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Follow-Up of the Libby, Montana Screening Cohort:

A 17-Year Mortality Study

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Abstract

Objective: To evaluate mortality patterns among participants in a community-based screening program for asbestos-related disease.

Methods: We calculated standardized mortality ratios (SMRs) and stratified results by exposure group (three occupational exposure groups, household contacts and residents without occupational asbestos exposure) and by radiographic abnormality presence.

Results: All-cause mortality (15.8%; 1,429/8,043) was statistically lower than expected. Asbestosis was statistically elevated in all exposure groups. Lung cancer was moderately associated with vermiculite miner/miller employment. Mesothelioma was elevated in that same exposure group and among residents. Systemic autoimmune disease mortality was also elevated. Radiographic parenchymal abnormalities were associated with lung cancer mortality.

Conclusion: In addition to asbestos-related mortality in occupational exposure groups, this initial follow-up of this cohort also shows elevated mortality for some asbestos-related causes in non-occupational exposure groups.

Keywords

asbestos; asbestosis; chest radiograph; mesothelioma; mortality

Throughout much of the 20th century, Libby, Montana was the largest worldwide source of vermiculite, a mineral with commercially desirable properties.[1] However, by the end of the century it became widely known that vermiculite from the Libby mining and milling operation contained amphibole asbestos and other toxic fibers, generally referred to as Libby amphibole asbestos (LAA).[2] In addition to LAA exposure among workers at the vermiculite operation, non-occupational exposures also occurred as the result of wide use of vermiculite throughout the community.[3] These uses of vermiculite included covering of ball fields and

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outdoor athletic tracks, loose-fill insulation in buildings, and in gardening activities.[3] These community uses of vermiculite may have resulted in LAA exposures among Libby residents before and after closure of the vermiculite operation in 1990.

To gauge the public health impact of LAA, the US government via the Agency for Toxic Substances and Disease Registry (ATSDR) initiated a community-based health screening program during 2000 to 2001. Active measures to identify candidates for screening both locally and nationally netted 9,236 eligible persons, among whom 7,307 (79%) participated.[3] Notably, among 6,668 participants whose screening included evaluation of their chest radiograph, 17.8% had pleural abnormalities, a marker of asbestos exposure.[3] Further, the prevalence of pleural abnormalities increased with the number of self-reported exposure pathways.[3] During 2003 to 2008, the Montana Department of Public Health and Human Services (MDPHHS) continued to offer screening in Libby under a similar protocol in which 736 additional persons were screened.

Our goal for the present analysis was to conduct a mortality update of the screening cohort to determine if its mortality patterns differed from that of the US population and if mortality was associated with cohort subgroups.

METHODS

Study Population

The screening protocol used by ATSDR and MDPHHS to collect data for this analysis was approved by the Centers for Disease Control and Prevention (CDC) Institutional Review Board. The screening cohort comprised all participants in the ATSDR and MDPHHS programs. To have been eligible to participate in either screening program, one must have lived, worked, or played in Libby for at least 6 months prior to the end of 1990, when the mine ceased operation.[3] Screening included a standardized survey and spirometry and evaluation via chest radiography was offered to adults.[3] In the initial ATSDR screening, all radiographs were evaluated by two National Institute for Occupational Safety and Health (NIOSH) certified B readers[4] and an additional B reader conducted a third evaluation in instances where the first two readers' evaluations differed regarding the presence of pneumoconiosis.[5] In the MDPHHS program, each radiograph was evaluated by a single B reader.

We used survey and clinical measurement data collected through screening to create stratification variables for sex, age at baseline screening (categorized as 10 to 29, 30 to 49, 50 to 59, and 60 to 90 years), self-reported cigarette smoking status (current/ former smoker or never smoked), presence of radiographic pleural abnormalities, presence of radiographic parenchymal abnormalities, and LAA exposure category. For LAA exposure, we created five categories using the following mutually-exclusive hierarchy (from presumed most highly exposed group to least): workers employed by the W.R. Grace & Co. vermiculite operation (WRG workers), secondary contract workers to the vermiculite operation, persons reporting other potential occupational asbestos or LAA exposure, household contacts of WRG workers, and residents not falling into another category. We defined household contacts as persons reporting ever living with a WRG worker while the worker was employed by the

vermiculite operation. We defined workers with other potential occupational asbestos or LAA exposure as those reporting employment:

- with vermiculite exposure at other jobs, not including ones at the vermiculite mining and milling operation; or
- in a job involving mixing, cutting, or spraying asbestos material; or
- at any job that might have resulted in asbestos exposure; or
- exposure to asbestos during military service.

Statistical Analysis

Each participant's vital status and cause of death (coded using the International Classification of Disease, 10th Revision; ICD-10) were determined using the National Death Index (NDI).[6] Follow-up began on the date of initial screening, which occurred as early as 2000, and continued until December 31, 2016 or date of death. Unless otherwise specified, statistical analysis was conducted with SAS 9.4 (SAS Institute, Inc., Cary, NC).[7] We conducted Pearson chi-square tests of independence for each stratification variable with vital status. Next, we used the NIOSH computer program Life Table Analysis System (LTAS.NET)[8] to compute standardized mortality ratios (SMRs) comparing the screening cohort to age-, sex-, race-, and calendar-specific US mortality rates for underlying cause of death. Also using LTAS.NET we calculated directly standardized rate ratios (SRRs) for internal comparisons. For SMR and SRR calculations, person-years and deaths were stratified on age, race and calendar year (the standardizing variables) and smoking status, LAA exposure category, and radiographic abnormality. We present cause-specific SMRs that were either statistically significant or of interest because of their association with asbestos exposure in other settings and include asbestosis[4] and lung cancer and mesothelioma as well as colorectal, ovarian, laryngeal, pharyngeal, and stomach cancer.[9] We also present SMRs and SRRs for select asbestos-related causes of death stratified by exposure category or radiographic abnormality.

We conducted an ad-hoc SMR analysis for a disease class not included in 119 NIOSH disease categories used in LTAS.NET.[10] This disease class comprised a combination of three select systemic autoimmune diseases (SAIDs henceforth): systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), or scleroderma (coded in ICD-10 as M32, M04/M06, and M34, respectively). These SAIDs were previously associated with asbestos exposure in the Libby screening cohort.[11] Expected deaths were calculated from aggregated US multiple cause data from 1999 to 2016 obtained from the CDC database Wide-ranging ONline Data for Epidemiologic Research (WONDER; https://wonder.cdc.gov/) and person-years for the screening cohort obtained from LTAS.NET. We then used the STDRATE procedure in SAS 9.4 to calculate the SMR for SAIDs. Due to concern that the underlying cause of death might not accurately capture SAIDs, we used multiple cause of death to calculate its SMR.

RESULTS

The cohort comprised 8,043 participants (7,307 and 736 from ATSDR and MDPHHS programs, respectively) and had 125,792 person-years of follow-up. During the follow-up

period, there were 1,429 (17.8%) deaths. Table 1 compares characteristics of decedents with living cohort members; differences were statistically significant ($P < 0.05$) for each stratifying variable and show a greater proportion of decedents among men, smokers, and those with radiographic pleural or parenchymal abnormalities. In addition, the proportion of decedents increased monotonically with age and putative LAA exposure.

Table 2 shows SMRs and 95% confidence intervals (CI) for selected underlying causes of death. The cohort experienced fewer deaths from all causes and from all cancers than expected (SMR 0.86 [95% CI 0.82, 0.91] and 0.85 [95% CI 0.77, 0.94], respectively). In addition, SMRs for heart disease (0.75; 95% CI 0.67, 0.84), diabetes (0.68; 95% CI 0.47, 0.96), and breast cancer (0.63; 95% CI 0.37, 0.99) were also statistically lower than expected. Among causes of death associated with asbestos exposure, only mesothelioma and asbestosis were statistically elevated. Deaths resulting from transportation injuries of the motor vehicle driver were also statistically elevated (SMR 2.90; 95% CI 1.75, 4.53).

Also in Table 2, deaths falling under the NIOSH category “other musculoskeletal disease” were also statistically elevated (SMR 3.69; 95% CI 2.15, 5.91). Further examination showed 11 of 24 of these decedents had lupus, scleroderma, or RA as the underlying cause of death. The ad hoc, multiple-cause-of-death SMR for these three SAIDs combined was 4.30 (observed = 16, expected = 3.73; 95% CI 2.19, 6.40). RA predominated ($n = 9$), followed by scleroderma ($n = 5$) and SLE ($n = 2$).

Table 3 shows SMRs and SRRs for selected underlying causes of death stratified by asbestos exposure category. The lung cancer SRRs were statistically elevated among WRG workers (2.83; 95% CI 1.10, 7.25) and workers of other asbestos occupations (1.69; 95% CI 1.04, 2.75). The SMR for lung cancer among WRG workers was statistically elevated (1.77; 95% CI 1.09, 2.70), and among residents was statistically lower than expected (0.73; 95% CI 0.54, 0.98). When stratified by smoking status, there were only three lung cancer deaths among non-smokers and the SRR for smokers compared to non-smokers was 37.95 (95% CI 11.91, 120.94).

The SMR for mesothelioma was statistically elevated among WRG workers (25.58; 95% CI 12.34, 56.32) and residents (4.25; 95% CI 1.16, 10.89). The SRR for mesothelioma among WRG workers compared with residents was statistically elevated (15.12; 95% CI 3.32, 68.90).

The SRR for asbestosis was statistically elevated for WRG workers (2.42; 95% CI 1.11, 5.26) and household contacts (2.58; 95% CI 1.01, 6.58) compared with residents. Asbestosis SMRs were >80 for all exposure groups.

SMRs for the category “other musculoskeletal disease” were elevated for all exposure groups but reached statistical significance only among residents (3.96; 95% CI 1.90, 7.27). SRRs for this cause of death were not statistically significant.

Table 4 shows SMRs and SRRs for selected underlying causes of death stratified by radiographic abnormality. Compared with participants without pleural abnormalities, the SRR for lung cancer among those with pleural abnormalities was statistically elevated (1.73;

95% CI 1.15, 2.61). Similarly, the SRR for lung cancer among those with parenchymal abnormalities was also statistically elevated (3.94; 95% CI 2.10, 7.38). The SMR for lung cancer among participants with parenchymal abnormalities was statistically elevated (3.61; 95% CI 1.97, 6.06) and among participants without parenchymal abnormalities was statistically lower than expected (0.81; 95% CI 0.67, 0.98).

SRRs for mesothelioma were not statistically significant for either pleural or parenchymal abnormalities. Mesothelioma SMRs were statistically elevated for participants both with (8.27; 95% CI 3.04, 18.01) and without pleural abnormalities (4.18; 95% CI 1.68, 8.60). The mesothelioma SMR was statistically elevated for participants without parenchymal abnormalities (5.62; 95% CI 2.99, 9.61).

SRRs for asbestosis were statistically significant for participants both with pleural abnormalities (2.52; 95% CI 1.39, 8.93) and with parenchymal abnormalities (38.82; 95% CI 6.16, 241.24). Asbestosis SMRs were statistically elevated for participants both with (230.74; 95% CI 165.56, 313.03) and without pleural abnormalities (69.07; 95% CI 42.74, 105.58). Asbestosis SMRs were also statistically elevated for participants with (383.78; 95% CI 175.49, 728.53) and without parenchymal abnormalities (115.56, 95% CI 86.62, 151.27).

When the radiographic data for MDPHHS screening participants were excluded, SMRs and SRRs were similar to those in Table 4 (results not shown), suggesting that radiographic assessment of MDPHHS participants was robust.

DISCUSSION

These results represent the first mortality update of the Libby screening cohort, with more than 15 years of follow-up for participants in the initial round of screening (2000 to 2001). Not unexpectedly, given the numerous occupational and non-occupational asbestos exposure pathways that occurred in Libby, cohort deaths from asbestosis and mesothelioma were in excess compared with those of the United States. Despite excess deaths from these two causes, the number of deaths from all causes and all cancers as well as from heart disease, diabetes, and breast cancer were statistically lower than expected. This may be due to participation bias resulting from screening participants potentially having better general health, increased concern for their health, engagement in healthy lifestyle choices, and active pursuit of medical care and preventive treatment. Participation in the screening program also may have been a factor in improving mortality outcomes by increasing the likelihood of participants receiving follow-up health services (ie, secondary prevention). The other SMRs in Table 2 are largely consistent with those from a recent mortality survey of Libby residents, except for mesothelioma which was previously elevated but not statistically significant.[12]

Results stratified by exposure category suggest varying past asbestos exposures. For example, SRRs for mesothelioma indicate WRG workers had a 15-fold increase in risk compared with residents, although it should be noted the SMR among residents was also statistically elevated. Because mesothelioma can be used as a marker of past asbestos exposure,[13] the elevated mesothelioma SMR for residents suggests that residents in the

screening cohort may have had significant asbestos exposure. Similarly, the asbestosis SRRs were statistically elevated only for WRG workers and household contacts, although the SMR for each exposure category was extremely high. These extreme SMRs may be due in part to increased awareness of asbestos-related effects among Libby death certifiers compared with the rest of the country.[12] As evidence for this heightened awareness in Libby, screening participants who died in Montana were 2.6 times more likely to have asbestosis recorded as a cause of death compared with those that died in another state after adjusting for sex, age at death, and exposure category (95% CI 1.2, 5.8). When interpreting the SRRs for mesothelioma and asbestosis, it should be noted that SMRs among residents (the reference category) were statistically elevated, resulting in lower SRRs for the other exposure categories than would have occurred if in fact residents were unexposed.

Statistically significant SMRs for asbestosis and mesothelioma among residents in this study suggest relatively high exposures to LAA in that group. This observation, that LAA can affect persons without occupational exposure, is supported by results from at least one study of a cohort exposed to LAA outside of Libby. Libby vermiculite was widely disseminated to more than 250 sites across the United States where it was processed for commercial use.[14] In a study of community members living near such an urban site in Minneapolis, Minnesota, the prevalence of pleural abnormalities was 10.8% and correlated with metrics of asbestos exposure levels.[15] In another study of a sample of other processing sites, significant excesses of mesothelioma and pleural cancer cases and deaths were found at several sites, although that study could not rule out their association with occupational asbestos exposure.[14] However, in contrast with results from Libby, statistically significant excesses of asbestosis mortality were not found in that study.[14] LAA exposures have also been associated with pulmonary symptoms among persons with childhood exposure[16,17] and radiologic changes and pulmonary function decrements have been documented in Libby patients who would have been classified in the present study as residents.[18]

A significant finding from the 2000 to 2001 screening round was the prevalence of parenchymal and, especially, pleural abnormalities (0.8% and 17.8%, respectively).[3] The results from the present study show the presence of pleural abnormalities is associated with lung cancer and asbestosis mortality. Consistent with the results in Table 4, it has been established that parenchymal abnormalities are associated with an increased risk of lung cancer.[19] Additionally, the statistically significant SRR for pleural abnormalities and lung cancer in Table 4 is consistent with the finding by Pairon et al[20] that pleural plaque may be an independent risk factor for lung cancer mortality. Interestingly, both the presence and absence of parenchymal and pleural abnormalities at baseline screening were strongly associated with asbestosis mortality (Table 4). This suggests a high sensitivity and low specificity of these radiographic abnormalities for asbestosis mortality, which (again) may be related to heightened awareness of asbestos-related disease in the Libby community, as well as the diagnostic limitations of chest radiographs for asbestos-related findings.[4] It is also possible that parenchymal abnormalities for some asbestosis decedents became radiographically apparent only after they were screened.

Our finding of a strong association between smoking and lung cancer mortality is consistent with national US data in which 82% of lung cancer deaths, not including those associated

with secondhand smoke, are attributed to smoking.[21] Remaining lung cancer deaths are thought to be due to other causes, including environmental exposure to substances such as asbestos. However, the results from the present study do not distinguish the contribution of asbestos to lung cancer mortality. Of note, while the lung cancer SMR for WRG workers was statistically elevated (Table 3), the lung cancer SMR for the entire cohort showed a weak inverse and statistically insignificant association (Table 2).

During initial meetings with the community, ATSDR learned of concerns about a potential cluster of autoimmune disease among Libby residents. The existence of such a cluster and its association with asbestos exposure was supported by subsequent epidemiologic and laboratory investigations.[11,22] Among screening participants during 2000 to 2001, 494 (6.9%) reported SAID diagnosis compared with less than 1% expected based on national prevalence data.[11] This relative self-reported SAID prevalence is consistent with the SAID SMR (4.30) reported here. Noonan et al[11]found that in the screening cohort, self-reported SAIDs were associated with vermiculite exposure pathways. An increased frequency of positive antinuclear antibody tests among Libby residents further support this association.[22] In general, the cause of SAIDs is unknown, but may involve a gene–environment interaction in which an exposure to a sensitizing agent, such as LAA, triggers autoantibody production.[23]

Limitations of this study include our use of crude exposure categories, potential diagnostic bias (ie, death certificate certifiers in the Libby community may have been overly aware of asbestos-related disease), and the accuracy of death certificate data. We used broadly defined exposure categories without regard to length or intensity of LAA exposure at the person level. This may have obscured effects among participants with different exposure profiles within an exposure category,[24] particularly among household contacts and residents. Further, the lack of quantitative exposure data may have resulted in underestimates of associations. While the community, local health care providers and death certificate certifiers are likely very aware of past asbestos exposures in Libby, asbestos exposures may not be widely recognized nationwide. Combined, this may have contributed to the extremely high asbestosis SMRs observed here and in other mortality studies of Libby WRG workers and the community.[12,25,26] In general, there is evidence that cause of death accuracy is good for cancer on US death certificates (although there may be differences between tumor types).[27] However, death certificate causes of death may be less reliable for chronic, non-cancer diseases, particularly among older decedents with multiple diseases.[28]

In conclusion, the Libby screening cohort may be experiencing better-than-expected health with significantly fewer deaths than expected for all cancers, heart disease, diabetes, and breast cancer. However, as expected, SMRs for asbestosis and mesothelioma were statistically elevated. While WRG workers were the only exposure group with a statistically significantly elevated lung cancer SMR, all exposure groups had statistically elevated asbestosis SMRs and the mesothelioma SMRs for WRG workers and residents were also statistically elevated. Radiographic parenchymal abnormalities were associated with lung cancer mortality in this cohort. The SMR for SAIDs was statistically significant and supports results of studies indicating this disease class is associated with LAA exposure. A study of SAID incidence in this cohort should be considered to evaluate patient factors

associated with autoimmune disease. Future study of this cohort should refine exposure classifications, especially among those with residential-only exposure. These findings support the inclusion of lung cancer screening for asbestos-exposed populations, as is being done in the current Libby screening program using low-dose computed tomography (LDCT).[29]

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Clinical Significance:

In addition to excesses of asbestos-related mortality among screening participants with occupational asbestos exposure, excesses were also found among household contacts of workers and residents with no known occupational exposure. These results can be used by health care professionals treating patients with occupational or non-occupational asbestos exposure.

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TABLE 1.

Characteristics of the Screening Cohort

	Total, *n*	Alive, *n* (Row Proportion)	Deceased, *n* (Row Proportion)	*P* Value
All	8,043	6,614 (0.82)	1,429 (0.18)	–
Male	3,958	3,185 (0.80)	773 (0.20)	<0.0001
Female	4,085	3,429 (0.84)	656 (0.16)	
Age at baseline, yrs				
10–29	1,379	1,356 (0.98)	23 (0.02)	<0.0001
30–49	2,971	2,812 (0.95)	159 (0.05)	
50–59	1,703	1,458 (0.86)	245 (0.14)	
60–90	1,990	988 (0.50)	1,002 (0.50)	
Smoking status				
Never	4,054	3,640 (0.90)	414 (0.10)	<0.0001
Current/former	3,989	2,974 (0.75)	1,015 (0.25)	
Exposure				
WRG worker	413	272 (0.66)	141 (0.34)	<0.0001
Secondary contractor	453	335 (0.74)	118 (0.26)	
Other asbestos occupation	1,671	1,334 (0.80)	337 (0.20)	
Household contact	1,086	885 (0.81)	201 (0.19)	
Resident	4,420	3,788 (0.86)	632 (0.14)	
Pleural abnormality				
Yes	1,020	589 (0.58)	431 (0.42)	<0.0001
No	6,374	5,395 (0.85)	979 (0.15)	
No radiograph evaluated	649	630 (0.97)	19 (0.03)	
Parenchymal abnormality				
Yes	137	62 (0.45)	75 (0.55)	<0.0001
No	7,257	5,922 (0.82)	1,335 (0.18)	
No radiograph evaluated	649	630 (0.97)	19 (0.03)	

TABLE 2.

Standardized Mortality Ratios for Select Underlying Causes of Death in the Libby Screening Cohort, 2000 to 2016

	Observed	Expected	SMR (95% CI)
All causes	1,429	1,652.13	**0.86** (0.82, 0.91)
All cancers	382	448.60	**0.85** (0.77, 0.94)
Asbestos-related causes			
Lung cancer	123	137.73	0.89 (0.74, 1.07)
Asbestosis	62	0.48	**128.15** (98.25, 164.29)
Intestinal cancer	27	33.78	0.80 (0.53, 1.16)
Mesothelioma	15	2.41	**6.21** (3.48, 10.25)
Ovarian cancer	12	11.24	1.07 (0.55, 1.87)
Stomach cancer	5	7.95	0.63 (0.20, 1.47)
Rectal cancer	3	7.48	0.40 (0.08, 1.17)
Laryngeal cancer	2	3.12	0.64 (0.08, 2.31)
Pharyngeal cancer	1	3.05	0.33 (0.01, 1.83)
Other causes			
Heart disease	317	420.74	**0.75** (0.67, 0.84)
Diabetes	34	49.69	**0.68** (0.47, 0.96)
Injury, motor vehicle driver	19	6.55	**2.90** (1.75, 4.53)
Breast cancer	18	28.67	**0.63** (0.37, 0.99)
Other musculoskeletal disease *	17	4.60	**3.69** (2.15, 5.91)

Statistically significant (P<0.05) SMRs are in bold font. CI, confidence interval.

* More than 100 ICD-10 codes comprise this NIOSH cause-of-death category which excludes arthritis, spondylitis, osteomyelitis, and periotitis. It and other cause categories are described at https://www.cdc.gov/niosh/ltas/pdf/niosh-119-table-2007.pdf

TABLE 3.

Standardized Mortality Ratios and Standardized Rate Ratios for Select Underlying Causes of Death, Stratified by Asbestos Exposure Category

Cause	Exposure Category	Obs	Exp	SMR (95% CI)	SRR (95% CI)
Lung cancer	WRG worker	21	11.89	**1.77** (1.09, 2.70)	**2.83** (1.10, 7.25)
	Secondary contractor	6	12.15	0.49 (0.18, 1.07)	0.44 (0.19, 1.03)
	Other asbestos occupation	35	35.14	1.00 (0.69, 1.39)	**1.69** (1.04, 2.75)
	Household contact	15	15.83	0.95 (0.53, 1.56)	1.29 (0.56, 3.00)
	Resident	46	62.73	**0.73** (0.54, 0.98)	1
Mesothelioma	WRG worker	8	0.28	**28.58** (12.34, 56.32)	**15.12** (3.32, 68.90)
	Secondary contractor	0	0.28	0 (0.00, 13.10)	–
	Other asbestos occupation	3	0.73	4.12 (0.85, 12.03)	1.19 (0.26, 5.45)
	Household contact	0	0.18	0 (0.00, 20.18)	–
	Resident	4	0.94	**4.25** (1.16, 10.89)	1
Asbestosis	WRG worker	12	0.07	**179.79** (92.79, 314.08)	**2.42** (1.11, 5.26)
	Secondary contractor	8	0.06	**125.62** (54.24, 247.53)	2.10 (0.87, 5.06)
	Other asbestos occupation	13	0.16	**82.11** (43.68, 140.41)	1.16 (0.55, 2.46)
	Household contact	13	0.02	**558.46** (297.07, 955.05)	**2.58** (1.01, 6.58)
	Resident	16	0.17	**93.15** (53.21, 151.28)	1
Other musculoskeletal disease	WRG worker	1	0.23	4.27 (0.11, 23.78)	0.44 (0.06, 3.46)
	Secondary contractor	1	0.22	4.46 (0.11, 24.83)	0.43 (0.05, 3.39)
	Other asbestos occupation	3	0.87	3.44 (0.71, 10.07)	1.56 (0.42, 5.77)
	Household contact	2	0.75	2.68 (0.32, 9.68)	0.52 (0.11, 2.40)
	Resident	10	2.53	**3.96** (1.90, 7.27)	1

Statistically significant ($P < 0.05$) SMRs and SRRs are in bold font. CI, confidence interval; SMRs, standardized mortality ratios; SRRs, standardized rate ratios. Person-years for each exposure category: WRG worker 6,005; secondary contractor 6,821; other asbestos occupation 26,044; household contact 17,066; resident 69,855.

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TABLE 4.

Standardized Mortality Ratios and Standardized Rate Ratios for Select Underlying Causes of Death, Stratified by Presence of Pleural and Parenchymal Abnormalities on Chest Radiographs (CXR)

Cause	CXR Category	Obs	Exp	SMR (95% CI)	SRR (95% CI)
Lung cancer	Pleural	44	32.98	1.33 (0.97, 1.79)	**1.73** (1.15, 2.61)
	No pleural	78	104.01	**0.75** (0.59, 0.94)	1
	Parenchymal	14	3.88	**3.61** (1.97, 6.06)	**3.94** (2.10, 7.38)
	No parenchymal	108	133.11	**0.81** (0.67, 0.98)	1
Mesothelioma	Pleural	6	0.75	**8.27** (3.04, 18.01)	2.39 (0.15, 28.14)
	No pleural	7	1.68	**4.18** (1.68, 8.60)	1
	Parenchymal	0	0.09	0 (0.00, 41.75)	NR
	No parenchymal	13	2.31	**5.62** (2.99, 9.61)	1
Asbestosis	Pleural	41	0.18	**230.74** (165.56, 313.03)	**2.52** (1.39, 8.93)
	No pleural	21	0.3	**69.07** (42.74, 105.58)	1
	Parenchymal	9	0.02	**383.78** (175.49, 728.53)	**38.62** (6.16, 241.24)
	No parenchymal	53	0.45	**115.65** (86.62, 151.27)	1

Statistically significant ($P < 0.05$) SMRs and SRRs are in bold font. CI, confidence interval; SMRs, standardized mortality ratios; SRRs, standardized rate ratios. Person-years for each CXR category: pleural abnormality 14,162; no pleural abnormality 101,684; parenchymal abnormality 1,723; no parenchymal abnormality 114,123.