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DISCUSSION PAPER: ASBESTOS FIBER EXPOSURES
IN A HARD ROCK GOLD MINE

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INTRODUCTION

The study reported by Gillam *et al.*¹ demonstrated a significantly increased risk of both malignant and nonmalignant respiratory disease among a cohort of miners at a hard rock gold mine in Lead, South Dakota. That study also reported data that showed low-level exposures to fibrous grunerite (amosite) in the mine. The following paragraphs describe in further detail the laboratory analyses undertaken by the National Institute for Occupational Safety and Health (NIOSH) to determine the fiber exposures reported by Gillam *et al.*

METHODS AND MATERIALS

In 1974, during the industrial hygiene survey conducted at the subject mine by the U.S. Bureau of Mines' Mining Enforcement and Safety Administration (MESA), approximately 200 air samples were taken to determine 8-hr time-weighted average fiber exposures, as analyzed by the NIOSH phase-contrast optical microscope counting technique.² The average fiber concentration was found to be 0.25 fibers greater than 5 μ m in length per cubic centimeter of air, and the highest single concentration was 2.8 fibers/cm³. No fiber identification was done in that study, however.

To further characterize the fiber exposures in the subject mine, portions of all of the 200 air samples collected by MESA during its survey were made available to NIOSH. Twenty-five of the underground personnel personal air samples with the highest fiber concentrations, as determined by MESA by phase-contrast optical microscopy, were chosen for detailed analysis by analytic electron microscopy. The membrane (Millipore type AA) filter samples were mounted on formvar and carbon-coated copper electron microscope grids (200 mesh) by means of the direct clearing method, as described by Fraser.³ Samples for scanning electron microscope analysis were further coated with a thin (100-150 Å) gold layer by vacuum evaporation.

A JEOL, JEM 100B transmission/scanning electron microscope equipped with an EDAX energy-dispersive x-ray spectrometer (specimen to detector distance of approximately 10 mm) was used for all analyses. This instrument allowed for simultaneous observation of single-fiber electron diffraction patterns

* Mention of commercial products does not constitute endorsement by NIOSH.

and fiber chemical composition by x-ray microanalysis. An acceleration voltage of 100 kV was used for transmission electron microscopy, and 20 kV was used for scanning electron microscopy.

Two criteria were used as necessary and sufficient data to classify fibers in these air samples: observation of the single-fiber electron diffraction pattern and observation of semiquantitative elemental analysis spectra with x-ray microanalysis techniques.⁵

Typical fibers, electron diffraction patterns, and x-ray spectra for fibers in the air samples and appropriate amphibole fiber standards were photographed; integrated x-ray counts from the x-ray spectra were recorded for further analysis. Cleavage patterns for typical fibers in mine-settled dust samples were observed and then photographed with scanning electron microscopy. Approximately 250 separate fiber analyses were conducted.

Airborne fiber concentrations and size distributions were determined by counting and sizing fibers directly on the microscope's viewing screen at a magnification of 10,000 $\times$. Fiber diameter and length were simultaneously determined by comparison with calibration marks engraved on the screen. Counting-field areas were defined by means of the electron grid openings, the areas of which were calibrated. A total of 2111 fibers were counted and sized.

RESULTS

FIGURE 1 shows a typical micrograph of fibers seen in a grunerite standard, obtained from the Smithsonian Institution, along with the single-fiber amphibole electron diffraction pattern and x-ray spectra for the same fiber. FIGURE 2 shows the same data for a typical fibrous grunerite (amosite) fiber seen in the air samples collected in the hard rock gold mine. This fiber demonstrates a chemical composition (x-ray spectrum) identical to UICC amosite and a definite amphibole diffraction pattern. These data demonstrate the presence of amosite asbestos in the mine's work atmosphere.

Other fibers, defined as particles with an aspect ratio of at least three to one, identified in these samples include cummingtonite and hornblends. TABLE 1 shows the relative proportion of the various fibers observed. As can be seen, 80-90% of the airborne fibers demonstrated an amphibole electron diffraction pattern. Of these amphiboles, 60-70% had chemical compositions identical to that of fibrous grunerite (amosite), 1-2% were fibrous cummingtonite, and 10-15% had compositions of common hornblends, that is, aluminum in the fiber structure.

TABLE 1
RESULTS OF MICROSCOPIC IDENTIFICATION OF AIRBORNE FIBERS
BY ELECTRON DIFFRACTION AND X-RAY SPECTROMETRY,
HARD ROCK GOLD MINE, SOUTH DAKOTA

Amphibole by Electron Diffraction (%)	Unidentified by Electron Diffraction (%)	Percentage of Amphibole Fibers in Each Category		
		Grunerite	Cummingtonite	Hornblends
80-90	~20	60-70	1-2	10-15

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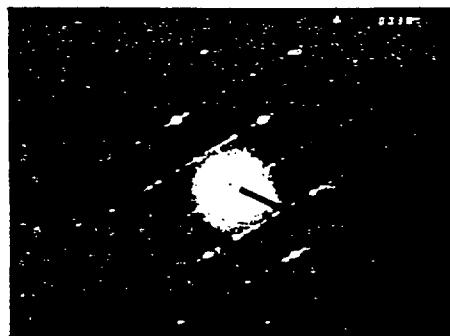
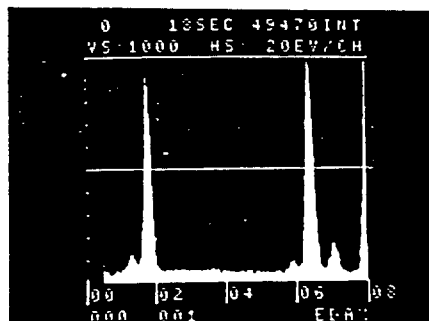


FIGURE 1. Grunerite standard. *Top*, electron micrograph ($\times 10,000$); *middle*, amphibole diffraction pattern; *bottom*, x-ray spectrum.



Approximately 20% of the airborne fibers were either too thick or too thin to give identifiable diffraction patterns and were, therefore, not classified. However, many of these fibers also had chemical compositions identical to that of amosite asbestos.

The fibrous nature of the amphibole particles was also demonstrated by using scanning electron microscopy. FIGURE 3 shows scanning micrographs of a settled dust sample that possesses fibers with amphibole cleavage patterns, namely, cleavage parallel to length.

Results of the fiber concentration measurements are shown in TABLE 2. By

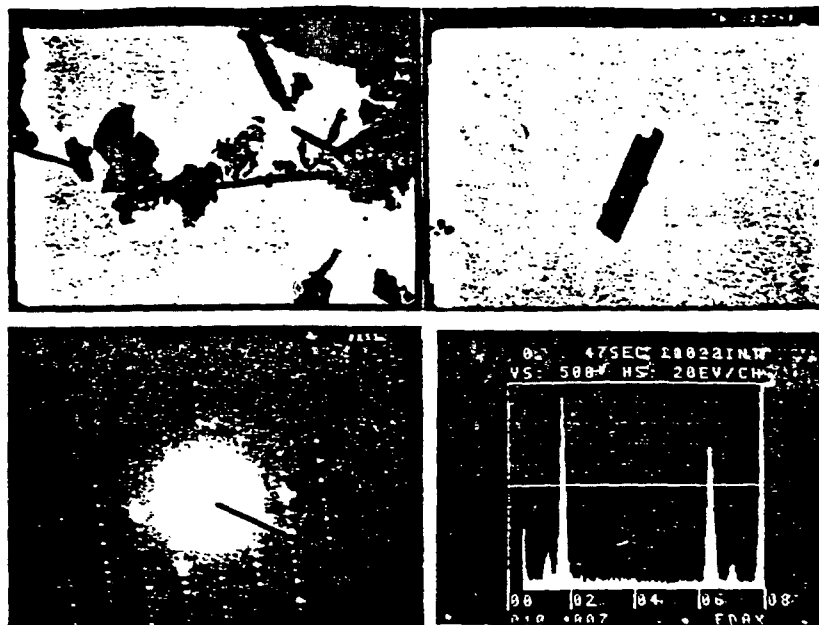


FIGURE 2. Fibrous grunerite (amosite) present in air sample. Top, electron micrographs ($\times 10,000$); bottom left, amphibole diffraction pattern; bottom right, x-ray spectrum.

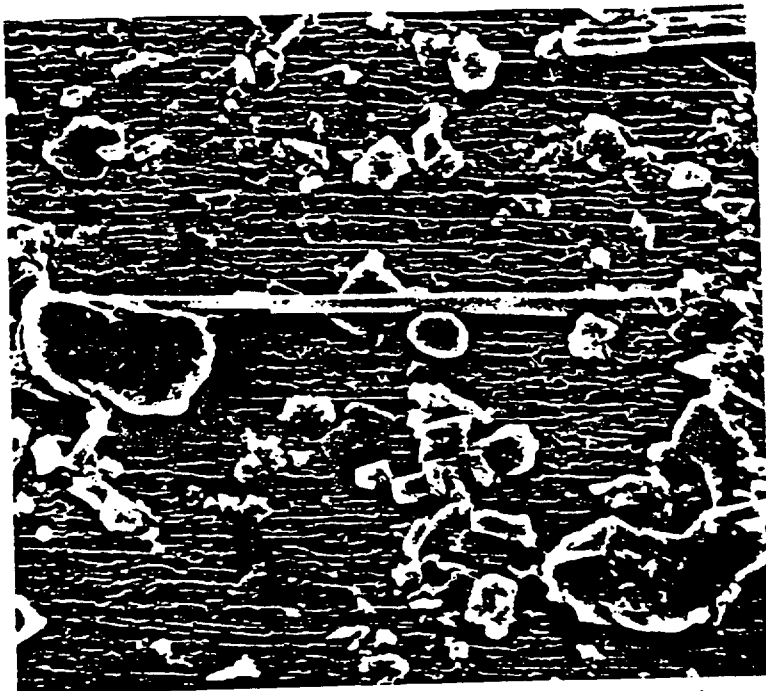
use of electron microscopy, fiber concentrations were seen to be low, with an average concentration of 0.36 fibers/cm³ greater than 5 μ m in length. The average total fiber concentration was found to be 4.82 fibers/cm³. Airborne fiber length and diameter data, as determined by electron microscopy, are shown in TABLE 3. Median fiber diameter and length were found to be 0.13 and 1.1 μ m, respectively; 94% of the fibers were less than 5 μ m in length.

DISCUSSION

Fibrous grunerite (amosite) has been shown to be present at low concentrations in this hard rock gold mine; most fibers are shorter than 5 μ m in length. TABLE 4 compares fiber length distributions of fibers from this mine to those from other asbestos-using industrial operations. As can be seen, the proportion of fibers longer than 5 μ m in length ranges from 1 to 51%, depending on the operation and on the type fiber present. It is now well recognized that the NIOSH phase-contrast fiber-counting method for asbestos, in which only fibers longer than 5 μ m in length are counted, represents only an "index" of the true

FIGURE 3. Scanning electron micrographs of amphibole fibers in settled dust samples ($\times 3000$).

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TABLE 2
AIRBORNE FIBER CONCENTRATIONS AS DETERMINED BY ELECTRON MICROSCOPIC
FIBER COUNTS AT 10,000 $\times$ MAGNIFICATION,
HARD ROCK GOLD MINE, SOUTH DAKOTA *

Measure	Total Fibers	Fibers > 5 μ m
Mean $\pm$ SE	4.82 $\pm$ 0.68	0.36 $\pm$ 0.08
Range	0.66-11.79	0.07-1.29

* Fiber concentrations in mine, average of 22 samples (fibers/cm³).

TABLE 3
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, (μ m)	0.13	1.10
Geometric standard deviation	3.13	2.70
95% confidence interval for count median size (μ m)	0.128-0.141	1.07-1.15

TABLE 4
COMPARISON OF AIRBORNE FIBER LENGTH DISTRIBUTIONS
IN VARIOUS ASBESTOS OPERATIONS

Operation	Fiber Type	Median Length	% > 5 μ m
Textile *	chrysotile		
Fiber preparation and carding		1.4	4
Spinning, twisting, weaving		1.0	2
Friction *	chrysotile		
Mixing		0.9	2
Finishing		0.8	2
Asbestos-cement pipe *	chrysotile		
Mixing		0.9	2
Finishing		0.7	1
Pipe insulation †	amosite		
Pipe forming		4.9	51
Gold mining	amosite		
Miners		1.1	7

* From Reference 6.

† Unpublished NIOSH study.

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Section I

INTRODUCTION

The Scope of Work for Work Order 1.9 requires that Northrop Services, Inc., provide the services, facilities, and materials (except government furnished equipment) necessary to determine the health effects of air pollutants on laboratory animals from toxic compounds such as asbestos and asbestos-like particles.

Section II contains background information on the properties of asbestos and asbestos-like particles and the epidemiological and experimental history of asbestos toxicity in man and animals, as well as the study objective and research approach to be used in future studies. Section III includes the engineering design describing the capabilities of the facilities. Section IV describes the laboratory procedures in detail.

Section II

TECHNICAL PROTOCOL

This section consists of two basic parts. The first part contains background information on the properties of asbestos and asbestos-like particles and the epidemiological and experimental history of asbestos toxicity in man and animals. The second part contains the study objective and the research approach to be used in future studies.

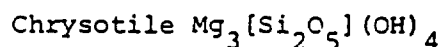
2.1 ASBESTOS

More than nine-tenths of the earth's crust is comprised of silicates. Of the 30 mineral silicates which can crystalize in a fibrous form, some are called asbestos.¹ Mineralogically, asbestos is divided into two classes: serpentine, characterized as a sheet-shaped formation of silicate; and amphibole, built up of double chains of tetrahedral formations.

The amphiboles constitute an important group of rock-forming minerals which are estimated to comprise as much as 3 percent of the earth's crust and are associated with both igneous and metamorphic rocks. Amphiboles are divided into three groups: iron-magnesium (for example, anthophyllite and amosite), calcium (for example, tremolite and actinolite), and alkali (for example, crocidolite).¹

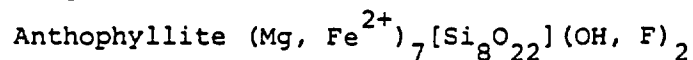
The chemical compositions of serpentine and amphibole are:

Serpentine

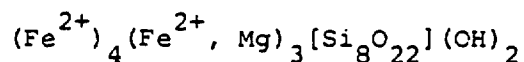


Amphibole

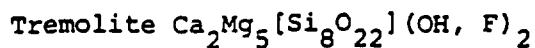
Iron-magnesium



Grunerite (Amosite)

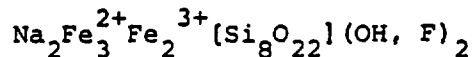


Calcium



Alkali

Riebeckite (crocidolite)



2.2 PATHOGENICITY IN MAN

Long-term epidemiological studies have now revealed beyond a reasonable doubt that inhalation of asbestos dust can lead to pulmonary fibrosis and carcinoma of the lung and diffuse mesotheliomas of the pleura and peritoneum. Most data have been reported by groups associated with various asbestos-involved occupations. These professions include mining and milling operations, manufacture of asbestos-containing products (textiles, construction materials, and so forth), and jobs involved with application or removal of asbestos-containing insulating materials. Also, sufficient data on populations residing in areas adjacent to these industries are available.

Although asbestosis, a diffuse interstitial fibrosis of the lung, was first reported by Murray in 1907,² its relationship to human exposure to the mineral was not clearly recognized until 1920. The disease has since been investigated in epidemiological studies in several countries. Among the countries regarded suitable for such studies are the following: Australia (crocidolite), Canada (chrysotile), Cyprus (chrysotile), Finland (anthophyllite), Italy (chrysotile), South Africa (amosite, chrysotile, and crocidolite), United States (chrysotile and tremolite), and the Soviet Union (chrysotile). All forms of asbestos have been shown capable of producing asbestosis.³ Studies evaluating comparisons of effects associated with various sizes and types of fibers have been carried out with workers in mining operations, as well as people residing in areas adjacent to such sites. By investigating the one type of fiber mined at a particular site, comparisons between mines have been made possible.

Pleural plaques are non-neoplastic lesions consisting of collagen arranged to give a characteristic "basket-weave" appearance which ceases abruptly at the

plaque margin. Pleural plaques are associated with occupational as well as nonoccupational exposure to asbestos. Epidemiological studies show that 40 percent of the population over 15 years of age in locations where anthophyllite was mined in Finland had pleural plaques.^{4,5} Asbestos bodies were present in the lungs of 86 percent of the subjects with bilateral plaques. It was noticed, however, that if asbestos were present in abundance, plaques were regularly encountered.

Pleural plaques were also found in asbestos workers in Dresden^{6,7}, London⁸, Bulgaria⁹, and in Czechoslovakia¹⁰. Pleural calcification was observed in asbestos insulators in Copenhagen¹¹, New York City¹², and in Belfast¹³, as well as in nonoccupationally exposed populations living in the neighborhood of anthophyllite mines in Finland¹⁴, crocidolite and amosite mines in South Africa¹⁵, and chrysotile mines in Italy⁷⁸.

The first suggestion that asbestos might be causally related to cancer of the lung was made by Gloyne in England in 1933¹⁶ and Lynch and Smith in the United States in 1935.¹⁷ However, it was not until 1947 that Merewether¹⁸ proposed that asbestosis might be complicated by bronchial carcinoma. He reported 31 instances of cancer of the lung in 235 persons who died of asbestosis between 1924 and 1946. In 1951, Gloyne¹⁹ further reported that out of 132 asbestos workers, 121 who had asbestosis also had cancer of the lung. The clear proof of the association between asbestosis and bronchogenic carcinoma came in a study by Doll.²⁰ He analyzed the causes of death among 105 men with histories of at least 20 years of work in asbestos textile plants. He found 18 cases of lung cancer, a figure which is 10 times higher than that for the general male population. In a recent analysis, Selikoff, Hammond, and Seidman²¹ found that bronchogenic carcinoma accounted for approximately 45 percent of fatal neoplasms among asbestos insulation workers. It was suggested by the Senior Medical Inspectors Advisory Panel in 1967 that bronchial carcinoma is a complication of asbestosis rather than a result of exposure to asbestos.²² British asbestos workers have exhibited no excess of bronchogenic cancer in the absence of asbestosis. In the United States, however, an excess mortality from bronchogenic carcinoma has existed in the comparative absence of asbestosis in

certain groups of workers exposed to asbestos.^{23,24} In South Africa, bronchogenic carcinoma occurs in all South African asbestos areas.²⁵ These data have further been confirmed by several other investigators through epidemiological studies.^{23,26-38}

It is now believed that there is a relationship between cigarette smoking and exposure to asbestos and incidences of bronchogenic cancer. Selikoff, Hammond, and Churg³² demonstrated that asbestos insulation workers with a history of regular cigarette smoking suffered eight times the risk of lung cancer compared with cigarette smokers who were not exposed to asbestos and 90 times more than men who neither worked with asbestos nor smoked cigarettes. This effect was confirmed in a further study³⁹. Exposure to asbestos did not seem to lead to an extremely high risk of bronchogenic cancer among nonsmokers. Also, Berry, Newhouse, and Turok⁴⁰ examined mortality from lung cancer in the asbestos textile industry over a 10-year period and compared them with the number of deaths due to lung cancer in the general population of the area. No significant excess was found in asbestos workers, whether smokers or nonsmokers, with low to moderate exposure; but among workers who smoked and who were severely exposed, the excess was highly significant.

The occurrence of cancers of the peritoneum and pleura (mesotheliomas) in association with asbestos was first reported in 1933 by Gloyne,¹⁶ and then in 1960 by Keal⁴¹ and Wagner⁴². In 1963, Wagner⁴³ collected 120 cases of mesotheliomas of the pleura which were confirmed by biopsy or autopsy. A large proportion of cases had been exposed to crocidolite in the Northwestern Cape. He also noticed that more than half of the group had never worked in the industry but had lived in the vicinity of mines and mills. Statistical evaluation by Oettlé²⁸ reveals that the people in the Northwestern Cape exposed to crocidolite have a distinct risk of developing mesotheliomas. The Northeastern Transvaal, where amosite and crocidolite are mined, were in the low risk area. To date, only a few cases of mesothelioma with possible past exposure to amosite in the region have been reported. Among those cases are the ones who were exposed solely to amosite in the United States and who had bronchogenic cancer and mesothelioma. No mesotheliomas have been reported due to anthophyllite from Finland^{44,46}. Chrysotile seems to be the most actively involved etiological agent in the United States^{30,31,34} and in Canada³⁸. However, in

Swaziland and Rhodesia, where chrysotile is mined and milled, no cases of mesotheliomas have been reported⁴⁷. Similar results have been reported in the U.S.S.R., Italy, South Africa, and Cyprus³⁸. It is now accepted that mesothelioma can occur after a brief exposure to asbestos. In some cases, there was only a brief exposure in homes or simply as a result of living in the vicinity of asbestos factories or mills⁴⁸. To date, there is no association between cigarette smoking and production of mesothelioma.

2.3 PATHOGENICITY IN ANIMALS

The current data linking asbestos exposure to cancer in man has precipitated considerable experimentation with animals. Lesions such as fibrosis of lung, squamous cell carcinoma of lung, and mesotheliomas have been produced in several species of animals by administration of asbestos particles. As in the case of human exposure, attention to fiber type and size must be taken into account. In addition, one must consider the variables augmented by several species and strains of animals.

A causal relationship between exposure to asbestos and pulmonary fibrosis in animals has been demonstrated by several investigators.^{3,43,49-52} To date, the exact properties of the minerals that cause severe tissue damage and eventual fibrosis have not been adequately established. Miller and Harrington⁵³ have noted an increase in the amount of lysosomal enzymes after administration of experimental asbestos and suspect a causal relationship. The fibrogenic effect is not unique for asbestos. Davis⁵² found that mineral fibers beside chrysotile, fibrous brucite, silica fiber, glass fiber, man-made insulation fibers, and other minerals (chlorite, chromite, forsterite, magnetite, olivine, pyronone, and taconite) all produced granulomas in the pleural cavities of mice. There was, however, a great variation in the magnitude of cell responses. For example, long-fiber samples produced large cellular granulomas that were eventually replaced by large amounts of fibrous tissue, and short-fiber samples or nonfibrous dust and finely ground dust revealed much less reaction.

Wagner⁴³ observed asbestosis in guinea pigs that had inhaled chrysotile dust over a period of 2 years. Holt, Mills, and Young⁵¹ found that guinea pigs that had inhaled very fine particles of chrysotile asbestos showed a

well-developed bronchiolitis after a few days. Phagocytic multinuclear giant cells and monocytes were common features. After 30 weeks, there was cell degeneration and fibrosis. Collagen appeared first as a lining to respiratory bronchioles and alveolar ducts. Numerous asbestos bodies appeared later. Similar lung damages were produced by crocidolite, amosite, and anthophyllite. Asbestosis is experimentally produced in rats⁵⁴⁻⁵⁸, in hamsters⁷³, and in rabbits and monkeys⁴³.

The direct effect of exposure to asbestos and the synergistic effect of benzo(a)pyrene (BaP) (a known component of cigarette smoke) has been explored through animal experimentations by several investigators.⁵⁹⁻⁶² Miller, Smith, and Berliner⁵⁹ demonstrated that when amosite and chrysotile were injected intratracheally into hamsters, they produced squamous cell carcinomas of the lungs. When 5 milligrams of BaP was injected in conjunction with amosite, there was no increase in tumor production. When chrysotile was injected with the same dose of BaP, however, there was a considerable increase in tumor production. Vôsomaë⁶⁰ confirmed the promoting action of chrysotile established by Miller, Smith, and Berliner.⁵⁹ In Vôsomaë's studies, rats were administered intratracheal injections of BaP together with chrysotile (UICC standard reference sample). He demonstrated a considerably higher yield of malignant lung tumors in rats. Pylev⁶¹ obtained a similar effect with chrysotile and BaP. Recently, chrysotile from Ural mines was tested by injecting particles intratracheally into lungs of rats.⁶² Results revealed that when the rats were treated with chrysotile alone, an insignificant number of the animals developed precancerous lung lesions and no tumors were found. Analogous injections of chrysotiles together with adsorbed BaP resulted in a greater number of precancerous lung lesions, such as squamous metaplasia, reticulosarcoma and diffuse irregular hyperplasia of pleural mesothelium, and focal adenomatous growth of pleural mesothelium. The rats treated with chrysotile and BaP produced a larger number of precancerous lesions as well as lung papillomas, squamous cell carcinomas, and pleural mesotheliomas. The rats subjected to BaP alone produced precancerous lesions, but no tumors.

Diffuse mesotheliomas of the pleura have been produced by several investigators by means of intrapleural injections of asbestos. Wagner^{63,43} demonstrated that amosite, chrysotile, and crocidolite can produce mesothelial tumors in hamsters when 50 milligrams of dust is injected into the pleural cavity. Peacock and Peacock⁶⁴ described a similar effect in fowl after injecting crocidolite, amosite, chrysotile, and tremolite. Later, Smith and Hubert⁶⁵ successfully produced mesotheliomas in hamsters with amosite and chrysotile.

Mesotheliomas have been produced by intraperitoneal injections. Scheuer, Hunth, and Pott⁶⁶ demonstrated in separate experiments that when UICC samples of amosite, anthophyllite, chrysotile A, and crocidolite were injected intraperitoneally, over 50 percent of the animals produced various diseases. These maladies include multicentric and malignant abdominal tumors such as fibrosarcomas, sarcomas, mesotheliomas, reticulum cell sarcomas, and adenocarcinomas. An effort was also made to investigate whether there is any combined effect of asbestos and BaP as in intratracheal inoculation. The data failed to show such a relationship via intraperitoneal injections. Malignant mesotheliomas were induced by single intraperitoneal injection of chrysotile or crocidolite asbestos fibers⁶⁷. Davis⁶⁸ also produced mesotheliomas via intraperitoneal injections of crocidolite asbestos into rats and mice.

Comparative study of different UICC amphibole samples with different size was made by Wagner and Berry.⁶⁹ They used diameter distribution involving a decreasing order of fineness in crocidolite, amosite, and anthophyllite. The study demonstrated that anthophyllite produced the least number of mesotheliomas. Their interpretation of this study was that for a given weight of asbestos inoculated into the pleural cavity, anthophyllite provided fewer fibers than amosite, which in turn provided fewer fibers than crocidolite. Since chrysotile fibers are shaped like coils and amphiboles are shaped like needles, it is difficult to assess the state of chrysotile fibers in animals and to compare this with that of the amphiboles. Nevertheless, they pointed out that mesotheliomas are associated with the implantation of fine fibers in cells. The finer the fiber, the more tumors are produced.

Stanton and Wrench⁷⁰ and Stanton⁷¹ produced mesotheliomas by implanting a fiberglass sheet impregnated by test particles into the thoracic cavities of rats. They introduced UICC standard reference samples of crocidolite and chrysotile A, two samples of fine fibrous glass with diameters of 3 microns or less, and aluminum oxide whiskers. They demonstrated that all produced mesotheliomas. The ability of glass fibers to produce mesotheliomas has also been shown by Pott and Friedrichs.⁷² The authors suggest that the carcinogenicity is related to the ability of fibers to interact with the cell membrane without destroying the cell.

Respiratory tumors have also been produced in rats by chrysotile inhalation in work by Gross et al⁷³. The animals surviving more than 10 months had adenocarcinomas, fibrosarcomas, squamous cell carcinomas, and mesotheliomas. The actual carcinogens responsible for these effects were ascribed by the authors⁷³ to increased amounts of trace metals introduced in or on the fiber during hammer-milling of the specimens.

Recently, Wagner et al⁷⁴ studied the effects of UICC standard reference samples, amosite, anthophyllite and crocidolite, Canadian samples of chrysotile, and Rhodesian samples of chrysotile on the lungs of rats. Significant differences were observed between various samples. Inhalation of anthophyllite and Canadian chrysotile resulted in the most asbestosis after exposure of 6 months or longer. Amosite resulted in the least asbestosis. Crocidolite and Rhodesian chrysotile produced intermediate results. An analysis was carried out to determine whether there was any relationship between the severity of asbestosis and the presence of lung tumors. It was found that the animals with lung tumors had significantly more asbestosis than those without. No tumors of the lung were observed within 300 days of the start of exposure. Adenocarcinoma and squamous cell carcinoma with some metastases and mesotheliomas were observed.

The amount of chrysotile retained in the lungs did not show any clear increase with increased dose in rats exposed for longer than 3 months. The weight of asbestos found in the lungs of rats exposed to amphibole was three

times greater than in those exposed to chrysotile. In spite of this fact, there was no evidence of either less carcinogenicity or less asbestosis in the groups exposed to chrysotile than those exposed to the amphiboles. UICC Canadian chrysotile produced as many mesotheliomas as the UICC crocidolite. Wagner et al⁷⁵, however, showed that after intrapleural inoculation, the risk of a mesothelioma occurring with UICC crocidolite is three times the risk incurred with chrysotile. No tumors were found in sites other than lungs.

2.4 STUDY OBJECTIVE

Massive amounts of noncommercial asbestos are found in the United States in mines used as sources of other minerals which have commercial value. The epidemiological and experimental data presented earlier give a clear indication of adverse health effects associated with exposure to asbestos. These data also raise an awareness that the health hazards may not be unique to commercial forms, but may also be associated with asbestos-like fibers found in other rocks and in man-made fibers. The most important question in the case of persons with nonoccupational exposures to asbestos and other amphiboles is whether there is an increased risk of malignancies. Since amphibole rock is estimated to comprise about 3 percent of the earth's crust, human beings may be at risk in many mining, quarrying, tunneling, and other hard-rock mining activities. Furthermore, non-occupational exposure is possible by means of dust generated by rock crushing and milling as well as from tailing of mine wastes gaining access in the ambient air and water.

One of the mines containing amphibole matrices which is currently being exploited for taconite ore is the Reserve Mining Company's Peter Mitchell Pit in Minnesota. It was questioned by the Environmental Protection Agency (EPA) whether such a project is hazardous to the health of the exposed population in the surrounding community and litigation ensued. The defense in the EPA vs Reserve Mining case claimed that although the fibers were similar chemically to amosite asbestos, they were not amosite asbestos, stating that the fibers were too small to be regarded as asbestos fibers. Further, the mining company's representatives contended that there were insufficient medical data for the fibers to be regarded as a health threat.

It is pertinent that the potential toxicity of materials released in the mining and processing of taconite ore is yet to be determined. Such investigation should consist of studies of the physio-chemical properties of amphiboles and their biological effects with respect to geological classification. Moreover, this information should be of value not only to the specific problem associated with the particular taconite mine, but also should provide a means of judgment when similar problems arise from other mines and rock moving and processing generally.

2.5 RESEARCH APPROACH

The purpose of the proposed investigation is to evaluate whether amphiboles and other minerals processed during extraction and refining of taconite ore from Peter Mitchell Pit (PMP) are carcinogenic and/or cocarcinogenic. PMP particles and UICC ~~chrysotile~~^{amosite} particles will be administered to animals via intratracheal and intrapleural injections. The biological effects due to the PMP particles will be compared with those obtained by UICC ~~chrysotile~~^{amosite} samples in the presence and absence of a known carcinogen, benzo(a)pyrene. The following criteria will be used to evaluate such data:

- General health of the animals
- Lung pathology demonstrating asbestosis, fibrosis, bronchogenic carcinoma, and mesothelioma
- Metastasis to other organs
- Gastrointestinal tumors.

A backup contract has already been awarded to Illinois Institute of Technology Research Institute (IITRI) in Chicago to procure raw rock face and to identify them chemically and geologically. After such analysis, the contractor is to supply each geological type to Mt. Sinai Hospital, New York, for confirmation. A selection of samples will then be made according to the size, shape, and chemical properties of the particles. The samples will then be sent to this laboratory for the biological evaluation. All specimens will be held in Mt. Sinai laboratory as reference material.

2.5.1 Animal Selection

The type of animal to be used for asbestos lung studies was considered by the UICC Working Party in 1965. Various types of lesions, similar to those

fiber exposures. This is due to two factors: most airborne asbestos fibers are shorter than 5 μm in length and are, therefore, not counted, and many fibers, although longer than 5 μm , may be too small in diameter to be detected by optical microscopy due to resolution limitations. Phase-contrast fiber counts represent at best, therefore, a crude method of *true total fiber exposure*.

Anderson *et al.*⁴ presented data that demonstrated the occurrence of mesothelioma and asbestosis among household contacts of amosite asbestos workers. Such "take home" exposures would be expected to be of low concentration. Moreover, the lifetime cumulative dust exposures among such household contacts would certainly be far below that experienced by a typical asbestos worker exposed at concentrations at the OSHA 1976 standard of 2.0 fibers greater than 5 μm in length per cubic centimeter. The results of Anderson *et al.*⁴ and of the NIOSH study of hard rock gold miners thus point to the inadequacy of the OSHA 1976 asbestos occupational exposure standard to protect against asbestos-related malignant and nonmalignant diseases.

SUMMARY AND CONCLUSIONS

Amosite asbestos fibers of short length have been identified in the study mine at concentrations far below the 1976 OSHA standard of 2.0 fibers greater than 5 μm in length per cubic centimeter. Moreover, the mortality study presented by Gillam *et al.*¹ has shown significantly increased risks of both cancerous and noncancerous respiratory disease in that mining population, findings that are consistent with an asbestos-related etiology.

Three specific conclusions can be drawn when the results of the present study are considered in combination with the data presented at this conference^{1, 4}:

The OSHA 1976 asbestos standard of 2.0 fibers greater than 5 μm in length per cubic centimeter may not be adequate to protect against asbestos-induced respiratory neoplasia.

The adverse health implications of asbestos fibers shorter than 5 μm in length in asbestos disease etiology must be paid greater attention in the development and setting of asbestos exposure standards. A routine sampling method that more adequately accounts for such fibers must be developed.

Noncommercial asbestos fibers, namely, asbestos fibers present as contaminants in other products, are capable of producing disease. This finding is of paramount importance in regard to several occupational and environmental issues, such as air pollution by asbestiform fibers in many areas of the United States and of other countries and asbestos contamination of commercial talc products, in which fibrous anthophyllite and tremolite asbestos are frequently found.

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