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ARTICLES

Follow-Up Study of Chrysotile Asbestos Textile Workers: Cohort Mortality and Case–Control Analyses

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Previous studies of mortality among white males employed in a Charleston, South Carolina asbestos textile plant using chrysotile demonstrated significant excess mortality due to asbestos-related disease and a steep exposure–response relationship for lung cancer. This cohort was further studied by adding 15 years of follow-up and including mortality among white female and black male workers. Nested case–control analyses were undertaken to further explore possible differences in lung cancer risk by textile operation as well as possible confounding by mineral oil exposures. Preliminary data for white males have been previously published. White males experienced statistically significant excess mortality due to lung cancer (standardized mortality ratio [SMR] = 2.30; confidence interval [CI] = 1.88–2.79), all causes (SMR = 1.48; CI = 1.38–1.58), all cancers (SMR = 1.50; CI = 1.29–1.72), diabetes mellitus (SMR = 2.05; CI = 1.18–3.33), heart disease (SMR = 1.41; CI = 1.26–1.58), cerebrovascular disease (SMR = 1.50; CI = 1.08–2.02), pneumoconiosis and other respiratory diseases (SMR = 4.10; CI = 3.10–5.31), and accidents (SMR = 1.49; CI = 1.15–1.91). Among white females, statistically significant excesses occurred for lung cancer (SMR = 2.75; CI = 2.06–3.61), all causes (SMR = 1.21; CI = 1.11–1.32), pneumoconiosis and other respiratory diseases (SMR = 2.40; CI = 1.53–3.60), and other respiratory cancers (SMR = 14.98; CI = 4.08–38.7). Among the total cohort of black males, the only statistically significant excess observed was for pneumoconiosis (SMR = 2.19; CI = 1.23–3.62). Based on historical exposure measurements at the plant, there was a positive exposure–response relationship for both lung cancer and pneumoconiosis. Data for the entire cohort demonstrate an increase in the lung cancer relative risk of 2–3% for each fiber/cc-year of cumulative chrysotile exposure. This relationship was more consistent for the white male workers. The excess risk for lung cancer among white males and females appeared to occur at cumulative exposures lower than those for black males. Possible reasons for the lesser lung cancer risk among black males include less smoking and differences in airborne fiber characteristics experienced by black males as a result of plant job placement patterns. The case–control analysis found employment in preparation and carding operations (where most of the black males worked) to be associated with a slightly reduced lung cancer risk, although not statis-

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tically significant, whereas spinning and twisting employment was associated with a statistically significant increased lung cancer risk compared to other plant operations. Airborne fiber size data, determined by transmission electron microscopy, demonstrated slightly longer fibers in spinning and twisting compared to other textile operations. Case–control analyses demonstrated little effect of mineral oil exposures on the lung cancer exposure–response estimates. Two deaths due to mesothelioma were observed among this cohort.

INTRODUCTION

In previous publications, the exposure and mortality of workers in a South Carolina textile plant processing chrysotile asbestos were reported [Dement et al., 1981, 1983a,b]. Based on a retrospective cohort mortality analysis of the white male work force, strong exposure–response relationships were observed for lung cancer and nonmalignant respiratory disease. The slope of the exposure–response relationship seen in this cohort was among the highest seen in asbestos exposed cohorts irrespective of fiber type or industry. Independent study of these same chrysotile asbestos textile workers corroborated the results obtained by Dement et al. [McDonald et al., 1983a]. Subsequent nested case–control analyses of this population were conducted to examine possible confounding effects from the use of mineral oil in the process of textile manufacturing [Dement, 1991]. These analyses concluded that mineral oil exposure was not a plausible explanation for the observed risk and confirmed the role of chrysotile exposure as the most likely cause for the increase in lung cancer and the strong exposure–response pattern observed. A similar lung cancer exposure–response pattern was observed in another textile plant studied by McDonald et al. [1983b]. In contrast, the lung cancer exposure–response pattern observed among chrysotile miners and millers was much less dramatic [McDonald et al., 1980].

In order to further refine the risk estimates associated with chrysotile exposure, the cohort has been updated and reanalyzed by adding 15 years of observation and by including the mortality experience of white female and black male workers. Preliminary results of the updated followup have been previously published [Dement and Brown, 1993; Brown et al., in press]. This manuscript presents final mortality data for white males, white females, and black males, as well as updated estimates of the lung cancer mortality exposure–response relationship using data from the entire cohort. Also examined are mortality from other causes of death that may be related to chrysotile exposure. Other causes of death of a priori interest include asbestosis and other nonmalignant respiratory diseases, as well as cancers of the pancreas, gastrointestinal tract, kidney, larynx, mesothelioma [National Research Council, 1984; EPA, 1986; WHO, 1987; OSHA, 1986; CPSC, 1983; Smith et al., 1989], and ischemic heart disease [Sanden et al., 1993].

In addition to the cohort mortality analysis, a nested case–control study was undertaken to further examine possible differences in lung cancer exposure–response by textile operation. These analyses also re-examined the possible role of mineral oil exposures using the larger number of deaths available for study.

MATERIALS AND METHODS

Cohort Mortality Study

A complete description of the textile plant which is the subject of this study is given in previous publications [Dement et al., 1981, 1983]. Briefly, the plant was

located in South Carolina and began production of asbestos products in 1896. Asbestos textiles were first produced in 1909. Chrysotile, received from Quebec, British Columbia, and Rhodesia, was the only type of asbestos processed as a raw fiber. Crocidolite yarn was used in small quantities to make tape or braided packing from 1950s until 1975. The total quantity of crocidolite used was approximately 2,000 pounds compared to 6–8 million pounds per year of chrysotile during the same time period. Crocidolite was never carded, spun, or twisted; thus, the predominant exposure by far at this plant was to chrysotile asbestos.

This plant is unique in that most changes to control dust exposures were in place by 1940, and the plant processes remained fairly constant during the period from 1940 until production of asbestos textiles ceased in the late 1970s. Chrysotile exposure levels (expressed as fibers longer than 5 μm/cc of air) by areas of the plant (department and operations), specific textile jobs, and calendar years were estimated based on historic data and were used in the previously published analyses [Dement et al., 1981, 1983b]. Individual lifetime cumulative exposures were calculated and used for estimating exposure–response relationships. These same exposure estimates were used in the present analyses as well.

In the original analysis, the study cohort was defined as all white male workers employed in the textile production operations for at least 1 month between January 1, 1940 and December 31, 1965 with vital status follow-up through December 31, 1975. The updated cohort includes white and black males and white female workers who met the same employment requirements with follow-up of vital status extended through December 31, 1990. All of the basic data coded for the original study, including the work histories at the plant and follow-up information through 1975, were used in the update. Vital status follow-up was accomplished by matching the individuals in the cohort with the death master files of the Social Security Administration (SSA) and with the National Death Index (NDI). Since the NDI files are complete from 1979 to 1990, the SSA files were primarily used for identifying deaths from 1976 through 1978. If a worker was known to be alive as of 1975 based on the original study, he/she was assumed to be alive as of 1990 if there was no match with SSA or NDI files. If the vital status of the worker was unknown (i.e., lost to follow-up) in the original study and there was no match with the SSA or NDI files he/she was considered to have unknown vital status in this study. An attempt was also made to ascertain vital status of those considered unknown by searching Internal Revenue Service files. Death certificates were obtained from the state vital records offices and the underlying cause of death was coded by a qualified nosologist according to the revision of the International Classification of Diseases (ICD) in effect at the time of death.

The life-table analysis sytem (LTAS) of the National Institute for Occupational Safety and Health (NIOSH) [Steenland et al., 1990] was used to calculate the standardized mortality ratios (SMR) for specific causes of death. The LTAS distributes person-years at risk (PYAR) of dying for each worker into strata by 5-year age groups and 5-year calendar time periods; and further stratifies PYAR by length of employment (or by exposure) and time since first employment (latency). The PYAR for each worker were accumulated from the time 1 month of employment was achieved to the end of the study (December 31, 1990) or the date of death, whichever occurred first. For those lost to follow-up, PYAR were accumulated to the date they were last known to be alive which was the date last employed in most cases. Those who died after December 31, 1990 were considered alive during the study period. PYAR in each age

and calendar strata were multiplied by the corresponding U.S. mortality rates specific for each race and sex group to calculate expected deaths by cause. For comparison, analyses were completed using South Carolina death rates as well. For the analyses using South Carolina rates, PYAR and observed deaths that occurred prior to 1960 were not included because South Carolina death rates were not available prior to that date. The 90% confidence interval (CI) was computed for each cause-specific SMR using the Byar approximation when the deaths were eight or more or the Fisher exact method when the deaths were fewer than eight [Rothman and Boice, 1979].

SMR values presented in this report are slightly different from preliminary data previously published [Dement and Brown, 1993] due to differences in handling those lost to follow-up. In the preliminary analyses, those lost to follow-up were assumed to be alive as of December 31, 1990 thus contributing the maximum number of person-years to the calculation of expected deaths. As described above, the current analyses use the generally preferred procedure of terminating person-years for those lost on the date they were last known to be alive. The results for white males are essentially the same irrespective of method used due to the small number lost to follow-up.

Methods used to calculate SMRs by cumulative exposure were the same as described in the original analyses [Dement et al., 1981, 1983b]. Briefly, fiber concentrations, expressed as fibers longer than 5 μm/cc, were estimated for all textile jobs and calendar time periods. For each worker, time spent in each job (in terms of days) was multiplied by the corresponding estimated exposure level to give cumulative exposures (fiber/cc-days). As the worker was followed throughout the study period, the cumulative exposure was calculated and the PYAR were assigned to the appropriate exposure strata. Cumulative exposure strata were defined a priori.

Standardized rate ratios (SRR) for lung cancer among white males and females were also calculated from the LTAS. The lowest exposure stratum (<1,000 fiber/cc-days) was used as the referent group for comparison with the higher exposure strata. For this analysis, the midpoints of each cumulative exposure stratum were used except for the last category which was equal to the lower bound plus 50% (e.g., 40,000 + 20,000 = 60,000 fiber/cc-days). SRRs for black males were not calculated because of the lack of observed deaths in the low exposure strata. The analysis by SRRs allows for direct comparison of risks across strata as well as a test for linear trend and calculation of the exposure–response slope [Rothman, 1986].

In addition to SRR calculations described above, linear statistical models were fitted to the SMR exposure–response data using procedures similar to those employed by the EPA and OSHA in their most recent risk assessments [EPA, 1986; OSHA, 1986]. For these analyses, the lung cancer SMR was used as the dependent variable and cumulative asbestos exposure was used as the independent variable. Midpoints of each cumulative exposure stratum were the same as those used for the SRR analyses described above. Cumulative exposure was expressed as fiber/cc-years by dividing fiber/cc-days by 365.5. Weights used for each observation were the inverses of the estimated SMR variances.

Nested Case–Control Study

In order to further examine possible differences in lung cancer mortality by plant operation and to explore possible confounding due to mineral oil exposures, a nested case–control study was undertaken. Cases included cohort members with an

underlying cause of death of cancer of the trachea, bronchus, or lung (9th Revision ICD code 162). A total of 126 lung cancers were included (74 while males, 38 white females, 14 nonwhite males). Five controls were chosen for each lung cancer case by incidence density matching on the race, sex, and age at death of the index cases [Beaumont et al., 1989]. This program terminates the work history of the controls when they reach the ages of death of their matched index case.

Work histories of cases and controls were used to assign plant operation and mineral oil exposure variables. Plant operation and exposure zone groupings were determined a priori based on expected similarity in airborne fiber characteristics [Dement, 1980]. Analyses were conducted by assigning cases and controls to the plant operation or zone in which they were employed for the greatest period of time. Qualitative mineral oil exposure categories were the same as previously used by Dement [1991].

Lung cancer odds-ratios by plant operations and exposure zones, as well as qualitative mineral oil exposure categories, were estimated using conditional logistic regression [Pearce et al., 1989a,b]. Variables included in the conditional regression models included year of death of the index case in five groupings (1940–1949, 1950–1959,. . . . 1980–1990), time since first exposure in two categories (<15 years, $>=15$ years), and five groupings of exposure expressed in fiber/cc-days ($<1{,}000$, 1,000–2,499, 2,500–9,999, 10,000–39,999, $>=40{,}000$). These analyses thus used the lowest cumulative exposure category of $<1{,}000$ fiber/cc-days as the internal reference group for calculation of lung cancer odds-ratios. Both continuous and categorical exposure variables were tested in logistic regression models; however, categorical rather than continuous exposure variables were used in the final models for several reasons. First, the exposure–response relationship between lung cancer and asbestos exposure is best described as linear [OSHA, 1986; EPA, 1986]. Checkoway et al. [1989] have shown that categorical exposure variables are more appropriate in logistic models when the exposure–response relationship is linear. Second, categorical analyses allowed more direct comparisons with results from the cohort study. Conditional logistic regression was used to estimate lung cancer odds-ratios related to employment in plant operations and mineral oil categories after controlling for other variables in the models as described above. Logistic models were fitted using the SAS PHREG maximum likelihood estimation procedure [SAS, 1991].

RESULTS

Overall Cohort Mortality

The current update of the original study population added 15 years of observation. Results of the update for vital status information are given in Table I. Among white males, the number of deaths almost doubled from 308 to 607 and an additional 11,000 PYAR (from 33,141 to 44,131) were included. The update includes 289 total deaths among black males and 363 among white females. There was a fairly high percentage of black males with unknown vital status (7.6%) and with missing death certificates (7.6%). As is the case in other occupational cohort mortality studies, the follow-up for females was less complete than it was for males. Among the white females, 22.8% had an unknown vital status. This is partially due to married name changes, use of their spouse's Social Security Number (especially in the 1940s), and because of the greater tendency among women to leave the work force, making it

TABLE I. Vital Status of Asbestos Textile Cohort: 1940–1990

Vital status	White males (%)	White females (%)	Black males (%)	Total
Alive	621 (49.8)	586 (47.7)	214 (39.2)	1,421 (47.0)
Dead	607 (48.7)	363 (29.5)	289 (53.0)	1,259 (41.7)
Missing death certificates	17 (2.8)	40 (11)	22 (7.6)	79 (6.3)
Unknown	19 (1.5)	280 (22.8)	43 (7.8)	342 (11.3)
Total	1,247	1,229	546	3,022
PYAR[a]	44,131	52,489	19,254	115,874
Average years of observation	35	43	35	38
Median cumulative exposure (fiber/cc-days)	1,462	1,531	5,316	1,919

[a]PYAR, person-years at risk.

more difficult to determine vital status from the SSA, NDI, or Internal Revenue Service record systems.

The PYAR distribution by cumulative exposure strata for the cohort with at least 15 years of latency is given in Table II. This distribution was restricted to 15 years of latency since this criterion was used in the exposure–response analysis. The distribution for white males and females was similar but black males have a lower percentage of PYAR in the lowest exposure stratum and a higher percentage in the highest exposure stratum.

Table III gives the results of the mortality analysis for major causes of death by race and sex based on use of U.S. mortality rates to generate expected deaths. Among the white males, the excess mortality for all causes combined (SMR = 1.48; CI = 1.38–1.58) and for several of the major causes of death is statistically significant including: cancer (SMR = 1.50; CI = 1.29–1.72), diabetes mellitus (SMR = 2.05; CI = 1.18–3.33), heart disease (SMR = 1.41; CI = 1.26–1.58), cerebrovascular disease (SMR = 1.50; CI = 1.08–2.02), nonmalignant respiratory diseases (SMR = 2.30; CI = 1.82–2.86), and accidents (SMR = 1.49; CI = 1.15–1.91). The category, "nonmalignant respiratory diseases" is primarily composed of pneumoconiosis (including asbestosis) and other respiratory diseases (primarily chronic obstructive pulmonary disease). The SMR for this subcategory was 4.10 (CI = 3.10–5.31). Approximately 50% (21/41) of these deaths were due to pneumoconiosis. Using South Carolina death rates to estimate expected deaths, the SMRs for most causes were similar to those using U.S. death rates except mortality for cerebrovascular disease (SMR = 1.23; CI = 0.88–1.66) and accidents (SMR = 1.13; CI = 0.80–1.55) was elevated but no longer statistically significant.

Among white females, risks for all causes combined (SMR = 1.21; CI = 1.11–1.32) and pneumoconiosis and other nonmalignant respiratory diseases (SMR = 2.40; CI = 1.53–3.60) were statistically significant. Seven of the 17 deaths in this latter category were due to pneumoconiosis. Use of South Carolina death rates yielded similar results; however, the excess risks for all cancer combined (SMR = 1.35; CI = 1.13–1.60) and for diseases of the digestive system (SMR = 1.58; CI = 1.02–2.34) were statistically significant.

Among black males, the only statistically significant excess was for the category, pneumoconiosis and other respiratory diseases (SMR = 2.19; CI = 1.23–

TABLE II. PYAR by Cumulative Exposure for the Asbestos Textile Cohort With Greater Than 15 Years Latency*

Cumulative exposure fiber/cc-days (fibers/cc-yrs)	White males PYAR (%)	White females PYAR (%)	Black males PYAR (%)	Total PYAR (%)
<1,000 (<2.7)	10,772 (41)	9,596 (37)	1,533 (15)	21,901 (35)
1,000–2,500 (2.7–6.8)	5,318 (20)	4,823 (18)	1,719 (17)	11,860 (19)
2,500–10,000 (6.8–27.4)	4,372 (16)	5,120 (20)	3,483 (34)	12,975 (20)
10,000–40,000 (27.4–109.5)	4,491 (17)	5,223 (20)	2,198 (21)	11,912 (19)
>40,000 (>109.5)	1,651 (6)	1,275 (5)	1,390 (13)	4,316 (7)
Total	26,604 (100)	26,037 (100)	10,323 (100)	62,964 (100)

*PYAR, person-years at risk.

3.62). Six of the 11 deaths in this category were due to pneumoconiosis. There was little change in these results using South Carolina death reates except that a statistically significant deficit for all causes mortality (SMR = 0.89; CI = 0.80–0.99) and for cerebrovascular disease (SMR = 0.54; CI = 0.35–0.80) was observed.

Table IV gives the mortality results for malignant neoplasms of specific sites. White males had a statistically significant increase in all respiratory tract cancer (SMR = 2.30; CI = 1.89–2.78), primarily due to lung cancer (SMR = 2.30; CI = 1.88–2.79), and a statistically significant increase in liver/gall bladder/biliary tract cancer (SMR = 4.12; CI = 1.79–8.13). The mortality excess for lung cancer based on South Carolina death rates was reduced but still statistically significant (SMR = 1.80; CI = 1.44–2.22).

Among white females, the only statistically significant increase in malignant neoplasms was for all respiratory tract cancer (SMR = 2.87; CI = 2.17–3.72) including lung cancer (SMR = 2.75; CI = 2.06–3.61) and other respiratory cancers (SMR = 14.98; CI = 4.08–38.7). Based on South Carolina rates, the lung cancer excess was slightly higher (SMR = 3.36; CI = 2.51–4.40).

There was no statistically significant excesses by specific cancer site among the black males. The overall lung cancer SMR was 0.78 (CI = 0.47–1.22). Based on South Carolina death rates, the risk was slightly higher (SMR = 0.92; CI = 0.56–1.43).

With regard to the other a priori diseases of interest, cancers of the pancreas (7 observed vs. 4.7 expected), urinary organs (7 observed vs. 4.6 expected), and larynx (3 observed vs. 1.3 expected) were observed in excess among white males as well as cancers of the pancreas (4 observed vs. 2.8 expected) among black males, although these excesses were not statistically significant. For the whole cohort combined, there was a statistically significant excess for pancreatic cancer (5 observed vs. <1 expected) at the highest exposure level (>40,000 fiber/cc-days). There was a statistically significant increase in risk for ischemic heart disease among white males (SMR = 1.43; CI = 1.26–1.61) and elevated but not statistically significant risks among white females and black males.

All death certificates with an underlying cause of death that may have been related to mesothelioma were reviewed using methods described by Lilienfeld and Gunderson [1986]. Mesothelioma was recorded on the death certificates of two white males who had latency periods of 37 and 34 years and who worked at the plant for

TABLE III. Mortality by Major Cause of Death Categories Among the Asbestos Textile Cohort Based on U.S. Death Rates

Cause of death category	White males		White females		Black males		Total	
(9th Revision ICD codes)	OBS[a]	SMR[a]	OBS	SMR	OBS	SMR	OBS	SMR
Tuberculosis (010–018)	6	1.67 (0.72–3.29)[b]	0	0	6	0.76 (0.33–1.50)	12	0.86 (0.49–1.38)
All cancer (140–208)	137	1.50 (1.29–1.72)	98	1.09 (0.91–1.29)	48	0.88 (0.68–1.11)	283	1.19 (1.08–1.32)
Diabetes mellitus (250)	12	2.05 (1.18–3.33)	10	1.37 (0.74–2.33)	4	0.84 (0.29–1.92)	26	1.45 (1.01–1.99)
Heart disease (390–398,402,404, 410–414,420–429)	226	1.41 (1.26–1.58)	106	1.13 (0.95–1.32)	82	0.96 (0.79–1.16)	414	1.22 (1.12–1.32)
• Ischemic heart disease (410–114)	188	1.43 (1.26–1.61)	76	1.10 (0.90–1.33)	57	1.02 (0.81–1.27)	321	1.25 (1.13–1.37)
Cerebrovascular disease (430–438)	31	1.50 (1.08–2.02)	22	0.93 (0.63–1.32)	22	0.93 (0.66–1.40)	75	1.12 (0.91–1.35)
Respiratory disease (460–466,470–478, 480–487,490–519)	58	2.30 (1.82–2.86)	22	1.25 (0.84–1.78)	25	1.47 (1.02–2.05)	105	1.75 (1.47–2.05)
• Pneumoconiosis and other (470–478, 494–519)	41	4.10 (3.10–5.31)	17	2.40 (1.53–3.60)	11	2.19 (1.23–3.62)	69	3.11 (2.52–3.80)
Digestive Disease (520–579)	23	1.10 (0.75–1.55)	19	1.31 (0.85–1.92)	10	0.74 (0.40–1.26)	52	1.06 (0.83–1.33)
Accidents (E800–E949)	47	1.49 (1.15–1.91)	9	0.93 (0.48–1.62)	21	1.03 (0.69–1.49)	77	1.25 (1.02–1.51)
All other and unknown	67	1.30	76	1.83	71	1.34	214	1.43
All causes	607	1.48 (1.38–1.58)	362	1.21 (1.11–1.32)	289	1.04 (0.94–1.14)	1,258	1.28 (1.21–1.33)

[a]OBS, observed; SMR, standardized mortality ratio.
[b]Numbers in parentheses are 90% confidence interval.

25 and 32 years, respectively. Both of these workers were primarily employed in spinning operations. An additional mesothelioma case (not included in the cohort) which occurred after 1990 was observed in a white male employed in mostly non-textile operations at this plant. In addition, four deaths coded as "cancers of other parts of the respiratory system" (ICD-8 code 163×) occurred; three among white females (3 observed vs. 0.20 expected; SMR = 14.98; CI = 3.12–29.6) and one among white males (1 observed vs. 0.39 expected; SMR = 2.64; CI = 0.14–12.5). A close review of these deaths found that all mentioned cancer of the lung but without mention of specific site within the lung. Information from death certificates thus suggests that these tumors were lung cancers rather than mesotheliomas; however, no autopsy or other medical reports were available for further confirmation of the primary cancer site.

Cohort Study of Exposure–Response

In order to further examine the exposure–response relationship for lung cancer, the risk was examined by cumulative exposure. These analyses were restricted to

TABLE IV. Mortality From Selected Malignant Neoplasms Among the Asbestos Textile Cohort Based on U.S. Rates

Cause of death category (9th Revision ICD codes)	White males		White females		Black males		Total	
	OBS[a]	SMR[a]	OBS	SMR	OBS	SMR	OBS	SMR
All digestive and peritoneum (150–159)	23 (0.66–1.37)[b]	0.97	12 (0.32–0.91)	0.56	18 (0.70–1.61)	1.08	53 (0.67–1.07)	0.86
• Stomach (151)	3 (0.21–2.00)	0.77	0 (E = 2.38)[c]	—	6 (0.69–3.15)	1.60	9 (0.47–1.56)	0.90
• Liver and biliary (155.0,155.1,156)	6 (1.79–8.13)	4.12	0 (E = 1.66)	—	0 (E = 1.20)	—	6 (0.60–2.73)	1.38
• Pancreas (157)	7 (0.70–2.80)	1.49	4 (0.33–2.20)	0.96	4 (0.50–3.31)	1.45	15 (0.79–1.98)	1.28
All respiratory (160–165)	78 (1.89–2.78)	2.30	41 (2.17–3.72)	2.87	15 (0.48–1.20)	0.78	134 (1.71–2.29)	1.99
• Larynx (161)	3 (0.63–5.96)	2.31	0 (E = 0.29)	—	1 (0.05–4.84)	1.02	4 (0.53–3.55)	1.55
• Trachea, bronchus, lung (162)	74 (1.88–2.79)	2.30	38 (2.06–3.61)	2.75	14 (0.47–1.22)	0.78	126 (1.69–2.28)	1.97
• Other respiratory (160,163–165)	1 (0.14–12.5)	2.64	3 (4.08–38.7)	14.98	0 (E = 0.21)	—	4 (1.73–11.5)	5.04
Lymphatic and hematopoietic (200–208)	9 (0.54–1.80)	1.03	7 (0.44–1.77)	0.94	3 (0.22–2.13)	0.82	19 (0.63–1.40)	0.96
All other	27	1.07	38	0.90	12	0.78	77	0.87
All cancer (140–208)	137 (1.29–1.72)	1.50	98 (0.91–1.29)	1.09	48 (0.68–1.11)	0.88	283 (1.08–1.32)	1.19

[a]OBS, observed, SMR, standardized mortality ratio.
[b]Numbers in parentheses are 90% confidence interval.
[c]E, expected deaths.

those with at least 15 years of latency as this was the time period used in the original analysis and considered important for occupational cancer risk. Table V gives the lung cancer mortality by cumulative exposure as measured in fiber/cc-days (and fiber/cc-years). Generally, there was an increase in risk (SMR and SRR) for lung cancer with increasing cumulative exposure, although there was some inconsistency in this trend among black males where few deaths were observed in the low exposure stratum. The lung cancer risk pattern among white females at low exposure was different from white males in that a statistically significant excess was observed among females in the lowest exposure stratum.

Based on the SRR analysis, there was a statistically significant positive trend for white males ($Z = 2.88$; $p < 0.01$) but not for white females ($Z = 1.71$; $p > 0.05$). When the white males and females were combined, the trend was more consistent and highly significant ($Z = 4.25$; $p < 0.01$), and the trend for the total cohort, including the black males, also was significant ($Z = 3.56$; $p < 0.01$). The slopes are also reported in Table V. For white males, the slope was 9.093×10^{-8} or an increase in lung cancer of 0.009/100,000 for each fiber/cc-days, which is equivalent to 3.29/100,000 for each fiber/cc-year, and the relative risk increases approximately 3% per

TABLE V. Lung Cancer Mortality by Cumulative Exposure Among the Asbestos Textile Cohort With at Least 15 Years Latency*

Cumulative exposure fiber/cc-days (fiber/cc-years)	<1,000 (<2.7)	1,000 2,500 (2.7–6.8)	2,500 10,000 (6.8–27.4)	10,000 40,000 (27.4–109.5)	>40,000 (>109.5)	Total
White males	11/12.3	15/5.8	10/5.1	16/5.2	20/2.4	72/30.8
	0.89	2.59[a]	1.96[b]	3.08[a]	8.33[a]	2.34[a]
	1.00	2.63	2.03	2.95	6.60	3.35
		[1.20–5.75]	[0.86–4.80]	[1.33–6.57]	[2.97–14.66]	[1.75–6.39]
White females	10/5.0	4/2.6	8/2.6	13/2.6	3/0.7	38/13.5
	2.00[b]	1.54	3.08[a]	5.00[a]	4.29[b]	2.81[a]
	1.00	0.74	1.36	2.17	1.63	1.62
		[0.23–2.37]	[0.54–3.48]	[0.93–5.07]	[0.39–6.75]	[0.78–3.34]
White males and females	21/17.3	19/8.4	18/7.7	29/7.8	23/3.1	110/44.4
	1.21	2.26[a]	2.33[a]	3.72[a]	7.42[a]	2.48[a]
	1.00	1.70	1.70	2.57	4.16	2.50
		[0.91–3.18]	[0.90–3.21]	[1.44–4.60]	[2.16–8.01]	[1.55–4.03]
Black males	0/2.5	3/2.9	2/5.9	1/3.5	8/2.3	14/17.2
	—	1.03	0.34	0.29	3.48[a]	0.81
Total	21/19.8	22/11.3	20/13.6	30/11.4	31/5.4	124/61.5
	1.06	1.95[a]	1.47	2.63[a]	5.74[a]	2.02[a]
	1.00	2.05	1.80	2.68	4.87	2.78
		[1.12–3.76]	[0.96–3.35]	[1.51–4.78]	[2.64–8.99]	[1.74–4.47]

*Each cell includes observed/expected deaths, SMR, SRR, and (95% CI) for SRR. Based on SRR analyses: white males—slope = 9.093×10^{-8} with a standard error = 3.16×10^{-8}, Z = 2.88; $p < 0.01$. white females—slope = 2.527×10^{-8} with a standard error = 1.477×10^{-8}, Z = 1.71; $p > 0.05$. white males and females combined—slope = 5.867×10^{-8} with a standard error = 1.381×10^{-8}, Z = 4.25; $p < 0.01$. Total cohort— slope = 5.654×10^{-8} with a standard error = 1.588, Z = 3.56; $p < 0.01$.

[a] $p < 0.01$.

[b] $p < 0.05$.

fiber/cc-years. For white females, the increase in lung cancer rate was 0.91/100,000 for each fiber/cc-year, and the relative risk increases by 1% for each fiber/cc-year. For the whole cohort, the increase in lung cancer rate was 2.06/100,000 for each fiber/cc-year, and the relative risk increases by 2% for each fiber/cc-year. Results for the weighted regression analyses using the SMR data for the whole cohort yielded comparable results, demonstrating a 3% increase in the lung cancer relative risk for each fiber/cc-year of exposure (SMR = 1.0 + 0.031 [fiber/cc-year], standard error of slope = 0.005).

The inconsistency in the exposure–response relationship among the females may be a result of the unequal distribution of those lost to follow-up, most of which occurred among workers with short employment duration and thus low cumulative exposure. Among the 280 white females lost to follow-up, 36% worked less than 3 months, 18% worked 3–6 months, and 17% worked 6 months–1 year. If it is assumed all of those lost to follow-up were alive at the end of the study, the exposure–response for the white females would be altered by lowering the risks in the low exposure stratum yielding the following SMRs for the five exposure strata: 1.28, 1.03, 2.50, 4.64, and 4.29 (the last three SMRs were statistically significant). Black males were affected to a lesser extent. Because of the high percentage of lost to follow-up among

TABLE VI. Pneumoconioses Mortality by Cumulative Exposure Among the Asbestos Textile Cohort With at Least 15 Years Latency*

Cumulative exposure fiber/cc-days (fiber/cc-years)	<1,000 (<2.7)	1,000 2,500 (2.7–6.8)	2,500 10,000 (6.8–27.4)	10,000 40,000 (27.4–109.5)	>40,000 (>109.5)	Total
White males	9/3.7	3/1.8	3/1.7	11/1.7	13/0.8	39/9.6
	2.43	1.67	1.76	6.47[a]	16.25[a]	4.06[a]
White females	4/2.5	2/1.3	1/1.2	5/1.4	5/0.4	17/6.9
	1.60	1.53	0.83	3.57[b]	12.50[a]	2.46[a]
Black males	0/0.7	0/0.8	3/1.6	3/1.0	5/0.6	11/4.6
	—	—	1.88	3.00	8.33[a]	2.39[a]
Total	13/6.9	5/3.9	7/4.5	19/4.1	23/1.8	67/21.2
	1.88[b]	1.28	1.56	4.63[a]	12.78[a]	3.16[a]

*Pneumoconioses and other respiratory diseases (9th revision ICD codes 470–478 and 494–519). Each cell includes observed/expected deaths and SMR.
[a]$p < 0.05$.
[b]$p < 0.01$.

the females (and to a lesser extent among the black males), more confidence and emphasis should be placed on the exposure–response analysis for white males.

Regardless of these problems with follow-up, there appears to be a difference in the overall lung cancer risk as well as the exposure–response relationship for white workers compared to black workers. Among white males and females, a statistically significant increase begins at a cumulative exposure much lower than that for black males. Although this was based on few observed deaths in the low exposure stratum for black males, there were enough data to detect a statistically significant deficit in risk (SMR = 0.40; 6 observed vs. 14.8 expected deaths) below 40,000 fiber/cc-days.

Pneumoconiosis was the other asbestos-related disease found in excess and therefore examined in more detail. This category of disease, as defined by the NIOSH LTAS used in this study, includes pneumoconioses, such as asbestosis, as well as chronic obstructive pulmonary disease. The underlying cause of death for approximately 50% of these deaths was from pneumoconiosis. Tables VI gives the results of mortality from pneumoconiosis by cumulative exposure. Like lung cancer, a positive exposure–response was observed in all race/sex groups and, similar to the lung cancer analysis, there was some inconsistency in the trend at low exposures.

Nested Case–Control Analyses

A summary of demographic and exposure variables for the lung cancer cases and controls is shown in Table VII. Since age at death was the incidence density matching variable, cases and controls were nearly identical for this parameter. Cases and controls also showed close agreement with regard to year of birth, date first employed, and years since first employed. As expected, cases experienced higher mean cumulative asbestos exposures than controls although the distribution of cumulative exposure values was highly skewed toward lower cumulative exposures. With regard to average exposure levels among lung cancer cases, black males were the highest (12.0 fiber/cc) followed by white males (5.5 fibers/cc) and white females

TABLE VII. Summary of Demographic and Exposure Variables for Lung Cancer Cases and Controls in Cohort of Asbestos Textile Workers, 1940–1990

Demographic or exposure variable	Mean value and (standard deviation)					
	White males		White females		Black males	
	Cases	Controls	Cases	Controls	Cases	Controls
Number	74	370	38	190	14	70
Year of birth	1913 (9.3)	1911 (10.9)	1914 (10.3)	1909 (8.1)	1917 (7.3)	1911 (10.8)
Date first employed	1941 (7.8)	1941 (6.3)	1943 (6.0)	1942 (4.6)	1944 (3.5)	1942 (3.5)
Date last observed	1974 (11.2)	1972 (11.0)	1982 (8.3)	1977 (10.6)	1981 (6.3)	1976 (10.0)
Years since first emp	34.1 (9.1)	31.1 (11.3)	38.9 (8.7)	35.1 (11.1)	37.4 (6.3)	33.1 (9.8)
Age at risk	61.9 (7.5)	61.8 (7.5)	67.6 (10.6)	67.6 (10.5)	64.4 (8.2)	64.6 (8.1)
Cumulative exposure[a]	24.5 (29.6)	14.6 (33.9)	13.2 (15.5)	11.9 (17.4)	38.4 (37.1)	16.4 (20.8)
Average exposure[b]	5.5 (4.1)	5.2 (5.5)	4.9 (2.3)	4.9 (2.0)	12.0 (9.7)	11.9 (7.8)

[a]Cumulative exposure expressed in 1,000 fiber/cc-days. The distribution of exposures is highly skewed and approximately lognormal.
[b]Average exposure level (fibers/cc) calculated as the cumulative exposure divided by the number of days employed.

(4.9 fibers/cc). These differences in average exposure values reflect differences in job assignment patterns by race and sex.

Results of the overall exposure–response analyses obtained from the case–control study with and without control for mineral exposures are shown in Table VIII. Significant excess lung cancer risk was observed for all cumulative exposures using the category <1,000 fiber/cc-days as the internal referent. The lung cancer odds-ratio increased with cumulative exposure, reaching a value of 7.11 (CI = 3.51–14.40) among workers achieving a cumulative exposure of 40,000 fiber/cc-days or greater. Results of the exposure–response analyses adjusting for qualitative mineral oil category demonstrate little effect of mineral oil exposure. These results are similar to those obtained by Dement [1991] using a much smaller number of lung cancer cases and controls. Mineral oil exposure does not appear to be a significant confounder in the risk estimates associated with cumulative asbestos exposure. Race and sex interactive terms with exposure were entered into the regression models but failed to achieve statistical significance. The small number of lung cancer deaths for black males in the low exposure stratum make it difficult to look at differences by race.

Lung cancer odds-ratios by the longest department worked, after adjusting for cumulative exposure, are shown in Table IX. These data suggest that workers in preparation and carding may be at slightly reduced lung cancer risk compared to other operations although the odds-ratio was not statistically different from 1.0. Employment in spinning and twisting operations was associated with significantly increased lung cancer risk compared to other plant operations (OR = 1.75, CI = 1.07–2.86).

DISCUSSION

The primary focus of this study was lung cancer mortality among chrysotile asbestos textile workers. This update of the original study more than doubled the number of lung cancer deaths among the white males and includes data for white

TABLE VIII. Case–Control Analyses of Lung Cancer Exposure-Response Controlling for Mineral Oil Exposure Among Asbestos Textile Workers

Cumulative asbestos exposure, fibers/cc days (fiber/cc-years)	Odds-ratio without control for mineral oil exposure	Odds-ratio with control for mineral oil exposure[a]
<1,000 (<2.7)	1.00	1.00
1,000–2,499 (2.7–6.8)	2.13 (1.12–4.07)	2.04 (1.07–3.90)
2,500–9,999 (6.8–27.4)	2.14 (1.06–4.33)	2.05 (1.00–4.13)
10,000–39,999 (27.4–109.5)	3.27 (1.71–6.24)	3.26 (1.71–6.22)
40,000 or greater (>109.5)	7.11 (3.51–14.40)	7.03 (3.47–14.24)

[a]Conditional logistic regression odds-ratio and 95% CI controlling for year of death of index case, time since first exposure, and mineral oil exposure.

TABLE IX. Lung Cancer Odds-Ratios by Longest Employment in Plant Operations and Exposure Zones in Asbestos Textile Workers

Plant operations and exposure zones	Odds-ratio[a]
Preparation	0.88 (0.36–1.78)
Carding	0.80 (0.39–2.00)
Spining and twisting	1.75 (1.07–2.86)
Winding	0.78 (0.41–1.49)
Weaving	0.94 (0.59–1.50)

[a]Conditional logistic regression odds-ratios and 95% CI controlling for year of death for index case, time since first exposure, and cumulative asbestos exposure categories.

female and black male workers. With the increase in the number of lung cancer deaths, it was possible to calculate more precise estimates of risk, especially at low cumulative exposures. Results of the cohort analysis for white males confirm findings from the original study. Among white females, the overall risk for lung cancer, as well as the observed exposure–response, further corroborates the findings for white males. In contrast, the lung cancer mortality pattern among black males appears to be different compared to white males and females. The overall lung cancer risk for black males was less than expected and a statistically significant excess occurred only at the highest cumulative exposure although data in the low exposure stratum for black males were sparse. There are at least two possible explanations for this observation: reduced smoking among black males at this plant compared to U.S. black males used as the referent population and/or differences in risk due to different plant employment patterns for blacks vs. whites.

Based on two surveys conducted by the U.S. Public Health Service in 1964 and 1971 and on data collected by the company, the prevalence of smoking was estimated for a sample of the cohort (N = 292 white males, N = 124 white females, and N = 113 black males) [Dement, 1980]. Among the white males, 52.4% were current smokers, 22.3% were past smokers, and 25.3% nonsmokers; among white females 42.7% were current smokers, 6.5% were past smokers, and 50.8% were nonsmokers; and among black males 38.1% were current smokers, 14.1 were past smokers, and 47.8% were nonsmokers. These smoking patterns may affect lung cancer risk estimates in two ways. First, the prevalence of smoking among black males in the United States in 1965 was 60.8% [Public Health Service, 1979], which was much higher

TABLE X. Summary of Airborne Asbestos Fiber Size Data by Textile Operation Determined by Electron Microscopy*

	Fiber diameter Percent less than size			Fiber length Percent less than size		
Textile operation	<0.1 μm	<0.5 μm	<1.0 μm	<0.5 μm	<5.0 μm	<10.0 μm
Preparation	29.8	88.6	92.7	4.3	72.7	85.4
Carding	41.2	94.2	98.3	7.9	86.0	92.4
Spinning and twisting	34.5	87.3	94.9	6.8	74.4	83.8
Winding	44.2	91.2	96.4	0.6	80.5	88.2
Weaving	66.8	96.0	97.8	21.2	89.7	94.6

*Data from Dement [1980]. More than 500 fibers were sized by transmission electron microscopy using five to eight individual air samples for each operation.

than the 38.1% in the cohort, whereas the prevalence in the United States for white males was 51.5% or almost the same as the cohort, and the prevalence among white women was 34.2%, which was lower than the prevalence among the cohort. Since U.S. mortality rates were used in the analysis, these smoking patterns may have biased the results yielding an underestimate of the lung cancer risk for black males, an overestimate of the risk for white females, and no effect for white males. Second, smoking is known to have a synergistic effect with asbestos in causing lung cancer [Hammond et al., 1979]. The statistically significant risk for lung cancer seen for white males and white females at lower levels of exposure compared to black males may be due to the synergistic effect related to cigarette smoking.

Another possible reason for the difference in lung cancer risk between the white and the black workers may be related to plant job and process assignments and resulting fiber exposure characteristics. Characteristics of airborne fibers may have differed depending on the specific textile operation [Dement, 1980; Dement and Wallingford, 1990]. As raw fiber was further processed in textile operations, fiber bundles were broken into finer diameter fibers. An analysis of plant work assignments by race and sex found that most of the black males (70%) were employed in the preparation and carding operations where raw chrysotile fibers were first received from the mine and prepared for further processing. In contrast, most of the white males (93%) and white females (98%) were employed in spinning, twisting, winding, weaving, and finishing.

Nested case–control studies were undertaken in order to further examine lung cancer mortality risks by plant operation. While employment in all textile operations was found to be associated with excess risk of lung cancer, these analyses generally support a slightly lower lung cancer risk among workers employed in the initial operations of fiber preparation and carding, as well as higher risks for spinning and twisting. Table X is a summary of the electron microscopic fiber size analyses of airborne dust samples collected in this plant between 1965 and 1971 [Dement, 1980]. Fiber preparation was found to have a smaller fraction of fibers <0.1 μm in diameter compared to other textile operations whereas fiber lengths were more similar. Interestingly, weaving operations were found to produce fibers which were both smaller in diameter and shorter in length. Spinning and twisting operations produced more fibers longer than 10 μm.

Taken at face value, the available airborne fiber size data provide only moderate

support for airborne fiber characteristics as a possible explanation for differences in lung cancer exposure–response by race seen in this study. However, it must be realized that samples available for study were taken during 1965–1971; thus, these data may not be representative of past airborne fiber characteristics. For example, raw, unopened fiber was received and processed at this plant until approximately 1965. Opening of these fibers was accomplished using a rotary pan crusher. It is likely that airborne fiber characteristics for this operation were different than represented by the airborne fiber size data shown in Table X. Dreessen et al. [1938] studied airborne fibers characteristics in textile plants which used the pan crushing method in fiber preparation. Using oil immersion optical microscopy ($\times$ 1,000), Dreessen et al. [1938] found that airborne dust samples from preparation and carding "contained few fibers and considerable particulate matter." Median fiber lengths also were shorter in preparation and carding compared to twisting and weaving. It thus appears that airborne fiber characteristics could partially explain, in conjunction with reduced cigarette smoking, the lower lung cancer risk observed among black males most of whom were employed in fiber preparation and carding.

Interestingly, it has been suggested that the lower lung cancer risk observed among chrysotile miners compared to textile workers may be due to a difference in fiber sizes [McDonald et al., 1983a; Dement, 1991]. This is consistent with the data in this study, since the raw asbestos from the mine was first handled by those in preparation and carding. Textile operations have been shown to produce airborne fibers which are generally longer in length than other operations using chrysotile [Dement and Wallingford, 1990]. Airborne fiber size measurement data for chrysotile mining and milling demonstrate a significantly greater proportion of fibers (95–98%) less than 5 μm in length [Gibbs and Hwang, 1980] compared to data from this textile plant shown in Table X. Such airborne size differences are generally supported by pathological studies which have examined fibers in the lungs of workers from this plant compared to fibers in the lungs of chrysotile miners and millers [Churg and Harley, 1984; Sébastien et al., 1989].

The observation of excess nonmalignant respiratory disease mortality at low cumulative exposures is interesting and deserves further study. OSHA estimated a lifetime incidence of asbestosis of 0.5% at the current standard of 0.2 fibers/cc. While exposure levels in the plant studied were generally much greater than 0.2 fibers/cc, these results none-the-less suggest increased risk at low cumulative exposures. These findings are consistent with other data which demonstrate increased pulmonary fibrosis in pathological specimens among workers at this plant who had low cumulative exposures [Green et al., 1986].

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