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A risk assessment for exposure to grunerite asbestos (amosite) in an iron ore mine

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ABSTRACT **The potential for health risks to humans exposed to the asbestos minerals continues to be a public health concern. Although the production and use of the commercial amphibole asbestos minerals—grunerite (amosite) and riebeckite (crocidolite)—have been almost completely eliminated from world commerce, special opportunities for potentially significant exposures remain. Commercially viable deposits of grunerite asbestos are very rare, but it can occur as a gangue mineral in a limited part of a mine otherwise thought asbestos-free. This report describes such a situation, in which a very localized seam of grunerite asbestos was identified in an iron ore mine. The geological occurrence of the seam in the ore body is described, as well as the mineralogical character of the grunerite asbestos. The most relevant epidemiological studies of workers exposed to grunerite asbestos are used to gauge the hazards associated with the inhalation of this fibrous mineral. Both analytical transmission electron microscopy and phase-contrast optical microscopy were used to quantify the fibers present in the air during mining in the area with outcroppings of grunerite asbestos. Analytical transmission electron microscopy and continuous-scan x-ray diffraction were used to determine the type of asbestos fiber present. Knowing the level of the miner's exposures, we carried out a risk assessment by using a model developed for the Environmental Protection Agency.**

We evaluate the potential for any risk to health in miners that might arise after the release of grunerite asbestos from a seam in an iron ore mine. None of the analytical criteria required for the mineral's identification were ambiguous (the objects studied were asbestos fibers, not cleavage fragments). A geological survey of the asbestos seam indicated localization in a relatively small area of the mine. No asbestos of any other variety was detected in the blast pattern and drill core samples. To evaluate the potential for asbestos exposure, an air sampling program that included area and personal samples was initiated. Both types of samples were analyzed by phase-contrast optical microscopy and analytical transmission electron microscopy (ATEM). The risk assessment calculations were referenced to the fibers ≥ 5 μm long, with fiber counts obtained by phase-contrast optical microscopy using standard National Institute of Occupational Safety and Health–Mine Safety and Health Administration (MSHA) methods.

The grunerite asbestos identified in the iron ore mine is a known human carcinogen and merits special attention, although its presence in the mine appears to be an anomaly. The best evidence for the pathogenicity of grunerite asbestos has come from epidemiological studies of workers in factories where predominantly this fiber type was used. The mortality studies of lung cancer, mesothelioma, and asbestosis among grunerite asbestos exposed workers are reviewed.

In addition, lung content analysis using ATEM was used to characterize the fiber concentrations found in lung tissues of individuals who developed asbestos-related diseases after exposure. The results of the air sampling program are used to calculate the mine work required to inhale a similar number of fibers as that found in the lungs of mesothelioma cases.

The exposures measured in the iron ore mine are several factors of ten lower than the occupational exposures that occurred in the studied groups. Unlike the comparisons of lung content described above that assumes a threshold, the Environmental Protection Agency (EPA) model assumes a linear dose-response, where each exposure is associated with an incremental increase in risk.

Brief Review of the Occupational Health Effects Associated with Asbestos Exposure. The earliest reports on the health effects of exposure to asbestos occurred among individuals who were exposed predominately to chrysotile asbestos (1). The first case in the English literature of asbestos-related pulmonary fibrosis described as asbestosis was reported in 1927 and occurred in a chrysotile textile worker. Although the first medical indications of any effect of asbestos on health was reported in 1906 in France and the United Kingdom, it (as with other diseases, like silicosis) was frequently complicated by the presence of tuberculosis. However, by 1938, asbestosis was generally accepted by industry and government health units as an occupational disease with distinct clinical, radiological, lung function, and pathological characteristics.

Case reports of lung cancer accompanying asbestosis first began to appear in the literature during the 1930s. The evidence associating these diseases was greatly strengthened by the information Merewether provided for the 1947 Report of the Chief Inspector of Factories (England). He reviewed the accumulated data from 1923–1946 and found a 13.2% prevalence of lung cancer among the 235 autopsies of individuals know to have died with asbestosis, compared with 1.3% in 6,884 cases of silicosis. A high prevalence of lung cancer was found among other autopsy series of asbestosis cases, such as Wyer (1949), where 14.8% lung cancer was found among 115 asbestosis deaths (1), although at a meeting in Zagreb in 1953, Merewether (2) expressed doubt about the relationship between asbestosis and cancer of the lung, perhaps because of the limitations of an autopsy series.

In 1955, Sir Richard Doll published a comprehensive epidemiological survey of employees of chrysotile asbestos textile plant in Rochdale, England (3). Individuals employed for 20 or more years experienced lung cancer $\approx$14 times more frequently than the general population (11 cases observed/0.8 expected). The results became available at the same time that

Abbreviations: ATEM, transmission electron microscopy; SMR, standardized mortality ratio; OSHA, Occupational Safety and Health Administration; MSHA, Mine Safety and Health Administration.

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the association between lung cancer and cigarette smoking was being established. Defining the increase in the risk of developing lung cancer when an individual's exposure to chrysotile asbestos is insufficient to produce asbestosis is mostly theoretical. Changes in the diagnostic criteria of asbestosis have further complicated the matter.

In 1960, Wagner *et al.* (4) reported 33 cases of a malignant tumor known as mesothelioma, which he attributed to crocidolite exposure. The discovery focused attention on the question of asbestos fiber type and disease. This rare tumor was the last of the three major asbestos-related diseases to be identified. The potency of chrysotile to induce this tumor in humans remains a subject of considerable controversy. It also is clear that exposure to crocidolite asbestos, actinolite-tremolite asbestos, and grunerite asbestos produce considerably higher incidence of this disease, sometimes even after exposures that are considered quite low. The patterns of mesothelioma depending on asbestos fiber type are strikingly different in that a high mortality for mesothelioma is never found among individuals exposed only to chrysotile asbestos (5), although from time to time, individuals present with pleural mesothelioma and high concentrations of chrysotile are found to be present in the pulmonary tissue by lung content analysis (6).

Geological Survey of the Area of the Mine Containing Grunerite Asbestos. The grunerite asbestos is confined to quartz–ankerite–grunerite veins of the host rock. These veins contain medium- to coarse-grained quartz, ankerite, stilpnomelane, and grunerite fiber distributed throughout a specific bench face (Fig. 1). The veins range up to 3 feet thick. The major veins occur within a magnetite–chert–silicate unit at the contact of the host rock and metadiabase sill units. The larger veins generally conform to the compositional banding of the host rock, but smaller veins commonly cut across the structure. Long-fibered asbestos mineral development is restricted to the thicker conformable veins.

Grunerite asbestos is developed within the quartz–ankerite–stilpnomelane veins and along its contact with the host rock and sills. The veins were deformed structurally, exhibiting signs of shearing, brecciation, faulting, and folding. Minor quartz–carbonate veins occur, which lack asbestos-like minerals.

The grunerite asbestos is discontinuous along the strike of the veins. Locally, recrystallization or replacement within the host rock has resulted in relatively coarse-grained acicular amphibole. The coarse-grained amphiboles are most notable in the silicate layers, but occur occasionally within the magnetite–chert bands, particularly near grunerite asbestos. Fibrous amphiboles occur irregularly in cross-cutting and concordant vein-like structures over a gradational zone from the host wall rock, with fairly coarser grained amphiboles, to quartz-ankerite–stilpnomelane–grunerite veins. The coarse grunerite asbestos occurs discretely within, and immediately adjacent to, the quartz–ankerite–stilpnomelane veins (Fig. 2). Strongly sheared horizons in the host rock close to the veins have formed platy, bladed, and fibrous mineral habits, only some of which are asbestiform. At several places along the strike of the quartz–ankerite–stilpnomelane–grunerite veins, the host rock has been tightly folded immediately adjacent to the vein (several inches on both sides). Essentially no deformation is observed just inches away from tight folding.

Banded, vuggy, quartz–fluorite–pyrite–chalcopyrite veins occur locally (most notably at the extreme southern end of the mapped bench) possibly in association with the quartz–ankerite–stilpnomelane–grunerite veins. The mineralogy and appearance of the sulfide veins indicate a different generation of development, but no clear cross-cutting relationships were observed. Minor quartz–magnetite–pyrite–chalcopyrite veins and veinlets occur.

Analysis of Bulk Samples. Three bulk samples, selected from highly fibrous seams, were analyzed by polarized light microscopy, continuous-scan x-ray diffraction, and ATEM. In the United States, MSHA and the Occupational Safety and Health Administration (OSHA) regulate six minerals under the asbestos standard (Table 1). Five are amphiboles. These minerals have diverse elemental compositions (7). Each of the named minerals can exist in three different morphological forms or habits (8) that have been shown to effect their biological potential (9). In the asbestos habit, the fiber occurs as parallel fibrils, which form polyfilamentous bundles. It is this habit that is believed to cause cancer, and only this asbestos habit is regulated by MSHA and OSHA. The two other habits are nonasbestiform, occurring as splintery fiber, and massive anhedral nodules. When crushed, however, the nonasbestiform amphiboles may form elongated cleavage fragments that morphologically resemble fibers. Difficulties arise when cleavage fragments occur in association with amphibole asbestos.

Two of the asbestos minerals (cummigtonite–grunerite and tremolite–actinolite) form a solid solution series in which Fe^{2+}

FIG. 1. The grunerite asbestos occurred along the wall and on top of the lower bench on the left.

FIG. 2. The coarse grunerite asbestos vein occur within, and immediately adjacent to the quartz–ankerite–stilpnomelane veins.

and Mg^{2+} substitute. Although actinolite, grunerite, and tremolite do occur in nature as asbestos minerals, an occurrence of cummingtonite asbestos has not been reported.

All three of the highly fibrous samples were analyzed by polarized light microscopy, continuous-scan x-ray diffraction, and ATEM. None of the analytical criteria required for the mineral's identification are ambiguous (10). The asbestos seam is localized to a relatively small area of the mine. No other asbestos fiber type was detected in 24 blast pattern and drill core samples collected to evaluate the depth to which the seam extends.

Evaluation of Air Samples from the Mine. To evaluate the potential for asbestos exposure by inhalation, an air sampling program (including both area and personal samples) was initiated. The personal samples were job classification-specific and sufficient in number to evaluate the range of exposures that would occur during mining of the ore. Of the 179 personal air samples collected, the mean concentration was 0.05 fiber per ml (all fiber ≥5 μm), and the highest exposure was 0.39 fiber per ml (all fiber ≥5 μm) (Table 2). None exceeded the MSHA asbestos standard (2 fiber per ml) (all fiber ≥5 μm) or action level, although 13.4% did exceed the current OSHA asbestos standard of 0.1 fiber per ml (all fiber ≥5 μm) (Table 3).

Comparison of Epidemiological Studies of Workers Exposed to Iron Ore Dust and Those Exposed to Asbestos Dust. The four epidemiological studies described cover mortality. Such studies of causes of death,are used to determine whether a cohort (a group of individuals defined by exposure to some agent) dies more frequently from a particular disease than would otherwise be expected (based on rates in the reference population, e.g., everyone in the U.S.A.). Diseases such as lung cancer occur with a natural background. Cigarette smoking elevates the expected background death rate, and cancer incidence may be further increased by exposure to certain environmental agents. The assumption is made that the fraction of people that smoke is the same in the exposed as the control group. Epidemiological cohort studies allow for the determination of association between exposure to some agent and an increase in the occurrence of a specific disease. The standardized mortality ratio (SMR) is the number of deaths observed of a specific disease in the cohort divided by the number of deaths from that cause expected for the reference population, multiplied by 100. As the years of exposure increases, the SMR should also rise because of the increase in dose.

A cohort of 17,800 asbestos insulation workers in the United States and Canada was followed from January 1, 1967 until the end of 1986 (11, 12). At the end of 1986, after almost 302,000 person-years of observation, 4,951 deaths occurred, while only 3,453 deaths were expected. The increased incidence of lung cancer accounted for >50% of the excess deaths (Table 4). The SMR (100 × observed/expected cases) for lung cancer was 435, whereas 8.6% and 9.3% of the deaths were caused by asbestosis and mesothelioma, respectively. Although the insulators were exposed to all of the commercial asbestos fiber types, the major fiber type retained in the worker's lung tissue was grunerite asbestos (12).

Table 2. Results of the analysis of three hundred and twenty-six air samples collected while mining a grunerite asbestos (amosite) seen by the NIOSH-7400 methods

Air sample	No. of samples	Concentration of fibers per ml† (all fibers ≥5 μm in the air)	Range of fiber concentration
Area			
During mining	137	0.02 ± 0.02	0.001–0.20
During blasting	10	0.01 ± 0.01	0.002–0.02
Total	147		
Personal			
Drilling	110	0.06 ± 0.05	0.001–0.23
Shovel	22	0.06 ± 0.09	0.008–0.39
Production truck	23	0.04 ± 0.05	0.005–0.24
Track dozer	20	0.05 ± 0.04	0.005–0.17
Blast	2	0.03 ± 0.03	0.013–<0.05
Unidentified Sample	2	0.05 ± 0.03	0.028–0.07
Total	179	0.05 ± 0.05	0.001–0.39
Total samples analyzed	326	0.04 ± 0.05	0.001–0.39
Field	11	5.2‡	
Laboratory	7	2.7‡	
Unspecified	2	1.6‡	
Not analyzed	3		
Total samples taken	349		

†Values given as arithmetic mean ± SD—the fiber concentrations a log normal.
‡Controls, values are fibers per mm^2 of filter area.

Table 1. Mineralogy of the six minerals regulated under the asbestos standard in the United States. Amosite is occasionally referred to as cummingtonite–grunerite asbestos

Commercial name	Mineral name	Mineral group	Chemical formula
Amosite	Grunerite	Amphibole	$(Fe^{2+}, Mg)_7[Si_8O_{22}]\ (OH)_2$‡
Anthophyllite asbestos	Anthophyllite*	Amphibole	$(Mg, Fe^{2+})_7[Si_8O_{22}]\ (OH)_2$
Chrysotile	Chrysotile	Serpentine	$Mg_3[Si_2O_5]\ (OH)_4$
Crocidolite	Riebeckite	Amphibole	$Na_2Fe^{3+}{}_2(Fe^{2+}, Mg)_3[Si_8O_{22}]\ (OH)_2$
Tremolite asbestos	Tremolite*†	Amphibole	$Ca_2Ma_5[Si_8O_{22}]\ (OH)_2$
Actinolite asbestos	Actinolite*†	Amphibole	$Ca_2(Mg, Fe^{2+})_5[Si_8O_{22}]\ (OH)_2$

*These minerals do not have separate names for their asbestos analogs. Mineralogists now refer to amosite as grunerite asbestos and cocidolite as riebeckite asbestos, although the commercial names persist in the literature.
†Tremolite–actinolite also form a solid-solution series between a calcium–magnesium-end member (tremolite) and a calcium–iron magnesium-end member (actinolite).
‡For amosite (grunerite asbestos) the Fe^{2+} is present in at least 5 of the 7 available x structural sites.

Table 3. United States regulations concerning occupational exposure to asbestos fiber

Regulatory agency	Standard for an 8-hour time-weighted average, asbestos fibers per ml	Excursion level, fibers per ml	Action level, asbestos fibers per ml
MSHA	2.0	≤10‡	1.0
OSHA*	0.2	None Allowed	0.1
OSHA†	0.1	None Allowed	0.05

All data refer to all fibers ≥5 μm.
*Department of Labor Reg. 1986, 29CFR 1910-1926. Effective July 21, 1986.
†Department of Labor Reg. 1994, 29CFR 1910, Effective October 11, 1994.
‡In a 15-min period.

Vermiculite Ore Containing Tremolite Asbestos. The mineral vermiculite has the generalized chemical formula $(Mg, Ca)_{0.35}(Mg, Fe, Al)_3(Al, Si)_4O_{10}(OH)_2nH_2O$. On heating, the mineral loses water rapidly and expands to form a lightweight aggregate used for various purposes, e.g., insulation, soil conditioning, and filter medium. Various amphibole minerals associated with vermiculite have been the focus of health concerns, rather than vermiculite itself.

The health effects among the miners and millers in Libby, Montana exposed to vermiculite containing tremolite asbestos have been studied by two groups of investigators (13–17). Each investigation was designed as a mortality study and a cross-sectional chest radiographic survey. Slightly different criteria were used to define each cohort: the McDonald study (13, 14) contained 406 men with 165 deaths, and the Amandus study (15–17) contained 575 men with 161 deaths. Both research groups used historical air samples to estimate exposure indices for the cohort members. The dust levels in the past were made with a device called a midget impinger, and the unit of concentration of dust was expressed in millions of particles per cubic foot (mppcf) of air. Conversion factors have been used to change the mppcf unit to an approximate number of fibers per milliliter of air (fibers per ml ≥5 μm), the units used in modern risk assessment (13, 15, 18).

The exposure in the mill before the installation of dust control equipment in 1964, was estimated to be 400 and 168 fibers per ml (all fiber ≥5 μm), respectively. Dust levels between 1965 and the closure of the mill in 1974 were estimated by McDonald *et al.* and Amandus *et al.* to ≈20 and ≈33 fibers per ml (all fiber ≥5 μm), respectively. These were the highest exposures measured except for 20% higher dust levels during floor sweeping.

McDonald and colleagues calculated the SMR for total mortality as 117, with 23 lung cancers observed against 9.4 expected (SMR = 245) and 4 mesotheliomas (2.4%). The SMR for the total mortality in the Amandus cohort was 110, with 20 lung cancers where ≈9 cases were expected (SMR = 223) and 2 mesotheliomas (1.2%). The lung cancer SMR for >20 years since first exposure for all exposure levels were 242 and 279 for the McDonald and Amandus cohorts, respectively. Both cohorts had an SMR of 250 for nonmalignant respiratory disease.

Two Cohort of Minnesota Iron Ore Workers. Taconite is a term used particularly in the Lake Superior region of Minnesota for certain iron-containing rocks from the Biwabik Iron Formation. A high-grade ore concentrate is obtained from commercial-grade taconite that contains enough magnetite (Fe_3O_4) to be economically processed by fine grinding and wet-magnetic separation. Taconite is a hard, dense, fine-grained metamorphic rock that contains substantial quartz (20–50%) and magnetite (10–36%) and various mineral constituents, including hematite, carbonates, amphiboles (principally of the cummingtonite–grunerite series, although actinolite and hornblende also occur), greenalite, chamosite, minnesotaite, and stilpnomelane.

Reserve Mining Company. Analysis of mortality data obtained on men who were employed from 1952–1976 has been reported (19). The study was initiated by concerns in the early 1970s that asbestos was released into the air and dumped into lake water during processing of the taconite rock (20, 21). It was inferred that this dust posed a risk to the miners as well as to the general public. Silver Bay and Duluth obtained their drinking water from Lake Superior, into which the pulverized waste rock (or tailings) from the pellet plant was deposited at Silver Bay. The U.S. Department of Justice considered this a potential health hazard. The Department alleged that the amphibole in the waste rock (cummingtonite–grunerite) was asbestos and the exposures would cause gastrointestinal cancer through ingestion and lung cancer from inhalation of the water- and airborne fibers (although they had done no calculation of this).

The Reserve cohort consisted of 5,751 men, of which 907 had worked for the company for >20 years and 298 were deceased. The men had been exposed to respirable dust concentrations from 0.02 to 2.75 mg/M^3, the modal range being 0.2–0.6 mg/M^3. The fibrous particulate content of the dust was occasionally >0.5 fibers per ml (all fibers ≥5 μm), but usually the concentration was much lower.

The observed and expected deaths and SMR for all men who had worked one year or longer from 1952–1975 are given in Table 5. There was no relationship between the mortality observed and lifetime exposure to silica dust (that was as high as 1,000 mg/M^3 × years). There was no suggestion that deaths from cancer increased after 10 or 20 years of latency. No deaths from mesothelioma or asbestosis were reported.

Table 4. Deaths from lung cancer asbestosis and mesothelioma* among 17,800 asbestos insulation workers in the United States and Canada (1967–1986)*

Years from onset of exposure	Person-years	Lung Cancer		SMR	Asbestosis		Mesothelioma	
		E	O	O/E	O	No. per 100,000 per yr	O	No. per 100,000 per yr
<15	61,655	3.9	9	232	1	1.6	0	0
15–19	52,709	11.6	37	318	14	26.6	5	9.5
20–24	57,595	27.5	95	346	31	53.8	18	31.8
25–29	50,518	46.6	183	393	52	102.9	73	144.5
30–34	37,165	57.4	281	490	59	158.8	105	287.5
35–39	20,340	46.8	239	511	84	413.0	91	447.4
40–44	10,200	30.8	155	503	80	784.3	59	578.5
45–49	5,256	18.8	75	399	33	627.8	58	1103.4
>50	6,151	25.4	94	370	73	1,186.8	49	796.6
Total	301,593	268.7	1168	435	427	141.6	458	151.9

*Best evidence: Causes of death categorized after review of best available information (autopsy, surgical, and clinical). E, expected; O, observed.

Table 5. Selected causes of mortality for men who worked one year or longer for the Reserve Mining Company

Cause of death	ICD*	Deaths		SMR†
		Expected	Observed	
All causes	000–E999	343.7	298	87
Cardiovascular disease	402, 404, 410–429	123.8	112	90
Cancers				
All	140–209	63.4	58	92
Respiratory	160–163	17.9	15	84
Digestive	150–159	17.6	20	114
Urinary	188–189	3.0	3	101
Genital	180–187	3.3	3	91
Selected nonmalignant respiratory diseases	470–474, 480–486, 490, 491, 493, 510–519.	6.8	4	59
All external causes	E800–E998	72.8	76	104
Motor vehicle accidents	E810–E823	31.2	38	122

Source: Higgins *et al.* (1983)
*International Classification of Causes of Death, 8th Revision.
†Standardized mortality ratio, based on white male mortality in Minnesota, 1952–1976.

Minnesota Taconite Miners. A second epidemiological study of Minnesota taconite workers employed at the Erie and Minntac mines was reported (22). The study cohort, followed from 1947–1988 with a minimum observation period of 30 years for all participants, was made up of 3,341 men, of which 1,058 were deceased. Dusts in the two mines are reported as containing 28–40% and 20% quartz at Erie and Minntac mine, respectively. Concentrations of fibrous particulates were nearly always <2 fibers per ml (all fibers ≥5 μm). These fibrous particulates included elongate cleavage fragments and are assumed to be similar to those objects reported at Reserve Mining. The total number of deaths was significantly fewer than expected, SMR = 83 (based on U.S. male rates) and 91 (based on Minnesota male rates). SMR for all cancer (including lung cancer), diseases of the circulatory system, and nonmalignant respiratory disease were fewer than expected when compared with both reference groups (Table 6).

There was one reported case of mesothelioma in a 62-year-old worker whose exposure to taconite had begun only 11 years before his death. Although latency periods as short as 15 years have been reported among insulation workers, mesothelioma generally occurs following a long latency period of 25 years or more (23). This person had previously been employed in the railroad industry, as a locomotive fireman and engineer, an occupational environment where both amosite and crocidolite asbestos insulation was used and opportunity for exposure existed (12). It is unlikely that this particular taconite exposure contributed to the appearance of mesothelioma.

Analysis of the mortality data, with a minimum latency period of 30 years, provided no evidence to support any association between exposure to quartz or elongated cleavage fragments of amphibole with lung cancer, nonmalignant respiratory disease, or any other specific disease.

Comparison of Occupational Cohorts Exposed to Iron Ore and Asbestos. The American and Canadian asbestos insulation workers are generally thought to have had exposure to the three principal commercial asbestos fiber types—grunerite asbestos, crocidolite, and chrysotile (12). The tremolite asbestos in the vermiculite at Libby, Montana has never been extensively used in commerce in the United States. The vermiculite workers are an example of the effect of amphibole asbestos at concentrations of ≈1% in the ore. The mortality experience of the two asbestos-exposed groups are distinctly similar. Each shows an elevated risk of lung cancer, mesothelioma, and asbestosis (a nonmalignant respiratory disease). Of the 1,058 deaths reported in the most recent study of Minnesota taconite workers, one would have expected about 250 lung cancer (23.6%) and about 98 mesotheliomas (9.3%) if their mortality experience was similar to American and Canadian insulators (11). Instead, the actual number of lung cancer and mesotheliomas (Table 6) was 65 (6.1%) and 1 (0.09%), respectively.

Actually 32 fewer lung cancer occurred than the 97 expected (SMR = 67) using the rates for U.S. white males. The one mesothelioma that did occur had a latency of ≈11 years in taconite mining. In the large insulation cohort (17,800 workers), no mesothelioma was reported with a latency <15 years, indicating the present case was unlikely to be related to his taconite dust exposure (11, 23). The mortality experience of the iron ore workers is, in fact, overall less than expected, indicating they are healthier than the general population. This healthy workers effect is commonly observed among many employed groups.

Epidemiological and Lung Content Analysis of Grunerite Asbestos-Exposed Workers. Before the United States entering

Table 6. Deaths by major causes (1948–1988) in taconite miners and millers exposed for 3 months or more before 1959

Cause of death (ICD, 7th Revision, 1955)	Deaths		SMR
	Expected	Observed	
All causes (001–998)	1,272.5	1,058	83
All malignant neoplasms (140–205)	267.7	232	87
Digestive organs and peritoneum (150–159)	70.5	66	94
Stomach (151)	12.0	11	92
Large intestine (153)	23.9	26	109
Respiratory system (160–164)	97.0	65	67
Bronchus, tracheas, lung (162–163)	92.2	62	67
Kidney (180)	6.8	12	177
Lymphopoietic (200–205)	25.8	29	112
All diseases of circulatory system (400–468)	575.1	477	83
Arteriosclerotic heart disease (420)	481.8	368	76
Cirrhosis of liver (581)	35.5	24	68
Nonmalignant respiratory disease (470–527)	77.2	55	71
All external causes of death (800–998)	112.3	114	102
All accidents (800–962)	74.4	79	106
Motor vehicle accidents (810–835)	33.4	32	96
Suicide (963, 970–979)	27.3	32	117
Cause unknown		19	
Number of workers		3,431	
Number of person-years		10,055	
Deaths per 1,000 person-years		10.5	
Adjustment of cause-specific SMRs for missing Certificates		+1.8%	

Source: Cooper *et al.* (22)

Table 7. Fiber count (given in millions of fibers per gram of dried lung tissue) by type of pathology

Pathology	Total		Grunerite Asbestos (Amosite)		Number of Cases
	Mean	SD	Mean	SD	
Lung Cancer	1,483	2,568	1,433	2,590	14
Mesothelioma	1,035	1,039	1,000	1,013	5
Other	358	490	297	463	24

Source: Gibbs *et al.* (25).

World War II, a grunerite asbestos factory was established in Paterson, New Jersey to supply the U.S. Navy with asbestos insulation for the pipes, boilers, and turbines in ships. From 1941–1945, 933 men were recruited to work in this plant, which operated until November 1954. Of these, 820 men formed a cohort and provided a unique group of individuals with an intense short-term exposure and a long-term follow-up (24).

Among these individuals, no mesotheliomas occurred with less than a 6-month exposure history or a latency of <20 years. Although the concentration of asbestos fibers in the air of the Paterson plant was never determined, few occupational health experts would estimate the exposure at <30 fibers per ml (all fibers ≥5 μm). Therefore, 6 months of work at the plant is equivalent to 15 fibers per ml × years. The mean fiber levels in the iron ore mine are 0.05 fibers per ml. Therefore, it would require about 300 years of exposure in the iron ore mine to reach the 15 fiber per ml × years level.

For the workers in the Paterson plant the concentration of grunerite asbestos present in the lung tissue of any individual with an asbestos-related disease has not been reported. However, in a report about workers in a British grunerite asbestos factory, lung tissue taken at autopsy from 14 lung cancer and 5 mesothelioma cases were examined for fiber levels (25). The mineral fibers were separated from the lung tissue and analyzed by using ATEM. Although the factory principally used grunerite asbestos, a small amount of chrysotile had also been used. Of the 43 cases in which sufficient tissue was available for fiber analysis, grunerite asbestos was present at a 20-fold higher concentration than the three other commercial asbestos fiber types. In both the lung cancer and mesothelioma cases, ≈97% of the total fiber burden was grunerite asbestos (Table 7). The mean fiber concentration was about 1.483×10^9 and 1.035×10^9 fibers per gram of dry lung tissue for lung cancer and mesothelioma, respectively. The mean fiber concentration was ≈45% higher in the lung cancer cases than in the mesothelioma cases.

Assuming the total dry weight of an average pair of human lungs to be ≈150 gm, the mean total concentration of fiber in the five mesothelioma cases would be 1.5×10^{11} fibers (25). The mean fiber concentration in the air of the iron mine was 0.05 fibers per ml (all fibers ≥ 5μm). The fiber number in the lung tissue represents fibers of all lengths, whereas the air data is only for those ≥5 μm. The 0.05 fibers per ml (all fibers ≥5 μm) represents an index of the fibers present in the air.

The fibers <5 μm and ≥5 μm but too thin to be visible by phase-contrast microscopy were not counted. One method to approximate the total number of fibers per ml is to interpolate from data where the total size distribution of grunerite asbestos has been reported, as at the Penge Mine in the Republic of South Africa (26). Using the length and diameter data from Penge and assuming 0.05 fibers per ml represents the fibers ≥5 μm in lengths and ≥0.25 μm in diameter, a multiplication factory of 6.2 was interpolated. The total fiber concentration in the iron mine is therefore assumed to be 0.05 fibers per ml × 6.2, or 0.33 fibers per ml (all fibers). A second method is to add the fiber counts of 11 air samples from the mine analyzed by phase-contrast optical microscopy and ATEM to estimate total exposure. When the two values were added, the mean exposure was 1.18 ± 0.57 fibers per ml (all fibers). The exposure is 3.6-fold greater than that estimated by using the size distribution of grunerite asbestos in the Penge mining environment, although the mean exposure for the 11 air samples was 0.08 ± 0.05 fibers per ml (all fibers ≥5 μm), which exceeds the average of the 179 personal air samples of 0.05 ± 0.05 fibers per ml (all fibers ≥5 μm). All of the grunerite asbestos fibers counted by ATEM were <5 μm long.

To inhale a concentration of fibers similar to the concentration in the lung tissue of the mesothelioma cases (1.5×10^{11} fibers) would require inhaling 4.7×10^{11} ml of air in the iron ore mine, assuming an exposure of 0.33 fibers per ml. For the purpose of this model, we pessimistically assume *no* clearance, although the lung has mechanisms to clear inhaled particles that can be very effective. Assuming on average an individual inhales 10,000 ml of air per minute, this is 600,000 ml per hour, or 4,800,000 ml per 8-hour shift. This seems a very large number, but it would require ≈98,000 days in the iron ore mine with an exposure of 0.33 fibers per ml (at 1.18 fibers per ml exposure, it would require 27,000 days) just to inhale a similar number of fibers to that found in the only series of lung content analysis of grunerite asbestos-related mesotheliomas. The range is 75–265 years of daily 8-hour shifts of exposure to

Table 8. Risk of death in a lifetime for some selected environmental exposures

Activity	Lifetime risk per 100,000
Heavy cigarette smoking	
All causes of death	35,000
Lung cancer only	9,000
Total	23,000
U.S. motor vehicle accidents	
All deaths	1,200
Pedestrian deaths	100
U.S. air pollution (calculated deaths from assumed correlation)	2,000
Frequent airline passenger, 200,000 miles for 35 years	
Accident	400
Cosmic ray cancers	300
U.S. natural radiation background at sea level (cancers) excluding radon gas	200
U.S. home deaths	
All	600
Falls (mostly over age 65)	200
Drowning deaths (nontransport causes)	80
Diagnostic x-ray in USA (cancer)	200
Person living with a smoker (cancer)	100
Person in brick building, added natural radiation	70
One transcontinental round-trip flight per year	
Accident	15
Cosmic rays	15
Upper level of risk EPA claims to regulate	15
Falling meteorite	5
Drinking water with 100 mg/ml choloroform (EPA level)	5
Eating 1.1 kg charcoal-broiled steak per week (cancer only)	2
World Health Organization (1974) acceptable risk for drinking water	1
Struck by falling airplane (average over entire U.S.)*	0.4
Smoking three cigarettes in a lifetime (all deaths)*	0.3
Lower level of risk EPA claims to regulate*	0.1
Lightning*	0.1–0.15

*Those activities with lifetime similar to the lung cancer and mesothelioma risk calculated for the iron ore miners. All other activities listed pose a higher risk.

Table 9. Lifetime risk for 1-year continuous exposure per 100,000 people for 0.001 fibers per ml

Age on onset of exposure	Lung Cancer Risk				Total Cancer Risk	
	Average*	Nonsmoker	Smoker	Mesothelioma Risk	Nonsmoker	Smoker
30	0.31	0.06	0.62	0.21	0.27	0.83
45					0.1	0.6
50	0.25	0.05	0.50	0.03	0.08	0.53

*From Table 6-3 EPA (1986) for men.

inhale a similar number of fibers to that found in the lung tissue of the factory mesothelioma cases.

Risk Assessment from Mining in the Iron Ore Mine. In the past, workers were exposed to aerosols containing high concentrations of asbestos fibers. To obtain a quantitative risk estimate from the low exposures, we used a model developed for the Environmental Protection Agency to quantify the risk of asbestos-related disease (27). This model is developed to fit the type of data described above, the exposures during mining of the iron ore are orders of magnitude lower than the occupational exposures which occurred in the cohorts used to parameterize the dose component in the equations of the risk models. Nonetheless, the high exposure-response relationships of the past were used to interpolate the risk to the current low exposures encountered in the iron ore mine in linear (proportional) relationships. We know of no scientist who has argued that this linear dose-response model underestimates the risk. The risk assessment model requires that the concentrations of asbestos fibers in the air be determined. Risk assessment is based on counting all fibers ≥ 5 μm in length in the occupational environment by phase-contrast microscopy, at $\approx \times 500$ magnification (Table 2).

Risk estimates were considered for the following two scenarios: (*i*) A bench containing approximately 1 million tons of rock was removed in 22 days. Assuming the average employee is 45 years old, what is the lifetime risk for lung cancer and mesothelioma? No air sampling was done at that site, and it is uncertain whether any asbestos exposure took place. Assume the fiber levels are similar to those given in Table 2. (*ii*) Approximately 30 days of drilling remain to be done on the bench containing the seam of grunerite asbestos (28 days in the sill and two days in the waste iron formation). Assuming the sill contains no asbestos (so far none has been found), what would be the lifetime risk to the drillers for lung cancer and mesothelioma assuming they are 45 years old?

Table 6-3 from the EPA risk model (27) was used. This table is for an exposure to a concentration over a long time. It can be used for a 2- or 22-day exposure if it is assumed that the exposure integrated over time is the relevant parameter. (*i*) There is a linear dose-response relationship. Any proposed biological mechanism of which we are aware involves the exposure integrated over time. (*ii*) If the peak exposure is the parameter of concern, the risk is proportional to the frequency of peak exposures. The integrated exposure is also proportional to the total time of possible exposure and goes down with time.

The average lung cancer risk among smokers and nonsmokers was reported by the EPA. The risk number found in the EPA Table 6-3 is the average for smokers and nonsmokers, but the actual lung cancer risk from asbestos exposure is five times less for nonsmokers and double for smokers. Because mesotheliomas are assumed not to be related to smoking, the number applies to both smokers and nonsmokers.

Exposure. The average of the exposures monitored is appropriate for calculating the risk to a worker not otherwise identified. The mean airborne concentration of 179 personal air samples was 0.05 fibers per ml (all fibers ≥ 5 μm) (Table 2). This value assumes all the fibers were asbestos and that each person was continuously exposed (8-hour time-weighted average) over a 22-day period. The EPA calculated for continuous exposure over different periods of time, and therefore the iron ore mining exposure is converted to be equal to the exposure average over 1 year, $<E>$. $<E> = 22/365 \times 8/24 \times 0.05 = 0.01$ fibers per ml (all fibers ≥ 5 μm). The life-time risk can be read directly from Table 6-3 (27) at 30 and 50 years of age at onset of exposure (45 years of age is interpolated) (Table 9).

Scenario I. The total cancer risk for the individual exposure beginning at 45 years of age is 0.1 and 0.6 in 100,000 for nonsmokers and smokers, respectively (see Table 8 for comparison with selected different lifestyles and environmental exposures). This assumes a linear dose-response. If all of the cancer risk is assumed to be lung cancer, it is equivalent to smoking 2 or 12 cigarettes in a lifetime for 0.1 and 0.6 in 100,000 people respectively. The risk for someone smoking one cigarette is 0.05 per 100,000 people (or, smoking 2 cigarettes is associated with a lung cancer risk of 1 in 1 million).

Scenario II. In this scenario, there will be a 2-day exposure (not the 22-day of Scenario I), so the risk becomes 2/22 or 1/11 of the risk of Scenario I (0.1 in 1,000,000 for nonsmokers, and 0.6 in 1,000,000 for smokers) (Table 10).

These are risks accumulated in a lifetime. Note also that according to the assumption pertaining to the risk calculation; each new exposure adds to this risk independent of the past risk. Of course, if asbestosis is a precondition for lung cancer, there exists a lung cancer threshold (28, 29). Although new exposures can add to past ones, they only increase the risk where the total exposure exceeds the threshold. That the EPA model overestimates the risk of lung cancer is widely believed (30). Although the above is a best estimate, an important consideration is how much larger could the risk be to that individual. An examination of Table 2 indicates the extreme exposure level of 0.39 fibers per ml (all fibers ≥ 5 μm) was seven times larger than the mean 0.05 fibers per ml (all fibers ≥ 5 μm). This suggests the most extreme risk is seven times greater than given above. These risks are put into perspective in Table 8.

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Table 10. Lifetime risk for two mining scenarios in the iron ore mine compared to selected relative risks*

Activity	Lifetime risk per 100,000
Iron Ore Mining	
Scenario I	
Nonsmokers	0.1
Smokers	0.6
Scenario II	
Nonsmokers	0.01
Smokers	0.06
Lung cancer heavy cigarette smoking	9,000
US motor vehicles, all deaths	1,200
Drowning deaths, nontransport caused	80
Upper limit of risk EPA claims to regulate	15

*See Table 8 for additional comparison.

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