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Mortality of workers employed in two asbestos cement manufacturing plants

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ABSTRACT In a study of the mortality experience of 6931 employees of two New Orleans asbestos cement products manufacturing plants over 95% were traced. Chrysotile was the primary fibre used in both plants. Plant 1 also used small amounts of amosite and, later, crocidolite irregularly whereas plant 2 used crocidolite steadily in pipe production. Previously reported exposure concentration estimates were revised, based on additional air sampling data and re-evaluation of these data. Workers in the two plants had similar duration of employment (overall, a mean of 3·8 years) and estimated exposure concentration (a mean of 7·6 million particles per cubic foot (mppcf)). Mortality was similar for these plants and comparable with Louisiana rates for all causes combined, non-malignant causes, and primary cancers of specified sites other than lung. Short term workers from both plants showed raised and similar risk of lung cancer, but risk among longer term workers differed—for example, for workers employed over one year there was no excess in plant 1 (16 observed, 17·2 expected) but a significant excess in plant 2 (52 observed, 28·9 expected, $p < 0·001$). After excluding short term workers, risk of lung cancer in plant 2 showed a significant trend with estimated cumulative asbestos exposure; using a conversion of 1·4 fibres/ml = 1 mppcf, the slope of the line was 0·0076. The slope for plant 1 was 0·0003. Among all workers (the 6931, plus 167 early employees) ten mesotheliomas had occurred up to 1984: two from plant 1, eight from plant 2. In plant 2 a case-control analysis found a relation between risk of mesothelioma and duration of employment ($p < 0·01$) and proportion of time spent in the pipe area ($p < 0·01$), thus adding to the evidence of a greater risk of mesothelioma from crocidolite than chrysotile asbestos. A review of the mortality findings of eight cohorts of asbestos cement workers is presented.

Although world wide consumption and production of asbestos is reported to have peaked in 1978–9,[1] asbestos continues to be used extensively, especially in less developed countries. In fact, there has been an increasing trend of asbestos consumption in Asian countries since 1980 and this trend is expected to continue. Approximately 85% of this demand is for asbestos cement products, primarily for construction materials.[1] Because of this continuing use of asbestos cement and because of the evidence that the risk of asbestos related lung cancer varies with industrial process (after controlling for cumulative asbestos exposure), further evaluation of the potential risk from this segment of the industry continues to be an important need in the area of asbestos associated health risk.

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This paper reports the mortality experience up to 1982 of workers in two asbestos cement manufacturing plants in New Orleans, Louisiana, USA. Earlier reports of this population,[2 3] with follow up to 1974, showed a dose response relation between the risk of lung cancer and estimated cumulative asbestos exposure, and found suggestive but inconclusive evidence of a greater risk of lung cancer among workers exposed to a mixture of chrysotile and crocidolite asbestos than among those with exposure to chrysotile only. Since only two cases of mesothelioma had occurred, no useful comparison of mesothelioma risk by fibre type exposure could be made.

A possible limitation of the earlier study was the assumption that the 24% of the cohort with neither a death claim nor confirming transaction with the Social Security Administration (SSA) were, in fact, alive. Nevertheless, the study's findings were sup-

ported by the observation of essentially identical dose response relations based on the entire population and on the long term employees, of whom over 90% had been traced.

Environmental conditions

The two plants, both of which opened in the 1920s and produced asbestos cement building materials, have been described previously.[2] The smaller (plant 1), located in a commercial area of the city, employed about 150 workers in the early 1940s and reached maximum employment of over 500 workers in 1951 and 1952. The larger plant, located outside the city, employed about 300 workers in the early 1940s, with a maximum exceeding 900 in 1949 and 1950.

A complete review of the dust exposure data and revision of the exposure estimates were undertaken as part of this updated study. During this review, additional exposure measurements for plant 2, some made in the 1950s, which had not been available for the earlier analyses were located and incorporated into the estimates of past exposure levels. Air sampling data, collected by industry, insurance company, and government personnel using the midget impinger (recorded in millions of particles per cubic foot (mppcf)), were first collected in both plants in 1952 (table 1). A total of only 100 impinger measurements, most during the 1960s, were available for plant 1. In plant 2, 248 measurements were made during the 1950s and more than 1100 during the 1960s. Membrane filter sampling (in fibres per millilitre (f/ml)) began in 1969. Since much of the employment of this population was in the 1940s and 1950s, all exposure concentration estimates were made in mppcf.

The exposure estimates made during the earlier study were based on both the air sampling data then available and anecdotal information from company management and long term employees. This approach was taken to adjust for the small number of measurements recorded for some job titles and to make maximal use of available information concerning exposure conditions. Consequently, in the previous study there were instances in which jobs with relatively similar exposure measurements were assigned different exposure concentration estimates because of anecdotal information. In the current study anecdotal information was used only for combining jobs into categories of likely similar exposure levels and for contrasting the relative exposure conditions of the 1940s with those of the 1950s, the period with the earliest exposure measurements.

Table 1 *Number of air samples pre-1970 by year and plant*

Year	*Plant 1*	*Plant 2*
1952–3	7	56
1955–8	0	175
1959–60	19	90
1961–4	25	506
1965–8	49	594
1969	0	243
Total	100	1664

For each period, all jobs in a category were assigned the same estimated concentration level, which was taken to be the mean of the available exposure measurements for these jobs. In calculating this mean very high measurements found to be statistical outliers based on a lognormal distribution were first recoded to be equal to the highest non-outlying value. This procedure was adopted as preferable to either retaining these possibly invalid measurements or deleting them entirely. Deletion seemed inappropriate since the possible validity of these high values was supported by their tendency to occur during the earliest years of sampling, when peak employment was attained and high exposure levels may, in fact, have occurred.

For some jobs, the availability of additonal dust measurements and the decreased reliance on anecdotal information concerning relative dustiness of jobs resulted in revised exposure estimates substantially different from the earlier estimates. Table 2 provides a summary comparison of the earlier and revised estimates by calculating the average estimated

Table 2 *Mean estimated asbestos exposure concentration (mppcf) of workers during first five years' employment, by plant, year of initial employment, and exposure assignment method. (Number of workers in parentheses)*

	Plant 1			*Plant 2*		
Year of initial employment	*Previous estimates*		*Revised estimates*	*Previous estimates*		*Revised estimates*
1940–9	6·8	(1571)	10·0	21·7	(3041)	8·0
1950–9	5·2	(696)	7·2	17·9	(648)	9·1
1960–9	3·4	(298)	1·3	7·0	(462)	3·9

exposure concentrations of workers during the first five years after hire—that is, over each person's career or first five years of employment, whichever was less—by decade of initial employment. In plant 1 the revised and earlier estimates were reasonably similar, though the revised estimates tended to be slightly higher than the earlier estimates for the 1940s and 1950s and somewhat lower for the 1960s. Revision had a substantial effect on the plant 2 estimates, however: revised estimates are about one third the previous estimates up to the 1950s and about one half afterwards.

In both plants chrysotile was the primary type of asbestos used. In plant 1 which consisted of one building, amosite was also used (in corrugated siding) from the early 1940s until the late 1960s, and crocidolite was used occasionally for about ten years beginning in 1962. This information concerning amosite usage differs from that reported earlier,[1 2] when it was believed that amosite had not been introduced into the plant until the late 1950s. The latest information, however, indicates that corrugated siding always contained a small amount of amosite.

Plant 2 consisted of four separate buildings, each manufacturing different products. Pipe production, which opened in 1946, used crocidolite in addition to chrysotile. All other areas used chrysotile only. Amosite was never used.

Population and methods

Workers were identified for the study by abstracting all job records on file in the two plants in 1970. The study population consisted of all men who had been employed for at least one month before 1970 and for whom a valid Social Security number was available from company records. Because of record keeping procedures in the plants, workers who qualified for the study but who began work in plant 1 before 1942 (n = 39) or in plant 2 before 1937 (n = 128) were those still employed in these years. These workers therefore constitute a survivor population and, as such, may not be representative of all early employees. Unless otherwise stated, all results will be presented only for workers hired during the stated years or later, to be referred to as the primary cohort.

Excluding the 167 early workers, the primary cohort consisted of 6931 men, of whom 2565 (37%) were employed in plant 1 and 4366 (63%) in plant 2. Overall, the population was 54% black, 46% white. An additional 321 women employees are not included in this report.

To assess the completeness of employee identification, copies of plant 2 SSA quarterly reporting forms were obtained for the years 1942, 1945, and 1948. (Because of a change in ownership in 1952, forms for plant 1 could not be obtained.) Of the 1291 employees listed on these forms for the first two quarters of any one year (implying employment for at least one month), 1244 (95·6%) were included in the study population. Thus inclusion of the plant 2 target population was judged to be satisfactory.

Sixty one per cent of plant 1 workers were initially employed during the period 1942–9, 74% of plant 2 workers during 1937–49 (table 3). The two plants were similar on the basis of average estimated exposure concentration (7·8 and 7·5 mppcf for plants 1 and 2, respectively) and duration of employment (both means under four years, medians less than one year). Overall, approximately 60% of the cohort was employed for one year or less. On average, age at initial employment was higher in plant 1 (31·7 years) than in plant 2 (26·8 years) and was especially high in plant 1 during the war years (39·0 years).

Follow up was up to 1982 or age 80, whichever was earlier. Over 96% of the population was traced (table 4) through the help of federal, state, and local agencies. Trace was better among those employed for more than one year (97·7%) than for other workers (95·1%). Of the 2143 identified as dead, death certificates for 2014 (94%) were obtained from the individual states. These were coded by a nosologist according to the eighth revision of the International Classification of Diseases (ICD).[4] Although the same

Table 3 *Description of cohort, by plant and year of initial employment*

	Plant 1				*Plant 2*			
Initial employment year	*No*	*Mean initial age (years)*	*Mean average concentration* (mppcf)*	*Mean (median) years employed*	*No*	*Mean initial age (years)*	*Mean average concentration* (mppcf)*	*Mean (median) years employed*
1937–41				—	447(10%)	25·8	6·4	5·5(1·3)
1942–5	368(14%)	39·0	8·9	5·7(2·3)	925(21%)	29·8	9·0	2·1(0·4)
1946–9	1203(47%)	30·3	9·6	3·6(0·5)	1884(43%)	26·0	7·4	5·2(0·6)
1950–9	696(27%)	30·8	6·8	2·9(0·7)	648(15%)	26·5	8·7	2·8(0·7)
1960–9	298(12%)	31·0	1·3	2·5(1·6)	462(11%)	25·6	3·9	2·0(1·1)
Total	2565(100%)	31·7	7·8	3·6(0·7)	4366(100%)	26·8	7·5	3·9(0·6)

*Average concentration for each worker calculated over his plant career or first 20 years, whichever was shorter.

Table 4 *Trace status*

	Plant 1		*Plant 2*		*Plants combined*	
	No	*%*	*No*	*%*	*No*	*%*
Alive	1592	62·1	2936	67·2	4528	65·3
Dead	886	34·5	1257	28·8	2143	30·9
Untraced	87	3·4	173	4·0	260	3·8
Total	2565	100·0	4366	100·0	6931	100·0

nosologist was used as in the earlier study, all certificates, including those obtained previously, were coded for this study. (Few of those coded both times were coded differently; these discrepancies, none of which involved malignancies, were resolved by consultation with the nosologist.) In the analyses deaths for which certificates were not obtained were allocated to categories of causes of death in the same proportion as those with certificates.

Mortality experience was primarily compared with that expected based on Louisiana mortality rates, although United States rates were also used. Louisiana rates were obtained both from the State of Louisiana Department of Health and from Marsh and Preininger.[5] Age adjusted 1960–79 Louisiana malignancy rates are known to be higher than United States rates: state rates for all cancers combined are approximately 8% higher than United States rates; state lung cancer rates are 29% higher for whites and 9% higher for blacks.[6]

Approximately 85% of the death certificates were obtained from Louisiana and of these, approximately 75% were from the two parishes (counties) in which the plants were located. Malignancy mortality rates for these two parishes are higher than for the state: parish rates for all cancer combined are approximately 14% higher than state rates, and lung cancer rates are 10% and 38% higher for whites and blacks, respectively.[6] Parish rates were obtained for this study, but since the age specific, race specific, and cause specific rates were based on small numbers in some cases, these rates were variable and therefore judged too unstable for use.

Standardised mortality ratio (SMR) analyses[7] were carried out using a computer program written in Britain (J Peto). Case-control analysis[8] of the mesothelioma cases was executed using the logistic regression routine of the statistical software package BMDP.[9] All dose response relations were assessed using iteratively weighted least squares regression.[10]

In analysing risk in relation to estimated cumulative asbestos exposure, during each five year period (20 or more years after initial exposure) each person contributed person-years to the cumulative exposure category attained ten years previously. In this way relatively recent exposures (10–15 years previously) were disregarded in determining the cumulative exposure category for each worker.

Results

For these two plants, the ratios of observed number of deaths to expected numbers (O/E) were similar (20 or more years after initial exposure) for all causes combined (0·91 and 0·95), and for all malignancies (1·12 and 1·14) (table 5).

Among plant 1 workers, the excess malignancies were primarily due to lung, colon-rectal, urinary, and residual cancers, though none of these excesses was statistically significant. The 20 residual cancers were primarily cancers of unspecified sites (n = 8) and secondary respiratory/digestive cancers (n = 5). Two workers had pneumoconiosis listed as the cause of death.

In plant 2 there were statistically significant excesses of lung cancer (107 observed, 74·3 expected, $p < 0·01$) and residual cancers (42 versus 29·4, $p < 0·01$) and a non-significant excess of stomach cancers (15 observed, 12·0 expected). The 42 residual cancer were primarily cancers of unspecified/ill defined sites (n = 26) and secondary respiratory/digestive cancers (n = 6). Five deaths were due to pneumoconiosis in the primary cohort (table 5) plus another death among workers hired before 1937.

Of the eight workers from the two plants with pneumoconiosis listed as the cause of death, duration of employment ranged from 19 to 35 years.

Use of United States rather than Louisiana men as a comparison population resulted in lower expected numbers and therefore higher relative risks (relative to United States men): for plants 1 and 2, respectively, values of 1·00 and 1·04 for all causes combined, 1·21 and 1·24 for all malignancies (both statistically significant), and 1·33 and 1·69 for lung cancer (both statistically significant).

DURATION OF EXPOSURE

The risk of cancer for plant 1 by duration of employment (table 6) showed no trend. The shortest term workers experienced the highest risk for all malignancies, lung cancer, and residual cancer. The relative risk of lung cancer (relative to Louisiana men) was

1·39 (26 observed, 18·7 expected) among those employed six months or less compared with a value of 0·98 (22 observed, 22·5 expected) for other workers. The risk of bladder and kidney cancer, while raised overall (7 observed, 4·1 expected), was not statistically significant and did not increase with duration of employment. Colon rectal cancer was raised (non-significantly) among workers employed for five years or less but not for those employed longer.

In plant 2 overall mortality, all cancers combined, lung cancer, and residual cancers showed a general trend of increasing risk with duration of employment (table 7). Workers employed for three months or less, however, experienced an excess risk of lung cancer (34 observed, 24·3 expected, $p < 0·05$), whereas those employed for four to 12 months did not. The non-significant overall excess risk of stomach cancer did not show an increasing trend with duration. In the category of residual cancers, if the three mesotheliomas are excluded, there is no clear trend of risk

Table 5 *Observed and expected deaths 20 or more years after initial exposure by plant*

	Plant 1 (n = 1898)			*Plant 2 (n = 3594)*			*Plants combined (n = 5492)*		
	*O**	*E**	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*
All causes	477	522·2	0·91	874	922·7	0·95	1351	1444·9	0·94
All malignancies (140–209)†	127	113·9	1·12	226	199·1	1·14‡	353	313·0	1·13‡
Respiratory (162–163)	48(1)	41·2	1·17	107	74·3	1·44§	155(1)	115·5	1·34§
Larynx (161)	2	2·0	1·00	1	3·4	0·30	3	5·4	0·56
Buccal/pharynx (140–149)	5	4·4	1·13	6	7·8	0·77	11	12·2	0·90
Digestive (150–159)	26	29·2	0·89	44	49·7	0·89	70	78·9	0·89
Oesophagus (150)	5	5·0	1·00	7	7·9	0·89	12	12·9	0·93
Stomach (151)	8	7·5	1·07	15	12·0	1·25	22	19·5	1·13
Colon, rectum (153–154)	10	8·3	1·20	11	15·0	0·73	21	23·3	0·90
Other digestive (152–159)	3	8·4	0·36	11	14·9	0·74	15	23·3	0·64
Prostate (185)	10	9·5	1·06	14	13·6	1·03	24	23·1	1·04
Bladder (188)	3	2·3	1·33	2	3·9	0·52	5	6·2	0·81
Kidney (189)	4	1·8	2·25	3	3·5	0·86	7	5·3	1·32
Lymphatic (200–209)	9	7·1	1·27	7	13·5	0·52	16	20·6	0·78
Residual cancers	20	16·2	1·23	42(3)	29·4	1·43‡	62(3)	45·8	1·35‡
Cardiovascular (390–448)	218	265·4	0·82	420	462·9	0·91	638	728·3	0·88
Influenza, pneumonia, bronchitis, emphysema, asthma (470–474, 480–486, 490–493)	19	19·4	0·98	29	33·9	0·86	48	53·3	0·90
Pneumoconiosis (515)	2			5	—	—	7	—	—
External causes (800–998)	40	41·6	0·96	75	83·2	0·90	115	124·8	0·92
Residual causes	71	81·9	0·87	119	143·6	0·83	190	225·5	0·84

*O = Observed number, E = expected number based on Louisiana rates (number of mesotheliomas in parentheses).
†ICD 8th revision code.
‡Observed significantly higher than expected, one tailed $p \leq 0·05$, based on a Poisson distribution with parameter E.
§Observed significantly higher than expected, one tailed $p \leq 0·01$, based on a Poisson distribution with parameter E.

Table 6 *Observed and expected deaths for selected causes, plant 1 employees, 20 or more years after initial exposure, by duration of exposure*

Duration	*≤6 months (n = 902)*			*7 months–1 year (n = 253)*			*>1–5 years (n = 376)*			*>5–15 years (n = 157)*			*>15 years (n = 210)*		
	*O**	*E**	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*
All causes	218	227·7	0·96	55	62·5	0·88	91	104·9	0·87	55	51·0	1·08	58	76·1	0·76
All malignancies	65	50·1	1·30‡	14	14·1	0·99	21	22·4	0·94	12	10·5	1·14	15	16·7	0·90
Respiratory	26(1)	18·7	1·39	6	5·3	1·13	6	8·0	0·75	4	3·5	1·15	6	5·7	1·05
Digestive	11	12·6	0·87	2	3·6	0·56	6	5·8	1·04	3	2·8	1·07	4	4·5	0·89
Oesophagus	2	2·2	0·89	0	0·7	—	1	0·9	1·06	0	0·4	—	2	0·7	2·71
Stomach	5	3·1	1·62	0	0·9	—	1	1·5	0·67	2	0·8	2·62	0	1·2	—
Colon, rectum	2	3·6	0·56	2	1·0	2·01	4	1·7	2·38	1	0·8	1·20	1	1·2	0·81
Other	2	3·7	0·55	0	1·0	—	0	1·7	—	0	0·8	—	1	1·3	0·78
Kidney, bladder	3	1·7	1·72	1	0·5	2·07	1	0·8	1·22	1	0·4	2·48	1	0·6	1·71
Lymphatic	3	3·2	0·95	1	0·9	1·15	3	1·4	2·14	1	0·7	1·52	1	1·0	0·98
Residual†	22	13·9	1·58‡	4	3·9	1·03	5	6·4	0·78	3	3·2	0·94	3	4·8	0·63
Pneumoconiosis	0	—	—	0	—	—	0	—	—	0	—	—	2	—	—

*O = Observed, E = expected based on Louisiana rates (number of mesotheliomas in parentheses).
†Residual cancer also contains larynx, buccal, and prostate cancer, which had been separate in table 5.
‡Observed significantly higher than expected, one tailed $p \leq 0·05$; see footnote table 5.

for residual cancer with duration of employment. Nevertheless, it is possible that some of these cancers are related to asbestos, possibly lung cancers.

Risks of lung cancer (20 or more years after initial employment) among the shortest term workers in the two plants were almost identical: relative risks of 1·39 among 902 plant 1 workers employed six months or less and 1·40 among 1175 plant 2 workers employed three months or less. There was no evidence of higher exposure concentrations for these groups when compared with other workers hired during the same periods.

CUMULATIVE ASBESTOS EXPOSURE

For plant 1, if the shortest term workers (six months or less) are included in the cumulative exposure analysis then the results are similar to those for employment duration: an excess respiratory cancer rate only in the lowest exposure category. If they are excluded from the analysis (table 8) then there are no statistically significant excesses. It will be noted that the boundaries for the first two duration categories differ for the two plants. For both plants analyses were carried out classifying workers employed ⩽1 year into three categories: one to three months, four to six months, and seven to 12 months. In both cases about one half of these workers were in the one to three months group. In plant 2 because results for the four to six and seven to 12 months groups were similar, with neither group exhibiting excess risk of lung cancer, these two groups were combined (table 7). In plant 1 results for the four to six months group were more similar to the shorter term group (and these two groups were therefore combined in table 6): a non-significant excess risk of lung cancer in the one to three months group (n = 619, 16 lung cancers observed, O/E = 1·23), a marginally significant excess in the four to six months group (n = 283, 10 lung cancers, O/E = 1·78, p = 0·06), and a slight excess in the seven to 12 months group (n = 253, 6

Table 7 *Observed and expected deaths for selected causes, plant 2 employees, 20 or more years after initial exposure, by duration of exposure*

Duration	*⩽3 months (n = 1175)*			*4 months–1 year (n = 1003)*			*>1–5 years (n = 710)*			*>5–15 years (n = 204)*			*>15 years (n = 502)*		
	*O**	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*
All causes	271	308·6	0·88	239	268·4	0·89	190	174·3	1·09	47	42·3	1·11	127	129·0	0·98
All malignancies	70	66·0	1·06	54	57·8	0·94	49	37·8	1·30†	14	9·2	1·53	39	28·3	1·38†
Respiratory	34	24·3	1·40†	21	21·2	0·99	20	14·4	1·39	8	3·6	2·24†	24	10·9	2·20‡
Digestive	16	16·7	0·96	11	14·6	0·75	13	9·4	1·38	0	2·3	—	4	6·9	0·58
Oesophagus	1	2·7	0·38	4	2·4	1·68	2	1·5	1·38	0	0·4	—	0	1·1	—
Stomach	6	4·1	1·46	4	3·6	1·10	4	2·2	1·82	0	0·5	—	1	1·5	0·65
Colon, rectum	5	5·0	1·00	2	4·3	0·47	1	2·8	0·36	0	0·7	—	3	2·1	1·43
Other	4	4·9	0·81	1	4·3	0·23	6	2·8	1·77	0	0·7	—	0	2·1	—
Kidney, bladder	1	2·5	0·41	2	2·1	0·96	0	1·4	—	1	0·3	2·9	1	1·1	0·93
Lymphatic	2	4·4	0·45	3	3·9	0·78	1	2·6	0·38	1	0·6	1·56	0	2·0	—
Residual§	17	18·2	0·93	17(1)	16·1	1·06	15	10·1	1·49	4	2·4	1·67	10(2)	7·5	1·33
Pneumoconiosis	0	—	—	0	—	—	0	—	—	0	—	—	5	—	—

*O = Observed, E = expected based on Louisiana rates.
†O/E significantly higher than 1·0, p ⩽ 0·05; see table 5 footnote.
‡O/E significantly higher than 1·0, p ⩽ 0·01; see table 5 footnote.
§Residual cancers also contains larynx, buccal, pharynx, and prostate cancer, which had been separate in table 5.

Table 8 *Observed and expected deaths for selected causes, plant 1 employees with more than six months' employment, 20 or more years after initial exposure, by cumulative asbestos exposure category*

*Cumulative asbestos exposure (mppcf-yrs)**	*<6 (4)† (160)‡*			*6–24 (13) (384)*			*25–49 (35) (155)*			*50–99 (74) (141)*			*⩾100 (183) (156)*		
	O	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*
All causes	30	33·0	0·91	94	100·8	0·93	41	49·3	0·83	52	54·5	0·95	42	56·9	0·74
All malignancies	9	7·5	1·20	24	22·0	1·09	8	10·6	0·76	11	11·4	0·97	10	12·4	0·81
Respiratory	3(1)	2·9	1·04	9	8·0	1·12	2	3·7	0·55	3	3·8	0·78	5	4·1	1·23
Digestive§	1	1·8	0·55	6	5·6	1·08	3	2·8	1·08	3	3·1	0·98	2	3·4	0·59
Kidney, bladder	0	0·3	—	2	0·8	2·57	0	0·4	—	1	0·4	2·35	1	0·4	2·33
Lymphatic	1	0·5	2·08	3	1·37	2·19	0	0·7	—	1	0·7	1·42	1	0·7	1·36
Residual	4	2·0	2·01	4	6·1	0·66	3	3·1	0·97	3	3·3	0·91	1	3·8	0·26
Pneumoconiosis	0	—	—	0	—	—	0	—	—	0	—	—	2	—	—

*During each five year period of follow up, each person contributed person-years to the cumulative asbestos exposure category which he had attained ten years previously (see text).
†Mean cumulative exposure for category.
‡Number for whom exposure category is maximum attained.
§Breakdown of specific sites omitted due to the small numbers.

lung cancers, O/E = 1·13). Thus raised risk of lung cancer was observed in both plants among short term workers—in fact, among those employed for six months or less. The reported categories (tables 6 and 7) were therefore chosen as most simply presenting the observation of an excess risk of lung cancer among short term workers without an excess in one or more categories of longer term employment.

Although no exposure category in table 8 showed a significant risk of lung cancer, there is somewhat of an increasing trend in the first, second, and highest categories. The iteratively weighted least squares regression line is O/E = 0·93 + 0·0009 x, where x is cumulative asbestos exposure (mppcf-yrs) and O/E is the ratio of observed to expected lung cancer cases. The intercept was not significantly different from 1·0 (using a goodness of fit test); fitting with an intercept of 1·0 yields the equation O/E = 1·0 + 0·0004 x. The slope was not significantly different from zero in either fit.

Based on data collected in one of these plants[11] the best factor for converting mppcf to f/ml will be assumed to be 1·4 f/ml = 1 mppcf. Using this conversion factor the slope of the above regression line when x is expressed in f/ml-yrs is 0·0003.

In plant 2, after excluding those with three months employment or less, there was a generally increasing trend of risk with exposure cateogory for all causes, all cancers, lung cancer, and, to a lesser extent, residual cancers (table 9). The regression line for lung cancer risk was O/E = 1·17 + 0·0085 x; the intercept was not significantly different from 1·0 (using a goodness of fit test). Forcing an intercept of 1·0 yields the equation O/E = 1·0 + 0·0107 x (x in mppcf-yrs). For x expressed in f/ml-yrs, this equation is O/E = 1·0 + 0·0076 x.

The cumulative exposure analysis was also performed including the short term workers, most of whom comprised an even lower cumulative exposure category (less than 3 mppcf-yrs). The results were similar to those for duration of employment, with an excess risk of lung cancer in the lowest cumulative exposure category but not in the next higher category.

AGE AND RACE

For each plant, risk in relation to employment duration and cumulative asbestos exposure was investigated separately for the two races and for two (for plant 1) or three (for plant 2) categories of age at hire. No effect of these factors was observed.

LUNG CANCER AND TYPE OF ASBESTOS FIBRE

All operations in plant 1 were housed in one building and there was only limited variability in recorded job titles in this plant (55% were listed only as "labourer"). Therefore, although amosite was used only in the production of corrugated siding, no attempt was made to categorise workers according to type of asbestos fibre exposure.

Workers in plant 2 were categorised into two groups: those who held one or more jobs in the pipe production building, who were assumed to have been exposed in these jobs to a mixture of crocidolite and chrysotile asbestos, and those never assigned to this building, assumed to have had only exposure to chrysotile. Overall, 37% of employees had been assigned for some time to the pipe area. Half the pipe workers had spent over 98% of their employment time assigned to the pipe area; 78% had spent at least half their employment time there.

Comparing pipe and non-pipe workers, estimated average concentration levels were similar (a mean of 6·7 for pipe workers versus 7·8 mppcf) but the pipe

Table 9 *Observed and expected deaths for selected causes, plant 2 employees with more than three months' employment, 20 or more years after initial exposure, by cumulative asbestos exposure category*

*Cumulative asbestos exposure (mppcf-yrs)**	*<6 (3)* (885)†*			*6–24 (12) (732)*			*25–49 (36) (252)*			*50–99 (71) (263)*			*⩾100 (164) (287)*		
	O	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*	*O*	*E*	*O/E*
All causes	211	238·7	0·88	189	177·5	1·07	67	72·5	0·91	75	67·4	1·11	61	58·0	1·05
All malignancies	49	51·3	0·95	45	38·4	1·17	22	15·6	1·41	22	14·5	1·51‡	18	13·2	1·37
Respiratory	20	18·9	1·06	19	14·5	1·31	12	6·0	2·00‡	10	5·5	1·81‡	12	5·2	2·31§
Digestive	10	13·0	0·77	13	9·5	1·36	1	3·8	0·26	3	3·6	0·83	1	3·1	0·32
Oesophagus	4	2·1	1·91	2	1·5	1·32	0	0·6	—	0	0·6	—	0	0·5	—
Stomach	3	3·2	0·93	4	2·3	1·74	1	0·8	1·18	0	0·8	—	1	0·7	1·50
Colon, rectum	2	3·9	0·51	1	2·8	0·36	0	1·2	—	3	1·1	2·73	0	1·0	—
Other	1	3·9	0·26	0	2·9	2·09	0	1·2	—	0	1·1	—	0	1·0	—
Kidney, bladder	1	1·9	0·54	1	1·4	0·72	1	0·6	1·65	0	0·5	—	1	0·5	2·00
Lymphatic	3	3·4	0·87	0	2·6	—	1	1·1	0·90	0	1·0	—	1	0·9	1·10
Residual	15(1)	14·1	1·06	12	10·4	1·15	7	4·1	1·71	9	3·9	2·31‡	3(2)	3·5	0·86
Pneumoconiosis	0			0	—	—	0	—	—	1	—	—	4	—	—

*See footnotes, table 8.
†Number for whom exposure category is maximum attained.
‡O/E significantly higher than 1·0, $p \leqslant 0·05$; see footnote, table 5.
§O/E significantly higher than 1·0, $p \leqslant 0·01$; see footnote, table 5.

workers had been employed for longer than other workers (a mean of 7·7 versus 1·7 years). Median years of initial employment were 1947 and 1945 for the pipe and non-pipe workers, respectively.

The risk of lung cancer in these two groups showed similar levels and trends with duration of employment (table 10), with both short term groups experiencing an excess.

Excluding the shortest term employees (three months or less), the risk of lung cancer showed an increasing trend with cumulative asbestos exposure category among both groups (table 11). Although the numbers are small in some cells, within the higher cumulative exposure categories those with exposure to a mixture of fibre types showed a higher risk than those who had been exposed to chrysotile only. The fitted dose response relations, however (iteratively weighted, with a forced intercept of 1·0) were O/E = 1·0 + 0·0100 x for the chrysotile group and O/E = 1·0 + 0·0106 x for the mixed fibre group. Thus the two slopes were similar, indicating no significant difference in risk of lung cancer by fibre type exposure after adjusting for cumulative asbestos exposure.

Since crocidolite was not used until 1946, there was a maximum of 35 years of follow up from a worker's initial exposure to crocidolite. The above results concerning risk of lung cancer by fibre type, however, were similar when follow up was ended at 35 years after initial exposure for all workers.

LUNG CANCER DOSE RESPONSE USING EARLIER EXPOSURE ASSIGNMENTS

The data for the current study were also analysed using the earlier exposure concentration estimates. The results were qualitatively similar to those using the revised estimates, with no trend of risk with estimated cumulative exposure level among plant 1 employees but a trend in plant 2. For plant 2, use of the earlier assignments yielded a regression line (with forced intercept of 1) of O/E = 1·0 + 0·0055 x, where x is in mppcf-yrs. The slope 0·0055 is about half the slope of 0·0107 obtained using the revised estimates.

Table 10 *Observed and expected deaths from lung cancer among plant 2 employees, 20 or more years after initial exposure, by employment duration and fibre type*

	Chrysotile only					*Chrysotile and crocidolite**				
Duration of employment (years)	*No*	*Concentration† (mppcf)*	*O*	*E*	*O/E*	*No*	*Concentration† (mppcf)*	*O*	*E*	*O/E*
⩽0·25	949	8·9	28	20·8	1·34	226	9·3	6	3·4	1·74
0·25–1	779	7·9	18	17·7	1·02	224	8·8	3	3·5	0·86
1–5	477	7·3	16	10·9	1·46	233	8·3	4	3·5	1·16
5–15	69	6·2	3	1·5	1·95	135	6·6	5	2·0	2·45‡
>15	89	6·4	5	2·2	2·26	413	5·9	19	8·7	2·18§
Combined	2363	8·0	70	53·2	1·32‡	1231	7·5	37	21·1	1·75§

*Employees with any employment in the pipe production area.
†Mean of the average estimated exposure concentrations (mppcf) of workers in each group, calculated over each worker's career up to ten years before end of follow up.
‡O/E significantly higher than 1·0, $p \leqslant 0·05$; see footnote, table 5.
§O/E significantly higher than 1·0, $p \leqslant 0·01$; see footnote, table 5.

Table 11 *Observed and expected deaths from lung cancer among plant 2 employees with more than three months' employment, 20 or more years after initial employment, by cumulative asbestos exposure and fibre type*

Cumulative asbestos exposure (mppcf-years)*	*Chrysotile only*				*Cumulative asbestos exposure (mppcf-years)*	*Chrysotile and crocidolite†*			
	No	*O*	*E*	*O/E*		*No*	*O*	*E*	*O/E*
<3	373	8	8·8	0·91					
					<6	199	4	3·1	1·31
3–5	313	8	7·0	1·14					
6–24	511	17	11·1	1·54	6–24	221	2	3·4	0·59
25–49	110	5	2·9	1·71	25–49	142	7	3·1	2·28‡
⩾50	107	4	2·6	1·56					
					50–99	201	8	3·9	2·04‡
					⩾100	242	10	4·2	2·37‡
Combined	1414	42	32·4	1·30		1005	31	17·7	1·75§

*See footnote table 8.
†Employees with any employment in the pipe production area.
‡O/E significantly higher than 1·0, $p \leqslant 0·05$; see footnote, table 5.
§O/E significantly higher than 1·0, $p \leqslant 0·01$; see footnote, table 5.

Table 12 *Cases of mesothelioma among all identified workers, sorted by lag time**

Case	Type	Plant	Initial employment		Duration of employment (years)	Lag time (years)
			Year	Age		
1	Pleural	1	1955	40	0·8	14·6
2	Pleural	2	1941	31	13·5	18·8
3	Pleural	2	1946	26	23·4	28·8
4	Pleural	1	1948	24	0·3	30·1
5	Pleural	2	1947	21	22·3	32·6
6	Pleural	2	1948	19	0·4	32·8
7	Peritoneal	2	1947	19	22·8	35·9
8	Pleural	2	1946	22	23·5	36·2
9	Pleural	2	1937	20	32·8	47·0
10	Pleural	2	1927	16	42·3	54·0

*Years from initial employment in plant to death.

This difference is consistent with the data in table 2 indicating that, overall, the original exposure estimates for plant 2 were about two to three times higher than the revised estimates.

MESOTHELIOMA

Six mesotheliomas (four occurring 20 years after initial employment) are known to have occurred by the end of 1981 among the primary cohort—that is, workers initially employed in plant 1 during 1942–69 or plant 2 during 1937–69. Four occurred among plant 2 workers, two in plant 1. In addition to these six cases four others have occurred, all among plant 2 workers: one among those initially employed before 1937 (a possible survivor population) and three within the primary cohort but occurring after 1981. Since trace after 1981 is not complete, it is possible that cases arising since 1981 have been missed.

The ten workers with mesothelioma are described in table 12. The two plant 1 employees had been employed for only short periods: three months and ten months. The ten month worker began employment at age 40 (his death certificate listed his usual occupation as longshoreman), so he may have been exposed to asbestos before beginning work in the plant. Duration of employment for the eight cases at plant 2 ranged from five months to 42 years.

As seen in table 12, there was an inverse relation between lag time and age at initial employment: those exposed at the earliest ages had the longest lag times between initial employment and death.

Of the eight cases among plant 2 workers, seven (88%) were employed in the pipe production area, compared with 37% overall. The remaining case had been employed continuously from 1927 until 1970; his job record listed assignment only to the shingle production area, indicating exposure only to chrysotile. Six of the seven cases from the pipe area had been employed more than ten years (five more than 20 years). The seventh case had worked only five months in the plant but was subsequently employed for more than 30 years in a heating and air conditioning company, thus raising the possibility of later exposure to asbestos.

Since the pipe area workers were employed, on average, about four times as long as other workers, the possibility was considered that this apparently greater risk to pipe workers was due to longer exposure to asbestos rather than exposure to crocidolite. To assess the effects of duration of employment and fibre type on the risk of mesothelioma, a case-control analysis of the eight plant 2 cases of mesothelioma was carried out. Assuming no differential in the identification of existing cases by fibre type, this analysis should yield a valid assessment of fibre type effect. For seven of the eight cases, five controls were randomly selected from among all plant 2 employees who: (1) were born within one year of the case, (2) started work at the plant within one year of the case, or (3) were alive one year before the death of the case.

There were no matches satisfying the above criteria for the case who was hired in 1927. Four matches, however, were obtained who had been born within six years of this case and hired within three years.

The distribution of the eight cases of mesothelioma and the 39 controls by categories of duration of employment and proportion of time assigned to the pipe area appears in table 13. The odds ratios show an increasing trend with each of these variables. Using the actual values of each variable for each person, a matched analysis[8] was used to test for the statistical significance of the observed relation between these factors and the risk of mesothelioma. After accounting for the statistically significant factor of employment duration ($p < 0{\cdot}004$), proportion of time in the pipe area was also statistically significant ($p < 0{\cdot}008$), as was the fact of pipe employment (yes/no; $p < 0{\cdot}04$).

Table 13 also presents the proportion of subjects in each cell who have mesothelioma. Within each employment duration category if there were no relation between risk of mesothelioma and pipe area employment, then the proportions of cases within a

Table 13 *Distribution of plant 2 mesothelioma cases and controls by categories of employment duration and proportion of time in pipe area*

Proportion of time in pipe area	Duration of employment (years)												Odds ratio‡
	⩽1		1–15		15–25		>25		All durations				
	N1*	N2*	N1	N2	N1	N2	N1	N2	N1		N2		
0	0 (0)†	14	0 (0)	7	0 —	0	1 (0·20)	4	1 (0·04)		25		1·0
0·01–0·75	0 —	0	0 (0)	2	0 (0)	2	1 (0·33)	2	1 (0·14)		6		4·2
>0·75	1 (0·20)	4	1 (0·33)	2	2 (0·50)	2	2 (1·00)	0	6 (0·43)		8		18·8
Combined	1 (0·05)	18	1 (0·08)	11	2 (0·33)	4	4 (0·40)	6	8 (0·17)		39		
Odds ratio§	1·0		1·6		9·0		12·0						

*N1 = Number of cases, N2 = number of controls.
†Proportion of cell members which are cases of mesothelioma.
‡Relative to those with no time in pipe area.
§Relative to those employed ⩽1 year.

cell should be about the same for the three pipe categories. For example, among the ten subjects employed over 20 years, 40% were cases. Rather than observing cases constituting approximately 40% of each pipe employment group, however, there is a trend with time spent in the pipe area: 20%, 33%, and 100%.

Discussion

Workers in these two asbestos cement manufacturing plants were similar with respect to the important risk factors of employment duration, estimated exposure concentration, and years of hire. Known differences between the two plant groups included: the types of asbestos used (though both used primarily chrysotile); the average age at hire (plant 1 workers were, on average, five years older than plant 2 workers); size and racial composition of the work force (plant 1 was smaller, with 63% black, compared with 49% black in plant 2); physical layout (plant 1 consisted of one large building; plant 2 had four separate buildings); and location of the plants (plant 1 in the city, plant 2 outside).

The mortality experience of these two groups (20 or more years after initial exposure) was similar for all causes combined, all malignancies combined, pneumoconiosis, and non-malignant causes. For both plants, mortality due to all causes, non-malignant causes, and malignancies other than lung and mesothelioma were close to that expected based on Louisiana mortality rates. Both groups of workers had an excess of lung cancers in those in the shortest employment categories. The lung cancer experience for longer term employees, however, differed considerably in the two plant populations: plant 1 workers employed for more than one year had no excess lung cancer (16 observed, 17·2 expected) nor excess residual cancers (primarily unspecified sites and secondary respiratory/digestive; 11 observed, 13·5 expected); but comparable plant 2 workers had statistically significant excesses of both lung cancer (52 observed, 28·9 expected) and residual cancers (29 observed, 18·6 expected), with lung cancer and, to a lesser extent, residual cancer, showing dose-response relations.

Regarding cigarette smoking habits in these plants, a cross sectional study[12 13] of over 95% (n = 908) of workers employed in these plants in 1969 found comparable prevalences of smoking: 52% current, 25% ex, and 23% never smokers in plant 1 compared with 49% current, 26% ex, and 25% never smokers in plant 2. These rates of current smoking are only slighly less than the estimate of approximately 55% for all United States men in 1969.[14] Although information concerning the smoking habits of earlier workers in these plants is not available, the 1969 data suggest that similar smoking levels is a reasonable assumption, and that smoking differences do not explain the differing risks of lung cancer in the two plants.

Within each plant all results were similar for the two races and for separate categories of age at hire, indicating no effect of these two variables on risk. Therefore, plant differences in race and initial age cannot explain the differing experience of lung cancer of their employees.

The lack of an effect of age at initial exposure is consistent with a recent summary of the evidence from North American insulation workers and British textile workers.[15] By contrast, in a cohort of French asbestos cement workers the risk of lung cancer was found to be higher among workers hired before age 25 than among those hired later.[16]

The different fibre types used in the two plants must be considered as a possible explanation of the

differing risk of lung cancer among their employees. In addition to chrysotile as the primary fibre type used in both plants a small amount of amosite was used in plant 1 and a small amount of crocidolite was occasionally used from the early 1960s. In plant 2 amosite was never used but crocidolite has been used continuously in the pipe production building since 1946. Best estimates indicate that more amphibole was used in the plant 2 pipe area than in plant 1. Doll and Peto, making across study comparisons of 22 cohort studies, presented evidence of a difference in lung cancer risk by fibre type, with risk of lung cancer increasing with the relative amount of amphibole fibre used.[15] The risk of lung cancer in the two plants in this study is consistent with this reported trend.

In plant 2, however, the dose response relations between risk of lung cancer and cumulative asbestos exposure were similar for workers exposed only to chrysotile and those exposed to both chrysotile and crocidolite. The accuracy of job records in reflecting actual work area and exposure to fibre is critical to this analysis but cannot be assessed. The detailed nature of many of the job records from this plant and the fact that here, as elsewhere, pay rates varied with job assignments suggest reasonable accuracy. The fact that pipe area workers were employed, on average, considerably longer than other workers hired during the same period raises the possibility that these two groups of workers differed in ways other than type of fibre exposure. Possibilities include personal factors associated with job stability or undetected differences in working conditions. Such differences are impossible to identify but could confound any existing differences in fibre type effects.

The observation in this study of similar dose response relations by fibre type exposure is consistent with the finding of comparable relations in two United States asbestos textile manufacturing plants, one using only chrysotile, the other using chrysotile, crocidolite, and amosite.[17-19] We conclude that although the across study comparisons of Doll and Peto[15] suggest greater risk of lung cancer from exposure to amphibole compared with chrysotile alone, firm conclusions on this issue cannot yet be drawn.

The observation in this study of an excess risk of lung cancer among the shortest duration workers (approximately 40% excess in each plant) has also been made in several other studies of asbestos workers,[20-24] as well as in studies in other industries.[25] Possibly personal lifestyles cause this group to be at higher risk than longer term employees. As Doll has pointed out, "... short term employees are a peculiar group, whose experience is particularly hard to evaluate."[25]

In an earlier follow up of this population (up to 1974) the short term workers in neither plant experienced an excess risk of lung cancer.[2,3] Reanalysis (with 96% trace) confirmed this earlier observation, finding that the excess among these workers occurred after 1974.

Although no significant excess risk of lung cancer was detected among plant 1 workers with more than three months employment, two cases of pleural mesothelioma occurred in plant 1. Both were employed before crocidolite was used in this plant, so exposure was only to chrysotile and amosite. Both were employed for less than a year, although one began work in the plant at age 40 and therefore could have had previous exposure to asbestos.

Among plant 2 workers, one pleural mesothelioma occurred among the 63% of the workers whose job histories indicated exposure only to chrysotile, whereas seven (six pleural, one peritoneal) occurred among the 37% exposed to a mixture of chrysotile and crocidolite. Three of the mesotheliomas occurred after the date of full follow up, so the possibility remains that other cases have occurred since 1981 which have not been detected. Further trace is continuing.

Workers ever assigned to the pipe department of plant 2 (where both chrysotile and crocidolite were used) were employed on average four times as long as other workers. A case-control analysis, however, found that assignment to the pipe area (yes/no) and proportion of employment time spent in this area were significantly related to the risk of mesothelioma after adjusting for duration of employment (also significantly related to risk).

The evidence from plant 2 of a greater risk of mesothelioma from a mixture of chrysotile and crocidolite fibres than from chrysotile alone is consistent with much of the epidemiological evidence to date. A case-control study of ten pleural mesotheliomas in a friction product manufacturing plant found more frequent assignment to the crocidolite area among cases than controls.[26] A study of asbestos cement workers in Wales found two cases of mesothelioma, both occurring among the 15% of the cohort employed during the years when crocidolite was used.[27] Across study comparisons of cases of mesothelioma as a percentage of observed excess lung cancer have found the lowest percentage for populations exposed to chrysotile, the highest for exposures to crocidolite, and intermediate values for exposure to mixture of chrysotile with amphiboles.[28] Tissue analysis studies have supported this trend, finding greater numbers of crocidolite fibres but similar numbers of chrysotile fibres in the lungs of patients with mesothelioma compared with non-cases.[29,30]

The accumulating evidence of greater risk of mesothelioma associated with crocidolite than with

chrysotile exposure has been recognised by most industrialised countries by differences in allowable exposure levels for these fibre types, and in some instances, the banning of amphibole use. The existing evidence is so convincing that continued failure to differentiate between fibre types by governmental regulatory agencies such as the United States Occupational Safety and Health Administration is difficult to justify.

In this report, after excluding the short term workers, comparison of the results for the two plants found greater excess risk of lung cancer among plant 2 workers and a steeper dose response relation with cumulative asbestos exposure. If trace is continued only up to 1974,[2 3] however, the dose response relation for lung cancer risk (with both employment duration and cumulative asbestos exposure) are not significantly different between the two plants. In the earlier reports the lung cancer dose response relations among plant 1 workers resulted entirely from an excess in the small group of workers employed the longest: among those employed for over 15 years, five lung cancers were observed compared with 2·5 expected (Louisiana rates) by the end of 1974, whereas no excess occurred among any other duration group. By the end of 1981, however, six cases had occurred, compared with 5·7 expected. We conclude that the earlier observation of an excess within the longest employed plant 1 workers was probably a result of small numbers; with further follow up no significant excess risk of lung cancer was observed.

There is considerable variability concerning risk of lung cancer in eight groups of workers employed in the asbestos cement industry; see table 14 (a ninth study[34] has been reported only in preliminary form, with little information concerning the exposure of the cohort). Among these eight groups (excluding short term workers from this study), three exhibited little or no excess risk of lung cancer whereas relative risks in the other five ranged from 1·46 to 4·90.

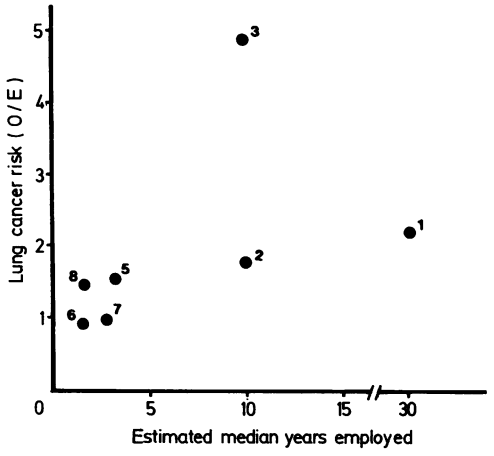
The highest risk of lung cancer was observed in the Canadian study,[32] which also found high mesothelioma risk: 11 cases in a cohort of 535 workers compared with a total of 20 cases among more than 13 000 workers in the other cohorts combined. The high risk of cancer in this population showed no sensible relation with estimated cumulative asbestos exposure in either this cohort or in a subgroup of longer term employees.[35] Exposure estimates were based on membrane filter sampling started in 1969; backward extrapolation was used to estimate earlier exposure levels. Because of the lack of an exposure response relation in this study, it has been concluded that its results "cannot be interpreted in terms of exposure response" and that no "effort at quantification would be justified."[36]

Little information was provided concerning duration of employment in the Canadian reports, but based on the available information a median duration of ten years will be assumed for the full cohort.[32] Plotting lung cancer risk by estimated median employment duration for the seven asbestos cement cohorts for which the median could be estimated

Table 14 *Lung cancer and mesothelioma in eight cohorts of asbestos cement workers*

Study (country)	*Fibre type (years of employment)*	*Cohort No (% traced)*	*Estimated median years employed (minimum)*	*Estimated concentration (f/ml)*	*No of deaths (minimum latency)**	*Lung cancer (O/E)†*	*No† of mesotheliomas*
Alies-Patien, *et al*[16] (France)	Ch, Cr (1940–)	941 (100)	25 (5)	NA	107 (20)	12/5·5 (2·17)	4
Clemmesen and Hjalgrim-Jensen[31] (Denmark)	Ch, Cr, A (1944–76)	5686 (NA)	10‡ (None)	NA	NA (None)	47/27·3 (1·72)	3
Finkelstein[32] (Canada)	Ch, Cr (1948–59)	535 (84)	10§ (1)	9‡	108 (20)	26/5·3 (4·90)	11
Lacquet and van der Linden[33] (Belgium)	Ch, Cr, A (1963–77)	NA (NA)	NA (1)	NA	201 (None)	22/22·4 (0·98)	1
Ohlson and Hoqstedt[20] (Sweden)	Ch, Cr, A (1943–76)	1176 (99)	3 (0·25)	1–2	130§ (20)	9/5·7 (1·58)	0
Thomas *et al*[27] (Wales)	Ch (1937–77)	1291 (97)	1·5‡ (0·5)	NA	268 (None)	23/23·8 (0·97)	0
	Ch, Cr (1935–6)	249 (97)	1·5§ (0·5)	NA	83 (None)	7/9·2 (0·76)	2
This study (US)							
Plant 1	Ch, Cr, A (1942–69)	996 (97)	2·6 (>0·5)	12·1§	259 (20)	22/22·5 (0·98)	2
Plant 2	Ch, Cr (1937–69)	2419 (96)	1·4 (>0·25)	10·4§	603 (20)	73/50·1 (1·46)	8

*Number of deaths after minimum latency (minimum elapsed time from initial employment).
†O/E = Observed number of lung cancers/expected number (after minimum latency); mesotheliomas are those reported regardless of latency time.
‡Not reported; estimated from other information provided.
§Using a conversion of 1 mppcf = 1·4 f/ml.
Ch = Chrysotile; Cr = Crocidolite; A = Amosite; NA = Not available.



Observed lung cancer risk in seven cohorts of asbestos cement workers, by estimated median duration of employment (mean duration could not be estimated in some cases). Numbers in plot identify study, as listed in Table 14.

shows that results are reasonably consistent for six of these seven cohorts (though unexplained variability remains) but that the Canadian study result is an outlier (figure).

The disparity of the results for the Canadian plant and plant 2 of this study are particularly perplexing since both were in operation at about the same time and both were owned by the same company. Smoking information was not available for either cohort, but more extensive smoking among the Canadian workers could not be expected to account for much of this difference.

The availability of air sampling data from plant 2 beginning in 1952 and the observation of the expected generally increasing trend of lung cancer relative risk with estimated cumulative asbestos exposure support the validity of this study's exposure estimates, at least on an ordinal scale.

Although exposure estimates were revised for use in this report, primarily because more measurements had become available, these could be underestimates of exposure conditions in plant 2, especially during the 1940s and 1950s, when much of the cohort was employed. The revised estimates do not make use of extensive anecdotal information used in our previous report to augment air sampling data. Much of the anecdotal information suggested that exposure levels were frequently higher than the data from area monitoring indicates and that average concentration levels in plant 2 during the 1940s were probably higher than the estimated mean of about 12 f/ml. Whether air sampling data alone or such data augmented by anecdotal information more accurately reflect actual exposure conditions is an important issue in deriving exposure estimates for any study but, at least in this study, is impossible to assess. For plant 1, anecdotal information had little effect on the exposure estimates, but for plant 2 this information increased the estimates substantially. If the information for plant 2 was valid then use of air sampling data alone would have overestimated the slope of the dose response relation by a factor of two. The variability of estimated risk resulting from this degree of uncertainty in exposure estimates, however, is not likely to influence policy decisions regarding asbestos health effects.

The estimated slope of 0·0076 based on plant 2 is consistent with the observation[37 38] of apparently intermediate slopes for asbestos construction product manufacturing when compared with textile manufacturing (steep slopes) and mining or friction product manufacturing (shallow). On the other hand, the finding of no overall excess of lung cancer and a very shallow slope in plant 1 is more consistent with observations in friction product workers.[21 39]

We conclude that the lung cancer dose response relation observed in plant 2 provides a reasonable (though possibly high) estimate of lung cancer risk in the asbestos cement industry. This relation (RR = 1 + 0·0076 x, for x in f/ml-yrs) would predict a relative risk of 1·038 for workers exposed to 0·2 f/ml for 25 work years, or about two lifetime lung cancers per 1000 workers based on United States male lung cancer rates. If the recent decline in smoking among United States men continues the background risk of lung cancer will decrease (as has already been observed among young men), resulting in corresponding lower estimates of excess risk due to exposure to asbestos.

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