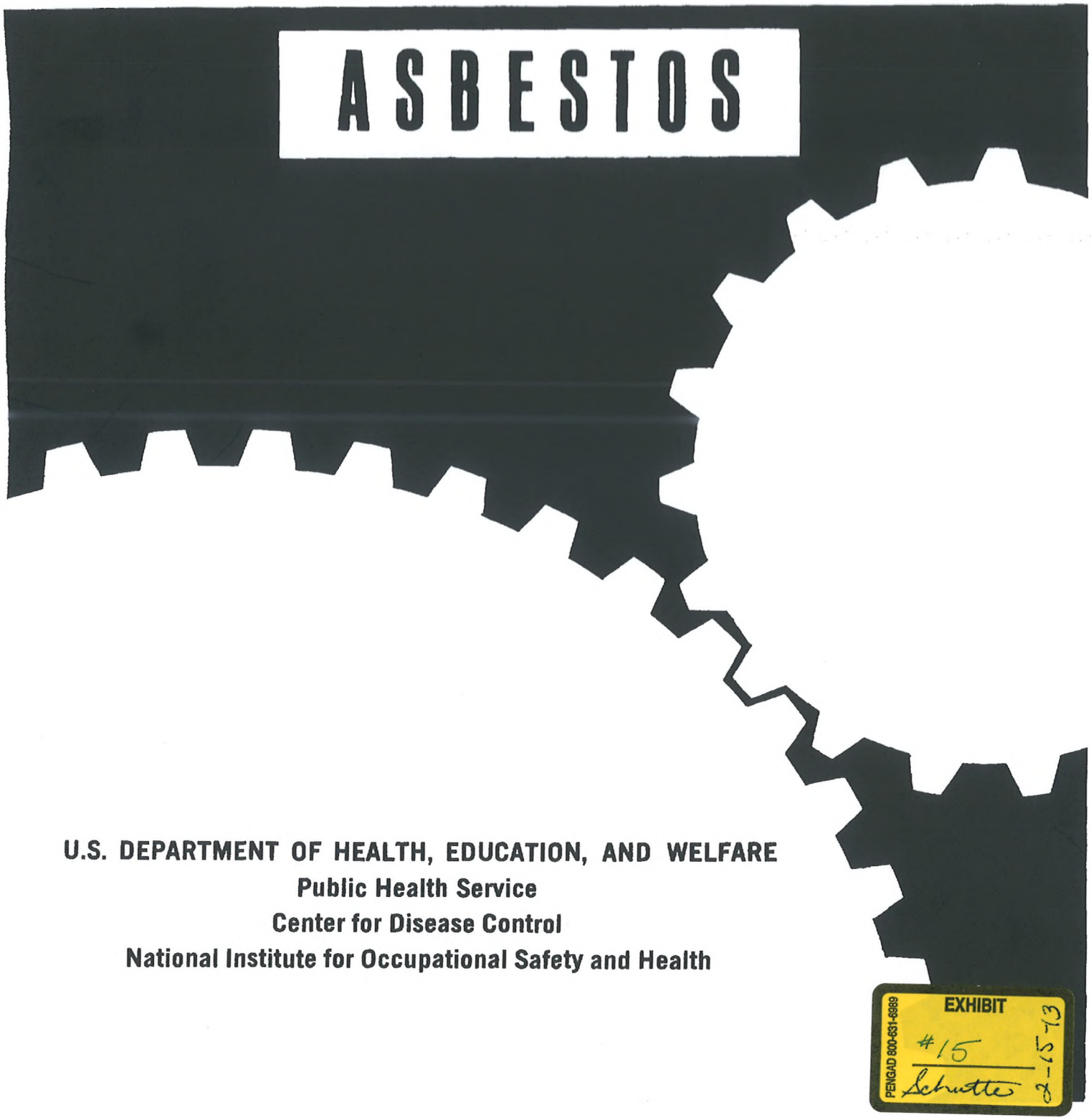
criteria for a recommended standard . . . .

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

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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

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PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health of workers exposed to an ever increasing number of potential hazards at their workplace. To provide relevant data from which valid criteria and effective standards can be deduced, the National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytic methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this first report on asbestos by members of my staff, and the valuable constructive comments by the Review Consultants on Asbestos. A list of these contributors and reviewers appears on pages iii and iv. The contributions of others are also acknowledged:

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR ASBESTOS

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I. RECOMMENDATIONS FOR AN ASBESTOS STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to asbestos dust in the workplace be controlled by requiring compliance with the following sections. Control of worker exposure to the limits stated will prevent asbestosis and more adequately guard against asbestos-induced neoplasms. The standard is amenable to techniques that are valid, reproducible, and available to industry and governmental agencies. It will be subject to review and will be revised as necessary.

Section 1 - Environmental (work place air)

(a) Concentration

Occupational exposure to airborne asbestos dust shall be controlled so that no worker shall be exposed to more than 2.0 asbestos fibers per cubic centimeter (cc) of air based on a count of fibers greater than 5 micrometers (>5 μm) in length ((determined by the membrane filter method at 400-450X magnification (4 millimeter objective) phase contrast illumination, as described in Appendix I)), determined as a time-weighted average (TWA) exposure for an 8-hour work day, and no peak concentration of asbestos to which workers are exposed shall exceed 10.0 fibers/cc >5 μm as determined by a minimum sampling time of fifteen minutes.

(b) Sampling

Procedures for sampling, calibration of equipment, and analysis of asbestos samples shall be as provided in Appendix I.

(c) It is recommended that this Section I become effective two years after promulgation as a standard, and that until the date of

publication, the present emergency standard for exposure to asbestos dust (29 CFR 1910.93a) shall be in effect. This period is believed necessary to permit installation of necessary engineering controls.

Section 2 - Medical

Medical surveillance is required, except where a variance from the medical requirements of this proposed standard have been granted, for all workers who are exposed to asbestos as part of their work environment. For purposes of this requirement the term "exposed to asbestos" will be interpreted as referring to time-weighted average exposures above 1 fiber/cc or peak exposures above 5 fibers/cc. The major objective of such surveillance will be to ensure proper medical management of individuals who show evidence of reaction to past dust exposures, either due to excessive exposures or unusual susceptibility. Medical management may range from recommendations as to job placement, improved work practices, cessation of smoking, to specific therapy for asbestos-related disease or its complications. Medical surveillance cannot be a guide to adequacy of current controls when environmental data and medical examinations only cover recent work experience because of the prolonged latent period required for the development of asbestosis and neoplasms.

Required components of a medical surveillance program include periodic measurements of pulmonary function (forced vital capacity (FVC)), and forced expiratory volume for one second (FEV_1), and periodic chest roentgenograms (postero-anterior 14 x 17 inches). Additional medical requirement components include a history to describe smoking habits and details on past exposures to asbestos and other dusts and to determine presence or absence of pulmonary, cardiovascular, and gastrointestinal symptoms, and a physical examination, with special attention to pulmonary rales, clubbing of fingers, and other signs related to cardiopulmonary systems.

Chest roentgenograms and pulmonary function tests will be performed at the employer's expense, at least every 2 years on all employees exposed to asbestos. These tests will be made annually to individuals, (1) who have a history of 10 or more years of employment involving exposure to asbestos or, (2) who show roentgenographic findings (such as small opacities, pleural plaques, pleural thickening, pleural calcification) which suggest or indicate pneumoconiosis or other reactions to asbestos, or (3) who have changes in pulmonary function which indicate restrictive or obstructive lung disease.

Preplacement medical examinations and medical examinations on the termination of employment of asbestos exposed workers are also required.

Section 3 - Labeling

(a) A warning label for asbestos as shown in Figure 1 shall be used.

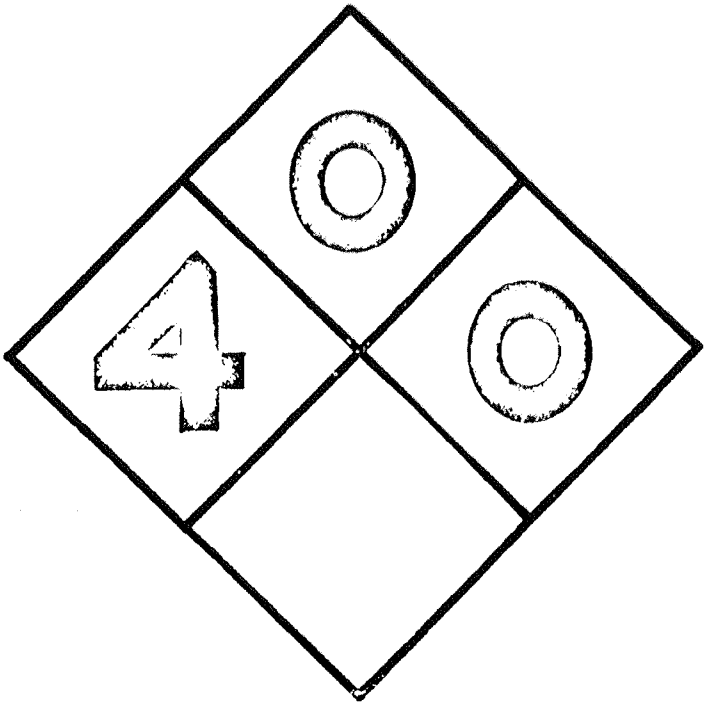
(b) Numerical designations indicate the following:

(i) 4= Health Hazard (color code, blue). Inhalation may cause asbestosis, pleural or peritoneal mesothelioma, or lung cancer.

(ii) 0= Fire Hazard (color code, red). Asbestos is non-flammable and has negligible vapor pressure, volatility, flash point, and explosive limits.

(c) The details of the numerical hazard rating system are found in Appendix II.

ASBESTOS



HARMFUL: May Cause Delayed Lung Injury

(Asbestosis, Lung Cancer).

DO NOT BREATHE DUST

Use only with adequate ventilation and
approved respiratory protective devices.

Section 4 - Personal Protective Equipment and Clothing

This section shall apply whenever a variance from the standard set in Section I is granted under provisions of the Occupational Safety and Health Act.* Use of respirators can be decided on the basis of time-weighted average or peak concentration. When the limits of exposure to asbestos dust prescribed in paragraph (a) of Section 1 cannot be met by limiting the concentration of asbestos dust in the work environment, an employer must utilize as provided in subsections (a) and (b) of this Section a program of respiratory protection and furnishing of protective clothing to effect the required protection of every worker exposed.

(a) Respiratory Protection

(i) For the purpose of determining the class of respirator to be used, the employer shall measure the atmospheric concentration of airborne asbestos in the workplace when the initial application for variance is made and thereafter whenever process, worksite, climate or control changes occur which are likely to affect the asbestos concentration. The employer shall test for respirator fit and/or make asbestos measurements within the respiratory inlet covering to insure that no worker is being exposed to asbestos in excess of the standard either because of improper respirator selection or fit.

(ii) As noted above, the use of respirators and protective clothing can be decided on the basis of either time-weighted average or peak concentrations. For determining usage or compliance, the peak concentration of 10 fibers/cc is preferable.

*Variance procedures will not be required for emergency and occasional short-term exposures in excess of the environmental standard. However, the use of respirator equipment as indicated in this Section (4) will be required under conditions in excess of the standard.

(iii) For an atmosphere containing not more than 10 fibers/cc greater than 5 μm in length over an 8-hour average or more than 50 fibers/cc over any 15 minute period, a reusable or single use filter-type air-purifying respirator, operating with a negative pressure during the inhalation phase of breathing, approved under the provisions of 30 CFR 14 (Bureau of Mines Schedule 21B) or valveless respirators providing equivalent protection shall be used.

(iv) For an atmosphere containing not more than 100 fibers/cc greater than 5 μm in length over an 8-hour average or more than 500 fibers/cc over any 15 minute period, a powered air-purifying positive-pressure respirator approved under the provisions of 30 CFR 14 (Bureau of Mines Schedule 21B) shall be used.

(v) For an atmosphere containing more than 100 fibers/cc greater than 5 μm in length over an 8-hour average or over 500 fibers/cc for any period in excess of 15 minutes, a type C positive-pressure supplied air respirator approved under the provisions of 30 CFR 12 (Bureau of Mines Schedule 19B) shall be used.

(vi) The employer shall establish a respirator program in accordance with the requirements of the American National Standard for Respiratory Protection Z88.2--1969.

(b) Protective Clothing

(i) The employer shall provide each employee subject to exposure in a variance area with coveralls or similar full body protective clothing and hat, which shall be worn during the working hours in areas where there is exposure to asbestos dust.

(ii) The employer shall provide for maintenance and laundering of the soiled protective clothing, which shall be stored, transported and disposed of in sealed non-reusable containers marked "Asbestos-Contaminated Clothing" in easy-to-read letters.

(iii) Protective clothing shall be vacuumed before removal. Clothes shall not be cleaned by blowing dust from the clothing or shaking.

(iv) If laundering is to be done by a private contractor, the employer shall inform the contractor of the potentially harmful effects of exposure to asbestos dust and of safe practices required in the laundering of the asbestos-soiled work clothes.

(v) Resin-impregnated paper or similar protective clothing can be substituted for fabric type of clothing.

(vi) It is recommended that in highly contaminated operations (such as insulation and textiles) provisions be made for separate change rooms.

Section 5 - Apprisal of Employees of Hazards from Asbestos

Each employee exposed to asbestos shall be apprised of all hazards, relevant symptoms, and proper conditions and precautions concerning use or exposure. Each exposed worker shall be informed of the information which is applicable to a specific product or material containing 5% or more asbestos (see Appendix III for details of information required). The information shall be kept on file and readily accessible to the worker at all places of employment where asbestos materials are manufactured or used in unit processes and operations. It is recommended, but not required, that this information be provided for asbestos processes and operations where the asbestos content is less than 5%.

Information as specified in Appendix III shall be recorded on U. S. Department of Labor Form OSHA-20, "Material Safety Data Sheet", (see page X-3 and X-4), or a similar form approved by the Occupational Safety and Health Administration, U. S. Department of Labor.

Section 6 - Work Practices

(a) Asbestos cement, mortar, coatings, grout, and plaster shall be mixed in closed bags or other containers.

(b) Asbestos waste and scrap shall be collected and disposed of in sealed bags or other containers.

(c) All cleanup of asbestos dust shall be performed by vacuum cleaners or wet cleaning methods. No dry sweeping shall be performed.

Section 7 - Monitoring and Recordkeeping Requirements

Employers will be required* to maintain records of environmental exposure to asbestos based upon the following environmental sampling and recordkeeping schedule. Personal exposure samples will be collected at least annually by specific maximum-risk work operations from a number of employees. The first sampling period will be completed within 180 days of the date of this standard. These selected samples will be collected and evaluated as both time-weighted and peak concentration values. The personal sampling regime shall be on a quarterly basis for maximum-risk work areas under the following conditions:

(a) The environmental levels are in excess of the standard.

(b) There are other conditions existing that necessitate the requesting of a variance from the Department of Labor.

Records of the type of respiratory protection in use during the quarterly sampling schedule must also be maintained. Quarterly sampling, monitoring and recordkeeping will be required only until environmental levels comply with the standard.

*Except where a variance for monitoring and recordkeeping has been granted.

II. INTRODUCTION

This report presents the criteria and the standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to asbestos dust. The necessary relevant data are made available for use by the Secretary, Department of Health, Education, and Welfare in accordance with the provision of the Occupational Safety and Health Act of 1970 requiring the development of criteria by "The Secretary, Department of Health, Education, and Welfare...on the basis of such research, demonstrations, and experiments and any other information available to him...to effectuate the purposes of this Act."..., "..: by providing medical criteria which will assure insofar as practicable that no employee will suffer diminished health, functional capacity, or life expectancy as a result of his work experience"...

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultations with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any recommended criteria for a standard should enable management and labor to develop better engineering controls and more healthful work practices and should not be used as a final goal.

These criteria for a standard for asbestos dust are the first of the criteria developed by NIOSH. The criteria and standard speak only to the processing, manufacture, and use of asbestos products as applicable under the Occupational Safety and Health Act of 1970.

The occupational safety and health aspects of the mining and milling of asbestos ores are covered by provisions of the Federal Metal and Nonmetallic Mine Safety Act (30 US.C. 725 et seq.) under which provisions the Bureau of Mines has promulgated applicable regulations. Relevant data, however, bearing on the safety and health hazards from exposure to asbestos dust in the mining and milling of ores were considered in this document.

These criteria were developed to assure that the standard based thereon would, (1) protect against asbestosis and asbestos-induced neoplasms, (2) be amenable to techniques that are valid, reproducible, and available to industry and official agencies, and (3) be attainable with existing technology.

The recommended standard is designed primarily to prevent asbestosis. For other diseases associated with asbestos, there is insufficient information to establish a standard to prevent such diseases including asbestos-induced neoplasms by any all-inclusive limit other than one of zero. Nevertheless, a safety factor has been included in arriving at the concentration level that will reduce the total body burden and should more adequately guard against neoplasms.

Asbestos has been mined, milled, processed, and used for many years, and as a result, a number of workers have experienced significant accumulative exposure to asbestos dust over a working lifetime. It has been recognized that biological monitoring (by periodic chest roentgenograms) and removal from further exposure after initiation of fibrosis, calcification or neoplasia will not absolutely prevent

further progression of asbestosis or the clinical development of neoplasms. Therefore, it is absolutely essential that a low level of concentration be set to preclude the initiation of diseases resulting from exposure to asbestos. And of necessity, any prolonged delay in the establishment of the standard may require a more stringent standard in the future to assure the reduced total body burden of employees which is necessary to protect their safety and health.

III. BIOLOGIC EFFECTS OF EXPOSURE TO ASBESTOS

Asbestos is a generic term that applies to a number of naturally occurring, hydrated mineral silicates incombustible in air and separable into filaments. The most widely used in industry in the United States is chrysotile ($3MgO.2SiO_2.2H_2O$), a fibrous form of serpentine. Other types include amosite ($(FeMg)SiO_3$); crocidolite ($NaFe(SiO_3)_2.FeSiO_3.H_2O$); tremolite ($Ca_2Mg_5Si_8O_{22}(OH)_2$); anthophyllite ($(MgFe)_7Si_8O_{22}(OH)_2$); and actinolite ($CaO.3(MgFe)O.4SiO_2$).

Extent of Exposure

Almost one million tons per year of asbestos are used in the United States. In 1965, approximately 74 percent of the asbestos produced was used in the construction industry (532,300 tons) while 26 percent was used in non-construction industries (187,400 tons). Approximately 92 percent of the half million tons used in the construction industry is firmly bonded, i.e., the asbestos is "locked in" in such products as floor tiles, asbestos cements, and roofing felts and shingles; while the remaining 8 percent is friable or in powder form present in insulation materials, asbestos cement powders, and acoustical products.[1] As expected, these latter materials generate more airborne fibers than the firmly bonded products. The 187,400 tons of asbestos used in non-construction industries in 1965 were utilized in such products as textiles, friction material including brake linings, and clutch facings, paper, paints, plastics, roof coatings, floor tiles, and miscellaneous other products.

Mining and milling of asbestos in the United States is a small industry, employing fewer than a thousand workers. The health and safety

aspects of mining and milling operations are not covered under the Occupational Safety and Health Act of 1970.

The construction industry has, in recent years, applied asbestos insulation materials by spraying, a method of application that generates more airborne asbestos fibers than older conventional methods. This technique at present utilizes only a small percentage of the total asbestos produced and its use is decreasing.

There are approximately 40,000 field insulation workers in the United States who are exposed to asbestos dust. The activities of these workers cause secondary exposures to an estimated three to five million other building construction and shipyard workers.[2]

Since the dust exposure to the individual worker is extremely variable and the number of asbestos workers at any one location is small, the primary and secondary asbestos dust exposures to all workers have never been satisfactorily estimated.

An estimated 50,000 workers are involved in the manufacture of asbestos-containing products. This figure does not include secondary manufacture of products which contain asbestos, such as electrical or thermal insulation, or products which include previously manufactured components containing asbestos.[3]

The following information, furnished by the Pennsylvania Division of Occupational Health, shows the number and variety of plants using asbestos in which potential exposures can occur. These figures are based on a survey of a total of 18,439 manufacturing plants in that

State as of August 22, 1969, and represents about 1.4 percent of all manufacturing operations in Pennsylvania. Service facilities such as garages are not included.

	No. of Plants
Insulation, including cutting, drilling, and tape manufacture	75
Manufacturing and processing	16
Brakes and friction	10
Cement, clay	18
Miscellaneous*	146
TOTAL	265

*Gaskets
Signs
Safety equipment
Laminated material
Paint and roofing materials
Shipbuilding and shipbreaking
Impregnating resin and urethane
Textile
Undercoating material
Ironing board covers
Flooring

Early Historical Reports

The widespread use of asbestos fibers did not begin until the last quarter of the nineteenth century.[2] With the increasing use of asbestos materials and increasing reports of asbestos related disease there developed concern over the role of these minerals as factors in human disease. Differentiation of the type of asbestos fiber was not made in most studies related to occupational exposure. In the United States the exposures of greatest concern usually involve more than one type of fiber, although chrysotile predominates. To refine our knowledge of the biological actions of asbestos, it is imperative that the character of the exposure as to concentration, size, and type of fiber be known. At present, data of this complexity are scanty or often non-existent with respect to human exposure.

The first record of a case of asbestosis was reported in England by Montague Murray in 1906.[4] The first complete description of asbestosis and of the "curious bodies" seen in lung tissue appeared in 1927 when Cooke[5] reported on a case of asbestosis and McDonald[6] reported on the same and another case. Each author gave reasons for believing that these "curious bodies" originate from asbestos fibers that reach the lungs.

Many of the people exposed to asbestos dust develop the disease "asbestosis" if the dust concentration is high or the duration of their exposure is long. This has been documented by the following studies: Merewether and Price, 1930; Fulton et al., 1935; and Dreessen et al., 1938. In 1918, Hoffman[7] reported that it was the practice of

American and Canadian insurance companies not to insure asbestos workers due to the assumed health-injurious conditions of that industry. In 1917, Pancoast, Miller and Landis[8] reported on X-ray appearances of pneumoconiosis in 15 individuals exposed to asbestos.

Mills' publication[9] in 1930 was the first report on a case of asbestosis published in the United States, and in that same year, Lynch and Smith[10] reported on "asbestosis" bodies* found in the sputum of asbestos workers. In Merewether's review of asbestosis,[11] emphasis was placed on the relation of asbestosis to dusty working conditions.

The clinical aspects of asbestosis are well documented. Gloyne[12] discussed the pathology of asbestosis and methods for diagnosing asbestos bodies and asbestosis. Selikoff and Hammond[13] analyzed 1,975 autopsies in three large New York City hospitals and found asbestos bodies in 942 (47.7%). Broadly considered, 40 percent of housewives, 50 percent of "white collar" males, and 50 percent of "blue collar" males showed asbestos bodies; but males who had a history of shipyard or construction work had higher incidence of asbestos bodies, i.e., 90 of 129 cases or 70 percent. Selikoff's observations also suggest that asbestos bodies were as frequently present 38 years ago as now.

Although a large percentage of the lungs of adult urban dwellers may be found to contain ferruginous bodies (depending on the method of examination), the significance of this is as yet unknown.

*"Ferruginous bodies" is a more descriptive term. This and other aspects of the biologic effects of asbestos are well documented in the Annals of the New York Academy of Science.[1]

The core fibers have not been systematically identified to indicate how many are asbestos bodies, and there are little data bearing on possible health effects associated with the low concentrations of fibers found in ambient air.

An abnormality, occurring with unusually greater frequency in populations exposed to inhalation of asbestos fiber, is that of localized thickening, or plaques, of the pleura with or without calcification of the plaques. The role of the asbestos fiber in this manifestation is not clear.

The medical aspects of exposure to asbestos and the development of the occupational disease, asbestosis, are characterized by:

(1) A pattern of roentgenographic changes consistent with diffuse interstitial fibrosis of variable degree and, at times, pleural changes of fibrosis and calcification.

(2) Clinical changes including fine rales and finger clubbing. These may be present or absent in any individual case.

(3) Physiological changes consistent with a lung disorder.

(4) A known history of occupational exposure to airborne asbestos dust. In general, a considerable time lapse between inhalation of the dust and appearance of changes as determined by X-ray.

The several clinical abnormalities listed above appear to occur with unusual frequency in those environments where airborne asbestos fibers, often in association with other substances, exist. One of these abnormalities, a diffuse chronic inflammation and scarring

of the lung, is the one recognized early in this century and referred to as "asbestosis."

Epidemiological Studies

Harries[14] in 1968 suggested that first impressions would lead one to believe that only workers continuously exposed to asbestos are at risk of developing asbestosis, however, a number of trades experiencing intense intermittent exposures are also suspect. These other trades involve work with asbestos insulation in confined spaces onboard ship. Work in these trades has been accepted by the Pneumoconiosis Panel of the United Kingdom as associated with asbestosis. Selikoff,[15] however, in a study of 232 former insulation plant employees reported positive X-ray findings among individuals having had known exposures to asbestos as short as one day (Table XXVII).

In the late 1940's a frequency of bronchogenic cancer greater than that expected on the basis of the general male population was manifest among persons who worked in the manufacture of asbestos products.[16] This excess of bronchogenic cancer was also demonstrated among a group of workers in the United States exposed to airborne asbestos fibers in the installation of insulation.[17,18] Among 632 asbestos insulation installers observed from 1943 to 1967 there were 99 excess deaths (above that expected on the basis of the U. S. white male population) for three types of malignancies-- bronchogenic (63), gastrointestinal (26) and all other sites combined (10). Elmes and Simpson[19] recently reported findings of similar magnitude among men employed as insulators and pipe coverers in Belfast. Newhouse[20] found an excess of lung cancer

in a study of over 4,500 male workers employed at an asbestos factory making both textile and insulation materials. This excess of lung cancer was demonstrated among those workers with jobs which entailed heavy exposure irrespective of the duration of employment.

More recent observations by Selikoff in the United States indicate a lung cancer risk for workers exposed to amosite asbestos in the production of insulation material.[21]

The possibility that the carcinogenic role of asbestos is solely that of a cocarcinogen has been suggested by Wright.[2] This suggestion stems from the observation by Selikoff and associates[17] that among 370 asbestos insulators, exposure to asbestos dust does not greatly increase the risk of bronchogenic cancer in the absence of regular cigarette smoking. More recent observations among this same group of workers,[22] however, demonstrate that this interpretation is largely a function of sample size as one lung cancer death vs. 0.02 expected was observed among non-smokers as contrasted with 27 vs. 2.83 expected among cigarette smokers. Moreover, Decoufle[23] demonstrated that the excess of lung cancer mortality among several subgroups of retired asbestos workers could not be explained by cigarette smoking alone.

Concerning mesothelioma, 80 percent of the cases studied in South Africa and the United Kingdom have been shown to have an occupational or para-occupational association with asbestos fibers.[2] In the United States, Selikoff and co-workers have reported the occurrence of 14 deaths from mesotheliomas among 532 asbestos insulation workers studied in retrospect from 1943 to 1968 compared to no deaths which

would be expected in the same number of similar individuals in the general population.[17,18] Information is insufficient at this time to set an exposure standard (other than zero) which would assure prevention of mesothelioma in all workers, as the disease may occur following a very limited exposure 20-30 years earlier.

An increased rate of occurrence of mesothelioma of the pleura or peritoneum was reported in some populations in 1959 and in subsequent years. The possibility that asbestos may play a role in this distribution has been raised. Investigations of the distribution of mesothelioma in populations occupationally exposed to asbestos indicate a strong relationship between exposure to asbestos fiber and the presence of mesothelioma.[18,20,24,25]

Neoplasms, such as mesothelioma, may occur without radiological evidence of asbestosis at exposure levels lower than those required for prevention of radiologically evident asbestosis. This may be of particular importance when consideration is given to short-term, high levels of exposure, and may result in the development of mesothelioma before or after completion of a normal span of work either in or out of the asbestos industry.

This is illustrated by several case studies, including two cases of malignant mesothelioma, one a "family" and the other a "neighborhood" case.[26] In another "family" case, a woman washed the overalls of her three daughters at home; all three daughters worked for an asbestos company with possible heavy exposures to asbestos.

The time lapse between onset of exposure and mesothelioma in 344 deaths among asbestos insulation workers was studied. Mesothelioma developed after a longer lapse of time from onset of exposure to asbestos than was the case in the development of asbestosis (Table XXVIII).[15] Knox[27] reported 4 cases of mesothelioma in men and women with less than 10 years exposure, one with only seven months exposure, with the latent time for the development of the mesothelioma from 23 to 53 years.

D. L. Cran[28] indicated that mesothelioma did occur in cases of asbestosis, but that in most cases of mesothelioma that he had seen, the occurrence of asbestosis was not found. He postulated that the difference being the long periods of exposure required to produce asbestosis, while mesothelioma could occur long after a short intensive exposure. The 27 cases of mesothelioma in children under 19 years of age indicates the latent time period for development of mesothelioma may be shorter than first estimated.[29]

Fifteen cases[30] of pleural mesothelioma associated with occupational exposure were reported in Australia. The relationship between the mesothelioma development and asbestos was based upon occupational histories and finding of asbestos bodies in the tissue. In some of these cases, the relationship to occupational exposure could not be developed with any degree of certainty, but included patients whose exposure was as short as six months. No patient was regarded clinically or radiologically as suffering from asbestosis; one person had pleural plaques that were radiologically visible.

Stumphius,[31] between 1962 and 1968, found 25 cases of mesothelioma on Walcheren Island. Of these cases, 22 had been employed in the shipyard trades. Stumphius noted that the shipyard employed about 3000 men. This would result in a rate of mesothelioma of approximately 100 per 100,000 males per year. He also noted that the rate for Dutch provinces with heavy industry is 1.0 per 100,000 per year.[31] In the same study, examination of sputum from 277 shipyard workers showed that 60% had asbestos bodies. The frequency varied from 39% of those with no obvious exposure to 100% among those with slight but definite asbestos exposure.

McEwen[32] found that the incidence of mesothelioma in Scotland was similar to that found in other parts of the United Kingdom and confirmed the association between the development of the tumor and occupational exposure to asbestos.

In 1968 Stumphius and Meyer[33] concluded that asbestos exposure may lead to asbestosis, to carcinoma of the lungs and digestive tract, and to mesothelioma. They further stated that there may be no indication of definite exposure to asbestos. It must be pointed out that a clear picture of the relationship between the type of asbestos and the production of asbestosis, neoplasms, and mesotheliomas is not defined in the exposures reported. In many cases mixed exposures have occurred; e.g., the cases from the Naval dockyards in Great Britain where exposures have occurred in unknown amounts to crocidolite and amosite.

Animal Toxicity

Experimental Animal Studies. Experimental exposure of animals to asbestos has been in progress for more than 40 years. During this time, a precise experimental animal model, from which could be derived dose-response relationships that could be used in estimating the appropriate value for a work place air standard has not yet been reported.

The rate of development of asbestotic pulmonary fibrosis and of induction of pleural mesotheliomas is so slow that the animals die before onset of the condition. Accordingly, to develop either condition, experimenters have had to use inordinately high exposure levels or abnormal modes of administration or both, thus nullifying the animal model. The classical demonstrations of diffuse pulmonary fibrosis in guinea pigs with accompanying asbestos bodies by Gardner and Cummings[34] and by Vorwald et al.[35] became possible only by using fiber levels of from 1,400 to 5,000/cc (39 million to 138 million fibers/cubic foot); and the uniform production of mesotheliomas in rats by Wagner and Berry[36] was attained only after administering the asbestos by intra-pleural injection at the extraordinarily high dose of 20 mg.

Stanton et al.[37] were unable, even when aided by chemical means, to induce neoplasms of any type in a tumor-susceptible strain of rats at low dosages of asbestos (type unspecified); but Gross et al.[38] did produce in rats malignant pulmonary tumors of several types from exposure at very high doses (ca. 22,000 fibers/cc 86 mg/m^3) of chrysotile asbestos that had been hammermilled to an increase in cobalt of 145%; nickel, 82%; and chromium, 34%.

Differences in animal responses to "harsh" and "soft" chrysotile asbestos were seen by Smith et al.[39]: granulomatous and fibrous pleural adhesions were thicker, and pleural mesotheliomas appeared more rapidly in response to harsh chrysotile. (Harsh chrysotile was characterized as appearing in thicker bundles and was hydrophobic whereas the soft chrysotile was hydrophilic).

There are no experimental animal dose-response data that can be used in estimating a work place air standard for asbestos.

Contributions to Occupational Exposure Standards from Animal Studies. Of possible value in estimating occupational exposure limits are data regarding the relative disease-producing potency of the various forms and types of asbestos.

Wagner[40] found in the three species exposed (guinea pigs, rabbits, and monkeys) that amosite produced more marked interstitial fibrosis than chrysotile and the lesions occurred earlier. No statement on relative potency of crocidolite could be made because of the impure nature of the test specimen. On the other hand, amosite was found by the same investigator[36] to be about one-half as potent in the production of mesotheliomas in rats as chrysotile and crocidolite, if numbers and rate of production are used as indicators. An incidental finding was no evidence for difference in effect between natural and oil-extracted forms of crocidolite, a subject considered as a possible factor in the induction of asbestos cancers.[41]

Naturally Occurring Effects in Lower Animals. No evidence appears to exist that domestic or wild animals can provide criteria for standards,

or for controlling asbestos emissions, although a few confirmatory reports have been made that asbestosis can occur in such animals. Webster[42] has demonstrated fibrosis with associated asbestos bodies and fibers in wild rodents in South Africa, in one of a troop of baboons, and in two donkeys that had either worked in, or lived around, crocidolite mines or mills. And Schuster[43] reported pulmonary asbestosis, without asbestos bodies, in a dog that had lived for about 10 years in a London asbestos factory as a rat catcher. The magnitude or the type of exposure was not reported in any instance.

Factors Influencing Pathogenesis-- Experimental Animal. Experimental animal studies have been informative in elucidating the factors that modify or explain the biologic action of asbestos. At least six factors have been investigated: (1) fiber length and bundle size; (2) cytotoxicity; (3) red cell hemolytic activity; (4) asbestos hydrocarbons; (5) morphologic changes; and (6) trace metals in asbestos.

(1) Fiber length and bundle size. The relation between length of fibers and of fibers to motes (nonfibrous particles) and asbestos induced disease has been one of continuing experimental inquiry. Gardner and Cummings[34] and Gardner[44] found that longer fibers appeared to have a greater fibrogenic effect, although fibrosis developed in animals exposed to dusts which were composed of but one to 1.5 percent fibers. The high exposure concentration of 100 mppcf (ca. 3,600 fibers/cc) makes any decision on the relative potency of fibers vs. motes virtually impossible; however, when animals were exposed to short-fiber asbestos dust, although the type and rate of tissue reaction

were essentially the same, the extent of involvement was very much less than that of longer fibers. Inasmuch as exposure concentrations in these comparable studies were about the same, the conclusion can reasonably be made that longer fibers are more fibrogenic, but that the motes are not without fibrogenic potential.

In experiments with rabbits, King, Clegg, and Rae[45] using Rhodesian chrysotile fibers averaging 2.5 μm and 15 μm in length, concluded that the shorter fibers produced generalized interstitial fibrosis, whereas the longer fibers produced nodular lesions. This finding was not confirmed by one of the investigators (King) in another animal species.[46] Later repetition of the investigations, with "fine" chrysotile and amosite (85% and 82.6% respectively, less than 1 μm in length) by Wagner[40] yielded definite fibrosis with both dusts, thus confirming the original work of Gardner that short fibers or motes have fibrogenic potential.

This experimental work has significance for industrial air standards in indicating the need to support additional research on the "greater than 5 μm in length" specific requirement and the more general relation of fiber length to cancer induction, which has never been determined experimentally.

(2) Cytotoxicity. Both chrysotile and crocidolite were found to be markedly toxic to guinea pig macrophages *in vitro*.[47] The fibrous fraction showed a high, and the particulate, a moderate toxicity, thus providing evidence in conformity with the relative biologic potencies of fibrous and nonfibrous forms found in *in vivo* studies.

(3) Hemolytic Activity. In a similar effort to discover the initial stages of biologic activity of asbestos, and in particular to account for the iron-staining character of asbestos bodies, the hemolytic action of four asbestos types was determined. Whereas chrysotile proved to be potently hemolytic, crocidolite, amosite and anthophyllite were either completely inactive or only weakly.[48] No attempt was made, however, to correlate the greater hemolytic activity of chrysotile with the iron-staining intensity of its asbestos bodies relative to those from other asbestos forms.

(4) Asbestos Hydrocarbons. As chrysotile proved to be most adsorptive of iron, so was it most adsorptive of benzpyrene; compared with 100% adsorption for chrysotile, crocidolite and amosite absorbed from solution 40% and 10% respectively.[49] On this basis, chrysotile should prove the most potent cocarcinogen of the three forms if its action is mediated through exogenous benzpyrene. This has not been demonstrated as yet in humans. A 10% desorption from chrysotile by serum in three days was demonstrated,[49] a condition considered an essential first step in hydrocarbon carcinogenesis.

(5) Morphologic Changes. Electron microscopy of animal tissues has greatly enlarged understanding of the processes that occur following contact of pulmonary cells with asbestos. Examination by light, phase, and electron microscopy by Suzuki and Churg[49] of subcellular tissue of hamsters intratracheally exposed to chrysotile revealed the successive steps that occurred in the cytoplasm of certain pulmonary cells. Particularly informative for the mode of chrysotile action was the description of the formation and the ultrastructure of the asbestos

body, and the indication that instilled fibers tend to split longitudinally with time. The suggestion that chrysotile breaks up into short fragments on the evidence that the majority of the fibers found in the alveoli were less than one-sixth the injected length, one and two years later, is open to the alternative interpretation that, inasmuch as longer particles are more readily phagocytosed, what is actually observed is the residual, smaller, nonphagocytosed chrysotile.[50] Thus, despite the detailed, in-depth information furnished by electron microscopy, no body of knowledge yet exists that permits the assigning of relative risk factors to fibers of differing lengths.

In respect to asbestos bodies, it should be noted that "ferruginous bodies" produced in guinea pigs in response to other fibrous material, fine fibrous glass and ceramic aluminum silicate were identical in fine structure to that of asbestos bodies,[51] thus rendering firm diagnostic decisions difficult in cases of multiexposures to different fibrogenic fibers in the electron and light microscopic range.

(6) Trace Metals. Harington and Roe[41] and later Cralley et al.[52] reported large amounts of nickel, chromium, manganese, and iron are intimately associated with certain forms of chrysotile. On the possibility that trace metals may be associated with the induction of asbestos, cancer studies in animals were performed[53] which supported the hypothesis that, in the induction of asbestos cancers, trace metals play an active cocarcinogenic role along with the exogenously derived carcinogen benzpyrene, while asbestos plays a passive role as a metal carrier.

Correlation of Exposure and Effect

Available information on the relationship of asbestos exposure and the risk of asbestosis and/or bronchogenic carcinoma is somewhat

extensive, indicating a strong association between the diseases and such exposure under a variety of conditions[2,15,22,28] and evidence of dose-response relationship.

Enterline and associates[54] have recently demonstrated convincing evidence for an exposure-response relationship between asbestos as measured in terms of million parts per cubic foot years (mppcfyr), and the risk of malignant and non-malignant respiratory disease. Specifically, the risk of respiratory cancer increases from 166.7 (standardized mortality ratio) at minimal exposures to 555.6, at accumulative exposures in excess of 750 mppcfyr (Table XXX).

Knox et al.[27] suggested that in one asbestos plant where environmental levels varied between 1 and 8 particles/cc $>$ 5 μm in length, the risk to bronchial carcinoma may have been largely eliminated, but that insufficient data were available to estimate the extent of the risk that may remain. The different textile operations were fiberizing, carding, spinning, weaving, and plastering. When environmental samples collected by operation in 1961 and 1966 were summed, the averages were between 4 to 6 fibers/cc. Operational averages were from a low of 2.5 fibers/cc in weaving to a high of 6.5 fibers/cc in carding.

In 1968, Balzer and Cooper[55] reported asbestosis among insulation workers exposed at levels not exceeding the time-weighted average of 5 mppcf.

McDonald et al.[56] reported in May 1971, on 129 primary thoracic neoplasms in the workers employed in Quebec chrysotile asbestos mines and mills out of a total of 9304 former employees; five of these cases were mesothelioma. The authors concluded that the additional data

supports evidence of other studies that even heavy exposure to asbestos in mining and milling carries only modest risk of contracting lung cancer and less still of contracting malignant mesothelioma. McDonald et al. suggest that any increased risk of respiratory cancer or pneumoconiosis at a dust-index below 200 would not be detectable and would still be in doubt below 400. At a dust index of 200 an employee could work for 40 years at a dust concentration of 5 mppcf. The author assumes that the fiber content of the dust is about 10% and he states that this is equivalent to about 12 fibers/cc.

Wright[57] pointed out that others have noted the striking differences in the health experiences of workers in mines and mills as compared to other workers, specifically in comparison to insulation operations, but that he felt the question was still unresolved. In contrast to populations exposed to mixed environments, those engaged in the mining and milling of asbestos fibers showed no augmented frequency of bronchogenic cancer.[2]

Selikoff,[15] however, indicated that McDonald's "heavily exposed" group had 5 times as much lung cancer as the "lightly exposed" workers. Furthermore, lung cancer among insulation workers was found to be about 7 times greater than expected compared to the general non-exposed population.[15] A non-exposed group was not reported by McDonald.[56]

Although it has been suggested that the risks associated with asbestos exposure may be less in mining than in industrial operations, additional study will be necessary to confirm if such is true, based upon the comparison made by Selikoff.[15]

Consideration must be given to McDonald's analysis of levels of exposure of 12 fibers/cc. At this level, he assumes that some degree of asbestosis may occur. The mathematical assumption made to arrive at this environmental level leaves a great deal to question, even without attempting to relate this information to the asbestos industry in general. Two primary considerations lack the evidence necessary to make general comparisons of these data with other reported work: the assumption as stated by McDonald[56] that the fiber content of the dust is 10%, and the method used to convert from mppcf to fibers/cc is not explained in the paper.

Murphy et al.[58] found that asbestosis was 11 times more common among pipe coverers in new ship construction than among a control group. The asbestosis was first found after 13 years of exposure or about 60 mppcf years. The prevalence was 38% after 20 years. The asbestosis was defined by the presence of at least three of the following signs: (1) basular rales in two or more sites, (2) clubbing of the fingers, (3) a vital capacity of less than 80% of the predicted, and (4) roentgenography consistent with moderately advanced, or advanced asbestosis, and (5) dyspnea on climbing one flight of stairs. The environmental level was based upon samples collected in an impinger and all the results were time-weighted average exposures and these were averaged over several different operations. The highest average concentration was with hand-saw cutting at 10.0 mppcf and the lowest average was 0.8 mppcf when mixing mud. The average of all operations was 5.2 mppcf. One-hundred and one workers were

in the exposed group with 94 used as controls matched for age, duration of employment and smoking habits. Both amosite and chrysotile were used in these operations while crocidolite was not. Murphy states that in his study no asbestosis was found for men exposed to 60 mppcf-years while 20% of those exposed for 75 to 100 mppcf-years were considered to have asbestosis. Consideration must be given to averaging the time-weighted average values of the environmental samples over what seem to be several different sampling locations or operations. Were workers who were classified as suffering from asbestosis exposed in the hand-saw cutting, or mixing mud, or both, and for what time interval? Answer to this question would have a major effect upon the relationship between the development of asbestosis and environmental levels, and the relation of these impinger counts to fibers/cc.

In a recent unpublished paper, Williams, Baier, and Thomas compiled data from the Pennsylvania Department of Health files on exposure levels at various textile processing operations in two plants. The data included dust concentrations from 1930 through 1967 in one plant and from 1948 through 1968 in the second plant. Even though controlled exposures were for the most part below 5 mppcf and in many cases below the 1968 ACGIH Notice of Intended Change to 2 mppcf, 64 cases of asbestosis were reported from these two asbestos textile plants. The authors conclude that: "If asbestosis is to be prevented, airborne asbestos dust must be stringently controlled in the working environment. From these data a TLV of 3 mppcf would provide inadequate protection and the proposed 2 mppcf may not be substantiated."

Thus, considerable evidence exists indicating that the prevention or reduction of the occurrence of asbestosis among workers requires that the concentration of asbestos fibers to which they are exposed be reduced.

There is at this time, however, only scant correlation of epidemiological data with environmental exposure data upon which a definitive standard can be established.

Champion[26] reported two cases of malignant mesothelioma in two men, 31 and 32 years old, following exposure to asbestos. In the first case, the only documented exposure of the patient was from his father, who at 68 years of age, had severe asbestosis following employment as a pipe lagger in Scotland. In this case, no special precautions were taken to protect the children from contact with the father's work clothing, which was washed at home. The man smoked about 20 cigarettes per day for sixteen years and had a brief history of breathlessness and other signs which could have been related to asbestos exposure. The second case involved a patient who had moved to Asbestos, Quebec, where he lived for the next 23 years. This patient had worked for 10 years as an asbestos prospector and had worked for a short period in open-pit mining. Seven years before his death in 1968, he moved away from the area and became a salesman in a department store. The patient smoked 20 to 30 cigarettes per day for 14 years. In this case, it was believed that he was exposed only to chrysotile and primarily in mining operations. Champion's two cases seem to support earlier data of family cases[15] with reasonably short and/or low levels of exposure.

Murphy et al.[59] presented data concerning two cases of workers exposed to asbestos. One case on biopsy confirmed mesothelioma and the other case had extensive pleural calcification. Both workers had frequently sanded asphalt and vinyl tile floors prior to installation of new floor covering. A technique to simulate normal work practice was developed and levels of 1.2 and 1.3 fibers/cc >5 μm in length resulted. The authors noted that under other work conditions these values may be higher. In the case involving mesothelioma, the worker was 44 years old and had no other history of occupational exposure to asbestos, although he had worked in a shipyard in a "non-dusty" gyroscope repair area from 1945-1947. The repair area would practically have to be considered a clean room operation in view of the precision involved in gyroscopic instrument repairs. He had smoked one package of cigarettes a day between the ages of 17 and 30 and had worked from 1948-1967 as a floor tile installer. The second case involved a 61-year-old worker who had been a floor tile installer for the last 30 years and had smoked one pack of cigarettes per day for the last 45 years. This second patient had no history of other asbestos exposure different from the first; however, some question may be raised of a possible neighborhood exposure even if it only concerned going to work. The possibility of such exposure must be considered in view of the neighborhood case noted by Selikoff,[15] Table XXIX.

The possibility of the development of asbestos-related diseases in floor tile installation must be considered, and special attention must be given to this operation when considering the low levels of

exposure that may be related to these two cases. If even in actual practice, levels were found to be 10 times those found by the investigators, it would substantiate the low levels of exposure recommended in this standard. The time interval for sanding as compared to tile installation must be small, and, if this is true, then, in fact, any level found would be very low if based on a time-weighted average exposure. This increases the weight of consideration that must be given to this possibly exposed occupational group and the relationship of these low exposures to asbestos to the development of disease.

Consideration must also be given related to the effect that may have resulted from exposure to other material in the floor tile. The level of, and effect of such material as asphalt and any decomposition products from sanding must be considered.

Isolated clinical case reports are difficult to interpret in terms of dose-time response relationship and can only be used to indicate other possible problem areas and to highlight what may prove to be practicable areas for further study.

IV. ENVIRONMENTAL DATA

The use of asbestos has changed with the addition of new products and with changes in the industrial processes. These changes and a growing awareness of the health effects from exposure of the worker to asbestos have resulted in a changing work environment within the asbestos industry. The lack of environmental data for previous years and the changes in technology used to collect samples, now and in the past, have resulted in the availability of comparable environmental data for only the last few years. Thus, the scant data and the long latent period for the development of bronchogenic cancer and mesothelioma do not permit the establishment of the dose-response relationship at this time. However, as has been indicated, the development of the diseases has been proven in workers exposed to asbestos and environmental data does exist for the last several years.

Table XIV shows the average concentration of asbestos fibers to which a number of insulation workers were exposed in 1969. The results shown are not time-weighted averages, but are averages of concentrations found for individual exposures during the time samples were collected (usually 15, 30, or 60 minutes). Although the average concentrations are reasonably low, with the exception of spraying, individual exposures varied from 0 to 100 fibers/cc. The latter occurred during a 60-minute period while a workman sprayed asbestos fiber on a turbine.

McClure[60] summarized results of a preliminary survey conducted by the U. S. Department of Labor during the period July, 1969, to January, 1970, at nine private shipyards as follows: 37 of 74 samples

collected during various operations of preparing and applying insulation were above 2 fibers/cc (50%) and 19 of 74 were about 12 fibers/cc (26%). These were not time-weighted average exposures, but represented average fiber concentrations during the sampling period. Furthermore, none of these samples represented workers' exposures while tearing out old insulation and lagging--an operation that has been previously found to produce more dust than the application of the insulation.

A summary of some of the environmental data collected by NIOSH is presented in Table I through XII. The environmental data presented in this document represent only that collected in the last few years and reported in fibers/cc>5 μm as counted by phase contrast light microscopy. As pointed out by Ayer et al.[61], "It is obviously impossible to give any single ratio that would accurately represent all processes at all times in each plant." As a result, little correlation, if any, can be made between early data (collected with an impinger where settled particles were counted) with current data (collected with a personal sampler and counted under a microscope equipped with a 16 mm 10X objective).

These data represent only the levels found during the time the samples were actually being taken. The sampling times were usually between 15 minutes to one hour, and should not be considered as time-weighted average exposures even though credence could be given to this approach due to the large number of samples collected.

Levels of exposure in the manufacture of asbestos are given in Table I through XII. In a total of 7 asbestos cement pipe plants, a range of individual samples was from 13.4 in coupling finishing, to levels too low to count in pipe forming, curing, pipe finishing,

coupling finishing, packing and miscellaneous operations (Table I).

It should also be noted in Table I that when consideration is given to feasibility of engineering control, in coupling finishing, the individual highest sample was 13.4 and the lowest and second lowest samples were zero. Warehousing and mixing (6.3 fibers/cc>5 μm) and packing (6.1 fibers/cc>5 μm) were the highest means by operation (Table II), and the lows were both 0.4 fibers/cc>5 μm. These data indicate the possibility of controlling these operations to below the proposed standards.

These wide ranges of individual samples and means by operations were also shown in asbestos friction plants (Tables III and IV), cement shingle, millboard, and gasket operations (Tables V and VI), insulation (Tables IX and X), and from asbestos paper, packing and asphalt products (Tables VII and VIII).

In textile operations, while the individual low and second lowest concentrations were, in all cases, below 1.0 fiber/cc (except fiber preparation, 1.4 fibers/cc), the means by operations exceeded 2.0 fibers/cc in fiber preparation (7.4 fibers/cc), carding (6.1 fibers/cc), spinning (3.7 fibers/cc), and twisting (3.2 fibers/cc). In the second lowest group, all operations except finishing exceeded 2.0 fibers/cc. These values, when considered with the highest means and highest individual samples (143.9 fibers/cc in carding and 123.2 in weaving), indicate that present methods of control practiced in the textile industry are not adequate for the standard proposed.

This is probably true in insulation operations as well. Even though levels were below the level of 2.0 fibers/cc>5 μm, the individual samples and operational means were high.

The individual sample high (Table IX) was 208.4 in finishing and 188.9 fibers/cc in mixing. Table XXV shows that in at least one insulation plant, 100 percent of all samples taken were less than or equal to 2 fibers/cc>5 μm, and in one other, all but the mixing operations met the 5 fibers/cc>5 μm value. In textiles, under present operating conditions, none of the plants met the 2 fibers/cc>5 μm criteria (Table XXV). This does not imply that industry could not meet the proposed standard of a time-weighted average exposure of 2.0 fibers/cc>5 μm, but only that it is not meeting it at the present in the insulation and textile plants, and it probably could meet the standard if given time to clean-up the plant operations.

Secular trends indicate that there is a wide variation between a few samples taken over large intervals of time. The evaluation of these trends, if indeed they are trends, would be open to question. however, it does point out that much can be done in the improvement of plant operations. It is not reasonable to associate these differences with changes in field sampling methods, counting techniques, or locations of sampling devices when similar trends are not apparent in cement pipe (Table XV), friction (Table XVI), or shingle, millboard and gasket operations (Table XVII). Variation in trends in insulation and textile plants (Tables XIX to XXI) indicate stable plants in some areas and not in others. The comparatively low values in textiles is somewhat surprising.

At most of the operations in the well-controlled plants, it is possible to meet the proposed standard with only small changes in engineering practices (Table XII). This is also true to a lesser degree

in friction operations (Table XXIII), and shingle, millboard, and gasket operations (Table XXIV), and true in only a few operations in textiles and insulation operations (Tables XXV-XXVI).

It must be noted that in asbestos plants having the same operations, some have been able to meet the proposed standard, while others have exhibited environmental values at higher levels, which suggests the need for engineering control - not the lack of engineering feasibility to meet the standard.

It will not be easy to control exposure in the insulation and textile industries, where higher levels of asbestosis, lung cancer, and mesothelioma are known to occur. There is a high priority requirement to protect the workers in these industries to assure that excessive asbestosis, lung cancer, and mesothelioma will not continue and, at the same time, give the worker the type of protection that is required at once. Table XIII gives an indication of the dramatic reduction in time-weighted average exposures that could be accomplished if peak or ceiling exposures were eliminated. In this case, reducing the peaks in insulation operations to the ceiling of 10 fibers/cc reduced the time-weighted average to near 2 fibers/cc.

V. DEVELOPMENT OF STANDARD

Various criteria have been used for categorizing the dustiness of the environment. Recent developments have made it clear that a method utilizing the capture and direct estimation of fibers of asbestos should be utilized for environmental measurement of exposure to asbestos. In the past, in the United States, asbestos fibers were measured by the impinger method which included counting particles as well as asbestos fibers.

The question still exists as to whether or not different varieties of asbestos fibers may have varying biological effects. This will not be answered until more definitive information is available on the specific etiological agent(s) and mechanisms of injury involved. The consumption of asbestos in this country is overwhelmingly in the form of chrysotile. Where other forms of asbestos are used, such as crocidolite and amosite, they are often mixed with chrysotile and are encountered alone, mainly in research and specialty situations. It would be extremely difficult on the basis of current information on biological effects and industrial practices to establish and administer separate standards for different types of asbestos.

The question also arises on the validity of basing standards on the number of respirable fibers in the air greater than 5 micrometers in length. It is fully realized that the fiber-size spectrum of respirable asbestos fibers in any particular industrial environment will range from that of bundles of fibrils in the upper respirable size to those of the individual fibrils in the sub-micron size. The type and grade of fibers, nature of processing, and controls in existence will greatly

influence the fiber-size spectrum (fiber length and diameter) in any given environment. The problem is further complicated by the lack of definitive information on the biologic response to fibers of different sizes. It is known, however, that the longer fibers show a dose-response relation to asbestosis, and may have a different behavior and degree of response than the shorter size fibers which may, in the lower and sub-micron range, tend to resemble more the physical behavior of non-fibrous respirable particulates. Since it would not be feasible to have a standard on the total respirable fibers which would necessitate the routine use of expensive and time-consuming techniques including electron microscopy, an index of exposure must be selected which, as nearly as possible, relates to the predominant biologic activity and dose-response of the size spectrum of fibers most commonly encountered. It is assumed for the present that the factor of safety associated with the standard will allow for differences in the size spectrum of respirable fibers that may be encountered.

The British, in evaluating respirable chrysotile fiber exposures in relation to the ongoing epidemiologic studies in the textile industry and for the basis of a standard for chrysotile, established as an index of exposure, fibers greater than 5 micrometers in length.[62] A substantial amount of information on the biologic effects of asbestos has, and is, being obtained using this parameter of exposure measurement. A review of the research in Britain, with concurrence on the rationale involved, made it prudent that we use the same definition of index-of-exposure on which to base criteria for standards. These criteria should be re-evaluated when, (1) more definitive information on the biologic response of asbestos including the agent(s) and dose-response data on different lengths of fiber is

available, (2) the spectrum of fiber lengths encountered in industry by types of asbestos and operations is ascertained, and (3) more precise epidemiologic data are developed.

To prevent fibrosis and excessive rates of neoplasia, such as mesothelioma, respiratory cancer, and gastrointestinal cancer, a standard for asbestos dust should be based on a concept of dose-response that includes not only the factor of fiber count times years of exposure but also that for total asbestos dust fibers retained over a number of years.

Thus, the effect after several decades of a one-time acute dose of limited duration which overwhelms the clearing mechanism, and is retained in the lungs, may be as harmful as the cumulative effect of lower daily doses of exposure over many years of work.

Basis for Previous Standards

The first standard for controlling exposure to asbestos dust was recommended by Dreessen et al.[63] in 1938 following a study of 541 employees in four asbestos textile plants where massive exposures occurred. A tentative limit for asbestos dust in the textile industry of 5 million particles per cubic foot (mppcf), determined by the impinger technique, was recommended. They found numerous well-marked cases of pneumoconiosis where concentrations exceeded 5 mppcf, but only three doubtful cases where concentrations were under 5 mppcf. However, only five persons had been exposed for more than 10 years to concentrations from 0.0 to 4.9 mppcf. None of the 39 persons exposed to concentrations below 2.5 mppcf showed evidence of asbestosis; but only six of these had been employed more than five years.

The study by Dreessen et al. had unavoidable limitations such as the fact that 333 of the 541 employees studied had worked less than five years in these textile mills, only 66 were employed as long as 10 years, and only 2 for more than 20 years. Furthermore, the average age of these asbestos textile workers was 32.1 years and only one of the four plants studied had been in operation for more than 15 years. Thus, the first standard established was based upon limited data. The authors recognized the limitations and stated that . . . "5 mppcf may be regarded tentatively as the threshold value for asbestos-dust exposure until better data are available."

The American Conference of Governmental Industrial Hygientists' (ACGIH) Threshold Limit Value (TLV) for asbestos dust was 5 mppcf

from 1946 to 1970. This limit was based on the study by Dreessen et al.[63] and subsequent investigations by others. In 1968 and 1969, ACGIH published notices of intended changes to lower the TLV to 12 fibers/ml >5 μm in length or 2 mppcf and they published in 1970 and 1971 a still lower limit of 5 fibers/ml >5 μm in length as a notice of proposed intended change. The conversion of data from mppcf to fibers/ml in all asbestos operations can only be done with considerable risk to the validity of the results. Lynch et al.[64] pointed out in 1970 the need for such conversion data and that the data reported in 1965[61] of the 12 fiber/ml equivalent to 2 mppcf relationship was obtained in textile mills and should not be applied to other product areas. Estimates of risk of disease in other product areas should be based on fiber counts since this method yields a more direct estimate of airborne asbestos concentration.

In 1968, the Committee on Hygienic Standards of the British Occupational Hygiene Society (BOHS) after reviewing medical evidence, results of studies made by the asbestos industry in the United Kingdom, and epidemiological data from the United States, published Hygienic Standards for Chrysotile Asbestos Dust.[62] It stated:

"1. As long as there is any airborne chrysotile dust in the work environment there may be some small risk to health. Nevertheless, it should be realized that exposure up to certain limits can be tolerated for a lifetime without incurring undue risks.

"2. The committee believes that a proper and reasonable objective would be to reduce the risk of contracting asbestosis to 1 percent of those who have a lifetime's exposure to the dust. By 'asbestosis'

this committee means the earliest demonstrable effects on the lungs due to asbestos.

"It is probable that the risk of being affected to the extent of having such early clinical signs will be less than 1 percent for an accumulated exposure of 100 fiber years per cm^3 or 2 fibers/cm^3 for 50 years, 4 fibers per cm^3 for 25 years or 10 fibers per cm^3 for 10 years.

"3. It is recommended that exposures which lie in certain ranges of dustiness be designated by categories according to the following scheme:

DUST CATEGORY	CONCENTRATION AVERAGED OVER 3 MONTHS (FIBERS/cm^3)
Negligible	0-0.4
Low	0.5-1.9
Medium	2.0-10.0
High	Over 10.0

"4. The levels are expressed in terms of the number of fibers per cm^3 greater than 5 μm in length as determined with the standard membrane filter method. Any other method can be used provided it is accompanied by appropriate evidence relating its results to those which would have been obtained with the standard membrane filter method.

"5. When it is necessary to work intermittently in a 'high dust' area an approved mask should be worn, provided that the concentration is no more than 50 fibers per cm^3 a higher standard of respiratory protection should be provided such as a pressure-fed breathing apparatus.

"Additional Recommendations

"1. It is recommended that where practicable an up-to-date employment record card be kept of every person which indicates, every calendar quarter, the category or categories in which he or she has been employed and in which he or she is recommended to work.

"2. All employees exposed to risk should be medically examined before employment. Periodic examinations should be made thereafter, annually.

"Notes:

"These hygienic standards are subject to review in the light of new evidence and improved methods of measurement.

"The standards are, in our opinion, the best that can be drawn from the existing data. These data are scanty and based on factory experience of continuous exposure during working hours. Due caution should be exercised in applying these standards to other patterns of exposure. As far as possible the dust exposures have been estimated conservatively and, in particular, in the period 1933-1950 the average hours of work were substantially greater than 40 per week.

"It is hoped to supplement the existing data in due course, when the standards will, if necessary, be modified. These standards will be formally reviewed in three years."*

In an unpublished paper, Williams, Baier, and Thomas compiled data from the Pennsylvania Department of Health files on exposure levels at

*As of 1/6/72 their standards as effective in May 1970 had not been revised. Per telephone conversation with Dr. S. Holmes, Secretary to the Asbestosis Research Council.

various textile processing operations in two plants. Their data included dust concentrations from 1930 through 1967 in one plant and from 1948 through 1968 in the second plant. Even though controlled exposures were, for the most part, below 5 mppcf and in many cases below the 1968 ACGIH Notice of Intended Change to 2 mppcf, 64 cases of asbestosis were reported from these two asbestos textile plants. The authors conclude that: "If asbestosis is to be prevented, airborne asbestos dust must be stringently controlled in the working environment. From these data a TLV of 3 mppcf would provide inadequate protection and the proposed 2 mppcf may not be substantiated."

Gee and Bouhuys,[65] in December, 1971, pointed out that on the basis of "reasonable probability," decisions must be made to control exposure to asbestos rather than from a precise definition of dose-response relationship, and "the present threshold limit value for asbestos should be lowered far below some recent proposal."

U. S. Emergency Standard

The present emergency standard for exposure to asbestos dust (29 CFR 1910.93a) published in the Federal Register, Vol. 36, No. 234, page 23207, December 7, 1971) is as follows:

> "The 8-hour time-weighted average airborne concentration of asbestos dust to which employees are exposed shall not exceed 5 fibers per milliliter greater than 5 microns in length, as determined by the membrane filter method at 400-450X magnification (4 millimeter objective) phase contrast illumination. Concentrations above 5 fibers per milliliter but, not to exceed 10 fibers per milliliter, may be permitted up to a total of 15 minutes in an hour for up to 5 hours in an 8-hour day."

The 1971 ACGIH tentative threshold limit value is 5 fibers/ml $> 5\ \mu m$ in length. Both are higher than the British standard of 2 fibers/cc by at least a factor of 1.5 times.

Basis for Recommended Standard

The number of studies that have collected both environmental and medical data and with a significant number of exposed workers is not sufficient to establish a meaningful standard based upon firm scientific data. The requirement to protect the worker exposed to asbestos is defined in a number of studies outlined in this document. The general recognition of the increasing number of cases of asbestosis, bronchogenic cancer, and mesothelioma indicates the urgent need to develop a standard at the present time.

NIOSH recognizes that these data are fragmentary and, as a result, a safety factor must be included in any standard considered. On this basis the research that did include both environmental and medical data, or where a standard or limit had been proposed, was given a careful and detailed study to determine its particular contribution to the development of a national standard.

The development of a standard for asbestos dust[66] in Great Britain and the evaluation made by the British Occupational Hygiene Society (BOHS) Sub-committee on Hygiene Standards for Asbestos,[62,66] which considered data to reduce the risk of asbestosis, was given great weight in the development of this asbestos standard. The BOHS fitted the data available to a dose-response curve and the conclusion was drawn that an accumulated exposure of 100 fiber-years/cm^3 would reduce early clinical signs to less than 1%. This would be 2 fibers/cm^3 for 50 years of exposure or 4 fibers/cm^3 for 25 years. According to Roach,[67] "The British Occupational Hygiene Society Standards Sub-committee on Asbestos expressed the view that a proper and reasonable objective would be to reduce exposures to below this level and thereby reduce the risk of

contracting asbestosis to less than 1% of those who have a lifetime exposure to the dust. For such workers, who may possibly work for 50 years, the long-term average concentration to which they are exposed would need to be less than 2 fibers/cm^3. For others, who will be exposed to asbestos dust in air for shorter periods, the long-term average concentration need not be so low, as long as their exposure will amount to less than 100 fiber-years/cm^3."

It is recognized that the British standard is based upon data not as precise as desired, but it does offer a mechanism for comparison with the ACGIH TLV and after three years of use no change has been recommended. The British standard was primarily based upon a study of 290 men employed for 10 years or longer between 1933-1966 in an asbestos textile mill. The environmental dust concentrations to which different workers had been exposed were estimated to have varied from 1 to 27 fibers/cm^3. The risk-exposure relationships were developed based upon basal rales and X-ray changes. In this study, basal rales were considered the key symptom since all workers exhibiting X-ray changes also exhibited basal rales.

In reviewing the values on the basis of the 100 fiber-years/cm^3 proposed by the British Hygiene Standards Committee, the following comparisons can be made between the British Standard and the Emergency U. S. Standard. Each standard is normalized to 100 fiber-years to account for differences in the working lifetime of the average asbestos worker. The Emergency U. S. Standard is based upon the ACGIH TLV which, in turn, is based upon an exposure time of 30 years to 5 fibers/ml> 5 um in length[68],

and the British, 50 years of exposure at 2 fibers/cm^3 > 5 um in length.

In summary:

	British	U. S. Emergency ACGIH
	2 fibers/cc	5 fibers/ml
Fiber-yrs/cc	100	150

The validity of this type of comparison has already been questioned in this document, i.e., the "K" factor used to change ACGIH impinger data to fiber counts.[61,64]

However, on this basis, data suggest that the ACGIH value is higher than the British value.

In addition to consideration of the British data, the comparison of British and ACGIH data suggests that the 30-year exposure value for a U. S. Standard should be about 3 fibers/cc 5 μm in length in order to assure that less than 1% of the workers exposed are at risk of developing the earliest clinical signs of asbestosis.

However, additional consideration must be given to the concepts of carcinogenesis as they relate to the determination of a standard for asbestos exposure. Any carcinogen (initiator) must be assumed, until otherwise proven, to have discrete, dose-dependent, irreversible and additive effects to cells that are transmissible to the cell progeny. Thus, initiation of malignancy following single small exposures to asbestos is possible, but of a low probability. With frequent or chronic exposure and a low dose-rate, the probability of initiation of malignancy is increased. Yet, even under optimal conditions of cell proliferation (in the presence of promotors) these malignant

transformations do not lead to instantaneous cancer, but remain insidious for a number of years (latent).

In protracted exposure, some of the total accumulated exposure is "wasted" (or irrelevant) as far as the initiator of cancer is concerned. Exposures in excess of the minimal initiation dose conceivably may shorten the latent period to some extent by substituting for other contributing factors that would have eventually been effectual in converting the latent tumor into a frank malignancy. Analytic methods used in the epidemiology of asbestos-induced cancers are unable to discriminate between the initiating dose and subsequent (wasted) exposure.

Consideration must also be given to the concept that an inverse relationship exists between dose-rate and the latent period. As the dose-rate becomes progressively lower, the latent period may approach or exceed the life span of exposed individuals.

Adherence to these concepts would argue toward reducing asbestos exposure substantially below those levels currently demonstrated to be associated with the disease. Such a course of action is consistent with the Surgeon General's ad hoc Committee on Evaluation of Low Levels of Environmental Chemical Carcinogens statement that, "for carcinogenic agents, a safe level for man cannot be established by application of our present knowledge."

Work practices in industries should be encouraged to develop work practice standards by the consensus method so that the lowest feasible environmental levels can be obtained. The following work practice standards are included in the emergency standard for asbestos and are included in the recommended standard:

(a) Asbestos cement, mortar, coatings, grout, and plaster shall be mixed in closed bags or other containers.

(b) Asbestos waste and scrap shall be collected and disposed of in sealed bags or other containers.

(c) All cleanup of asbestos dust shall be performed by vacuum cleaners or by wet cleaning methods. No dry sweeping shall be performed.

The need in industry for a proper precautionary label for asbestos and for other hazardous materials associated with the mining, production, and use of chemical compounds has existed for a number of years. The development of a labeling system for use as an occupational hazard warning system overlaps into so many other labeling areas, e.g., transportation of chemicals, fire fighting, use by the military, etc., that it would be necessary either to develop a separate system for use in relation to occupational exposures only, or to combine all the present systems into one.

The addition of one more labeling system compounds the multi-labeling requirement presently imposed on industry and creates one more labeling system the worker must recognize. Combining all systems into one requires the coordination of many governmental, professional, trade, manufacturing, and international and local organizations. Time required to accomplish this task is prohibitive in relation to the requirement for the immediate development of an occupational health standard for asbestos. As a result, NIOSH recommends as an interim system the adoption, with modification, of the system for the Identification of the Fire Hazards of Materials of the National Fire Protection Association and the Guide to Precautionary Labeling of

Hazardous Chemicals of the Manufacturing Chemists Association.

It is recognized that this system may not be the most appropriate system and may require additional development to permit the worker, himself, to use it to identify the hazards to which he is exposed and to learn the necessary precautions to assure him safe working conditions. (See Appendix II for the details and modification of the labeling system).

Summary of the Basis for the Recommended Standard

The recommendation for an environmental standard for asbestos is based upon health considerations and limited engineering feasibility data. The overriding considerations are the health effects.

Evidence indicates that past and current standards for fiber concentrations in the working places where asbestos fibers occur, though undoubtedly contributing to reduction of the severity and frequency of asbestosis, have not provided complete protection from exposure to asbestos, necessitating development of a new standard.

Consideration was given to previous reports and studies, recent data, and the present "state-of-the-art." It is recognized that additional data would be desirable to support an asbestos standard, but because of immediate need for worker protection, it is necessary to make a recommendation based on available studies and data. The following constraints in applicability of research data were considered in the development of the recommendations:

(a) Few epidemiological studies or clinical reports with supporting environmental data are available in the exposure range that must be considered.

(b) Environmental data on practically all studies were collected only over the last few years and/or they were collected by other techniques and expressed in terms other than fibers/cc.

(c) The environmental samples were expressly collected in many cases for control purposes rather than for research and, as a result, meaningful evaluations cannot be made.

(d) There is a lack of data to define with any degree of precision the threshold of development of neoplasms resulting from exposure to asbestos and the relationship of the latent period between exposure and development of neoplasms.

The standard recommended in this document is similar to the standard adopted by Her Majesty's Factory Inspectorate in 1969[66] (still in effect as of December 29, 1971), and more stringent than the recent U. S. Emergency Standard. It is felt to be feasible technologically for the control of the exposure to the worker and effective biologically for protection of the worker against asbestos-induced diseases.

Considerations of carcinogenesis indicated the need for a measure of prudence. As a result of this rationale, a factor was added to reduce the time-weighted average exposure to 2.0 fibers/cc > 5 um. A ceiling value of 10.0 fibers/cc > 5 um that was not to be exceeded was included to reduce the possibility of the short-term heavy exposures to asbestos that have been reported to cause mesothelioma. In addition, this should reduce the likelihood of diseases (malignant and non-malignant) resulting from exposures in excess of 30 years or with very long latent periods.

VI. COMPATIBILITY WITH EMISSION STANDARDS

The proposed national emission standard for asbestos was published in the Federal Register, Vol. 36, No. 235, pages 2342-2343 (40 CFR 61.20-61.24) by the Environmental Protection Agency. The emission standard will be applicable to asbestos mines, mills; building structures, or facilities within which manufacturing or fabricating operations involving the use of commercial asbestos; buildings or structures which have been or will be constructed or modified using asbestos insulation products; roadway facilities which would be surfaced or resurfaced using asbestos tailings.

The standards are based upon information derived from many sources, including health effect levels, meteorology, technical analysis of control capability, and consideration of economic impact. The overriding considerations are health effects. These standards are based upon specific operations and physical conditions and are limited in general to emissions to the atmosphere.

1. Emissions shall not exceed those which would be emitted from operations if proper engineering control had been installed (i.e. fabric filter, cyclone gas cleaning devices).

2. Visible emissions of particulate

3. Spraying of asbestos

4. Use of asbestos for surfacing or resurfacing of roads.

The use of procedural standards and visible emissions as the basis for evaluation for compliance with the standard are designed to minimize emission to the atmosphere. EPA determined that there

is no suitable technique for sampling and analysis of asbestos in ambient air or emission gases. This determination was made as only limited information had been developed from measuring fibers in community air. The use of high volume samplers for collection of samples and counting by light microscopic techniques similar to industrial hygiene methods has shown only small numbers of fibers in urban areas.[69]

It was felt that these values were low when compared to occupational health experience and values to few too use with confidence.[69]

As a result there is no direct comparison possible between the proposed national emission standards for asbestos and the recommended criteria for occupational exposure except to say that the levels of exposure to the general public on a 24-hour day, 7 days a week, basis would be lower, as would be expected, than occupational standards based on an 8-hour day, 40-hour work week.

The Illinois Pollution Control Board on November 30, 1971,[70] published a notice of proposed final draft of emission standards for asbestos that can be more easily related to the recommended occupational standard than those proposed by EPA. Illinois includes a provision that, "After June 30, 1972, a factory, plant or enterprise which engages in the processing or manufacturing of any asbestos-containing product shall discharge no visible emission of particulate matter from such manufacturing or processing into the ambient air and shall emit no concentrations of asbestos fiber in excess of 2 fibers per cubic centimeter of air."

The method of counting the asbestos fibers is that proposed by Edwards et al.[71] and similar to the technique proposed in Appendix I of this report. This proposed Illinois standard places a ceiling value of 2 fibers/cc on emissions from processing on manufacturing of asbestos containing products. In the explanation of the revision of the proposed Illinois regulation they state:

> "IV. Part V, controlling manufacturing sources, is changed to require an emission standard of 2 fibers per cubic centimeter and no visible emissions. While some testimony indicated the difficulty in measuring compliance with a numerical emission standard, overall the evidence establishes both the need (protection against the great proportion of invisible fiber) and the ease of measurement of such a criterion. A "no visible emission" standard has been added to the numerical standard to simplify enforcement against exceptionally dirty emission sources. A grace period, until June 30, 1972, has been added to permit acquisition of the necessary control equipment to attain the emission standard."

This air quality standard is, as it should be, more restrictive that an occupational standard due to differences in exposure time.

This proposed occupational standard would seem to be compatible with the proposed emission standard and each should complement the other in the control of asbestos exposure.

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VIII. APPENDIX I

Air Sampling Methods

In the study of asbestosis conducted by Dreessen et al.[63] midget impinger count data were used as an estimate of dust exposure. All of the dust particles seen, both grains and fibers, were counted since too few fibers were seen to give an accurate measurement. The resulting count concentration was a measure of overall dust levels rather than a specific measurement of the asbestos concentration. This method was satisfactory at that time since exposures were massive and the control measures installed to reduce overall dust levels also reduced the asbestos dust levels.

As dust levels were reduced, it became necessary to measure the biologically appropriate attribute of the dust cloud. At equal levels overall dustiness, the concentration of asbestos could vary considerably from textile manufacture (75-85%) to insulation (5-15%). Furthermore, if the limit were lowered below the 5 mppcf used previously and dust counts taken by the impinger technique, it would be necessary to consider the effect of background dust, which could be as high as 1 mppcf.

A number of methods for measurement of asbestos dust concentrations have been used in the NIOSH epidemiological study of the asbestos product industry.[73,74,75,76] Based on these data, the preferred index of asbestos exposure is the concentration of fibers longer than 5 μm counted on membrane filters at 430X with phase contrast illumination.[71,72] This index is utilized in the method adopted as the standard field sampling method by the Public Health Service.

Fibers longer than 5 μm in length are counted in preference to counting all fibers seen in order to minimize observer/microscope resolving power variability. Furthermore, the British define a "fibre" as a particle, "of length between 5 μm and 100 μm and having a length-to-breadth ratio of at least 3:1, observed by transmitted light by means of a microscope at a magnification of approximately 500X."[62]

Although the British have refrained from standardizing on a single method of measurement, recent measurements have been performed by a method essentially identical to the fiber-count method described in detail below, and the British hygiene standards for use with their asbestos regulations are stated in these terms.[62]

Principles of Sampling

A dust sampling procedure must be designed so that samples of actual dust concentrations are collected accurately and consistently. The results of the analysis of these samples will reflect, realistically, the concentrations of dust at the place and time of sampling.

In order to collect a sample representative of airborne dust, which is likely to enter the subject's respiratory system, it is necessary to position a collection apparatus near the nose and mouth of the subject or in his "breathing zone".

The concentration of dust in the air to which a worker is exposed will vary, depending upon the nature of the operation and upon the type of work performed by the operator and the position of the operator relative to the source of the dust. The amount of dust inhaled by a worker can vary daily, seasonally, and with the weather. In order to obtain representative samples of workers' exposures, it is necessary to collect samples under varying conditions of weather, on different

days, and at different times during a shift.

The percentage of working time spent on different tasks will affect the concentration of dust the worker inhales since the different tasks usually result in exposure to different concentrations. The percentage can be determined from work schedules and by observation of work routines.

The daily average weighted exposure can be determined by using the following formula:

$$\frac{(\text{Hours X conc. task A}) + (\text{Hours X conc. task B}) + \text{etc.}}{\text{8 Hours (or actual hours worked)}}$$

The concentration of any air contaminant resulting from an industrial operation also varies with time. Therefore, a longer sampling time will better approximate the actual average.

With the following recommended sampling procedure, it is possible to collect samples at the workers' breathing zones for periods from 4 to 8 hours, thus permitting the evaluation of average exposures for a half or full 8-hour shift--a desirable and recommended procedure. Furthermore, dust exposures of a more normal work pattern result from the use of personal samplers. In evaluating daily exposures, samples should be collected as near as possible to workers' breathing zones.

Collecting Sample

The method recommended in this report for taking samples and counting fibers is based on a modification of the membrane filter method described by Edwards and Lynch.[71]

The sample should be collected on a 37-millimeter Millipore type AA* filter mounted in an open-face filter holder. The holder should be fastened to the worker's lapel and air drawn through the filter by means of a battery-powered personal sampler pump similar to those approved by NIOSH under the provisions of 30 CFR 74. The filters are contained in plastic filter holders and are supported on pads which also aid in controlling the distribution of air through the filter. To yield a more uniform sample deposit, the filter-holder face-caps should be removed. Sampling flow rates from 1.0 liter per minute (lpm) up to the maximum flow rate of the personal sampler pump (usually not over 2.5 lpm) and sampling time from 15 minutes to eight hours are acceptable provided the following restraints are considered:

(a) In order to obtain an accurate estimate of the number of fibers the statistical error resulting from the random distribution of the fibers must be kept to an acceptably low level. Since fiber counts follow a Poisson distribution, a count of 100 fibers in a sample would have a standard deviation of $\sqrt{100}$ or 10 fibers or $\pm$ 10%. Thus the 95% confidence limits would be approximately 2 standard deviations or $\pm$ 20%. Since the 37 mm filter has an effective collecting area of 855 mm^2 and the projected field area of the Porton reticle is 0.005 mm^2, each field represents 1/171000 of the sample. Based on this ratio the following number of fields must be counted to measure the various limits in various sampling times:

*Mention of commercial products does not constitute endorsement by the Public Health Service or U. S. Department of Health, Education and Welfare.

Sampling Time Minutes	Flow Rate 1pm	Number of Fields for 100 Fibers 0.2 fibers/ml	2.0 fibers/ml	10 fibers/ml
10	2	4350	435	91
15	2	2860	286	58
30	2	1430	143	29
90	1	1000	100	20
90	2	500	50	10
240	1	260	26	7
240	2	180	18	4
480	1	180	18	4

(b) Do not count a field containing over 20 fibers because in addition to the fibers being counted, there are also present a number of grains, which interfere with the accuracy of the count.

Based on these restraints, i.e., number of fields to be counted and maximum number of fibers per field, acceptable sampling parameters for the various limits are underlined in the above table.

The following conclusions may be drawn from this analysis:

(1) The short-term limit should be for a period of at least 15 minutes and preferably 30 minutes.

(2) The 2.0 fiber/cc limit may be evaluated over periods of from 90 to 480 minutes.

As many fields as required to yield at least 100 fibers should be counted. In general the minimum number of fields should be 20 and the maximum 100.

Mounting Sample

The mounting medium used in this method is prepared by dissolving 0.05 g of membrane filter per ml of 1:1 solution of dimethyl phthalate

and diethyl oxalate. The index of refraction of the medium thus prepared is ND = 1.47.

To prepare a sample for microscopic examination, a drop of the mounting medium is placed on a freshly cleaned, standard (25 mm X 75 mm), microscopic slide. A wedge-shaped piece with arc length of about 1 cm is excised from the filter with a scalpel and forceps and placed dust-side-up on the drop of mounting solution. A No. 1-1/2 coverslip, carefully cleaned with lens tissue, is placed over the filter wedge. Slight pressure on the coverslip achieves contact between it and the mounting medium. The sample may be examined as soon as the mount is transparent. The optical homogeneity of the resulting mount is nearly perfect, with only a slight background granularity under phase contrast, which disappears within one day. The sample should be counted within two days after mounting.

Evaluation

The filter samples mounted in the manner previously described are evaluated in terms of the concentration of asbestos fibers greater than 5 μm in length. A microscope equipped with phase-contrast optics and a 4-mm "high-dry" achromatic objective is suitable for this determination. 10X eyepieces, one of which contains a Porton or other suitable reticle at the level of the field-limiting diaphragm, should be used. The left half of the Porton reticle field serves to define the counting area of the field. Twenty fields located at random on the sample are counted and total asbestos fibers longer than 5 μm are recorded. Any particle having an aspect ratio of three or greater is considered a fiber.

The following formulae are used to determine the number of fibers/ml:

$$(1)\quad \frac{\text{Filter area } (mm^2)}{\text{Field area } (mm^2)} = K$$

$$(2)\quad \frac{\text{Average net count X K}}{\text{Air volume sampled (ml)}} = \text{fibers/ml}$$

For example, assume the following: area of the filter used was 855 mm^2, counting area of one field under the Porton reticle was 0.005 mm^2; average net count per field of 20 fields was 10 fibers; and sample was collected at 2 liters per minute for 90 minutes: Then:

$$\frac{855 mm^2}{0.005\ mm^2} = 171{,}000\ (K)$$

$$\frac{10 \text{ fibers x } 171{,}000}{2{,}000 \text{ ml/min x } 90 \text{ min}} = 9.5 \text{ fibers/ml}$$

Calibration of Personal Sampler

The accuracy of an analysis can be no greater than the accuracy of the volume of air which is measured. Therefore, the accurate calibration of a sampling device is essential to the correct interpretation of an instrument's indication. The frequency of calibration is somewhat dependent on the use, care, and handling to which the pump is subjected. Pumps should be calibrated if they have been subjected to misuse or if they have just been repaired or received from a manufacturer. If hard usage is given the instrument, more frequent calibration may be necessary.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, a 1-liter burette or wet-test meter should be used. In the field, a rotameter is the most convenient

instrument used. The actual set-up will be the same for all of these instruments. The calibration instrument will be connected in sequence to the filter unit which will be followed by the personal sampler pump. In this way, the calibration instrument will be at atmospheric pressure. Connections between units can be made using the same type of tubing used in the personal sampling unit. Each pump must be calibrated separately for each type of filter used, if, for example, it has been decided to use a filter with a different pore size. The burette should be set up so that the flow is toward the narrow end of the unit.

Care must be exercised in the assembly procedure to insure adequate seals at the joints and that the length of connecting tubing be kept at a minimum. Calibration should be done under the same conditions of pressure, temperature and density as will be encountered. The rotameter should be used only in the field as a check if the diaphragm or piston pumps are not equipped with pulsation dampeners. The pulsating flow resulting from these type pumps causes the rotameter to give results which are not as accurate as that obtained with a burette or wet-test meter. Calibration can be accomplished with any of the other standard calibrating instruments, such as spirometer, Marriott's bottle, or dry-gas meter. The burette and wet-test meter were selected because of their accuracy, availability, and ease of operation.

IX. APPENDIX II

NUMERICAL HAZARD RATING SYSTEM

The numerical hazard ratings given to products for each category of hazard shall be in accordance with the following criteria. Figure 2 graphically illustrates the hazard identification system.

Health hazards shall be rated as follows:

The health hazard rating of a material shall be determined by evaluating the potential for exposure and the relative toxicity of the most toxic ingredient of a compound or mixture. For this evaluation, the following relative toxicity criteria* for absorbed or exposure dose will be used:

Commonly Used Term	LD_{50} Simple Oral Dose Rats mg/kg	Inhalation 4-hr. Vapor Exposure, Rats Mortality of 2/6 to 4/6 ppm	LD_{50} - Skin Rabbits mg/kg
Extremely toxic	≤ 1	≤ 10	≤ 5
Highly toxic	1.1 to 50	11 to 100	5.1 to 43
Moderately toxic	50.1 to 500	101 to 1000	44 to 340
Slightly toxic or practically non-toxic	501 to 15,000	1,001 to 100,000	350 to 22,600
Relatively harmless	15,000	100,000	22,600

Degree 4: Extremely Hazardous.

Materials, which on very short exposure, can cause death or major permanent injury, even though prompt medical treatment were given, including those which are too dangerous to be approached without specialized

*(Reference: A.I.H.A. Quarterly, Vol. 15, No. 2, June 1954. "Safe Handling Procedures for Compounds Developed by the Petro Chemical Industry," p. 141.)

protective equipment, such as self-contained breathing apparatus or a hose mask with blower, and impervious clothing. This rating includes:

(a) Carcinogens

(b) Materials capable of producing sensitization

(c) Extremely toxic materials which can penetrate ordinary protective clothing.

(d) Extremely hazardous materials, when under normal conditions give off gases that are extremely toxic or corrosive through inhalation or by contact with or absorption through any body surface.

Degree 3: Highly Hazardous.

Materials which on short exposure can cause serious temporary or residual injury, even though prompt medical treatment were given, including those requiring protection from all bodily contact. This rating includes:

(a) Materials giving off highly toxic combustion products

(b) Materials giving off highly toxic gases or vapors, under normal conditions

(c) Materials corrosive to living tissue or highly toxic by skin absorption

Degree 2: Hazardous.

Materials which on continued exposure can cause temporary incapacitation or possible residual injury unless prompt medical treatment is given. This rating includes:

(a) Materials giving off moderately toxic combustion products

(b) Materials which either under normal conditions or under fire conditions give off moderately toxic vapors lacking warning properties.

Degree 1: Slightly hazardous.

Materials, which on exposure at normal conditions, would cause irritation but only minor residual injury even if no treatment is given.

This rating includes:

(a) Materials which under fire conditions give off slightly toxic or irritating combustion products

(b) Materials which on the skin could cause irritation without destruction of tissue

Degree 0: Harmless.

Materials which on exposure by skin contact, inhalation, or ingestion are relatively harmless or which under fire conditions offer no hazard beyond that of ordinary combustible materials.

Flammability hazards shall be rated as follows:

Degree 4.

Materials which will rapidly or completely vaporize at atmospheric pressure and normal ambient temperature or which are readily dispersed in air, and which will burn readily. This degree should include: Gaseous materials: Cryogenic materials; any liquid or gaseous material which is a liquid while under pressure and having a flash point below 73°F (22.8°C) and having a boiling point below 100°F (37.8°C). (Class 1A flammable liquids.)

Materials which on account of their physical form or environmental conditions can form explosive mixtures with air and which are readily dispersed in air, such as dusts of combustible solids and mists of flammable or combustible liquid droplets.

Degree 3.

Liquids and solids that can be ignited under almost all ambient temperature conditions. Materials in this degree produce hazardous

atmospheres with air under almost all ambient temperatures, are readily ignited under almost all conditions. This degree should include: Liquids having a flash point below 73°F (22.8°C) and having a boiling point at or above 100°F (37.8°C) and those liquids having a flash point at or above 73°F (22.8°C) and below 100°F (37.8°C). (Class 1B and Class 1C flammable liquids);

Solid materials in the form of coarse dusts which may burn rapidly but which generally do not form explosive atmosphere with air;

Solid materials in a fibrous or shredded form which may burn rapidly and create flash fire hazards, such as cotton, sisal and hemp;

Solids which burn with extreme rapidity usually by reason of self-contained oxygen (e.g., dry nitrocellulose);

Materials which ignite spontaneously when exposed to air.

Degree 2.

Materials that must be moderately heated or exposed to relatively high ambient temperatures before ignition can occur. Materials in this degree would not under normal conditions form hazardous atmospheres with air, but under high ambient temperatures or under moderate heating may release vapor in sufficient quantities to produce hazardous atmospheres with air. This degree should include:

Liquids having a flash point about 100°F, but not exceeding 200°F; solids and semisolids which readily give off flammable vapors.

Degree 1.

Materials that must be preheated before ignition can occur. Materials in this degree require considerable preheating, under all ambient temperature conditions, before ignition and combustion can occur.

This degree should include:

Materials which will burn in air when exposed to a temperature of 1500°F for a period of five minutes or less;

Liquids, solids and semisolids having a flash point above 200°F; this degree includes most ordinary combustible materials.

Degree 0.

Materials that will not burn. This degree should include any material which will not burn in air when exposed to a temperature of 1500°F for a period of five minutes.

Reactivity hazards shall be rated as follows:

Degree 4.

Materials which are readily capable of detonation or of explosive decomposition or explosive reaction at normal temperatures and pressures. This degree should include materials which are sentitive to mechanical or localized thermal shock at normal temperatures and pressures.

Degree 3

Materials which are capable of detonation or of explosive decomposition or explosive reaction but which require a strong initiating source or which must be heated under confinement before initiation. This degree should include materials which are sensitive to thermal or mechanical shock at elevated temperatures and pressures or which react explosively with water without requiring heat or confinement.

Degree 2.

Materials which are normally unstable and readily undergo violent chemical change but do not detonate. This degree should include materials which can undergo chemical change with rapid release of energy at normal

termperatures and pressures or which can undergo violent chemical change at elevated temperatures and pressures. It should also include those materials which may react violently with water or which may form potentially explosive mixtures with water.

Degree 1.

Materials which are normally stable, but which may react with water with some release of energy but not violently.

Degree 0.

Materials which are normally stable, even under fire exposure conditions, and which are not reactive with water.

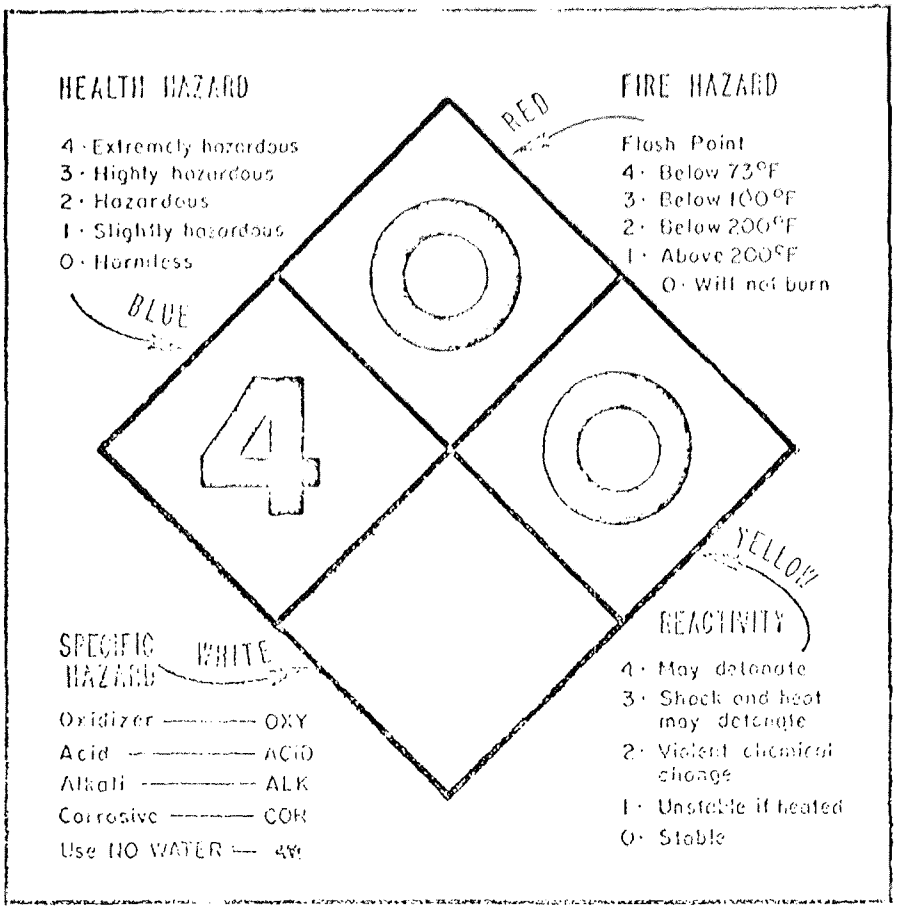
Specific hazards:

Oxidizing Material. A substance as chlorate, permanganate, peroxide, or a nitrate that yields oxygen to support combustion or which reacts readily to oxidize fuels or other combustible materials.

Corrosive Material. Acids, alkali or other material that will cause severe damage to living tissue or to other material it contacts.

Water Reactivity Hazard (Use No Water). Any material that may be a hazard because of its specific reactivity with water.

Figure 1. Hazard Identification System



COLOR AND DIMENSION

Color format for NFPA No. 704M designations are shown above. The colors indicated shall acceptably match in shade the applicable color of FED-STD-595 as follows:

Color	Class
Black	17038
Red	11105
White	17875
Yellow	13538
Blue	15102

Dimensions of the symbol and LAPI warning combination shall be optional but of such size and location as to be readily visible and legible.

The symbol and warning shall be applied by stenciling, painting, printing, lithographing, with fade-resistant materials.

X. APPENDIX III

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material containing 5% or more of asbestos shall be provided in the appropriate section of the Material Safety Data Sheet or approved form. If a specific item of information is inapplicable (i.e. flash point) initials "n.a." not applicable should be inserted.

(i) The product designation in the upper left hand corner of both front and back to facilitate filing and retrieval. Print in upper case letters in as large print possible.

(ii) Section I. Name and Source

(A) The name, address and telephone number of the manufacturer or supplier of the product.

(B) The trade name and synonyms for a mixture of chemicals, a basic structural material, or for a process material; and the trade name and synonyms, chemical name and synonyms, chemical family, and formula for a single chemical.

(iii) Section II. Hazardous Ingredients.

(A) Chemical or widely recognized common name of all hazardous ingredients.

(B) The approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range of maximum amount, i.e., 10-20% V; 10% max. W.

(C) Basis for toxicity for each hazardous material such as established OSHA standard (TLV), in appropriate units and/or LD_{50}, showing amount and mode of exposure and species or LC_{50} showing concentration and species.

(iv) Section III. Physical Data

(A) Physical properties of the total product including boiling point and melting point in degrees Fahrenheit; vapor pressure, in millimeters of mercury, vapor density of gas or vapor (air = 1), solubility in water, in parts per hundred parts of water by weight; specific gravity (water = 1); percent volatile, indicate if by weight or volume, at 70° Fahrenheit; evaporation rate for liquids (indicate whether butyl acetate or ether = 1); and appearance and odor.

(v) Section IV. Fire and Explosion Hazard Data.

(A) Fire and explosion hazard data about a single chemical or a mixture of chemicals, including flash point, in degrees Fahrenheit; flammable limits, in percent by volume in air; suitable extinguishing media or agents; special fire fighting procedures; and unusual fire and explosion hazard information.

(vi) Section V. Health Hazard Data.

(A) Toxic level for total compound or mixture, relevant symptoms of exposure, skin and eye irritation properties, principle routes of absorption, effects of chronic (long-term) exposure and emergency and first aid procedures.

(vii) Section VI. Reactivity Data.

(A) Chemical stability, incompatibility, hazardous decomposition products, and hazardous polymerization.

(viii) Section VII. Spill or Leak Procedures.

(A) Detailed procedures to be followed with emphasis on precautions to be taken in cleaning up and safe disposal of materials leaked or spilled. This includes proper labeling and disposal of containers containing residues,

contaminated absorbants, etc.

(ix) Section VIII. Special Protection Information.

(A) Requirements for personal protective equipment, such as respirators, eye protection and protective clothing, and ventilation such as local exhaust (at site of product use or application), general, or other special types.

(x) Section IX. Special Precautions.

(A) Any other general precautionary information such as personal protective equipment for exposure to the thermal decomposition products listed in Section VI, and to particulates formed by abrading a dry coating, such as by a power sanding disc.

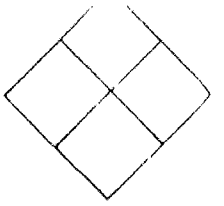
(xi) The signature of the responsible person filling out the data sheet, his address, and the date on which it is filled out.

(xii) The NFPA 704M numerical hazard ratings as defined in section (c) (5) following. The entry shall be made immediately to the right of the heading "Material Safety Data Sheet" at the top of the page and within a diamond symbol preprinted on the forms.

PRODUCT DESIGNATION

MATERIAL SAFETY
DATA SHEET

Form Approved
Budget Bureau No.
Approval Expires
Form No. OSHA



SECTION I SOURCE AND NOMENCLATURE	
MANUFACTURER'S NAME	EMERGENCY TELEPHONE NO.
ADDRESS (Number, Street, City, State, ZIP Code)	
TRADE NAME AND SYNONYMS	CHEMICAL FAMILY
CHEMICAL NAME AND SYNONYMS	FORMULA

SECTION II HAZARDOUS INGREDIENTS						
BASIC MATERIAL	APPROXIMATE OR MAXIMUM % WT. OR VOL.	ESTABLISHED OSHA STANDARD	LD_{50}		LC_{50}	
			ORAL	PERCUT.	SPECIES	CONC.

SECTION III PHYSICAL DATA	
BOILING POINT °F.	VAPOR PRESSURE mm Hg.
MELTING POINT °F.	VAPOR DENSITY (Air=1)
SPECIFIC GRAVITY (H_2O=1)	EVAPORATION RATE (_____=1)
SOLUBILITY IN WATER Pts/100 pts H_2O	VOLATILE % Vol. % Wt.
APPEARANCE AND ODOR	

SECTION IV FIRE AND EXPLOSION HAZARD DATA		
FLASH POINT	FLAMMABLE (EXPLOSIVE) LIMITS	UPPER
METHOD USED		LOWER
EXTINGUISHING MEDIA		
SPECIAL FIRE FIGHTING PROCEDURES		
UNUSUAL FIRE AND EXPLOSION HAZARDS		

PRODUCT DESIGNATION

SECTION V HEALTH HAZARD DATA	
TOXIC LEVEL	CARCINOGENIC
PRINCIPLE ROUTES OF ABSORBTION	SKIN AND EYE IRRITATION
RELEVANT SYMPTOMS OF EXPOSURE	
EFFECTS OF CHRONIC EXPOSURE	
EMERGENCY AND FIRST AID PROCEDURES	

SECTION VI REACTIVITY DATA
CONDITIONS CONTRIBUTING TO INSTABILITY
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
INCOMPATABILITY (Materials to Avoid)
HAZARDOUS DECOMPOSITION PRODUCTS

SECTION VII SPILL OR LEAK PROCEDURES
STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED
WASTE DISPOSAL METHOD

SECTION VIII SPECIAL PROTECTION INFORMATION	
VENTILATION REQUIREMENTS LOCAL EXHAUST	PROTECTIVE EQUIPMENT (Specify Types) EYE
MECHANICAL (General)	GLOVES
SPECIAL	RESPIRATOR
OTHER PROTECTIVE EQUIPMENT	

SECTION IX SPECIAL PRECAUTIONS
PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE
OTHER PRECAUTIONS

Signature _____ Address

Date _____

TABLE I

CEMENT PIPE PLANTS
NUMBER OF PLANTS = 7

INDIVIDUAL SAMPLES BY OPERATION AND SAMPLE SIZE

OPERATION	HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND LOWEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	LOWEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT
Warehousing & Mixing	7.0	2	O	5.6	2	O	0.2	3	P	0.2	4	Q
Pipe Forming	3.7	6	O	3.4	3	N	0.1	6	O	0.0	6	AA
Curing	2.6	3	O	2.1	15	BB	0.1	4	Z	0.0	6	P
Pipe Finishing	4.6	10	Z	4.0	5	Q	0.0	6	N	0.0	10	Z
Coupling Finishing	13.4	7	Z	10.5	7	Z	0.0	16	P	0.0	21	AA
Epoxy	4.7	1	N	2.1	6	BB	0.3	5	Z	0.2	6	BB
Packing	6.1	1	Q	2.5	7	Z	0.1	13	AA	0.0	13	AA
Miscellaneous	1.7	9	Z	1.4	9	P	0.1	9	P	0.0	9	Z

1 - All samples expressed as fibers > 5μ/cc counted by the standard method recommended in this document. (Latest Available NIOSH Data Collected during the Years 1969 through 1970).

TABLE II

ASBESTOS CEMENT PIPE PLANTS

NUMBER OF PLANTS = 7

MEANS BY OPERATION AND SAMPLE SIZE ( )

OPERATION	HIGHEST	PLANT	SECOND HIGHEST	PLANT	SECOND LOWEST	PLANT	LOWEST	PLANT
Warehousing & Mixing	6.3 (2)	O	2.7 (4)	N	0.7 (5)	AA	0.4 (3)	P
Pipe Forming	2.2 (3)	N	1.8 (4)	Z	0.5 (6)	AA	0.3 (4)	P
Curing	2.0 (3)	O	0.9 (15)	BB	0.4 (4)	Z	0.3 (6)	P
Pipe Finishing	1.7 (10)	Z	1.3 (5)	Q	0.6 (9)	AA	0.5 (6)	N
Coupling Finishing	5.3 (7)	Z	3.8 (4)	O	0.6 (21)	AA	0.5 (16)	P
Epoxy	4.7 (1)	N	1.1 (6)	BB	0.6 (6)	P	0.3 (1)	AA
Packing	6.1 (1)	Q	1.1 (7)	Z	0.7 (6)	BB	0.4 (13)	AA
Miscellaneous	0.5 (9)	Z	0.5 (6)	BB	0.4 (9)	P	0.2 (3)	Q

1 - All samples expressed as fibers >5u/cc counted by the standard method recommended in this document. (Latest Available NIOSH Data collected during the years 1969 through 1970).

TABLE III

ASBESTOS FRICTION
NUMBER OF PLANTS = 5

INDIVIDUAL SAMPLES BY OPERATION AND SAMPLE SIZE

OPERATION	HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	LOWEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT
Mixing, Coating & Extruding	32.4	16	S	18.4	16	S	0.1	7	M	0.1	7	M
Forming	16.2	3	U	9.2	4	S	0.3	6	H	0.1	6	H
Hot Pressing	7.3	5	S	6.0	5	S	0.2	7	H	0.1	7	H
Baking	7.4	5	S	7.3	5	S	0.5	2	M	0.1	2	H
Grinding & Sanding	20.5	8	T	16.6	16	S	0.1	10	H	0.1	10	H
Cutting & Drilling	14.4	22	S	14.4	22	S	0.4	12	H	0.1	7	M
Bonding & Riveting	8.7	4	H	1.5	4	H	0.2	1	T	0.1	1	M
Inspection & Packing	11.1	4	S	9.9	13	H	0.1	13	H	0.1	13	H
Miscellaneous	6.4	9	H	6.4	9	H	0.1	5	T	0.1	9	H

1 - All samples expressed as fibers >5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH Data collected during the years 1968 through 1971).

TABLE IV

ASBESTOS FRICTION PLANTS

NUMBER OF PLANTS = 5

MEANS BY OPERATION WITH SAMPLE SIZE ( )

OPERATION	HIGHEST	PLANT	2nd HIGHEST	PLANT	2nd LOWEST	PLANT	LOWEST	PLANT
Mixing, Coating & Extruding	11.0 (16)	S	5.3 (2)	H	4.3 (2)	U	1.9 (7)	M
Forming	6.0 (3)	U	3.6 (4)	S	0.5 (2)	T	0.5 (6)	H
Hot Pressing	4.9 (5)	S	1.5 (2)	U	1.4 (4)	M	0.7 (7)	H
Baking	5.4 (5)	S	3.7 (1)	U	0.6 (2)	M	0.4 (2)	H
Grinding & Sanding	6.3 (4)	U	5.2 (16)	S	2.7 (7)	M	1.1 (10)	H
Cutting & Drilling	14.4 (1)	U	7.7 (22)	S	0.9 (7)	T	0.6 (7)	M
Bonding & Riveting	2.8 (4)	H			0.2 (1)	T	0.1 (1)	M
Inspection & Packing	5.1 (4)	S	3.7 (3)	U	1.0 (4)	M	0.9 (7)	T
Miscellaneous	2.2 (9)	H	1.4 (1)	M	0.8 (3)	U	0.5 (5)	T

1 - All samples expressed as fibers >5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1968 through 1971).

TABLE V

ASBESTOS CEMENT SHINGLE, MILL BOARD AND GASKET
NUMBER OF PLANTS = 3

INDIVIDUAL SAMPLES BY OPERATION AND SAMPLE SIZE

OPERATION	HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT
Warehousing	1.4	3	R	0.4	3	R	0.2	3	R	0.1	1	V
Mixing	16.6	15	R	9.5	6	W	0.5	6	W	0.3	15	R
Forming	6.4	18	R	3.7	3	W	0.1	18	R	0.0	18	R
Curing	2.5	2	R	1.6	2	V	0.4	1	W	0.2	2	R
Finishing	5.4	17	R	4.4	17	R	0.1	7	V	0.1	7	W
Packing	3.8	4	R	1.1	2	W	0.2	2	W	0.1	4	R
Miscellaneous	1.4	2	W	1.2	4	R	0.9	2	W	0.6	4	R

1 - All samples expressed as fibers > 5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1970).

TABLE VI

'ASBESTOS CEMENT SHINGLE, MILLBOARD AND GASKET
NUMBER OF PLANTS = 3
MEANS BY OPERATION AND SAMPLE SIZE ( )

OPERATION	HIGHEST	PLANT	2nd HIGHEST	PLANT	2nd LOWEST	PLANT	LOWEST	PLANT
Warehousing	0.7 (3)	R					0.1 (1)	V
Mixing	4.4 (6)	W	3.8 (15)	R			1.8 (1)	V
Forming	2.6 (3)	W	1.3 (18)	R			0.9 (6)	V
Curing	1.5 (2)	V	1.4 (2)	R			0.4 (1)	W
Finishing	1.9 (17)	R	1.5 (2)	W			1.0 (7)	V
Packing	1.2 (4)	R	0.7 (2)	W			0.5 (2)	V
Miscellaneous	1.2 (2)	W	1.0 (4)	R			0.9 (1)	V

1 - All samples expressed as fibers $>5\mu$/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1970).

TABLE VII

ASBESTOS PAPER, PACKING, AND ASPHALT PRODUCTS

INDIVIDUAL SAMPLES BY OPERATIONS AND SAMPLE SIZE*

PRODUCT AREA	INDIVIDUAL HIGH	OPERATION	INDIVIDUAL LOW	OPERATION
Asbestos Paper	10.9	Asbestos Mixing	0.0	Wood Mixing Paper Making
Asbestos Packing	18.9	Weaving	0.1	Braiding Mixing & Calender Forming Cutting & Trimming
Asbestos Asphalt Products	16.3	Dry Mixing	0.0	Dry Mixing Wet Mixing Forming Finishing Inspection & Packing

* IN THESE THREE ASBESTOS PRODUCT AREAS, INSUFFICIENT DATA PREVENTS TABULATING ENVIRONMENTAL LEVELS INTO HIGHEST AND LOWEST INDIVIDUAL SAMPLE CATEGORIES BY OPERATION. BASED ON A SMALL NUMBER OF PLANTS FOR EACH PRODUCT AREA, ONLY THE HIGH, LOW FOR INDIVIDUAL SAMPLES WERE DETERMINED.

1 - All samples expressed as fibers > 5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1970).

TABLE VIII

ASBESTOS PAPER, PACKING AND ASPHALT PRODUCTS

MEANS BY OPERATIONS AND SAMPLE SIZE*

PRODUCT AREA	HIGH MEAN	OPERATION	LOW MEAN	OPERATION
Asbestos Paper	3.4	Asbestos Mixing	0.7	Miscellaneous
Asbestos Packing	13.6	Weaving	0.2	Mixing & Calender
Asbestos Asphalt Products	2.4	Dry Mixing	0.2	Forming Finishing

*IN THESE THREE ASBESTOS PRODUCT AREAS, INSUFFICIENT DATA PREVENTS TABULATING ENVIRONMENTAL LEVELS INTO HIGHESTS, LOWEST MEAN CATEGORIES BY OPERATION. BASED ON A SMALL NUMBER OF PLANTS FOR EACH PRODUCT AREA, ONLY THE HIGH MEAN AND LOW MEAN WERE DETERMINED.

1 - All samples expressed as fibers $>5\mu$/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1970).

TABLE IX

ASBESTOS INSULATION PLANTS
NUMBER OF PLANTS = 5

INDIVIDUAL SAMPLES BY OPERATION AND SAMPLE SIZE

OPERATION	HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT
Mixing	188.9	11	X	169.7	11	X	0.4	3	DD	0.2	7	R
Forming	134.4	39	X	111.2	39	X	0.0	10	R	0.0	10	R
Curing	23.5	5	X	19.9	5	X	1.5	1	DD	0.1	1	CC
Finishing	208.4	26	X	97.3	26	X	0.1	4	CC	0.1	11	R
Inspection & Packing	92.3	15	X	73.6	15	X	0.1	11	R	0.0	11	CC
Miscellaneous	42.3	24	X	37.5	24	X	0.1	4	CC	0.1	4	CC

1 - All samples expressed as fibers >5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1971).

TABLE X

ASBESTOS INSULATION PLANTS

NUMBER OF PLANTS = 5

MEANS BY OPERATION AND SAMPLE SIZE ( )

OPERATION	HIGHEST	PLANT	2nd HIGHEST	PLANT	2nd LOWEST	PLANT	LOWEST	PLANT
Mixing	74.4 (11)	X	46.3 (7)	Y	4.1 (7)	R	1.7 (2)	CC
Forming	50.6 (39)	X	25.2 (32)	Y	0.7 (10)	R	0.2 (7)	CC
Curing	14.4 (5)	X			1.5 (1)	DD	0.1 (1)	CC
Finishing	39.5 (26)	X	15.0 (17)	Y	1.0 (11)	R	0.9 (4)	CC
Inspection & Packing	22.8 (15)	X	11.0 (19)	Y	0.5 (1)	R	0.3 (11)	CC
Miscellaneous	16.6 (24)	X	2.7 (5)	Y	2.6 (4)	DD	0.2 (4)	CC

1 - All samples expressed as fibers >5μ/cc counted by the standard method recommended in this document. (Latest available NIOSH data collected during the years 1966 through 1971).

TABLE XI

LATEST SURVEY RESULTS

ASBESTOS TEXTILE
NUMBER OF PLANTS = 8

INDIVIDUAL SAMPLES BY OPERATION AND THE SAMPLE SIZE INDIVIDUAL SAMPLE WAS TAKEN FROM

FIBERS/cc 5u

OPERATION	HIGHEST INDIVIDUAL SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND HIGHEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	SECOND LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT	LOWEST INDIVID. SAMPLE	SAMPLE SIZE FROM WHICH INDIVIDUAL SAMPLE DRAWN	PLANT
Fiber Preparation	120.3	12	B	40.9	4	A	1.4	9	K	0.4	9	K
Carding	143.9	30	B	72.2	30	B	0.7	22	K	0.4	40	J
Spinning	40.9	36	K	28.7	43	B	1.0	36	K	0.4	43	B
Twisting	31.1	7	A	25.3	8	K	0.5	8	K	0.2	8	G
Winding	18.4	24	B	17.9	40	K	0.1	40	K	0.0	40	K
Weaving	123.2	57	B	38.5	25	A	0.1	50	K	0.1	50	K
Rope, Wick, Braid & Cord	11.0	3	D	10.3	3	D	0.1	4	K	0.1	3	D
Finishing	5.6	3	G	3.8	28	B	0.2	28	B	0.1	28	B
Miscellaneous	37.0	2	A	22.7	2	A	0.1	45	K	0.1	45	K

TABLE XII

LATEST SURVEY RESULTS

ASBESTOS TEXTILE PLANTS

NUMBER OF PLANTS = 8

MEANS BY OPERATION WITH SAMPLE SIZE ( )
FIBERS/CC >5u

OPERATION	HIGHEST	PLANT	SECOND HIGHEST	PLANT	SECOND LOWEST	PLANT	LOWEST	PLANT
Fiber Preparation	22.3 (4)	A	20.3 (12)	B	7.6 (9)	K	7.4 (5)	J
Carding	27.3 (10)	A	26.4 (30)	B	7.1 (22)	K	6.1 (14)	G
Spinning	12.5 (36)	K	10.9 (11)	A	5.8 (11)	C	3.7 (2)	J
Twisting	14.5 (7)	A	10.7 (19)	B	4.8 (4)	C	3.2 (8)	G
Winding	9.7 (12)	A	5.9 (24)	B	2.8 (10)	J	2.0 (5)	G
Weaving	12.4 (25)	A	10.0 (16)	J	2.5 (11)	C	1.1 (3)	E
Rope, Wick, Braid & Cord	7.1 (3)	D	3.5 (2)	J	2.6 (4)	A	1.3 (4)	K
Miscellaneous	29.9 (2)	A	9.7 (2)	G	2.5 (4)	J	0.2 (2)	E
Finishing	2.5 (3)	G	1.8 (2)	C	1.3 (28)	B	0.1 (5)	E

TABLE XIII

ASBESTOS CONCENTRATION* BY OPERATION
FOR INSULATION WORKERS

Marine Construction Repair	No. of Samples	Actual Arithemetic Means	Recalculated Mean***	Previous Time-Weighted Average***	Recalculated Time-Weighted Average***
Prefabrication	7	30.4	8.7		
Application	25	6.2	2.6		
Mixing	19	21.2	6.4		
General	18	0.6	0.6	9.2	1.8
Tear Out	14	31.5	8.3		
Finishing	19	0.3	0.3		
Light and Heavy Industrial Construction					
Prefabrication	23	10.1	6.6		
Application	36	3.1	2.4		
Mixing	17	4.7	2.9	4.2	2.2
General	19	1.6	1.1		
Tear Out	10	12.8	7.1		
Finishing	16	0.9	0.9		

* Fibers/ml $> 5\mu$ in length
** Summarized from data
*** Personal communication, March 1970 from Balzer & Cooper[4].

TABLE XIV

ASBESTOS CONCENTRATION BY OPERATION*, 1969

Work practice	Environmental conditions	Average asbestos fiber levels: Personal Samples Fibers/ml	Area Samples Fibers/ml	Distance from Source
#1 Asbestos cement	High ceiling room. Louvre venting	2.4	.45	2'
#2 Asbestos cement	Low ceiling room. Poor ventilation	2.6	-	-
#3 Asbestos cement	Access tunnel	6.1	-	-
#4 Asbestos cement	Power house. Low Ceiling, poor ventilation	3.9	2.5	3-5'
Cutting calcium silicate, block, pipe #1	Table and hand saws, in power house - open	1.2	-	-
Cutting calcium silicate, block & pipe #2	Same - in industrial building. Good ventilation.	4.1	-	-
Cutting calcium silicate block & pipe	Apartment house boiler room. No ventilation. Work 3"-18" from breathing zone.	11.5	-	-
Cutting calcium silicate block & pipe #4	Limited ventilation	9.4	1.6	3-4'
Spraying insulation	Turbines in power plant - very high ceiling, good ventilation.	47.7	19.5	3'
			28.0	6'

Fibers/ml $>5\mu$ in length

Notes:

1. Conditions usually variable: Cement mixed dry - applied wet; rapid changes in local ventilation; composition of material may vary; number of men on job may vary.
2. Average of counts (excluding spray insulation): ≥ 5 fibers/ml = 64.5%; 5-12 fibers/ml = 25.5%; >12 fibers/ml = 10.0%.
3. Information prepared by Reitze, Nicholson, and Holaday.(5)

TABLE XV

ASBESTOS PLANT Z - CEMENT PIPE

PERSONAL SAMPLES - SECULAR TRENDS

MEANS BY OPERATION AND SAMPLE SIZE

OPERATION	1967 MEAN	1967 No. OF SAMPLES	1971 MEAN	1971 NO. OF SAMPLES
Warehousing & Mixing	6.2	4	2.3	2
Pipe Forming	2.1	15	1.8	4
Curing	1.3	8	0.4	4
Pipe Finishing	5.0	6	1.7	10
Coupling Finishing	12.8	9	5.3	7
Epoxy	2.6	2	0.9	5
Packing	1.7	6	1.1	7
Miscellaneous			0.5	9

1 - All samples expressed as fibers $>5\mu$/cc by the standard method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XVI

ASBESTOS PLANT S - FRICTION

PERSONAL SAMPLES - SECULAR TRENDS

MEANS BY OPERATION WITH SAMPLE SIZE

OPERATION	1966 MEAN	1966 NO. OF SAMPLES	1969 MEAN	1969 NO. OF SAMPLES	1971 MEAN	1971 NO. OF SAMPLES
Mixing, Coating & Extruding	7.5	24	8.0	6	11.0	16
Forming	5.7	7	0.5	3	3.6	4
Hot Pressing	13.1	15	1.8	3	4.9	5
Baking	9.1	1	2.6	4	5.4	5
Grinding & Sanding	10.8	34	4.7	10	5.2	16
Cutting & Drilling	11.0	31	2.8	8	7.7	22
Bonding & Riveting						
Inspection & Packing	9.6	21	1.9	5	5.1	4
Miscellaneous Friction	6.7	6	1.8	10		

1 - All samples expressed as fibers $>5\ \mu$/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XVII

ASBESTOS CEMENT SHINGLE, MILLBOARD AND GASKET

PERSONAL SAMPLES - SECULAR TRENDS

MEANS BY OPERATION AND SAMPLE SIZE

PLANT W

OPERATION	1967 MEAN	1967 NO. OF SAMPLES	1970 MEAN	1970 NO. OF SAMPLES
Warehousing	8.9	4		
Mixing	8.3	14	4.4	6
Forming	1.8	36	2.6	3
Curing			0.4	1
Finishing	4.3	35	1.5	7
Packing	2.5	22	0.7	2
Miscellaneous	2.3	13	1.2	2

1 - All samples expressed as fibers >5 μ/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XVIII

ASBESTOS, INSULATION PLANT X

PERSONAL SAMPLES - SECULAR TRENDS

THERMAL PIPE

MEANS BY OPERATION WITH SAMPLE SIZE

OPERATION	1967 MEAN	1967 NO. OF SAMPLES	1970 MEAN	1970 NO. OF SAMPLES	1971 MEAN	1971 NO. OF SAMPLES
Mixing	163.0	5	36.2	3	74.4	11
Forming	33.3	18	25.7	3	50.6	39
Curing	2.5	1	31.0	1	14.4	5
Finishing	44.6	3	34.8	4	39.5	26
Inspection & Packing	16.7	7	17.9	3	22.8	15
Miscellaneous			13.8	2	16.6	24
Office Worker						

1 - All samples expressed as fibers >5 μ/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XIX

ASBESTOS, INSULATION PLANT Y

PERSONAL SAMPLES - SECULAR TRENDS

THERMAL PIPE

MEANS BY OPERATION WITH SAMPLE SIZE

OPERATION	1967 MEAN	1967 NO. OF SAMPLES	1970 MEAN	1970 NO. OF SAMPLES	1971 MEAN	1971 NO. OF SAMPLES
Mixing	107.0	3	27.7	2	46.3	7
Forming	98.9	12	24.1	13	25.2	32
Curing						
Finishing	32.2	4	16.8	2	15.0	17
Inspection & Packing	13.3	2	13.0	8	11.0	19
Miscellaneous			21.0	14	2.7	5

1 - All samples expressed as fibers >5 μ/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XX

ASBESTOS TEXTILE PLANT A

PERSONAL SAMPLES - SECULAR TRENDS

MEANS BY OPERATION WITH SAMPLE SIZE

OPERATION	1964 MEAN	1964 NO. OF SAMPLES	1966 MEAN	1966 NO. OF SAMPLES	1970 MEAN	1970 NO. OF SAMPLES
Fiber preparation	13.6	6	9.6	4	22.3	4
Carding	14.5	4	52.2	7	27.3	10
Spinning	11.8	2	15.3	9	10.9	11
Twisting	5.4	7	9.2	8	14.5	7
Winding	9.5	5	13.8	4	9.7	12
Weaving	5.6	11	17.7	15	12.4	25
Rope, Wick, Braid & Cord	0.2	6	6.9	2	2.6	4
Finishing	5.7	2	7.5	1	29.9	2
Miscellaneous						

1 - All samples expressed as fibers >5 μ/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XXI

ASBESTOS TEXTILE PLANT J

PERSONAL SAMPLES - SECULAR TRENDS

MEANS BY OPERATION WITH SAMPLE SIZE

OPERATION	1965 MEAN	1965 NO. OF SAMPLES	1967 MEAN	1967 NO. OF SAMPLES	1971 MEAN	1971 NO. OF SAMPLES
Fiber preparation	6.4	7	15.7	5	7.4	5
Carding	8.1	17	12.6	11	7.8	40
Spinning	7.9	14	27.4	11	3.7	2
Twisting	7.3	20	17.7	9	6.9	35
Winding			3.4	3	2.8	10
Weaving	5.6	47	6.8	12	10.0	16
Rope, Wick, Braid & Cord					3.5	2
Miscellaneous					2.5	4

1 - All samples expressed as fibers >5 μ/cc by the Standard Method recommended in this document.

2 - Information prepared from NIOSH data.

TABLE XXII

ASBESTOS CEMENT PIPE
PERCENT OF SAMPLES LESS THAN OR EQUAL TO 2 FIBERS/CC,
5 FIBERS/CC AND 10 FIBERS/CC LONGER THAN 5u IN LENGTH,
BY PLANT AND OPERATION
( ) = NO. OF SAMPLES
REPRESENTS FIBERS LONGER THAN 5u

OPERATION	PLANT N			PLANT O			PLANT P			PLANT Q			PLANT Z			PLANT AA			PLANT BB		
	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10
Warehousing & Mixing	25	100 (4)	100	0	0 (2)	100	100	100 (3)	100	100	100 (4)	100	50	100 (2)	100	100	100 (5)	100	80	100 (5)	100
Pipe Forming	33	100 (3)	100	33	100 (6)	100	100	100 (4)	100	100	100 (2)	100	75	100 (4)	100	100	100 (6)	100	100	100 (10)	100
Curing	100	100 (3)	100	67	100 (3)	100	100	100 (6)	100	100	100 (1)	100	100	100 (4)	100	100	100 (5)	100	93	100 (15)	100
Pipe Finishing	100	100 (6)	100	100	100 (4)	100	100	100 (6)	100	80	100 (5)	100	70	100 (10)	100	100	100 (9)	100	100	100 (9)	100
Coupling Finishing	100	100 (4)	100	25	50 (4)	100	100	100 (16)	100	80	100 (5)	100	43	57 (7)	71	100	100 (21)	100	90	100 (10)	100
Epoxy	0	100 (1)	100	100	100 (1)	100	100	100 (6)	100	-	-	-	100	100 (5)	100	100	100 (1)	100	83	100 (6)	100
Packing	100	100 (4)	100	100	100 (1)	100	-	-	-	0	0 (1)	100	86	100 (7)	100	100	100 (13)	100	100	100 (6)	100
Miscellaneous	-	-	-	-	-	-	100	100 (9)	100	100	100 (3)	100	100	100 (9)	100	100	100 (8)	100	100	100 (6)	100

- Not Applicable

TABLE XXIII

ASBESTOS FRICTION PLANTS
PERCENT OF SAMPLES LESS THAN OR EQUAL TO 2 FIBERS/CC,
5 FIBERS/CC, AND 10 FIBERS/CC LONGER THAN 5μ
BY PLANT AND OPERATION
( ) = NUMBER OF SAMPLES

OPERATION	PLANT H			PLANT M			PLANT S			PLANT T			PLANT U		
	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10
Mixing, Coating & Extruding	0	50 (2)	100	71	86 (7)	100	13	19 (16)	44	-	-	-	0	50 (2)	100
Forming	100	100 (6)	100	-	-	-	75	75 (4)	100	100	100 (2)	100	67	67 (3)	67
Hot Pressing	100	100 (7)	100	50	100 (4)	100	0	40 (5)	100	-	-	-	100	100 (2)	100
Baking	100	100 (2)	100	100	100 (2)	100	0	40 (5)	100	100	100 (2)	100	0	100 (1)	100
Grinding & Sanding	90	100 (10)	100	57	86 (7)	100	13	56 (16)	94	88	88 (8)	88	0	25 (4)	100
Cutting & Drilling	50	75 (12)	83	100	100 (7)	100	5	32 (22)	64	86	100 (7)	100	0	0 (1)	0
Bonding & Riveting	75	75 (4)	100	100	100 (1)	100	-	-	-	100	100 (1)	100	-	-	-
Inspecting & Packing	54	69 (13)	100	100	100 (4)	100	50	50 (4)	75	86	100 (7)	100	0	67 (3)	100
Miscellaneous	67	78 (9)	100	100	100 (1)	100	-	-	-	100	100 (5)	100	100	100 (3)	100

- Not applicable

TABLE XXIV

ASBESTOS CEMENT SHINGLE, MILLBOARD AND GASKET
PERCENT OF SAMPLES LESS THAN OR EQUAL TO 2 FIBERS/CC,
5 FIBERS/CC, AND 10 FIBERS/CC LONGER THAN 5μ
BY PLANT AND OPERATION
( ) = NUMBER OF SAMPLES

	PLANT R			PLANT U			PLANT W		
OPERATION	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10
Warehousing	100	100 (3)	100	100	100 (1)	100	-	-	-
Mixing	53	67 (15)	93	67	100 (3)	100	50	67 (6)	100
Forming	83	94 (18)	100	100	100 (6)	100	33	100 (3)	100
Curing	50	100 (2)	100	100	100 (2)	100	100	100 (1)	100
Finishing	71	94 (17)	100	86	100 (7)	100	71	100 (7)	100
Packing	75	100 (4)	100	100	100 (2)	100	100	100 (2)	100
Miscellaneous	100	100 (4)	100	100	100 (1)	100	100	100 (2)	100

- Not Applicable

TABLE XXV

ASBESTOS INSULATION PLANTS
PERCENT OF SAMPLES LESS THAN OR EQUAL TO 2 FIBERS/CC,
5 FIBERS/CC, AND 10 FIBERS/CC LONGER THAN 5μ
BY PLANT AND OPERATION
( ) = NUMBER OF SAMPLES

OPERATION	PLANT R			PLANT X			PLANT Y			PLANT CC			PLANT DD		
	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10	%≤2	%≤5	%≤10
Mixing	57	71 (7)	86	0	0 (11)	18	0	0 (7)	0	100	100 (2)	100	67	67 (3)	67
Forming	90	90 (10)	100	0	0 (39)	15	0	9 (32)	13	100	100 (7)	100	100	100 (5)	100
Curing	-	-	-	0	0 (5)	40	-	-	-	100	100 (1)	100	100	100 (1)	100
Finishing	82	100 (11)	100	8	12 (26)	15	6	6 (17)	29	100	100 (4)	100	40	100 (5)	100
Inspection & Packing	100	100 (1)	100	13	27 (15)	40	0	16 (19)	63	100	100 (11)	100	63	100 (8)	100
Miscellaneous	-	-	-	21	46 (24)	54	40	100 (5)	100	100	100 (4)	100	50	100 (4)	100

- Not applicable

TABLE XXVI

ASBESTOS TEXTILE PLANTS
PERCENT OF SAMPLES LESS THAN OR EQUAL TO 2 FIBERS/CC,
5 FIBERS/CC, AND 10 FIBERS/CC LONGER THAN 5u
BY PLANT AND OPERATION
( ) = NUMBER OF SAMPLES

OPERATION	PLANT A			PLANT B			PLANT C			PLANT D			PLANT E			PLANT G			PLANT J			PLANT K		
	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10	%_2	%_5	%_10
Fiber Preparation	0	0 (4)	25	0	17 (12)	58	0	0 (3)	33	0	0 (3)	33	-	-	-	0	0 (2)	50	20	40 (5)	80	22	44 (9)	67
Carding	0	0 (10)	10	0	7 (30)	40	0	0 (4)	75	20	40 (10)	60	-	-	-	14	57 (14)	71	13	38 (40)	80	23	36 (22)	77
Spinning	0	9 (11)	36	5	26 (43)	77	0	36 (11)	91	0	13 (16)	44	-	-	-	0	33 (6)	100	0	100 (2)	100	3	14 (36)	42
Twisting	0	0 (7)	29	0	11 (19)	58	0	75 (4)	100	13	50 (8)	75	-	-	-	38	75 (8)	100	6	26 (35)	89	25	38 (8)	50
Winding	8	33 (12)	50	25	54 (24)	83	0	60 (5)	100	43	71 (7)	86	-	-	-	60	100 (5)	100	40	90 (10)	100	40	53 (40)	78
Weaving	4	16 (25)	44	16	58 (57)	86	36	100 (11)	100	8	33 (24)	88	67	100 (3)	100	8	50 (12)	100	0	25 (16)	56	26	76 (50)	96
Rope, Wick, Braid & Cord	50	75 (4)	100	40	100 (5)	100	-	-	-	33	33 (3)	33	-	-	-	-	-	-	0	100 (2)	100	75	100 (4)	100
Miscellaneous	0	0 (2)	0	33	67 (3)	100	-	-	-	0	50 (6)	83	100	100 (2)	100	0	50 (3)	50	50	100 (4)	100	80	91 (45)	100
Finishing	-	-	-	82	100 (28)	100	50	100 (2)	100	-	-	-	100	100 (5)	100	67	67 (2)	100	-	-	-	63	100 (8)	94

- Not Applicable

TABLE XXVII

Duration of employment and known exposure to Asbestos and the development of X-ray findings of Asbestosis in 232 employees of an Asbestos Insulation Factory, employed sometime in 1941-1945 and examined in 1969-1970.

DURATION OF EMPLOYMENT	TOTAL	X-RAY ASBESTOSIS 0	+ -	1+	2+	3+
1 DAY OR LESS	7	3	0	4	0	0
1 - 7 DAYS	13	4	3	5	1	0
1 - 4 WKS	15	5	3	6	1	0
1 - 3 MOS	35	6	5	23	1	0
3 - 6 MOS	35	8	3	19	5	0
6 - 12 MOS	31	5	3	15	5	3
1 - 2 YRS	48	7	5	25	8	3
2 - 5 YRS	36	3	8	16	6	3
5 - 14 YRS	12	1	0	5	4	2
	232					

ALL EMPLOYEES INCLUDED. EXPOSURES VARIED FROM "NONE" (OFFICE) THROUGH THAT OF MANAGEMENT, ENGINEERING AND SHIPPING, TO THAT OF PRODUCTION EMPLOYEES.

1 - Personal Communication Dr. Irving Selikoff, January, 1971.

TABLE XXVIII

Lapsed period from onset of exposure in 344 deaths among employees of an asbestos insulation factory, employed at some time in 1941-1945 and followed to 1970.

	Years from Onset							
Cause of Death	0-4	5-9	10-14	15-19	20-24	25+		TOTAL
Lung cancer	0	3	8	14	16	18	=	59
Mesothelioma	0	0	0	0	2	2	=	4
G. I. cancer	1	1	6	3	4	3	=	18
Asbestosis	0	2	1	8	8	5	=	24
All other cancer	1	3	9	7	6	5	=	31
All other causes	26	28	30	52	42	30	=	208
TOTAL	28	37	54	84	78	63	=	344

1 - Personal communication Dr. Irving Selikoff, January, 1971.

TABLE XXIX

SUMMARY OF 4 CASE HISTORIES OF EXPOSURE TO ASBESTOS AND SUBSEQUENT DEVELOPMENT OF MESOTHELIOMA

Race	W	W	W	W
Sex	M	F	M	F
Occupational History				
Before asbestos exposure	None	Student	None	None
Asbestos exposure				
Duration of exposure	Unknown	6 weeks	3 years	Unknown - at least several yrs
Type of Work	Engineer	Pipe Insulation	Neighborhood exposure	Family exposure
After asbestos exposure	Unknown	Housewife	Bookkeeper-floor manager	Housewife
Type of asbestos	Chrysotile-amosite-crocidolite	Chrysotile-amosite	Chrysotile-amosite	Amosite
Respirator protection	None	None	NA	NA
Mesothelioma History				
Age at death	74	41	30	52
Site	Peritoneal & Pleural	Right Pleural	Pleural	Left Pleural
Histological diagnosis	Biphasic	(Biphasic) epithelial & fibrous	Biphasic Pleomorphic	- [1]
Lapsed period since exposure	25 years	21 years	19 years	Unknown
Duration of illness	13 weeks	5 weeks	1 year	2 years
Concurrent asbestosis	Pleural Calcification	Grade I by X-Ray	None	None by X-Ray
Smoking history	0	40	20	NA
Duration of smoking history (years)	-	24	5 years Stopped in 1965	-

1 - Personal communication Dr. Irving Selikoff - January, 1971.

TABLE XXX

Observed and Expected Deaths Through December 31, 1969 by Cause and Dust Exposure Score, for 291 Males who Worked Primarily in Non-Asbestos Production and Maintenance Service Jobs and for 1464 Males who Worked Primarily in Asbestos Production and Maintenance-Service Jobs and Retired During 1941-1967, Showing Standardized Mortality Ratios (SMR's)

Accumulative exposure to asbestos in million parts per cubic foot years (mppcfyr)

Cause of Death and International List Number	Limited Exposure			< 125			125-250			250-500		
	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR
All Causes	114	129.9	87.8	365	344.8	105.8	162	139.8	115.9	184	156.7	117.4*
All Cancer (140-205)	22	22.3	98.6	74	56.3	131.4*	23	23.6	97.4	52	26.4	197.1*
Digestive System (150-159)	9	8.2	109.8	27	21.9	123.3	5	8.9	56.2	19	10.0	190.0*
Lung, Bronchus, Trachea & Pleura (162-163)	8	4.8	166.7	18	10.7	168.2	11	4.9	224.5*	16	5.4	296.3*
All Other Cancer	5	9.3	53.8	29	23.7	122.4	7	9.8	71.4	17	11.0	90.9
Cerebral Vascular Lesions (330-334)	15	14.8	101.4	31	41.4	74.3	14	16.2	86.4	15	18.2	82.4
All Heart Disease (400-443)	46	60.8	75.6	168	161.2	104.2	72	65.5	109.9	75	73.3	102.3
Coronary Heart Disease (420)	39	48.9	80.1	129	124.8	103.4	59	52.2	113.0	55	57.8	95.2
All Other Heart Disease	7	12.1	57.8	39	36.4	107.1	13	13.3	97.7	20	15.5	129.0
Diseases of the Respiratory System (470-527)	10	7.7	129.9	26	19.0	162.5*	11	80	137.5	17	8.9	191.0*
Pneumoconiosis & Pulmonary Fibrosis (523-525)	5	-	-	8	-	-	3	-	-	8	-	-
All Other Causes	22	24.5	89.8	66	66.6	99.1	42	26.5	158.5*	25	29.9	83.6

* SMR significantly different from 100 at 5% level.

Source: A Study of the Dose-Response Relationship Between Asbestos Dust and Lung Cancer by Philip Enterline, Pierre DeCoufle and Vivian Henderson (Unpublished Manuscript)

TABLE XXX (continued)

Cause of Death and International List Number	500-750			>750								
	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR
All Causes	77	50.2	153.4*	34	26.2	129.8						
All Cancer (140-205)	18	8.7	206.9*	9	4.5	200.0						
Digestive System (150-159)	6	3.3	181.8	2	1.7	117.6						
Lung, Bronchus, Trachea & Pleura (162-163)	9	1.8	500.0*	5	0.9	555.6						
All Other Cancer	3	3.6	83.3	2	1.9	105.3						
Cerebral Vascular Lesions (330-334)	5	5.6	89.3	3	3.0	100.0						
All Heart Disease (400-443)	36	23.5	153.2*	11	12.3	89.4						
Coronary Heart Disease (420)	24	13.8	127.6	8	9.9	80.8						
All Other Heart Disease	12	4.7	255.3*	3	2.4	125.0						
Diseases of the Respiratory System (470-527)	11	2.8	392.8*	9	1.5	600.0*						
Pneumoconiosis & Pulmonary Fibrosis (523,525)	8	-	-	5	-	-						
All Other Causes	7	9.6	72.9	2	4.9	40.8						

*SMR significantly different from 100 at 5% level.

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