PLAINTIFF'S EXHIBIT AIA-69
PENGAD-Bayonne,N.J.

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

December 21, 1976

NATIONAL INSTITUTE FOR OCCUPATIONAL
SAFETY AND HEALTH
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R. H. Mereness
Executive Director
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Dear Mr. Mereness:

As per your telephone request of December 20, 1976, enclosed is a photocopy of the NIOSH document entitled "Re-examination and Update of Information on the Health Effects of Occupational Exposure to Asbestos." This recommended standard was transmitted to the Department of Labor on December 15, 1976. The recommended environmental limit of 100,000 fibers>5μm in length per cubic meter is presented on page VI-2.

Sincerely yours,

Jon R. May

Jon R. May, Ph.D.
Chief, Criteria Development Branch
Division of Criteria Documentation
and Standards Development

Enclosure

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Re-examination and Update of Information on the Health Effects of Occupational Exposure To Asbestos

U.S. Department of Health, Education and Welfare
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
December, 1976

01-0203687

The Division of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health (NIOSH), having primary responsibility for development of a NIOSH position paper on health effects of occupational asbestos exposure, has critiqued all available data and prepared the following document for publication and transmittal to the Occupational Safety and Health Administration (OSHA), as requested by the Assistant Secretary for Labor. Primary responsibility for development of this document was shared by Richard A. Lemen and John M. Dement with technical consultation provided by Dr. Joseph K. Wagoner. Individuals who served as the NIOSH review committee were:

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I. INTRODUCTION

When the asbestos criteria document was first published in 1972, the National Institute for Occupational Safety and Health recommended a standard of 2.0 asbestos fibers per cubic centimeter (cc) of air based on a count of fibers greater than 5 micrometers (μm) in length. This standard was recommended with the stated belief that it would "prevent" asbestosis and with the open recognition that it would not "prevent" asbestos-induced neoplasms. Furthermore, data were presented supporting the fact that technology was available to achieve that standard and that the criteria would be subject to review and revision as necessary. Since the time of that asbestos criteria, published in 1972, sufficient additional data bearing on asbestos-related disease have been developed to warrant re-evaluation.

On June 7, 1972 the Occupational Safety and Health Administration (OSHA) promulgated a standard for occupational exposure to asbestos containing an 8-hour time weighted average (TWA) concentration exposure limit of 5 fibers longer than 5 μm per cubic centimeter of air, with a ceiling limitation against any exposure in excess of 10 such fibers per cubic centimeter. The standard provided further that the 8-hour TWA was to be reduced to 2 fibers per cubic centimeter on July 1, 1976.

As the result of a court case, OSHA decided that to achieve the most feasible occupational health protection, a re-examination of the standard's general premises and general structure was necessary. To this end, on October 9, 1975 OSHA announced a proposed rule-making to lower the standard to an 8-hour TWA airborne concentration of asbestos fibers of 0.5 fibers

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per cubic centimeter of air with a ceiling concentration of 5 fibers per cubic centimeter of air determined by a 15-minute sampling period. On December 2, 1975, Marshall L. Miller of OSHA requested NIOSH to re-evaluate the information available on the health effects of occupational exposure to asbestos fibers and to advise OSHA of the results of this study.

This document contains an updated review of the available information on the health effects of exposure to asbestos. In addition, NIOSH's proposal for a new numerical exposure limit is included.

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II. ANIMAL DATA

CARCINOGENICITY

INSTILLATION

Intratracheal Injection

This technique has been used to study co-carcinogenesis of chrysotile asbestos with benzo(a)pyrene in hamsters (Miller et al 1965) and rats (Vosamae 1972, Pylev 1972; Pylev and Shabad 1973; Shabad et al 1974). In both species, it was demonstrated that the effect of chrysotile was additive to that of benzo(a)pyrene for tumors of the respiratory tract.

Shabad et al (1974) in an extension of his study showed that intratracheal injection of 2 mg Russian chrysotile on which 0.144 mg benzo(a) pyrene was absorbed (3 times at monthly intervals), or 2 mg of Russian chrysotile together with 5 mg benzo(a)pyrene (single injection) produced lung papillomas, epidermoid carcinomas, reticulosarcomas, or pleural mesotheliomas in 6/21 and 6/11 rats respectively within 9-28 months. No lung tumors or mesotheliomas occurred in 49 rats given 3 doses of 2 mg chrysotile alone or in 19 rats given a single dose of 5 mg benzo(a)pyrene alone during or up to 28 months of observation.

Intraperitoneal (i p) Administration

Reeves et al. (1971) gave i.p. injections of 0.3, 0.5, or 1.0 ml of a solution of 20 mg/ml amosite, crocidolite or chrysotile into groups of 11, 13 and 13 Charles River CD rats respectively. Three peritoneal mesotheliomas were observed with chrysotile, 3 with crocidolite, and none with amosite after 7-17 months. No data on control animals were reported.

Maltoni and Annoscia (1973) injected 25 mg of crocidolite into 50 male and 50

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female Sprague-Dawley rats, 18 weeks old, and later observed 65 mesotheliomas: 31 in males and 34 in females.

Pott and Friedrichs (1972) and Pott et al (1974) injected fibrous and granular dusts into the peritoneal cavities of Wistar rats. The dosage, number of inoculations, and results are shown in Tables 1 and 2.

After injection of powdered chrysotile, the latent period for the induction of tumors was found to be longer than after injection of standard chrysotile. The rate of tumor occurrence was about 40% in both groups and was not distinctly influenced by the addition of benzo(a)pyrene. In another group, benzo(a)pyrene without asbestos induced tumors in 10% of the animals. Histologically, the types of tumors observed were connected with structures of the abdominal wall, including the serosa, and in isolated cases with those of the intestinal wall (Pott et al 1972).

Intrapleural Administration

All commercial types of asbestos have produced mesotheliomas in CD Wistar rats. A dose of 20 mg of the 5 UICC standard reference samples produced mesotheliomas in varying numbers - crocidolite, (61%); amosite, (36%); anthophyllite, (34%); Canadian chrysotile, (30%); Rhodesian chrysotile, (19%) (Wagner et al 1974). The lowest dose used (0.5 mg chrysotile or crocidolite) produced mesotheliomas (Wagner et al 1973). Stanton and Wrench (1972) using a dose of 40 mg asbestos dust on gelatin-coated fiber glass pledgets, found that 3 of the UICC samples, crocidolite, amosite and Rhodesian chrysotile, all produced mesotheliomas in about 60% of the Osborne-Mendel rats. Pyle and Shabad (1973) induced mesotheliomas with 60 mg of Russian chrysotile. In all these studies there was a long latent period between inoculation and

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appearance of the tumors. Evidence that the response was dose-related was provided by Wagner et al (1973) and by Stanton (1973). Mesotheliomas have also been produced by other workers: in rats (Donna 1970, Reeves et al 1971), in hamsters (Smith et al 1965) and in rabbits (Reeves et al 1971). Groth et al, (1975) reported no mesotheliomas or other neoplasms from chrysotile in 45 female discard-breeder albino rats, approximately 10 months old. However, all surviving tumor-free animals were killed at 90 or 150 days after injection, a time period insufficient for the development of mesothelioma as demonstrated by the experiments of Wagner and Berry (1969).

The suggestion has been made that natural oils and waxes (Harington, 1962) and contaminant oils from milling of the fiber (Harington and Roe 1965; Roe et al 1966) or from plastic storage bags (Commins and Gibbs 1969) contributed to the incidence of pleural tumors. However, samples from which the oils had been removed gave very similar results to untreated fiber (Wagner and Berry 1969; Wagner et al 1973).

Morgan and Holmes (1970) and Morgan et al (1971) showed that when asbestos was inoculated intrapleurally, the majority of the fibers were cleared from the lungs during the first ten days; subsequently there was also a very slow elimination through the gut. In feeding experiments almost all the fibers were eliminated. After intrapleural or subcutaneous inoculation, only a minute fraction of the finer fibers were translocated through the tissues. This finding was supported by the studies of Kanazawa et al. (1970).

The fiber diameter, length and shape may be important in disease production. All of the eight separate sub-samples which were pooled in the UICC Canadian

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chrysotile reference sample (Timbrell and Rendall 1972), when ground separately to a finer powder, produced a higher incidence of mesothelioma than the pooled sample. The highest incidence (66%) was produced by a separate superfine chrysotile sample (20 mg dose) fractionated from fine grade asbestos by water sedimentation (Wagner et al 1973). Using UICC crocidolite, Stanton and Wrench (1972) found that partially pulverized material gave fewer mesotheliomas than did the standard unpulverized fiber. Prolonged fine grinding is known to destroy fiber and crystalline structure (Occella and Maddalon 1963). Stanton (1973) showed that fibers of other materials, including glass could induce mesotheliomas, but only when the diameter was of the same order as that of asbestos when measured by light microscopy.

In addition to the UICC standard reference samples, other fibers were inoculated intrapleurally into rats by Wagner et al (1973). Out of 32 rats per group, mesotheliomas occurred in 18 animals injected with a sample of brucite, 3 injected with a ceramic fiber, 1 each with barium sulphate, glass powder, and aluminum oxide. None occurred with a coarse glass fiber.

Wagner et al (1976) conducted a series of experiments comparing the biological effects of a pure asbestos free cosmetic talc with the superfine chrysotile asbestos used in previous experiments. In an intrapleural inoculation experiment, 48 rats were inoculated with each dust. Eighteen of the chrysotile group developed mesotheliomas, but no tumors were seen in those given talc.

Further evidence on the importance of fiber diameter was provided by Wagner et al (1976) who reported on rats injected intrapleurally with

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glass fiber (Table 3). Two samples of glass fiber were used, one with a median fiber diameter of 0.12 μm and the other with median diameter of 1.8 μm. Four mesotheliomas were observed in 32 rats injected with the finer fiber and none with the coarser fiber. Also, the degree of mesothelial cell hyperplasia was more pronounced in the rats injected with the finer fiber. These results were comparable with those of the previous experiment.

Shabad et al. (1974) reported that when 20 mg of Russian chrysotile was injected intrapleurally 3 times into 67 rats, 31 developed mesotheliomas within 2 years.

INGESTION

Gross et al. (1974) reported the results of a series of feeding experiments with chrysotile and crocidolite fed to rats of various origins. In groups of rats varying in number from 10 through 35, no significant differences in tumor incidence were observed in comparison with controls. Survival rates were not reported, sample sizes were small (from 10 through 35) and no details of pathology were given.

In another experiment (Wagner et al 1976) fed groups of 32 Wistar SPF rats 100 mg/day of talc (5 days/week) or chrysotile in malted milk powder for 100 days over a 6-month period; sixteen controls were fed only malted milk. The mean survival from the start of feeding was 614 days for talc, 618 for chrysotile and 641 days for the controls. The only tumors which may have been associated with ingestion were two gastric leiomysarcomas; one in an animal fed talc and the other in one fed chrysotile. None occurred in the controls.

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INHALATION

Lynch et al (1957) exposed AC/F_1 hybrid mice by inhalation to a commercial preparation of chrysotile asbestos and observed a higher incidence of multiple pulmonary adenomas in the exposed group of animals, 45.7% (58/127), compared with the controls 36.0% (80/222). These results were reported as not statistically significant.

Reeves et al (1974) exposed groups of 30 Swiss mice to dusts of crocidolite, amosite and chrysotile for 4 hours/day, 4 days/week, for 2 years at a mean concentration of about 50 mg/m^3. Two of the animals exposed to crocidolite developed papillary carcinomas of the bronchus, as did one of the non-exposed controls.

Gross et al (1967) observed carcinomas of the lung in rats repeatedly exposed to chrysotile dust with a mean concentration of 86 mg/m^3 for 30 hours/week. Twenty of 72 rats surviving for 16 months or longer developed adenocarcinomas and 4 developed squamous-cell carcinomas, whereas no tumors occurred in 39 controls. The authors suggested that the presence of trace metals from the hammers of the mill used to prepare the fiber was a factor in the induction of these tumors. However, this suggestion was not confirmed by subsequent experiments (Reeves et al 1974; Wagner et al 1974), th leading Gross et al (1974) to retract the trace metal hypothesis for asbestos-induced neoplasia.

Reeves et al (1971) found squamous carcinomas of the bronchus in 2 of 31 rats which survived exposure to crocidolite for 2 years at a concentration of 49 mg/m^3 for 16 hours/week. Five rats in a group of 40 exposed to chrysotile developed pulmonary adenomatosis, but no malignant tumors were observed in rats exposed to either chrysotile or amosite.

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In a subsequent experiment, Reeves et al (1974) exposed groups of 69 Charles River CD rats to crocidolite, amosite and chrysotile for 4 hours/day , 4 days/week for 2 years, at mean concentrations of about 50 mg/m^3, Table 4. In addition, groups of 20 rabbits, 32 guinea pigs and 68 gerbils were exposed for 18 months to the same three asbestos dusts as the rats mentioned above. No tumors were observed, but mean survival times were not stated.

Wagner et al (1974) exposed groups of C/D Wistar rats to the five UICC asbestos samples at concentrations of about 12 mg/m^3 of dust for 7 hours/day, 5 days/week. for several lengths of exposure: one day (7 hours), 3 months, 12 months, and 24 months. At the end of the periods of exposure, the amount of dust in the lungs of animals exposed to the two chrysotile samples was much less than in the animals exposed to the three amphibole samples. However, all types of fiber produced asbestosis which was progressive after removal from the dust. Furthermore, whereas no tumors were found in the control group, carcinogenicity was demonstrated in the groups exposed to chrysotile (Canadian or Rhodesian) and the amphiboles (Table 5). An increasing incidence of neoplasms was observed with increasing exposures to each form of asbestos. Even as little as one day of exposure - providing the animals were allowed to survive and were observed - produced neoplasia. (Table 6) One-day exposures to Canadian chrysotile produced lung tumors. Mesotheliomas were observed in 11 rats, 2 of which were exposed only 1 day, one to amosite, and one to crocidolite.

Wagner et al (1976) compared rats exposed for a two year period to a pure non-fibrous cosmetic talc, with another group of rats exposed to superfine chrysotile. Similar degrees of fibrosis were found in each group

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while one adenocarcinoma was found in an animal exposed to the chrysotile.

FIBER ANALYSIS IN TISSUE

Following inhalation, asbestos fibers found in sections of lung tissue were usually <3 µm in diameter and <100 µm in length. Thicker or longer fibers are either not inhaled or are rapidly cleared from the respiratory tract. On a weight basis, only a very small proportion of inhaled fiber is retained. An account of the inhalation of fibers is given by Timbrell (1965, 1972). Electron-microscopy is essential for studies of asbestos in tissue as many of the fibers of chrysotile and amphiboles are too small in diameter to be seen with the light microscope (Langer and Pooley 1973).

The retention of different types of asbestos in animals following exposure to the same concentrations of respirable dust was described by Wagner et al (1974). For the amphiboles, there was a similar pattern with an almost proportional increase of lung dust with dose. Much less dust was found for the chrysotiles and no increase of dust content in the lungs was shown. Dust in the lungs of animals with 6 months exposure had been partially cleared 18 months after the inhalation period. About 74% of the amosite and crocidolite and 41% of the anthophyllite were eliminated. The elimination rate of chrysotiles could not be exactly determined because of their low content in the lung (Fig. 1) (Wagner et al 1974).

The penetration and clearance of radioactive UICC crocidolite has been studied in rats. After 30 days, the lung content of crocidolite was reduced to 75% of the initial value (Evans et al 1973).

In early experiments, it was demonstrated that guinea pigs and monkeys exposed to the four commercial types of asbestos developed fibrotic lesions of the lung and pleura similar to those seen in human cases of asbestosis

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(Vorwald et al 1951; Wagner 1963; Holt et al 1965). In more recent experiments this finding has been confirmed in rats (Wagner et al. 1973).

The question whether asbestos fibers can move from their site of primary deposition in the body and induce cancer in other sites is still a vexing one. Volkheimer (1973) and Schreiber (1974) have reported that particles and plant fibers ingested by experimental animals and man can penetrate the wall of the gastrointestinal tract and be transported throughout the body, possibly appearing in the urine. Westlake et al (1965) fed a diet containing 6% of chrysotile to rats and reported that the animals had fibers in the wall of the colon. Cunningham and Pontefract (1973) performed a similar experiment and reported that asbestos fibers appeared in the blood and various tissues. The more recent report by Gross et al (1974) referred to above, concluded, however, that there was no satisfactory evidence from their study of transmigration of fibers outside the gastrointestinal tract.

In studies in which chrysotile labelled intrinsically with radioactive trace metals by neutron irradiation was injected intrapleurally into rats, Holmes and Morgan (1967) found evidence for passage of a small amount of the fiber from the pleural cavity and lungs into such other organs as the liver. In a later, similar experiment, Morgan et al (1971) reported that a population of radionuclides consistent with that expected on the basis of the labelled chrysotile was found in the heart, the lungs, the diaphragm, and the chest muscles.

Karacharova et al (1969) and Friedrichs et al (1971) found some evidence for movement of asbestos fibers from an intraperitoneal site of injection into various tissues of the rats used. The latter group of

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investigators reported that movement was inversely related to the length of the fiber, becoming essentially zero for fibers 20 or more μm long.

Roe et al (1967) and Kanazawa et al (1970) found evidence for transport of asbestos fibers from subcutaneous sites of deposition to such organs as the spleen, the liver, kidneys, and the brain of mice. Cunningham and Pontefract (1973, 1974) reported that iv-injected asbestos localized mostly in the liver and the lungs. The later paper found further that chrysotile injected iv into pregnant rats crossed the placenta and appeared in the livers and lungs of the fetuses.

MUTAGENICITY

Sincock and Seabright (1975) found that chrysotile and crocidolite asbestos dust in a concentration of 0.01 mg/ml in culture medium induced chromosomal aberrations in Chinese hamster cells, however, these changes were not observed with glass fiber or glass powder.

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CARCINOGENICITY ANIMAL DATA

Author	Date	Finding	Type of Animal	Dosage	Type of Fiber
INTRATRACHEAL INSTILLATION					
Miller	1965	Tumors of respiratory tract	Hamster	Unknown	Chrysotile with benzo (a) pyrene
Vosamae	1972	Tumors of respiratory tract	Rats	Unknown	Chrysotile with benzo (a) pyrene
Pylev	1972	Tumors of respiratory tract	Rats	Unknown	Chrysotile with benzo (a) pyrene
Pylev & Shabad	1973	Tumors of respiratory tract	Rats	Unknown	Chrysotile with benzo (a) pyrene
Shabad et al	1974	Lung papillomas, epidermoid carcinomas reticulosarcomas, pleural mesotheliomas 6/21 and 6/11 rats within 9-28 months	Rats	2 mg. Russian chrysotile 5 mg. benzo (a) pyrene	Russian chrysotile
INTRAPERITONEAL ADMINISTRATION					
Reeves et al	1971	3/13 peritoneal mesotheliomas with chrysotile 3/13 peritoneal mesotheliomas with crocidolite 0/11 peritoneal mesotheliomas with amosite After 7-17 months	Rats	0.3, 0.5 or 1.0 ml of solution of 20 mg/ml.	Amosite Crocidolite Chrysotile
Maltoni	1973	31/50 mesothelioma in males 34/50 mesothelioma in females	Sprague-Dawley rats (18 weeks old)	25 mg crocidolite	Crocidolite
Potts and Friedricks	1972	40% tumor occurrence	Wistar rats	2, 6. 25, 25, 75, 100 mg	Chrysotile A
Pott	1974	40% tumor occurrence	Wistar rats	2, 10, 50 mg	Chrysotile A
INTRAPLEURAL ADMINISTRATION					
Wagner	1973	61% tumors with crocidolite 36% tumors with amosite 34% tumors with anthophyllite 30% tumors with Canadian chrysotile 19% tumors with Rhodesian chrysotile	Rats	20 mg.	Crocidolite Amosite Anthophyllite Canadian chrysotile Rhodesian chrysotile

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Carcinogenicity Animal Data (continued)

Stanton and Wrench	1972	Mesotheliomas in 60% rats	Rats	40 mg.	Crocidolite Amosite and Rhodesian chrysotile
Pylev and Shabad	1973	Mesotheliomas	Rats	60 mg.	Russian chrysotile
Grothetal et al	1975	No mesotheliomas-but animals sacrificed 90-150 days after injection-insufficient latent period	Albino Rats	Unknown	
Wagner	1976	18/48 mesotheliomas-chrysotile 0/48 mesotheliomas-talc	Rats		Chrysotile Talc
Reeves et al	1971	1/15 mesothelioma with crocidolite 2/12 mesothelioma with chrysotile	Rats	.5 ml	Amosite Crocidolite Chrysotile
Reeves et al	1971	2/13 mesothelioma with chrysotile	Rabbit	.8 ml	Chrysotile
Shabad et al	1974	31/67 mesotheliomas within 2 years	Rats	20 mg.	Russian chrysotile
INGESTION					
Gross et al	1974	No significant difference in tumor incidence observed; survival rates not reported; sample sizes were small	Rats	5% fiber by weight in food	Chrysotile and Crocidolite
Wagner et al	1976	2 gastric leiomyosarcomas, 1 in animal fed talc and 1 fed chrysotile	32 Wistar SPF rats	100 mg/day/ 5 day week 100 days over a 6 month period	Chrysotile or Talc
INHALATION					
Lynch et al	1957	45.7 (58/127) pulmonary adenomas in exposed group 36.0% (80/222) pulmonary adenomas in controls	AC/F. hybrid mice	Dust concentrations ranged from 150,000,000 to 300,000,000 particles per cc.	Chrysotile
Reeves et al	1974	2/30 bronchiogenic carcinoma with	Swiss mice	50 mg/m^3 4 hrs/ day, 4 days/wk. for 2 years	Crocidolite Amosite Chrysotile

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Carcinogenicity Animal Data (continued)

Gross et al	1967	20/72 rats surviving 16 months or longer developed adeno-carcinomas 4/72 rats developed squamous-cell carcinomas 0/39 tumors in controls	Rats	86 mg/m^3 for 30 hrs/week	Chrysotile dust
Reeves et al	1971	2/31 rats developed carcinoma of the bronchus with crocidolite exposure 5/40 rats developed adenomatosis with chrysotile exposure	Rats	49 mg/m^3 for 16 hrs/week for 2 years	Crocidolite Chrysotile Amosite
Wagner et al.	1974	Asbestosis produced with all types of fibers Lung Cancer / Mesothelioma / Fiber: 11/146 — 1/146 — amosite 16/145 — 2/145 — anthophyllite 16/141 — 4/141 — crocidolite 17/137 — 4/137 — chrysotile (Canadian) 30/144 — 0/144 — chrysotile (Rhodesian)	C/D Wistar rats	12 mg. dust 7 hrs/day -- 5 days/week for several lengths of exposure (1 day, 3 mo., 12 mo., 24 mo.)	Chrysotile Amosite
Sincock and Seabright	1975	Chromosal aberration in Chinese hamster cells	Hamster	0.01 mg/ml	Chrysotile Crocidolite

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Table 1: Tumours in abdomen and/or thorax after intraperitoneal injection of different fibrous and granular c

Dust	Form*	Dose i.p. (mg)	Effective Number of dissected rats	First tum. after ... days	Average survival time of rats with tum. (days after inj.)	Rats with tum.	Mesothelioma	Spindle-c.sarc.	Polym. c.sarc.	Carc...	Retic. c.sarc.**	Ben. tum.**
Chrysotile A UICC	f	2	37	431	651	16.2	4	2	-	-	1	-
"	f	6.25	35	343	501	77.1	24	3	-	-	-	-
"	f	25	31	276	419	80.6	21	2	1	1	-	-
"	f	4 x 25	33	323	361	54.5	16	2	-	-	-	-
"	f	3 x 25	33	449	449	3.0	-	-	1	-	-	-
" milled	f	4 x 25	37	400	509	32.4	9	3	-	-	-	-
Palygorscite	f	3 x 25	34	257	348	76.5	24	2	-	-	-	-
Glass fibres S + S 106	f	2	34	692	692	2.9	1	-	-	-	-	-
"	f	10	36	350	530	11.1	2	2	-	-	1	-
"	f	4 x 25	32	197	325	71.9	20	3	-	-	-	-
Gypsum	f	4 x 25	35	579	583	5.7	-	-	1	1	1	-
Nemalite	f	4 x 25	34	249	315	73.5	17	8	-	-	-	-
Actinolite	g	4 x 25	39	-	-	-	-	-	-	-	-	-
Biotite	g	4 x 25	37	-	-	-	-	-	-	-	-	-
Haematite (precipit.)	g	4 x 25	34	-	-	-	-	-	-	-	-	-
Haematite (mineral)	g	4 x 25	38	-	-	-	-	-	-	-	-	-
Pectolite	g	4 x 25	40	569	569	2.5	-	-	-	1	1	1
Sanidine	g	4 x 25	39	579	579	2.6	-	1	-	-	-	-
Talc	g	4 x 25	36	587	587	2.8	1	-	-	-	-	-
NaCl-Control	-	4 x 2m	72	--	-	-	-	-	-	-	-	-

*f = fibrous
g = granular

**not evaluated in tumour rates

Potts and Friedricks (1972)

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Table 2: TUMORS IN ABDOMEN AND/OR THORAX AFTER INTRAPERITONEAL INJECTION OF GLASS FIBERS, CROCIDOLITE AND CORUNDUM.

Dust	* Form	Dose i.p. (mg)	Effective Number of dissected rats	First tum. after ... days	Average survival time of rats with tum. (days after inj.)	Rats with tum. (%)	Mesothe-lioma	Spindle-c. sarc.	Polym. c. sarc.	Carc.	Retic. ** c. sarc.	Ben. tum. **
Glass fibres MN 104	f	2	73	421	703	27.4	17	3	-	-	1	1
"	f	10	77	210	632	53.2	36	4	-	1	3	-
"	f	2 x 25	77	194	367	71.4	47	6	2	-	-	-
Glass fibres MN 112	f	20	37	390	615	37.8	12	1	-	1	-	1
Crocidolite	f	2	39	452	761	38.5	12	3	-	-	2	1
Corundum	g	2 x 25	37	545	799	8.1	1	-	-	2	2	2

* f=fibrous
g=granular

** not evaluated in tumor vates

Pott et al. (1974)

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Table 3: PERCENTAGE OF RATS DEVELOPING MESOTHELIOMAS AFTER INTRAPLEURAL INOCULATION OF VARIOUS MATERIALS.

Material	Percentage of rats with mesotheliomas
SFA Chrysotile	66
UICC crocidolite	61
UICC amosite	36
UICC anthophyllite	34
UICC chrysotile (Canadian)	30
UICC chrysotile (Rhodesian)	19
Fine Glass fibre (code 100)	12
Ceramic fibre	10
Glass powder	3
Coarse glass fibre (code 110)	0

Wagner et al. (1976)

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Table 4: INHALATION CARCINOGENESIS FROM VARIOUS FORMS OF ASBESTOS.

Form of Asbestos	Number of Tumors
Controls	no tumors
Amosite	2 pleural mesotheliomas
Crocidolite	3 squamous-cell carcinoma, 1 papillary carcinoma and 1 adenocarcinoma, all of lungs.
Chrysotile	1 papillary carcinoma, 1 squamous-cell carcinoma of lungs, and 1 pleural mesothelioma.

Reeves et al. (1974)

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Table 6: NUMBER OF ANIMALS WITH LUNG TUMORS OR MESOTHELIOMA ACCORDING TO LENGTH OF EXPOSURE

Length of exposure	No. of animals	No. with Lung CA	No. with pleural mesotheliomas	% of animals with tumors
Controls	126	0	0	0.0
1 day	219	3	2	2,3
3 months	180	8	1	5.0
6 months	90	7	0	7.8
12 months	129	35	6	31.8
24 months	95	37	2	41.0

Wagner et al (1974)

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TABLE 5: NUMBER OF ANIMALS WITH LUNG TUMORS OR MESOTHELIOMA ACCORDING TO TYPE OF ASBESTOS

Dust	No. of animals	Tumor Type		
		Adenocarcinoma	Sq. Carcinoma	Mesotheliomas
Controls	126	0	0	0
Amosite	146	5	6	1
Anthopyllite	145	8	8	2
Crocidolite	141	7	9	4
Chrysotile (Canadian)	137	11	6	4
Chrysotile (Rhodesian)	144	19	11	0

Wagner et al. (1974)

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III. HUMAN DATA

NONMALIGNANT RESPIRATORY DISEASES

Historical Studies

The use of asbestos dates back thousands of years; however, the modern industry dates from about 1880, when it was used to make heat and acid resistant fabrics, (Hendry 1965; Hueper 1966). With the increasing use of asbestos materials, reports of asbestos-related disease emerged.

The first record of a case of asbestosis was reported in England by Montague Murray in 1906. Hoffman (1918) reported that it was the practice of American and Canadian insurance companies not to insure asbestos workers due to unhealthful conditions in that industry. Pancoast et al (1917) commented on x-ray changes resembling pneumoconiosis in 15 individuals exposed to asbestos. The first complete description of asbestosis and of the "curious bodies" seen in lung tissue appeared when Cooke (1927) reported on a case of asbestosis, and McDonald (1927) reported on the same and another case. Each author gave reasons for believing that these "curious bodies" originated from asbestos fibers that had reached the lungs. Mills (1930) reported the first case of asbestosis in the United States, and in the same year, Lynch and Smith (1930) reported on "asbestosis bodies"* found in the sputum of asbestos workers. Early studies led many investigators to conclude that people exposed to asbestos dust developed the disease "asbestosis" if the dust concentration was high or their exposure was long (Merewether and Price 1930; Merewether 1934; Fulton et al 1935, and Dreessen et al 1938).

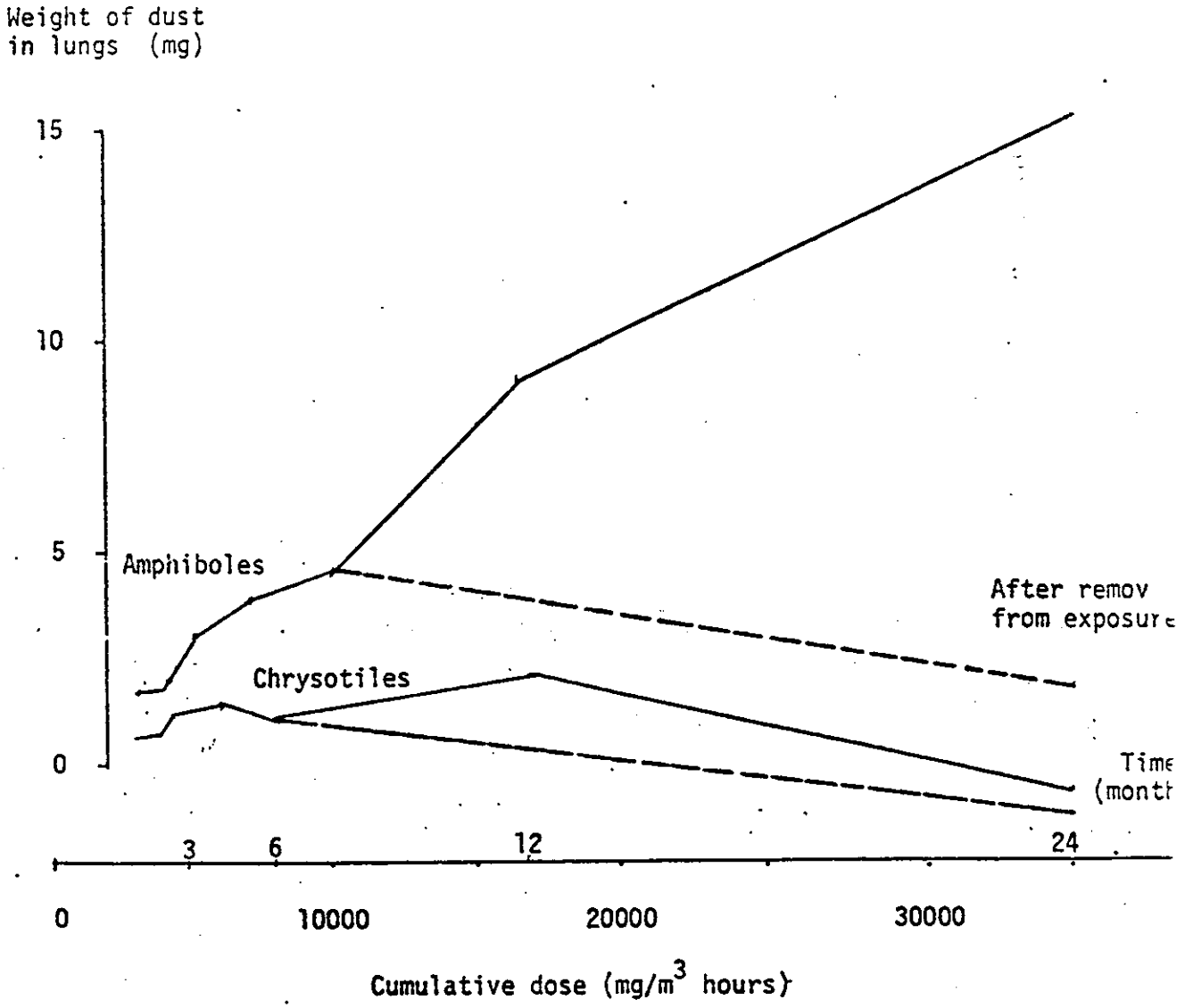
* "Ferruginous bodies" is a more descriptive term, as other inhaled fibers, e.g., fibrous glass, may also become iron coated.

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FIGURE 1

Effects of Inhalation of Asbestos in Rats



– Mean weight of dust in lungs of rats in relation to dose and time.

Wagner et al. (1974)

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Epidemiologic Studies

Harries (1968) reported that although first impressions would lead one to believe that only workers continuously exposed to asbestos are at risk of developing asbestosis, further consideration of the industry and processes should have suggested that many other workers were also at risk. For example, some trades worked in confined spaces where asbestos was used. Work in shipboard trades was accepted by the Pneumoconiosis Panel of the United Kingdom as associated with asbestosis.

Murphy et al (1971a) found that asbestosis was 11 times more common among pipe insulators involved in new ship construction than among a control group. Asbestosis first appeared 13 years after exposure or at about 60 mppcf-years. The prevalence was 38% after 20 years. They also reported a case of extensive pleural calcification in a worker whose only known asbestos exposure was during sanding asphalt and vinyl tile floors (Murphy et al. 1971b).

Lorimer et al. (1976) in a study of brake repair and maintenance workers exposed to asbestos found 25% of the workers showed evidence of x-ray abnormalities consistent with asbestosis. One quarter also had restrictive pulmonary function test findings.

Meurman et al. (1973) found a three-fold risk of dyspnea and a two-fold risk of cough for asbestos workers as compared with controls, after adjusting for smoking.

Weill et al (1975) reported a decreased lung function in relation to increasing cumulative dust exposure in a group of asbestos cement manufacturing workers. Ayer and Burg (1976) reported a decrease in pulmonary function in asbestos textile workers with less than ten years of exposure.

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In a study of 232 former insulation plant employees, Selikoff (1976a) reported positive x-ray findings among individuals having exposures to asbestos known to be as short as one day. More recently, Anderson et al (1976) reported x-ray findings consistent with asbestosis in household and family members having no known exposure to asbestos other than residing with a known asbestos worker. These two studies demonstrate the presence of asbestos disease in the absence of continuing new exposures.

Wagoner et al. (1973) demonstrated a significantly increased risk of death for nonmalignant respiratory disease and for diseases of the heart, which in part were secondary to pulmonary disease, among a cohort of workers in a major manufacturing complex using predominately chrysotile. Among those workers observed 20 or more years after onset of employment, a four-fol increased risk of death due to nonmalignant respiratory disease was observed. Further evaluation of these deaths revealed the majority occurred within 1 year after termination of employment and at an average age of 53.8 years.

Newhouse (1969) reported an increased risk of death from nonmalignant respiratory disease in male asbestos textile and insulation workers with low to moderate exposure.

Enterline and Henderson (1973) reported that for all ages, only 18 death occurred due to asbestosis in several asbestos plants studied from 1941 to 1969. It is of significance, however, to note that the state of New Jersey alone, in the years 1969-1970, had awarded workmen's compensation for asbestosis to 455 workers from one of the plants in the study. (Heymann 197 Serraino 1970)

Selikoff (1976a) reported a significant excess of deaths due to asbest among a group of workers in the U.S. and Canada. Out of 17,800

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asbestos insulation workers, there were 119 observed deaths attributed to asbestosis. Although it was not reported, the expected death rates from asbestosis in the general population would be virtually zero.

DESCRIPTION OF ASBESTOSIS

Asbestosis is a chronic lung disease due to the inhalation of asbestos fibers and is characterized by diffuse interstitial fibrosis, frequently associated with pleural fibrosis (thickening) or pleural calcification.

The characteristic x-ray changes of asbestosis are small irregular opacities in the lower and middle lung fields, often accompanied by pleural thickening and pleural calcifications.

The pulmonary fibrotic changes develop slowly over the years--often progressively even without further exposure--and their radiographic detection is a direct correlate of their extent and profusion. In some cases, minor fibrosis with considerable respiratory impairment and disability can be present without equivalent x-ray changes. Conversely, extensive radiographic findings may be present with little functional impairment.

Commonly found in asbestosis are pulmonary rales, dyspnea, finger clubbing and cyanosis, but any or all can be absent in any one case.

Pulmonary hypertension is frequently associated with advanced asbestosis and the resultant cor pulmonale (right-sided heart failure) may be the cause of death.

CARCINOGENICITY

OCCUPATIONAL EXPOSURE

Historical Studies

In 1935, 55 years after the start of large-scale usage of asbestos in industry, suspicion of an association between asbestosis and lung cancer

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was reported by Lynch and Smith (1935) in the USA and by Gloyne (1935) in the UK. About 10 years later, case reports of pleural and peritoneal tumors associated with asbestos appeared (Wedler 1943 a,b; Wyers 1946). Epidemiologic evidence from Doll (1955) showed a ten-fold excess risk of lung cancers in those UK asbestos textile workers who had been employed before 1930, before regulations produced improved dust conditions in factories. Similar findings were reported in the USA in 1961, but in addition, mesotheliomas were reported which were then published later (Mancuso and Coulter 1963; Selikoff et al 1964). Possible variations in risk with different types of fiber were rarely considered in the early reports. Since 1964, following the recommendations of the UICC Working group on Asbestos Cancers (UICC 1965) for new studies, there has been an expansion of epidemiological studies in many parts of the world.

Epidemiologic Studies

Lung Cancer, Pleural and Peritoneal Mesotheliomas

Mixed Types of Fiber

In most industrial processes different types of fiber are mixed, so that pure exposures to a single asbestos type are rare. Mortality studies of defined populations of asbestos-manufacturing, insulating, and shipyard workers have provided the most concrete evidence concerning the association between bronchial cancer, pleural and peritoneal mesotheliomas and exposure to asbestos. Reports have come from several countries; (UK) Newhouse 1969; (FRG) Bohlig et al , 1970; (USA) Selikoff et al 1970; (UK) Elmes and Simpson 1971; (The Netherlands) Stumphius 1971; (Italy) Rubino et al 1972.

A seven-fold excess of lung cancer was found in a group of insulation workers whose exposures had been to chrysotile and amosite but not crocidolite (Selikoff et al 1973). Enterline and Henderson (1973) reported a 4.4 X

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increased risk of respiratory cancer mortality among retired men who had worked as production or maintenance employees in the asbestos industry and had been exposed to mixed fibers. Among men with mixed exposure to crocidolite and chrysotile in the asbestos cement industry, the rate was 6.1 times the expected rate. In a British naval dockyard population, Harries (1976) showed that there had been a steep rise in mesotheliomas since 1964. However, the full biolocial effects of asbestos in shipyard workers would not have been expected to be detected until the 1970's and thereafter (Selikoff, 1976a).

Edge (1976) reported that shipyard workers with mixed asbestos exposure and pleural plaques (without evidence of pulmonary fibrosis) have a 2.5 times increased risk of developing carcinoma of the bronchus, when compared with matched controls without plaques. In a study of sheet metal workers (Cooper et al 1975) with measurable and mixed asbestos exposure, an excess of deaths from malignant neoplasms (24.7% of deaths for two cohorts selected for 5 or more years worked in the trade, 19.1% of deaths for a group with death claims where 14.5% was expected) was largely attributed to an excess of malignant tumors of the respiratory tract. Of the 307 deaths in the first cohort, 32 lung cancer deaths were significantly in excess (1.7 times the expected). One pleural mesothelioma was observed.

Additional confirmatory evidence of the association between mesotheliomas and past exposure to asbestos comes from many institutes and departments of pathology and cancer registers (e.g., (France) DeLajarte et al. 1973; (Italy) Gobbato and Ferri 1973; (South Africa) Webster 1973; (UK) Greenberg and Lloyd Davies 1974; (Fed. Rep. Germany) Hain et al. 1974, (Finland) Nurminen 1975; (German Democratic Republic) Sturm 1975; (The Netherlands) Zielhuis et al. 1975). These studies have shown an association between asbestos and mesothelioma even with exposures as brief as one day;

however, approximately 15% of mesotheliomas are not known to be related to exposure to asbestos. Three studies (McDonald et al. 1973, Greenberg and Lloyd Davies 1974, Newhouse et al 1972) showed a poor correlation between certified cause of death and histologic diagnosis of mesothelioma. There is still a need to reduce the inter-observer variation in the diagnosis of these rare and pleomorphic tumors (McCaughey and Oldham 1973).

The ratio of pleural to peritoneal tumors reported varies widely in different studies, and peritoneal tumors appear to be associated with heavier exposure (Newhouse et al 1973). Among a number of occupationally exposed groups studied, approximately 5 - 7% of deaths have been from mesothelioma (Gilson 1973; Hammond and Selikoff 1973: Selikoff 1976b). More recently however, an estimate has projected that 11% of asbestos workers' deaths in England will be from mesothelioma (Newhouse and Berry 1975).

Individual Types of Fibers

Crocidolite

In 1956, Wagner started investigating the occurrence of pleural and peritoneal mesotheliomas in the crocidolite mining areas of the Northwest Cape Province in South Africa. It was shown that these tumors occurred in the non-mining population living in the vicinity as well as among men working in the mines and mills and in the transportation and handling of the fiber (Wagner et al 1960). Asbestosis was not invariably present. The latent period between first exposure and clinical recognition of the tumor was long - a mean of 40 years. Subsequent surveillance of the mining population in all the asbestos-producing areas in South Africa has added support for a major difference in the incidence of mesothelioma within the crocidolite mining areas of that country, (Harrington et al 1971; Webster

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1973). The mining of crocidolite in northwest Australia has been associated with mesotheliomas (McNulty 1962). Jones et al (1976) have reported a high incidence of mesotheliomas among women who worked with crocidolite in a factory producing gas mask canisters during World War II.

Chrysotile

McDonald et al (1973, 1974) reported that the overall death rate among 11,500 workers born between 1891 and 1920 and employed in the chrysotile mines and mills of Quebec was lower than for Quebec Province as a whole. An increased lung cancer risk was found and considered to be dose-related, and those who had been most heavily exposed to the dust showed about a five-fold risk compared with the least exposed. Of the 3270 deaths, 134 were from respiratory cancer, with 129 being lung cancer and 5 mesothelioma. Now the authors (McDonald et al 1976) have observed 3938 total deaths among males through 1973, of which 224 were from lung cancer and 7 from mesothelioma. The authors suggest that the respiratory cancer mortality in the Quebec chrysotile industry as a whole was greater than that expected on the basis of regional mortality data.

Kogan et al (1972) investigated the cancer mortality among workers in asbestos mining and milling industries between 1948 and 1967. The total cancer mortality rate among workers was 1.6 times higher than in the general male population; for female workers the rates were 0.8 for those in mines and 1.3 for those in mills. The lung cancer risk for male miners and millers was twice that of the general male population. For females in mines and mills the risks were 2.1 and 1.4 times that of the general female population respectively. For those workers over 50 years of age the lung cancer risk was greater: for men in mining, 4.9; those in milling, 5.9; for women in mining, 9.5; and for those women in milling, 39.8 times

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that in the general population. No mesotheliomas were found, but Kogan indicates that this might be explained by the insufficient experience of pathologists with this rare type of cancer in that geographical area. Also, the numbers of people in the study populations were not reported.

Wagoner et al (1973) reported on the cancer risk among a cohort of workers in a major manufacturing complex utilizing predominately chrysotile asbestos in textile, friction and packaging products. An excess of respiratory cancer occurred among asbestos workers in each duration-of-employment category down to and including 1-9 years. They observed a statistically significant standard mortality ratio of 122 for all malignant neoplasms and 244 for malignant neoplasms of the respiratory system. The asbestos workers in this study were located in an area of predominately Amish Dutch population with known low frequencies of smoking. The authors, nevertheless, used the general white male U.S. population as a control group, which would tend to underestimate the degree of risk.

Enterline and Henderson (1973) found that for retired men who had worked as production or maintenance employees in the asbestos industry and who had reached 65 years of age, those who had been exposed only to chrysotile had a respiratory cancer risk 2 - 4 times than that expected. Among men within the asbestos cement industry exposed only to chrysotile, a one- to four-fold excess of respiratory cancer was found. Of 802 deaths, only one mesothelioma had been recorded in the several plants investigated. In contrast, a subsequent investigation by Borow et al (1973) found 70 cases of mesothelioma from only one of these plants. The discrepancy was due to methodological variations, for example Enterline and Henderson (1973) had limited their investigation to men of 65 or over, while many of the mesothelioma cases reported by Borow et al (1973) had died before that age.

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Amosite

In a study of a group of miners exposed to amphibole fibers in the cummingtonite-grunerite ore series, Gillam et al (1976) have demonstrated mortality from malignant respiratory disease three times that of the general population.

Exposures to amosite alone in a factory making insulation material were reported by Selikoff et al (1976 a & b). Ten mesotheliomas were found in addition to an increased risk of lung cancer in workers who were observed 20 years or longer. The excess lung cancer risk in the amosite workers was shown to increase with duration of employment. There was a three-fold increase in lung cancer among those with less than 3 months employment and among those with less than 1 month employment there was a 2.25-fold increase.

In a retrospective study of 914 men who had worked for various periods of time during World War II in a plant manufacturing insulating materials from amosite for the U.S. Navy, Seidman et al (1976) concluded that the group of 65 men who had worked for less than 1 month had experienced excess mortality, on the age-specific basis, from lung cancer during the 30 years since the beginning of their exposure, but not from all cancers or all causes of death. Men who had worked for a full month or longer had excess mortalities from all three causations examined, the risk of death from lung cancer increasing with duration of exposure.

Anthophyllite

In Finland, anthophyllite mining has been associated with an excess bronchial cancer risk of 1 - 4 times expectation overall, and about double this figure for those with more than 10 years' exposure (Meurman et al 1974).

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There was also a higher prevalence of dyspnea and cough in the miners. However, no mesotheliomas were found despite the presence in Finland of an unusually high incidence of pleural thickening and calcification as detected by radiographic and pathologic surveys (Kivilioto 1960; Meurman 1966).

Other Cancer

Epidemiologic studies of the already defined populations have consistently shown an excess risk of other cancers, especially of the gastrointestinal tract (Mancuso and El Attar 1967; Elmes and Simpson 1971; Kogan et al 1972; Newhouse 1973; Wagoner et al 1973; McDonald et al 1974; Selikoff et al 1974); however, it has been less than that of lung cancers.

Schneiderman (1974) in a literature review, with an emphasis on dose-response, concluded that "good dose-response data, with quantitative estimates of dose are uncommon; however, in all the literature reviewed only one paper did not support the conclusion that increased exposure to inhaled asbestos particles leads to increased digestive system cancer."

Stell and McGill (1973) found that out of 100 men with squamous carcinoma of the larynx, 31 had known exposure to asbestos compared with only three in matched controls. Similar associations have been reported by Morgan and Shettigara (1976). Newhouse and Berry (1973) found two cases of cancer of the larynx (ICD 161) in their cohort of over 4000 workers compared with an expected 0.4.

NONOCCUPATIONAL EXPOSURE

Household contact with asbestos is associated with an increased mesothelioma risk. Anderson et al (1976) have recently reviewed 34 such cases of mesothelioma from nine countries and reported four new cases among the traced family members of 1664 asbestos workers. Cases of mesotheliomas

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have also occurred in nonoccupationally exposed individuals living in the neighborhood of industrial sources of asbestos (Wagner et al 1960; Newhouse and Thompson 1966; Bohlig and Hain 1973). Studies of the geographical distribution of cases of mesothelioma in the UK over a 10-year period indicate that the new cases are nearly all from areas in which there has been a recognized industrial source of asbestos (Gilson 1970; Greenberg and Lloyd Davies 1974).

Results among nonoccupationally exposed persons in Finland have been reported where anthophyllite asbestos is mined. In this study 118, cases of the total 126 study cases of roentgenologically-diagnosed pleural calcification, excluding those individuals with hemothorax, emphysema, and tuberculosis, lived or have lived in areas immediately adjacent to asbestos mines (Kiviluoto 1960). The results of this study suggest a health hazard due to ambient community exposure to asbestos.

SYNERGISM

There is marked enhancement of the risk of lung carcinoma in those worker exposed to asbestos who also smoke cigarettes (Selikoff et al 1968; Doll 1971; Berry et al 1972; Hammond and Selikoff 1973); Hammond and Selikoff (1973) interpret the excess lung carcinoma risk from asbestos in non-smokers to be small. No link between cigarette smoking and mesotheliomas has been observed in a prospective study by Hammond and Selikoff (1973). A preliminar study (Lemen 1976) on female workers employed between January 1940 and December 1967, in a predominately chrysotile asbestos textile plant, revealec seven lung cancer deaths among 580 women when only 0.63 deaths were expected ($p<0.01$). One lung cancer death was observed in a smoker, two in women of undetermined smoking history, and four in "never" smokers as determined from hospital admission charts.

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It is important to note that the historic documentation of cigarette consumption patterns is lacking for most retrospective cohort studies of asbestos workers. It is further important to note that a sizable portion of the general population, the group usually selected for contrast in these studies, are cigarette smokers. Therefore, the risk of lung cancer demonstrated for these industrial groups exposed to asbestos are of such magnitude as to preclude the interpretation of an independent role for cigarette smoking etiology.

FIBER ANALYSIS IN TISSUE

The physical characteristics of asbestos fibers which penetrate to the lung parechyma have been studied by Timbrell (1965 and 1972) who demonstrated fiber respirability to be largely a function of fiber diameter.

Two kinds of data are relevant. Timbrell et al. (1971) and Timbrell (1972) have shown that the crocidolite mined in Northern Cape Province and in Western Australia is associated with a high incidence of pleural mesothelioma among the local populations and has finer and shorter fibers that the crocidolite or amosite mined in the Transvaal Province, which is associated with a relatively lower incidence of pleural mesothelioma among the exposed population. As crocidolite and amosite are similar in chemical composition, there is reason to assume that the risk difference may be attributable to the differing physical characteristics of fibers.

Preliminary studies (Fondimare et al 1974) concerning diameter and lengt of 5000 asbestos fibers from the lungs of 10 deceased persons who had been occupationally exposed, showed that these fibers were all less than 0.5µm micrometer in diameter. When seperated according to type of asbestos, 90% of chrysotile fibers and 70% of amphibole fibers were less than 5 µm in length

Asbestos bodies have been found in large numbers by light microscopy

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in occupationally exposed individuals (Ashcroft and Heppleston 1973). Numerous asbestos fibers, either of chrysotile or amphibole or both types, have been found by electron microscopy in lungs of industrially exposed men (Pooley 1972, 1973; Fondimare et al 1974). A quantitative topographic study of asbestos fibers in the lung has been carried out in 12 industrially exposed men which showed that heavily exposed cases with lung fibrosis and carcinomas had fewer fibers in the fibrotic lower lobes than in the less fibrotic upper lobes (Fondimare et al 1975). The fibers were mostly of the amphibole type. In less exposed cases with lung cancer but without lung fibrosis, a higher concentration of asbestos fibers, mostly of the chrysotile type, was clearly demonstrated in peripheral areas of the lung.

Optical and electron microscopic study of pleural plaques revealed the presence of some coated fibers and larger numbers of uncoated fibers, mostly short, ultimate fibrils of chrysotile (Fondimare et al 1975).

Pooley (1973) found that 93% of 120 mesothelioma cases studied had asbestos fibers in their lungs visible by electron microscopy versus less than 50% of 135 non-mesothelioma cases. Higher concentrations of fibers were observed in mesothelioma than in non-mesothelioma cases. In mesotheliom cases, the fiber types were either amphibole or chrysotile, or both, but amphibole was predominant; in non-mesothelioma cases, chrysotile fibers were predominant. In the three cases included in the study by Fondimare et al (1975), the percentage of chrysotile fibers was from 44 to 97% in the periphe areas of the lung. The ratio of amphibole to chrysotile has been found to decrease from the central toward the peripheral areas of the lung (Fondimare et al 1975; LeBouffant et al 1976).

Coated fibers ("asbestos" or "ferruginous bodies") have been found in the lungs of most adults who have lived in urban areas (Gross et al 1969;

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Bignon and Goni 1969; Selikoff et al 1972; Thompson et al 1966; Davis and Gross 1973; Oldham 1973). The number of coated fibers in the lung has been compared in cases with and without lung carcinoma. Meurman (1966), who took into account cigarette consumption, could find no significant difference.

Doniach et al (1975) found an increased incidence of asbestos bodies in men with stomach cancer and in women with breast cancer, but not in lung cancer cases. Warrock and Churg (1975) found that lung cancer cases had more coated fibers in their lungs, even though only one case had known occupational exposure. The variations in percentage are probably from methodologic differences. In general, methods involving the counting of fibers per unit of weight or volume of lung tissue have greater associations with health outcomes in epidemiologic studies. However, coated fibers are not specific to asbestos (Gross et al 1968) and cannot be related to asbestos unless the core has been identified as such by electron diffraction and/or x-ray analytical techniques (Pooley 1970; 1975; Langer and Pooley 1973, 1974; Fondimare et al 1975).

Transmission electron microscopy has demonstrated the presence of 1971 chrysotile fibers or fibrils in the lungs of most consecutive autopsy cases in London (Pooley et al 1970), New York (Langer et al 1971) and Pittsburgh (Gross et al 1973).

Although some differences in both the fibrotic and the carcinogenic responses to asbestos fibers have been suggested to depend on the type of fiber administered, all types have been shown definitely to have both these kinds of action (e.g., Karacharova et al (1969), Shin and Firminger (1973), Wagner et al (1976). Godwin and Jagatic (1970), Gross et al (1973), and Taskinen et al (1973) reported finding fibers in lymph nodes and in the spleen, abdomen, and intestinal mucosa of occupationally exposed patients

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with mesothelioma and pleural nodules. This emphasizes the practical importance of penetration and transport of the small fibers of asbestos from their initial sites of impaction. It also stresses the importance of guarding against the entrance of asbestos fibers into the body by any route.

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STUDIES OF HUMAN POPULATIONS
NON MALIGNANT RESPIRATORY DISEASE

Author	Date	Finding	Group and Exposure
Historical Studies			
Murray	1906	First reported case of asbestosis	Asbestos workers
Cooke	1927	Case of asbestosis reported	
McDonald	1927	Two cases of asbestosis reported	
Mills	1930	First case of asbestosis reported in U.S.	
Lynch and Smith	1930	Ferruginous or "asbestosis bodies" found in sputum	
Epidemiological Studies			
Murphy	1971	Asbestosis	Pipe insulators
Lorimer et al.	1976	X-ray abnormalities consistent with asbestosis and restrictive pulmonary function testing	Brake repair maintenance workers
Meurman et al.	1973	Dyspnea and cough	Asbestos workers
Weill et al.	1975	Decreased lung function	Asbestos cement manufacturing workers
Ayer and Burg	1976	Decrease in pulmonary function	Asbestos textile workers with less than 10 yrs. exposure
Selikoff	1976a	Asbestosis	Former insulation plant employees with as little as one day exposure
Anderson et al	1976	X-rays consistent with asbestosis	Household and family members of asbestos worker

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STUDIES OF HUMAN POPULATION CARCINOGENICITY

Author	Date	Finding	Group and Exposure
Occupational Exposure			
Historical Studies			
Lynch and Smith	1935	Suspicion of association	Asbestos workers
Gloyne	1935	Between asbestos and lung cancer	
Wedler Wyers	1943a,b 1946	Case reports of pleural and peritoneal tumors associated to asbestos	
Doll	1955	Lung cancer	Asbestos textile workers employed before 1930
Mancusco and Coulter	1963	Lung cancer and mesotheliomas	Asbestos workers
Selikoff	1964	Lung cancer and mesotheliomas	Asbestos workers
Epidemiological Studies			
Lung, Pleural and Peritoneum			
Mixed Types of Fibers			
Newhouse (UK)	1969	Bronchial cancer, pleural and peritoneal mesotheliomas	Asbestos manufacturing, insulation and shipyard workers
Bohlig et al (FRG)	1970		
Selikoff et al (USA)	1970		
Elmes and Simpson (UK)	1971		
Stumphius (Netherlands)	1971		
Rubino et al (Italy)	1972		
Selikoff et al.	1973	Lung cancer	Insulation workers, chrysotile and amosite asbestos exposure

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NON-MALIGNANT R IRATORY DISEASE (Continued)

Wagoner et. al	1973	Death due to nonmalignant respiratory disease and diseases of heart, in part secondary to pulmonary disease	Chrysotile workers
Newhouse	1969	Death due to nonmalignant respiratory disease	Male asbestos textile and insulation workers
Enterline and Henderson	1973	Death due to asbestosis	Asbestos plant workers
Selikoff	1976a	Death due to asbestosis	Insulation workers and factory workers exposed to asbestos
Bohs	1968	X-ray evidence of asbestos	Workers in asbestos industry in Britain after 1933 with perponderance of less than 20 years exposure
Lewinsohn	1972	X-ray abnormalities	Asbestos workers in Britain
Gillam et al	1976	Nonmalignant respiratory disease	Amosite miners

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Enterline and Henderson	1973	Respiratory cancer	Retired production and maintenance workers in asbestos industry
Harries	1976	Mesotheliomas	Naval dockyard workers
Edge	1976	Carcinoma of bronchus	Shipyard workers
DeLajarte et al (France)	1973	Evidence of association between mesotheliomas and past exposure to asbestos	Occupational exposures in some cases as brief as one day
Gobbato and Ferri (Italy)	1973		
Webster (South Africa)	1973		
Greenberg and Lloyd	1974		
Davies (UK)			
Hain et al (Fed. Rep. Germany)	1974		
Nurminen (Finland)	1975		
Stunn (Ger. Dem. Rep.)	1975		
Zielhuis (The Netherlands)	1975		
Newhouse et al	1973	Peritoneal tumors associated to heavy exposures	
Gilson	1973	5% to 7% asbestos workers' deaths due to mesotheliomas	
Hammond and Selikoff	1973		
Selikoff	1976		
Newhouse and Berry	1975	11% asbestos workers deaths due to mesotheliomas	

Single Types of Fibers

Crocidolite

Wagner	1960	Pleural and peritoneal cancer	Workers in mines, mills and in transportation and handling of crocidolite and population in vicinity of mines
Harrington et al	1971	Mesothelioma	Mining population of crocidolite mines
Webster	1973		

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McNulty	1962	Mesothelioma	Miners of crocidolite
Jones et al	1976	Mesothelioma	Women working with crocidolite in WWII gas mask canister factories
Chrysotile			
McDonald et al	1973, 1974	Lung cancer	Chrysotile mine and mill workers
Kogan et al	1972	Total cancer Lung cancer	Workers in asbestos mining and milling, men and women
Wagoner et al	1973	Respiratory cancer	Workers in manufacturing of textile, friction and packaging products using chrysotile
Enterline and Henderson	1973	Respiratory cancer	Men 65 yrs. and older, retired production or maintenance employees in asbestos industry exposed only to chrysotile
Borow et al	1973	Mesthelíomas	Workers at plant using chrysotile, all ages
Amosite			
Gilliam et al	1976	Malignant respiratory disease	Miners exposed to amphibole fibers in cummingtonite-grunerite ore series
Selikoff et al	1976a,b	Mesotheliomas, lung cancer	Insulation workers in factory using amosite
Anthophyllite			
Neurman et al	1974	Bronchial cancer, dyspnea and cough	Anthophyllite mining employees

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Other Cancer			
Mancuso and El Attar	1967	Cancer of gastrointestinal tract	Asbestos workers
Elmes and Simpson	1971		
Kogan et al	1972		
Newhouse	1973		
Wagoner et al	1973		
McDonald et al	1974		
Selikoff et al	1974		
Stell and McGill	1973	Squamous carcinoma of larynx	Workers with exposure to asbestos
Morgan et al	1976		
Newhouse and Perry	1973	Cancer of larynx	Asbestos worker
Non-Occupational Exposure			
Anderson et al	1975	Mesothelioma	Family members of asbestos workers
Wagner et al	1960	Mesothelioma	Individuals in neighborhood of industrial sources of asbestos
Newhouse and Thompson	1965		
Bohlig and Hain	1973		
Gilson	1970	Mesothelioma	New cases from areas with recognized industrial source of asbestos
Greenburg and Lloyd	1974		
Davies			
Kiviluoto	1960	Pleural plaques	Persons in farming region of Bulgaria where minute quantities of anthophyllite, tremolite and sepiolite in soil and non occupationally exposed persons in anthophyllite mining area of Finland
Other Studies			
Newhouse	1969,1973	Cancer, mesothelioma	Women factory workers exposed to chrysotile, amosite and crocidolite
Newhouse et al	1972		

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Howard et al	1976	Lung cancer	Workers in asbestos industry from 1933 to 1950 and after 1950
Cooper et al	1975	Lung cancer	Sheet metal workers with 5 or more years exposure

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IV. SAMPLING METHODS AND ENVIRONMENTAL DATA

REVIEW OF SAMPLING AND ANALYSIS TECHNIQUES FOR ASBESTOS

A variety of sampling and analysis techniques have been used to identify asbestos fibers and determine their concentrations in air, water, mineral samples and biological tissue. These include optical and electron microscopy, x-ray diffraction and differential thermal analysis. Asbestos fiber identification and quantitation in occupational and environmental air samples is difficult for a variety of reasons:

1) Asbestos fibers are generally present in low mass quantities even though fiber number concentrations may be high.
2) Many instrumental analytical techniques can not differentiate asbestos fibers from their non-fibrous mineralogic polymorphs.
3) Many airborne asbestos fibers are generally below resolution limits of the optical microscope. These fibers may only be detected by using electron microscopic methods.
4) For identification of the various asbestos fiber types by electron microscopy, electron diffraction and micro-chemical analyses must be performed which require expensive instrumentation and analysis time.

Electron Microscopy And Microchemical Analysis

Both transmission and scanning electron microscopy have been used for asbestos fiber identification and quantitation. In addition to morphologic observation, selected area electron diffraction and microchemical analytical techniques may be used for fiber identification.

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In addition to superior resolution capabilities, most modern transmission electron microscopes are equipped with electron diffraction facilities. Crystalline materials scatter electrons in regular patterns related to their crystal structure. The image of the scattered electrons is mainly predicted by Bragg geometry. In the transmission electron microscope, the diffraction image is formed in the back focal plane of the objective lens and is focused on the viewing screen by defocusing the intermediate lens. Visual observation of single fiber (single crystal) electron diffraction patterns may be used to differentiate chrysotile fibers from amphibole fibers (Langer et al 1974, Timbrell 1970). Chrysotile fibers produce streaked diffraction patterns (due to lattice defects), with the streaks or layer lines nearly perpendicular to the fiber length. The spacing between the layer lines denotes the fiber "a" axis of approximately 5.3 $\mathring{A}$. Reflections along the layer lines are usually very streaked and Debye-Scherrer rings are common. With progressive electron beam bombardment, the diffraction pattern may change due to fiber damage. The "central core" of chrysotile fibers may also aide in fiber identification with the precaution that the central core is not always discernable and may disappear with the beam damage (Langer et al 1974). Also, other fibrous minerals may have hollow cores.

The amphibole minerals are generally straighter in appearance than chrysotile fibers. In addition, light and dark banding (diffraction images) may cross the fiber at right angles (Langer et al 1974). Diffraction contrast figures have been observed on all amphibole fiber types. Selected area diffraction patterns for the amphibole asbestos minerals are all similar in appearance; therefore, visual observation of these patterns is sufficient only to classify the fiber as being a fibrou

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amphibole (Langer et al 1974, Cook et al 1974). Amphibole electron diffraction patterns show layers and sometimes streaks perpendicular to the fiber length with the spacing between the layer lines or streaks representing the fiber "c" axis (Langer et al 1974) of approximately 5.3 Å. In contrast to chrysotile, less streaking along the layer lines is observed with the spot repeat along the lines representing one of the two remaining lattice spacings ("b" or "a") depending on fiber orientation relative to the electron beam. Typically, approximately 30 seconds is needed to perform a selected area electron diffraction analysis on a single fiber.

In addition to visual observation of electron diffraction patterns for fiber identification, photographs can be made of the diffraction patterns and crystal "d" spacings measured from the plate and calculated using the instrument camera constant (Timbrell 1970). Both "spot" and polycrystalline patterns may be measured. It must be bornein mind that intensities may not be the same as observed for x-ray powder patterns and additional reflections may be present.

Electron beam microchemical analytical techniques may sometimes be used to identify asbestos fibers from other fibrous particles (Rubin and Maggiore 1974, Ferrell et al 1975, Langer et al 1975, Maggiore and Rubin 1973). The most common system presently in use is the energy dispersive x-ray detector in combination with a scanning or transmission electron microscope. Wavelength x-ray analyzers and the conventional electron microprobe have been used; however, their routine application is limited due to data acquisition times (Langer et al 1975). On the other hand, data acquisition times with energy dispersive analyzers are far less ranging from 20 to 80

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seconds per analysis.

Semi-quantitative microchemical analysis in the electron microscope is based on the fact that a beam of high energy electrons incident on an asbestos fiber generates x-rays characteristic of the elements present in that fiber. The generated x-rays are detected by a detector (lithium-drifted silicon crystal) placed in the electron microscope column close to the specimen. The energy of the x-ray photon is converted to a voltage pulse which is amplified, digitized and stored in a multi-channel analyzer or a mini-computer. The content of the memory is usually displayed on a CRT (Maggiore and Rubin 1973). With the energy dispersive detector, all elements with atomic numbers of sodium or higher may be analyzed. Continuous background or brehmsstrahlung radiation is always present with the x-ray spectrum.

Each of the asbestos minerals has an x-ray spectrum which is usually characteristic enough, when combined with fiber morphology, to allow it identification (Rubin and Maggiore 1974, Ferrell et al 1975, Dement et al 1975). Visual observation of the semi-quantitative fiber x-ray spectra is usually sufficient for fiber identification; however, three component diagrams have been used after subtracting the continuous background from the semi-quantitative x-ray spectrum (Ferrell et al 1975). For asbestos fiber analysis, matrix corrections are rarely used. Typically, iron, magnesium and silicon are plotted on the three component diagram and compositional boundaries for the asbestos minerals established. This technique suffers from inability to use all compositional data obtained, such as presence or absence of sodium, calcium, aluminum and manganese, which aid in identification.

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With energy dispersive x-ray techniques, possession of proper elemental intensities may not be sufficient for positive identification as many fibrous minerals show similar elemental intensities. For example, chrysotile, anthophyllite and fibrous talc, which have similar elemental compositions, may be difficult to differentiate. However, these materials may easily be distinguished using selected area electron diffraction. In addition, unique identification of the various fibrous amphiboles usually requires both selected area diffraction and microchemical analysis. Transmission electron microscopes equipped with an energy dispersive x-ray detector are now available which allow simultaneous observation of morphology, crystal structure and elemental composition. These microscopy systems have been used to study asbestos fibers in environmental and material samples. (Cook et al 1974, Dement et al 1975).

Quantitative analysis of asbestos fiber concentrations in environmental and tissue samples has been accomplished using electron microscopy. Environmental samples (water and air) are generally collected by first concentrating the sample by filtration, centrifuging, etc (Cook et al 1974, Nicholson 1974). The filters (Millipore) and polycarbonate filters (Nuclepore) are prepared for electron microscopic analysis by various methods. For scanning electron microscopy, Nuclepore filters, due to their smooth surface, may be directly coated with an appropriate metal (gold, etc.) and analyzed (Porter and Berggren 1974). Millipore filters have a rough surface texture and are not generally suitable for direct coating for scanning electron microscopy as small fibers may escape detection due to impaction below the filter surface (Nicholson 1974).

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For transmission electron microscopy, the filter substrate must be removed and the particles mounted on suitable electron microscopy grids. A wide variety of mounting techniques has been used. The two most commonly used methods are the Jaffe Wick and condensation washing techniques. The techniques offer simplicity in addition to maintaining the original particle size distribution of the sample. Different investigators have reported particle losses up to 60% with Millipore filters while using the condensation washing method with rapid filter dissolution, whereas losses with the Jaffe Wick method have been reported to be considerably less (<10%) (Beaman and File 1975). Lesser particle loss has been observed with the condensation washing method when longer times for dissolution of the filter are used. Ortiz and Loom (1974) reported that a modification c the Jaffe Wick method, whereby the filter is first coated with silicon monoxide and carbon by vacuum evaporation prior to dissolving the Millipore filter, minimized particle loss. Several investigators have reported minimal particle loss with Nuclepore filters when the filter is first carbon coated prior to dissolving the filter substrate (Cook et al 1974, Maggiore and Rubin 1973).

In addition to the so called direct clearing/mounting techniques mention above, a variety of other techniques has also been used for preparing environmental samples. Selikoff et al (1972) have used a so called "rub-out" technique whereby the Millipore filter is ashed in a low temperature asher to remove organic or carbonaceous material. The residue is then dispersed on a microscope slide using a solution of 1% Nitrocellulose in amyl acetate. After grinding with a watch glass to liberate individual fibers, the sample is dispersed evenly between two microscope slides to form a thin film which is transferred to standard electron microscope grids. Particle losses

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averaging 50% have been reported with this technique. This technique also increases the apparent number of fibers present due to breaking up of fiber bundles. Asbestos fiber levels in environmental samples and biological tissue are usually expressed as asbestos fibers per unit volume of sample (fibers/m^3, fibers/liter, fibers/gm dry lung etc.) These concentrations are determined by counting fibers within calibrated areas on the electron microscope viewing screen or counting fibers from photographs. Asbestos fiber concentrations in water samples determined by laboratories using the same mounting techniques have been reported to vary by a factor of 2-3 (Cook et al 1974). Much larger variations have been reported between laboratories using different techniques.

Asbestos mass (chrysotile) concentrations in environmental samples have also been determined using electron microscopy. This is accomplished by measuring the length and diameter (volume) of each fiber and calculating the mass using the appropriate density (Selikoff et al 1972). The accuracy of this technique has not been studied in detail.

Electron microscopic techniques represent the "best available" methods currently available for asbestos fiber analysis. However, application of these techniques to routine samples is not practical due to extremely high analysis costs ($200-$400 per sample), long analysis times, and limited equipment availability.

X-Ray Diffraction

X-ray powder diffractometry is one of the standard mineralogic techniques used in the analysis of solid crystalline phases. X-ray diffraction has been widely used for identification and quantitation of asbestos fibers in bulk materials such as talc (Stanley and Norwood 1974;

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Rohl and Langer 1974) and other industrial materials (Crable and Knott 1968, Keenan and Lynch 1970).

X-ray diffraction has also been used to study amphibole asbestos contamination of water samples (Cook et al 1974). X-ray diffraction is generally considered more sensitive for asbestos than light microscopy, although less sensitive than electron microscopy (Rohl and Langer 1974).

Diffraction lines and relative intensities for each of the asbestos minerals have been published and may be found in the ASTM Powder Diffraction File. Variations in asbestos fiber chemical composition, especially for the amphiboles, may result in slight peak shifts from reported x-ray diffraction data.

Quantitative determinations of asbestos fiber levels in material samples (talc, etc.) require that particle size first be reduced to an average of 0.1 - 10 μm. Preferred orientation and surface roughness must also be eliminated.

A number of techniques has been used to minimize preferred orientation effects including binder and slurry mounting methods, sifting and backloading of dry powders and several other techniques. To minimize preferred orientation, Rohl and Langer (1974) have developed a method for filtering an aqueous slurry through Millipore filters using a filtration adapter attached to a hypodermic syringe. Other investigators have used the backloading technique with multiple x-ray diffraction scans.

Using conventional scan rates (0.5 to 1 degrees 2 theta per minute), lower limits of detection of asbestos by x-ray diffraction of 5% in bulk samples have been reported (Crable and Knott 1966). Automated step scanning procedures by which diagnostic reflections are slowly scanned and integrated

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counts recorded, have been reported to significantly reduce detectable limits. Rohl and Langer (1974) have detected anthophyllite at 2.0%, chrysotile at 0.25%, and tremolite at 0.10% by weight in a talc matrix using external dilution standards for calibration. Similar lower detectable levels have been reported by Stanley and Norwood (1974).

Application of x-ray diffraction for routine asbestos fiber analysis of environmental samples has been limited. Birks et al (1975) have reported a feasibility study concerning quantitative analysis of airborne asbestos. Their technique involved alignment of the asbestos fibers in an electrostatic field to enhance diffraction intensity followed by x-ray counting in a specially designed diffraction apparatus with two x-ray detectors. A lower limit of detection of 0.4 - 0.5 µg was reported. This technique has not been applied to actual environmental samples.

Amphibole and cummingtonite-grunerite mass concentrations in water samples have been semiquantitatively determined using x-ray diffraction with step scanning (Cook et al 1974). This technique involves filtering the water through 0.45 µm Millipore filters followed by step scanning a major amphibole diffraction peak (110) and a peak specific to cummingtonite-grunerite (310). The integrated peak count above background is recorded and mass concentrations determined using external dilution standards.

Proper selection of diagnostic reflections to maximize detection sensitivity and minimize interference due to other mineral phases is necessary for proper use of x-ray diffraction. It must also be recognized that x-ray diffraction methods are not capable of differentiating between asbestos fibers and their non-fibrous mineralogic polymorphs. This fact, combined with relatively poor detection levels, suggests that alternate techniques such as electron microscopy should be combined with x-ray analysis.

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Differential Thermal Analysis

Differential thermal analysis has been used to determine asbestos fiber levels in talc samples (Schlez 1974). Chrysotile (serpentine minerals) shows a dehydroxylation endotherm at approximately 650°C and an exotherm at approximately 820°C, associated with the formation of forsterite. These peaks may be used for quantitative analysis. Using a 140 mg sample holder with an exposed loop differential thermcouple and a 10°c/min. heating rate, Schlez (1974) reported that a 1% concentration of chrysotile could be detected in pharmaceutical grade talc. A dynamic helium atmosphere was maintained to sweep out gaseous mineral decomposition products and to prevent oxidative reactions.

Differential thermal analysis has not been used for environmental samples as lower limits of mass detection are extremely poor. Differential thermal analysis, like x-ray diffraction, is not capable of differentiating between asbestos fibers and their non-fibrous mineralogic polymorphs.

Optical Microscopy

A number of optical microscopic techniques have been used to identify and/or quantitate asbestos fibers in environmental samples. These include petrographic and phase contrast microscopy. Petrographic microscopic techniques may be used to identify asbestos fibers greater than approximately 0.2 - 0.3 μm in diameter. Using the polarizing microscope, various optical crystallographic measurements such as refractive index, extinction angles and sign of elongation may be measured and compared with data reported for standard asbestos reference samples. Typical optical data for selected asbestos minerals are shown in Table 1. (Julian and McCrone 1970)

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Dispersion staining with polarized light has been used to identify asbestos fibers as reported by Julian and McCrone (1974.) With this technique the fibers are immersed in a mounting medium with a steeper dispersion curve than the fibers. A central or annular stop is used in the objective lens back focal plant to allow either the wavelength of light at which the index of the particle matches that of the mounting media or complements to that color to reach the observers eye. Using plane polarized light, asbestos fibers show two characteristic dispersion staining colors; one for the light vibration parallel, and another for that perpendicular, to the fiber length. The dispersion colors depend on the refractive index media in which the fibers are mounted, as shown in Table 2. Dispersion staining colors may change slightly depending on the geographic area from which the asbestos was mined and subsequent treatment. Fibers less than 0.5 µm in diameter may not be identified by this technique due to difficulties in distinguishing colors.

Phase contrast optical microscopy is the technique specified for determining compliance with the Occupational Safety and Health Administration asbestos standard (U.S. Department of Labor 1975). The method consists of collecting breathing zone samples during 15 minute to 8 hour periods on membra filters (Millipore AA). Samples are analyzed by first clearing the membrane filter to make it optically transparent followed by fiber counts at 400-500x magnification by phase contrast optical microscopy. Asbestos fibers are defined as those particles with a length greater than 5 µm and a length-to-diameter ratio of 3 : 1 or greater. This technique, by which only fibers longer than 5 µm are counted, is recognized as only an index of total fiber exposure and does not imply that shorter fibers do not pose a health hazard. The relative proportion of airborne fibers longer than 5 µm has

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been shown by Dement et al. (1975) to vary from 1 to approximately 50% depending on industrial operation and asbestos fiber type. In addition to problems of detecting short fibers, phase contrast microscopy may not be specific to asbestos fibers in industrial operations where mixed fiber types are encountered.

Despite its limitations, phase contrast microscopy represents the only technique, presently available, that reasonably can be used for routine asbestos fiber sampling and analysis. It is adaptable to personal sampling where low air volumes are sampled and analysis equipment is readily available.

Minimum detectable fiber concentrations by phase contrast microscopy depend on a number of factors such as air volume sampled, microscope field counting area, number of microscopic fields counted and presence or absence of non-fibrous particles. Theoretical minimum detectable concentrations may be calculated assuming one fiber longer than 5 μm is observed per 100 microscopic fields (after filter background subtraction). Table 3 shows theoretical minimum detectable fiber concentrations as a function of sample period for a typical microscope arrangement. For a 15-minute sampling period, 0.04 fibers >5μm/cc may be detected; however, with an 8 hour sample, 0.001 fibers/cc can be detected. These minimum concentrations are similar to those reported by Corn and Sansone (1974). These authors reported that 0.01 fibers/cc could be detected with a 2-hour sample period (40 microscopic fields counted).

The above calculations represent theoretical minimum detectable concentrations, not considering the many factors affecting precision and accuracy of the technique. There are many sources of variability in the

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laboratory analysis technique. The major sources of variability are as follows:

1) Variability of fiber distribution across the filter surface.

2) Variability of fiber distribution on a given filter wedge being analyzed.

3) Variability due to differences between microscopes.

4) Variability due to differences between individual counters.

5) Variability among laboratories.

Leidel and Busch (1974) found that the fiber distribution on a given filter section could best be described by the Poisson-distribution. However, Conway and Holland (1973) found that the distribution of fibers on filters was not uniform and were more disperse than predicted by the Poisson distribution, so that concentrations between sections could vary by as much as 50-60% Similar results were found by Rajhans and Bragg (1975) in Series I of their study.

If the Poisson distribution is taken to adequately describe fiber distributions on a filter sections, the standard deviation of the fiber count may be estimated from the square root of the count. In order to maintain an acceptable Coefficient of Variation (CV) (below 20%), a minimum of 25 fibers must be counted. For a typical industrial asbestos sample of 2 hours (2 lpm flow), this would correspond to a concentration of 0.13 fibers/cc.

The precision of the entire sampling and analysis procedure (all sources of variability) has been estimated by Leidel et al , (1975) These authors estimated the total Coefficient of Variation to be 22%.

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COMPARISONS OF ASBESTOS MASS CONCENTRATIONS
(ng/m^3) AND FIBER NUMBER CONCENTRATIONS (fibers/cc)

In order to relate ambient asbestos levels, which are generally expressed as ng/m^3, to occupational exposures, which are expressed as fibers >5 μm in length/cc, a conversion factor is needed. Attempts to formulate such a conversion have generally been unsuccessful because of exceptionally large variability. This is to be expected as ambient levels are generally determined using electron microscopy whereas phase contrast microscopy is used to measure occupational exposures. In addition, techniques used to prepare samples for electron microscope observation may cause alterations in fiber size (diameter and length) distributions.

Lynch and Ayer (1966) presented results of environmental studies in the asbestos textile industry where fiber concentrations were determined using phase contrast optical microscopy and fiber size distributions determined using electron microscopy. The mass of chrysotile on the filter was estimated by using atomic absorption spectroscopy to determine the magnesium content of the sample and asbestos content was calculated; assuming a 25% magnesium content for chrysotile. These data are summarized in Table 4. Based on the magnesium analysis the authors concluded that one nanogram of asbestos was roughly equivalent to five fibers greater than 5 μm in length by optical microscopy although much variability about this value was observed. By using fiber size data determined by electron microscopy to calculate the mass of a typical fiber, the authors concluded that one nanogram of asbestos corresponded to 8 fibers (all lengths) by optical microscopy.

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In a subsequent paper, Lynch et al (1970) published results of count to weight comparisons for other industrial operations using the sample techniques previously described. These data are summarized in Table 5. Again, large variations in the relationships were observed as evidenced by large geometric standard deviations. From Table 5, it can be seen that one nanogram of asbestos may be roughly equivalent to 6.7 - 46.5 fibers > 5 μm depending on the operation.

In their study of asbestos contamination in commercial building, Nicholson et al (1975a) compared the results of asbestos concentrations (ng/m^3) determined by electron microscopy to fiber concentrations determined by phase contrast microscopy for the same samples. These data were highly variant showing no consistent relationship. One nanogram of asbestos was shown to range from none detected to 6570 asbestos fibers >5 μm by phase contrast microscopy. By averaging data, it was calculated that one nanogram was equivalent to 52 asbestos fibers >5 μm in length.

Air samples collected in communities surrounding the Reserve Mining Company, Silver Bay, Minnesota, have been analyzed by electron microscopy and concentrations expressed in ng/m^3 by mass calculation and $fibers/m^3$ by direct counts (Nicholson 1973). These results showed one nanogram of amphibole fibers to be equivalent to 640 - 108,000 total amphibole fibers by electron microscopy, with an average value of 30,600 fibers/ng.

A study recently published by Dement et al (1975) provides additional data for the conversion of mass concentration to fiber number for amphiboles. In this study, 22 air samples collected in an underground gold mine were analyzed by phase contrast optical microscopy and electron microscopy to determine fiber concentrations. A direct clearing technique which preserved the original fiber size distribution was used to prepare samples for electron microscopy. In addition to fiber counts by electron microscopy,

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each fiber was sized (length and diameter) so that the mass could be calculated (assuming a density of 2.5 gm/cm^3). These data are summarized in Table 6. From these data, approximate relationships between mass concentrations and fiber count concentrations were calculated. One nanogram was calculated to be equivalent to approximately 1,200 total fibers by electron microscopy or 400 fibers >5 μm in length by phase contrast microscopy.

The above studies have not shown a consistent conversion factor for fiber mass to fiber count. Bruckman and Rubino (1975) have suggested a conversion ratio of 20 asbestos fibers >5 μm in length, as determined by optical microscopy, per nanogram of asbestos. Based on the above review, the validity of such a general conversion may be seriously questioned.

NONOCCUPATIONAL EXPOSURES - AMBIENT LEVELS

Asbestos air pollution in urban areas has been studied. Levels of chrysotile asbestos at various locations in New York City, Philadelphia, Ridgewood, N.J. and Port Allegany, Pa. have been studied using electron microscopy (Selikoff et al 1972). Sample sites were chosen which were distant from any known significant source of asbestos. Study results which are summarized in Table 7, show concentrations ranging from 11 to 100 nanograms per cubic meter of air (ng/m^3). These authors point out that one nanogram of asbestos could represent a million chrysotile fibrils.

Ambient samples have been collected in the cities of Reading and Rochdale, England, Bochum and Dusseldorf, Germany, Prague and Pilsen, Czechoslovakia, Johannesburg, South Africa, and Reykjavik, Iceland (Holt and Young 1973). Although no effort was made to quantitate levels, electron microscopy studies revealed the presence of chrysotile asbestos in most samples.

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Results of electron microscopy studies of ambient samples in the United Kingdom are summarized in Table 8. Chrysotile concentrations of 1 to 10 ng/m^3 were observed (Richards 1973).

Asbestos levels in major U.S. cities during 1969-1970 have been determined under contract with the U.S. Environmental Protection Agency (Nicholson 1971). Samples were collected on three or four different occasions for each city and analyzed by electron microscopy. Results are summarized in Table 9 and show that mean concentrations for the samples range from 0.7 to 24.3 ng/m^3; however, 48% of the cities had average concentrations less than 2.0 ng/m^3. The highest mean, 24.3 ng/m^3, was observed in Dayton, Ohio, where numerous plants processing asbestos are located. The highest concentration of 95 ng/m^3 was also observed in Dayton.

Results of chrysotile measurements within buildings insulated with asbestos and ambient levels in the vicinity of these buildings have been presented (Nicholson et al 1975). Chrysotile concentrations were determined using electron microscopy techniques as in previous studies (Selikoff et al. 1972). Ambient levels were found to range from 0 to 46 ng/m^3. Using phase contrast optical microscopy, fiber levels (ambient and indoor) were found to range from 0.000 to 0.027 fibers > 5 μm/cc, with an average of 0.006 fibers/cc. Average concentrations within the buildings sampled ranged from 2.5 to 200 ng/m^3, indicating the possibility of fiber erosion from insulated air plenums. The same report indicates that asbestos concentrations in excess of 100 ng/m^3 may often be found in the homes of asbestos workers, with the highest measured concentration being 5,000 ng/m^3. These authors suggest that exposure in excess of 100 ng/m^3 may be associated with an observable risk of asbestos disease.

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Nicholson et al (1975a) published data indicating that 35 rooms in 17 office buildings in Boston, New York, Chicago and San Francisco-Berkeley had a mean concentration of asbestos fibers in their airs of 11,600/cu m whereas the intake airs for 15 of these buildings (all for which such data was given) contained a mean of 6000 fibers/cu m. One room had a concentration of 102,800 fibers/cu m, all the other having fiber counts below 60,000 per cu m. Samples of air from plenums in 11 of these buildings contained a mean concentration of 5100 fibers/cu m. In an earlier report (1975b), the same investigators stated that two buildings in New York in which no asbestos was known to have been used as a fireproofing or anechoic material had a mean concentration of asbestos within their circulating airs considerably above that of the intake airs for these buildings. These findings indicate that, although pick-up of asbestos from linings applied to air-ducts and plenums may be a factor in the distribution of these fibers within buildings, these linings are not a major source of the asbestos fibers found in the air circulating within buildings.

A survey carried out in the U.K. (Wagg, quoted by Meyer, 1976) has shown that 82% of 73 buildings examined had airborne concentrations of asbestos fibers of up to 20,000/cu m. Only 4% had concentrations of asbestos in the range 50,000-80,000 fibers/cu m. No higher concentrations were reported. The higher concentrations were found in office buildings, residences and miscellaneous types of buildings. Really high concentrations of asbestos in air (of the order of 1-1000 ng/cu m) have been found only within a few hundred meters downwind of asbestos processing plants (Richards and Badami 1971, 1973; Simecek, 1967, Meyer, 1976).

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Asbestos fiber levels in communities surrounding the Reserve Mining Companys' milling operations in Silver Bay, Minnesota, have been reported by numerous investigators. A recent preliminary report of air sampling results has been reported for ten stations located between the Reserve Mining Companys' pollution source and several population centers (Fairless 1974). Samples were collected each sixth day, beginning on November 6, 1974, (for a one year period). These samples were submitted blind to one or more of three laboratories where asbestos fiber concentrations were determined by electron microscopy. Results of these preliminary analyses are summarized in Table 10. Mean concentrations of amphibole fibers ranged from 2.6 to 8.9 x 10^3 fibers/m^3. In addition to amphibole fibers, chrysotile concentrations for individual samples ranged from none detected to 10.4 x 10^4 fibers/m^3. Analyses of all samples collected have not been completed.

Concentrations of amphibole fibers have also been reported near specific point emission sources of the Reserve Mining Company (Nicholson et al 1974). Concentrations as high as 11 x 10^6 fibers per cubic meter of air were reported.

The National Institute for Occupational Safety and Health has performed two studies of fiber concentrations in the air of public buildings using the phase contrast microscopy counting technique (Wallingford et al 1973; Zumwalde 1973). Samples were collected over 6-8 hours at 7 - 10.5 liters per minute. These data are summarized in Table 11. Mean concentrations of 0.004 and 0.001 fibers >5 μm were observed, with the highest single concentration observed being 0.008 fibers >5 μm/cc.

In summary, ambient asbestos levels as determined by electron microscopy techniques are generally less than 10 ng/m^3 with occasional peaks as high as 100 ng/m^3. Only a few studies of ambient levels have been performed using

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phase contrast optical microscopy. These studies indicate ambient levels to be generally less than 0.01 fibers >5 µm/cc, with some peak values as high as 0.03 fibers >5 µm/cc.

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Table 1

TYPICAL OPTICAL DATA FOR ASBESTOS MINERALS

Asbestos Type	Crystal System	Refractive Indices	Extinction Angles	Sign of Elongation
Chrysotile	monoclinic	1.49-1.57	$\gamma \wedge L^* = 0^{\circ}$	+
Anthophyllite	orthorhombic	1.60-1.66	$\gamma \wedge L = 0^{\circ}$	+
Amosite	monoclinic	1.66-1.70	$\gamma \wedge L = 14\text{-}21^{\circ}$	+
Crocidolite	monoclinic	1.69-1.71	$\gamma \wedge L = 3\text{-}15^{\circ}$	-
Tremolite**	monoclinic	1.60-1.65	$\gamma \wedge L = 10\text{-}21^{\circ}$	+
Actinolite**	monoclinic	1.62-1.68	$\gamma \wedge L = 10\text{-}15^{\circ}$	+

*L = long direction of fibers
** Tremolite and actinolite form a continuous mineralogical series.
Values shown are for end members.

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Table 2

DISPERSION STAINING COLORS FOR ASBESTOS MINERALS
USING PLANE POLARIZED LIGHT

Asbestos Type	Refractive Index Liquid	Dispersion Staining Colors	
Chrysotile	1.560	light blue	magenta
Anthophyllite	1.610	blue-green	golden yellow
Amosite	1.670	red magenta	golden yellow
Crocidolite	1.700	magenta	blue magenta

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Table 3

THEORETICAL MINIMUM DETECTABLE FIBER CONCENTRATIONS BY PHASE CONTRAST OPTICAL MICROSCOPY

Sampling Period (Minutes)	Minimum Detectable Conc. fibers > 5 µm/cc
15	0.04
30	0.02
60	0.01
90	0.007
120	0.005
240	0.003
480	0.001

* Based on a sample flow rate of 2.01 lpm and a microscope counting field area of 0.0071 mm^2.

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Table 4

ASBESTOS COUNT/WEIGHT RELATIONSHIPS

DETERMINED BY LYNCH AND AYER (1966)

FOR ASBESTOS TEXTILE PLANTS

Type Count By Phase Contrast Microscopy	Fibers per Nanogram of Asbestos
Total Fibers	11
> 5 μm Fibers	5

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Table 5

ASBESTOS COUNT/WEIGHT RELATIONSHIPS

DETERMINED BY LYNCH AND AYER (1966) FOR

VARIOUS INDUSTRIAL OPERATIONS

Product	Type Fiber Count	Geometric Mean fibers/ng	Geometric Standard Deviation
Textile			
	Total	14.5	2.5
	> 5 µm	6.7	3.3
Friction			
	Total	26.3	3.4
	> 5 µm	13.9	3.6
Pipe			
	Total	46.5	2.8
	> 5 µm	22.5	2.9

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Table 6

SUMMARY OF FIBER COUNT/MASS

RELATIONSHIPS CALCULATED FROM DATA

BY DEMENT ET AL (1975)

Analysis Method	Average Conc. (range)	Units of Measure
Total Fibers by Electron Microscopy	4.82 (0.66 - 11.79)	fibers/cc
Asbestos Mass by Electron Microscopy	3,900 (540 - 9600)	ng/m^3
Fibers >5 μm by Optical Microscopy	1.51 (0.16 - 2.8)	fibers/cc

Approximate Relationships:

1 ng ≅ 1,200 total fibers by electron microscopy

1 ng ≅ 400 fibers >5 μm in length by phase contrast microscopy

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Table 7

SUMMARY OF AMBIENT ASBESTOS LEVELS IN VARIOUS CITIES AS DETERMINED BY SELIKOFF ET AL. (1972)

Sample Site	Asbestos Conc. 10^{-9} gm/m^3
New York City	
Manhattan	25-60
Bronx	25-28
Brooklyn	19-22
Queens	18-29
Staten Island	11-21
Philadelphia, Pa.	45-100
Ridgewood, N.J.	20
Port Allegany, Pa.	10-30

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Table 8

SUMMARY OF AMBIENT CHRYSOTILE LEVELS

IN THE UNITED KINGDOM AS DETERMINED

BY RICHARDS (1973)

Sample Site	Chrysotile Conc. 10^{-9} gm/m^3
Rochdale (Factory Grounds)	1-10
Rochdale (Town Center)	10
Lancashire/Yorkshire	1-10
Industrial Site (Oldbury)	10

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Table 9

SUMMARY OF AMBIENT ASBESTOS LEVELS

IN 49 CITIES FOR 1969-70 AS DETERMINED BY

NICHOLSON ET AL. (1971)

Conc. 10^{-9} gm/m^3	Cumulative % of City Mean Conc. ≤ Given Conc.
0.1-1.9	12
1.0-1.9	48
2.0-2.9	64
3.0-3.9	72
4.0-4.9	86
5.0-5.9	94
> 6.0	6%

* Highest Mean - 24.3 ng/m^3 observed in Dayton, Ohio

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Table 10

SUMMARY OF AMPHIBOLE FIBER CONCENTRATIONS

FOR TEN SAMPLE SITES IN THE VICINITY OF RESERVE MINING

AS DETERMINED BY FAIRLESS (1974)

Sample Site	Amphibole Conc. 10^3 fibers/m^3	
	Mean	Range
Duluth	7.5	0-17
Duluth (Residence)	2.6	0- 8
Silver Bay (Residence)	11	0-30
Babbit (Residence)	13	0-82
Hoyt Lake	8.5	0-31
Hibbing	5.6	0-19
Cloquet	6.8	0-30
Pengilly	6.6	0-17
Virginia	4.2	0-12
Mt. Iron	8.9	0-45

Overall Mean = 7.6 X 10^3 fibers/m^3

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Table 11

SUMMARY OF FIBER CONCENTRATION DETERMINATIONS
IN THE AIR OF PUBLIC BUILDINGS USING PHASE
CONTRAST OPTICAL MICROSCOPY AS DETERMINED BY WALLINGFORD ET AL ,
(1973) AND ZUMWALDE (1973)

Building Location	fibers >5 µm in length/cc Mean and Range ( )
Baltimore, Maryland and Washington, D.C.	0.004 (0.001-0.008)
Towson, Maryland	0.001 (0.000-0.003)

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V. BASIS FOR A STANDARD

The first modern approach to the setting of an asbestos standard was proposed by the British Occupational Hygiene Society (BOHS 1968) in terms of fiber concentration. In 1968, a subcommittee of the Society evaluated data on 290 men at work in an asbestos factory. This data was provided by company sources. All the men had been employed after January, 1933, following implementation of dust control measures mandated by the Factory Inspectorate in 1931. Estimates were also provided by the company of the fiber exposure of these workmen. Of the 290 individuals, 8 were stated to have x-ray evidence of asbestos disease and 16 to have rales. Noteworthy in the 1968 data was the preponderance of individuals who had been employed less than 20 years. Only 118 of 290 had worked for longer than 20 years and a scant 13 had been employed for 30 of more years.

After a review of these data, the BOHS proposed a standard which was adopted with minor modifications by the British government in 1969, and implemented in May, 1970. All fibers between 5 and 100 microns in length were counted by light microscopy. The standard required no action to be taken below 2 fibers/cc. Between 2 fibers/cc and 12 fiberc/cc, control measures commensurate with the exposure circumstances (time and frequency of worker exposure) were prescribed; above 12 fibers/cc, full application of control measures, including respiratory protection, was mandatory. The BOHS predicted that the risk of being affected, to the extent of having the earliest clinical signs of asbestos exposure (rales), would be less than 1% for an accumulated exposure of 100 fiber-years/cc (2 fibers/cc for 50 years, 4 fibers/cc for 25, etc.). Data

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(Lewinsohn 1972) from the same factory which formed the basis for the BOHS Standard demonstrate that a greater prevalence of x-ray abnormalities now exist (Table 1). These data in addition to demonstrating a dose-response relationship for radiographic abnormalities consistent with asbestosis, further showed a 17% prevalence of abnormal radiographic findings (6% consistent with asbestosis) among individuals employed since 1950.

Weill et al (1975) when considering lung function and irregular small opacities, reported that there was little evidence of a dose-response relationship below 100 mppcf-years. They further concluded that a concentration of 5 fibers/cc could be cautiously considered as "safe". Ayer and Berg (1976), however, report data which suggest that the BOHS standard, of an average cumulative exposure of 100 fiber-years/cc, for chrysotile asbestos may prevent significant decreases in pulmonary function only when combined with periodic spirometry and further reduction of exposure for affected workers. Holmes (1973) has since stated that the data upon which the BOHS standard was based were inadequate for the purpose of setting a standard to prevent asbestosis. The BOHS-recommended standard of 2 fibers/cc was based on data related only to asbestosis and the Society clearly cautioned that, since a quantitative relationship between asbestos exposure and cancer risk was not known, it was not possible at that time to specify an air concentration which was known to be free of increased cancer risk. (BOHS 1968)

Howard et al (1976), in a follow-up of the textile workers previously studied by Doll, (1955) and Knox et al (1965, 1968) for cancer, and by Lewinsohn (1972) for asbestosis, reported a statistically

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significant increase in the risk of developing lung cancer (1.8 times the expected) among those first entering scheduled areas from 1933 to 1950. In the same study, they also reported an excess of deaths due to lung cancer (1.9 times the expected) after 15 or more years from initial exposure among those who started work subsequent to 1950, a period of improved industrial engineering control technology and regulation.

In a study of miners exposed to amphibole fibers (amosite) in the cummingtonite-grunerite ore series, with airborne concentrations of less than 2.0 fibers/cc (average concentration, 0.25 fibers/cc) and 94% of the fibers shorter than 5 μm in length, Gillam et al (1976) have demonstrated three-fold increases in the risks of mortality from both malignant and nonmalignant respiratory diseases.

Newhouse (1969, 1973) and Newhouse et al (1972) have shown that the cancer risk following mixed exposure of factory workers to chrysotile, amosite, and crocidolite is dose-related. Those women reported to have heavier exposures (as judged by their occupations) showed a six-fold excess of cancer following only 15 years' latency, whereas those with moderate or low exposures required 25 years' latency to demonstrate an excess. The rate of mesothelioma increased with both the severity and the length of exposure. However, even with as little as two years of asbestos exposure, six mesotheliomas occurred among female employees.

McDonald (1973) states that the risk of developing lung cancer was essentially confined to persons with a dust index above 200 mppcf-years and Enterline et al (1973) showed no direct dose response for respiratory cancer below 125 mppcf-years. In a review of these two papers, Schneiderman (1974) concluded that, instead of being consistent with a threshold level at which

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no cancer risk exists, these data did not provide evidence for a threshold or for a "safe" level of exposure. He pointed out that in the paper by Enterline et al. (1973) there is no dose group for which the SMR is below 100 (100 = normal), but that the 95% confidence limits on the SMR's included 100 for two of the three dose groups below 125 mppcf-years. One of the dose groups (25-62.4) had a statistically significant excess mortality from lung cancer, whereas for the other two this mortality rate was insignificantly elevated above the expected values. Regarding McDonald's paper, Schneiderman stated that it is hard to determine what is excess since no expected numbers for each group are given upon which to base this comparison.

Among amosite workers with 3 months or less employment, Selikoff (1976) reported excess cancer risks of 3.87, 1.68, and 1.65 times those expected for cancer of the lung, colon and rectum, and all sites, respectively.

Anderson et al (1976) have reported a significant excess of radiographic abnormalities of the chest characteristic of asbestos exposure (pleural and/or parenchymal) 25 - 30 years following the onset of household contamination. These abnormalities were observed in 35% of 326 otherwise healthy household contacts of amosite asbestos workers. In addition, four pleural mesotheliomas were found in this group.

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VI. RECOMMENDED STANDARD

Available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have been demonstrated to follow exposure to chrysotile, crocidolite, amosite, and anthophyllite. Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have also been demonstrated to occur in individuals living in the neighborhood of asbestos factories; in persons living with asbestos workers; and in persons living near crocidolite deposits. Asbestosis has been identified among persons living near anthophyllite deposits.

Likewise, all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas following intrapleural or intraperitoneal injection. Mesotheliomas and lung cancers were induced following even one day's exposure by inhalation.

The size and shape of the fibers are important factors; fibers less than 0.5 μm in diameter are most active in producing tumors. Other fibers, of a similar size, including glass fibers, can also produce mesotheliomas following intrapleural or intraperitoneal injection.

There are data that show that the lower the exposure, the lower the risk developing cancer. Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure. Based upon the available data, no assessment can presently be made concerning the existence of a level of asbestos exposure

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below which an increased risk of cancer could not be detected.

In view of the above, the standard should be set at the lowest level detectable by available analytical techniques, an approach consistent with NIOSH's most recent recommendations for other carcinogens (i.e. arsenic and vinyl chloride). Such a standard should also prevent the development of asbestosis.

Since phase contrast microscopy is the only generally available and practical analytical technique at the present time, this level is defined to be 100,000 fibers >5 μm in length per m^3 (0.1 fibers/cc), on an 8-hour-time-weighted average basis with peak concentrations not exceeding 500,000 fibers >5 μm in length per m^3 (0.5 fibers/cc) based on a 15-minute sample period. Sampling and analytical techniques should be performed as specified by NIOSH publication USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers - T.R. 84 (1976).

This recommended standard of 100,000 fibers >5 μm in length per m^3 is intended to (1) protect against the non-carcinogenic effects of asbestos, (2) materially reduce the risk of asbestos-induced cancer (only a ban can assure protection against carcinogenic effect of asbestos) and (3) be measured by techniques that are valid, reproducible, and available to industry and official agencies.

This recommended standard poses some difficulties in that specific work practices and innovative engineering control or process changes are needed. However, because of the well documented human carcinogenicity from all forms of asbestos, these difficulties should not be cited as cause for permitting continued exposure to asbestos at concentrations above 100,000 fibers >5 μm in length per m^3.

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This standard was not designed for the population-at-large, and any extrapolation beyond general occupational exposures is not warranted. The standard was designed only for the processing, manufacturing, and use of asbestos and asbestos-containing products as applicable under the Occupational Safety and Health Act of 1970.

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TABLE 1

B.O.H.S. ASBESTOS STANDARD
X-RAY FINDINGS IN AN ASBESTOS TEXTILE FACTORY
DECEMBER 1970 (MALES)

		X-RAY FINDINGS			
YEARS OF EXPOSURE	NO.	NORMAL	PLEURAL FIBROSIS*	PULMONARY FIBROSIS*	TOTAL ABNORMAL**
0 - 9	613	548	10	0	65(11%)
10 - 19	189	122	18	20	67(36%)
20 - 29	114	51	30	21	63(55%)
30 - 39	42	9	17	17	33(78%)
40 - 49	12	2	6	3	10(83%)

*Consistent with asbestos exposure

**Including changes not considered due to asbestos exposure

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