A clinical assessment and lung tissue burden from an individual who worked as a Libby vermiculite miner

Brad Black, Ronald F. Dodson, James R. Bruce, Lee W. Poye, Claudia Henschke & Gregory Loewen

To cite this article: Brad Black, Ronald F. Dodson, James R. Bruce, Lee W. Poye, Claudia Henschke & Gregory Loewen (2017) A clinical assessment and lung tissue burden from an individual who worked as a Libby vermiculite miner, Inhalation Toxicology, 29:9, 404-413, DOI: 10.1080/08958378.2017.1372536

To link to this article: http://dx.doi.org/10.1080/08958378.2017.1372536

Published online: 17 Oct 2017.

Submit your article to this journal

Article views: 85

View related articles

View Crossmark data
A clinical assessment and lung tissue burden from an individual who worked as a Libby vermiculite miner

Brad Black, Ronald F. Dodson, James R. Bruce, Lee W. Poye, Claudia Henschke and Gregory Loewen

ABSTRACT
During its days of operation (1920s–1990), the world's largest source of vermiculite was extracted from a mine located near Libby, Montana. The material mined at this site was shipped for various commercial applications to numerous sites in the United States. There was a “fibrous” component with toxic potential within the vermiculite deposit that has resulted in “asbestos-like” diseases/deaths being reported in numerous studies involving miners as well as residents of the town of Libby. The present case involves the clinical assessments of an individual who worked at the mine from 1969 to 1990. He had no other known occupational exposures to fibrous materials. He developed a clinical picture that included “asbestos-like” pathological features and eventually an adenocarcinoma. The clinical assessment including radiographic features will be presented. The evaluation will also include the analytical evaluation of the fibrous/ferruginous body composition of the lung tissue. This is to our knowledge the first time such an extensive evaluation has been conducted in a vermiculite miner from Libby, Montana.

Introduction
The Rainy Creek mineral complex near Libby, Montana was defined by Meeker and associates (2003) as containing amphibole-rich rock within the largest mined vermiculite deposit in the world. The mine and mill near Libby, Montana operated from the early 1920s until 1990 (Antao et al., 2012). The significance of the volume of vermiculite mined from this source can be appreciated from an EPA document stating 80% of the world’s supply of vermiculite came from this source (U.S. Environmental Protection Agency, 2006). There were many commercial applications for the vermiculite as described by Antao and associates (Antao et al., 2012) and Lockey et al. (Lockey et al., 1984). According to Horton and colleagues (Horton et al., 2006), the vermiculite mined in the Libby facility was shipped from the Libby mine to approximately 200 domestic processing and receiving facilities in different cities in the United States. Fibrous dust from the vermiculite potentially provided exposure to workers in the facilities, as well as those living around the facilities and consumers who used the products containing the vermiculite.

Historically, individuals exposed to vermiculite dust as miners and millers, as well as people living in the community of Libby, have developed diseases consistent with those associated with exposure to asbestos, as documented in clinical reports such as reported by Peipins et al. (2003) and Whitehouse et al. (2016). The toxic fibrous component of the vermiculite inducing diseases evolved based on changing of the mineralogical classification of the elongated particulates. The original asbestiform structures were defined in several articles as “tremolite asbestos.” Lockey et al. (1984) discussed the pulmonary changes associated with vermiculite containing “tremolite.” These pathological changes included benign pleural effusion, as well as other radiographic findings in the lungs.

McDonald and associates (1985a) also reported radiographic findings in the lungs and “probability” of exposure resulting in pleural thickening. These findings were attributed to tremolite exposure within the mined vermiculite. A companion study of individuals who had worked as vermiculite miners (thus exposed to “tremolite”) was reported by the same group (McDonald et al. 1985b). Their conclusions were that there was higher mortality from vermiculite mining in the Libby cohort than was observed in the cohort of individuals exposed to chrysotile mining (where tremolite was a component of the mined material). Interestingly, in this paper, an observation was made that the elemental chemical analysis of fibers by energy dispersive spectrometry (EDS) indicated some fibers were compatible in elemental composition with tremolite–actinolite series; however, the authors reported that the sodium-rich fibers often displayed no calcium response (potentially being magnesioriebeckite) or intermediate calcium response suggesting richterite.

The suggestions that “asbestos” content of the vermiculite was causing the “asbestos type” diseases seemed reasonable until Meeker (2009) discussed a historical perspective of the
nomenclature for the fibrous composition of vermiculite. Meeker found only a limited component of the fiber content within the Libby amphiboles that met the “regulated term for asbestos” which was tremolite in an asbestiform habit. Under Occupational Safety and Health Administration (OSHA) definitions (1994) and Asbestos Hazard Emergency Response Act (AHERA) definitions (1987), the regulated structures refer only to fibrous forms of the tremolite, actinolite, amosite, anthophyllite, crocidolite and chrysotile. National Emission Standards for Hazardous Air Pollutants (NESHAP, 1990) defines asbestos slightly more specifically, stating that the regulated form of the same six minerals is designated as “asbestiform,” indicating a crystalline habit defining the morphological growth pattern of the crystals. According to Meeker (2009), the evolving classification of the asbestos fiber material in the vermiculite formation was impacted by the International Mineralogical Association’s revised recommendation for amphibole minerals. In this reclassification, much of the previously defined “tremolite” in Libby materials was changed to winchite. This mineral is not a regulated entity under the definition of asbestos based on previously defined asbestos types in federal regulations. The actual tremolite component of the vermiculite samples was agreed upon in the legal process involving the mine and was determined to be less than 10%. (Meeker, 2009).

There is no doubt that exposure to “Libby amphiboles” is responsible for the development of asbestos-type diseases in miners, processors and the nonoccupationally exposed population of Libby. To our knowledge, however, there is no data as to the composition and numbers of fibrous particulates in the lung tissue of actual miners. In fact, the only assessment of which we are aware that evaluates material derived secondarily from the lung tissue was published by Sebastien et al. in the form of evaluation of the presence of “asbestos bodies” within the samples of sputa (1988). Sebastien and associates (1988) state that 128 of 170 subjects produced specimens which had identifiable “asbestos bodies” which they attribute to tremolite exposure of the cohort.

Obviously, from the above data, we would have preferred to use the term “ferruginous bodies,” since the potential of the cores being formed on tremolite in this exposure is small since tremolite is now recognized to constitute a minority fibrous component of the elongated component of the vermiculite and actual core definition requires the use of Analytical Transmission Electron Microscopy. Ferruginous bodies, if meeting specific features, can be considered “as consistent with the morphological features of asbestos cored ferruginous bodies” when seen by optical microscopy, but one cannot conclusively define the core material except in generalities (as will be discussed in detail in our findings in this case).

The content of the tissue from the left lower lung described in this report was also referred to the laboratory of one author (RFD) for assessment of both fiber and ferruginous body burden as determined by combined light microscopy of tissue sections of lung parenchyma for the presence of ferruginous bodies as well as optical and transmission electron microscopy evaluation of digested tissue for the presence of ferruginous bodies and uncoated fibers. The attending physicians had received authorization to utilize the information for this publication from the family of the deceased. The mechanisms by which inhaled elongated particulates induce disease have been covered extensively in two special editions of the Journal of Toxicology and Environmental Health; part B (2010 and 2016). The present paper will not therefore discuss the mechanisms and refer the reader to the aforementioned editions.

**Clinical history**

**Background**

The Center for Asbestos Related Disease, established in July, 2000 in Libby, Montana, has provided ongoing asbestos health screening for Libby amphibole exposure within Lincoln County, Montana. Asbestos screening is currently funded by a CDC grant. Based on the most recent NIOSH report, Lincoln County experiences the highest age-adjusted rate of asbestosis mortality in the United States through 2009. In addition, lung cancer rates have been 20–30% higher than the Montana rate excluding the Libby area (ATSDR Final Public Health Assessment, 2003). Beginning in 2013, the grant began funding for low-dose CT (LDCT)-based lung cancer screening in the highest risk members of the CARD Clinic population. Patients between ages 55–84 years with asbestos related disease (ARD) and at least a 20 pack-year history of smoking were offered LDCT. The patient in this case report was identified with early cancer (Figure 1) in this screening program, allowing for the opportunity to obtain tissue at surgery for fiber analysis.

**Clinical description**

The case involves a 74-year-old former resident who had moved to Libby, Montana in 1969 and worked as a millwright at the vermiculite mine until the mine closure in 1990. He moved out of the area in 1991 and experienced no...
other known fiber exposure. His duties did not involve work with boilers or steam pipes, and he had no known appreciable exposure to other fibrous material. His occupational exposure was estimated at 64 fiber-years by WR Grace Company documentation (Alpha Employer History List, 11 April 1986). He also had a smoking history of 34 pack-years, having quit in 1984. He developed increasing dyspnea on exertion (DOE) and was evaluated in 1988 by a pulmonologist and diagnosed with ARD and asthma. CXR revealed right pleural thickening and left lower lobe interstitial fibrosis. Pulmonary functions were reported as normal. In 1993, he presented with left-sided pleuritic chest pain of more than two months duration, considered secondary to pleural inflammation. Evaluation at that time included a HRCT which showed pleural thickening but no suggestion of mesothelioma. At follow-up evaluation in 2004, he continued to experience increased DOE and left lateral pleuritic chest pain associated with a pleural rub on examination. The pleuritic chest pain and DOE persisted, and intensified in 2008, prompting an emergency room evaluation and cardiac catheterization which ruled out a cardiac basis for his chest pain. Chest CT at that time revealed early lamellar (thin layer) pleural thickening, primarily in the posterior lung bases, which typifies the subtle pleural fibrosis observed in Libby amphibole exposure, that is thin, noncalcified and easily escapes recognition. In 2011, he developed increasing right posterolateral chest pain. A HRCT study revealed bilateral lamellar pleural thickening (Figure 2). There was no evidence of mesothelioma. Pain management was episodic, as needed, for more severe thoracic pain.

In 2013, lung cancer screening detected a new, solid nodule which had grown on the follow-up CT scan in 2014 (Figure 1). Pulmonary function tests at the time were normal.

Percutaneous needle biopsy revealed invasive adenocarcinoma, and PET imaging (not shown) suggested Stage IA adenocarcinoma of left lower lung. The patient was referred for lung resection, and subsequently underwent VATS lobectomy.

Direct inspection of the pleural space at surgery revealed a thin, white parietal pleural layer of fibrotic tissue (Figure 3). Pleural thickening was not appreciated on the visceral pleura. Histology of pleural biopsy confirmed the presence of pleural fibrosis. Left lower lobectomy was completed, and the patient was discharged postoperatively without incident.

Surgical pathologic examination confirmed the diagnosis of stage IA adenocarcinoma of the lung (T1N0M0).

The patient was followed for two years following lung resection and remained free of recurrent disease from lung cancer. He was later hospitalized for peritonitis and died from complications that arose from sequelae of sepsis. Thus, his official cause of death was determined to be nonasbestos related.

Methods

Methods and materials – morphological evaluation and tissue burden analysis

A slide consisting of approximately a 6 cm² section of lung parenchyma was evaluated on an H and E prepared slide by two of the authors (RFD and JRB) for the general morphology of the tissue, as well as the presence of particulates and/or ferruginous bodies.

The tissue block and companion H&E stained slide was submitted to the laboratory of one author (RFD) for the evaluation of the sample for potential usefulness for tissue digestion in order to determine the presence of ferruginous bodies by light microscopy. The tissue was considered appropriate for digestion. The second charge was to evaluate the digested material for the presence of ferruginous bodies, compositions as to the cores of ferruginous bodies, and determine the presence, types and numbers of uncoated fibers by analytical transmission electron microscopy (ATEM). The tissue on the slide defined as “left lower lobe of the lung” consisted of two pieces of parenchymal tissue. The section of lung tissue was scanned by light microscopy and numbers of classical ferruginous bodies were found throughout the section by both readers (RFD and JRB). Approximately one half of each piece of tissue on the companion block was dissected for processing. The pieces of lung tissue were processed by a modified bleach digestion procedure (Williams et al., 1982).

The digestion pool for the left lung sample contained 0.4242 g wet de-paraffinized-weight of tissue. The digestate was collected on either 0.22 μm pored mixed cellulose ester
(MCE) filters for analysis by light microscopy for the presence of ferruginous bodies or on 0.2 μm polycarbonate filters (PC) for analysis in the analytical transmission electron microscope (ATEM) for the presence of uncoated fibers and core identification of ferruginous bodies if such a structure was observed.

A wedge of the MCE filter was mounted on a glass slide, cleared using an acetone vapor and then scanned by light microscopy at 200x to 400x in an American Optical (AO) light microscope. If a structure was found by light microscopy that conformed morphologically to an asbestos cored ferruginous body, then that entity was referred to as a classical ferruginous body. The cores of such structures cannot be further identified by light microscopy.

Strips of the carbon-coated PC filters were collected on 100-mesh copper marker grids and the filter matrix was dissolved using chloroform via the use of a Jaffee wick method. This resulted in the production of a carbon replica on the grids which could be analyzed by ATEM for fibrous particulates and ferruginous body content. The fibrous particulate/ferruginous body burden was extremely heavy which necessitated the use of a filter with a lower concentration of digestate than normally selected. Five randomly selected grid squares on three grids were evaluated at a 15,000x magnification in a JEOL 1200EX ATEM for the presence of fibrous particulates and ferruginous bodies. An elongated particle to be analyzed consisted of a fibrous structure that has an aspect ratio of greater than or equal to 5:1, substantially parallel sides for most of the length and a length of greater than or equal to 0.5 μm. The dimensions and definition chosen for a “fiber” is consistent with that definition of the count scheme under AHERA-1987 for those evaluations where ATEM is designated. The higher magnification evaluation of the tissue burden by this ATEM using this definition permits inclusion of both short and/or thin elongated particulates that are often the majority of the elongated structures seen in both tissue burdens (Dodson, 2011) and air samples (Langer et al., 1991). Any structures meeting the described definition as a fiber or cores of ferruginous bodies were evaluated as to crystalline characteristics by selected area electron diffraction (SAED) as is necessary for confirming an amphibole pattern and elemental composition by energy-dispersive spectroscopy. The elemental composition of the elongated particulates was determined by IXRF Systems IXRF analyzer model 550i containing EDS 2008 software and an AAT light element detector with 144-eV resolution. A second analysis was conducted at 2000x on 20 grid squares for the presence of ferruginous bodies and analysis of the core material of these structures.

The laboratories of RFD and LWP conduct background evaluations to assure the cleanliness of each laboratory and assuring that the material found on the filters came from the prepared tissue rather than contamination from some background source. Data comparisons of the elongated particulates were made with asbestos standards obtained from NIST/HSE. For Libby amphiboles, comparisons were made using samples as used by Federal Agencies and was obtained from USGS (defined as LA2007B). The physician of record for the patient obtained the approval from the family to permit evaluation of the tissue for content of fibrous structures and ferruginous bodies.

Results

Pathological evaluation of the tissue section

An examination by one author (JRB) of a 6 cm² section of lung parenchymal tissue revealed evidence of fibrosis associated with both membranous (Figure 4) and respiratory bronchioles (Figure 5). Ferruginous bodies were identified with features of classical asbestos bodies (Figure 6). Descriptively the “typical asbestos body” is characterized by a straight and fibrous, optically clear core, averaging approximately 0.5 μm in diameter. The core is surrounded by a variably segmented, golden brown coating that stains for iron and the ends of fibers are often knobbled by ferruginous material as described by Crouch and Churg (1984). A rare focus of alveolar septal fibrosis is identified in the first tier of alveoli. These findings permit classification of the pathological feature as Grade 1 “asbestosis” consistent with the definition in the CAP-NIOSH grading scheme as described by Craighead and associates (1982). Although there are concerns regarding the definition of asbestosis in the CAP criteria offered in the 2010 version (Hammer & Abraham, 2015), there are areas within the tissue sections...
that meet even the 2010 definition of asbestosis (Roggli et al., 2010). There were also anthracotic pigments identified by both reviewers (JRB and RFD) in the walls of many of the bronchioles.

**Tissue burden analysis**

Further confirmation as to the core material of ferruginous bodies was confirmed as per required ATEM analysis. The preparation of the digested material that was collected on an MCE filter that was cleared and evaluated by light microscopy offers a more unimpaired view of the morphology of the ferruginous bodies than afforded in tissue sections. The isolated structures are collected on a flat plane rather than various orientations in a tissue section and the cleared filter permits no obstruction when viewing the structures as may be present from tissue components in a tissue section. In this preparation, the structures were consistent with the appearance as defined previously for an “asbestos body.” The calculation of the numbers of ferruginous bodies from the count on the cleared portion of the MCE was 150,830 ferruginous bodies/gram de-paraffinized wet weight of tissue.

A sample of a standard of “Libby amphiboles” was collected on a polycarbonate filter from which a replica was prepared and evaluated by ATEM for diffraction and EDAX comparisons with the fibers found in the tissue digestion. The digested material from the lung sample was collected at two concentrations on the PC filters. When the replicas of the preparations were evaluated by ATEM, the level of tissue burden of fibers and ferruginous bodies was such in the heavier concentration preparation that a dust overload existed (Figure 7). The analysis for quantitative purposes conducted at 15,000x in the ATEM resulted in a “stop count” reached when the last grid square was complete in which 100 structures had been reached. All uncoated fibers analyzed in the preparation were Libby amphiboles (Figure 8) as defined by SAED and when the elemental composition was compared were consistent with the elemental composition of standard USGS LA2007B (Figure 9). The 100 Libby amphibole fibers were equivalent to 5,377,090 fibers/gram de-paraffinized wet weight of tissue. The average length of the uncoated Libby amphiboles was 13.03 μm. There were eighteen of the uncoated fibers that were less than five micrometers in length and thus would not be counted based on length alone in NIOSH’s 7400 count scheme, where fibers must be 5 μm or longer to meet the count definition of a fiber. The longest Libby uncoated fiber was 63 μm in length. There were three ferruginous bodies observed in the area scanned at 15,000x and the cores were Libby amphiboles. An additional low magnification scan at 2000x was conducted to find ferruginous bodies and thus analyze the core composition. There were seventeen ferruginous bodies found at low magnification and these were formed on cores determined to be Libby amphiboles (Figure 10).

**Discussion**

This case is, to our knowledge, the first in which lung tissue from a Libby miner has been evaluated for fiber analysis. There are numerous unique findings from this tissue evaluation. There are numerous “classical ferruginous bodies” found within the tissue section from this case. This observation is highly relevant in that of the pathological diagnosis of asbestosis in tissue requires the presence of such structures in areas of interstitial fibrosis (Craighead et al., 1982). However, as Crouch and Churg (1984) noted “there has been a tendency on the part of pathologists to equate the term ‘ferruginous body’ with ‘asbestos body,’ asbestos bodies are actually only one type of ferruginous body and several classes of minerals, including all types of asbestos, sheet silicates, carbon, rutile, fly ash and even iron, have been found to undergo ferrugination in the human lung.” They offer examples of these types of structures. However, many of these cores are relatively easy to distinguish by light microscopy when ferruginous bodies are isolated from tissue or lavage material, since they do not have a transparent clear core that is evident as being formed on a fibrous core (Dodson et al., 1994). This definition of an “asbestos body” gets a bit more complex in tissue sections when one mixes in variations in orientation of such structures due to a plane of section, as well as complications of tissue components.
that induce interference with the ability to clearly visualize the core material.

In the present case, the ferruginous bodies are totally consistent in appearance with those formed on "asbestos cores"; however, these are not formed on regulated "asbestos" as previously defined in the reference to OSHA, AHERA (1987) and NESHAP. This poses another interesting issue in how one uses the term "asbestosis" (in several variations of the pathological definition) which depends on the presence of "asbestos bodies" associated with areas of interstitial fibrosis (Craighead et al., 1982). In fact, Churg (1993) has even suggested that "when asbestosis is present and the patient is also a cigarette smoker, then both smoking and asbestos exposure should be considered etiologic agents (of lung cancer). But when asbestosis is absent, the asbestos exposures should not be implicated, and some other cause for the lung cancer, usually cigarette smoking, must be sought." We confirm the presence of Libby amphibole fibers in an individual with exposure to Libby vermiculite and that the ferruginous bodies are formed on asbestiform structures that do not presently conform by type to "regulated asbestos" (as defined in the aforementioned federal regulations). This poses an interesting question applicable not only in this patient, but for those with other occupational or consumer exposures to vermiculite products as well as those exposed to other "nonregulated" forms of asbestiform minerals in that if the patient has radiographic and surgical findings consistent with "asbestosis" as well as ferruginous bodies associated with areas of interstitial fibrosis then should the clinical status be literally not "asbestosis?" It seems that the regulations might be appropriately inclusive of additional asbestiform structures than those presently included under the general definition "asbestos." If not, then what should clinical changes such as in the present case be called other than a general term of interstitial fibrosis which describe outcomes in numerous diseases? For this reason,
from the clinical standpoint, we would reasonably take the position that “asbestosis” must be used to describe the presence of ferruginous bodies in the presence of interstitial fibrosis when the core materials of the ferruginous bodies are Libby amphiboles.

It is clear that without the suggestions from the occupational history the findings of large numbers of classical ferruginous bodies in the tissue sections could be appropriately concluded based on morphology as from exposure to a “regulated form of asbestos.” The presence of “asbestos bodies” defines a critical component in pathological definition of asbestosis (Craighead et al., 1982). We have chosen to use this document for the definition of asbestosis since it is a document including participation by NIOSH, unlike the 2010 version of Committee of the College of American Pathologists (CAP) and Pulmonary Pathology Society’s update on diagnostic criteria (PPS) (Roggli et al., 2010).

There are also issues raised regarding the “new definition of pathologically defined asbestosis” in the 2010 paper as defined in the commentary by Hammer and Abraham (2015). We are in complete agreement with their points. Two of the authors (RFD and LWP) hold licenses/credentials by afore mentioned agencies that require the use of the term “asbestos” required under compliance definitions to mean one of the six types of elongated mineral structures listed in the regulations. Winchite and richterite series, the majority of the fibrous material in the Libby vermiculite dust, is not listed under the definition of asbestos in the regulations.

Nevertheless, the individual in this study has appreciable numbers of “classical ferruginous bodies” in his tissue, thus indicating that at some time in the past a heavy exposure occurred to fibrous dust and that a portion of that fiber exposure consisted of longer fibers since only longer fibers stimulate formation of ferruginous coatings (Dodson, 2011).
The evaluation of Respirable Elongated Mineral Particles (REMP’s) (Aust et al., 2011) in the lung of this individual reflects a snapshot in time of particulates left in the tissue following years of particulate removal by clearance. The clearance mechanism in man is a very efficient process for removal of inhaled materials with the smaller particles being more likely to be removed first (Gross & Detreville, 1972). Thus, classical ferruginous bodies when compared to uncoated fibers of the same fibrous mineral, are larger and more difficult particles to clear. The level of elevated numbers of classical ferruginous bodies per gram in the tissue in the digestate (150,830 ferruginous bodies/gr. de-paraffinized wet weight) can be appreciated as greatly elevated when a comparison is made with the numbers of ferruginous bodies found in our laboratory in tissue from general population groups which range from 20–0 (non-detect) per gram wet weight of tissue (Dodson et al., 1999).

As another basis for comparison, the number of classical ferruginous bodies per gram de-paraffinized wet weight of tissue from this individual would have been the second highest level found in lungs from a series of twenty individuals with alleged occupational exposure to asbestos and having developed lung cancer (Dodson et al., 2004). Also, when a comparison is made of the number of Libby amphiboles found in the present case (5,377,090 per gram de-paraffinized wet weight) with the total asbestos fibers in the lung cancer study, it was determined that the number would have been the second highest. It must be reiterated that just as with the ferruginous bodies, the uncoated fibers of Libby amphibole reflect what is left in the lung after years of clearance. This may explain why a small percentage of Libby amphiboles in the lung are less than five micrometers since, reasonably, the shorter fibers are more mobile and over time more likely to clear. That means that numbers of all sizes have been
eliminated by the time the tissue sample was taken. As a synopsis, this miner had very elevated numbers of ferruginous bodies in his lung tissue and which, when viewed in tissue sections by light microscopy, would be considered as “asbestos bodies.” There were also large numbers of uncoated “Libby fibers” in the tissue and the clinical evaluations presented in the paper have shown that exposure to Libby amphiboles can cause all types of asbestos related diseases including lung cancer from which this individual suffered.

Conclusions

This case study of an individual who worked in the Libby, Montana vermiculite mine offers unique observations. Figures 2 and 3 demonstrate pleural soft tissue changes that would not be readily identified on CT scan using current methodologies but clearly correlate with diffuse parietal pleural fibrosis as seen during thoracoscopy. Based on these observations, advances in the detection of pleural fibrosis using a standardized CT reading methodology, similar to B reading used for chest x-rays, would be useful for CT reading. The clinical changes reflect features consistent with those associated with asbestos exposures. The pattern of thin, non-calciﬁed diffuse pleural thickening is typically observed in occupational and environmental exposure to Libby vermiculite associated amphiboles. However, the major fiber exposures in this case are not deﬁnable as “asbestos” under federal regulations in that they are not one of the six types of ﬁbrous forms of minerals regulated under the asbestos regulations. The case provides to our knowledge, the ﬁrst tissue burden analysis by light and transmission electron microscopy of a vermiculite miner from this cohort. There is considerable ferruginous body and uncoated ﬁber burden within the tissue. The ferruginous bodies seen in the light microscopy evaluation are consistent with the appearance of asbestos cored ferruginous bodies (asbestos bodies). However, since the bodies are not formed on one of the elongated mineral structures deﬁned in the regulations as “asbestos,” a question arises as to what one should call the ﬁbrosis induced by these ﬁbers. If they were on asbestos cores then the pathological changes consistent with their presence in areas of ﬁbrosis would be called “asbestosis.” The ﬁndings within the tissue of an individual exposed to the ﬁbrous materials at the mine also raises additional issues of clinical awareness relevant to those individuals exposed in the use of the vermiculite in manufacturing of vermiculite-containing products and users of these products.

Acknowledgements

The authors would like to acknowledge Dr. Steven J. Nisco of Northwest Heart and Lung Surgical Associates in Coeur d’Alene, Idaho for the collection of tissue and thorascopic photos. The authors would also like to acknowledge Tracy McNew of CARD for her assistance editing and formatting this manuscript.

Disclosure statement

The individual in this case, and thus, the clinical data were generated by clinicians involved in the management of his illness (BB, GL, CH).


