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Exposures and Mortality Among Chrysotile Asbestos Workers. Part II: Mortality

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A retrospective cohort mortality study was conducted among a cohort of 1,261 white males employed one or more months in chrysotile asbestos textile operations and followed between 1940 and 1975. Statistically significant excess mortality was observed for all causes combined (standardized mortality ratio [SMR] = 150), lung cancer (SMR = 135), diseases of the circulatory system (SMR = 125), nonmalignant respiratory diseases (SMR = 294), and accidents (SMR = 134). Using estimated fiber exposure levels in conjunction with detailed worker job histories, exposure-response relationships were investigated. Strong exposure-response relationships for lung cancer and asbestos related nonmalignant respiratory diseases were observed. Compared with data for chrysotile miners and millers, chrysotile textile workers were found to experience significantly greater lung cancer mortality at lower lifetime cumulative exposure levels. Factors such as differences in airborne fiber characteristics may partially account for the large differences in exposure response between textile workers and miners and millers.

Key words: asbestos, chrysotile, lung cancer, asbestosis, exposure-response

INTRODUCTION

The companion paper in this volume described the facility and presented methods used to reconstruct exposure levels for an asbestos textile operation using chrysotile. Using linear statistical models to account for textile processes and controls, worker exposures between 1930 and 1975 were estimated. These data were combined with an assessment of mortality to study exposure-response relationships for lung cancer and nonmalignant respiratory diseases. Each employee's detailed employment history provided the necessary link with the exposure estimates to allow analyses of exposure-response. This manuscript presents the overall mortality assessment and the observed exposure-response relationships.

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MATERIALS AND METHODS

Although the plant under study began production of asbestos products in 1896, detailed personnel records were first maintained beginning in approximately 1930. The record system has remained remarkably unchanged since that time. For each worker, an employment card was completed at initial employment giving name, date of birth, sex, race, social security number, marital status, and address. This same card also contained the detailed work history giving exact dates of employment by plant department and specific jobs. All information from these cards was entered onto a computer data file.

The cohort was limited to 1,261 white males employed one or more months in textile production operations with at least one month of plant employment between January 1, 1940, and December 31, 1965. The cohort was followed through December 31, 1975. The 1965 cut-off date for cohort entry was chosen to insure that all workers would have a minimum "latency" of ten years as of the study end date. The one-month entry criteria was used to allow comparisons with other mortality studies of chrysotile workers [McDonald et al, 1980].

An attempt was made to determine the vital status of all cohort members as of December 31, 1975. The primary sources of information used for this follow-up included the Social Security Administration (SSA), Internal Revenue Service (IRS), US Postal Mail Correction Service, state drivers license files, and state vital statistics offices. Individuals not located through these primary sources were traced using local records such as telephone listings, Polk directories, property records, voter records, records of funeral homes, and various other local sources.

Cause-specific standardized mortality ratios (SMRs) were calculated using a life-table analysis based on the technique developed by Cutler and Ederer [1958]. Person-years at risk of dying were distributed by five-year age, calendar time, and time since initial employment (latency) groups. Person-years were accumulated for each cohort member beginning when all requirements for cohort entry were met until the date of death or December 31, 1975. Those whose vital status remained unknown were assumed alive as of the study cut-off date, thereby contributing their maximum possible person-years to the analysis.

The follow-up period for this study spans the fifth through eighth revisions of the International Lists of Diseases and Causes of Death (ICDA). Death certificates were coded by a qualified nosologist according to the ICDA revision in effect at the time of death. All death codes were then grouped into 89 death categories based on the seventh revision for purposes of standardization. Individuals known to be deceased but for whom no death certificates were available were assumed to be deceased, cause unknown.

The number of expected deaths, standardized for sex, age, race, and calendar time, were calculated by application of cause-specific death rates for the total United States to the person-years at risk of dying. Death rates specific to the 89 seventh-revision death groups were calculated from yearly tallies of deaths and census data.

For evaluating exposure-response, cumulative exposures were calculated for each worker using detailed work histories contained in plant personnel records combined with the estimated level of exposure for each job held. A worker's cumulative dust exposure at any time during the follow-up period was expressed as the cumulative product of the estimated average dust concentrations for particular jobs held by the worker and the time duration in those jobs.

The estimated fiber concentrations for the period 1930–1975 are presented in the companion manuscript in this volume and are expressed as fibers longer than 5 μm in length per cubic centimeter of air (fibers/cc). Time spent in each job was calculated as the difference between dates of job changes and expressed in days; therefore, the cumulative exposure was expressed as fibers/cc $\times$ days. Using this method, weekends and holidays are not eliminated; thus the true number of "work days" in each job was overestimated. This was done to provide conservative estimates of exposure and to account for periods of work longer than eight hours. A few workers began employment before 1930. Pre-1930 exposure levels for these workers were estimated by assigning exposure levels prior to implementation of control measures for each job held before 1930.

Mortality in relation to exposure was investigated by creating cumulative exposure strata through which a worker was moved as his cumulative exposure increased during the follow-up period [Breslow, 1976; Lundin et al, 1971]. This method allowed full use of each cohort member's survival experience for the entire follow-up period. Cause of specific SMRs were calculated for each exposure stratum using appropriate age, race, sex, and calendar time specific death rates.

Statistical significance of observed excess or deficit mortality was evaluated using the Poisson distribution [Pearson and Hartley, 1958].

RESULTS

Overall Mortality

Results of the follow-up efforts are summarized in Table I. Vital status was determined for all but 26 (2.1%) of the 1,261 cohort members. Of the 308 deaths, all but 17 death certificates were obtained.

A total of 33,141 person-years at risk were experienced by this cohort between January 1, 1940, and December 31, 1975. Observed and expected deaths by cause are given in Table II.

A total fo 308 deaths were observed, whereas only 205.66 were expected (SMR = 150, $p < 0.05$). Observed and expected deaths by time interval since initial employment are shown in Figure 1. No statistically significant excess mortality was observed until after 15 or more years since initial employment, a finding consistent with other occupational mortality studies.

Examination of cause-specific mortality in Table II shows significant excess mortality for malignant neoplasms (SMR = 168, $p < 0.05$), diseases of the circulatory system (SMR = 125, $p < 0.05$), nonmalignant respiratory diseases (SMR = 294, $p < 0.05$), and accidents (SMR = 134, $p < 0.05$). Increased mortality was also observed for

TABLE I. Vital Status for White Males With One or More Months Textile Employment

Vital status as of Dec 31, 1975	No.	Percent
Known alive	927	73.5
Known deceased	308	24.4
Certificate obtained	(291)	(94.5)
Certificate not obtained	(17)	(5.5)
Unknown vital status	26	2.1
Total	1261	100

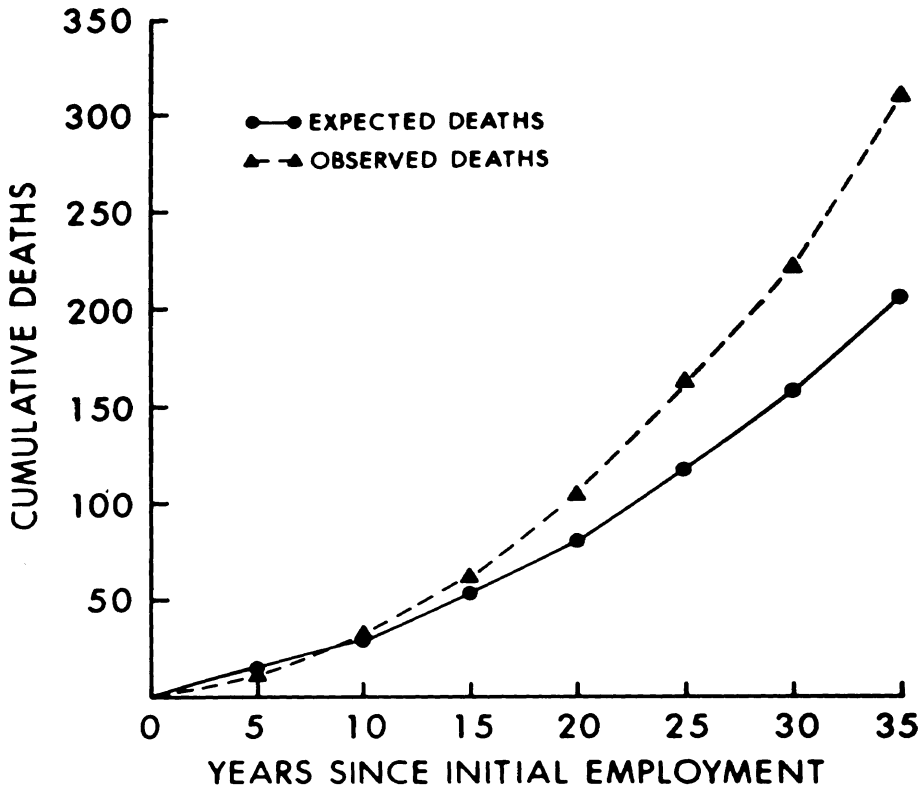

Fig. 1. Observed and expected deaths for all causes by time interval since initial employment.

TABLE II. Observed and Expected Deaths by Cause for White Male Asbestos Textile Workers 1940–1975

Cause of death	ICDA 7th list no.	Observed	Expected	SMR
All causes		308	205.66	150[a]
Malignant neoplasms		59	35.06	168[a]
Digestive system	150–159	13	9.89	131
Trachea, bronchus, lung	162–163	35	11.10	315[a]
All other sites		11	14.07	78
Vascular lesions affecting the central nervous system	330–334 345	15	10.97	137
Diseases of the circulatory system	400–468	105	83.74	125[a]
All tuberculosis	001–019	6	3.48	172
Nonmalignant respiratory diseases		28	9.53	294[a]
Acute upper respiratory infection	470–475	0	0.03	—
Influenza	480–483	0	0.04	—
Pneumonia	490–493	4	4.19	95
Bronchitis	500–502	0	0.55	—
Other respiratory disease	510–527	24	4.35	552[a]
Accidents	800–962	34	25.38	134[a]
Other violent deaths	963–964 970–985	9	9.37	96
All other known causes		29	26.91	108
Unknown causes including 17 missing death certificates		23	2.55	—

[a]$p < 0.05$.

diseases of the central nervous system and tuberculosis; however, these excesses were not statistically significant.

The elevated SMR for malignant neoplasms shown in Table II is largely accounted for by cancer of the trachea, bronchus, and lung, and cancers of the digestive system. A total of 35 lung cancers were observed and only 11.10 were expected (SMR = 315, $p < 0.05$). Tables III and IV show lung cancer mortality by time interval since first employment (latency) and duration of employment. No lung cancers were observed prior to 10 years latency and 17 of the 35 lung cancers occurred after 30 or more years latency. Table IV demonstrates an increasing trend in the lung cancer SMR with increased employment with an SMR of 976 for those employed 20–29 years.

Of the 28 deaths attributed to nonmalignant respiratory diseases, 24 fell into the category "other respiratory diseases" (ONMRD) which includes asbestosis. Of the 24 deaths in this category, asbestosis or pulmonary fibrosis was the underlying cause for 17. ONMRD mortality by latency and duration of employment is given in Tables V and VI. The SMR for ONMRD was not statistically elevated until greater than ten years employment but increased dramatically for those employed more than ten years.

Increased mortality due to cardiovascular diseases is a consistent observation among asbestos workers and represents a combined stress on the cardio-pulmonary system. A review of death certificates for the 105 deaths found that six mentioned asbestosis or pulmonary fibrosis as a contributory condition.

TABLE III. Lung Cancer (ICDA 162,163) Mortality by Time Interval Since Initial Employment

Years since initial employment	Observed	Expected	SMR
< 10	0	0.47	—
10–19	6	2.08	288[a]
20–29	12	4.76	252[a]
≥ 30	17	3.79	449[a]
Total	35	11.10	315[a]

[a]$p < 0.05$.

TABLE IV. Lung Cancer (ICDA 162,163) Mortality by Duration of Employment

Years employed	Observed	Expected	SMR
< 10	15	8.09	185[a]
10–19	5	1.05	476[a]
20–29	12	1.23	976[a]
≥ 30	3	0.73	410
Total	35	11.10	315[a]

[a]$p < 0.05$.

TABLE V. Mortality Due to "Other Nonmalignant Respiratory Diseases" (ICDA 510–527) by Time Interval Since Initial Employment

Years since initial employment	Observed	Expected	SMR
< 10	1	0.26	—
10–19	4	0.74	541[a]
20–29	10	1.77	565[a]
≥ 30	9	1.58	570[a]
Total	24	4.35	552[a]

[a]$p < 0.05$.

TABLE VI. Mortality Due to "Other Nonmalignant Respiratory Diseases" (ICDA 510-527) by Duration of Employment

Years employed	Observed	Expected	SMR
< 10	6	3.13	192
10–19	5	0.39	1282[a]
20–29	9	0.51	1765[a]
≥ 30	4	0.32	1250[a]
Total	24	4.35	552[a]

[a]$p < 0.05$.

Only one mesothelioma was observed among this cohort. This was a peritoneal mesothelioma confirmed by autopsy. The interval (latency) between initial employment and death was 34 years. There were several other deaths which mentioned "cancer of the abdomen" that may be suspect; however, no autopsy or other confirmatory data were available.

Exposure-Response Relationships

Both lung cancer and asbestosis require lengthy periods from initial exposure to become clinically evident. For exposure-response studies based on mortality it is important to restrict the analyses to those achieving sufficient latency to be "at risk" of dying from lung cancer or asbestosis. For this reason, exposure-response analyses were restricted to those achieving 15 or more years since initial employment (latency). This was accomplished by beginning accumulation of person-years for each worker after the 15-year latency period was satisfied; however, cumulative exposures began at employment. Those dying before reaching 15 years latency were excluded.

Results of the exposure-response analyses for all causes, diseases of the circulatory system, lung cancer, and digestive system cancer are given in Tables VII and VIII. Significant excess overall mortality was observed for all cumulative exposure strata except the highest, where observed numbers were small. Although increased numbers of circulatory system deaths were observed for each exposure stratum, statistically significant excesses were observed in only one. There appeared to be no consistent increasing trend of circulatory system mortality with exposure.

Table VIII demonstrates a strong exposure-response relationship for lung cancer. Statistically significant excess lung cancer mortality was observed for all but the lowest exposure stratum where five lung cancers were observed versus 3.58 expected. Lung cancer SMRs increased with increasing cumulative exposure with an SMR of 1,818 in the highest exposure stratum. A plot of lung cancer SMRs by cumulative exposure is given in Figure 2. A linear function appears to adequately describe these data. Although digestive system cancers were not excess overall, an increasing trend in the SMR was observed with exposure; however, none of the exposure strata demonstrated a statistically significant excess.

Exposure-response relationships for ONMRD are given in Table IX. Significant excess mortality was observed in all except one exposure stratum with the SMR increasing consistently with increased exposure. Shown in parentheses are those deaths with an

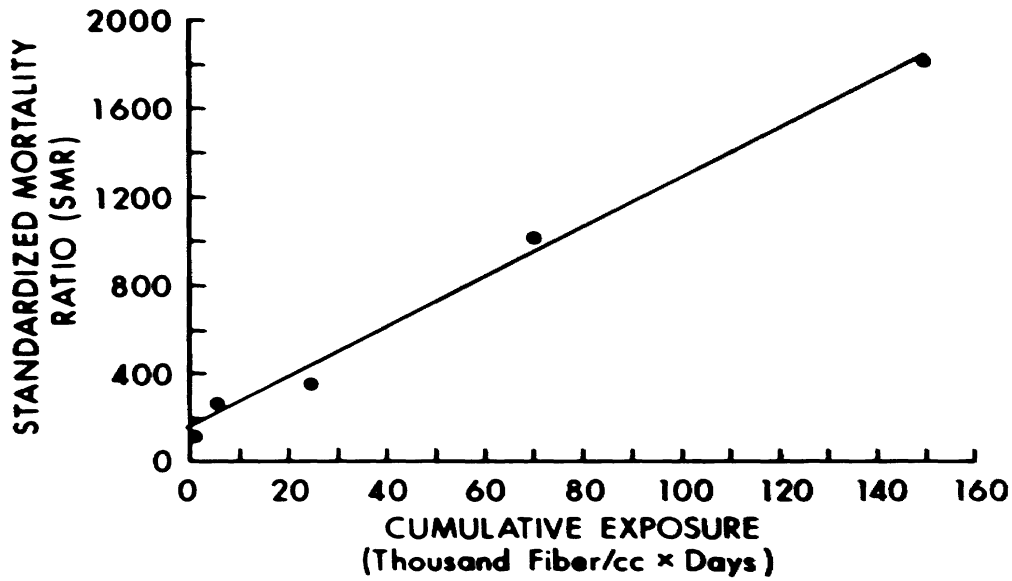

Fig. 2. Exposure-response for lung cancer among white males achieving 15 or more years latency.

TABLE VII. Exposure-Response Relationship for All Causes and Diseases of the Circulatory System Among White Males Achieving 15 or More Years Latency

Cumulative exposure fiber/cc × days	All causes			Diseases of the circulatory system (ICDA 400–468)		
	Observed	Expected	SMR	Observed	Expected	SMR
< 1,000	79	55.01	144	34	24.40	139
1,000–10,000	67	48.62	138[a]	24	21.52	112
10,000–40,000	60	32.77	183[a]	24	15.19	158[a]
40,000–100,000	33	13.59	243[a]	8	6.50	123
> 100,000	6	2.50	240	2	1.27	157
Total	245	152.49	161[a]	92	68.88	134[a]

[a]$p < 0.05$.

TABLE VIII. Exposure-Response Relationships for Lung Cancer and Digestive System Cancer Among White Males Achieving 15 or More Years Latency

Cumulative exposure fiber/cc × days	Lung cancer (ICDA 162,163)			Digestive system (ICDA 150–159)		
	Observed	Expected	SMR	Observed	Expected	SMR
< 1,000	5	3.58	140	2	2.85	70
1,000–10,000	9	3.23	279[a]	1	2.54	–
10,000–40,000	7	1.99	352[a]	4	1.78	225
40,000–100,000	10	0.91	1099[a]	3	0.77	390
> 100,000	2	0.11	1818[a]	0	0.14	–
Total	33	9.82	336[a]	10	8.08	124

[a]$p < 0.05$.

underlying cause attributed to asbestosis or pulmonary fibrosis. Of the five deaths in the lowest exposure stratum, only two were attributed to asbestosis or pulmonary fibrosis.

Exposure-response relationships for asbestosis and pulmonary fibrosis were investigated separately by calculating incidence density (cases per 1,000 person-years) for each exposure stratum. These data are given in Table X and shown graphically in Figure 3. A strong increasing trend in incidence density with exposure was observed with the incidence in the highest exposure stratum approximately 50 times that of the lowest. The exposure-response pattern shown in Figure 3 suggests that the incidence of asbestosis and pulmonary fibrosis increases more rapidly with exposure than predicted by a linear function. This is only a tentative observation since only two cases were observed in the highest category with a resulting wide confidence interval for the incidence density.

DISCUSSION

Statistically significant excess mortality was observed for lung cancer and nonmalignant respiratory diseases among chrysotile asbestos textile workers. Furthermore, using cumulative exposure as the exposure variable and the SMR as the measure of disease risk, strong exposure-response relationships were observed for lung cancer. A linear, no-threshold model appears to adequately describe the form of the exposure-response function for lung cancer. Convincing exposure-response relationships were also

TABLE IX. Exposure-Response Relationships for Other Nonmalignant Respiratory Diseases (ONMRD) Among White Males Achieving 15 or More Years Latency

	ONMRD deaths (ICDA 510–527)		
Cumulative exposure (fiber cc × days)	Observed[a]	Expected	SMR
< 1,000	5 (2)	1.38	362[b]
1,000–10,000	1 (1)	1.19	—
10,000–40,000	7 (6)	0.78	897[b]
40,000–100,000	7 (6)	0.38	1842[b]
> 100,000	2 (2)	0.08	2500[b]
Total	22 (17)	3.86	570[b]

[a]Figures in parentheses indicate number of observed deaths attributed to asbestosis or pulmonary fibrosis.
[b]$p < 0.05$.

TABLE X. Incidence Density for Asbestosis or Pulmonary Fibrosis as Underlying Cause of Death Among White Males Achieving 15 or More Years Latency

Cumulative exposure fiber cc × days	Cases	Person-years at risk	Cases per 1,000 person-years	95% confidence interval[a]
< 1,000	2	6,254	0.32	0.04– 1.16
1,000–10,000	1	5,661	0.18	0.03– 5.57
10,000–40,000	6	3,027	1.98	0.73– 4.32
40,000–100,000	6	1,002	5.99	2.20–13.06
> 100,000	2	126	15.87	1.92–57.29
Total	17	16,070	1.06	0.62– 1.70

[a]Based on Poisson distribution.

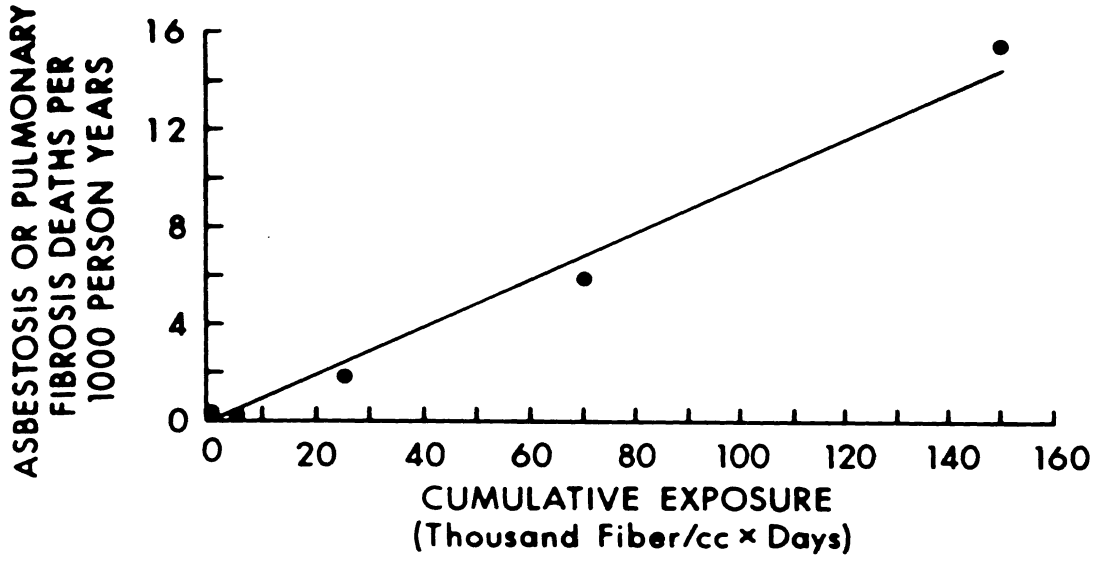

Fig. 3. Incidence density for asbestosis and pulmonary fibrosis mortality among white males achieving 15 or more years latency.

TABLE XI. Comparison of Lung Cancer Mortality Rates (ICDA 162,163) Between County in Which Study Plant Was Located and Contiguous Counties, 1950–1969

	Age-adjusted deaths/100,000 (white males)
United States	37.98
State in which plant was located	37.83
County in which plant was located	66.5
Contiguous counties (range)	40.1–53.4
Counties one removed (range)	25.9–44.1

observed for nonmalignant respiratory disease mortality including asbestosis and pulmonary fibrosis.

There are several factors which need to be considered in evaluating the occupational contribution to observed mortality patterns. The most important of these are the choice of standard population death rates to estimate expected deaths and cigarette smoking patterns among the cohort. Other potential confounders such as age, race, sex, and calendar time period were dealt with in the study design.

The standard population death rates chosen for this study were those for white males for the entire United States. Table XI gives a comparison of lung cancer (ICDA 162,163) age-adjusted mortality rates for 1950–1969 for counties in the same area as the study plant with state and US rates [Mason et al, 1975]. Lung cancer death rates for the state in which the plant was located were nearly equal to US rates. On the other hand, rates for the county where the plant was located were 75% higher than US rates for white males.

The choice of an appropriate comparison population for mortality analyses is difficult, and arguments could be made for using rates for a set of counties contiguous to the county in which the plant was located. However, there are serious limitations to

this approach which were considered in this study and resulted in rejecting the use of local county rates. First, the county in which the plant was located is the site of a large shipyard industry. Employees for this industry were largely drawn from the local population. Many of these workers are thought to have been exposed to asbestos during ship construction and repair. In an ecological study, Blot et al [1978] demonstrated an association between county lung cancer rates and shipyard employment. In a more refined case-control study, Blot et al [1979] demonstrated a summary odds ratio of 1.6 for shipyard employment and lung cancer after adjusting for smoking, other occupations, age, race, and county of residence. These data suggest that lung cancer death rates in the area in which the plant was located are likely to be elevated by local shipyard employment. In addition, the effect of the plant under study on county lung cancer mortality must be considered.

The effects of shipyard and asbestos plant employment make the use of local death rates inappropriate for this study. However, even if rates for contiguous counties had been used (Table XI), the expected lung cancer rates for white males would have been increased by only approximately 15%; not nearly sufficient to account for the observed excess lung cancer risk among the study cohort. Detailed plant work histories as well as prior occupational histories (collected by the US Public Health Service in 1964 and 1971) were reviewed for lung cancer and nonmalignant respiratory disease cases. No association with either prior shipyard employment or plant employment in rubber operations was observed.

Cigarette smoking is a known risk factor for respiratory cancer, and smoking and asbestos exposures have been shown to act in a synergistic manner to greatly increase the risk of lung cancer [Hammond et al, 1979]. Respiratory-symptom questionnaires including questions on smoking history were administered by the US Public Health Service (USPHS) to active workers in this plant in 1964 and again in 1971. In addition, smoking data available from plant medical records were also collected. While smoking histories are not available on all cohort members, these data were useful in estimating the prevalence of smoking in this plant for comparison with smoking patterns among US males who were used as the standard population for estimation of expected lung cancer deaths.

The prevalence of cigarette smoking habits among the asbestos study cohort is given in Table XII. These data largely represent the smoking prevalence found by the 1964 USPHS survey since the cohort was limited to those achieving 1 month of employment before 1965. The USPHS 1971 data and company data were used only for those missed in 1964. Among white males, 52.4% were found to be current smokers, 25.3% nonsmokers, and 22.3% past smokers.

Table XII also compares smoking prevalence among the study cohort members with comparable data for US adults [USPHS, 1979]. These data show the prevalence of smoking among white males in the study cohort to be nearly identical to that of US white males. The 22.3% prevalence of past smokers is also identical to US figures. Available smoking data for this cohort suggest that the observed lung cancer and nonmalignant mortality excess among white males cannot be explained by cigarette smoking independent of asbestos exposure. This conclusion is also supported by the exposure-response data. While smoking cannot explain the observed lung cancer excess, an interactive effect with asbestos exposure is likely.

Although mortality among asbestos workers has been extensively studied, there are a few studies of populations exposed to only chrysotile. Mortality among Quebec

TABLE XII. Summary of Cigarette Smoking Patterns for White Male Asbestos Textile Workers and Comparison With Data for US White Males

	Current smoker (%)	Past smoker (%)	Nonsmoker (%)
Asbestos workers (N = 292)	52.4	22.3	25.3
US white adult males (1965)	51.5	22.1	26.4

chrysotile miners and millers has been extensively studied [McDonald et al, 1980]. The most recent report for this cohort included 10,939 men who had been employed one or more months and followed between 1926 and 1975. An overall SMR for lung cancer of 125 was observed; 42 deaths (1.3%) were due to asbestosis and 11 (0.3%) due to mesothelioma. Increased mortality was also observed for cancer of the stomach and esophagus but no other gastrointestinal sites. Similar patterns of lung cancer and asbestosis mortality have been reported for Italian chrysotile miners and millers where an SMR for lung cancer of 206 was observed among those with sufficient latency [Rubino et al, 1979].

The McDonald et al studies demonstrated a relatively modest increase in lung cancer risk even in the highest exposure group. Nicholson et al [1979] reported larger excesses for lung cancer and asbestosis in their study of chrysotile miners and millers in Quebec. This latter study cohort consisted of 544 miners and millers with at least 20 years seniority followed between 1961 and 1977. A total of 28 lung cancers were observed versus 11.1 expected (SMR = 252). There were 30 deaths due to noninfectious respiratory diseases, whereas only 6.7 were expected. Of these 30 deaths, 26 were due to asbestosis. Only one mesothelioma (pleural) was observed.

Studies of factory populations exposed to only chrysotile are rare. Weiss [1977] studied a small cohort of 264 workers in a plant producing asbestos millboard and reported no excess cancer mortality. However, there were only 66 deaths (two of which were due to asbestosis).

There are a few other published reports with which to compare the exposure-response data obtained in this study. In fact, there are no other reports of exposure-response using exposures expressed as fibers/cc by the phase-contrast method; all other reports have used impinger (MPPCF) data [Henderson and Enterline, 1979; McDonald et al, 1980]. For comparison with other published data, approximate impinger exposure values, expressed as MPPCF × years, were calculated for data from the current study using the impinger-membrane filter conversions. Estimated exposure-response for lung cancer based on these estimates are given in Table XIII along with other published data.

The data in Table XIII show the SMR for lung cancer at a given cumulative exposure for the present study to be much higher than other published values. However, there are differences in the designs of the three studies which may account for some of this apparent discrepancy. For example, the McDonald et al [1980] study included persons exposed to extremely high airborne-fiber levels, thus competing asbestosis risk may be important. The study by Henderson and Enterline [1979] consisted of retirees 65 years or older. In the present study, only eight of 35 lung cancer deaths were 65 or older. The Henderson and Enterline study may be a survivor population with less lung cancer risk for those surviving to age 65.

TABLE XIII. Comparison of Exposure-Response Relationships for Lung Cancer With Other Published Data

Present study		Henderson and Enterline [1979]		McDonald et al [1980][a]	
Approximate MPPCF × yrs	SMR	MPPCF × yrs	SMR	MPPCF × yrs	SMR
<0.9	140				
0.9- 9.1	279			30	104
9.1–36.5	352			100	114
36.5–91.3	1099				
>91.3	1818	<125	197.9		
		125–249	180.0	300	142
		250–499	327.6	500	170
		500–749	450.0		
		>750	777.8	1200	268

[a]Based on cumulative exposures until age 45 years. SMRs calculated from regression line provided by authors.

TABLE XIV. Comparison of Lung Cancer Mortality by Duration of Employment for Chrysotile-Exposed Cohorts

	Current study[a]			McDonald [1980][a]		
Duration of employment	Observed	Expected	SMR	Observed	Expected	SMR
1 mo–5 yr	11	5.32	207	76	83.39	91
5–20 yr	3	1.22	246	50	36.50	137
>20 yr	15	2.06	728	104	64.60	161

[a]Data in this table represents mortality after 20 or more years latency.

Differences in lung cancer exposure-response relationships between this study and that reported by McDonald et al [1980] are significant. Estimation of historic exposure levels is a difficult task, and it is possible that part of the apparent disparity between the two studies reflects imprecision of these estimates. However, large differences are also noted using duration of employment as a measure of exposure. Table XIV shows such a comparison for the two studies. In each duration of employment stratum, much larger lung cancer SMRs were observed in the current study. These differences were very large for those achieving more than 20 years employment. These data suggest that imprecision of exposure estimates does not account for observed differences in exposure-response. Other factors such as differences in airborne-fiber characteristics (length, diameter, etc) may be important. Compared with other asbestos-processing operations, textile processing has been shown to produce a greater airborne fraction of long (>5 μm in length), thin (<1.5 μm in diameter) fibers [Dement and Harris, 1979]. Animal studies have shown these fibers to be more capable of producing tumors upon pleural implantation than are shorter, thicker fibers [Stanton et al, 1981].

The current Occupational Safety and Health Administration asbestos exposure standard of 2.0 fibers/cc is based on an allowable lifetime cumulative exposure of 100 fibers/cc × years (ie, 2.0 fibers/cc for a 50-year working lifetime). Based on data from this study, significantly elevated mortality risks are predicted for lung cancer and for asbestosis at cumulative exposures of 100 fibers/cc × years in the textile industry. This observation is based on use of cumulative exposures as a summary exposure measure to account for both exposure level and duration. Further analyses of these data are planned to investigate the separate effects of exposure level and duration.

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