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DRAFT SUMMARY

ESTIMATES OF THE FRACTION OF CANCER INCIDENCE IN THE UNITED STATES ATTRIBUTABLE TO OCCUPATIONAL FACTORS

NATIONAL CANCER INSTITUTE

AND

NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES

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This statement will address the question: "What fraction of the cancer incidence in the United States is attributable in whole or part to occupational exposure to carcinogens in the workplace?" The conventional estimates have been that this fraction is quite small, and figures of between one percent and five percent are often quoted. However, as we will show below, these estimates were admittedly speculative and were incomplete or deficient in several respects. If the full consequences of occupational exposures in the present and the recent past are taken into account, estimates of at least 20 percent appear much more reasonable and may even be conservative.

Basis of the Estimation that 20 Percent of Cancer Deaths will be Associated with Occupational Exposure to Chemicals

I. Asbestos as a Well-Studied Example

The consequences of occupational exposure to asbestos in the United States have only been fully recognized in the past year. According to estimates made by the U.S. Department of Health, Education, and Welfare, between eight and eleven million workers have been exposed to asbestos in the U.S. since the beginning of World War II. Of that total, approximately 1.5 to 2.5 million are presently employed, while the remainder -- between 6.5 and 8.5 million workers -- were formerly employed in environments with significant asbestos exposure, including 4.5 million who worked in shipyards during World War II. Of these workers, approximately four million are believed to have had heavy exposure to asbestos. Based on epidemiological studies of workers, it is estimated that 20-25 percent of heavily exposed workers die of lung cancer, 7-10 percent of pleural or peritoneal mesothelioma, and 8-9 percent of gastrointestinal cancers. These figures are probably underestimates of lifetime risks, because relatively few workers have yet been followed to the end of their normal lifespan. The total fraction of heavily exposed workers likely to die of these cancers is probably between 35-44 percent.

Of the four million heavily exposed workers, approximately 1.6 million are thus expected to die of asbestos-related cancers. Assuming that the excess risk to the 4-7 million less heavily exposed workers is one-quarter of that to the heavily exposed workers, the total number of cancers associated with asbestos in the less-heavily exposed group would be expected to be about 0.55 million, raising the total to about 2.15 million. Since most of these cancers will be manifested in the next 30-35 years, the expected average number of cancers attributable to asbestos per year in that period will average about 67,000. Such numbers would represent about 17 percent of all cancers detected annually in the United States.

II. Other Less Well Studied Examples

Arsenic

Number of workers potentially exposed: about 1,500,000.
Risk ratios*: 3-8 for lung cancer.
Estimated number of excess cancers per year: 2,100 - 7,300.

Benzene

Number of workers potentially exposed: about 2,000,000.
Risk ratios: 2-3 to 7 for leukemia.
Estimated number of excess cancers per year: 240 - 1,400.

Coal Tar Pitch Volatiles and Coke Oven Emissions

Number of workers potentially exposed: about 60,000.
Risk ratios: 2-6 for cancer of the lung, larynx, skin, and scrotum.
Estimated number of excess cancers per year: 160-800.

* Risk Ratio:

The ratio of cancers to the number expected in a normal population. A risk ratio of two means a doubling of the risk.

Vinyl Chloride

Number of workers potentially exposed: about 2,260,000.
Risk ratios; 200,4 and 1.9 respectively for hemangiosarcoma, brain and lung cancer.
Estimated number of excess cancers: 1,940.

A total of 1-5 percent of the cancers occurring in a year will be associated with these four substances..

III. Substances for which excess cancer incidence has been recorded but for which estimates of the number of workers exposed may be less accurate.

Chromium

Number of workers potentially exposed: about 1,500,000.
Risk ratios: 3-40 for nasal cavity and sinus, lung and larynx.

Estimated number of excess cancers per year: 2,400-46,000.

Iron Oxide

Number of workers potentially exposed: about 1,600,000.
Risk ratios: 2-5 for lung and larynx.
Estimated number of excess cancers per year: 1,300-5,000.

Nickel

Number of workers potentially exposed: about 1,370,000.
Risk ratio: 5-10 for lung.
Estimated number of excess cancers per year: 3,800-5,000.

Petroleum Distillates

Number of workers potentially exposed about 3,000,000.
Risk ratios: 2-6 for lung and larynx.
Estimated number of excess cancers per year: 2,400-12,000.

A total of 3-18 percent of the cancers occurring in a year will be associated with these four substances.

IV. Totals.
Thus the total excess incidence would be from 21 to 38 percent. We choose to use the figure 20 percent in order to be conservative.

Summary and Conclusions

1. The oft-quoted estimatess that only 1 percent to 5 percent of total cancers in the United States are attributable to occupational factors have not been scientifically documented.

2. Most cancers have multiple causes: it is an error to attempt to assign each cancer to an exclusive single cause.

3. Because cancer incidence is strongly dependent on age and upon duration of exposure, most cancers resulting from exposure to carcinogens will occur late in life: many epidemiological studies detect only a small fraction of early-developing cancers.

4. Past exposure to asbestos is expected to result in over 2 million premature cancer deaths in the next three decades: this corresponds to roughly 17 percent of the total cancer incidence expected in that period.

5. Reasonable projections of the future consequences of past exposure to established carcinogens suggests that at least 8 other substances may contribute substantially to cancer incidence comparable in their total effect to asbestos.

6. The projections suggest that occupationally-related cancers may comprise 20 percent or more of total cancer incidence in forth-coming decades. (This does not include cancers attributable to ionizing radiation.)

7. Although exposure to some of the more important occupational carcinogens has been reduced in recent years, there are still many unregulated carcinogens in the U.S. workplaces; a number of occupations are characterized by excess cancer risks which cannot yet be attributed to specific agents.

8. There is no sound reason to assume that the future consequences of present-day exposure to carcinogens in the workplace will be less than those of exposure in the recent past.

9. Patterns and trends in total cancer incidence (and mortality) in the U.S. are consistent with the hypothesis that occupationally-related cancers comprise a substantial and increasing fraction of total cancer incidence.

OSHA's Asbestos Standard— A Status Report

OSHA's current standard on asbestos was promulgated on June 7, 1972. It defined "asbestos" as chrysotile, amosite, crocidolite, tremolite, anthophyllite, and actinolite and included every product containing any of these minerals. For regulatory purposes, an asbestos fiber was defined as a particulate form of asbestos, longer than 5 microns, with a length-to-diameter ratio of at least 3-to-1 and a maximum diameter of 5 microns.

The 1972 standard originally established a maximum 8-hour time-weighted-average (TWA) concentration of 5 asbestos fibers per cubic centimeter of air, and a ceiling exposure limit of 10 fibers per cubic centimeter. On July 1, 1976, a further provision of the standard took effect, lowering the permissible 8-hour TWA to two fibers per cubic centimeter of air. The two-fiber limit remains in effect today.

On October 9, 1975, OSHA proposed a new regulation for asbestos. Among other things, the proposal would lower the permissible 8-hour TWA exposure level to 0.5 fibers per cubic centimeter and would reduce the permissible ceiling exposure level to 5 fibers per cubic centimeter for any 15-minute period. This proposal would not apply to the construction industry, which would be required to follow a different standard to be proposed later.

A separate asbestos standard for the construction industry has not yet been proposed, nor have hearings on the 1975 proposal been scheduled. An economic impact statement concerning the proposed revision is nearing completion.

Meanwhile, on December 2, 1975, OSHA asked NIOSH to reevaluate available information on the health effects of occupational exposure to asbestos. Completed in December of 1976, the NIOSH reexamination was forwarded to OSHA in May. Finding no evidence of a "safe" level of asbestos exposure, NIOSH recommended treating asbestos like other carcinogens by allowing only the lowest exposure level detectable by available analytical techniques. NIOSH recommended a 0.1 fiber TWA and a 0.5 fiber ceiling limit for occupational exposures to asbestos.

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Polycyclic Aromatic Hydrocarbons in Soils of a Mountain Valley: Correlation with Highway Traffic and Cancer Incidence

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■ Analyses of soils in the vicinity of a Swiss mountain town show a correlation between the content of polycyclic aromatic hydrocarbons (PAH) and the proximity to a highway. PAH contents range from 300 mg/kg dry soil near the highway to 4–8 mg/kg in the surrounding higher alps. The PAH composition ranges from three- to eight-membered rings and to heavily alkyl-substituted derivatives. The PAH mixtures are far more complex than was assumed in the past and resemble that of automobile exhaust. The low values in town close to industry but remote from the highway, and high PAH values outside of town but near the highway suggest a correlation between automobile traffic and PAH content of soils. These results indirectly suggest also a correlation between the automobile traffic and the observed mortality from cancer in this area.

An epidemological study of a Swiss mountain town has demonstrated a strong correlation between cancer incidence among the residents and the proximity of their residences to the highway (*1*). The town of 3000 inhabitants is located within the 1-km-wide base of a deep valley with predominant winds along its axis and with frequent thermal inversions. It is divided by a 40-m-tall alluvial cone into the older main section with residential housing immediately adjacent to the heavily traveled highway (4000–5000 vehicles per day) and a newer section, about 400 m from the highway and shaded from it by the alluvial cone. Until recently, this section of town was serviced only by a dead-end road.

During the period of the original study (1958–1970), death from cancer was nine times as frequent for residents near the highway. A total of 72 persons died in the old part of town of various forms of cancer, whereas only three cases occurred in the traffic-free area. Cancer mortality near the highway is higher for all groups of residents, without correlation with age, sex, occupation, and smoking habits. Thus, 32 women died; none of them had smoked.

This suggested a link between cancer incidence and environmental carcinogens, associated with the highway traffic, among them petroleum- and coal tar-based road asphalt, tire particles, lubricants, asbestos, and the components of automobile exhaust (lead compounds, polycyclic hydrocarbons, and other reactive chemicals).

We have now surveyed the soils of this valley and of the surrounding mountains for their content of polycyclic aromatic hydrocarbons (PAH), as a possible group of traffic-linked carcinogens.

Samples were taken immediately below the grass within the uppermost humus layer; those representative of the soils near the highways were collected within 1 m from the edge of the road. A dust sample was gathered from a high windowsill in the town church, 50 m from the main highway, and a soot sample was taken from the exhaust pipe of a small car and from the chimney of a residence heated with fuel oil. Most samples were handcarried to the analytical laboratory; they were kept under refrigeration in clean glass containers until extraction. Isolation of the PAH fraction and its final analysis followed the procedure of Giger and Blumer (*2*), except for the more efficient distillation into the mass spectrometer source from a glass capillary with restricted opening (*3*). During each distillation 40 spectra were obtained at 12 eV and inspected on a Finnigan 3200 mass spectrometer with a 6000 data system; the total spectrum for each sample was reconstructed by the summation of all spectra containing appreciable intensities (*3, 4*) of the PAH molecular ions.

Table I presents the total weights of the purified PAH fractions. These values are reproducible (*2*), but they may include the weights of some non-PAH impurities that were not rejected during the separation. Lower values are obtained by UV analysis, since they neglect the presence of alkylated PAH series and of still unidentified PAH components that are evident from the mass spectra.

In our interpretation we consider the 12-eV mass spectra, the UV spectra, chromatographic mobilities, and the relative volatilities as observed in the probe distillation. We believe that our structural assignments (Table II) are sound, since they rest on the correlation of these different and independent analytical parameters. The mass spectra of the PAH fraction demonstrate a nearly uniform composition in all of the soil samples. Unsubstituted hydrocarbons predominate and range from phenanthrene to seven- and eight-membered aromatic ring systems that have not been identified before in environmental samples. Each unsubstituted hydrocarbon is accompanied by alkyl-homologs in gradually decreasing concentrations to C_5 and beyond, with nearly identical abundance patterns in every series. Many isomers, differing in ring arrangement and in position and structure of the side chains, may be present. Some well-known carcinogens (benzo[a]-pyrene, benzanthracene) occur together with other carcinogens and cancer initiators, whose presence in environmental samples is rarely considered (methylchrysenes). In addition, vast numbers of aromatic hydrocarbons are present whose structures are not sufficiently well known to assess their biological effect. Among these there may be many still unknown carcinogens and mutagens.

The composition of natural PAH assemblages is influenced by the processes involved in their formation (*4–6*), and chemical analysis can therefore distinguish between contributions from different sources. For instance, the relative abundance of alkylated PAH derivatives is influenced by the formation temperatures of pyrolytic PAH mixtures. At high temperatures, such as in the coking of coal, only the unsubstituted hydrocarbons are formed or survive, while at lower temperatures, for instance during petroleum formation, highly alkylated products predominate. These compositional fea-

[1] Deceased.

FMSI 05063

tures survive the processing to road asphalt and pitch (7). The mass spectra of the soil hydrocarbons demonstrate an alkylation pattern different from those of coal tar or petroleum (4, 5). The predominance of the unsubstituted hydrocarbons and the gradual decrease in concentration toward the more alkylated members speak for a pyrolytic origin at intermediate temperatures and enable us to rule out a major contribution from petroleum- or coal tar-based road asphalt or from lubricants.

In spite of the general compositional uniformity, we note some subtle differences between samples. The soot from the exhaust pipe of a car contains the same extended PAH series as the soils (a finding in disagreement with earlier analyses obtained with less highly resolving methods). The PAH mixture is depleted in the lowest boiling hydrocarbon series. Apparently, this series is not retained within the hot soot deposits in the exhaust system of the engine. Rather, these compounds pass into the atmosphere, which is consistent with the observation that the phenanthrene and pyrene series are considerably more abundant in the dust from the church, even if that sample has a similar overall composition in terms of ring systems and alkyl-derivatives. Correspondingly, the content of low boiling hydrocarbons in the soils near the highway is intermediate between the soot from the exhaust system of the car and the material collected in the church.

The soot from the chimney of a residence heated with fuel oil has a very different PAH composition. There, alkylated members of lower molecular weight PAH series predominate, and the unsubstituted hydrocarbons are in the minority. Higher ring-number series are present at low concentration or altogether absent. Thus, this soot sample reflects the compositional features that are characteristic for crude oil and its distillates, rather than for high-temperature pyrolysis products, as is the case for car exhaust.

The aromatic hydrocarbon composition of these Swiss soils is very similar to that of recent marine sediments and soils from the U.S. northeast coast. We believe that two different processes, but operating at similar temperatures, have produced a similar set of hydrocarbons. Those in the U.S. samples, and possibly also in the high Swiss Alps, originate in natural fires; they are transported through the troposphere on soot particles and enter the sediments with fallout (4, 8). Extended air transport would result in depletion of the low boiling hydrocarbons. This is observed both in the U.S. samples and those from the high alps. These hydrocarbon assemblages at concentrations near 5 ppm may well represent a worldwide PAH background. The high PAH levels at the bottom of the Swiss valley, on the other hand, cannot be attributed to the same source, especially since their concentration far exceeds the levels in the immediately surrounding alps. The association of such high PAH levels with the proximity to the highway (Table I) suggests that they are produced in internal combustion engines.

A major contribution from other PAH sources linked to the town or the highway can be excluded. Industrial and domestic heating produces some PAH. However, the low levels in town close to industry but remote from the highway, and the high level at the highway outside the town, speak for the association of the PAH production with the traffic. Aromatic hydrocarbons are also associated with the carbon used as filler in automobile tires. A comparison between tire life and gasoline consumption of automobiles suggests that even a minor conversion of the fuel into carbon can produce soot much more rapidly than it would be released by tire wear.

In combination, the geographic distribution of the hydrocarbons, the correlation between structure and processes of formation, and the chemical agreement in the PAH composition of soot in automobile exhaust with the soil hydrocarbons provide a powerful argument that car exhaust is responsible for the observed PAH accumulation in the Swiss Valley.

This work has many consequences. Our new analytical data,

Table I. Total Polycyclic Aromatic Hydrocarbons in Soils and Sediments

	PAH, mg/kg dry wt
Within town	
Center of town (470 m elev.), at highway	110
North end of town, at highway	220
South end of town, at highway	85
South end of neighboring town, at highway	300
Dust from church, center of town, 50 m from highway	100
Outlying section of town, light traffic, 400 m from main highway	21
Outlying section of town, dead-end road, 250 m from main highway, 100 m from foundry	6
Secondary road in village, at road	18
Open country	
At main highway, 750 m south of town	120
300 m from main highway in valley	15
700 m from main highway in valley	5
1000 m from main highway, alluvial plain	5
Alpine soils, side valleys	
Camp ground, 850 m elev., no through traffic	8
Mountain pass, no road, 1200 m elev.	6
Alpine meadow, 1600 m elev.	4
Soils, marshes, and sediments, USA	
Maine, forest, 115 m from secondary road	7
Cape Cod, forest, 750 m from highway, 400 m from secondary road	13
Air base, Cape Cod, sandy soil, 2 m from highway	2
Marsh, Cape Cod, 750 m from highway	19
Buzzards Bay, Mass., marine sediments, surface	4–5

Table II. Aromatic Hydrocarbon Series in Soils

Initial mass	Extent of series	Representative compounds[a]
178	To C_6	Phenanthrene (UV)
202	To C_6	Pyrene (UV), fluoranthene (UV)
228	To C_6	Benzanthracene (UV), chrysene (UV), triphenylene
252	To C_6	Benzo[a]pyrene (UV), benzo[e]pyrene (UV), perylene (UV)
276	To C_9	Anthanthrene (UV), benzo[ghi]perylene (UV)
278	To C_9	Picene, dibenzanthracene, dibenzophenanthrene
300	To C_7	Coronene (UV)
302	To C_7	Dibenzofluoranthene
326	To C_7	Heptacyclic PAH, e.g., dibenzoperylene
350	To C_7	Octacyclic PAH, e.g., benzocoronene
352	To C_6	Tribenzofluoranthene (MS, chrom., dist.)
376	To C_5	Octacyclic PAH, e.g., tribenzoperylene

Relative abundance of series, at bottom of valley: 202 > 178 ≳ 228 > 252 > 276 > 278 > 302 > 300; for other samples, see text

[a] *Structural evidence was derived in all instances from mass spectra, from the chromatographic position, and the relative volatility; "UV" indicates further confirmation from ultraviolet spectra.*

FMSI 05064

obtained with much improved resolution, demonstrate that automobile exhaust and environmental PAH mixtures are far more complex than was assumed in the past. Therefore, earlier analyses now appear much more limited in their power to correlate with, or to predict, public health effects. Numerous additional components of exhaust and of environmental samples must now be considered in their possible roles as carcinogens, tumor inducers or promoters, and mutagens. The demonstrated correlation between highway traffic and the production of carcinogens strengthens indirectly also the correlation between highway traffic and the observed mortality from cancer. The implications for public health, for city and highway planning, and for efforts to control engine exhaust are considerable.

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Determination of Elemental Sulfur by Gas Chromatography

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■ Elemental sulfur was determined by combining electron capture detection with cyclohexane extractions of coal, particulate, and soil samples and with resin sorption of water samples. The sensitivity for sulfur permitted its determination in environmental samples at sub parts per billion levels. The extraction procedures allowed for a minimum of cleanup prior to the rapid and selective gas chromatography.

The usual procedures for the determination of elemental sulfur are reduction to the sulfide or oxidation to the sulfate. These techniques generally lack the selectivity and sensitivity of reported gas-liquid (*1–7*), thin-layer (*8*), and liquid chromatographic (*9*) procedures. These are apparently useful for sulfur determinations, but none has been applied to the quantitation of elemental sulfur in environmental samples.

This paper describes the methodology for the determination of elemental sulfur in stack particulate, soil, coal, and water samples using gas-liquid chromatography for the separation from other components present in the sample and electron capture for the selective and sensitive detection.

Experimental

Apparatus. A Tracor Model 550 equipped with a Ni 63 electron capture detector (ECD) and a Beckman Model GC-5 equipped with a helium discharge ECD were used for the gas chromatography.

Glass columns, 2 m × 4 mm i.d., were packed with the solid supports and liquid phases listed in Table I. These columns were silanized with four injections of 25 μL each Silyl 8 (Pierce Chemical Co.) before use.

A Du Pont 21-490-1 gas chromatograph–mass spectrometer (GC–MS) was used for positive identifications of the elemental sulfur extracted from various environmental samples.

Reagents. Cyclohexane (J. T. Baker Chemical Co.), 98% grade, was further purified by distillation. Sulfur standards used for quantitation were prepared by volumetric dilution of a solution having 10 mg of 99.999% sulfur in 50 mL of cyclohexane.

The 60–100 mesh Florisil (Floridin Co.) used to clean up the sample extracts was calcined at 540 °C by the manufacturer and activated for 5 h at 130 °C prior to use.

Analytical Procedures

Coal, Particulate, and Soil Extractions. One-gram samples of crushed coal which had passed a 60 mesh sieve were Soxhlet extracted for 24 h. Large 35 × 90 mm glass thimbles were used to prevent plugging of the Soxhlet device by the fines from the coal samples. The 90 mL of cyclohexane used for the extraction were then quantitatively transferred to volumetric flasks and diluted to 100 mL. Five-μL aliquots of this cyclohexane solution were subjected to gas chromatography without further cleanup.

Particulate samples were collected from 4-in. sampling ports located approximately half-way up the stack of a local power plant. Three types of samples were collected. Particulates #1 were from the accumulation in the ports. Particulates #2 were collected by drawing the atmosphere from inside the stack through a glass tube containing a glass wool plug. Particulates #3 represented that portion which settled onto horizontal trays placed inside the stack.

Ten grams of particulates were extracted in a Soxhlet for 24 h in 25 × 85 mm glass thimbles using approximately 50 mL of cyclohexane. The cyclohexane was transferred to volumetric flasks and diluted to 50 mL with cyclohexane. Five-μL aliquots of this solution were gas chromatographed without further cleanup.

Ten-gram amounts of local soils were Soxhlet extracted with cyclohexane as above. The extracts were concentrated

Table I. Gas Chromatographic Data for Elemental Sulfur

Liquid phase	Solid support[a]	t_R, min	Column temp, °C	Flow, mL/min
5% OV-210[b]	C	2.3	180	75
4% SE-30/ 6% OV-210[b]	G	5.2	200	75
3% OV-1[b]	C	1.7	120	75
5% OV-1[b]	C	1.8	120	75
1.5% OV-17/ 1.95% OV-210[c]	G	2.5	200	160
10% DC-200[c]	G	3.6	200	160

[a] C is Chromosorb W HP, 80–100 mesh; G is Gas Chrom Q, 100–120 mesh. [b] Tracor Model 550; detector, 340 °C; injector, 220 °C. [c] Beckman GC-5; detector, 310 °C; injector, 240 °C.

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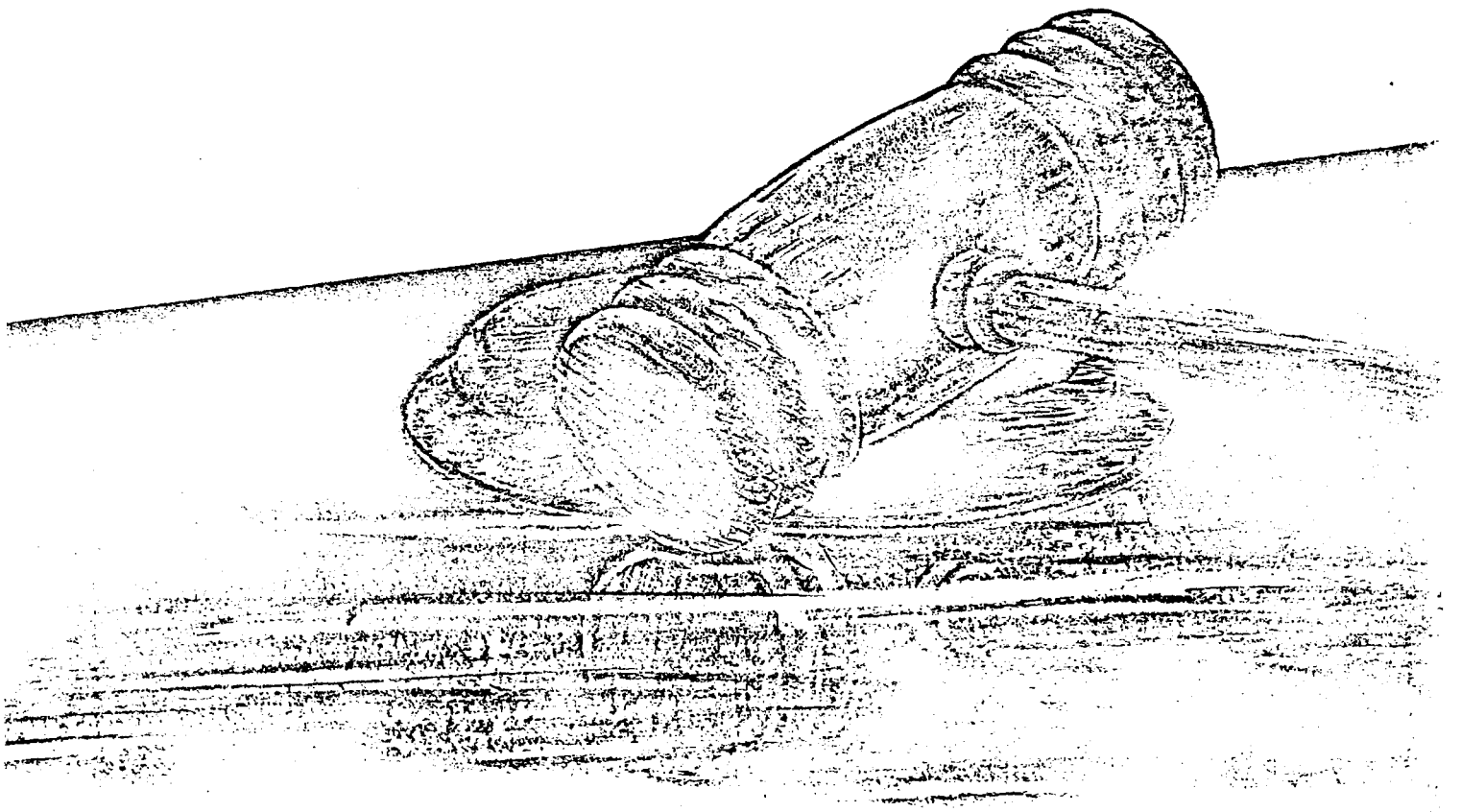
A GUIDE TO THE WORK-RELATEDNESS OF DISEASE

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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A GUIDE TO

THE WORK-RELATEDNESS OF DISEASE

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PREFACE

The goal of the National Institute for Occupational Safety and Health (NIOSH) is to protect the health and safety of working men and women. Within the context of this program are NIOSH efforts that are directed toward the identification of those disease conditions that are causally related to occupation, as necessary prerequisite to their prevention.

This guide is presented primarily as an aid to State agencies and others concerned with occupational disease compensation. The Guide presents one method for assembling and evaluating evidence that may be relevant in determining the work-relatedness of a disease in an individual. Information on five disease-producing agents is presented to illustrate the decision-making process. It should be noted that such information may not be complete and does not necessarily reflect the most recent data regarding health standards and epidemiologic studies.

NIOSH will welcome suggestions for improvement of the Guide based upon experience with its use.

FMSI 05069

ABSTRACT

This Guide discusses various factors associated with establishing the relationship between disease and occupation. Prepared as an aid to State agencies, physicians, and others concerned with workers' compensation for occupational disease, the publication describes a method for collecting, organizing, and appraising medical, occupational, and other evidence with the aim of determining the probable work-relatedness of a given disease. Illustrative material on five disease-producing agents is included. The Guide also contains a list of occupations with potential exposure to selected agents, and other information that may be useful to those with decision-making responsibility in cases of occupational disease.

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ASBESTOS

Introduction

Asbestos is a mineral fiber, and is the name given to about thirty silicate compounds. Of these, only the following 5 are of significance in industry:

- Chrysotile (white asbestos)
- Amosite
- Tremolite
- Crocidolite (blue asbestos)
- Anthophyllite

Chrysotile accounts for about 97 percent of all the asbestos used in this country.

Asbestos is widespread in the environment because of its extensive use in industry and the home. Over 3,000 products contain asbestos.

Because of this wide usage, it may be difficult at times to determine if a disease arising from asbestos is occupational in origin. For example, the air of some relatively new apartment buildings has been found to contain more asbestos fibers than the maximum recommended levels in industry. The source of the fibers in the apartment buildings is the insulating materials used in the ventilating system.

Exposure to asbestos can produce a lung fibrosis called asbestosis. The onset of asbestosis is usually gradual, developing over a period of 10 to 30 years of exposure to significant concentrations of asbestos. Occasionally, from very massive exposures, it may develop more quickly.

Asbestos is also a cancer producing agent (bronchogenic carcinoma, mesothelioma) and can cause certain specific skin diseases (asbestotic subcutaneous granulomatosis and asbestotic cutaneous verruc a). Heavy exposure to dust containing asbestos can cause skin irritation. Epidemiologic studies (experience with groups of people) and animal studies have shown that increased exposure to any of the types of asbestos increases the risk of lung cancer (bronchial carcinoma). This carcinoma appears to be related to the degree of exposure to asbestos, the type of asbestos and cigarette smoking. It is also significant that cigarette smoking in men and women greatly increase the risk of lunk cancer in those who are exposed to asbestos. Smoking is a factor that should be considered when determining whether lung cancer is caused, wholly or in part, by an occupational exposure to asbestos.

Mesothelioma, a rare malignant tumor of the membrane which lines the chest cavity and the abdominal cavity, is occurring with increasing frequency in workers with exposure to asbestos. The development of this tumor apparently is not related to the amount of asbestos inhaled and it is found in persons not having asbestosis. Levels of exposure which are within accepted standards for protection against asbestosis, may not

FMSI 05071

protect against mescthelioma.

An increased incidence of malignancy of the stomach and colon has been reported among insulation workers using asbestos.

Occupations with Potential Exposure to Asbestos

Acoustical Product Makers
Acoustical Product Installers
Air filter makers
Asbestos-cement products makers
Asbestos-cement products users
Asbestos-coatings makers
Asbestos-coatings users
Asbestos-grout makers
Asbestos-grout users
Asbestos-millboard makers
Asbestos-millboard users
Asbestos-mortar makers
Asbestos-mortar users
Asbestos millers
Asbestos miners
Asbestos-paper makers
Asbestos-paper users
Asbestos-plaster makers
Asbestos-plaster users
Asbestos sprayers
Asbestos workers
Asphalt mixers
Automobile repair garage workers
Brake lining makers
Building demolition workers
Carders (asbestos)
Caulking compound makers
Caulking compound users
Clutch facing makers
Cobbers (asbestos)
Construction workers
Crushers (Asbestos)
Fiberizers (Asbestos)
Fireproofers
Firemen
Furnace filter makers
Gasket makers
Heal resistant clothing makers
Insulation workers
Inert filter media workers
Ironing board cover makers
Laboratory hood installers
Laggers
Paint makers
Pipe insulators
Plastics makers
Pump packing makers
Roofers
Roofing materials makers
Rubber compounders
Shingle makers
Ship builders
Ship demolition workers
Spinners (Asbestos)
Talc miners
Talc workers
Textile flameproofers
Textile workers
Undercoaters
Vinyl-asbestos tile makers
Vinyl-asbestos tile installers
Weavers (asbestos)

Medical Evaluation

(Also, See Decision-Making Process)

In addition to the usual medical history, the following should be considered:

1. Any history of diseases of the heart or lung or abnormal tissue growth should be carefully evaluated to determine the relationship between the previous disease and the claimant's present condition.

FMSI 05072

2. A respiratory questionnaire, a sample of which is shown in Appendix, can be useful in evaluating the extent and importance of respiratory symptoms such as:

   - breathlessness
   - phlegm (sputum) production
   - chest pain
   - cough
   - wheezing

Asbestosis

Shortness of breath upon exertion is usually the first symptom, frequently accompanied by a dry cough. This symptom develops after several years of progressive pulmonary fibrosis. As asbestosis progresses, the following signs and symptoms are observed:

- cough with production of sputum
- anorexia (loss of appetite)
- secondary respiratory infections that are difficult to control
- rapid breathing
- repetitive end-inspiratory crackles (crackling sounds heard in the lower part of the lungs through stethoscope when employee completes each of a series of inhaled breaths)
- orthopnea (breathing difficulty in a recumbent position)
- cyanosis (change in skin color to bluish, grayish, slatelike or dark purple)
- decrease of chest expansion
- digital clubbing (rounding of the ends, and swelling of the fingers and/or toes)
- sequelae (other resultant diseases) including cor pulmonale (right heart failure), bronch ogenic carcinoma (lung cancer), stomach or intestinal cancer, or pleural carcinoma (cancer of the membrane lining the chest)

Fibrosis results in alveolo-capillary block (impaired ability of the lungs to transfer oxygen into the blood). This impairment is often more severe than is indicated by chest x-rays.

Mesothelioma

In cases of mesothelioma, the rare malignancy noted above, there may be a long latent period, as much as 40 years, between initial exposure to asbestos and the development of the tumor.

Mesothelioma of the peritoneum (membrane surrounding the abdominal organs) is usually accompanied by abdominal swelling and pain that is not concentrated in a particular area. Signs and symptoms of this type of tumor (which may be associated with asbestos exposure) include:

- weight loss
- obstruction of the bowel
- excessive accumulation of fluid in the abdominal cavity (ascites) is almost always present

This malignant tumor of the peritoneum may spread to the chest cavity.

With mesothelioma of the pleura, complaints include chest pain and breathlessness. Signs and symptoms of pleural mesothelioma include:

- pleural effusion (accumulation of fluid in the space around the lungs)
- the tumor may grow outward through the chest wall in the form of a lump beneath the skin (subcutaneous lump)
- the tumor may spread to involve bone, lymph glands (nodes) mediastinum (area between the right and left lungs), and pericardium (the sac enclosing the heart). As a result, the supraclavicular nodes may become enlarged, ribs may develop tumors, and obstruction of the superior vena cava (major vein draining the upper portion of the body) may occur.
- in addition, pericardial effusion (fluid in the heart cavity) may occur, causing tamponade.

Laboratory

(See Decision-Making Process)

Additional tests which will assist in arriving at a correct diagnosis are:

Chest X-rays

Findings should be classified according to the ILO/UC 1971 Classification of the Radiographs of the Pneumoconioses. (Appendix B)

Findings for asbestosis vary, but the usual picture shows a density in both lungs, with the lower one-third of the lungs involved. In the affected area there is a "ground glass" appearance.

As asbestosis progresses, more and more of the lung is involved, except the apices (tips of the lungs). The X-rays will show gradual obscuring of the border between the lungs and the diaphragm. It may show shadows from the presence of nodules.

X-ray findings usually will show the following as the asbestosis progresses:

- reduced radiographic volume
- formation of cysts combined with increased size of the heart, dilation (enlargement) of the proximal pulmonary arteries (arteries which lead from the heart to the lungs)

Lung Function Tests

Reduced lung capacities and other lung changes do not differ from those resulting from other forms of lung fibrosis, both occupational and nonoccupational. Therefore, the results of lung function tests alone or chest X-ray findings alone do not lead to diagnosis of asbestosis. Asbestos bodies in lymph nodes indicate exposure, but not necessarily asbestosis.

- Asbestosis causes a reduction in the vital capacity (VC) of the lungs and a reduction in total lung capacity (TLC). These capacities are further reduced as the disease progresses.
- The residual volume (RV)of the lungs will be normal or slightly increased.
- The lungs' diffusing capacity for carbon monoxide (D_L) will be reduced.

Other lung function test results which are found in asbestosis include:

- Increased minute ventilation (amount of air breathed in one minute)
- Reduced oxygenation of the arterial blood (arterial hypoxemia)
- Increased static transpulmonary pressures
- Decreased lung compliance

An exercise test will result in an increased amount of air required during physical effort, decreased oxygen in the blood, leading to cyanosis.

Sputum Examination

Asbestos fibers or bodies may be found in the sputum. These indicate asbestos exposure, but not necessarily asbestosis. Where cancer cells are present in the sputum, and chest X-ray findings are normal, bronchoscopy may be necessary to confirm and locate the lung tumor.

FMSI 05075

Skin Tests--The following tests should be performed by the physician to exclude possible infectious diseases:

1. PPD (tuberculin test)
2. blastomycin
3. histoplasmin
4. coccidioidin

Epidemiological Data

Various epidemiologic studies have demonstrated the relationship between asbestos and lung disease, including mesothelioma, in such trades and occupations as mining, insulation installation, textiles, paint, electrical industries, and many other occupations as a result of the widespread use of this substance.

The available information indicates evidence of a dose-response relationship for asbestos exposure and the risk of asbestosis and/or bronchogenic carcinoma. However, much of this information is epidemiolcgical in nature and there is little correlation between epidemiologic data and environmental exposure data. For this reason and others, including the long latent period for the development of carcinomas, it is difficult to develop a specific dose-response relationship. This should be taken into consideration when referring to the following material:

Enterline[1] has reported an exposure-response relationship between asbestos exposure (evaluated as millions of particles per cubic foot years) and the risk of malignant and nonmalignant respiratory disease. Enterline's data indicates that the risk of respiratory cancer increased from 166.7 (standardized mortality ratio) at minimum exposure to 555.6 at cumulative exposures exceeding 750 million particles per cubic foot years. Enterline's data is summarized in a table by NIOSH[2].

Murphy[3] reported that asbestosis was 11 times more common among pipe coverers in new ship construction than in a control group. The first asbestosis was found after 13 years of exposure to an estimated cumulative dose of about 60 million particles per cubic foot years. After 20 years, asbestosis prevalence was 38%. Murphy reported no asbestosis for men exposed to 60 mppcf years but 20% asbestosis in men exposed to 75-100 mppcf years. Murphy reports atmospheric dust concentrations ranged from 0.8-10.0 mppcf depending on the different operations evaluated. Asbestosis was considered present if the worker had at least three of the following: vascular rales in two or more sites, clubbing of the fingers, vital capacity of less than 80% predicted, roentgenography consistent with moderately advanced or advanced asbestosis, shortness of breath on climbing one flight of stairs.

The Pennsylvania Department of Health[4] reported a study of asbestos dust concentrations in two plants (one studies from 1930-1967 and the other from 1948-1968). 64 cases of asbestosis were reported. In the two plants, the study indicates that the air concentrations of particulates were generally less than five mppcf and in many cases less than two mppcf.

Epidemiological evidence is also available relating the development of mesothelioma with exposure to asbestos. Selikoff[5,6] reported 14 deaths

FMSI 05076

from mesothelioma in 532 abestos insulation workers from 1943-1968. No deaths from mesothelioma would be expected from the same number of individuals in the general population.

Evidence of Exposure

Historically, there have been two air sampling and analysis methods to determine the quantity of asbestos in the workplace environment. The earlier light field impinger count method allowed only a measure of the overall dust level in the air rather than focusing on the amount of asbestos fibers in the air. The current fiber count method satisfactorily determines the amount of asbestos fibers in the air. It is performed by collecting airborne materials on a membrane filter and then counting the fibers using a phase contrast microscope at a 400 to 450 times magnification ratio (400X-450X).

Asbestos fibers occur in varying lengths and diameters. As of the publication of the guide, the Occupational Safety and Health Act (OSHA) establishes maximum allowable limits for asbestos fibers greater than five micrometers (um) in length. OSHA limits such asbestos fibers to no more than five fibers per cubic centimeter of air (based on an eight hour time-weighted average exposure).

OSHA further requires that no workers be exposed to more than 10 asbestos fibers (greater than five um in length) during any one 15 minute period of time.

For samples collected by the field impinger count method, results may be compared to the pre-1970 limit (TLV) of five million particles per cubic foot of air.

Occupational exposure to asbestos fibers five um in length or greater, at quantities averaging more than five fibers per cubic centimeter of air or frequent exposures to more than 10 such fibers during a 15-minute period of time is evidence of a possible causal relationship between disease and occupation.

Toxicological

(See References 1-6, Appendix A)

Conclusion

The diagnosis of occupational asbestosis is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.

2. X-ray findings compatible with those indicating asbestosis according to ILO/UC 1971 "Classification of Radiographs of the Pneumoconioses."

3. Pulmonary impairment, particularly a decrease in lung diffusing capacity and an increase in alveolar-arterial oxygen difference,

FMSI 05077

as demonstrated by lung function tests.

The diagnosis of occupational mesothelioma is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.

2. Pathological evidence of mesothelioma.

FMSI 05078

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FMSI 05079

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FMSI 05080

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FMSI 05081

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FMSI 05082

C. SAMPLE RESPIRATORY QUESTIONNAIRE

Use the actual wording of each question. Put X in the appropriate space after each question. When in doubt, record "NO."

PREAMBLE: I am going to ask you some questions mainly about your chest. I should like you to answer 'YES' or 'NO' whenever possible.

	YES	NO	N/A
1. Do you usually cough first thing in the morning or on getting up? (Count a cough with first smoke or on first going out of doors. Exclude throat clearing or a single cough.)	___	___	
2. Do you cough like this on most days for as much as three months each year?	___	___	___
3. Do you cough at work?	___	___	
4. Do you usually bring up some phlegm from your chest first thing in the morning or on getting up? (Count phlegm with the first smoke or on first going out of doors. Exclude phlegm from the nose. Count swallowed phlegm.)	___	___	

FMSI 05083

5. Do you bring up phlegm like this on most days for as much as three months each year? ___ ___ ___

6. In the past three years, have you had a period of (increased) cough and phlegm lasting 3 weeks or more? ___ ___

7. Have you had more than one such period? ___ ___

8. Does your chest ever feel tight or your breathing become difficult? ___ ___

9. Do you get this apart from colds? ___ ___

(If YES: specify...(Interviewer to code)

(a) With Exercise ___ ___

(b) At Work ___ ___

(c) Any Other Time ___ ___

If disabled from walking by skeletal or other physical disability put 'X' here. ___

10. Are you troubled by shortness of breath, when hurrying on the levels or walking up a slight hill? ___ ___

(If 'NO' omit questions 11 and 12)

11. Do you get short of breath walking with other people of your own age on level ground? ___ ___

(If 'NO' omit question 12)

12. Do you have to stop for breath when walking at your own pace on level ground? ___ ___

13. Do you usually have a stuffy nose or catarrh at the back of your nose in the winter? ___ ___

14. Do you have this in the summer? ___ ___

(If "NO" to both questions 13 and 14, go to question 16)

15. Do you have this on most days for as much as three months each year? ___ ___

16. During the past 3 years have you had any chest illness which has kept you off work or from your usual activities for as much as a week? ___ ___

17. Did you bring up more phlegm than usual in any of these illnesses? ___ ___

18. Have you had more than one illness with phlegm like this in the last 3 years? ___ ___

HAVE YOU EVER HAD:

(Give relevant details after each positive answer.)

19. An injury or operation affecting your chest? ___ ___

_____

_____

20. Heart trouble? ___ ___

_____

_____

FMSI 05085

21. Bronchitis? _____ ___ ___

_____

22. Pneumonia? _____ ___ ___

_____

23. Pleurisy? _____ ___ ___

_____

24. Pulmonary Tuberculosis? _____ ___ ___

_____

25. Bronchial Asthma? _____ ___ ___

_____

26. Eczema? _____ ___ ___

_____

27. Dermatitis? _____ ___ ___

_____

28. Pneumoconiosis? _____ ___ ___

_____

29. Byssinosis? _____ ___ ___

_____

30. Other chest troubles? _____ ___ ___

_____

31. Have you ever smoked? _____ ___ ___

(Record 'NO' if subject has never smoked as much as one cigarette a day, or 1 oz. tobacco a month, for as long as one year)

FMSI 05086

32. Age when stopped_____years. Was this in the last month? ___ ___

If 'YES' to 31 and 32, fill in figures below:

	AMOUNT SMOKED	
	NOW	BEFORE STOPPING
Cigarettes/day (Average including weekends)	___	_____
Oz. tobacco/week (handrolled)	___	_____
Cigars/week (large)	___	_____
Cigars/week (small)	___	_____

OCCUPATION (1st Interview Only)

(Record on lines the years in which subject has worked in any of these industries, e.g., 1960-1963)

		YES	NO
33.	Have you ever worked in a dusty job? _____	___	___
34.	In a coal mine _____	___	___
35.	In any other mine? _____	___	___
36.	In a quarry? _____	___	___
37.	In a foundry? _____	___	___
38.	In a pottery? _____	___	___
39.	In a cotton, flax or hemp mill? _____	___	___
40.	With asbestos? _____	___	___

FMSI 05087

41. In any other dusty job? _____ ___ ___

If 'YES', specify _____

_____

42. Have you been exposed regularly to irritating gas or chemical fumes? ___ ___

_____

If 'YES', give details of nature and duration _____

_____

OCCUPATION (Follow-Up only)

43. What is your present job? _____

44. How long have you been doing it? _____

45. What was your previous job in the factory? _____

_____

Taken with minor changes from Operating and Medical Codes of Practice for Safe Working with Toulene Diisocyanate, Health Advisory Committee, British Rubber Manufacturers' Association Ltd.

FMSI 05088

SEP 15 1977

ASBESTOS: THE PHOENIX OF OCCUPATIONAL MEDICINE*

Harvey B. Snyder, M.D.

EXXON COMPANY, U.S.A.

The Phoenix was a large sized, male, fabulous mythical bird with a sweet voice and gorgeous plumage. It was sacred to the Sun, was the only one of its kind and lived for a long time.

At the end of its life it made a nest of the twigs of spice trees on which it died by setting the nest on fire and burning itself alive. From its body or its ashes or the nest which it had fertilized came forth another phoenix, perfect, but first in the shape of a white grub. The young bird, as soon as it was strong enough, took up the charred body of its father, covered itself in spices, took ashes, nest and all and flew to Heliopolis in Egypt where it deposited them on the altar of the Sun. It then died, but not before spontaneously producing an offspring.

This significant symbol of immortality, albeit totally legendary, has a distinct relationship to the magic mineral asbestos which has been said to have a half-life of infinity.

Asbestos has been called the 20th Century mineral for its production and utility has increased 20 times more than petroleum in the past 60 years. This is startling when we recognize that the mineral is practically indestructible and is capable of producing disease after a period of 20 or more years after exposure. Is the prediction of increasing incidence of asbestosis in the future exaggerated?[16]

Asbestos was known to the Ancient World. Plutarch refers to the "perpetual" lamp wicks used by the Vestal Virgins. Pliny writes of a shroud for cremation of nobility and calls asbestos "the funeral dress of kings".

Asbestos use for modern industry was begun in 1868 when 200 tons were produced in Italy. The mineral is of two varieties. Pyroxene is a serpentine fiber, an example of which is chrysotile, and is the bulk of commerical asbestos used. Amphiboles represents the second variety of asbestos and is represented by crocidolite, amosite, anthophylite, tremolite and actinolite.

* Presented at the European Medical Directors Meeting, Brussels, Belgium, 1976.

FMSI 05089

2-15 (16)

The world production is increasing annually. The largest commercial producer is Canada, which has approximately two-thirds of the total world production. Crocidolite, or "blue asbestos", is mined in South Africa and Australia. Amosite is found mostly in South Africa. The largest United States production is in Maryland and California. This variety is anthophylite, $(MgFe)_7Si_8O_{22}(OH)_2$, which has long coarse fibers of low tensile strength and thus of little commercial importance. Most varieties of asbestos have these elements present in their chemical compostion.

Physical Characteristics of Asbestos Fibers

Asbestos has an almost infinite ability to subdivide itself into smaller and more fibers. Asbestos fibers may be 2 inches or more in length but their attrition is unavoidable when separating fibers from non-fibrous rock. This results in fibers so short that the length is hardly greater than the diameter. Similarly, the diameter of bunches of fibers may be 1/32" or more in fairly crudely processed specimens but break down to 100-1000 particles — many too small to be counted with the optical microscope. In an air stream a fiber is generally unpredictable since, rather than conforming to the direction of motion like an arrow, it may spin round and round, collecting other fibers to it forming balls of fluff. In addition, asbestos fibers may divide incompletely at the ends, forming "parachutes" that stay suspended in the air. The size of the fiber is critical. Recent studies have shown that fiber penetration of the lung depends on "diameter and curliness". Harshness thus may be important as it may affect the ability of the fiber to curl.[1]

There are many classifications of diseases of the respiratory system. Asbestos fits into the causative scheme of clinical lung disease as a producer of a type of pneumoconiosis and is also a cause of malignant disease of the lungs and pleura.

Clinical Asbestosis

The characteristic feature of asbestosis is diffuse pulmonary fibrosis (alveolar-capillary block), which contrasts strongly with the nodular fibrosis of silicosis. As a rule, the lesions of asbestosis are most pronounced in the lower and middle lung zones, whereas emphysema often predominates in the upper zones. In addition to these parenchymatous changes, asbestos often causes a proliferation of the connective tissues in the visceral pleura, particularly in the basal and medial regions. There is no correlation between the

FMSI 05090

severity of pulmonary fibrosis and the pleural changes. Severe pulmonary fibrosis may be present without pleural lesions and severe pleural changes with only mild pulmonary fibrosis. In progressive asbestosis there may be gross pleural thickening with horn-like plaques giving the roentgenogram a highly characteristic appearance. Calcified plaque formation, when bilateral, is said to be pathognomonic of asbestosis, but it does not necessarily develop into the malignant changes of mesothelioma.

Theories of Pathogenesis

1. *The Original Theory of Physical Irritation*
   The presence of the asbestos fibers in the terminal spaces causes irritation and damage to the walls of the alveoli, leading to fibrosis.

2. *The Solubility Theory*
   The fibrosis is due to the effects of the silicic acid and metal ions leaching out from the asbestos fibers. Asbestos is relatively more soluble than quartz and coal.

3. *The Autoimmune Theory*
   Two concepts are entertained:
   a. The presence of the asbestos in the lungs and its reaction within the alveolar phagocytes or fibroblasts either produces or localizes abnormal globulin, the presence of which can be discerned by the presence of "rheumatoid factor" in the circulating blood and in the tissues by immunofluorescent techniques.
   b. The effect of the fiber on the pulmonary phagocytes combined with their being entrapped in the respiratory bronchioles may be a factor in the initiation of fibrosis. The lysis, then, of the phagocyte at this site releases a "substance" that is not accepted as "self".

4. *Stagnation of Phagocytes Theory*
   This is similar to (3.) but involves no immunologic mechanism in the tissue destruction and fibrosis. The trapped phagocytes disintegrate and release sclerosing agents, lipids and lipoproteins.

Pathology

Asbestosis occurs in two main anatomical forms:

1. Diffuse Fibrosis — honeycomb (cystic) lung
2. Solid Fibrosis

In either case there appears to be first an aggregation of macrophages in the alveolar spaces, followed by a desquamation of the lining epithelium. Asbestos fibers coated with hemosiderin (asbestos bodies) may be seen in a period as short as 16 days after exposure.

The characteristic lesion in asbestosis is the asbestos body, more appropriately called the ferruginous body. [3, 4, 10]

Asbestos fibers, unlike shorter particles called asbestos dust, are converted into asbestos bodies (ferruginous bodies) by deposition of yellow globules of protein and iron, producing an easily recognized and quite characteristic structure. But, before this happens, many of the thin and sharp asbestos fibers move downwards to the lung bases owing to lung movements and to gravity. This downward movement is likely to be more efficient and more rapid in the healthy lungs of the active young. Once a fiber has become a ferruginous body it is unlikely to move further, as it is usually too large to be phagocytosed or transported by lymphatics and its sharp points are now blunted by the protein globules.

In recent years the term asbestos body has been altered by some to "asbestosis body". This is an unfortunate and unwarranted change, because the great majority of lungs which show asbestos bodies have no pulmonary asbestosis at all, as the findings of many studies clearly demonstrate. This supplies a good reason to call the unique pathological lesion a ferruginous body. Recently Dr. I. J. Selikoff has stated that virtually 100% of New York City adults have ferruginous bodies. Others[5] have also made this assertion. Fortunately there is far less incidence of asbestosis.[6]

There is today an increasing amount of evidence suggesting that less intense grades of pulmonary asbestosis and even minor or trivial amounts are more likely to be associated with pleural rather than with pulmonary changes. Pleural thickening associated with asbestosis is usually calcified and has a significant frequency of malignant mesothelioma of the pleura or the peritoneum.

FMSI 05091

There is a prediction of an increase of limited basal asbestosis, malignant mesothelioma of the pleura, and mesothelioma of the peritoneum.

Signs and Symptoms

The predominant symptom is dyspnea. This is not surprising in view of the earlier statement that the basic pathophysiology is an alveolar-capillary block. Asbestosis may also present with signs and symptoms of pleural effusion.[6, 7, 8]

Lung function tests[9] are highly characteristic and constant, including:

1. Small inspiratory capacity.
2. Decreased MBC but well maintained relative to lung volume.
3. High FEV_1 relative to vital capacity.
4. Reduced vital capacity. This is the most sensitive index of the progression of the disease.
5. Reduced diffusing capacity.
6. Loss of compliance.
7. No significant changes in blood gas studies.

Radiological Findings*

1. Interstitial fibrosis.
2. Pleural thickening—plaques.*
3. Basal findings > Apical findings.
4. Obliteration of the costophrenic angles.
5. Shaggy cardiac shadow.
6. Thickening of interlobar septa.
7. "Ground glass" appearance, composed of linear markings and small discrete opacities in the lower lobes.
8. Honeycomb appearance just above the diaphragm.

* In one study of asbestosis with 1117 cases, there were 150 cases of pleural plaques. They were found to b more prevalent on the left side. When calcification was extensive it was usually bilateral. Calcificatic usually involved the parietal pleura primarily. Calcification of plaques correlated to an exposure of mar years before, often 20 or more.

1. Distinction between asbestosis and other forms of diffuse fibrosis.
   - Scleroderma
   - Idiopathic diffuse interstitial fibrosis
   - Cystic lung — pneumoconiosis
   - Eosinophilic granuloma
   - Sarcoidosis
   - Solid fibrosis — M.P.F.
2. Distinction between other forms of silicatosis (nodular whorled fibrosis)
3. Distinction between ferruginous bodies and hemosiderosis of elastic tissues which may mimic asbestosis. Rheumatoid asbestosis.

In summary, there are a number of short "clinical pearls" which were gleaned from a course on Environmental Lung Disease, given in New York City, 10-12 June 1976.

1. The latent period in asbestosis is long — >20 years.
2. A single exposure may be causative. This gives some credence to the concept of "Bystander's Disease". Laws of dose-response are not necessarily followed.
3. Lung cancer is seldom seen in asbestos workers who do not smoke. It is 92 times more frequent in smokers.
4. Smoking is not related to mesothelioma.[11]
5. Air pollution is not a significant factor in cancer of lung.
6. Compared to non-smokers with the same disease
   a. Fifteen times as many cases of emphysema are smokers
   b. Ten times as many cases of cancer of lung are smokers
   c. Two times as many cases of coronary artery disease are smokers
7. Eighty-five percent of mesotheliomas have a history of asbestos exposure.
8. Sputum cytology is positive prior to any x-ray evidence of carcinoma of lung.

While the first cases of pulmonary fibrosis caused by asbestos inhalation were reported in 1929,[12] and the first case of lung cancer associated with asbestos was reported in 1935,[13] it was not until 1968[14] that epidemiologic studies established the role of asbestos in the development of lung cancer in cigarette smoking asbestos workers.

We should remember a point which Professor Ted Hatch has emphasized on several occasions, "When the dust levels are very high, almost any measurement is good enough to show a change for the better. But when the dust levels are getting low but not low enough, much more attention must be given to using those measurements which are biologically most appropriate." This is almost certainly the position we are likely to have to face in controlling the asbestos dust hazard in the future.

FMSI 05092

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FMSI 05093

EXXON ASBESTOS CONTROL RECOMMENDATIONS*

EXXON CORPORATION

Although the importance of eliminating asbestos from new construction and of minimizing exposure to "old asbestos" has been emphasized in both formal and informal communications between the corporate and regional/affiliate medical departments, no formal policy statement has been issued at the corporate level. In spite of the existence of different local regulations and a recognition of the influence of local customs and economic factors, the need for a corporate position seems evident. Exposure to asbestos must be eliminated or reduced to minimal levels by eliminating its use in new construction where possible and establishing procedures for the handling of asbestos-containing material already in use so there is no health risk to employees or others.

The following recommendations are issued for the guidance of all regional affiliate management and medical personnel:

- Non-asbestos containing materials should be used in all new construction unless their use would create a greater health and safety hazard by failure to meet insulating requirements.
- The Research & Environmental Health Division, Exxon Medical Department, has and will maintain and distribute an updated list of non-asbestos containing products available on a world wide basis or in specific operating areas.
- Existing units should be surveyed by maintenance personnel to identify where asbestos was used in prior construction. This will enable exposure controls to be instituted when these units are to be worked on and there is the possibility of a release of asbestos fibers.
- Protective clothing and respiratory protective equipment must be available to employees who have to work on units when and where exposure to asbestos is a risk.
- Adequate signs must be posted in areas where asbestos is being used or disturbed. These notices should clearly state that a potential health hazard exists and that personnel should avoid the area unless they are wearing necessary protective equipment.
- Acceptable housekeeping procedures must be instituted to minimize the spread of contamination where asbestos-containing materials are being handled. Such procedures include such steps as adequate wetting of the materials before sawing or otherwise disturbing old insulation, the use of vacuum equipment to collect finely divided material, the use of plastic drop cloths, the collection of debris in plastic bags and the burial of old material in adequately marked disposal areas.
- A monitoring program should be established to assess exposures to asbestos at regular intervals.
- Employees who are or have been potentially exposed to asbestos should be identified and included in a medical program including a yearly chest x-ray to determine if adverse health effects are occurring.
- A hazard information sheet should be developed to instruct employees who have to work with asbestos-containing materials about the potential health effects of the material and preventive measures in effect.
- Contractors and their employees who have to work with asbestos-containing materials should be provided with the same information given to Exxon employees and should be required to adhere to the same protective work practices. Their compliance should be assured by adequate monitoring.

* Issued September 9, 1976.

FMSI 05094

ASBESTOS HANDLING PROCEDURE*

Asbestos fibers have been deemed a serious respiratory hazard and control of worker exposure is considered necessary to prevent asbestosis and asbestos-induced neoplasms. Various Exxon affiliates have taken positive action limiting the use of and requiring substitutes for asbestos-containing materials including talc, asbestos insulation materials (all high temperature insulation can be assumed to be asbestos or contain 5% or more asbestos as a binder), asbestos fire blankets, etc. However, asbestos has been used for many years and its presence will be noticed each time insulation must be removed, during turnaround work and sometimes in new construction when asbestos insulation is the only suitable material. At scheduled turnaround, the breaking of insulation and reinstalling same after piping completions, etc. will entail some exposure to asbestos fibers.

Despite the existence of widespread knowledge concerning the potential health hazards of this material, there is significant variation in control measures in effect at the operating affiliates. As a result, Exxon Corporation Medical Department has issued a Policy on the control of asbestos as a guide to the elimination or reduction to minimal levels in the use of asbestos in new construction where possible and to establish procedures for the handling of asbestos-containing material already in use. The following work practices are recommended:

(1) Asbestos-containing cements, mortars, coatings, grout and plaster should be mixed in closed bags or other containers. Manufacturers have high temperature mortars packed in plastic bags so that water can be added directly to the bags through a spout and moistened before opening. Moistened mortar is removed as required and mixing is completed in the shipping container. All waste is replaced in this container and sent to disposal at the end of the shift.

FMSI 05095

(2) Removal of asbestos insulation or ripping-off procedures must be done in a manner which will minimize scatter, dusting or dispersion of asbestos. After removing the wire clips and bands holding the jacket, and the jacket from the insulation, the surface insulation should be wetted. A plastic sheet placed below the piping or structure will serve subsequent clean-up. As many large pieces should be removed as feasible to minimize the number of cuts required.

(3) All waste insulation should be placed in dust-proof bags and all clean-up of asbestos dust shall be performed by vacuum cleaners or by wet cleaning methods. No dry sweeping should be allowed.

(4) There should be no spraying of asbestos material without precautions to limit the spread of overspray.

(5) Where possible, restrict movement into areas where asbestos dust is being generated by roping and signs which read "Keep Out—Authorized Entry Only."

(6) Special protective clothing should be provided to employees working in areas where asbestos dust is being generated. Care should be exercised in laundering and other handling of contaminated clothing.

Effective July 1, 1976 the OSHA time weighted average (8-hour) standard for asbestos is 2 fibers (>5μm in length) per ml of air. A 15-minute ceiling limit of 10 fibers (>5μm/ml) also applies. Since air monitoring may not always be possible the approximate guide in Table II has been prepared as an aid to respirator selection. However, whenever there is any doubt whether these recommendations are adequate or where air monitoring data is available, respirators should be selected to insure that exposure does not exceed recommended standards.

Air monitoring techniques to determine concentrations recommended in the standard require some sophisticated equipment and skill. Accordingly to minimize exposures, a series of procedures are recommended in lieu of air monitoring. These are as follows:

All persons within the work area shall wear personal respiratory protective equipment selected on the basis of potential airborne concentrations of asbestos dust exceeding a TWA limit of 2 fibers/ml as time weighted average with a ceiling limit of 10 fibers/ml.

FMSI 05096

Respirators supplied for the specific application should be cleaned or disposed, maintained and serviced regularly. When not in use they should be stored in dust proof containers, e.g., a sealed plastic bag.

The following classification has been adopted for the purpose of recommending appropriate respirators for the type of asbestos material involved in each operation (See Table 1).

Table 1. Classification of Asbestos Containing Materials

Group I	(Applied dry) Molded asbestos Molded calcium silicate/asbestos Molded magnesia (85%) Molded high temperature insulating block
Group II	(Applied wet) Calcium silicate (plastic) 85% magnesia (plastic) Hard-setting asbestos cement Sprayed asbestos
Group III	Cement asbestos (hard) Roofing sheets Drain pipes Building boards
Group IV	Asbestos rope, yarn tape and sealing materials (not impregnated) Asbestos blankets and clothing Asbestos millboard and paper Filter mats and gauzes
Group V	Resin-bonded and asbestos reinforced material (e.g., friction linings and electrical insulating materials) Asbestos gland packing (impregnated) Compressed fiber jointing Gaskets and preformed packings

Table 2. Respiratory Protection Equipment Selection Guide

Operation	Type of Asbestos Material Used	Respiratory Protection Required: Dry Operation — With Vent.[a]	Dry Operation — No. Vent.	Wet Operation[a] — With Vent.	Wet Operation[a] — No. Vent.
Spraying/Demolition (Confined space)	I,II	D	D	C	D
Tearing Out Insulation (Confined space)	I,II	C	D	C	C
(Outdoor[b])		C	C	Fullface B	C

Table 2. (Continued)

Operation	Type of Asbestos Material Used	Respiratory Protection Required: Dry Operation — With Vent.[2]	Dry Operation — No. Vent.	Wet Operation[3] — With Vent.	Wet Operation[3] — No. Vent.
Housekeeping					
(Confined space)	All groups				
Sweeping	All groups	C	D	Fullface B	C
Vacuuming	All groups	Fullface B	C	A or B	A or B
(outdoor)					
Sweeping	All groups	Fullface B	Fullface B	B	B
Vacuuming	All groups	A or B	Fullface B	A or B	A or B
Application					
(Confined space)	I,II	Fullface B	C	Fullface B	Fullface B
(Outdoor)	I,II	A or B	Fullface B	A or B	A or B
Material Handling					
(Confined space)					
Mixing	I,II	Fullface B	C or D	A or B	A or B
Waste disposal	I,II	A or B	Fullface B or C	A or B	A or B
Charging	I,II	Fullface B	C or D	A or B	A or B
(Outdoor)					
Mixing	I,II	A or B	C	A or B	A or B
Waste disposal	All groups	A or B	Fullface B	A or B	A or B
Charging	I,II	B	Fullface B	A or B	A or B
Grinding & Sanding	III,V				
(Confined space)		A or B	C	None	A or B
(Outdoor)		A or B	A or B	None	—
Cutting & Drilling	III,V				
(Confined space)		None	A or B	None	A or B
(Outdoor)					
Cutting & Drilling	IV				
(Confined space)		None	A or B	None	None
(Outdoor)		None	A	None	None
Sanding & Rivetting	III,V				
(Confined space)		None	A or B	None	None
(Outdoor)		None	A or B	None	None
Application	III,IV,V				
(Confined space)		None	None	None	None
(Outdoor)		None	None	None	None

A — Single use ½ face respirator with or without exhalation valve
B — Air purifying ½ face respirator with replaceable particulate filter
C — Powered air purifying respirator, with high efficiency filter
D — Supplied air respirator with full facepiece, continuous flow or pressure demand

Notes:

1 — Refers to any operation performed outside any confined space where natural ventilation is available

2 — Ventilation refers to any mechanical exhaust or dilution unit. When applied to "outdoor" it refers to mechanical ventilation in addition to already present natural ventilation.

3 — This implies a thorough soaking of the material before any operation is begun. Superficial wetting will not provide adequate control.

FMSI 05097

The following respirators or their equivalent are recommended for use when working with asbestos containing materials.

A. Single Use Respirators (Disposable)

Manufacturer*	Model Number
3M	8710
Willson	1400
A.O.	R1050
Safeline	5350
3M	8800

B. Air Purifying Respirators with Replacement Filter (Reusable)

Manufacturer*	Model Number	Replacement Filter
MSA	459440	459595(10)
MSA	459438	459321
Willson	560	R60
Willson	1210	R10
Willson	1211	R11
A.O.	R5030	R30
A.O.	R6030	R30
Safeline	5265	5905
Welsh	7506	7500-6
Welsh	7406L	7400-6
Cover	1482-F100	F100
CESCO	94R20	R20
Glendale	GR2000	F10

C. Supplied Air Respirators With Full Facepiece

Manufacturer*	Model Number	Operating Mode
SurvivAir	9011-13	Pressure demand
	9811-13	Pressure demand
3M	W840	Continuous flow
Scott	4616-11	Continuous flow
	801548	Pressure demand
MSA	460862	Continuous flow
Pulmosan	A460#10168	Continuous flow
Willson	1810	Continuous flow
Bullard	46	Continuous flow
A.O.	R6099	Continuous flow
Lear Siegler	0501-A	Pressure demand

*Addresses of manufacturers are found in the following section.

NAMES AND ADDRESSES OF MANUFACTURERS AND DISTRIBUTORS

American Optical Corp., Safety Products Div., 100 Canal St., Putnam, Connecticut 06260.

Bullard, E.D., Co., 2680 Bridgeway, Sausalito, California 94965.

Cesco Safety Products, Parmalee Industries, Inc., P.O. Box 1237, Kansas City, Missouri 64141,

Cover, H.S. Co., 107 East Alexander St., Buchanan, Mississippi 49107.

Glendale Optical Co., 130 Crossways Park Drive, Woodbury, N.Y. 11797.

Lear Siegler, Inc., 714 North Brookhurst St., Anaheim, California 92803.

Mine Safety Appliances Co., 400 Penn Center Blvd., Pittsburgh, Penna. 19535.

Pulmosan Safety Equipment Corp., 30–48 Linden Place, Flushing, N.Y. 11354.

Safeline Products, P.O. Box 550, Putnam, Connecticut 06260.

Scott Aviation Division of ATO, Inc., Lancaster, N.Y. 14086.

SurvivAir, Division of U.S. Divers Co., 3323 W. Warner Ave., Santa Ana, California 92702.

3M Company, 3M Center, St. Paul, Minnesota 55101.

Welsh Manufacturing Co., 9 Magnolia Street, Providence, Rhode Island 02909.

Willson Products Div., ESB Inc., P.O. Box 622, Reading, Pennsylvania 19603.

In the U.K. the following respirators have been approved for use in atmospheres containing asbestos fibers. The list is reproduced from the Asbestos Research Council *Control and Safety Guide—No. 1.* Please note that the protection factors recommended in this list are much higher than those recommended by U.S. standards for similar respiratory devices. The U.S. has adopted a more conservative posture on protective factors as a result of a study by Edwin C. Hyatt entitled *Respiratory Protection Factors,* Report No. LA-6084-MS. In this study the protection factors were calculated on the basis of average penetration of DOP obtained from test subjects during five basic exercises designed to simulate a wearer's head and facial movements during actual working conditions.

PART I

Respirators approved for use in asbestos concentrations up to 40 fibers per milliliter (4 fibers/ml for crocidolite). These respirators may substitute A or B in Table II.

1. Duralair dust respirator, fitted with Type R 1100 dust filter

   Safety International
   British American Optical Co. Ltd.
   Radlett Road
   Watford, Herts, WD2 4LJ
   England

2. Filtasafe single cartridge dust respirator, fitted with FC_1 fine dust filter

   Siebe Gorman & Co. Ltd.
   Davis Road
   Chessington
   Surrey KT9 1TW
   England

3. Filtasafe twin cartridge dust respirator, fitted with type FC_1 or FC_2 fine dust filter

   Siebe Gorman & Co. Ltd.
   Davis Road
   Chessington
   Surrey KT9 1TW
   England

4. Baxter Filtrex dust respirator, fitted with an encapsulated filter

   The Leyland & Birmingham Rubber Co. Ltd.
   Leyland, Nr. Preston
   Lancashire, PR5 1UB
   England

5. Baxter Pneuseal dust respirator, fitted with an encapsulated filter

   The Leyland & Birmingham Rubber Co. Ltd.
   Leyland, Nr. Preston
   Lancashire, PR5 1UB
   England

FMSI 05098

6. Martindale Type T dust respirator*

Martindale Protection Ltd.
Neasden Lane
London NW10 1RN
England

7. Martindale Type U dust respirator*

Martindale Protection Ltd.
Neasden Lane
London NW10 1RN
England

8. Martindale Type W dust respirator*
fitted with an encapsulated "Ultron" Type B dust filter*

Martindale Protection Ltd.
Neasden Lane
London NW10 1RN
England

9. Martindale Type X dust respirator, fitted with an encapsulated Type B fine dust filter

Martindale Protection Ltd.
Neasden Lane
London NW10 1RN
England

10. Martindale Type Y dust respirator, fitted with encapsulated Type B filters, part No. 18006

Martindale Protection Ltd.
Neasden Lane
London NW10 1RN
England

11. Safirmatic dust respirator, fitted with a yellow encapsulated dust (Type B) filter

Chapman and Smith Ltd.
Safir Works
East Hoathley, Nr. Lewes
Sussex, BN8 6EW
England

12. Purair dust respirator, fitted with a yellow encapsulated dust (Type B) filter

Chapman and Smith Ltd.
Safir Works
East Hoathley, Nr. Lewes
Sussex, BN8 6EW
England.

FMSI 05099

13. Protective RQ 1000 dust respirator fitted with Safeair No. 54 filter cartridge*

Protector Safety Products (UK) Ltd.
Protector House
719 Banbury Avenue
Slough
Berkshire, SL1 4LL
England

14. Protector RQ 2000 dust respirator fitted with Safeair No. 74 filter cartridge*

Protector Safety Products (UK) Ltd.
Protector House
719 Banbury Avenue
Slough,
Berkshire, SL1 4LL
England

15. Protector RQ 200 dust respirator fitted with either RC 54 or RC 74 filter cartridge*

Protector Safety Products (UK) Ltd.
Protector House
719 Banbury Avenue
Slough
Berkshire, SL1 4LL
England

16. Protector RQ 100 dust respirator fitted with RC 54 filter cartridge*
with exhalation valve identified by the part No. X210/D*
Nos. 13–16 are also marketed under the name "Protectair"

Protector Safety Products (UK) Ltd.
Protector House
719 Banbury Avenue
Slough
Berkshire, SL1 4LL
England

17. Dustfoe 66 dust respirator fitted with fine dust filter, the respirator identified by gold coloring of body and marking thereon of part No. 260846

Mine Safety Appliances Co. Ltd.
Queenslie Industrial Estate
Glasgow G33 4BT
Scotland

18. Angus DM 662 dust respirator fitted with Angus filter cartridges type "B"

    Angus Fire Armour
    George Angus & Co. Ltd.
    Bentham
    Lancaster, LA2 7NA
    England

19. RP 1620 dust respirator fitted with RP 4 or RP 14 fine dust filters

    James North & Sons Ltd.
    P.O. Box 3, Hyde,
    Cheshire, SK14 1RL
    England

20. RP 1610 dust respirator fitted with single RP 4 filter and twin exhalation valves carrying the mark SP 1022

    James North & Sons Ltd.
    P.O. Box 3, Hyde,
    Cheshire, SK14 1RL
    England

21. RP 1660 dust respirator fitted with single RP 4 filter and single exhalation valve carrying the mark SP 1022

    James North & Sons Ltd.
    P.O. Box 3, Hyde,
    Cheshire, SK14 1RL
    England

22. Draeger Normalair dust respirator, 74/815, fitted with a fine dust filter Type DN 815

    Draeger Normalair Ltd.
    Kitty Brewster, Blyth
    Northumberland, NE24 4RH
    England

23. Phoenix dust respirator, fitted with a yellow encapsulated dust (Type B) filter

    Phoenix Accessories (Milnsbridge) Ltd.
    77 Waterloo Road, Pudsey,
    Yorkshire, LS28 8DQ
    England

24. Staysafe dust respirator with yellow encapsulated dust (Type B) filter

    Staysafe & Co., Ltd.
    909 Wolverhampton Road
    Warley
    Worcestershire, B69 9RR
    England

FMSI 05100

PART II

High efficiency respirators for use in asbestos concentrations up to 800 fibers per milliliter (80 fibers/ml for crocidolite)—(see note below). This respirator may substitute Fullface B in Table II.

1. MSA Type 3S High Efficiency Respirator, fitted with an Oval Ultra filter cartridge Part. No. 140-0013

   Mine Safety Appliances Co. Ltd.
   Queenslie Industrial Estate
   Glasgow, G33 4BT
   Scotland

PART III

Positive pressure powered respirators approved for use in asbestos concentrations up to 200 fibers per milliliter (20 fibers/ml for crocidolite)—(see note below). This respirator may also substitute Fullface B in Table II.

1. Martindale Mark II positive pressure powered respirator (fitted with a fine dust filter) Previously listed as the Martindale positive pressure powered respirator

   Martindale Protection Ltd.
   Neasden Lane
   London NW10 1RN
   England

PART IV

High efficiency positive pressure powered respirators upon which no limits of dust concentration have been placed—(see note below). This respirator may substitute C in Table II.

1. Powermask positive pressure powered respirator

   Siebe Gorman & Co. Ltd.
   Davis Road, Chessington
   Surrey, KT9 1TW
   England

NOTE: When selecting the respiratory protective equipment in parts II, III and IV above, due regard should be given to the circumstances in which it will be used which may affect visibility and maneuverability, e.g., work in confined spaces and on scaffolding.

SUBSTITUTES FOR ASBESTOS*

In view of the requirements of the recently issued Exxon asbestos control recommendations, concerning available substitutes for asbestos containing insulating materials, a survey has been made to update our lists of asbestos-free substitutes. Initially only suppliers in the U.S. and the U.K. have been surveyed. Additional surveys will be made in other countries, to complete our files.

The list of asbestos-free products obtained from the U.S. survey reflects products currently available. The U.K. survey results reflect products available as of January, 1973. This list will be updated at a later date. Since many of the quoted products change periodically, both the manufacturer and supplier should be contacted to insure that the appropriate product is available. Complete manufacturer product literature is kept in REHD Files and detailed inquiries on specific products will be available on request.

We would like to acknowledge the assistance and efforts of J.T. Sanderson, who provided information on suppliers in the U.K.

*Issued September 10, 1976 by Exxon Corporation, Medical Department, Research and Environmental Health Division, (J.F. Stelluto).

FMSI 05101

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: PIPE/INSULATION			
J-M Flame Safe	Glass Fiber	0 - 500°F.	Johns-Manville
Aerotube	Closed Cell Foamed Plastic	220°F.	Johns-Manville
J-M Flame Safe AP	Glass Fiber With Thermo Setting Resin	0 - 500°F.	Johns-Manville
J-M Flame Safe AP-T	With Pressure Sensitive Tape Closure System	0 - 500°F.	Johns-Manville
Micro-Lok	Fiber Glass	Max. 650°F.	Johns-Manville
Flame Safe ML	Metal Jacketed Fiber Glass	500°F.	Johns-Manville
Metal-On	Thermo-12 Calcium Silicate With Aluminum Pre-Jacket	1500°F.	Johns-Manville
Temp-Mat Ins.	Glass Fiber	Max. 1200°F.	Pittsburgh Corning
Foam Glas H/T	Sealed Glass Cells	Max. 1200°F.	Pittsburgh Corning
UNI-JAC	Vinyl Jacketed Foam Glass	-60°F. to +450°F.	Pittsburgh Corning
TAB-LOK	Mineral Fibers Between Steel Sheet	1400°F.	Eagle-Picher
Epitherm 1200	Mineral Fibers and Binder	1200°F.	Eagle-Picher
CPR 9005-2	Urethane Rigid Foam	250°F.	UpJohn
TRYMER CPR9545	Urethane Rigid Foam	300°F.	UpJohn
CPR 9006-3	Urethane Rigid Foam	300°F.	UpJohn
CATEGORY: SPRAYABLE			
Kaowool Spray Mix	Alumina-Silica	2300°F.	Babcock & Wilcox
Deck-shield CF	Mineral fibers and Binder		CAFCO/USM
Heat Shield	Mineral Fibers and Binder		CAFCO/USM
Power Shield C/F	Non-Crystalline Refractory Fibers	1400°F.	CAFCO/USM
ISONATE CPR425	Urethane Foam	+225°F.	UpJohn
TRYMER CPR421	Urethane Foam	+300°F.	UpJohn
ISONATE CPR485	Urethane Foam	+225°F.	UpJohn
In the U.K.			
Ceramospray			Cascade Service Co
Insumat			Gloster Saro Co.
Spray			
Mondoval Ltd. - "Mandax D4 Vermiculite" - Need respiratory and eye protection when applying or stripping.			
CATEGORY: INSULATING AND FINISHING CEMENTS			
No. 375 Cement	Hydraulic Setting	1800°F.	Johns-Manville
No. 460 Cement	Mineral Fiber	1800°F.	Johns-Manville
MW One	Mineral Wool Base	1000°F.	Celotex
MW 50	Mineral Wool Base	1800°F.	Celotex

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: INSULATING AND FINISHING CEMENTS (Cont'd.)			
Fibrous Adhesive	Fibers, Silicate of Soda and Fillers	800°F.	Celotex
Super 190	Mineral Wool and Clay	1900°F.	Keene Corp.
Hilcote Finishing Cement	Mineral Wool	1200°F.	Babcock & Wilcox
No. 1 Insulating Cement	Mineral Wool	1700°F.	Babcock & Wilcox
2 Super "66" Ins. Cement	Mineral Fiber	1700°F.	Eagle-Picher
One-Cote Cement	Mineral Fiber with Fillers	1200°F.	Eagle-Picher
Bond Seal (Spray)	Mineral Fibers and Cementitious Binder		CAFCO/USM
Cominco Cement			CAFCO/USM
Ceramic Adhesive	Refractory Fibers and Binder	3200°F.	Cotronics

In the U.K.

Plastic Compositions

"Potrite 6 G" used up to 593°C. (1100°F) - Potters Insulations Ltd.
"Potrite 3G Printing" used up to 593°C. (1100°F) - Potters Insulation Ltd.
"Magnesia 66" used up to 316°C. (600°F) - Chemical & Insulating Co. Ltd.

Hard Setting Compositions

Cape Insulations Co. Ltd.
Wm. Kenyon & Sons Ltd.
Potters Insulation Ltd.

Self-Setting Compositions (based on cements)

Newalls Insulation Co. Ltd.
Cape Insulation Ltd.
Chemical & Insulating Co. Ltd.
Joseph Nadin Ltd.
Potters Insulation Ltd.

CATEGORY: BLANKET-FLEXIBLE			
Microlite and R Series Microlite	Fiber Glass With Thermo-setting Resin	250°F. Faced 350°F. Unfaced	Johns-Manville Johns-Manville
Hilblanket	Silica Based Mineral Wool	0 - 900°F. 0 - 1200°F.	Babcock & Wilcox Babcock & Wilcox
Mineral Fiber Blanket	Mineral Fiber With Metal Fabric	1400°F.	Eagle-Picher
Kaowool Blanket	Kaolin	2300°F.	Babcock & Wilcox
Kaowool 2600	Alumina Silica Fireclay	2600°F.	Babcock & Wilcox
Ceramic Fire Blanket	Refractory Fibers	Max. 3200°F.	Cotronics

In the U.K.

Mattresses, Blankets & Rolls (Mineral Wool)

"Rocksil Mineral Wool" used to 593°C. (1100°F.)
"Eldonite Mineral Wool" - Cork Insulation & Asbestos Co. Ltd.
"Stillite Mineral Wool" - Stillite Products Co. Ltd.
"1100 B/S" 11-lbs/ft.³ used up to 1250°F. - Union Insulation Co. Ltd.

FMSI 05102

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: RIGID BOARD			
Marinite XL	Calcium Silicate and Inert Fillers		Johns-Manville
DU-ALL	Semi-Rigid Fiber Glass	500°F.	Johns-Manville
600 Series Spin-Glas	Rigid Fiber glass		Johns-Manville
Linacoustic R Plenum Liner	Fiber Glass	250°F.	Johns-Manville
Tank Top Insulation	Mineral Composition Board	250°F.	Johns-Manville
Hilboard	Silica Based Mineral Wool Fibers	1200°F.	Babcock & Wilcox
Superglas Board	Mineral Fibers	1800°F.	Eagle-Picher
MT Boards	Mineral Fibers	1050°F.	Eagle Picher
M Board	Alumina Silica Fireclay	2000 - 3200°F.	Babcock & Wilcox
Kaowool 3000	Alumina Fibers	1000°F.	Babcock & Wilcox
Cellofoam	Polystyrene	75°C.	CAFCO/USM
Ceramic Board	Refractory Fibers	3200°F.	Cotronics
CATEGORY: PAPER			
Paper	Alumina Silica Fireclay	2000 - 3000°F.	Babcock & Wilcox
Ceramic Paper	Refractory Fibers	Max. 3200°F.	Cotronics
CATEGORY: PIPE SECTIONS - TUBULAR ROUND			
Micro-Aire Rigid Round	Fiber Glass - Preformed	Max. 250°F.	Johns-Manville
CATEGORY: PIPE SECTIONS - FLEXIBLE ROUND			
Micro-Aire Flx	Fiber Glass	Max. 250°F.	Johns-Manville
CATEGORY: PIPE SECTIONS - RIGID ROUND			
Transite Air Duct	Mineral Fiber Cement		
CATEGORY: BLOCK			
H.T. Banroc	Mineral Fiber With Clay Binder	Max. 1900°F.	Johns-Manville
Glasrock Foam 50 & 30	Fused Silica Foam	Max. 2000°F.	Glasrock Product
Hilblok	Silica Based Mineral Fibers	1900°F.	Babcock & Wilcox
"PV" Supertemp Block	Mineral Fibers Bonded	1900°F.	Eagle-Picher
Epitherm 1200	Mineral Fibers Bonded	1200°F.	Eagle-Picher
Ceramic Fiber Block	Aluminum Silicate Fibers	2300°F.	Eagle-Picher
Kaowool Block	Kaolin	2300°F.	Babcock & Wilcox
Block Insulation	Glass Fiber Mineral Wool	Max. 1900°F.	Babcock & Wilcox

In the U.K.

"Potrite Calcium Silicate" - Potters Insulation Ltd.
"Calcium Silicate" - Joseph Nadil Ltd.
"Calcium Silicate" - Chemical & Insulating Co. Ltd.
All the above may be used at temperatures up to 593°C. (1100°F.)
"F.S. 1100 B/S" 11-lbs/ft.³ used up to 1250°F. - Union Insulation Co.
"Fiberoc HD" 12-lbs/ft.³ used up to 1350°F. - Union Insulation Co.
"Rocksil 144 HD" 9 - lbs/ft.³ used up to 1247°F - Cape Insulation Co.
"LR. 144" 9 - lbs./ft.³ used up to 1247°F. - Cape Insulation Co.
"Stillite Mineral Wool (Stilcaid)" 10 - lbs/ft.³ used up to 1382°F. - Stillite Products Ltd.

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: FLEXIBLE SHEET			
Aerotube	Closed Cell Foamed Plastic Sheet	Max. 220°F.	Johns-Manville
Flex White	Silica Base Mineral Wool	1200°F.	Babcock & Wilcox
Superglas Felt	Mineral Fibers	1800°F.	Eagle-Picher
Superglas Moldable Felt	Inorganic Fibers	1800°F.	Eagle-Picher
Wet Felt	Alumina Silica Fireclay	2300°F.	Babcock & Wilcox
CATEGORY: PIPE FITTINGS INSULATION			
Zeston	PVC Covered Fiber Glass	0 - 500°F.	Johns-Manville
CATEGORY: DUCT INSULATION - FLEX AND NON-FLEXIBLE			
Micro-Aire M/F	Fiber Glass	Max. 250°F.	Johns-Manville
Therm-Lock Closure System	Aluminum Backed Solid Polymer	Max.170°F.	Johns-Manville
Flex-Met	Aluminum	-100°F to 400°F.	Johns-Manville
Linacoustic	Fiber Glass	Max. 250°F.	Johns-Manville
800 Series Spin-Glass	Fiber Glass With Thermo-setting Resin	350°F. Unfaced 250°F. Faced	Johns-Manville
CATEGORY: REFLECTIVE INSULATION - METALLIC			
J-M Reflective	Stainless Steel or Aluminum	750°F.	Johns-Manville
J-M Encapsulated	Stainless Steel, Aluminum Inconel or Titanium	Up to 2200°F.	Johns-Manville
J-M Thermo 12	Calcium Silicate and Aluminum	Up to 1500°F.	Johns-Manville
CATEGORY: INSULATED PANEL			
Metal On	Spin Glass Insulation With Aluminum Sheets	450°F.	Johns-Manville
CATEGORY: SURFACE COATINGS			
Pittcote 300	Glass Fabric Reinforced Mastic	200°F.	Pittsburgh Corning
Pittcote 400	Vinyl Acrylic	140°F.	Pittsburgh Corning
Pittcote 800	Asphaltic	250°F.	Pittsburgh Corning
Pittcote 807AL	Aluminum Pigmented Asphaltic	250°F.	Pittsburgh Corning
Kaowool Cement	Ceramic Fiber Alumina-Silica	1600°F.	Babcock & Wilcox
CATEGORY: BLANKET AND BOARD			
1000 Series Spin-Glass	High Temperature Felted Spun Glass Fiber	Max. 850°F.	Johns-Manville
CATEGORY: LOOSE-BULK			
Bulk Wool	Sandstone and Blast Furnace Slag Melted Together	2300°F.	Babcock & Wilcox
Kaowool Bulk	Kaolin	2300°F.	Babcock & Wilcox

In the UK.

Fiber - (flock filling for mattresses)

Turner Bros. Ltd.
Fibreglass Ltd.

FMSI 05103

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: EXPANSION JOINT AND SEAL INSULATION			
Pitt Seal 111		- 40°F. to + 180°F.	Pittsburgh Corning
Pitt Seal 222		-20°F. to + 180°F.	Pittsburgh Corning

In the U.K.

Packing

Scandura Ltd. - "BBA Glass Rope"
Morganite Ceramic Fibres Ltd. - "Triton Kaowool"
Carborundum Ltd. - "Fibrefax"
Darlington Insulation Co. - "Refrasil"
Johns-Manville - "Cerafelt"

CATEGORY: ROPE AND WRAPPING			
CCM and CGM Cords	Carbon and Graphite	3000°C.	The Carbone Corp.
RCM and RGM Ribbon	Carbon and Graphite	3000°C.	The Carbone Corp.
TCM and TGM Cloth	Carbon and Graphite	3000°C.	The Carbone Corp.
RVC And RVG Felts	Carbon and Graphite	3000°C.	The Carbone Corp.
Fabri-Therm III	Varnished Glass Cloth and tape	Max. 180°C.	General Electric
Silicone Treated Glass Cloth and Tape	Silicone Resin Coated Glass Cloth	Max. 180°C.	General Electric
76599		Max. 180°C.	General Electric
76511A		180°C.	General Electric
Gemat Composites	Glass Web/Polyester Film Laminate	Max. 180°C.	General Electric
Gemat Rope 76520	Glass Yarn	150°C.	General Electric
Fusa-Fab	Cloth Coated With Polyester	2300°F.	Babcock & Wilcox
Kaowool Strip	Kaolin		

In the U.K.

Cloth (Glass) - Mattress-making, temporary lagging, expansion joints, bellows pieces and applications requiring flexibility.

Scandura Ltd.
Fothergill & Harvey Ltd.
Turner Bros. Ltd.

Tape

Scandura Ltd. - "BBA Glass Tape" - various widths, etc.
Turner Bros. Ltd. - "Glass Webbing"
Turner Bros. Ltd. - FORTEX Asbestos Textile, manufactured from liquid dispersed asbestos. Has minimal dust hazard in handling. Acceptable where glass cloth or ceramic fiber is not practicable.

Yarn - Thread

Turner Bros. Ltd.
Fothergill & Harvey Ltd.

CATEGORY: JACKETING			
Jaxsan 600	Thermosetting Polymer	220°F.	Plastic Coatings L
Hydrocal B-11	Gypsum	500°F.	U.S. Gypsum Co.
Ultracal 30	Gypsum	500°F.	U.S. Gypsum Co.
Densite	Gypsum	500°F.	U.S. Gypsum Co.

Product	Composition	Temperature Limit Reported By Manufacturer	Manufacturer
CATEGORY: JACKETING (Cont'd.)			
Pittwrap	Asphalt Impregnated Glass Fabric With Aluminum	Max. 350°F.	Plastic Coatings Inc.
Weatherproofing Jackets	Asphalt Saturated Mineral Fiber	250°F.	Johns-Manville
CATEGORY: PIPE AND BLOCK			
Thermo-12	Hydrous Calcium Silicate	Max. 1500°F.	Johns-Manville
Celo Temp. 1500	Expanded Perlite	1500°F.	Celotex
Carey Temp. Elbow Ins.	Expanded Perlite	1500°F.	Celotex

Suppliers in the United States

Eagle-Picher Industries Inc.
Chemicals and Fibers Division
P.O. Box 779
Cincinnati, Ohio 45201

General Electric
Chemical and Metallurgical Division
Schenectady, New York 12306

Pittsburgh Corning
Mechanical Systems Insulation
800 Presque Isle Drive
Pittsburgh, Pennsylvania 15239

Babcock & Wilcox
Refractories Divison
Augusta, Georgia 30903

Johns-Manville Insulation Center
P.O. Box 5108
Denver, Colorado 80217

Keene Corporation
Insulation Division
P.O. Box 145
Princeton, New Jersey 08540

UpJohn
CPR Division
555 Alaska Avenue
Torrance, California 90503

The Celotex Corporation
P.O. Box 22602
Tampa, Florida 33622

The Carbone Corporation
400 Myrtle Avenue
Boonton, New Jersey 07005

Glasrock Products, Inc.
Fused Silica Division
2210 Marietta Boulevard, N.W.
Atlanta, Georgia 30318

Cotronics Corporation
37 West 39th Street
New York, New York 10018

CAFCO
USM Products Co.
Stanhope, New Jersey 07874

FMSI 05104

Suppliers in the United Kingdom

Mandoval Ltd.
Potters Insulations Ltd.
Chemical & Insulating Co. Ltd.
Cape Insulation Co. Ltd.
Wm. Kenyon & Sons Ltd.
Newalls Insulation Co. Ltd.
Joseph Nadin Ltd.
Cork Insulation & Asbestos Co. Ltd.
Stillite Products Co. Ltd.
Union Insulation Co. Ltd.
Scandura Ltd.
Morganite Ceramic Fibres Ltd.
Carborundum Ltd.
Darlington Insulation Co.
Johns-Manville
Fothergill & Harvey Ltd.
Turner Bros. Ltd.