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RESEARCH ARTICLE

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# Case-fatality study of workers and residents with radiographic asbestos disease in Libby, Montana

Albert Miller MD[1] | Charles B. Black MD[2] | Gregory Loewen DO[3] | Curtis W. Noonan PhD[4] | Tracy McNew MPA[2] | Alan C. Whitehouse MD[5] | Arthur L. Frank MD, PhD[6]

[1]Icahn School of Medicine at Mount Sinai, New York, New York, USA

[2]Center for Asbestos Related Disease, Libby, Montana, USA

[3]Washington State University, Spokane, Washington, USA

[4]University of Montana, Missoula, Montana, USA

[5]Drexel University, Philadelphia, Pennsylvania, USA

[6]Department of Environmental and Occupational Health, Drexel University School of Public Health, Philadelphia, Pennsylvania, USA

**Correspondence**
Tracy McNew, MPA, Center for Asbestos Related Disease, 214 East 3rd St, Libby, MT 59923, USA.
Email: tracy@libbyasbestos.org

## Abstract

**Background:** Vermiculite ore from Libby, Montana contains on average 24% of a mixture of toxic and carcinogenic amphibole asbestiform fibers. These comprise primarily winchite (84%), with smaller quantities of richterite (11%) and tremolite (6%), which are together referred to as Libby amphibole (LA).

**Methods:** A total of 1883 individuals who were occupationally and/or environmentally exposed to LA and were diagnosed with asbestos-related pleuropulmonary disease (ARPPD) following participation in communitywide screening programs supported by the Agency for Toxic Substances and Disease Registry (ATSDR) and followed up at the Center for Asbestos Related Disease (CARD) between 2000 and 2010. There were 203 deaths of patients with sufficient records and radiographs. Best clinical and radiologic evidence was used to determine the cause of death, which was compared with death certificates.

**Results:** Asbestos-related mortality was 55% ($n$ = 112) in this series of 203 patients. Of the 203 deaths, 34 (17%) were from asbestos-related malignancy, 75 (37%) were from parenchymal asbestosis, often with pleural fibrosis, and 3 (1.5%) were from respiratory failure secondary to pleural thickening.

**Conclusions:** Asbestos is the leading cause of mortality following both occupational and nonoccupational exposure to LA in those with asbestos-related disease.

KEYWORDS

asbestos-related pleuropulmonary disease (ARPPD), case fatality, lamellar pleural thickening (LPT), Libby amphibole (LA), mortality, vermiculite

## 1 | INTRODUCTION

Libby, Montana represents a community of about 10,000. Mining of vermiculite ore seven miles northeast of Libby began in the 1920s, and W.R. Grace and Co. acquired the open cut mine and milling facilities from the Zonolite Company in 1963. Commercial operations continued until 1990. The processed vermiculite ore concentrate was loaded onto railroad cars and brought into the town of Libby, which is on the railroad line between Seattle and Minneapolis. W.R. Grace operated a vermiculite expansion plant, a bagging plant, and other

facilities near the railroad tracks in Libby. The material was also sent for further processing to over 240 sites in North America.

No serious health effects from uncontaminated vermiculite have been reported.[1] In contrast, the vermiculite originating from Libby was contaminated with naturally occurring amphibole asbestiform fibers. Tremolite was the only regulated asbestos fiber found in the raw vermiculite ore,[2] but in 2003, Meeker et al. determined that the Libby amphibole (LA) was composed of toxic nonregulated amphibole fibers, including winchite (84%), and richterite (11%), in addition to tremolite (6%).[3] Several occupations in the Libby area have been associated with mortality from exposure to LA, including occupations directly related to vermiculite mining and processing as well as other nonmining occupations.[4–6] Other local nonmining occupations in Libby resulted in high likelihoods of exposure to LA, including lumber mill workers.[7] Ward et al. (2012) documented large amounts of LA in tree bark when lumber was harvested from the local area, and simulation studies indicated high potential for exposure among loggers and mill workers.[8] At the lumber mill in Libby, LA-contaminated vermiculite was used in many ways, including as insulation on the top of plywood dryers.

A variety of historical nonoccupational LA exposure pathways have also been identified in the community. The town of Libby was chronically dusty from mining and processing operations. Air sampling by W.R. Grace and Co. in 1975 detected asbestiform fiber levels well above the then current Occupational Safety and Health Administration's occupational limit of 0.1 f/cm$^3$ over 8 h of exposure. Nonoccupational exposure pathways for LA also included home insulation as well as interaction with yards and parks where amphibole-containing vermiculite was supplied by the mine as a soil amendment.[7]

Diffuse pleural disease (DPD) is the signature radiographic abnormality in this patient group. Whitehouse, in 2004, reported on the radiographic findings and pulmonary function in 123 miners, family members of miners and residents and found a significant decline in pulmonary function.[9] In this study, 55% of patients with LA disease had only pleural disease on chest X-ray, and most of the remaining patients had minimal interstitial disease. The average annual loss was 2.2% for forced vital capacity (FVC), 2.3% for total lung capacity (TLC), and 3.0% for carbon monoxide diffusing capacity (DLCO). The rates of pulmonary function loss were the same in those with DPD with or without interstitial changes. Peipins et al. also reported that on chest radiographs, pleural abnormalities were present in 17.8% of the 6688 participants exposed to LA, and the frequency of pleural disease increased with exposure intensity.[10]

In 2002, the U.S. Environmental Protection Agency (EPA) placed the Libby Asbestos Superfund site on their National Priorities List for cleanup due to high levels of LA in and around the area. In 2009, the EPA declared Libby a Public Health Emergency, the first and only such public health emergency in the United States. The Center for Asbestos Related Disease (CARD) was established in Libby in 2000 as part of the local hospital, in coordination with the Agency for Toxic Substances and Disease Registry's (ATSDR) health screening program, to follow identified asbestos-related abnormalities. CARD later took on the comprehensive community survey initiated by ATSDR in 2000 and continued by Montana state public health officials from 2003 to 2009. In 2009, computed tomography (CT) was integrated into this screening in addition to plain X-rays. It is recognized that CT detects both pleural and parenchymal abnormalities often undetected by plain X-ray. As of 2010, physicians at CARD had diagnosed asbestos-related pleuropulmonary disease (ARPPD) in 1883 persons (47%) out of the 4028 subjects seen at the clinic. Epidemiological observations of elevated mortality, even with nonoccupational exposure, led to this investigation. The purpose of this study is to provide a more detailed clinical description of the mortality observed in both workers and nonworkers exposed to LA who were CARD enrollees.

## 2 | METHODS

### 2.1 | Exposure

LA exposure histories were categorized as occupational (mine and mine-related workers or lumber mill workers), and nonoccupational (families of workers, and community members exposed to LA while living in Libby). Exposure was not quantified.

### 2.2 | Sources of mortality data

All persons in the study were seen at least once at CARD between July 1, 2000 and February 22, 2010. Those included in this study were diagnosed with ARPPD from LA exposure and died by February 22, 2010. CARD was informed of deaths occurring both in the community and nationwide by obituary notices in the local newspapers for current and past area residents, by families and friends of patients who had moved but retained close ties to the community, and by routine follow-up telephone calls from the clinic. During this time period, 237 patient deaths were reported. February 22, 2010 was an arbitrary cutoff date to allow for the collection of death certificates and medical records. Of the 237 patient deaths, 20 were excluded because the death certificates were unobtainable, and 14 had insufficient medical records available for review. The remaining 203 deaths are included in this report. For each case, the medical records, death certificates, and most recent available chest radiographs and chest CT scans were reviewed.

### 2.3 | Causes of mortality

Asbestos-related cancer deaths were based on a review of pathology reports for mesothelioma, lung cancer, laryngeal cancer, and ovarian cancer per the International Agency for Research on Cancer (IARC).[11]

The methods of Selikoff and Seidman were used for determining best evidence of an asbestos death.[12] Death certificates from all states were obtained through public records. Clinical charts, radiographs, and deidentified data were provided in compliance with requirements of the Health Insurance Portability and Accountability Act. Institutional review board (IRB) exemption was obtained for this mortality study.

After all initial data were recorded, two investigators reviewed all radiographs and records of the nonmalignant asbestos-related deaths and concurred on all deaths using death certificates, radiographs, pulmonary function tests (PFT), and clinical findings. Each case was carefully reviewed and a cause of death was determined for all of the 203 subjects in this study based on clinical and radiographic data. Deaths not related to malignancy were designated as asbestos-related mortality when ARPPD was visible on chest radiography (diffuse or circumscribed pleural thickening and/or interstitial disease) AND asbestos-related death was recorded on a death certificate, OR when the review of the medical records, radiographs, and pulmonary function provided best evidence for an asbestos-related death. Many death certificates seemed to be inaccurate, typically because they were unwitnessed deaths at home and the coroner attributed death to arteriosclerotic heart disease even if there was no clinical evidence of heart disease. Twenty-five of the 203 death certificates were completed by nonphysician coroners or undertakers. Deaths were recorded by investigators as either (1) asbestos deaths per death certificate; (2) asbestos deaths by best evidence; or (3) nonasbestos deaths, even though asbestos changes were present on radiograph.

## 3 | RESULTS

Of 203 deaths, 77 were asbestos-related by death certificate and were confirmed by chart review and chest radiography. An additional 35 deaths were determined to be asbestos-related from best evidence, based on medical records and radiograph review. The overall asbestos-related mortality was 55% ($n = 112$) in this cohort of 203 patients. Of these 112 asbestos-related deaths, 78 proved to be from asbestosis or asbestos-related pleural fibrosis, and 34 were from asbestos-related cancers. Asbestos-related cancers included 4 mesotheliomas, 26 lung cancers, 1 laryngeal cancer, and 3 ovarian cancers. Of the 78 nonmalignant deaths, 45 had asbestosis noted on their death certificates and 33 did not, but all died of asbestos-related disease by best evidence, based on radiographs, pulmonary function, and chart review. Although asbestos-related mortality for cancer was greater in smokers than in nonsmokers (20% vs. 7%), mortality was virtually identical for asbestosis/pleural fibrosis (38% vs. 39%) as is shown in Table 1.

Exposures to LA are divided into occupational ($n = 103$) and nonoccupational ($n = 100$) histories (Table 1). Occupational history included WR Grace mine and processing workers, lumber mill workers, and those who worked in both. Nonoccupational history included family members of workers ($n = 22$), and community members exposed to asbestos prevalent in the environment as home insulation, in the air from nearby mining and processing operations, and from lawn and garden maintenance ($n = 78$). Compared with those with a history of occupational exposure, those with nonoccupational exposure had a greater proportion of deaths attributed to nonasbestos disease (53% vs. 37%). Nevertheless, 47 of the 100 deaths among ARPPD patients with nonoccupational exposure history were asbestos-related deaths.

Table 1 shows the demographics and disease results of all 203 subjects. More than three quarters (77%) of the subjects were current or former smokers. Mine workers were considered to have the highest exposures to LA, with lumber mill workers less, and

**TABLE 1** Demographics and exposure characteristics of 203 deceased patients with radiographic findings of asbestos-related pleuropulmonary disease

| | | | Asbestos-related deaths | |
|---|---|---|---|---|
| | Total deaths | Nonasbestos deaths | Asbestosis/ pleural fibrosis | Asbestos-related cancer |
| Gender | | | | |
| Male | 135 (67%) | 61 (45%) | 54 (40%) | 20 (15%) |
| Female | 68 (33%) | 30 (44%) | 24 (35%) | 14 (21%) |
| Smoking history | | | | |
| Current or Former Smoker | 157 (77%) | 66 (42%) | 60 (38%) | 31 (20%) |
| Smoked until last year | 31 | 9 | 13 | 9 |
| Former smoker | 126 | 44 | 52 | 30 |
| Nonsmoker | 46 (23%) | 25 (54%) | 18 (39%) | 3 (7%) |
| Exposure History | | | | |
| Occupational Exposures (mine workers, mill workers, and both) | 103 (51%) | 38 (37%) | 48 (47%) | 17 (17%) |
| Nonoccupational Exposures (community members and families of workers) | 100 (49%) | 53 (53%) | 30 (30%) | 17 (17%) |
| Total number | 203 | 91 (45%) | 78 (38%) | 34 (17%) |

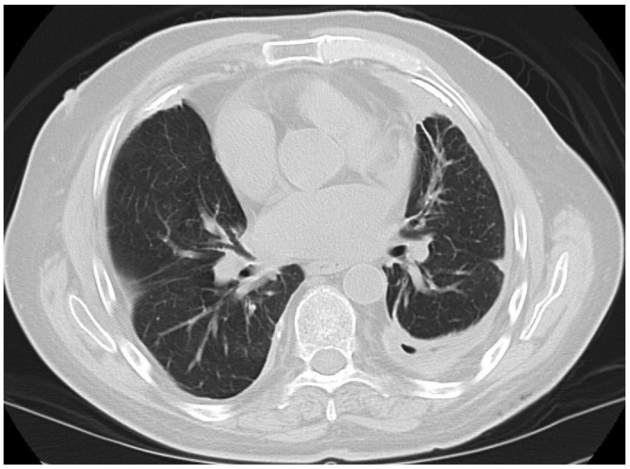

**FIGURE 1** CT findings in a 70-year-old man who died from ventilatory failure secondary to pleural disease: LPT with calcification is seen, more prominent on the left side. Note the decreased volume of the left hemithorax. FVC 54% predicted,[13] FEV1 48% predicted,[13] Ratio 0.65, DLCO 75% predicted.[14] CT, computed tomography; DLCO, carbon monoxide diffusing capacity; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; LPT, lamellar pleural thickening

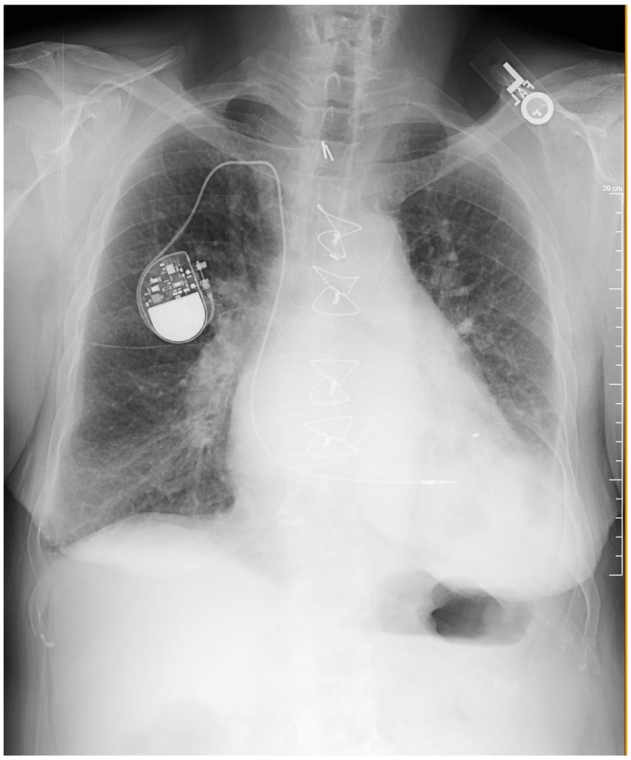

**FIGURE 2** Chest X-ray of a 77-year-old female who died from ventilatory failure secondary to pleural disease. Note bilateral LPT less obvious than seen in Figure 1, although restrictive spirometric impairment was greater. FVC 38% predicted,[13] FEV1 34% predicted,[13] Ratio 0.71. DLCO was 22% predicted.[14] It is not unusual for the severity of impairment to be greater than the extent of pleural thickening on imaging. Sternotomy wires, pacemaker, and slight cardiomegaly are noted. DLCO, carbon monoxide diffusing capacity; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; LPT, lamellar pleural thickening

community members the least,[7] but actual quantification of exposures in terms of airborne fibers has not been established.

The vast majority (75/78) of nonmalignant asbestos-related deaths were from a mixture of interstitial pulmonary fibrosis (asbestosis) and pleural fibrosis. The latter included typical circumscribed and diffuse thickening, and a progressive diffuse thickening which was described as lamellar. Two deaths were from lamellar pleural thickening (LPT) (Figures 1 and 2) and one death was from extensive circumscribed pleural thickening (Figure 3) without significant interstitial disease on plain chest radiograph or CT scan. These cases demonstrated severe restrictive ventilatory and diffusion impairment.

## 4 | DISCUSSION

The present series was limited to patients who had been evaluated at CARD in Libby and found to have radiographic evidence of asbestos-related disease. Our findings confirm that approximately half of the deaths observed following both occupational (64%) and community-wide nonoccupational exposure (47%) to LA are from asbestos-related diseases. Pathological evaluation of a Libby miner[15] and a community resident[16] have confirmed the presence of LA in both of their lungs.

This case fatality series of nonmalignant and malignant asbestos-related deaths in those with radiographic evidence of nonmalignant disease shares some similarities with two iconic asbestos-exposed cohorts, the insulator worker cohort and the miners and residents of Wittenoom, Australia. The Libby cohort included a mixture of residents occupationally- and environmentally exposed to LA, whereas the insulator workers were exposed primarily to chrysotile. As with the Libby cohort that included individuals with radiographic ARPPD, almost all of the insulator workers showed asbestos-related changes on plain X-ray.[17] The Wittenoom, Australia cohort was exposed to crocidolite rather than LA, and cohort entry was not based on radiographic ARPPD.[18] Similar to the Libby cohort, however, the Wittenoom cohort includes a mix of occupationally- and residentially exposed individuals. The 30% mortality from nonmalignant asbestos-related disease in nonoccupationally exposed Libby residents was remarkably greater than the insulators' (8.6%) and the Wittenoom workers' (2.1%) or residents (0.2%).[17,18]

The clinically documented best evidence approach for assessment of mortality cause in this study is a notable strength compared with other mortality studies in this population that have relied on death certificate to determine the cause of death. Nevertheless, our findings are consistent with prior findings of high mortality risk from asbestos-related causes in this population.[19] McDonald et al. studied mortality in 406 mine workers to find elevated standard mortality ratios (SMR) for lung cancer (SMR = 2.40, confidence interval 95% CI: 1.74–3.22) and nonmalignant respiratory disease (SMR = 3.09,

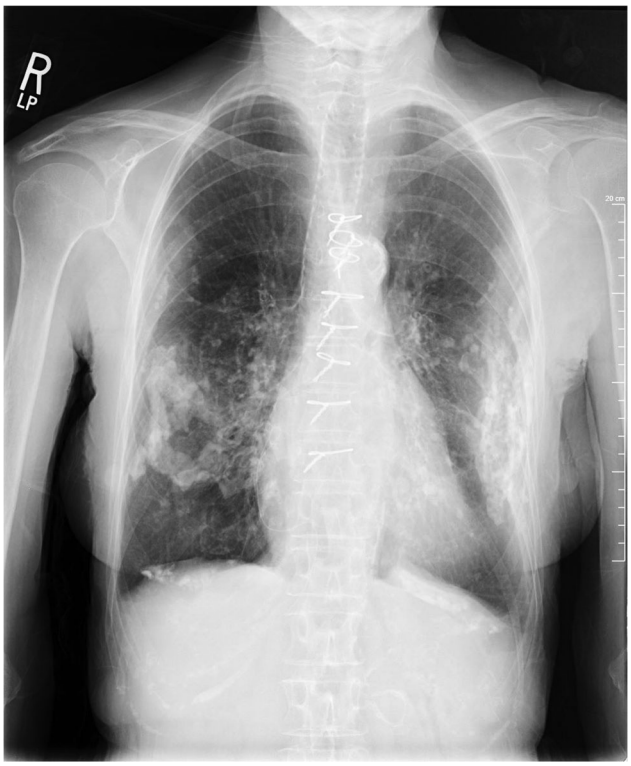


**FIGURE 3** Chest X-ray of a 72-year-old female who died from ventilatory failure secondary to pleural disease. Extensive bilateral circumscribed pleural thickening is seen. FVC 45% predicted,[13] FEV1 56% predicted,[13] ratio 0.94, DLCO 34% predicted.[14] DLCO, carbon monoxide diffusing capacity; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity

95% CI: 2.30–4.06).[4] Sullivan looked at a larger cohort of 1672 Libby miners, millers, and processors and found elevated SMRs for asbestosis (SMR = 165.8, 95% CI: 103.9–251.1), lung cancer (SMR = 1.7 95% CI 1.4–2.1), and mesothelioma (SMR 15.1, 95% CI 1.8–54.4).[5] Our series includes three patients who died from thin lamellar or extensive circumscribed pleural thickening, without significant interstitial findings, although two of the three had decreased DLCO. This pleural fibrosis is different both from the frequently observed circumscribed pleural thickening, and from the less common diffuse pleural thickening that has been described with other asbestos exposures. The two deaths from LPT included one male (occupational exposure) and one female (nonoccupational exposure). The exposure of the patient with extensive circumscribed pleural thickening was nonoccupational.

The rapidly advancing LPT associated with winchite, richterite, and tremolite exposure has been previously reported in this population, and this distinctive pleural disease results in pleuritic chest pain with progressive decline in pulmonary function.[20,21] Progression to respiratory failure and death has also been reported from the diffuse pleural thickening associated with conventional asbestos exposure, but is far less common.[22] It is very rare even with extensive circumscribed pleural thickening although ventilatory impairment is proportional to extent of pleural thickening.[23]

In the early stages, LPT is thin and difficult to discern on CT, but it becomes thicker and often calcifies. Half (96/198) of the subjects who had grown up in Libby and then left 30 years previously (The Pre-Adult Latency Study), showed pleural thickening on CT. In half of these (47/96), the pleural thickening was lamellar.[24] The LPT then is often accompanied by progressive loss of pulmonary function and pleuritic pain. The DPD associated with chrysotile exposure is much thicker, resembling the fibrothorax following empyema or pneumothorax therapy, and often follows an acute pleural effusion.[22] Diffuse pleural thickening generally is an uncommon feature of chrysotile asbestos exposure, but is more frequent following amphibole exposure.[25] Diffuse pleural thickening is described as a frequent feature of crocidolite amphibole disease seen in the Wittenoom population.[26]

The majority of our cohort were smokers, and the frequency of lung cancer in particular is consistent with the established interaction between tobacco smoke and asbestos. That mortality from nonmalignant asbestos-related causes was similar in smokers (38%) and nonsmokers (39%) is surprising given the greater frequency and severity of asbestosis in long-term insulators who smoked.[27] This may reflect the greater frequency and severity of the lamellar pleural fibrosis characteristic of LA.

Disease from nonoccupational exposure to asbestos has long been recognized.[28] Individuals in our series (with the exception of the miners) were all believed to have much lower fiber exposure, which underscores the apparent toxicity of winchite and richterite in combination with small amounts of tremolite. Roughly 100,000 tons of LA contaminated vermiculite left Libby and was processed in over 245 domestic sites across North America.[29] This toxic material was then widely distributed for multiple applications including commercial and domestic insulation.[30] LA has been attributed to outbreaks of mesothelioma[31] and pleural thickening was seen in both Ohio and Minnesota vermiculite processing workers.[32,33] Whitehouse reported a series of 31 cases of mesothelioma from his Libby practice.[34] Because the nonmalignant radiographic manifestations of LA are subtler, it is likely that this disease is under-recognized and underreported. Similar naturally occurring deposits of amphibole, containing winchite, richterite, and actinolite have been identified elsewhere, including southern Nevada, resulting in excess mesothelioma cases.[35] Airborne fibers of winchite and richterite in that area have the potential to cause exposures to large populations in the Las Vegas area. For these reasons, the mortality associated with LA disease in our series is relevant to other geologic amphibole exposures. Our findings expand clinical and epidemiological understanding of the elevated mortality following LA exposure and support that asbestos regulation should be expanded to include winchite and richterite fibers.

There are several limitations of this report. It is a case fatality study of patients who presented to a widely known survey open to the entire community. The CARD clinic was the only source of pulmonary specialty care within 80 miles, and attracted patients who were within this distance during the years of this study, and who may have been more likely to have pulmonary symptoms. Although asymptomatic patients with minimal or no disease who would

decrease the observed mortality may have been less likely to be included, our observation of such a high percentage of deaths from asbestosis and from pleural disease underscores the toxicity of LA. The original method of screening for ARPPD used chest radiographs read by B-readers. While this strategy was the only practical approach to this public health problem at the time, it is acknowledged that the subtle pleural disease seen in these patients is easily missed on the plain chest radiograph. For this reason, it is likely that the 1883 patients diagnosed with ARPPD represent an underestimate of frequency of LA disease in exposed individuals.

The strengths of our study include (1) logistical and financial support by the ATSDR and the National Institutes of Health (NIH); (2) involvement of the workers and community; (3) detailed documentation of causes of death beyond the death certificate; (4) demonstration of malignant (17%) and nonmalignant (30%) asbestos-related mortality in nonoccupationally exposed residents of the community; and (5) a large proportion of malignant (21%) and nonmalignant (35%) asbestos-related mortality among females, a population that is largely excluded from asbestos exposure research. These strengths were cited by Larsen of the ATSDR and Williamson of the Montana Department of Public Health and Health Services.[36]

Asbestos is a principal cause of mortality for those who are exposed to the LA mixture of winchite, richterite, and tremolite, both in occupational and nonoccupational settings. Our findings provide clinical detail for the elevation of asbestos-related mortality observed in Libby, Montana from multiple studies. Continued follow-up of this at-risk population for best evidence evaluation of mortality after 2010 is warranted.

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## CONFLICTS OF INTEREST

Authors Alan C. Whitehouse, Arthur L. Frank, Gregory Loewen, Albert Miller, and Charles B. Black have provided expert medical consultative services for several law firms in behalf of CARD or for other asbestos disease patients.

## DISCLOSURE BY AJIM EDITOR OF RECORD

John Meyer declares that he has no conflict of interest in the review and publication decision regarding this article.

## AUTHORS CONTRIBUTIONS

Doctors Alan C. Whitehouse, Arthur L. Frank, and Charles B. Black participated in the conception and design of the work. All authors participated in the acquisition, analysis, and interpretation of the data for this manuscript, drafting or revising it critically for important intellectual content, and final approval of the version to be published. All authors also agree to be accountable for all aspects of the work.

## DATA AVAILABILITY STATEMENT

Due to its private nature, supporting data can only be made available in a deidentified manner to bona fide researchers. Details of the data and how to request access are available from the Center for Asbestos Related Disease.

## ETHICS APPROVAL AND INFORMED CONSENT

An IRB exemption was obtained from the Providence Health Care Institutional Review Board in Spokane, Washington for this project.

## ORCID

*Gregory Loewen* http://orcid.org/0000-0002-7185-0796
*Tracy McNew* http://orcid.org/0000-0002-4302-1114

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