MAR 17 1976

xc: Messrs. F. H. Larrison
W. C. Thurber

4-23-76 EAP

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
March 12, 1976

NATIONAL INSTITUTE FOR OCCUPATIONAL
SAFETY AND HEALTH
U.S. POST OFFICE AND COURTHOUSE
CINCINNATI, OHIO 45202
Room 532

Mr. E.A. Piersall
Safety Director
Union Carbide Corporation
Metals Division
P.O. Box 1029
Grand Junction, Colorado 81501

Dear Mr. Piersall:

I've enclosed copies of two papers entitled "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral" and "Asbestos Fiber Exposures in a Hard Rock Gold Mine". These papers should give some insight into the recent NIOSH proposal for lowering the occupational airborne asbestos standard.

I hope you find this information helpful. If you have any questions, please give me a call. I can be reached at (513) 684-3255.

Sincerely yours,

Ralph Zumwalde
Environmental Investigations Branch

A 18017

PLAINTIFF'S EXHIBIT
Union Carbide 370

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

Presented at the
Conference on Occupational Carcinogenesis
The New York Academy of Sciences
New York, N.Y., U.S.A.
24 March 1975

By

J. Dean Gillam, M.A.[+]
John M. Dement, M.S.*
Richard A. Lemen, M.S.*
Joseph K. Wagoner, S.D. Hyg.*
Victor E. Archer, M.D.[+]
Héctor P. Blejer, M.D., D.I.H.*

National Institute for Occupational Safety and Health
Center for Disease Control, Public Health Service
Department of Health, Educatiom, and Welfare
*Cincinnati, Ohio 45202
[+]Salt Lake City, Utah 84108
U.S.A.

A 18013

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

By

J. Dean Gillam, M.A.[+]
John M. Dement, M.S.*
Richard A. Lemen, M.S.*
Joseph K. Wagoner, S.D. Hyg.*
Victor E. Archer, M.D.[+]
Héctor P. Blejer, M.D., D.I.H.*

National Institute for Occupational Safety and Health
Center for Disease Control, Public Health Service
Department of Health, Education, and Welfare
*Cincinnati, Ohio 45202
[+]Salt Lake City, Utah 84108
U.S.A.

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 Kiviluoto,[3] Meurman[4] and Meurman et al[5] reported significantly increased proportional mortality from bronchogenic carcinoma. As reported in 1972, Selikoff et al[6] demonstrated a seven-fold excess of lung cancer

A 18013

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/3

among insulation workers with 20 or more years since 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 micrometers (μm) in length per cubic centimeter (cc) of air, as an 8-hour 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, as well as asbestos fibers *shorter than 5 μm*, was brought out at recent court hearings concerning a mining company in Minnesota and its disposal of taconite tailings.[9] Expert testimony 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 or not a carcinogenic risk was associated with that ore dust which contained noncommercial forms of *amosite* asbestos in the cummingtonite-grunerite amphibole mineral category.

A:8020

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 microscopic data(11) which 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 in 1960 had been examined 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 those 439 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

A 18021

determined through records maintained by U.S. 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 those individuals not located through these data sources, other mechanisms 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 five-year calendar time periods, five-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 that 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 70 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

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOSH... Published by AsbestosClaims.Law
ACL Doc 20098 · Page 6 of 35 asbestosclaims.law/library/20098/6

(8 observed vs. 3.2 expected, $p<0.05$). A significant excess of deaths also occurred in the category "all other causes" ($p<0.05$). Of the nine observed deaths in this category, five (56%) had mention of pneumoconiotic disease in 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 there shows an excess of nonmalignant respiratory disease in general (8 observed vs. 2.56 expected, $p<0.01$), and specifically of pneumoconiotic disease (5 observed vs. 1.57 expected, $p<0.05$), to have occurred following 20 years since onset of underground 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, a carcinoma of the maxillary sinus; and one, 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

A 18023

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 arsenophyrites.(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 million particles per cubic foot (mppcf) in miner's breathing zones, whereas airborne and settled dust samples showed an average free silica content of 39 percent. Given a 39 percent free silica content, the Occupational Safety and Health Administration (OSHA) eight-hour time-weighted average standard for occupational exposure to free silica would be approximately 5 mppcf. Thus, free silica exposures were well below the present applicable OSHA standard. Semiquantitative x-ray spectrographic 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 having 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

A18024

this mine. Using 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 percent. All samples of mining personnel exposures showed average breathing zone dust concentrations 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 using 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 microscopic analytical method for asbestos fibers. The average fiber concentration was found to be 0.25 fibers greater than 5 μm in length/cc, (fibers/cc) with the highest single concentration found being 2.8 fibers/cc.

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

To carry out 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 highest fiber concentrations, as determined by optical microscopy,

A18025

were selected by NIOSH and analyzed by analytical transmission electron microscopy using 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 microscopic identification of airborne fibers present in this mine. As can be seen, 80 to 90 percent of the airborne fibers were fibrous amphiboles, and 60 to 70 percent of the latter were fibrous grunerite (amosite). Fibrous cummingtonite made up one to two percent of the amphibole fibers, and fibrous hornblends constituted an additional 10 to 15 percent of these fibers. Approximately 20 percent 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 amosite asbestos.

Average airborne fiber concentrations in this mine, as determined by electron microscopy, are shown on Table 4. Using the transmission electron miscoscope, total fiber concentrations were found to average 4.82 fibers/cc, whereas concentrations of fibers greater than 5 μm in length averaged 0.36 fibers/cc. In addition, the median fiber diameter and length were found to be 0.13 μm and 1.1 μm, respectively, with approximately 94 percent of the airborne fibers being 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

A 18020

the form of arsenic present in the airborne dust. All particles so analyzed were found to be arsenophyrite (FeAsS) at extremely low concentrations.

DISCUSSIONS

Engineering controls in this hardrock 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 also were 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 control 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 non-contributory, as demonstrated previously among underground potash miners.[16]

A 18027

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 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 percent.(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 etiological agent for the increased risk of respiratory cancer among this study cohort.

Additional 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 involving 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, using electron microscopy.(18) Table 6 presents a summary of those results and their comparison to the airborne concentrations found in the hardrock gold mine in South Dakota. The similarity both of the concentrations observed in these studies and of the relative prevalence

A 18028

of short fibers is striking: A mean concentration of 4.8 fibers/cc was noted in the South Dakota mine and 4.7 fibers/cc 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 to 10 percent 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: 1) amphibole fibers (amosite) in the cummingtonite - grunerite series; 2) airborne concentrations less than 2.0 fibers/cc as determined by the NIOSH phase contrast counting technique; and 3) 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, in the case of arsenic, chromium and nickel. The observed excess 1) of malignant respiratory disease can, therefore, be attributed to asbestos, singly or in combination with cigarette smoke; and 2) of nonmalignant respiratory disease, 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: 1) the adequacy of the OSHA standard of 2 fibers longer than 5 μm/cc; 2) the deleterious health effects of asbestos fibers shorter than 5 μm vis-a-vis any standard for occupational or environmental exposure to asbestos; 3) the adequacy of the present NIOSH asbestos sampling and analytical methods; and 4) the potential adverse health effects among general, nonoccupational populations exposed to industrial waste disposals and industrial process effluents containing trace concentrations of short-fiber, noncommercial asbestos minerals.

A 18029

REFERENCES

1. LYNCH, K.M. & W.A. SMITH, 1935. Pulmonary asbestosis III - carcinoma of lung in asbestos-silicosis. Amer. J. Cancer 24: 56-64.

2. WAGNER, J.C., C.A. SLEGGS & P. MARCHAND, 1960. Diffuse pleural mesothelioma and asbestos exposure in the North-Western Cape Province. Birt. J. Indust. Med. 17: 260-271.

3. KILVILUOTO, P. 1960. Pleural calcification as a roentgenologic sign of non-occupational endemic anthophyllite-asbestosis. Acta. Radiol. (Stockh.), suppl. 194: 1-67

4. MEURMAN, L.O. 1966. Asbestos bodies and pleural plaques in Finnish series of autopsy cases. Acta. Path. Microbiol. Scand. suppl. 181: 1-107.

5. MEURMAN, L.O., P. KILVILUOTO & M. HAKAMA. 1974. Mortality and morbidity of employees of anthophyllite asbestos mines in Finland. Brit. J. Indust. Med. 31: 105-112.

6. SELIKOFF, I.J., E.C. HAMMOND & H. SEIDMAN. Carcinogenicity of Amosite Asbestos. Arch. Environ. Health: 25, 183, 1972.

7. WAGONER, J.K., W. JOHNSON & R. LEMEN. Malignant and Non-Malignant Respiratory Disease Mortality Patterns Among Asbestos Production Workers. Congressional Record-Senate, 14 March 1973, 54660-54662.

8. NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH. 1971. Occupational exposure to asbestos-criteria for a recommended standard Publication No. HSM-1026. U.S. Government Printing Office, Washington, D.C., U.S.A.

9. U.S. DISTRICT COURT FOR MINNESOTA, 1973. United States versus Reserve Mining Company. No. 5-72-Civil-19.

A 18030

10. MASSON, T.L., F.W. MEKAY & R.W. MILLER. 1974. Asbestos-like fibers in Duluth water supply: relation to cancer mortality. JAMA 228: 1019-1020.

11. U.S. DISTRICT COURT FOR MINNESOTA. 1974. United States versus Reserve Mining Company. No.5-72-Civil-19. Feb. 25, 1974. Reserve Exhibit No. 466.

12. U.S. DISTRICT COURT FOR MINNESOTA. 1974. United States versus Reserve Mining Company. No.5-72-Civil-19, Feb. 25, 1974: 16009 Testimony by Raymond Erickson, Attorney for Reserve Mining Company.

13. U.S. DEPARTMENT OF THE INTERIOR, BUREAU OF MINES. 1960. Environmental study - Mine 30, Jan. 17 - Feb. 6, 1960. Internal report.

14. U.S. DEPARTMENT OF THE INTERIOR, BUREAU OF MINES. 1973. Onsite dust assessment at the Homestake Gold Mine, Lead, South Dakota. Internal report.

15. U.S. DEPARTMENT OF THE INTERIOR, MINING ENFORCEMENT AND SAFETY ADMINISTRATION. 1974. Fiber survey, Homestake Gold Mine, Homestake Mining Company, Lead, Lawrence County, South Dakota, Sept. 23 - Oct. 3, 1974. Internal report.

16. WAXWEILER, R.J., J.K. WAGONER & V.E. ARCHER. 1973. Mortality of Potash Workers. J. Occup. Med. 15: 486-489.

17. LUNDIN, F.E., J.K. WAGONER & V.E. ARCHER. 1971. Radon daughter exposure and respiratory cancer - quantitative and temporal aspects. National Institute for Occupational Safety and Health - National Institute for Environmental Health Sciences Joint Monograph No. 1. U.S. Government Printing Office, Washington, D.C., U.S.A.

18. U.S. DISTRICT COURT FOR MINNESOTA, 1973. United States versus Reserve Mining Company. No. 5-72-Civil-19, Sept. 6, 1973. Exhibit No. 62

A 18031

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 16 of 35

Table I

HARD ROCK GOLD MINE, SOUTH DAKOTA

OBSERVED AND EXPECTED DEATHS ACCORDING TO CAUSE AMONG WHITE MALES WHO HAD FIVE OR MORE YEARS OF UNDERGROUND MINING EXPERIENCE BY APRIL, 1960.

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 CNS	330-334	5	3.2
Disease of the Heart	400-443	25	25.2
Non-Malignant Respiratory Disease	470-493 500-527	8	3.2*
Influenza & Pneumonia	480-493	3	1.3
Other Respiratory Disease	470-479 500-527	5	1.9
Accidental Deaths	800-962	8	7.8
Motor Vehicle	810-835	0	2.5
Other	800-809 840-962	8	5.3
All Other Causes		9	3.9*
Total		70	52.9*

*Significant at the $p<0.05$
**Significant at the $p<0.01$

A-18032

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/16

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 17 of 35

Table II

HARD ROCK GOLD MINE, SOUTH DAKOTA

OBSERVED AND EXPECTED DEATHS DUE TO MALIGNANT AND NON-MALIGNANT RESPIRATORY DISEASE

AMONG WHITE MALES HAVING ACHIEVED FIVE OR MORE YEARS OF UNDERGROUND GOLD MINING

EXPERIENCE BY APRIL, 1960.

CAUSE OF DEATH	ICD	NUMBER OF YEARS SINCE ONSET OF UNDERGROUND EMPLOYMENT			
		5 - 19 Yrs.		≥20 Yrs.	
		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
Non-Malignant Respiratory Disease	470-493 500-527	0	0.59	8	2.56**
Influenza & Pneumonia	480-493	0	0.31	3	0.99
Other Respiratory Disease	470-479 500-527	0	0.28	5	1.57*

A:80033

*Significant at the p<0.05
**Significant at the p<0.01

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/17

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 18 of 35

Table III

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

PERCENT OF AIRBORNE FIBERS

Amphibole By Electron Diffraction	Fibrous Grunerite (Amosite)	Fibrous Cummingtonite	Fibrous Hornblend	Unknown
80 - 90	60 - 70	1 - 2	10 - 15	≈ 20

A-8037

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/18

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 19 of 35

Table IV

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

Exposure Measure	Fiber Concentrations, Fibers/cc	
	Total Fibers	Fiber >5μm
Mean ± S.E. Range	4.82 ± 0.68 0.66 - 11.79	0.36 ± 0.08 0.07 - 1.29

A 18035

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/19

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 20 of 35

Table V

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

Summary Statistic	Diameter	Length
Count Median Size (N = 2111)	0.13μm	1.10μm
Geometric Std. Dev.*	3.13	2.70
95% Conf. Internal For Count Median Size	0.128 - 0.141μm	1.07 - 1.15μm

A-8030

*Data fitted to log-normal distributions using probit analysis and linear regression ($\gamma > 0.90$).

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/20

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 21 of 35

Table VI

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 CONC., FIBER/CC		%> 5μm
	Mean	Range	
Study Mine (N = 22)	4.8	0.7 - 11.8	6%
Silver Bay (N = 6)	4.7	0.5 - 11.0	5 - 10%

A 18037

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/21

ASBESTOS FIBER EXPOSURES IN A HARD ROCK GOLD MINE

Presented at the

Conference on Occupational Carcinogenesis

The New York Academy of Sciences

New York, N.Y., U.S.A.

24 March 1975

By

John M. Dement, M.S.

Ralph D. Zumwalde

Kenneth M. Wallingford

National Institute for Occupational Safety and Health

Center for Disease Control, Public Health Service

Department of Health, Education, and Welfare

Cincinnati, Ohio 45202

U.S.A.

A 18038

ASBESTOS FIBER EXPOSURES IN A HARD ROCK GOLD MINE

By

John M. Dement, M.S.

Ralph D. Zumwalde

Kenneth M. Wallingford

National Institute for Occupational Safety and Health

Center for Disease Control, Public Health Service

Department of Health, Education, and Welfare

Cincinnati, Ohio 45202

U.S.A.

INTRODUCTION

The study, "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Material," reported by Gillam et al of the National Institute for Occupational Safety and Health (NIOSH), 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 showing low level exposures to fibrous grunerite (amosite) in the mine. The following paragraphs describe in further detail the laboratory analyses undertaken by 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-hour time-weighted average fiber exposures, as analyzed by the NIOSH phase contrast optical microscopic counting technique. The average fiber concentra-

A 18030

tion was found to be 0.25 fibers greater than 5 μm in length per cubic centimeter of air (fibers/cc); and the highest single concentration, 2.8 fibers/cc. No fiber identification was done in that study, however.

To carry out further characterization of the fiber exposures in the subject mine, portions of all 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 using analytical electron microscopy. The membrane (Millipore Type AA) filter samples were mounted on formvar and carbon-coated copper electron microscope grids (200 mesh) using the direct clearing method, as described by Fraser.[2] Samples for scanning electron microscopic analysis were further coated with a thin (100-150Å) 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 and fiber chemical composition by x-ray microanalysis. An acceleration voltage of 100 kilovolts (KV) was used for transmission electron microscopy and 20 KV for scanning microscopy.

Two criteria were used as necessary and sufficient data to classify fibers in these air samples: 1) observation of the single-fiber electron diffraction pattern, and 2) observation of semiquantitative elemental analysis spectra using x-ray microanalysis techniques.

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

A 18040

Typical fibers, electron diffraction patterns and x-ray spectra for fibers in the air samples as well as 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 using 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 10,000X. Fiber diameter and length were simultaneously determined by comparison with calibration marks engraved on the screen. Counting field areas were defined using the electron grid openings whose areas were calibrated. A total of 2,111 fibers were counted and sized.

RESULTS

Figure 1 shows a typical micrograph of fibers seen in a grunerite standard,obtained from the Smithsonian Institute, 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 fiber (amosite) 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 to 90 % of the airborne fibers demonstrated an amphibole electron diffraction pattern. Of these amphiboles, 60 to 70 % had chemical composi-

A:8041

tions identical to fibrous grunerite (amosite), 1 to 2 % were fibrous cummingtonite and 10 to 15 percent had compositions of common hornblends, that is, aluminum in the fiber structure.

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 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 which demonstrates fibers with amphibole cleavage patterns, viz cleavage parallel to length.

Results of the fiber concentration measurements are shown in Table 2. Using electron microscopy, fiber concentrations were seen to be low, with an *average* concentration of 0.36 fibers/cc *greater than 5 um in length.* The average *total fiber* concentration was found to be 4.82 fibers/cc. 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 µm, respectively, with 94% of the fibers being less than 5 µm in length.

DISCUSSION

Fibrous grunerite (amosite) has been shown to be present at low concentrations in this hard rock gold mine, with most fibers being shorter than 5 µm in length. Table 4 shows a comparison of fiber length distributions between those determined for this mine and those for other asbestos-using industrial operations. As can be seen, the proportion of fibers longer than 5 µm in length ranges from 1 to 51 %, depending on the operation and the type

A:8042

fiber present. It is now well recognized that the NIOSH phase contrast fiber counting method of asbestos--in which only those fibers longer than 5 μm in length are counted--represents only an "index" of the true fiber exposures. This is due to two factors: 1) most airborne asbestos fibers are shorter than 5 μm in length and are, therefore, not counted and 2) many fibers, although longer than 5 μm, may be to small in diameter to be detected by optical microscopy due to resolution limitations. Phase contrast fiber counts represent it best, therefore, a crude method of *true total fiber exposure*.

Anderson et al of the Mt. Sinai School of Medicine, City University of New York, presented data which demonstrated the occurrance 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/cc. The results of the Mt. Sinai study 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/cc. Moreover, the mortality study presented by Gillam et al, of NIOSH has shown significantly increased risks

A:8043

of both cancerous and noncancerous respiratory disease in that mining population, findings which are consistent with an asbestos-related etiology.

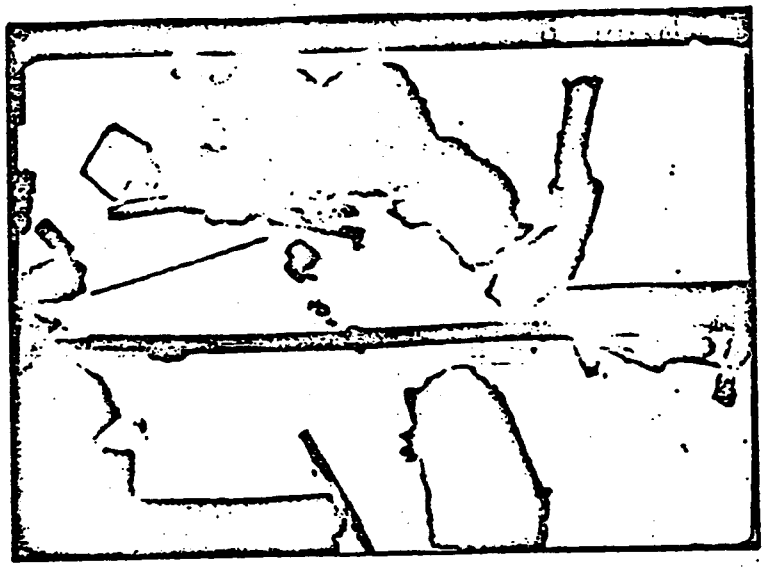
Three specific conclusions can be drawn when the results of the present study are taken in combination with the data presented at this conference by Gillam and Anderson.

1. The OSHA '1976' asbestos standard of 2.0 fibers greater than 5 μm in length/cc may not be adequate to protect against asbestos-induced respiratory neoplasia.
2. 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 which more adequately accounts for such fibers must be developed.
3. Noncommercial asbestos fibers--viz 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 a) air pollution by asbestiform fibers in many areas of the U.S.A. and of other countries, and b) asbestos-contaminantion of commercial talc products in which fibrous anthophyllite and tremolite asbestos are frequently found.

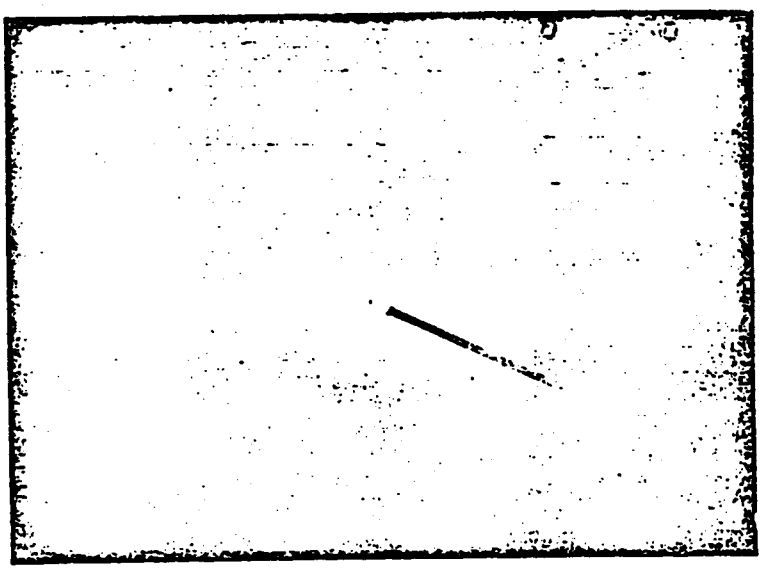
A 18044

FIG. 1: GRUNERITE STANDARD

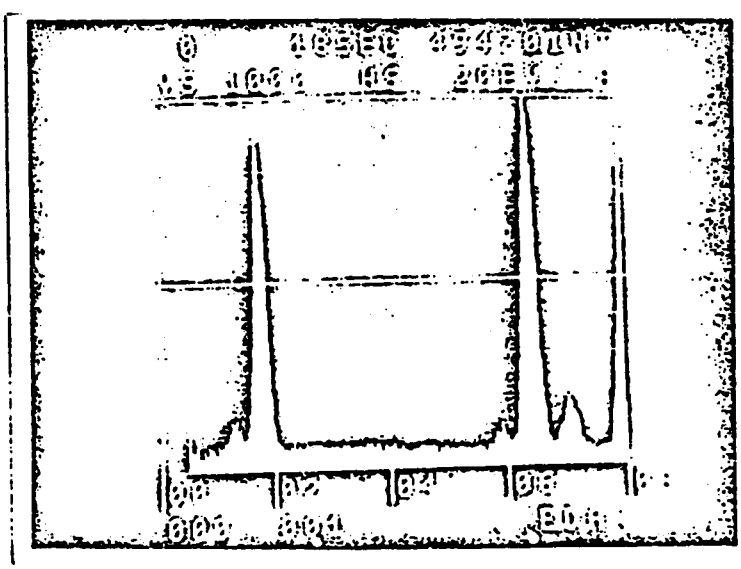
ELECTRON MICROGRAPH
10,000x



AMPHIBOLE DIFFRACTION
PATTERN



X-RAY SPECTRUM



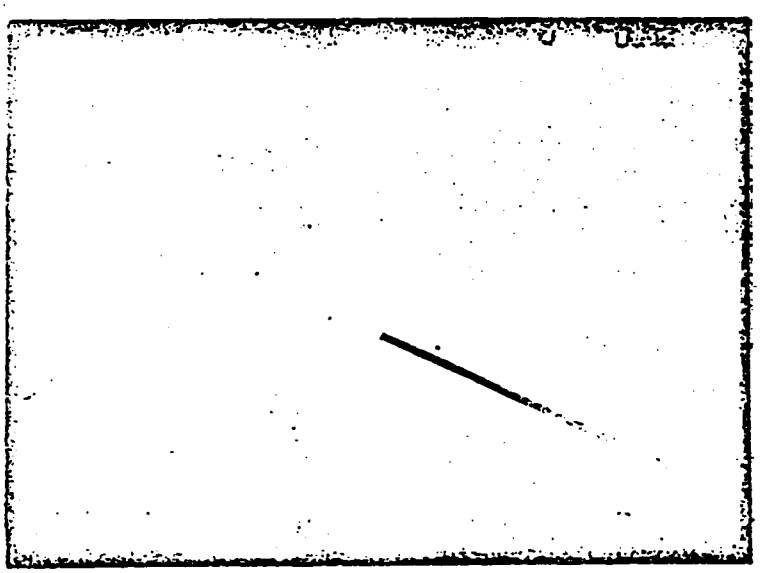
A 18045

FIG. 2: FIBROUS GRUNERITE (AMOSITE) PRESENT IN AIR SAMPLE

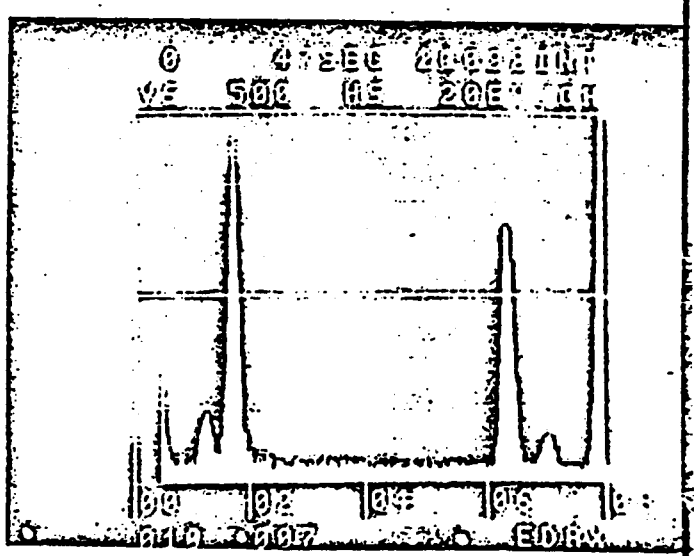
ELECTRON MICROGRAPHS, 10,000x



AMPHIBOLE DIFFRACTION PATTERN



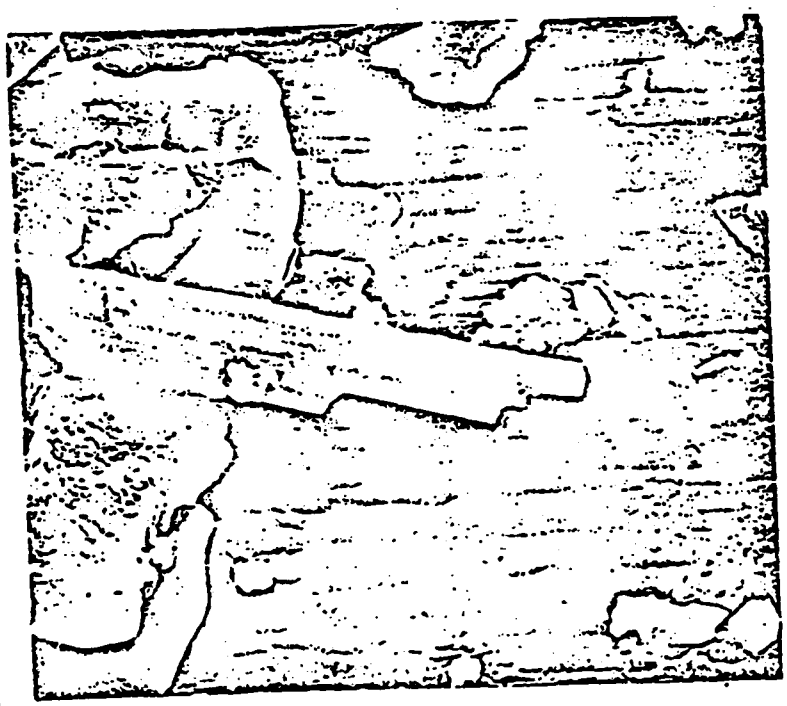
X-RAY SPECTRUM



A 18043

FIG.3: SCANNING ELECTRON MICROGRAPHS OF AMPHIBOLE FIBERS IN SETTLED DUST SAMPLES 3,000x





A 18047

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 32 of 35

TABLE 1

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

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

A:80430

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/32

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 33 of 35

TABLE 4

COMPARISON OF AIRBORNE FIBER LENGTH DISTRIBUTIONS
IN VARIOUS ASBESTOS OPERATIONS

Operation	Fiber Type	Median Length	% >5μm
Textile[A]	Chrysotile		
Fiber Prep and Carding		1.4	4
Spinning, Twisting, Weaving		1.0	2
Friction[A]	Chrysotile		
Mixing		0.9	2
Finishing		0.8	2
Asbestos-Cement Pipe[A]	Chrysotile		
Mixing		0.9	2
Finishing		0.7	1
Pipe Insulation[B]	Amosite		
Pipe Forming		4.9	51
Gold Mining	Amosite		
Miners		1.1	7

A. Taken from Reference 4.
B. Unpublished NIOSH study.

A-3041

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/33

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 34 of 35

TABLE 2

AIRBORNE FIBER CONCENTRATIONS AS DETERMINED BY ELECTRON MICROSCOPIC FIBER COUNTS AT 10,000X MAGNIFICATION
HARD ROCK GOLD MINE, SOUTH DAKOTA

FIBER CONCENTRATIONS IN MINE
AVERAGE OF 22 SAMPLES (FIBERS/CC)

MEASURE	TOTAL FIBERS	FIBERS > 5μm
Mean ± SE	4.82 ± 0.68	0.36 ± 0.08
Range	0.66 - 11.79	0.07 - 1.29

A-18043

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/34

Union Carbide trial exhibit Union Carbide 370: Ralph Zumwalde (NIOSH) to E. A. Piersall Transmitting Two NIOS...
ACL Doc 20098 · Page 35 of 35

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)	0.13 µm	1.10 µm
Geometric Standard Deviation	3.13	2.70
95% Confidence Interval for Count Median Size	0.128 - 0.141 µm	1.07 - 1.15 µm

A-18040

Published by AsbestosClaims.Law
asbestosclaims.law/library/20098/35