MORTALITY PATTERNS AMONG HARD ROCK GOLD MINERS EXPOSED TO AN ASBESTIFORM MINERAL *

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INTRODUCTION

The first suggestion that asbestos could be a carcinogen was made by Lynch and Smith in 1935,[1] as they had noticed an association between asbestosis and lung cancer. Since then, all forms of *commercial* asbestos have repeatedly been shown to be carcinogenic in man, and data from animal studies have corroborated these findings. In 1960 Wagner *et al.*[2] reported that pleural and peritoneal mesotheliomas occurred among asbestos miners occupationally exposed to crocidolite in certain areas of South Africa. From 1960 to 1973, studies of anthophyllite miners in Finland by Kilviluoto,[3] Meurman,[4] and Meurman *et al.*[5] reported significantly increased proportional mortality from bronchogenic carcinoma. In 1972, Selikoff *et al.*[6] demonstrated a sevenfold excess of lung cancer among insulation workers with 20 or more years since their first exposure to amosite asbestos. In 1973, Wagoner *et al.*[7] reported results of a mortality study of asbestos workers occupationally exposed predominantly to chrysotile and demonstrated significant increases in lung cancer and asbestosis.

In 1972, the National Institute for Occupational Safety and Health (NIOSH) recommended an occupational standard for asbestos exposure of 2.0 asbestos fibers greater than 5 μm in length per cubic centimeter of air, as an 8-hr time-weighted average daily concentration.[8] This standard was recommended with the recognition that it would "prevent asbestosis and more adequately guard against asbestos-induced neoplasms."[8] In developing this standard, NIOSH recognized the need for additional research due to "the lack of epidemiological studies or clinical reports with supporting environmental data in the exposure range that must be considered" and "the lack of definite information on the biologic response of fibers of different size."[8]

Further need for research on possible adverse health effects of occupational and nonoccupational exposures to *noncommercial* asbestos fibers, and asbestos fibers *shorter than 5 μm,* was brought out at recent court hearings for a mining company in Minnesota and its disposal of taconite tailings.[9] Expert testimony

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presented during that hearing identified the ore at the mining operation to be of the cummingtonite-grunerite *amphibole* (*asbestos*) mineral series.[9] Furthermore, considerable disagreement ensued as to whether a carcinogenic risk was associated with that ore dust, which contained noncommercial forms of *amosite* asbestos in the cummingtonite-grunerite amphibole mineral category. However, as those mining operations had begun in approximately 1957, the period of observation was adjudged insufficient to evaluate scientifically the presence or absence of a carcinogenic risk associated with such tailings.[10] At the same hearing, however, the South Dakota School of Mines presented x-ray diffraction and optical microscope data [11] that demonstrated the ore body of a hard rock gold mine in Lead, South Dakota to be of the cummingtonite-grunerite amphibole series. In addition, representatives of the Minnesota mining company stated that an occupational health study of a mining population with exposure to ores and dusts similar to those in their mining operation might answer questions as to the general public health implications of exposure to cummingtonite-grunerite dusts.[12] As a result of this recommendation and of the cited occupational health implications, NIOSH undertook a two-phase retrospective cohort mortality and industrial hygiene epidemiologic study of miners occupationally exposed to cummingtonite-grunerite at the Lead, South Dakota facility.

Retrospective Cohort Study

Methods

Selected for study were all male Caucasians who had been examined in 1960 by the U.S. Public Health Service during a silicosis survey of a hard rock gold mine in Lead, South Dakota and on whom social, demographic, and occupational data had been obtained in sufficient detail to permit follow-up of their health status. The study cohort was further restricted to the 440 individuals who in 1960 had achieved at least 60 months of underground mining at that facility and who had never mined underground elsewhere. Follow-up of all study group members was made from the time of initial examination in April 1960 to December 31, 1973. Vital status was determined through records maintained by federal, state, and local governmental agencies, including sources such as the Social Security Administration, state vital statistics offices, and state motor vehicle registration files. For individuals not located through these data sources, other data, such as city directories, post office mailing correction services, retail credit bureau searches, voter records, and various other local records were used. As a result of this follow-up program, no member of the study cohort was "lost-to-observation." Death certificates were obtained for those known to be dead, and causes of death were interpreted and classified by a qualified nosologist, according to the *Revision of the International Lists of Disease and Causes of Death* in effect at the time of death.

A modified life table technique was used to obtain person-years at risk of dying by 5-year calendar time periods, 5-year age groups, and number of years since onset of initial employment. Comparison was made between the observed number of deaths in the study cohort and the number expected on the basis of age, calendar time, and cause-specific mortality rates for the general white male population of South Dakota.

Results

As shown in TABLE 1, from April 1960 through December 31, 1973, a total of 71 deaths occurred among these gold miners, as contrasted with 52.9 expected deaths, an excess significant at $p < 0.05$. The distribution of these deaths was such that the only significant excess of mortality occurred in the respiratory system category. This excess was not specific to any response. Rather, it showed up both for respiratory malignancies (10 observed vs 2.7 expected, $p < 0.01$) and for respiratory nonmalignancies (8 observed vs 3.2 expected, $p < 0.05$). A significant excess of deaths also occurred in the category "other accidental deaths" ($p < 0.05$). Of the nine observed deaths in the "all other causes" category, 3 (33%) had mention of pneumoconiotic disease on the death certificate.

A more detailed analysis of respiratory disease mortality, both malignant and nonmalignant, by time interval since onset of underground employment at the mine, appears in TABLE 2. The mortality pattern in this Table shows an excess of nonmalignant respiratory disease in general (8 observed vs 2.56 expected, $p < 0.01$), and of pneumoconiotic disease specifically (5 observed vs 1.57 expected, $p < 0.05$), to have occurred after 20 years since onset of under-

TABLE 1

OBSERVED AND EXPECTED DEATHS ACCORDING TO CAUSE AMONG WHITE MALES WHO HAD 5 OR MORE YEARS OF UNDERGROUND MINING EXPERIENCE BY APRIL 1960, HARD ROCK GOLD MINE, SOUTH DAKOTA

Cause of Death	ICD	Total	
		Obs.	Exp.
Malignant neoplasms	140–205	15	9.7
Respiratory system	160–164	10	2.7 *
Other	140–159, 165 170–205	5	7.0
Vascular lesions of central nervous system	330–334	6	3.2
Diseases of the heart	400–443	25	25.2
Nonmalignant respiratory diseases	470–493 500–527	8	3.2 †
Influenza and pneumonia	480–493	3	1.3
Other respiratory diseases	470–479 500–527	5	1.9
Accidental deaths	800–962	8	5.2
Motor vehicle	810–835	0	2.5
Other	800–809 840–962	8	2.7 †
All other causes		9	6.5
Total		71	52.9 †

* Significant at $p < 0.01$.
† Significant at $p < 0.05$.

TABLE 2

OBSERVED AND EXPECTED DEATHS DUE TO MALIGNANT AND NONMALIGNANT RESPIRATORY DISEASE AMONG WHITE MALES WHO ACHIEVED 5 OR MORE YEARS OF UNDERGROUND GOLD MINING EXPERIENCE BY APRIL 1960, HARD ROCK GOLD MINE, SOUTH DAKOTA

		Number of Years Since Onset of Underground Employment			
		5–19 Years		≥20 Years	
Cause of Death	ICD	Obs.	Exp.	Obs.	Exp.
Malignant neoplasms	140–205	4	2.14	11	7.59
Respiratory system	160–164	3	0.56 *	7	2.18 †
Other	140–159, 165 170–205	1	1.58	4	5.41
Nonmalignant respiratory diseases	470–493 500–527	0	0.59	8	2.56 †
Influenza and pneumonia	480–493	0	0.31	3	0.99
Other respiratory diseases	470–479 500–527	0	0.28	5	1.57 *

* Significant at $p < 0.05$.
† Significant at $p < 0.01$.

ground gold mining. No such excess of nonmalignant respiratory disease risk was noted during the first 19 years after first exposure. A significant excess of respiratory tract cancer was demonstrable at each time interval since onset of underground mining: 3 observed vs 0.56 expected ($p < 0.05$) at less than 20 years and 7 observed vs 2.18 expected ($p < 0.01$) at 20 or more years since onset of such employment.

Analysis of the site of the 10 respiratory cancer deaths revealed that eight were primary bronchogenic carcinomas, one was a carcinoma of the maxillary sinus, and one was a mediastinal carcinoma (otherwise unspecified).

INDUSTRIAL HYGIENE STUDY

Occupational Exposures

In assessing what possible etiologic agent(s) contributed to the observed excess of deaths due to malignant and nonmalignant respiratory disease in this population, the environmental contaminants to which these miners were exposed must be considered. The ore body of the mine under study lies within metamorphosed precambrian sedimentary formations, in which the major mineral components are quartz, cummingtonite-grunerite, and sulfides, including arsenopyrites.[12] Other minor constituents of the ore are siderite, biotite, ankerite, chlorite, and hornblends.

Environmental conditions at this mine have been the subject of several industrial hygiene investigations. In 1960, the Bureau of Mines, during its silicosis study, collected impinger samples to evaluate free silica exposures to underground miners.[13] These samples showed an average airborne dust concentration of 1.7×10^6 particles per cubic foot in miners' breathing zones, whereas airborne and settled dust samples showed an average free silica content of 39%. Given a 39% free silica content, the Occupational Safety and Health Administration (OSHA) 8-hr time-weighted average standard for occupational exposure to free silica would be approximately 5×10^6 particles/ft^3. Thus, free silica exposures were well below the present applicable OSHA standard. Semiquantitative x-ray spectographic analysis of an underground settled dust sample taken during this survey for arsenic, chromium, and nickel showed trace concentrations (<0.01%). Radon daughters were not detected as sampled with instruments that had a lower limit of detection of 0.01 working levels, a level within the range of general ambient indoor residential exposure.

In 1973, the Bureau of Mines,[14] under its Mine Enforcement and Safety Administration (MESA) program, conducted further environmental studies at this mine. With personal samplers, a total of seven working shifts were sampled for respirable and total dust concentrations. X-ray diffraction analysis of the respirable dust showed an average free silica content of 13.1%. All samples of mining personnel exposures showed average breathing zone dust concentrations

TABLE 3

RESULTS OF MICROSCOPIC IDENTIFICATION OF AIRBORNE FIBERS BY MEANS OF ELECTRON DIFFRACTION AND X-RAY SPECTROMETRY, HARD ROCK GOLD MINE, SOUTH DAKOTA

Amphibole by Electron Diffraction (%)	Fibrous Grunerite (Amosite) (%)	Fibrous Cummingtonite (%)	Fibrous Hornblend (%)	Unknown (%)
80–90	60–70	1–2	10–15	≃20

below the OSHA standard for free silica. In addition to air samples, material samples of the ore were analyzed for asbestos by scanning electron microscopy by means of energy-dispersive x-ray techniques. Numerous fibrous particles were observed. Their identification was not determined, however.

Concern for possible worker exposure to asbestiform minerals at this mine then spurred a 1974 investigation by MESA.[15] During that study, approximately 200 personal membrane filter samples were collected over full working shifts. Fiber concentrations were determined with the NIOSH phase-contrast optical microscope analytic method for asbestos fibers. The average fiber concentration was found to be 0.25 fibers greater than 5 μm in length/cm^3; the highest single concentration found was 2.8 fibers/cm^3.

In addition, the content of arsenic, chromium, and nickel compounds of the ore at this time was quantified by x-ray fluorescence analysis to be less than 0.015, 0.035, and 0.016%, respectively.

To perform further analysis of the fiber exposures at this mine, portions of all 200 air samples collected by MESA during this environmental survey were made available to NIOSH. Twenty-five of these 200 air samples with the highest fiber concentrations, as determined by optical microscopy, were selected

TABLE 4

AIRBORNE FIBER CONCENTRATIONS AS DETERMINED BY ELECTRON MICROSCOPE FIBER COUNTS AT 10,000 × MAGNIFICATION, HARD ROCK GOLD MINE, SOUTH DAKOTA (N=22)

Exposure Measure	Fiber Concentrations (fibers/cm³)	
	Total Fibers	Fiber >5 μm
Mean ± SE	4.82±0.68	0.36±0.08
Range	0.66±11.79	0.07±1.29

by NIOSH and were analyzed by analytic transmission electron microscopy via selected-area electron diffraction and energy-dispersive x-ray analysis to determine fiber identification, concentration, and size distributions.

TABLE 3 shows the results of the NIOSH electron microscope identification of airborne fibers present in this mine. As can be seen, 80–90% of the airborne fibers were fibrous amphiboles, and 60–70% of the latter were fibrous grunerite (amosite). Fibrous cummingtonite comprised 1–2% of the amphibole fibers, and fibrous hornblends constituted an additional 10–15% of these fibers. Approximately 20% of the fibers were either too thin or too thick to give definitive amphibole diffraction patterns by direct observation on the electron microscope viewing screen. A large majority of these fibers, however, had x-ray spectra identical to those of amosite asbestos.

Average airborne fiber concentrations in this mine, as determined by electron microscopy, are shown in TABLE 4. With the transmission electron microscope, total fiber concentrations were found to average 4.82 fibers/cm³, whereas concentrations of fibers greater than 5 μm in length averaged 0.36 fibers/cm³. In addition, the median fiber diameter and length were found to be 0.13 and 1.1 μm, respectively; approximately 94% of the airborne fibers were less than 5 μm in length. Summary statistics for the fiber size analyses are shown in TABLE 5.

In addition to asbestos fiber analyses of these samples, energy dispersive x-ray analyses were performed on airborne particles to determine the form of arsenic present in the airborne dust. All particles so analyzed were found to be arsenopyrite (FeAsS) at extremely low concentrations.

TABLE 5

SUMMARY OF AIRBORNE FIBER SIZE DISTRIBUTIONS AS DETERMINED BY ELECTRON MICROSCOPY, HARD ROCK GOLD MINE, SOUTH DAKOTA

Summary Statistic	Diameter	Length
Count median size, N=2111 (μm)	0.13	1.10
Geometric Standard Deviation *	3.13	2.70
95% confidence interval for count median size (μm)	0.128–0.141	1.07–1.15

* Data fitted to log normal distributions by probit analysis and linear regression (γ >0.90).

Discussion

Engineering controls in this hard rock gold mine have been quite good for many years. During its 1960 study,[13] the Bureau of Mines observed extensive use of mine ventilation, wet drilling, and wetting of the work site to control dust exposures. Ore pockets and transfer points were also equipped with water sprays. The effectiveness of these controls is apparent by observation of the low free silica exposures and radon daughter air levels measured during the 1960 survey [13] and the MESA 1973 study.[14]

These methods would have been equally effective in controlling exposures to all dusts, including asbestos fibers, compounds of arsenic, and trace metals. Based on observations of present and past use of engineering controls and their effectiveness, as demonstrated by the Bureau of Mines 1960 [13] and 1973 [14] dust surveys, it may be adjudged that past exposures to asbestos fibers were not significantly different from those found by the 1974 MESA survey.[15] Further evidence to support low present and past airborne exposures to arsenic and trace metals are the low arsenic and trace metal contamination levels found in mine ore and settled dust samples [13, 14] and the low respirable mass concentrations measured by the Bureau of Mines in 1973.[14]

The role of such extremely low levels of radon daughters in the etiology of respiratory tract cancer has been found to be noncontributory, as demonstrated previously among underground potash miners.[16]

The role of cigarette smoking also must be taken into account. Smoking histories obtained during the 1960 silicosis study of this hard rock gold mine indicated that these miners smoked far less than did underground uranium miners. Although a known factor in the causation of lung cancer, it has been estimated in studies of underground uranium miners that such smoking by itself would increase the expected lung cancer death risk by no more than 49%.[17] Cigarette smoking, therefore, could not account per se for the observed increased respiratory cancer risk among these gold miners. Consequently, exposure to fibrous grunerite (amosite) stands out as the prime etiologic agent for the increased risk of respiratory cancer among this study cohort.

In addition to the occupational health implications of the above findings, parallels may be drawn between these findings and potential health effects on general, nonoccupational populations as a result of exposure to similar industrial waste and out-plant process effluents. At hearings for a Minnesota mining company and its disposal of taconite tailings and process discharges, data were presented on airborne asbestos concentration measurements made in the vicinity but not in the property of that company. Those samples had been collected by the U.S. Environmental Protection Agency and analyzed by the Mount Sinai School of Medicine, City University of New York by means of electron microscopy.[18] Table 6 presents a summary of those results and their comparison to the airborne concentrations found in the hard rock gold mine in South Dakota. The similarity both of the concentrations observed in these studies and of the relative prevalence of short fibers is striking: a mean concentration of 4.8 fibers/cm^3 was noted in the South Dakota mine and 4.7 fibers/cm^3 in the vicinity of the Minnesota company. Six percent of the fibers in the latter mine were longer than 5 μm in length, whereas 5–10% were longer than 5 μm in the former area. In addition, the fibers from both facilities have been shown to have identical compositions (fibrous grunerite).

Conclusions

A study among a group of miners exposed to amphibole fibers (amosite) in the cummingtonite-grunerite series, airborne concentrations less than 2.0 fibers/cm^3 as determined by the NIOSH phase-contrast counting technique, and fibers shorter than 5 μm in length has demonstrated significant risks of mortality from both malignant and nonmalignant respiratory disease. Exposures to known carcinogens in the mine, other than asbestos, did not exceed normal ambient residential levels for radon daughters or were adjudged to be negligible for arsenic, chromium, and nickel. The observed excess of malignant respiratory disease can, therefore, be attributed to asbestos, singly or in combination with cigarette smoke, and that of nonmalignant respiratory disease can, therefore, be ascribed to asbestos, with a possible additive role from low exposures to free silica dust.

The findings of this study point to the need for reevaluation of the adequacy of the OSHA standard of two fibers longer than 5 $\mu m/cm^3$, of the deleterious health effects of asbestos fibers shorter than 5 μm vis-à-vis any standard for occupational or environmental exposure to asbestos, of the adequacy of the present NIOSH asbestos sampling and analytic methods, and of the potential adverse health effects among general, nonoccupational populations exposed to industrial waste disposals and industrial process effluents that contain trace concentrations of short-fiber noncommercial asbestos minerals.

Table 6

Comparison of Airborne Asbestos Fiber Concentrations in the Vicinity of a Minnesota Mining Company with Those Observed in the Study Mine, South Dakota

Source	Total Fiber Concentration (fiber/cm^3) Mean	Range	% >5 μm
Study mine ($N=22$)	4.8	0.7–11.8	6
Silver Bay ($N=6$)	4.7	0.5–11.0	5–10

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