. For respiratory cancer, however, the SMR for the lowest exposure group (less than 10 mpcf.y) was 115.5 in South Carolina but only 69.9 in Pennsylvania. By contrast with South Carolina, where the SMRs tended to be above 100 for causes unrelated to asbestos, and for all causes in very short term employees,² the opposite was true in Pennsylvania. It seems likely that in both cohorts lack of comparability with the relevant state populations may be the explanation. Having regard for this possibility, the use of relative risks is perhaps more appropriate than SMRs for comparing the respiratory cancer mortality of the two cohorts. Table 7 shows that the relative risks of death from all causes, respiratory cancer, and pneumoconiosis in the two plants were extraordinarily similar. In both cohorts the relationships of respiratory cancer to exposure were essentially linear (figure) with slopes that were nearly identical (South Carolina, $RR = 1 + 0.059$ mpcf.y; Pennsylvania, $RR = 1 + 0.051$ mpcf.y).

A conceivable explanation for depression of the SMRs in the Pennsylvania plant was that the em-

Table 7 Relative risks based on SMRs by cumulative exposure in two plants

	mpcf.y				
	<10	10 < 20	20 < 40	40 < 80	≥ 80
All causes:					
South Carolina plant	1.0	1.09	1.36	1.48	2.29
Pennsylvania plant	1.0	0.88	1.35	1.88	2.31
Respiratory cancer					
South Carolina plant	1.0	1.28	2.13	2.93	7.21
Pennsylvania plant	1.0	1.25	2.33	2.39	6.22
	<i>(1.32)</i>	<i>(1.68)</i>	<i>(2.80)</i>	<i>(3.86)</i>	<i>(9.49)</i>
	<i>(1.26)</i>	<i>(1.58)</i>	<i>(2.94)</i>	<i>(3.03)</i>	<i>(7.87)</i>
Bronchitis, pneumonia, and pneumoconiosis:					
South Carolina plant	1.0	1.81	6.40	21.36	19.67
Pennsylvania plant	1.0	2.79	6.03	14.29	38.74

Figures in italics are relative risks calculated from SMRs at zero exposure derived from fitted line.



Relative risk of respiratory cancer and accumulated dust exposure in two mainly textile plants. (Lines fitted by FIDK Liddell using the methods of Hanley and Liddell.)

ployees had smoked fewer cigarettes than the general population of the state, perhaps because of the high proportion of Mennonites in the local community. We ascertained, however, that in fact very few Mennonites were employed in the plant. Smoking histories had been recorded in a standard manner for all those at work in 1978 or later in the two plants and also in the plant in Connecticut where the third cohort was studied.³ Of men born in 1910–9 (who would have been included in the three cohorts) the proportions who had never smoked were: 25% (of 36) in Pennsylvania, 11% (of 95) in South Carolina, and 16% (of 206) in Connecticut. Thus the proportion of non-smokers (25%), though somewhat higher than in the other two cohorts, is based on very small numbers and no firm conclusion can be drawn.

Similar proportions of all deaths in the two cohorts were from malignant disease (17% in South Carolina and 18% in Pennsylvania) but the types of malignancy differed. In South Carolina respiratory cancer accounted for 47%, abdominal 25%, and other types 28% whereas in the Pennsylvania plant the corresponding proportions were reversed, 27%, 29%, and 44%. Moreover, in South Carolina no systemic relationship with exposure was seen for abdominal or other types of malignant disease whereas in Pennsylvania there was evidence of such a relationship.

The increased risk of mesothelioma in the Penn-

sylvania plant (14 cases in 1392 male deaths (1%) compared with one case in 867 (0.1%) in South Carolina) raises the question of whether the abdominal and more particularly other types of cancer included undiagnosed cases of mesothelioma. There is some support for this idea in the substantial number coded to ICD 199 (malignant disease of other and unspecified sites) and the fact that 17 of these deaths occurred before 1964 when malignant mesothelioma started to become more generally recognised. Once again we find evidence in this study of the special risk of mesothelioma associated with exposure to even quite small proportions of amphibole, in this case predominantly amosite.

The very similar exposure-response relationships for respiratory cancer and asbestosis observed in this and the South Carolina plant support our previous conclusion that the risks of these diseases in chrysotile production (mining and milling) and in textile manufacture are quite different. In the third plant studied, a friction materials plant in Connecticut, there was little or no excess risk of respiratory cancer or asbestosis.³ This was also true in a friction materials plant in the United Kingdom.⁹ Possible reasons for the striking epidemiological differences—fibre size distributions in particular—have been discussed elsewhere.^{2,10}

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