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Dust exposure and mortality in an American chrysotile textile plant

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ABSTRACT Three parallel cohort studies of asbestos factory workers were undertaken to investigate the effects of mineral fibre type and industrial process on malignant mesothelioma, respiratory cancer, and asbestosis. This report describes the mortality of a cohort of 2543 men, defined as all those employed for at least a month from 1938 to 1958 in a textile plant in South Carolina in which chrysotile was the only type of asbestos used. Of these, 863 men (34%) had died before 31 December 1977, one from malignant mesothelioma. Twenty one deaths were ascribed to asbestosis and 66 to cancer of the lung. Compared with the number expected from South Carolina, there was an excess of 30 deaths from respiratory cancer (ICD 160–164) in men 20 or more years after first employment (SMR 199·5). In men employed five years or more, no SMRs for this category rose above 300. Individual exposures were estimated (in mpcf × years) from recorded environmental measurements. Life table analyses and "log-rank" (case-control) analyses both showed a steep linear exposure-response that was some 50-fold greater at similar accumulated dust exposures than in Canadian chrysotile mining and milling. These findings agree closely with those from another study in this plant and confirm that mesothelioma is rarely associated with chrysotile exposure. Cigarette smoking habits did not greatly differ between the textile workers and the Canadian miners and millers. The far greater risk of lung cancer in the textile industry, if not attributable to other identified cocarcinogens, may be related to major differences in the size distribution of fibres in the submicroscopic range which are not detected by the usual fibre or particle counting procedures.

This study was planned in 1976 when it had become clear that there was a substantially lower risk of mesothelioma in workers employed in mining and milling chrysotile than in most other groups of asbestos workers.[1] As workers employed in the manufacture or use of asbestos products are usually exposed to amphibole fibres in addition to chrysotile the difference could well have been due to fibre type, as suggested many years earlier by Wagner *et al.*[2] There remained the possibility, however, that, for chrysotile at least, the explanation might also be related to the industrial process, with lower risks at the point of production than in manufacturing or use when the fibres are probably much finer as a result of mechanical forces. The experimental work of Pott, Stanton, and others (see review by Stanton[3]), had already underlined this possibility; however, direct epidemiological evidence based on factory workers exposed only to chrysotile was too scanty to settle the matter. Accordingly we identified three factories, the one described here which used only chrysotile for textile manufacture, another which used only chrysotile for friction products, and a third which used chrysotile, amosite, and crocidolite in textile and various other processes. So far as mesothelioma is concerned the salient findings have been published[4]; they showed that this tumour was much more common in workers exposed to amphiboles, only one case being identified among 2341 deaths in the employees working only with chrysotile compared with 18 cases among 1429 deaths in those who had worked with mixed fibres.

In the meantime the chrysotile textile factory dealt with in this report had also been investigated by Dement and colleagues of the US Public Health Service (NIOSH).[5,6] Their results on exposure-

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response for respiratory cancer were striking, linear in form but with risk many fold higher than in chrysotile production.[7] The importance of these findings and the need for their confirmation added significance to our own study, covering as it does a much larger cohort, followed over a longer period, with independently collected and analysed observations.

Materials and methods

PROCESS

The factory in question is located on the outskirts of a major Atlantic port in South Carolina. It began the manufacture of asbestos containing gaskets and packings in 1895 and introduced textile processes for the production of asbestos yarn and cloth in 1909. The factory moved to its present site in 1914 and the methods remained much the same until 1978 when an entirely new extrusion process for yarn production was introduced. During the period immediately before this change most of the older textile machinery was moved to another plant.

The type of fibre used for the main products has always been chrysotile, mainly from Canada but also Rhodesia. A small amount of imported crocidolite yarn (less than 2000 pounds a year) was used from the early 1950s until 1972. It was processed at one location, wet or lubricated with oil or graphite, to unite woven tape and braided packing. In the late 1950s a very small amount of amosite was used experimentally in carding but this was soon abandoned. The textile process itself was traditional in nature. Raw chrysotile was brought in as crude or semiopened fibre and passed to the preparation department for cleaning, further opening, blending with other grades, and mixing with cotton. There followed the usual processes of carding, winding, and spinning to produce strong yarn. Some yarn was sold but most went for weaving into braided and unbraided cloth and tape, some of which was later rubberised. Asbestos waste was collected at each stage and returned to the preparation area for reuse.

STUDY GROUP

The cohort studied comprised all those men or women, black or white, ever employed for one calendar month or more, who met two criteria. They had to have been employed before 1 January 1959 and to have had a social security number and name that matched with data in the US Social Security Administration files. In this cohort, race (colour) had been recorded in the employment records. Social Security numbers were allocated in 1937. The cohort could thus be considered in two groups, those already working before 1 January 1938 and those first employed on or after that date. A distinction between these two groups was maintained in the analysis because of the unknown effect of the selective process of survival required by those who qualified for the first group. In the event only minor differences in outcome were apparent so, in this paper, the results are combined.

With help from the US Social Security Administration and additional local inquiries, we tried to ascertain the vital status of all members of the cohort as of 31 December 1977. A search was then made for death certificates from vital statistical departments of states and countries where death had occurred. The registered causes of death were coded by a single qualified nosologist according to the seventh revision of the International Classification of Diseases (ICD). From a total of 2543 men, 863 (34%) are known to have died and certificates were found in 827 (95%); for the 1175 women, the corresponding figures were lower, 160 (14%) and 147 (92%). This paper will deal only with the 857 male deaths of known age shown by age and cause in table 1. It may be noted that whereas the analysis of Dement *et al*[6] examined 26 deaths in white men ascribed to respiratory cancer (ICD 162–3), all of which should be included in the present study, we shall be considering 66 deaths (white and black) in a similar diagnostic group (ICD 162–4).

EXPOSURE

The information available to us on dust exposure was essentially the same as that used by Dement *et al*—namely, over 5000 environmental samples taken since 1930, mainly using the impinger method. The Metropolitan Life Insurance Company undertook five surveys from 1930 to 1939 and the United States Public Health Service two more in 1968 and 1971. The company had carried out measurements from the 1930s and routinely from 1956. In this paper we shall consider exposure estimated in dust particle concentrations and duration, and shall do no more than speculate in the discussion on possible equivalence in terms of fibre concentrations. The environmental data were reviewed in detail by the hygienist of our team (AJW) who also spent some time at the plant discussing past working conditions with the older employees, including some who were responsible for the actual measurements. Each operation was considered separately and as fully as possible, noting changes in volume and practices of work, the introduction of ventilation, and other control measures. Taking all this into account, and without reference either to health and mortality information or to the estimates made by Dement,[6] the dust concentrations prevailing for each job, year by year, were assessed (see table 2). Thus we have

Table 1 *Male deaths by age and certified cause*

Cause of death (ICD code)	*Age at death*			*Total*
	<45	45–64	≥65	
All causes	178	502	177	857
Malignant neoplasms:				
Lung (162–164)	1	47	18	66
Oesophagus and stomach (150–151)	0	13	2	15
Colon and rectum (152–154)	2	3	4	9
Other abdominal (155–159)	0	10	2	12
Larynx (161)	0	2	1	3
Other (140–48; 160; 165–205)	6	23	12	41
Heart disease (400–443)	38	189	70	297
Respiratory tuberculosis (001–008)	8	4	2	14
Other respiratory (470–522; 525–527)	10	27	11	48
Pneumoconiosis (523–4)	2	12	7	21
Cerebrovascular (330–334)	5	30	21	56
Accidents (800–999)	67	42	3	112
Other known causes	24	89	19	132
Causes not known	15	11	5	31

Table 2 *Estimated average prevailing dust concentrations (mpcf) in main departments: 1930–70*

	1930		*1940*		*1950*		*1960*		*1970*
Preparation	3·5	*	2·0	*	1·0	*	0·8		
Carding	3·1	*	1·5	*	1·2	*	0·9		
Spinning	3·5	*	2·0	*		1·6			
Winding	2·8	*	1·7	*		1·1			
Twisting	6·1	*	4·0	*	1·1				
Weaving	2·1	*	1·5			*	1·2		
Finishing and inspection	1·4	*	1·0	*	0·8	*	0·5		

*Apparent improvement usually associated with technical change.

available for each man estimates of dust concentration in millions of particles per cubic foot (mpcf) and duration of exposure in years or fractions thereof (y) expressed separately, or cumulatively (mpcf.y). This procedure was closely analogous to that followed in our studies of chrysotile miners and millers.

Among the many difficulties encountered in assessing historical exposure were work practices of much potential importance, also described by Dement,[5] during the years 1937–53. In this period the dust filtration system (receiving dust from ventilation inflow in the preparation and carding departments) consisted of burlap bags stretched across wooden frames. Every day, the waste house operators would beat the bags with buggy whips to dislodge the accumulated dust. At the end of the week, the dust was shovelled into containers and returned to the preparation department for reprocessing. This work and other clean up operations, which also entailed extremely high exposures, were carried out at weekends as optional overtime, for which there was no shortage of volunteers. During the years 1945–64, another practice entailing very high exposure occurred in the preparation department. The mixing of fibres which, until that time, was subject to some degree of control was transferred to a mezzanine floor where asbestos was moved around by men with pitch forks without any form of dust suppression: these mezzanine and baghouse exposures, which could neither be assessed nor identified with individual workers, have not been included in any analysis, neither ours nor that of Dement *et al.*

With these reservations, the data available on exposure are summarised in table 3 grouped by duration of exposure. This shows that the average

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

	Length of gross service (years)				
	<1	*1, <5*	*5, <20*	*≥20*	*Total*
No	950	574	421	465	2410*
Average age at start (years)	25·6	25·9	26·5	25·2	25·77
Gross service (years)	0·39	2·43	10·50	31·86	8·71
Net service (years)	0·37	1·81	7·55	29·51	7·59
Average dust concentration (mpcf)	2·11	1·86	1·67	1·23	1·80

*Excluding five whose employment histories were incomplete.

age at start of work and the average dust concentration over the net years of employment were similar in all four duration groups.

STATISTICAL ANALYSIS

The mortality and exposure data were analysed in two ways. The first followed the orthodox man-years life table approach of Hill[8] and others, whereby standardised mortality ratios (SMRs) are derived from comparison of *observed* numbers of deaths with numbers *expected* from mortality rates in a standard population. In this case age-, sex-, race (colour)-, year-specific rates for South Carolina were used. The second approach, essentially internal and case-control in type, followed the Mantel-Haenszel (or log rank) procedure,[9] yielding relative risks from entirely intracohort comparisons. In calculating SMRs a "lag time" of 10 years before death (or end of 1977) was imposed in determining exposure, and only deaths 20 years or more from first employment were included. In the Mantel-Haenszel analysis the same exclusions were applied, controls being selected from all other members of the cohort of the same sex and colour (black or white) who met the following criteria: (1) alive at death of case, (2) same year of birth, if in or after 1900, or within five years if before 1900, (3) within five years of date of first employment, before or after 1938. The statistical significance of differences between observed and expected numbers in this analysis and for departures from linearity were calculated as χ^2 values by the method of Peto and Pike.[10] Lines were fitted to exposure-response results by Liddell using the method of Hanley and Liddell (to be published).

Results

Table 4 summarises the mortality experience of the male cohort, based on the modified life table analysis. Overall, the SMR (all causes) is 27% above expectation and perhaps twice that in men employed

Table 4 *Male deaths 20 years after first employment, by cause, in relation to duration 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	159	107·4	113	122·7	120	156·1	178	136·7	570	127·4
Malignant neoplasms:										
Respiratory	8	78·2	10	163·9	15	304·1	26	317·3	59	199·5
Abdominal	6	107·9	5	146·4	7	240·3	8	151·4	26	151·7
Other	12	130·2	7	124·9	9	195·9	7	46·2	35	127·5
Heart disease	69	108·9	34	87·6	45	141·7	70	120·8	218	113·7
Respiratory tuberculosis	1	231·8	1	347·8	1	307·9	1	131·5	4	222·8
Other respiratory	3	53·3	3	85·6	2	78·3	27	557·5	35	207·3
Pneumoconiosis	(0)	—	(0)	—	(0)	—	(20)	—	(20)	—
Cerebrovascular	9	83·0	14	193·0	6	107·3	9	76·2	38	107·2
Accidents	18	121·2	8	89·7	5	75·8	9	85·0	40	97·0
Other known	30	116·9	28	175·5	23	177·7	21	92·3	102	132·4
Not known	3		3		7		0		13	

*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**	*Dust exposure (mpcf.y)*									
	<10		*10 <20*		*20 <40*		*40 <80*		*≥80*	
	0	*SMR*	*0*	*SMR*	*0*	*SMR*	*0*	*SMR*	*0*	*SMR*
All causes	376	115·5	55	125·5	63	156·9	43	170·8	33	264·4
Malignant neoplasms:										
Respiratory	31	143·1	5	182·7	8	304·2	7	419·5	8	1031·9
Abdominal	14	114·9	4	231·6	4	247·0	4	383·6	0	—
Other	28	140·0	3	109·2	1	44·9	0	—	3	383·5
Heart disease	143	103·5	28	143·6	29	166·6	10	88·6	8	149·9
Respiratory tuberculosis	3	264·4	0	—	0	—	1	634·4	0	—
Other respiratory:	8	65·9	2	119·5	6	421·7	13	1407·8	6	1296·0
Pneumoconiosis	(0)	—	(0)	—	(3)	—	(9)	—	(8)	—
Cerebrovascular	29	115·3	2	50·0	4	124·4	2	93·4	1	99·8
Accidents	31	99·2	2	54·1	5	152·9	1	49·4	1	120·0
Other known	79	140·4	9	116·9	4	630	5	111·5	5	263·3
Not known	10		0		2		0		1	

Table 6 *Dust exposure in male deaths from selected causes and controls (Mantel-Haenszel analysis[2])*

Linearity	*Dust exposure (mpcf.y) accumulated up to 10 years before death of case*					*Chi square*	
	<10	*10–19*	*20–39*	*40–79*	*≥80*	*Difference*	*Linearity*
Pneumoconiosis (ICD 523)							
Deaths	0	0	3	10	4	17·36	10·80
Expected	3·1	2·2	3·8	4·1	3·7		
Relative risk	—	—	—	—	—		
Lung cancer (ICD 162–4):							
Deaths	25	3	8	7	6	24·08	20·43
Expected	32·4	5·4	5·3	3·7	2·2		
Relative risk	1	0·98	2·95	4·32	15·00		
Abdominal cancer (ICD 150–9):							
Deaths	13	4	2	4	0	4·06	2·53
Expected	15·5	2·9	2·5	2·1	0		
Relative risk	1	1·64	1·30	7·63	—		
All causes:							
Deaths	331	45	53	37	24	14·42	10·63
Expected	348·0	46·2	48·5	32·4	15·0		
Relative risk	1	1·05	1·43	1·51	2·17		

five years or more. As there is (*a*) a 7% excess even in men employed less than one year, unexplained by any asbestos related cause of death, and (*b*) a 32% overall excess in deaths of "other known causes" the SMRs are probably somewhat inflated, mortality in South Carolina having presumably provided an imperfect basis for comparison. Much of the excess, however, is clearly attributable to respiratory cancer, pneumoconiosis, and gastrointestinal cancers. Table 5 shows the cohort mortality, related to dust exposure. There is a steady gradient from 115·5 to 264·4 for mortality (all causes) and a much steeper slope for respiratory cancer and also for selected other respiratory diseases (which include pneumoconiosis). No clear trend is apparent in the other diagnostic categories.

Table 6 shows the results of the a posteriori Mantel-Haenszel analysis for certain diagnostic groups only. The number of deaths included in this analysis falls short of those used in tables 4 and 5—for example, 490 compared with 570 from all causes; in the remainder no matching control could be found. There is clear confirmation of a statistically significant linear trend in lung cancer, pneumoconiosis, and deaths (all causes) but no convincing association for the abdominal cancers.

Only one death ascribed to mesothelioma was found—a man born in 1904 who died in 1967. He was first employed at the plant in 1925, worked as a mule spinner from 1933 to 1955 and as an oven helper until he left in 1965. The tumour was stated to be peritoneal but there was no necropsy.

Discussion

The *pattern* of mortality in this cohort of chrysotile textile workers is similar to that reported for Quebec chrysotile miners and millers, particularly those employed at Thetford Mines.[7] Overall, the SMRs for the factory workers are somewhat higher than for the miners (perhaps due in part to questions of comparability with the reference populations). There is the same scarcity of deaths attributed to mesothlioma and, in both cohorts, the relationship of lung cancer mortality to accumulated dust exposure is virtually linear. It is only when we examine the actual levels of exposure that the astonishing difference between the experience of these two chrysotile exposed cohorts is to be seen. This is illustrated in fig 1 where, to facilitate comparison, the SMRs in both cohorts are based on exposure accumulated to age 45 (see McDonald *et al*[7]). In fact, the slope of the exposure-response line for lung cancer in the textile

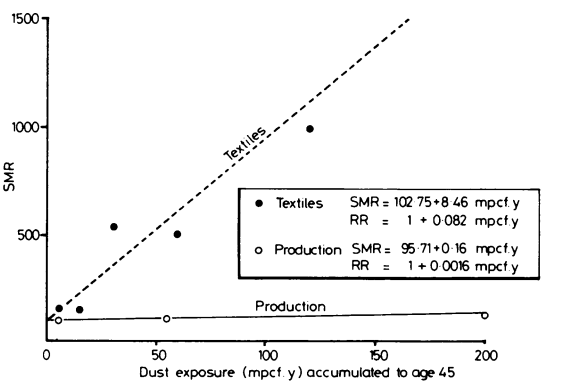

Fig 1 *Respiratory cancer SMRs in relation to dust exposure accumulated to age 45 in chrysotile production and textile manufacture.*

workers is 50 times more steep than that observed in miners and millers. We thus confirm almost exactly the findings of Dement *et al* in their smaller cohort from the same plant[6]; the agreement is very close (see fig 2). The data shown in this graph are based on mortality for white men only, 15 years or more from first employment and therefore differ somewhat from the figures in table 5.

The difference between fibre production and textile manufacture, if real, has important implications.[20] The possibility of error must therefore be considered. Our data on mortality and those of Dement *et al* are independent but those on exposure are not. The dust concentrations, although extraordinarily low when compared with mining and milling were numerous, reasonably consistent among several groups of observers, and similar to levels recorded in the other American textile plant we have studied[4 11] and probably also to the Rochdale textile plant,[12] although there are technical difficulties in making the latter comparison. The concentrations were also in the same range as those recorded in an American asbestos friction products plant in which little or no excess lung cancer mortality occurred.[4 21] These observations all raise questions of dust fibre conversion in these varied industries; this will be discussed below. In like manner, we must consider the possibility of error in the chrysotile mining and milling studies. In these there were far more environmental measurements, many made by our own team.[13] So far as mortality was concerned, our findings were consistent at four different points over a nine year period[14] and were essentially confirmed independently in a subcohort by Nicholson *et al.*[15]

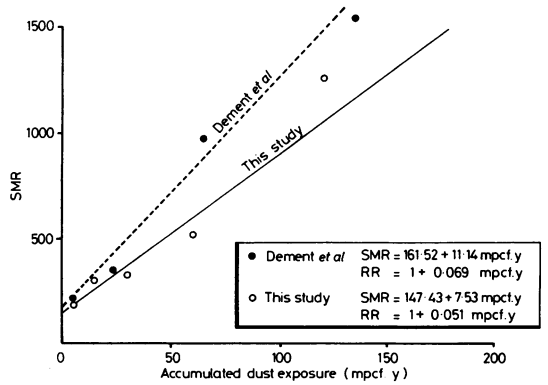

Fig 2 *Respiratory cancer SMRs in white men 15 years or more from first employment in relation to accumulated dust exposure. Comparison of this study and that of Dement* et al.[6]

It thus seems that error per se can be dismissed, but there remain two other possibilities. The first is that both Dement *et al* and we failed to give sufficient weight to the occasional extremely high exposures that, from all accounts, many or most employees in the plant experienced during overtime work "whipping the burlap" in the baghouse, 1937–53, and certain of them using pitch forks on the mezzanine, 1945–54. We are inclined to think that these casual exposures, although significant, are unlikely to explain a more than twofold error in accumulated exposure estimates.

The second possibility concerns the fibre equivalence of impinger dust counts. This problem can be considered first at the level of equivalence of fibres more than 5 μ in length per ml, as counted on a membrane filter. In the mines and mills, conversion factors varied greatly[16 17] but, for lung cancer mortality, an average factor of 3·64 appeared to us appropriate.[18] Nicholson accepted an earlier approximation of "about 3."[15] In the South Carolina textile plant Dement *et al* looked carefully at this question and used a factor of 3 for all operations except preparation, where they used 8.[6] In a much earlier investigation by Ayer *et al* it was concluded that 6 might be appropriate for textile processes in that era.[19] In our own review of the environmental measurements particularly during periods when both dust and fibre concentration were assessed, we observed a range from 1·3 to 10·0, with an average of about 6. These discrepancies are relatively minor and it thus seems improbable that, even allowing for *(a)* substantial underestimation of the actual exposure experienced by the textile workers, *(b)* the roughly twofold greater fibre equivalence in textiles than in mining and milling, and *(c)* some possible inflation of the SMRs resulting from use of the South Carolina mortality rates, the 50-fold difference in exposure response can be reduced to less than about 10-fold.

Of course, there remain other factors different in nature—for example, smoking habits or the presence of cocarcinogens. There is no convincing evidence of the latter, although various oils were often used in the textile plant for spraying yarn during spinning to prevent breakages. Only major differences in smoking habits could have much effect on the comparison between the textile and production workers. Our information on the question is fragmentary but does not suggest that this was so. Dement *et al* presented a table which showed that, in 1965, the smoking habits of a sample of workers in their cohort were almost identical with those for US white men, with about 25% non-smokers.[6] In October 1982 we surveyed the smoking records of 553 current and recently retired employees at the plant and found that 171 (31%) were non-smokers; however, of 246

men born before 1930 (and therefore more representative of our cohort), 44 (18%) were non-smokers and there were indications that the proportion was lower still in the older men. Data on the Quebec mining cohort suggest that the proportion of non-smokers also varied with date of birth, falling from 25% in those born 1891–95 to 9% in those born 1916–20. Once again, we find no major difference.

Thus we are left with two well established linear relationships between chrysotile dust exposure and risk of lung cancer, the one 50 times steeper than the other. Conversion from impinger particle counts to membrane filter fibre counts and underestimation of exposure might each halve the difference, and other possible sources of bias which have been discussed might reduce the difference further but hardly to less than 10-fold in all. That this may be the general situation as regards asbestos textile workers is supported by the results of the other cohort studies.[11 12] If cocarcinogens can be discounted the cause of the phenomenon must be in the nature of the chrysotile fibre exposure in these widely different industrial circumstances. Noting the experimental evidence in animals, we are led to speculate that the distribution of very fine fibres, seen only by electron microscope, may be different in the two situations. We have now embarked on research to test this hypothesis.

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