Georgia-Pacific

intracompany memo

Products

PLAINTIFF'S EXHIBIT

GP-1819

cc: Mr. T. R. Godfrey

to Mr. O. E. Burch location Portland

from Mr. C. W. Lehnert location Tigard

subject Consumer Product Safety Commission date June 6, 1977

Gene, sorry I am so late responding to your letter of 5/17/77. The only two items listed which we would be concerned with are HP 7618 dealing with drywall compounds containing asbestos and HP 77-9 dealing with tremolitic talc. We are no longer manufacturing products with either of these materials, so we shouldn't have to be concerned with the petitions unless they involve products which may still be in the marketplace.

C.W.L.

CWL:jr

FILE

SGP 0009270

UNITED STATES GOVERNMENT

Memorandum

[illegible] WASHINGTON, D.C. [illegible]

[illegible] : The Commission
[illegible] : [illegible]
Through: Michael A. [illegible]
[illegible] : Francine Shacter

[illegible] June [illegible], 1977

[illegible]

Attached for your information is material prepared by the Health Sciences group within Engineering and Sciences on asbestos.

Attachment

SGP 0009271

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Don Clay, Actg. AED for Engineering and Sciences

DATE: June 3, 1977

FROM : Robert M. Hehir, Actg. Deputy AED for Health Sciences RMH for JMH

SUBJECT: Engineering and Sciences Directorate Asbestos Tasks

Per our conversation on May 26, 1977, I am forwarding the following items (Attachments 1-5) which were requested at that meeting and subsequent to that meeting, by Ms. F. Shacter.

1. General Literature Review and Analysis (June Thompson) (Annotated Bibliography)

2. Background Material on Asbestos for F.R. Notice (Rita Orzel)

3. Medical Comment on the Asbestos Problem (Leo Duffy)

4. Risk of Respiratory Cancer Due to Low Level Exposure to Asbestos from Spackling and Joint Taping Compounds (Steven Bayard)

5. Difficulties Associated with Testing for Asbestos in Consumer Products (Gale Wyer)

Attachments: 5

*U.S. GOVERNMENT PRINTING OFFICE: 1974 733-533/3604 1-3

SGP 0009272

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Don R. Clay, Act. AED for Engineering Sciences

DATE: June 2, 1977

THRU : Robert M. Nehir, Ph.D, Dep. AED for Health Science

FROM : Leo T. Duffy, M.D., Office of Dep. AED for Health Science

SUBJECT: Medical Comment on the Asbestos Problem

There is ample evidence concerning the role of asbestos fibers as the etiological agents in the production of certain respiratory and gastro-intestinal pathology, including neoplasms. This may occur in relation to occupational, non-occupational, or intermittent exposures. It has also been shown that such pathology may be induced in either chronic long-term, or short-term exposures, and in varying concentrations. Therefore, no known threshold level of exposure is known, or considered, to be safe.

The use of consumer products, which may involve the liberation of "free" asbestos fibers, cannot be presumed to be safe, although it is recognized that because of the variablity of individuals, there may also be a varying susceptibility to any adverse effects.

It is considered that any retrospective action taken to cause a major impact in the reduction of consumer asbestos products, might very well aggravate the situation. However, the prudence of preventing further manufacture, sale or distribution of "free" asbestos consumer products, is without question.

*U.S. GOVERNMENT PRINTING OFFICE: 1974 733-533/3604 1-3

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Robert M. Hehir, Actg. Deputy AED for Health Sciences

DATE: June 2, 1977

FROM : E. June Thompson

SUBJECT: Literature Review and Analysis of Asbestos

Attached is a review and analysis covering the following:

- Definitions
- Characteristics of Asbestos
- Disease Associated with Asbestos Exposure
- Occupational Exposure
- Non-occupational Exposure
- Measurement Problems
- Other Pertinent Regulations
- Disposal
- Conslusion

Also attached is the annotated bibliography, requested by Ms. Francine Shacter, to be treated as an addendum to the Federal Register Notice.

Attachment

SGP 0009274

Definitions of Asbestos

For the sake of clarity and understanding by the public, medical community and regulated trade, it is recommended that the various agencies utilize a uniform definition of asbestos.

In the October 9, 1975 Federal Register Notice of Proposed Rule-making, OSHA has proposed to revise the definition of asbestos as follows:

(1) "Asbestos" includes fibrous chrysotile, amosite, crocido-lite, tremolite, anthophyllite and actinolite, and every product containing any of these minerals.

(2) "Asbestos Fiber" means a particulate form of asbestos, longer than 5 micrometers, with a length-to-diameter ratio of at least 3 to 1, and with a maximum diameter of 5 micrometers. (This reflects concern for the morphology and toxicity of the regulated substance rather than its geologic or mineralogic origin.)

OSHA's proposed definition is also consistent with that proposed by the American Industrial Hygiene Association, February, 1975.

Further, the "asbestos fiber" definition proposed by OSHA reflects fiber sizes known to be respirable and which can be counted using phase contract microscopy.

As of May 1977 this definition has not been finalized.

The definition currently in effect is:

(1) "Asbestos" includes chrysotile, amosite, crocidolite, tremo-lite, anthophyllite and actinolite.

(2) "Asbestos fibers" means asbestos fibers longer than 5 micro-meters.*

Draft Definition of Free Asbestos-containing Products

"Any products containing fibrous asbestos which is not bound, woven, or otherwise 'locked-in' by resins or other bonding agents, or those from which fibers can readily become airborne with any reasonable or foreseeable use."

*F.R. Vol. 19, no. 125, pp. 23543-23545, June 27, 1974.

Characteristics of Asbestos

One characteristic of asbestos minerals is their fibrous, spicular or needle-like shape, with a diameter-to-length ratio of 1:3 or longer. (This shape is not characteristic of the "platy" particles of talc or of the crystalline, but amorphous, silica particles of quartz, flint or sand, or the amorphous particles of soot or of ferric oxide.)

Another characteristic that the asbestiform minerals have in common are their ability to be broken into finer fibrils. Asbestos fiber refers to a particular crystallization of common nonfibrous rock-forming minerals, resulting in long fiber bundles, which continue to break down during crushing or processing into fibrils of smaller and smaller diameter until near molecular diameters are reached, while still maintaining relatively great elongation. The use of an aspect ratio (length to width) of 3 to 1 as the lower limit of a fiber (as defined in the Federal Register, NIOSH, and so forth), most certainly refers to fragments or pieces of much longer, previously recognizable asbestos fibers, since it is obvious that any other nonasbestos, nonfibrous minerals, such as feldspars, pyroxenes, amphiboles, calcite, gypsum, etc., could not be considered to have become fibrous on the basis that, during processing, they are reduced to particles or fragments which exceed an aspect ratio of 3 to 1. Many such minerals normally occur in elongated prismatic or acicular crystals, which have a tendency to elongate as they are subjected to attritioning but have little tensile strength, tend to be quite brittle, and are therefore not fibrous.

Other characteristics that the asbestos minerals have in common are their qualities of great elongation, resistance to heat and to biotransformation, flexibility, high-tensile strength and spinability.

The asbestos minerals all have nonasbestos counterparts of the same chemical composition and crystal structure which are more abundant and widespread than the asbestiform variety but the crystallization of these normally nonfibrous minerals into fibrous forms is a rare occurrence in nature.

The asbestiform varieties, as identified in OSHA's Asbestos Dust Standard (29 CFR 1910.93a), are as follows:

SGP 0009276

TABLE 1. - "Asbestos" minerals as listed in Federal Register

Asbestiform variety	Chemical composition	Nonasbestiform variety
Serpentine group: Chrysotile.....	$Mg_3(Si_2O_5)(OH)_4$.....	Antigorite, lizardite.
Amphibole group:		
Crocidolite.....	$Na_2Fe_3Fe_2(Si_8O_{22})(OH)_2$....	Riebeckite.
Amosite.....	$(Mg,Fe)_7(Si_8O_{22})(OH)_2$.....	Cummingtonite.
Anthophyllite asbestos.....	$(Mg,Fe)_7(Si_8O_{22})(OH,F)_2$...	Anthophyllite.
Tremolite asbestos.....	$Ca_2Mg_5(Si_8)_{22})(OH,F)_2$.....	Tremolite.
Actinolite asbestos.....	$Ca_2(Mg,Fe)_5(Si_8O_{22})(OH,F)_2$	Actinolite.

Chrysotile, the serpentine white asbestos is most abundant, and most widely used by industry (95%). Industry also uses the amphibole forms, blue asbestos (crocidolite) and amosite. Other amphibole asbestos forms, of less industrial importance, are tremolite, actinolite and anthophyllite, but some of these may appear in deposits of talc, and persist in marketed products.

Another characteristic of asbestos which is of significance, particularly in view of the petitions which have been reviewed by the Commission, is the transportability and/or suspendability of the fibers. In Rohl and Selikoff's study of asbestos fibers in consumer patching compounds, it was observed that significant concentrations of asbestos remained suspended in the air 15 minutes after the mixing (of the spackling compound) had ceased and was detected in the air 15 meters away in an adjoining room. In addition, cases of mesotheliomas have been reported following exposure to asbestos transported into the home on clothes dusty with asbestos fibers.

The degree to which a fiber will be inhaled and deposited into the respiratory system will depend to a great extent upon the fiber morphology and aerodynamics. Timbrell (1965) notes that for a fiber, deposition in the respiratory system is dependent upon free-falling speed, and further, that this speed is dependent upon diameter and not upon length. This explains why the largest compact particles found in the lungs are about 10 micrometers in diameter, but asbestos fibers of 50 micrometers to 200 micrometers in length may be found in the lung. Fibers longer than 200 micrometers will be trapped before reaching the lung. If a fiber is less than 3.5 micrometers in diameter, it will likely escape upper respiratory deposition and penetrate deeply into the lung.

In addition to fiber diameter, shape plays an important role. Those fibers which are symmetrical have a greater chance of penetrating. For example, the characteristically rectilinear shape of amphibole fibers appears to cause those fibers to penetrate more deeply into the lung (Timbrell 1973) than curly fibers.

With respect to fiber length, the efficiency of removal of fibers from the airstream through interception by nasal hairs increases with an increase in the fiber length (Timbrell 1965). Heavy deposition of long fibers not previously intercepted can be expected at the branching of respiratory bronchioles.

These characteristics have been used to explain the differences in mesothelioma development in the Northwestern Cape area after exposure to crocidolite dust when compared to relatively low tumor occurrence in the Transvaal, where both crocidolite and the closely related amosite are mined.

Electron microscopy has shown a consistent difference in the size of fibers in specimens obtained from these two areas. Transvaal fibers were found to be three times the diameter and three times the length of Northwestern Cape fibers. Air sampling has confirmed the electron microscopic findings and indicates for the Transvaal less liberation of respirable fibers, higher fiber settling rates and shorter times available for inhalation, and less efficient deep lung penetration when compared with the Northwestern Cape fibers (Timbrell 1973).

Harris and Fraser (1976) have developed a mathematical model for deposition of fibers in the human respiratory system. The model is based upon the aerodynamic behavior of thin straight rods. Through the use of this model, they predicted that as rod length increases, deposition in the nasopharyngeal compartment increases. They suggest that more than 90% of rods 200 micrometers or more in length will be deposited in the nose, leaving 1% of rods 200 micrometers long and 1 micrometer in diameter to be deposited in pulmonary spaces, and these data were validated by comparison with observations of fibers in human lungs reported by other authors.

Lippmann et al. (1976) used a hollow bronchial cast of a human lung to determine the deposition sites for fibrous particles. The casts allowed for measurement of deposition at bifurcations and along the lengths of airways from the larynx down to 2 mm bronchi. The case was coated with a thin layer of silicone oil so that particles adhere to the surfaces which they strike. The tests covered a range of aerodynamic particle diameters from 0.2 to 8.0 micrometers and inspiratory flowrates of from 15 to 60 liters per minute. Impaction of the particles was predicted to be the predominant deposition mechanism, and this was confirmed by the experimental data. In almost every case, bifurcations had a greater concentration of particles than length regions of the cast.

Diseases Related to Asbestos Exposure

The fibrogenic hazards of asbestos in the workplace were recognized in the early part of this century with increasing numbers of cases of pulmonary fibrosis associated with occupational exposure to asbestos. The name "asbestos" is given to this disease in 1927 by W. E. Cooke. Asbestosis, which is a classic occupational disease, is the progressive, restrictive pulmonary fibrosis associated with inhalation of asbestos fibers.

Certain malignancies are also related to exposure of asbestiform minerals. Both animal bioassay data and epidemiological studies support this conclusion.

All commercially available forms of asbestos which have been tested are carcinogenic in mice, rats, hamsters, and rabbits.

Some conclusions from a Review (Huff) of the major literature on asbestos are:

- Under experimental conditions, asbestos is pathogenic in animals and most is known to be carcinogenic in humans, causing lung cancer, mesotheliomas, and pleural lesions.
- The mechanisms of pathogenicity are poorly understood. The etiological significance of fiber size or type is controversial, and the physicochemical properties of various asbestos types as related to biological effects are incompletely defined.
- Little is known about the clearance rates of asbestos from tissues, the transport of asbestos within the organism, or the metabolic alteration of asbestos in the body.
- Animal models necessary to accurately predict the potential effects of asbestos in humans have not been developed.
- Quantitative dose-response relationships between asbestos inhalation and related diseases have not been determined for animals or humans, and minimal exposure levels required to cause disease are not known.
- Malignancies arise primarily after long-term occupational exposure of 20 years or more; however, they also reportedly result from indirect, nonoccupational exposure.
- Pathogenic synergism between asbestos and smoking as well as environmental pollutants is poorly defined. Apparently tobacco smoking increases the incidence of asbestosis and lung cancer among asbestos workers.
- Available data indicate that asbestos is a widespread environmental pollutant.

Occupational Exposure

Reports of pulmonary fibrosis or lung scarring, from inhalation of asbestos dust in factories began to appear in the literature in the early part of this century and by 1927 this lung scarring disease was known as "asbestosis". (Newhouse)

In 1930, the British Home Office Survey by Merewethier and Price found that approximately half of a population of asbestos factory workers were suffering from pulmonary fibrosis, leading to the introduction of the Asbestos Factory Regulations of 1931. (Ibid.) The objective of the Regulation, implemented in 1933, was to reduce the risk of asbestosis.

It was recognized, however, that in addition to asbestos-induced lung scarring, there was also increasing evidence of cancer associated with asbestos exposure. Some workers whose deaths were attributed to asbestosis were also found to have had lung cancer. (Nicholson)

By 1960, through epidemiological studies, an association between asbestos exposure and mesothelima had been demonstrated (Wagner) and was soon substantiated by additional epidemiological studies (Newhouse, Selikoff) and clinical diagnosis.

In 1968, the Committee of the British Occupational Hygiene Society evaluated worker exposure data in a large asbestos textile mill dating from implentation of the Asbestos Regulations of 1933 which indicated that there was "comparatively little clinical and/or roentgenological evidence of asbestosis" at that factory in a survey completed in 1966. Based on that data, the Committee concluded that by setting a dust exposure level at 100 fiber-years/ml (or 2 fibers/ml for 50 years) it would provide a full working lifetime without substantial risk of developing asbestosis. (OSHA, Oct. 1975)

The National Institutes of Occupational Safety & Health Administration (NIOSH) relied heavily on the British Standard in its recommendation for promulgation of the asbestos standard set by OSHA in 1972.

Subsequent to the implementation of the U.S. permanent asbestos standard on 1972, results of a new evaluation of the earlier British factory worker population showed many abnormal x-ray findings of the lung and pleural.

It is reported that there is now an excess lung cancer mortality rate among workers who became employed at the textile mill after 1933 and whose lack of clinical or x-ray evidence of asbestosis led to the development of the British asbestos standard.

Another report of an ongoing study of workers (and their family members) in a factory producing amosite asbestos between 1941 and 1954, when the factory closed , has shown a high incidence of x-ray abnormalities and 2 pleural mesothelioma deaths have thus far been identified.

It is also reported that clinical data are becoming available concerning asbestos lung scarring in individuals exposed at much lower than occupational levels.

It therefore appears that an exposure thought sufficiently low to reduce the risk of asbestosis has not prevented pathogenic changes, including lung cancer and mesothelioma.

OSHA is therefore re-evaluating the 1972 asbestos standard based on information about "...the occurrence of asbestos cancer among individuals exposed to low levels of asbestos, as in environmental circumstances or to brief or intermittent exposures to higher levels." (OSHA, Oct. 9, 1975)

Non-occupational Exposure

Direct and indirect evidence exists that individuals, other than those working directly with asbestos minerals are being exposed to asbestos. Firstly, asbestos fibers can be demonstrated at autopsy in the lungs of persons not occupationally exposed (Thompson). Substantial evidence exists which show that human lungs harbor thousands or millions of fibers. Some of these are chrysotile asbestos, and other types of asbestos minerals probably are there also. Studies indicate though that the numbers of fibers in persons not occupationally exposed are relatively small compared with the numbers found in occupationally exposed (Selikoff).

Further evidence of exposure is the fact that in a few geographic areas, pathologic changes considered to represent a reaction to asbestos (e.g. pleural calcification) have been found in populations with no history of occupational exposures. Moreover, as mentioned above, asbestos fibers have been demonstrated in ambient air.

A 1973 petition sent to the Federal Trade Commission by the Center for Science in the Public Interest regarding the hazards of asbestos noted the following: Readings taken by Selikoff's group at various sites in New York City (all locations being distant from known asbestos sources) found the chrysotile level of the ambient air to be as follows: Manhatten, 25-60 fibers of asbestos in 10^{-9} gm/cu m; Bronx, 25-28 fibers per 10^{-9} gm/cu m; Brooklyn, 19-22 fibers per 10^{-9} gm/cu m; Queens, 18-29 fibers per 10^{-9} gm/cu m; and Staten Island, 11-21 fibers per 10^{-9} gm/cu m. These amounts may appear at first reading to be rather small. However, chrysotile asbestos fragments very easily into fibrils 300-400 Å in diameter. Therefore, 10^{-9} gms of chrysotile asbestos could represent a million fibrils.

The chrysotile content of air in Philadelphia was found to be 45-100 fibers per 10^{-9} gm/cu m; in Ridgewood, New Jersey it was discovered to be 20 fibers per 10^{-9} gm/cu m. All readings were taken in 1972.

These figures establish a background of asbestos air contamination in urban areas. The contamination will be, of course, higher in areas adjacent to construction sites.

Secondly, there has been radiological evidence of pleural changes which are commonly associated with exposure to asbestos in individuals living in the vicinity of an asbestos mine or of a commercial user of asbestos.

Third, malignant mesothelioma has been diagnosed in non-occupationally exposed persons, including two patients whose only exposure to asbestos has been living in close proximity to an asbestos processing mill (Borow).

Indirect evidence is provided through epidemiologic studies of malignant mesotheliomas which are rare in the general population (Murphy) associated with individuals with no occupational exposure to asbestos, but who had lived in the vicinity of asbestos fields or mines. Some had moved away from the area as young children and had no known subsequent exposure prior to onset of disease symptoms (Wagner).

An ongoing study is tracing household members who were living with individuals who formerly worked in a factory producing amosite asbestos products, between 1941 and 1954. At the time of the reported study, 326 household members contacted who had no personal occupational exposure to asbestos, have thus far been examined. Among those traced, 2 pleural mesotheliomas have been identified and 2 other living family members have been diagnosed with mesotheliomas. The two deaths were daughters of asbestos workers who were first exposed to asbestos as children in the household of asbestos workers. Both had latency periods (first exposure to clinical appearance of mesothelioma) of over 30 years.

Another report was a case study of 83 patients from a London Hospital with confirmed mesotheliomas. Of these, 9 were relatives of asbestos workers. The most usual history was that of the wife washing her husband's work clothes, or from exposure to dusty work clothes. Of 36 patients with no occupational or domestic exposure, several had lived within 1/2 mile of an asbestos factory. Duration of exposure was reported to have been as little as 2 months to over 50 years. (Newhouse)

In an elaboration of the earlier London Hospital case study, it was reported that "the occurrence of tumors after exposure of less than 1 year's exposure ...suggests personal factors (may be) important in the etiology." (Newhouse)

The reported association of rare malignant mesotheliomas with asbestos exposure has given rise to the establishment of a Register maintained by the Employment Medical Advisory Service of Britain. Of 246 confirmed cases reported to the Register for 1967-68, 14 had non-occupational exposure histories including neighborhood, domestic or "hobby" exposure. Of these, one subject had relined brakes and clutches as a hobby, and

another was reported to have had one day's exposure resulting from sawing asbestos cement sheets to construct two sheds. (Greenberg)

Another investigation of the extent of asbestos exposure associated with 42 diagnosed mesothelioma cases was conducted in southeastern Pennsylvania. Of these, 8 were neighborhood exposures and 10 had "questionable" exposures. Among these was a 14-year old boy who had helped his father replace plaster board containing asbestos during extensive home remodeling.

Another report of a previous exposure to asbestos was a man who mixed and applied asbestos cement insulation to boilers in his home. His total exposure during these applications (according to the report) was only a matter of hours. (Lieben)

A study of 500 consecutive autopsies in Cape Town, South Africa and, 500 in Miami, Fla. both showed that 30% of the males and 20% of the females had evidence of inhaling asbestos fibers. This finding led to the speculation that there is asbestos contamination in urban areas and that it might be an increasing problem with increasing use of asbestos-containing products and in view of the virtual indestructibility of asbestos fibers.

It is also suggested that it is possible that inhalation of small numbers of asbestos fibers over a long period of time could result in focal concentration at the lung bases, possibly attaining fibrogenic or carcinogenic concentrations. (Thomson)

Substantiation of brief non-occupational exposures to asbestos which occurred 20 or more years prior to onset of disease is difficult, if not impossible and can only be considered indicative of hazards of exposure to asbestos at levels less than occupational exposures. In fact, some of the exposures, though brief, may have been massive -- as with children playing on asbestos waste dumps.

As pointed out in the 1971 National Academy of Sciences Estimation of Risk in Non-occupational Exposures to Asbestos, "One cannot extrapolate from the mortality experience ... of those directly or indirectly exposed to asbestos (to) ... the general public, who have had moderate or slight exposures ...". Further, "...there is evidence to suggest a gradient of effect from direct occupational, to indirect occupational, to family and neighborhood situations". (NAS)

However, since there is no known threshold exposure to asbestos, below which no adverse health effects will be manifest, unnecessary exposure should be eliminated wherever possible.

Fiber Identification Problems

The increasing awareness of the health hazards associated with exposure to airborne asbestos has focused attention on the problems of fiber identification and quantitative determination of asbestos in admixtures with many other constituents commonly found as air contaminants.

Currently, the electron microscope is the only instrument capable of measuring the morphology, chemistry, and crystal structure of asbestos fibers. Several laboratories perform the analysis of airborne asbestos fibers, and while they have reasonable internal self-consistency, the results obtained by the separate laboratories are often widely different. However, this methodology is expensive, extremely time-consuming and requires highly trained specialists for analyses.

Various other methods such as x-ray diffraction, infra-red spectroscopy, etc. have been utilized for quantitative determination of asbestos with varying degrees of success.

OSHA's sampling and evaluation method for identification of asbestos fibers is the NIOSH Membrane Filter Method whereby samples are collected by drawing air through a cellulose ester membrane filter by means of a battery powered personal sampling pump. The filter, after collection of the sample, is transformed from an opaque solid to a transparent, optically homogenous gel. The fibers are sized and counted by phase-contrast microscopy at 400-450 x magnification. (A copy of the method is attached.)

The Commission has been invited to participate in a workshop on "Asbestos: Definitions and Measurement Methods," jointly sponsored by the National Bureau of Standards and the Occupational Safety and Health Administration, in July 1977. It is the goal of this and subsequent interagency workshops to resolve problems where possible in fiber identification methodologies.

Free vs. Bound Fibers

It is important to note that the asbestos content of a given product is not necessarily the sole criterion of that product's relative health risk, for in numerous products the fibers are tightly bound to the matrix or are encapsulated. A potential health risk occurs when asbestos fibers become airborne, such as by mixing, sanding, or cleanup operations when using patching compounds. However, in terms of risk to the public health, a single individual engaged in such a process may cause exposure of other individuals in the vicinity not so engaged. The importance of such "bystander" exposure has been emphasized in several reports.

Consumer Prod.

Asbestos Regulations Affecting Consumer Products in Other Countries

1. Canada

In June 1976, Canada banned the sale and importation of asbestos-containing products for use by a child in learning or play which releases asbestos and exposes the child to a possible health hazard which may not become evident for many years. Also covered are modeling materials containing asbestos. Airborne asbestos fibers can be generated during two distinct operations: (1) the mixing of the modeling materials and (2) during abrasive processes such as the drilling, filing or sanding of the dried article.

Therefore, the following items were added to Canada's Hazardous Products Act schedule:

> "26. Products that are composed of, or contain actinolite, amosite, anthophyllite, chrysotile, crocidolite, cummingtonite, tremolite or any other type of asbestos and that
>
> (a) are for use by a child in learning or play, if they are made in such a way that asbestos may become separated from the products, or
>
> (b) are for use in modeling or sculpture."

A copy is attached.

2. England

A voluntary labeling scheme was introduced in April 1976 for products containing asbestos by the Department of Prices and Consumer Protection in an agreement with the Asbestosis Research Council and the Asbestos Information Committee.

The products affected are all "do-it-yourself" materials which contain asbestos and a range of household products which are to bear cautionary labels.

In addition, it was announced that the Asbestos Information Committee would make available leaflets for distribution of retailers of "do-it-yourself" asbestos-containing materials which "...set out ways of avoiding exposure to asbestos dust" in the use of such materials.

A copy of the voluntary label and suggested precautionary measures is attached.

3. Sweden

There is a ban of the use of asbestos in certain products in Sweden. The Commission's Office of the Secretary has requested a copy of this regulation but it has not yet been received.

Disposal

The original ceiling material in the Yale University Art and Architecture building was a spray-applied mixture of asbestos and fibrous-glass. This material caused occupant exposure to asbestos fibers under all conditions of normal activity. In some situations, the measured asbestos fiber concentration exceeded OSHA's allowable limits for industrial exposure. Since many air samples indicated potentially carcinogenic asbestos exposures, it was determined that the ceiling material would have to be removed from the building. Three methods of disposal were assessed on a trial basis for extent of asbestos contamination resulting therefrom, namely; dry removal, application of water to the ceiling followed by removal, and application of water and a surfactant to the ceiling followed by removal. It was found that the application of water containing a surfactant, or wetting agent, to enhance water penetration, resulted in the lowest amount of airborne asbestos during the removal process. The material removed in this fashion and all other debris were placed in plastic bags within 55-gal fiber drums for disposal. Disposal was by burial in accordance with guidelines of the Environmental Protection Agency.

Disposal by consumers of banned consumer products containing asbestos fibers should follow the same general guidelines, namely, use of water and a surfactant (e.g. household detergent) to wet the given product followed by placement of the material in a plastic bag and sealing of the bag for future disposal according ro EPA guidelines.

Conclusions

Asbestos is ubiquitous -- found in air, water and land in varying quantities. There exists then an unavoidable background concentration in our normal environment. Analyses of community air and home air for asbestos are far too limited to define the sources, concentrations and distribution in the environment. However, the asbestos fiber concentrations that have been measured in ambient air are small compared to those in industry. While there is a significant body of medical evidence linking chronic asbestos exposure with the induction of neoplasia, the data on consumer exposure is more tenuous. Such estimates as there may be on possible consumer exposure and concentrations have been derived from medical histories from asbestos workers' families or from case reports cited in the literature. Therefore, the actual consumer risk is still poorly defined. A review of the available evidence indicates that there is a gradient effect which suggests that there are individuals in the general population who inhale asbestos at low levels without detectable risk. At this point it is not known if there exists a range of respirable airborne asbestos fibers which has no measurable effect on health.

We have developed a risk assessment model which, of necessity, contains a number of assumptions. Nevertheless, the most important question in the mind of a person exposed to asbestos in a non-occupational setting is whether or not he or she runs an increased risk. Our risk assessment model is designed to address this question. Based upon the amount of exposure i.e., light exposure conditions, we would not expect to have an observable effect in 30-40 years. Under conditions of heavy exposure as defined in the model, there is an increased risk of death from respiratory cancer estimated at one per thousand. While it is clear that these estimates may be controversial, it is nonetheless prudent to discourage prospectively non-essential uses of asbestos in consumer products which are likely to contribute to future contamination levels.

ASBESTOS ANNOTATED BIBLIOGRAPHY

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   USA-USSR joint publ; Problem III, Topic I, co-operative program in Env. Health Res. 1974
   Mathematical projections in terms of temporal effects. Avoids impact of assessing risks in terms of excess cancer deaths - rather hardly tolerates anything greater than zero carcinogenicity.

2. Albert R.E. & Altshuler, B Considerations relating to the formulation of limits for unavoidable population exposures to environmental carcinogens.

   Proc 12th ANN Hanford Biology Symposium May 10-12, 1972
   Mathematical projection & discussion of carcinogen exposure risk deciding that "a more meaningful assessment of hazard should include age of occurrence and life shortening effects.

3. Anderson ... Selikoff Asbestos disease resulting from household exposure to occupational dusts

   Chest 66:3 (Sept 1974)
   Found that the dusts brought home by occupational workers settled in rafters and remained a long time.

4. Anderson, HA Household - Contact asbestos neoplastic risk

   Annals of the NY Acad Sci 271:311-323 (1976)
   An ongoing study tracing household contacts living of workers in a factory producing amosite asbestos products between 1941-45 (when the factory closed) 326 household members with no personal occupational exposure to asbestos have thus far been examined.

5. Bader et al (Selikoff) Pulmonary function and radiographic change in 598 workers with varying duration of exposure to asbestos

   Mt. Sinai Hosp. J 37:492:500 (1970)
   Ambulatory asbestos workers screened. Attempt made to relate grading of chest roentgenograms to vital capacity.

6. Berkley, C The detection & localization of mineral fibers in tissue.

   Ann of the NY Acad Sci. 132:48-63 (1965)
   Description of techniques for the detection in vivo & in vitro of individual fibers.

7. Berry G et al Combined effect of Asbestos exposure & smoking on mortality from lung cancer in factory workers

LANCET 2(775):476-479 (Sept. 2, 1972)
Heavy exposure to asbestos in combination with smoking produce a significant increase in cancer. This was not true of moderate exposures in smokers or non-smokers.

8. Borow, M. Mesothelioma & its Association with Asbestos

JAMA 201(8):93-97 (Aug 21, 1967)
Report of 17 cases of mesothelioma within a 3 year period diagnosed at a N.J. hospital within close proximity of a major asbestos producing plant.

9. Borow, M. et al Mesothelioma following exposure to Asbestos with a review of 72 cases

Chest 64(5): 641-645 (1973) Critical Rev.
Higher risk of occupational exposure because fibers are loose not bound as they are in products for general public. Discusses clinical aspects related to one of Selikoff's cohorts.

10. Bowes, Langer & Rohl Nature & Range of Mineral Dusts in the Environment

In press Trans. Roy Soc. London
A broad general background discussion of mineral dusts as pollutants in air, drinking H_2O, foodstuffs & drugs.

11. Bruckman, L., et al Asbestos and Mesothelioma incidence in Connecticut

J. Air Poll Cont. Assoc. 27(2):121-126 (1977)
Report of 133 diagnosed mesotheliomas to the Conn. tumor Registry between 1935-72 exhibiting a 10-fold increase of this rare cancer. Authors suggest this parallels asbestos increase in the state.

12. Davis, J.M.G. Histogenesis & Fine Structure of Peritoneal Tumors ...

J of NCI 52(6):1823-1837 (June 74)
Crocodolite was injected i.p. into rats & mice. The resulting tumors studied by light & EM microscopy suggested that growths arise from undifferentiated mesenchymal cells in submesothelial tissues.

13. Davis, JMG An electron microscope study of the response of mesothelial cells to the intraplural injection of asbestos dust

BR J Exp Path 55:64-70 (1974)
No mesothelial hyperplasia by chrysotile or crocidolite within 6 mos. in rats, mice or guinea pigs. Fibers in the majority occurred as crystal bundles & averaged 5 inches in length & 0.1 inches in width. Histological changes are described.

14. Englebrecht FM et al Biological effect of asbestos dust on the peritoneal viscera of rats

So. Afr. Med. J. 47:1746-1750 (1973)
This study used crocodolite & chrysotile. Authors postulate a soluble carcinogen from the fibers acting upon mesothilial cells to produce anaplastic changes.

15. Enterline, P.E. et al Asbestos dust exposures at various levels & mortality

Arch Env. Health 15:181-186 (1967)
Workers in the asbestos textile industry showed increased mortality over 16.5 years from respiratory & digestive cancer as well as pulmonale & asbestosis.

16. Enterline P & Henderson V A model for extrapolating to low levels of asbestos exposure

Abstract presented at Conference on Problems of Extrapolating the Results of Lab Animal Data to mes ... Pinehurst, N.C. Mar 11, 1976
Assumptions are made from other workers data & a mathematical model projected.

17. Evans, J.C. et al Studies on the deposition of inhaled fibrous material

Env. Res. 6:180-201 (1973)
Using radioactive tracer methods & nose only exposure in rats it was found that about 50% of the crocodolite deposited in the lower respiratory tract 17% was cleared in the 1st 30 days.

18. Gibson, J.C. Report of the Advisory Committee on Asbestos Cancers to the Directory of IARC

Brit J Ind Med 30:180-186 (1973)
The UICC working group on asbestos cancers made a series of recommendations for assessing the hazards of asbestos in terms of needed research and epidemiological studies.

19. Gibson, J.C. Asbestos Cancer: Past & Future Hazards

Proc Roy Soc Med 66:395-403 (1973)
Presents the hypothesis that fiber diameter of the different types of asbestos determine their point of impact in the lung and therefore their chances of causing cancer eg crocidolite smaller than amosite .. carried deeper into lung.

20. Greenberg Metal Mesothelioma Registry 1967-68

Brit J. Indust Med. 31:91-104 (1974)
Mesothelioma of the pleura & peritoneum are rare but the reported association with asbestos exposure has given rise to the establishment of a register maintained by the (now) Employment Medical Advisory Service of Britian. 246 confirmed causes.

21. Haley T.J. Asbestosis: A reassessment of the overall problem

J. Pharm Sci 64(9): 1435-49 (1975)
A review of sources of asbestos exposures both occupational and non-occupational & risks of asbestosis and/or cancer associated with these exposures. The author has cited a report of asbestosis from sawing wall boards without a respirator.

22. Holt P.F. Asbestos fibres in the air of towns

Atmospheric Env. 7:481-483 (1973)
Air sampling done in a number of cities from Iceland to S. Africa. Asbestos fibers found in minute amounts in all. Suggest a potential risk in the presence of higher concentrations because lung mechanisms cannot handle & coat large number of fibers.

23. Hueper, W.C. Occupational & non-occupational exposures to asbestos

NY Acad of Sci 132:184-195 (Dec 1975)
For an assessment of the potential scope of an environmental health hazard and occupational & non-occupational implications of asbestos exposure, it is necessary to determine the distribution pattern of the agent from the site of its original occurrence and production (also production figures are included).

24. Huff, J.E. Asbestos: An Overview

Env. Chemicals - Human & Animal Health 3rd Annual Conference Proceedings (1974)
A review of the literature on the occupational & environmental hazards of asbestos.

25. IARC Working Group on the Evaluation of the Carcinogenic Risk of Chemicals to Man: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Asbestos Intl. Agency for Research on Cancer. 1977.

A review of asbestos and associated health hazards. This includes experimental studies, sources of exposure and evaluation of the carcinogenic risk to man.

26. Kleinfield, M. Biologic Response to kind & amount of asbestos.

Joccup Med. 15(3): 296-300 (Mar 73)
Discusses various factors established & theoretical in the production of asbestosis and asbestos related cancers.

27. Kleinfeld, M et al Mortality among talc miners & millers in New York State

Arch. Env. Health 14:663-667 (May 1967)
Malignancies 4 times greater in this group of 220 with 15 years exposure than expected. No mesotheliomas. Significant increase occurred 20 yrs later (60-79 yrs) than in asbestos workers (40-59 yrs).

28. Kleinfeld, M et al A study of workers exposed to asbestiform minerals in commercial manufacture

Env. Res. 6:132-143 (1973)
In a total of 74 workers exposed to talc containing tremolite & anthophyllite fibers over a mean time of about 6 years, no malignancies are reported. Two different studies 2 exposure levels.

29. Langer, et al Identification of asbestos in human tissues

J. of Occ. Med 15(3): 287-295 (Mar 1973)
Discusses methodologies used in asbestos identification.

30. Langer, et al (Selikoff) Inorganic fibers including chrysotile in lungs at autopsy: preliminary report

Inhaled Part. Vap. 2:683:694 (1970)
Chrysotile fibers break down chemically and separate into 30-40 nm wide fibers in vivo. The amphiboles have not been observed to do this.

31. Langer, Rohl Selikoff et al Inorganic particles in cigarettes and cigarette smoke (1974)

Science 174:585 (1971) Inorganic particles in cigars & cigar smoke; Hammond, Langer, et al.
Briefly present some support for hypothesis that the inorganic components of tobacco smoke play some role in the etiology of lung fibrosis.

32. Langer, Selikoff, et al Chrysotile asbestos in the lungs of persons in NYC

Arch Env. Health 22:348-361 (Mar 1971)
Data from 28 consecutive NYC autopsies: Chrysotile found in 24.

33. Libshitz, H.I. et al Asbestosis of carcinoma of the larynx

JAMA 228:1571-72 (1974)
"Three patients had carcinoma of the larynx, history of asbestos exposures and evidence of intrathoracic changes characteristic of asbestosis. Suggest relation between asbestosis and laryngeal carcinoma.

34. Leben, J. Mesothelioma & asbestos Exposure

Arch Env. Health 14:559-563 (Apr 67)
Study of extent of relationship of asbestos exposure to mesothelioma diagnosed in S.E. Penn.

35. Lillington G.A. et al Conjugal Malignant Mesothelioma

New Eng. J. Med. 291(11): 583-84 (Sept. 12, 1974)
Report of an industrial worker with malignant mesothelioma and that of his wife whose exposure resulted from washing his dirty clothes.

36. Masson, T.J. Asbestos like fibers in diluted water supply

JAMA 228(8): 1019-1020 (1974)
No carcinoginic effect was noted from patterns of cancer mortality in a period of 14 years.

37. McCullagh S.F.: The Biological Effects of Asbestos
Med. J. Aust. 2:45-49 (1974).
In the urban atmosphere of both the U.S. and England, "The amount of asbestos is between one hundred thousandth and one millionth of that held to be safe in the workplace (according to the authors).

38. McLachlin, MSF et al Radiological diagnosis of crocidolite-induced pleural mesotheliomata in the rat.
Br. J. of Exp. Path. 55:164-168 (1974).

Thirty-six rats received crocidolite intrapleurally. Seven rats developed mesothelioma as diagnosed by X-ray. Three others were misdiagnosed.

39. Miller, Langer, Feinstein & Selikoff Non-Specific Interstitial Fibrosis.
New Eng J. of Med. 292(2): 91-93 (Jan. 9, 1975).

Report finding of chrysotile asbestos fibrils by electron microscopy in a patient described as having severe interstitial fibrosis with no known pathogenic dust exposure. Authors suggest EM examination of lung tissue in diagnoses of "idiopathic pulmonary fibrosis".

40. Mackish, D.E., et al: Exposure to Asbestos During Brake Maintenance.
Ann. Occup. Hyg. 10, pp.17-21 (1970).

Air samplings were made during blowing-out procedures of brake shoes and drums. Measurements of the dust cloud exceed the fiber standard but personal exposure measurements were below the standard.

41. Maggiori, Nicholson & Rubin Neighborhood Contamination with Asbestos. Analysis of settled dust as index of asbestos air pollution in prior Decades.

Environ. Sci. Lab. Annual Report (1974).

42. Martisching, K.J. et al Unsuspected Exposure to Asbestos and Bronchogenic Carcinoma.
Brit. Med. J. 1:746-749 (1977).

Asbestos regulations have reduced the risk of exposure to workers in scheduled industries, but asbestos-related diseases will probably be increasingly found among the many workers who nave only incidental exposures to asbestos.

43. Murphy R.L.H. et al. Effects of low concentration of asbestos

N. Eng. J. Med 285(23): 1270-1278
Shipyard pipe coverors, exposed to 5 million particles per cu. ft. for 13 yrs. showed asbestosis 11 times more commonly than the controls and a 38% prevalence after 30 yrs.

44. Murphy R.L. et al. Floor tile installation as a source of asbestos exposure

Am Rev. Resp. Dis. 100(4) Oct. 1971
Case studies of 2 long time floor tile installers with mesothelioma and pleural complications known to be associated with exposure to asbestos.

45. Newhouse, M.L. et al. Epidemiology of mesothelioma tumors in the London area

ANN of the NY Acad of Sci 132:579-588 (Dec. 65)
Elaboration of earlier case study report. Seems to be little doubt of risk of domestic and occupational exposure to asbestos.

46. Newhouse, M.L. et al. Predictions of mortality from mesothelial tumors in asbestos factory workers.

Brit J. of Ind. Med. 33:147-151 (1976)
It was projected that in the London asbestos textile factory cohort studied, 7-11% of the total mortality would be men and slightly more women would die of mesothelioma.

47. Newhouse, M.L.. Asbestos in the workplace and the community

ANN Occup Hyg 16:97-107 (1973)
Summary of many reports, case studies, exposure rates, asbestos in the environment and notes on non-occupational exposure.

48. Newhouse, M.L. et al. Mesothelioma of Pleura and Peritoneum following exposure to asbestos in the London area

Brit J. Ind. Med. 22:261-269 (1965)
A case study of 83 patients from London Hospital with confirmed mesotheliomas. Of these 9 were relatives of asbestos workers.

49. Nicholson & Rohl. Occupational and community asbestos exposure from wallboard finishing compounds (73-74)

To be publ. Health Res. Council of NYU 2331
Dry wall tapers and painters breathe in the dust from spackling compounds i.e. fiber counts during mixing and sanding operations varied from the TLV to several times.

50. Nicholson, Rohl & Weisman. Asbestos contamination of building air supply systems

From Env. Sci Lab, Dept. of Community Med., Mount Sinai School of Med of the City Univ of NY
Asbestos found in the air of those buildings previously fire proofed with asbesti form sprays. Recommendations are made for future monitoring and control.

51. Nicholson, Rohl Weisman. Asbestos contamination of the air in public buildings. Final Rpt to EPA, 1975

Same as asbestos contamination of building air supply sustems.

52. Nicholson & Rubin. Asbestos lung tissue burden following neighborhood exposure-evaluation of carbon extraction technique

To be pub support NCI-CP 43255
A vague outline of the technique is presented. It was considered inadequate for the purpose of the study.

53. Nicholson, W.J. Asbestos - the TCV approach

Annals of the NY Acad of Sci 271:157-169 (1976)
No known threshold for asbestos or other carcinogens, below which no health effect may be manifested. The TLV is risk limitation value.

54. Polednak, A.P. Latency periods in neoplastic diseases

J. Epid 100(5):354-6 (Nov 74)
Latency = period between exposure to a presumed causal agent and detection of (or death from) a neoplasm. Author suggests 2 models cumulative exposure; one-insult co-carcinogenic

55. Pooley, F.D. Asbestos bodies, their formation, composition and character

Env. Res 5:363-379 (1972)
Formation did not appear to take place or fibers less than 10 u in length and less on crocodolite.

56. Reeves, A.L. et al. Inhalation Carcinogenesis from various forms of asbestos

Env. Res 8:178-202 (1974)
Rats, rabbits, guinea pigs, gerbils and mice exposed to 3 air concentrations of chrysolite, crocodolite and amosite for 2 yrs. Only the rats showed lung or pleural carcinogenic response.

57. Richards, R.J. et al. Ultrastructural changes in lung fibroblast cultures exposed to chrysotile asbestos

But J. of Exp. Path. 55:275-284 (1974)
EM exam showed chrysotile fibers within some cells by 4 1/2 hours with nuclear and cytoplasmic changes.

58. Richards, R.J. et al. Collagen and mucopolysaccharide production in growing lung fibroblasts exposed to chrysotile asbestos

Life Sciences 12(II):441-451 (1973)
Cultures exposed to chrysotile and glass fibers for 24 days. Fiber size not stated. Glass caused slight reversible changes. The chrysotile caused increases in collagen and muco polysaccharide levels indicative of fibrogenesis.

59. Rohl, A. Mineralogical analysis of ore from a major US vermiculite deposit:preliminary investigation

Unpublished - Supported by Health Res. Council 4-12-72
Author claims that potentially hazardous concentrations of asbestos may be found but he had not yet quantitated his analysis.

60. Rohl et al. Asbestos in strange places; children's arts and crafts materials, paper products (1973)

To be pub. Support NIEHS ES 00928
Asbestos fibers of different types, forms and amounts were found in various paper products.

61. Rohl, Langer, Selikoff et al. Consumer talcums and powders: mineral and chemical characterization

J of Tox & Env Health 2:255-284 (1976)
Twenty-one commercial consumers U.S. Talcs & powders were analyzed between 1971 and 1975. Methods were instrumental and chemical. An appreciable number showed various forms of asbestiform fibers. No mention of product names nor the dates of acquisition.

62. Rohl, Langer, Selikoff & Nicholson. Exposure to asbestos in the use of consumer spackling, patching and taping compounds.

Science 189:551-553 (Aug. 15, 1975)
A number of consumer spackling compounds purchased in NYC were found to "contain asbestos minerals as well as other biologically active substances" ("quartz, talc and other minerals with disease potential")

63. Seidman, Lilis & Selikoff. Short term asbestos exposure and delayed cancer risk

Abstract from Mount Sinai School of Medicine of City Univ of NY, 5th Ave & 100th St., NY, NY 10029
Occupational exposure to amosite in a cohort followed from 1941-1945 to 12-31-74. Claim their figures support "fact" that exposure of less than one year duration "results in significant excess risk of lung cancer."

64. Selikoff, et al. Asbestosis and neoplasia

Am J. Med 42(4):487-496 (Apr 67)
What this group has written in more detail elsewhere. Presentation of data - 1973.

65. Selikoff, et al. Asbestos exposure smoking and neoplasia

JAMA 204 #2:106-112 (Apr 8, 1968)
Early paper relating incidence of malignancies in smoking or non-smoking insulation workers.

66. Selikoff & Hammond. Community effects of nonoccupational environmental asbestos exposure

Am J. Pub. Health 58(9):1658-1666 (Sept. 68)
A cautious and scientific evaluation of the many factors involved in properly assessing and assigning cause.

67. Selikoff, et al. Asbestos air pollution

Arch of Env. Health 25(1):1 (1972)
Discussion of the usual i.e. asbestos air pollution level of disease hazard, epidemiological consideration industry responsibility.

68. Selikoff. Widening perspectives of occupational lung disease.

Prev. Med 2:412-437 (1973)
A review of past and present work in the various pneumoconioses and possible relationship to lung cancer.

69. Selikoff. Cancer risk of insulation workers in the U.S. (1974)

Insulation Hygiene Progress Reports Vol 6 #3 (Fall 1974)
Reprint of a Chapter of IARC volume by Selikoff et al. Primarily a summary of what was said before relating to cancer in insulation workers.

77. Wegman, D.H. et al. Worker sponsored survey for asbestosis. Detection of occupational lung disease sans a control group

Arch Env. Health 27:105-109 (1973)
Fifty-seven wall board manufacturing workers were examined by questionnaire, pulmonary function tests and limited physical exam. By predictive equations and regression analysis, disease was proved without a control group.

79. Wagner, J.C. et al. Mesothelioma in rats after innoculation with asbestos and other materials

Br. J. Cancer 28:173-185 (1973)
Intrapleural injection of asbestos into rats produced mesotheliomata approx. proportional to the dose and apparently the finer fibers the more carcinogenic.

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Don Clay, AED for Health Sciences DATE: June 3, 1977

THRU : Robert Hehir, Deputy AED for Health Sciences RH for Grad
Joe McLaughlin, Director, Division of Toxicology and Medicine

FROM : Rita Orzel, Ph.D., Division of Toxicology and Medicine
C. T. Desmond, M.D., Division of Toxicology and Medicine

SUBJECT: Toxicological/Medical Input for proposed Federal Register Notice on Asbestos

The Toxicological/Medical input on asbestos is presented in the attached document as a suggested CPSC Federal Register Notice for Asbestos.

We recognize that this is not the usual format for presenting such material. The time constraints were such that, for the sake of expediency, utilization of research background material previously prepared by OSHA, Federal Register Notice, Occupation Exposure to Asbestos, Vol 40, No. 197, page 17652-665 was used as the basis for this document.

This was determined to be the best method for accomplishing our assignment.

SGP 0009301

*U.S. GOVERNMENT PRINTING OFFICE: 1974 733-533/3604 1-3

I. BACKGROUND

A. General

Asbestos is a generic term used to describe a number of naturally occuring fibrous, hydrated mineral silicates that differ in chemical composition. These may be divided into two mineral groups; (1) Serpentines, which include chrysotile ($3MgO \cdot 2SiO_2 \cdot 2H_2O$), the type most widely used in U.S. industry; and (2) amphiboles, including amosite ($(FeMg)\ SiO_3$), crocidolite ($(NaFe(SiO_3)_2 \cdot FeSiO_3 \cdot H_2O$), tremolite ($Ca_2Mg_5Si_8O(OH_{22})_2$), anthophyllite $(MgFe)_7Si_8O_{22}(OH)_2$, and actinolite ($(CaO \cdot 3MgFe)O \cdot 4SiO_2$). Asbestos fibers are generally characterized by high tensile strength, flexibility, heat and chemical resistance, and favorable frictional properties. Certain grades of asbestos can be carded, spun, and woven, while others can be laid and pressed to form paper, or used for structural reinforcement of materials such as cement, plastic, and asphalt.

Chrysotile (white asbestos) is the fibrous form of the mineral serpentine. It is the most common variety of asbestos, widely distributed geographically, with the largest deposits being in Canada, Russia, and Rhodesia. It accounts for over 90 percent of world consumption. Chrysotile can be readily crushed or fiberized into fine, white, silky fibers which may be processed into numerous products. The fibers have good heat resistance, but are destroyed by acids. Crocidolite (blue asbestos) is another important, although more specialized, form of asbestos.

It is the fibrous form of riebeckite, and has fine, resilient fibers of a characteristic blue color. It is mined in South Africa and Australia, and to a lesser extent, in Bolivia. Crocidolite is a strong, fast filtering fiber used especially in the manufacture of asbestos cement sheets and pressure pipes. It is also characterized by its high resistance to acids.

Amosite is the fibrous variety of the mineral grunerite, a ferrous magnesium silicate mined only in South Africa. Amosite can be readily broken down into long, somewhat harsh fibers, with a brownish-yellow to almost white color, depending upon the quality. It is used largely in the production of asbestos cement and heat-insulating products. It is characterized by a good resistance to acids and other chemicals.

Anthophyllite is a magnesium silicate of somewhat variable composition which has rather fragile, brownish or off-white fibers. It is rarer than other types described, but significant quantities have been mined in Finland, Kenya, and other countries. It is used primarily as an inexpensive filler, and for some specialized applications for which good heat or chemical resistance is required.

Tremolite, a calcium magnesium silicate, may be a component of industrial and commercial talc. It is mined in various parts of the United States including New York, Vermont and Montana.

Actinolite, a calcium magnesium iron silicate, is rarely used in industry. It is found in various parts of the world, but its low fiber strength makes it less desirable for industrial use.

Nearly one million tons of asbestos are consumed in the United States annually. According to the Bureau of Mines Mineral Yearbook, 1973,approximately 77 percent of asbestos products consumed in 1972 were used in the construction industries (624,000 short tons) while 23 percent are used in non-construction industries (186,000 short tons). Approximately 92 percent of the asbestos used in construction is firmly bonded, i.e., the asbestos is "locked in" in such products as floor tiles, asbestos cements, and roofing felts and shingles; while the remaining 8 percent is friable or in powder form present in insulation materials, asbestos cement powders, and acoustical products. These latter products generate more airborne fibers than the firmly bonded products. The 186,000 short tons of asbestos used in the non-construction industries in 1972 were utilized in such products as textiles, friction materials including brake linings and clutch facings, paper, paints, plactics, roof coatings, floor tiles, and miscellaneous other products.

II. TOXICOLOGICAL CONSIDERATIONS OF EXPOSURE TO ASBESTOS

A. EFFECTS OF ASBESTOS EXPOSURE

Asbestos, in its several commercial forms, has been shown in very recent times to be associated with the production

of a variety of disease entities. These include:

1. Asbestosis: a diffuse, interstitial, nonmalignant, scarring of the lungs;

2. Bronchogenic carcinoma: a malignancy of the interior of the lung;

3. Mesothelioma: a diffuse malignancy of the lining of the chest cavity (pleural mesothelioma), or of the lining of the abdomen (peritoneal mesothelioma);

4. Cancer of the stomach, colon, and rectum.

Asbestos may be evident, in its advanced stages, by characteristic manifestaions on x-ray films, by restrictive pulmonary function, or by clinical signs, of finger clubbing or rales (dry, cracking sounds within the lung). Its most important symptom is dyspnea, or undue shortness of breath. The disease is progressive, even in the absence of further exposure, as those inhaled fibers which have been trapped within the lung continue their biological action. In its severe forms, death results from the inability of the body to obtain requisite oxygen or from the heart's failure to pump blood through the scarred lungs.

SGP 0009305

Mesothelioma tumors are diffuse and spread rapidly throughout the cavity of origin. They have yet to be successfully cured by any type of treatment including chemotherapy, radiation, or surgery. Death usually results within a year of diagnosis. It may account for one death in several thousand in the absence of an environmental or occupational

asbestos exposure. In some groups of asbestos workers, it may account for one death in ten.

Once established, the other asbestos associated cancers differ little from those occuring in the general population, although there may be variations in the location of the primary site. Appropriate treatment and prognosis follow for the particular tumor. There is very limited long term survival from lung cancer therapy; and only somewhat better from treated cancer of the colon or rectum.

Asbestosis and asbestos cancer whether it be lung cancer, pleural mesothelioma, peritoneal mesothelioma, cancer of the stomach, colon, rectum-usually do not become clinically evident until more than 20 years have passed from onset of exposure. This time-interval is now widely recognized.

While some such cancers may appear during the second decade following onset of occupational exposure, peak incidence is often not noted until the 30-years-from-onset point, or later. This is true both with regular, long-term, brief or intermittent exposures. While variations in the time of occurrence may depend upon intensity and duration of exposure, with heavier exposure often being associated with shorter latency periods, variations among individual cases make it impossible to predict the latency period for the risk of any particular person.

B. RECENT DOCUMENTARY EVIDENCE OF OCCUPATIONAL EXPOSURE

(1) Asbestosis. Subsequent to the hearings on the

current Occupational Safety and Health Administration (OSHA) standard, uncertainty has arisen as to whether the existing British asbestos standard and the mandated 2 fiber/ml U.S. standard provide effective protection even against asbestosis. The data from Great Britain obtained in 1966 indicated that little clinical disease, including x-ray evidence of asbestosis, had occurred among workers first employed in that factory at some time after 1933, when important improvements in work practices had been achieved. In 1972, results of evaluation of new x-rays that had been taken in 1970, of the work force then employed in the same factory, were reported as showing that many now had abnormal findings either in the lung or in the coverings of the lung pleurae (Lewinsohn, 1972). There was thus a difference between the prevalence of abnormal x-ray findings among workers x-rayed in 1966 as reported to the British Occupational Hygiene Society, and evaluation of other x-rays of workers in the same factory four years later.

Additionally, clinical data are becoming available concerning asbestos lung scarring in individuals exposed at levels much lower than those of occupational circumstances. Among 210 family contacts of former asbestos factory workers, 38% have been reported to have x-ray changes characteristic of asbestos exposure (Anderson, Selikoff, Lilis and Daum, 1975).

(2) Cancer. In December 1972, important new information on the spectrum of asbestos cancers was presented

at the Conference on the Biological Effects of Asbestos, sponsored by the International Agency for Research on Cancer of the World Health Organization. At this conference, and subsequently, data on large groups of asbestos workers became available (Selikoff, Hammond and Seidman, 1973; Enterline, de Coufle and Henderson, 1972). As expected, the high risk of bronchogenic carcinoma and mesothelioma persisted among factory employees and insulators. Moreover, these later studies confirmed the excess gastrointestinal cancer that had been suggested earlier, and extended the spectrum of asbestos related cancers.

(a) Lung cancer. The most important cancer afflicting asbestos workers is cancer of the lung, although mesothelioma has attracted considerable attention because of the high frequency among asbestos workers and infrequent occurrence in the population as a whole.

In many groups of asbestos workers, approximately 20% of all deaths are caused by lung neoplasms. This has been true both among asbestos product factory workers (Selikoff, Hammond and Churg, 1972; and Nicholson, 1976) and among users of these products (Selikoff, Hammond and Seidman, 1973). The exact percentage varies with circumstances of exposure, age of the workers, duration of the workers' exposure and, and perhaps most of all, according to the duration from the onset of their asbestos work history. In addition, the last several years have seen the discovery of another critical variable affecting the incidence

of lung cancer among asbestos workers. In 1968, Selikoff, Churg and Hammond reported that lung cancer was not significantly increased in incidence among asbestos workers with no history of cigarette smoking, although when such history was present, the incidence of lung cancer increased markedly over what would be expected among other cigarette smokers, in the absence of asbestos exposure. Thus, these scientists calculated that an asbestos worker who smoked cigarettes had 92 times the risk of dying of lung cancer, as compared with like individuals without cigarette smoking or asbestos work. This finding has been confirmed by larger studies (Hammond and Selikoff, 1973) where, again, it was found that non-smoking asbestos workers had few lung cancers while those who smoked had much more lung cancer than would have been expected had they not been asbestos workers. Calculations suggest that cigarette-smoking asbestos workers have approximately eight times the risk of developing lung cancer compared to other smokers.

(b) Pleural and peritoneal mesothelioma. In 1960, Wagner, Sleggs and Marchand demonstrated an important association between asbestos exposure and pleural mesothelioma. This cancer, which appears to be unrelated to smoking, had previously been considered to be a very rare tumor. Numerous reports have confirmed the finding of Wagner and his colleagues that mesothelioma can be commonly associated with asbestos exposure. A subsequent report by Enticknap and Smither, 1964, concerning workers in a British asbestos factory demonstrated that the same tumor could be commonly

found in the abdomen (peritoneal mesothelioma), as well as in the chest.

The exact risk of death of these invariably fatal neoplasms has not been as well defined as has lung cancer, although recording of cases from hospitals near one large asbestos factory has indicated that it must be very common indeed (Borow, Conston, Livornese and Schalet, 1967). Information available from the experience of asbestos insulation workers suggests that approximately five to seven percent of deaths may be due to this neoplasm (Hammond, Selikoff and Churg, 1965; Selikoff, Hammond and Seidman, 1973). More recently, it has been suggested that this estimate is too low, on the basis of the experience of workers in a British asbestos factory where calculations predicted that between 10 and 11 percent of deaths would be due to mesothelioma (Newhouse and Berry, 1975).

(c) Gastro-intestinal cancer. Gastro-intestinal cancers (cancer of the stomach, colon and rectum) are also increased in incidence among asbestos workers, A number of studies now indicate that the increase is on the order of two or three times the number of expected tumors (Selikoff, Hammond and Seidman, 1973; Elmes and Simpson, 1971). Although this increased risk is relatively limited, especially when compared with lung cancer and mesothelioma, it is nevertheless of considerable importance since a two - or three-fold increase in such common tumors becomes an important cause of death for the workers involved.

It has been suggested that other tumors are also increased in incidence among asbestos workers, particularly cancers of the larynx (Stell and McGill, 1973; Newhouse and Berry, 1973), and of the esophagus (Selikoff, Hammond, and Seidman, 1973). However, data concerning these neoplasms are less extensive than for lung cancer, mesothelioma and gastro-intestinal cancer and further experiences are awaited. In any case, they are not very common tumors in general and any increase does not weigh heavily on the overall cancer risk of asbestos workers.

Considering all neoplasms, among some groups of asbestos workers, employed either in asbestos factory work or in the use of asbestos products, as much as 40 to 45 percent of all deaths have been due to one or another type of cancer, an approximately three-fold or four-fold increase.

Of significant importance, new data have recently been made available concerning the cancer risk of workers at the textile mill reviewed for the British standard, including those workers first emloyed after 1933 (Howard, Kinlen, Lewinsohn, Peto and Doll, 1975). It was found that there was excess mortality from lung cancer among those workers who entered scheduled areas after 1 January 1933. There was "clear evidence of some excess of lung cancer and respiratory deaths among those first exposed between 1933 and 1950." Equally important was the finding that "there still

appears to be an excess of deaths due to lung cancer after 15 or more years' exposure" even among those first exposed in 1951 and subsequently. Indeed, it is known that mesothelioma deaths have occurred among the specific group of 290 workers whose experience prior to 1966 had led to the development of the current standard as detailed above (Brody, 1974).

C. NON-OCCUPATIONAL, INDIRECT OCCUPATIONAL EXPOSURE OR INTERMITTENT OR BRIEF EXPOSURE

Recent data indicate the occurrence of asbestos cancer among individuals exposed to low levels of asbestos, as in environmental circumstances, or to brief or intermittent exposures to higher levels.

Analysis of the history of asbestos exposure among individuals in a large series of cases of mesothelioma in Great Britain and South Africa have provided evidence that brief or intermittent exposure to asbestos may, after the passage of decades, result in mesothelioma (Greenberg and Davies, 1974; Webster, 1973). In such circumstances, it appears that the lifetime exposure was less than 100 fiber-years/ml. The same discrepancy between present projected exposures and the risk of asbestos cancer exists when considering cases of mesothelioma resulting from household contact to asbestos among members of families of asbestos workers (Lillington, 1974) or among residents living in the vicinity of asbestos plants.

Of considerable industrial importance, has been the recent description of asbestos disease among shipbuilding and ship repair workers, few of whom actually work with asbestos, but many of whom were, in the past, inadvertently exposed to the asbestos dust resulting from the use of asbestos products by a relatively few of their work mates. In 1968, Harries of the Royal Navy reported cases of mesothelioma among shipyard workers at the Royal Navy dockyard in Devonport, in trades which did not directly involve worker exposure to asbestos, but in which there had been occasional opportunity for exposure merely by virtue of working in the same areas. This original finding has been widely confirmed and numerous cases of mesothelioma have since been reported in former shipyard workers (Whitwell and Rawcliffe, 1970; McEwen et al, 1970; Stumphius, 1971; Greenberg and Davies, 1974). Studies of populations of current shipyard workers have shown much radiological evidence of asbestos abnormalities among workers in trades only indirectly exposed to asbestos in the yards (Sheers and Templeton, 1968; Fletcher, 1972).

Gillam et al (1975), studying the mortality and reviewing the chest x-rays of 439 underground metal miners exposed to an asbestiform mineral, found three times the risk of malignant respiratory disease than expected. The fiber concentrations averaged 0.24 fibers/ml

Further, evidence has indicated that asbestos also acts as a lung carcinogen at levels much below those

which will produce asbestosis. Two surveys of shipyard workers who had x-ray evidence of pleural plaques, but generally not of pulmonary fibrosis, showed a 2.5-fold excess risk of death from lung cancer and high risk of mesothelioma. (Fletcher, 1972; Edge, 1975). In a study of the mortality experience of a large U.S. asbestos products manufacturing facility, it was found that workers in low-dust areas, with a minimum risk of death from asbestosis, had the same high risk of death from various cancers as workers in dustier areas (Nicholson, 1975).

Reports of the hazard, or potential for hazard, from exposure to asbestos resulting from use of asbestos-containing products are very limited. To determine the type and extent of non-occupational exposure, the investigator must generally rely on relatives or persons other than the exposed patient for exposure history.

Furthermore, it is extremely difficult, if not impossible, to document brief exposures reported to have occurred 20 or more years before the onset of symptoms. The only known quantitative study of asbestos levels in products regulated by this Commission was reported by Rohl, et al (1975) of asbestos fiber concentrations measured during the use of consumer spackling, patching and taping compounds. This study indicated that airborne fiber concentrations, exceeding the interim OSHA allowable excursion exposure level, were detected during application and cleanup operations. Fibers were detected in adjacent rooms during mixing

operations and it was reported that "... significant concentrations of asbestos remained suspended and could pervade living quarters for a considerable duration of time..." The authors suggest that the use of spackling and other patching compounds (in mixing, sanding and cleanup operations) may expose the user and other members of the household to "... significant concentrations of asbestos".

III. CERTAIN CONSIDERATIONS CONCERNING CARCINOCENICITY

In the case of asbestos, we are dealing with a substance that poses a range of health risks. These include the threat of cancer, as well as asbestosis. In considering the issue of carcinogenicity the data cited above should be considered along with leading scientific principles and opinions believed to reflect the research conclusions of international cancer experts.

A. LATENT EFFECTS

In humans, the latency period for chemical carcinogens may well extend between 20 to 40 or more years. Analogous periods exist for test animals. This means that the disease may undergo a long period of development before a tumor is actually detected. Prudence would seem to dictate that every reasonable measure should be taken to eliminate human exposure to chemical compounds as soon as their carcinogenic nature is identified.

B. INDIVIDUAL VARIATION

Cancer development may be influenced by such factors as the differing susceptibility of various body organs. In animal studies it has been found that individual variability in response to carcinogens is great depending upon factors such as age, sex, hormonal status, diet, and genetic factors. Thus, individuals biologically compromised, may be more susceptible than other groups.

C. "THRESHOLD" LIMIT

Because of the variability of individual response to carcinogens and other factors, the concept of a "no effect" or "threshold level" may have little real significance on the basis of existing knowledge. While some level, below which exposure to a carcinogen does not cause cancer, may conceivably exist for any one individual, other individuals may have cancer induced by doses so low as to be effectively zero. This is not to say that researchers will never find a threshold level for a carcinogenic substance, but it does mean that the threshold concept for carcinogens is, at present, more a matter of responsible regulatory policy than a precise, scientific determination.

These theoretical concepts have a bearing on the asbestos issue, particularly as the question of the existence, or nonexistence, of a threshold level of carcinogenic effect. A "no effect" level theoretically may exist, but it has not been demonstrated. Therefore, there is no known threshold level below which exposure to asbestos would be considered safe.

The use of asbestos as simulated ash in artificial fireplace logs and in consumer patching compounds poses an unreasonable risk of inhalation of potentially hazardous asbestiform fibers and are exposures which are avoidable.

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ATTACHMENT 2

Consumer and Corporate Affairs | Consommation et corporations

Consumer Standards Directorate | Direction générale des normes

Product Safety Branch | Sécurité des produits

(PSB-TC-011)

Your file / Votre référence

Our file / Notre référence 736

TRADE COMMUNIQUE

ASBESTOS IN TOYS
AND
MODELLING MATERIALS
ISSUE NO. 2

JUNE 1976

This will advise all interested parties of the inclusion of item 26 to Part I of the Schedule to the Hazardous Products Act on June 1, 1976. This item prohibits the importation, advertisement, or sale in Canada of products for use by a child which may release asbestos and of modelling materials which contain asbestos.

Asbestos is known to be a health hazard. Chronic lung disease and cancer are attributable to asbestos exposure.

SGP 0009321

This item covers products for use by a child in learning or play which release asbestos and expose the child to a possible health hazard which may not become evident for many years. Also covered are modelling materials containing asbestos. Airborne fibres of asbestos can be generated during two distinct operations, viz: the *mixing* of the modelling materials

COMMUNIQUE AUX ENTREPRISES

AMIANTE DANS LES JOUETS
ET
PATES A MODELER
PUBLICATION NO. 2

JUIN 1976

Toutes les parties intéressées sont avisées par la présente qu'l'article 26 est ajouté à la partie de l'annexe de la Loi sur les produits dangereux, à compter du 1er juin. Cet article interdit l'importation, l'annonce ou la vente au Canada de produits destinés aux enfants et qui peuvent dégager de l'amiante, ainsi que de pâtes à modeler qui contiennent de l'amiante.

Il est reconnu que l'amiante présente un danger pour la santé. L'exposition aux fibres d'amiante peut causer des maladies pulmonaires graves et le cancer.

Le présent article vise les produits destinés à l'éducation ou à la récréation des enfants et qui dégagent de l'amiante et peuvent être la cause de maladies dont les symptômes peuvent apparaître seulement quelques années plus tard. Les pâtes à modeler qui contiennent de l'amiante sont aussi visées par cet article. Des fibres aéroportées peuvent se dégager au cours de deux opérations distinctes, à savoir: le pétrissage

SCHEDULE

1. Part I of the schedule to the Hazardous Products Act is amended by adding thereto the following item:

"26. Products that are composed of or contain actinolite, amosite, anthophyllite, chrysotile, crocidolite, cummingtonite, tremolite or any other type of asbestos and that

(a) are for use by a child in learning or play, if they are made in such a way that asbestos may become separated from the products; or

(b) are for use in modelling or sculpture."

ATTACHMENT

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Francine Shacter, TAD/OSCA
Through: Assoc. Exec. Dir. for Compliance and Enforcement
Through: Director, Division of Inspection and Enforcement

DATE: 23 MAY 1977

FROM : Charles M. Jacobson, ECMI

SUBJECT: Commission Briefing on Proposed Ban of Patching Compounds and Artificial Fireplace Log Ashes Containing Asbestos

As we indicated in our memorandum of May 9, 1977, there are two aspects of the ban on the asbestos containing articles which are a concern to us from a compliance and enforcement standpoint. These are the questions of a prospective versus a retroactive ban and a finite level of asbestos at which products would be considered banned.

We would urge the Commission to propose that the effective date be prospective and ban only those products manufactured after the effective date. This is based on the considerations that the products which will be subject to this ban represent only a small portion of the consumers total exposure to asbestos fibers. Even with this ban in place, it is not going to reduce the consumer exposure to asbestos fibers from joint compounds which are already in place, fireplace log ashes which are already in use, asbestos from automobile brake shoes, and all other sources of occupational, environmental, and consumer product exposures to asbestos. We would also anticipate that through the rulemaking process of proposal, reviewing comments, promulgating a final order, with some future effective date that the affected industry will begin making the conversions fairly early, resulting in the amount of material on the market containing asbestos being significantly reduced by the effective date. To ban the offering for sale of anything after the effective date would result in a need to purge the marketplace from those limited numbers of items which may exist at that time. This would place a tremendous enforcement burden on the Commission in that we would have to enforce a ban which would stop thousands of retail outlets from continuing to sell products already on their shelves as opposed to the responsibility for seeing that only a limited number of manufacturers have ceased manufacturing these products. Even if we are successful in stopping all retail sales, we doubt that it could be demonstrated that in the total asbestos picture, there would be any significant amount of consumer protection attained over and above that which would be attained by stopping the manufacture and future marketing of these products.

SGP 0009324

*U.S. GOVERNMENT PRINTING OFFICE: 1974 731-[illegible] 1-3

Another consideration in the issue of a prospective versus a retroactive ban is the nature of the hazard posed by asbestos. While an acute hazard may well warrant a retroactive banning situation, a chronic hazard, based upon long term exposure, would not appear to justify a retroactive ban. It is doubtful that the economic impact upon the industry or serious resource commitment on the part of the agency to enforce a retroactive ban is justified if in fact the hazard presented by asbestos is chronic rather than acute in nature. An examination of our experiences with Tris may be helpful in ascertaining practical problems with a retroactive ban involving a chronic hazard.

Since asbestos is a ubiquitous mineral which appears in many forms and is derived from many sources, we would suspect that almost any product could have the potential of at least carrying some asbestos contamination even though asbestos is not added as an ingredient. Therefore, we feel that the final ban on the above mentioned products containing asbestos should be based on some finite level of asbestos at which they will be defined as banned. We are not aware of how this could be defined at this time. However, we do not feel that that is reason to hold up the proposal of the ban. If we have no level available to include in the proposal we would then suggest that the proposal solicite comments from interested parties in an attempt to establish such a level in the final order.

ATTACHMENT 4

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT
SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : *Robert M. Hehir, Ph.D., Acting Deputy AED for Health Sciences*
Thru: Gale D. Wyer, Dir., Division of Chemistry

DATE: *June 3, 1977*

FROM : *W.K. Porter, Jr., A.G. Ulsamer, Ph.D. and D.M. Kirkpatrick, Ph.D., Division of Chemistry*

SUBJECT: *Difficulties Associated with Testing for Asbestos in Consumer Products*

There has been recent concern in CPSC over the possible inhalation of asbestos fibers from artificial fireplace embers, spackling compounds, and textured paints. This concern stems from the well documented correlation between asbestos and asbestosis and lung cancer in the industrial environment. The purpose of this memorandum is to discuss some of the difficulties associated with the analysis of asbestos in the home environment and in consumer products.

Identification and quantitation of airborne asbestos in the home environment is a difficult problem due to the presence of other fiber types and the size of the fibers of biological significance. This problem is compounded further if the asbestos is contained within the matrix of an actual product, since non-destructive (to asbestos) isolation techniques are ill-defined and in any event would have to be customized for each type of product. The isolation of airborne fibers is a relatively simple operation involving use

SGP 0009327

*U.S. GOVERNMENT PRINTING OFFICE: 1974 733-511/3604 1-3

of impactor devices or appropriately sized membrane filters. Filters with an average pore size of 0.1-0.2 microns would appear to be best in order to trap the smaller, respirable fibers.

Once the fibers have been collected, there is the problem of identification and quantitation. At present, there is no apparent agreement as to the best way in which to proceed with an overall analysis for asbestos. The National Bureau of Standards (NBS) has recently become involved with this problem and has set up a 3-day workshop (July 18-20, 1977) for interested parties in an attempt to better define asbestos and to discuss the techniques for its analysis.

Several analytical methods for fibrous asbestos are presently in use. For regulatory purposes, the Occupational Safety and Health Administration (OSHA) uses optical microscopy at 430X with phase contrast illumination. The procedure defines all fibers greater than 5 microns in length as asbestos. This technique makes no distinction between the various types of asbestos, or between asbestos and non-asbestos fibers. Furthermore, fibers smaller than 0.5-1 micron are not seen, thus not counted or identified.

A more definitive analytical procedure makes use of transmission electron microscopy in conjunction with X-ray fluorescence to determine morphology and elemental composition of collected fibers. Scanning electron microscopy with adequate resolution and X-ray fluorescence

would also be expected to provide fiber morphology and elemental composition. The scanning electron microscope with X-ray fluorescence may not be as definitive as the transmission electron microscope with X-ray fluorescence due to the limited spatial resolution of existing scanning electron microscopes. The various types of asbestos fibers can also be identified from a determination of their crystalline structure. The crystalline structure is of importance in that it permits identification of chrysotile from amphiboles and further differentiates the various amphiboles. Such structural information is obtainable with transmission electron microscopes fitted with a selected area electron diffraction device, this cannot be done with the scanning electron microscopes. Some investigators however, have reported successful identification of asbestos using scanning electron microscopy, coupled with X-ray fluorescence determination of the elemental composition of selected fibers.

Other approaches to analysis have included X-ray fluorescence and atomic absorption spectrophotometry. Both require bulk samples and only elemental composition is obtained. Neither method differentiates between asbestos and similar minerals, nor do they provide information on either crystalline structure or fiber and non-fiber distribution within a sample. The use of X-ray diffraction alone requires a bulk sample and other minerals which may be present could result in an inconclusive analysis. The use of thermal analysis also requires a bulk sample rather than individual fibers, and does not differentiate between

fibrous and non-fibrous forms of the same material.

From what has been presented, it would appear that electron microscopy in combination with X-ray fluorescence and selected area electron diffraction is the best approach for identification of single fibers as asbestos. The quantitation of asbestos is still questionable, however, because electron microscopy techniques analyze only a small portion of the total sample. This feature of electron microscopy, analysis of only a small portion of total sample, can lead to statistically biased quantitative data. Thus, it would appear that some combination of optical and electron microscopy is required to approximate asbestos fiber concentrations over the range of respirable fibers.

In addition to dealing with how to analyze asbestos fibers, we must also consider whether such work should best be done by contract or in house, as well as the relative costs of each. For in-house fiber identification work on complex samples, the primary limiting factor is the availability of suitable additional laboratory space. The present laboratory facility in FOB-8 cannot accommodate the necessary electron microscopy equipment required for this project. Of even greater importance are questions about the suitability of the FOB-8 facility for electron microscopy operations. Beyond this are the less serious factors of a lack of trained personnel and funding. It is felt however that, providing a suitably trained light microscopist is available, bulk pure asbestos samples (fireplace embers) can be analyzed in existing laboratory facilities. The limiting factor in the extramural contract

route is the relatively restricted number of available contractors.

A cost breakdown for the in-house analysis of asbestos would include the following:

1) Personnel

Two (2) additional staff members (an electron microscopist and a chemist) would be required. There might be additional costs for training depending upon expertise.

2) Instrumentation Options

a) Transmission electron microscope equipped with selected area electron diffraction, X-ray fluorescence detection, microprobe and scanning attachment: $183,000 or (depending on further comparisons of transmission and scanning microscopy).

b) Scanning electron microscope equipped with X-ray fluorescence detection: $97,000.

If the extramural contract route is chosen, the approximate cost breakdown would be as follows for a 1 year effort assuming that the contractor is fully equipped:

Personnel (4)	$69,000
Equipment	0
Supplies	2,000
Overhead (100%)	71,000
	$142,000

For this price, it is estimated that isolation methods for 4 categories of asbestos-containing products could be developed and a total of 90 samples could be analyzed.

In conclusion, it is our feeling that further information is needed before a final choice between methods of analysis is made. The NBS workshop should provide a more complete overview of current technology. Immediate analytical needs with the exception of bulk, pure asbestos samples cannot be handled in house due to lack of space, equipment, and funds. Such needs could be handled on a sample to sample basis by contract until in-house capabilities for analysis can be developed.

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Don Clay, Acting AED for Engineering & Sciences DATE: June 3, 1977

THRU : Robert M. Hehir, Actg. Deputy AED for Health Sciences

FROM : Steven Bayard, Ph.D./SPSC

SUBJECT: Risk of Respiratory Cancer Due to Low Level Exposure to Asbestos from Spackling & Joint Taping Compounds

Summary & Discussion

A model for lifetime risk assessment of death from respiratory cancer due to consumer use of asbestos containing wall taping compounds is presented. Based on heavy exposure four times a year for 1 year, increased risk of death from respiratory cancer is estimated at 10 per million. For five years of exposure at these levels, however, the risk increases geometrically and is estimated at 1,000 per million or 1 per thousand.

For consumers using ready mixed spackling compounds to fill a few small holes and doing a little sanding, the model predicts negligible risk.

No quantitative risk assessment was made for asbestos exposure from the artificial ash adorning the emberized gas logs since there are no known measurements of the airborne fiber content. It can be assumed, however, that whatever air concentrations are present, they expose the home occupant to a repository of free fibers continuously vs. only intermittent exposure for the wall taping compounds. The risk from these ashes, therefore, may be considered at least as high as that from the wall taping compounds.

A. Assumptions

In order to compute a risk assessment of the use of asbestos containing wall joint compounds, many assumptions had to be made. The model used is mainly that of Enterline and Henderson (1976), which in turn was derived from data on amosite asbestos factory workers and asbestos insulation workers (Selikoff, Hammond and Seidman, 1973). Measurements of asbestos fibers longer than 5 microns from work with wall taping compounds were taken by Rohl et al (1975). Projections of consumer use of taping compounds are my own and age central death rates from respiratory cancer were based on the 1970-71 vital statistics of the United States.

The assumptions used in the risk assessment model are presented below; references for each are given.

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1. The dose-response relationship between asbestos and lung cancer is linear (Enterline and Henderson, 1976; McDonald, et al, 1974). This hypothesis assumes no threshold.

2. Time to tumor is dependent on dose and can be described by a log normal distribution with median time to tumor t :

$$t = 98.65\left(\frac{1}{D}\right)^{1/3}$$

where D = 8-hour time weighted average dose in fibers/cc and a standard deviation of 1.5 f/cc. (Enterline and Henderson, 1976, based on Jones and Grindon, 1975).

3. Competing risks of death for the first 40 years following exposure are considered to be normal.

4. Risk of asbestos caused death after the first 40 years following exposure is considered to be zero.

5. Effect of dose is cumulative and is assumed to have the same effect as if that dose had been accumulated in the first year of exposure.

6. Intermittent exposure with occasional high peaks has the same cumulative effect as continuous exposure at double the dose (Enterline, et al, 1972; Nicholson, 1976).

While assumptions 1-5 may seem a bit unclear, the total effect is to present a cumulative dose-response curve of the form log dose-log response. This is shown in Figure 1. Explanation of how these figures were derived is given below. It is emphasized, however, that this model is to be used for low exposure estimates. It does not fit the data for high or long term exposure data.

B. Derivation of Total Cases Caused by Asbestos Exposure

Besides the assumptions 1-6 above, the major data used to estimate the total cancer deaths attributable to dose were the Selikoff data on 294 factory workers who had been exposed to asbestos for 3-11 months during the years 1941-1945. Estimates of the concentration of asbestos dust during this period averaged 30 f/cc. Since the average exposure was only 5/8 year, the equivalent concentration was figured at 18.75 f/cc /day on a 1 year basis. By assumption 2, the median time to death from respiratory cancer is 37.1 years. Also, by assumption 2, the log normal distribution shows that for the 28 years of followup used in the Selikoff paper only 24.4% of these deaths would have occurred. Since the adjusted relative risk of these workers was 2.95 (Enterline, 1976), and the age central death rate from respiratory cancer (ages 35+) was 850/million, the number of respiratory deaths which could have been caused by the asbestos exposure was the solution to

$$\frac{28(.000850) + .244\ X}{28\ (.000850)} = 2.95$$

or X = .190205 or 190,205 deaths/million. But, since only 40 years of exposure are considered (assumptions 3 and 4), assumption 2 allows only 57.3% or 109,000 lifetime cancer respiratory deaths per million exposed.

By assumption 1, the number of potential cases by dose can then be calculated and risk estimates can be derived from these. This is shown in Table 1, along with the calculated relative risks. Here it is seen that excess deaths and relative risks do not increase linearly with increasing dose but in a geometric manner.

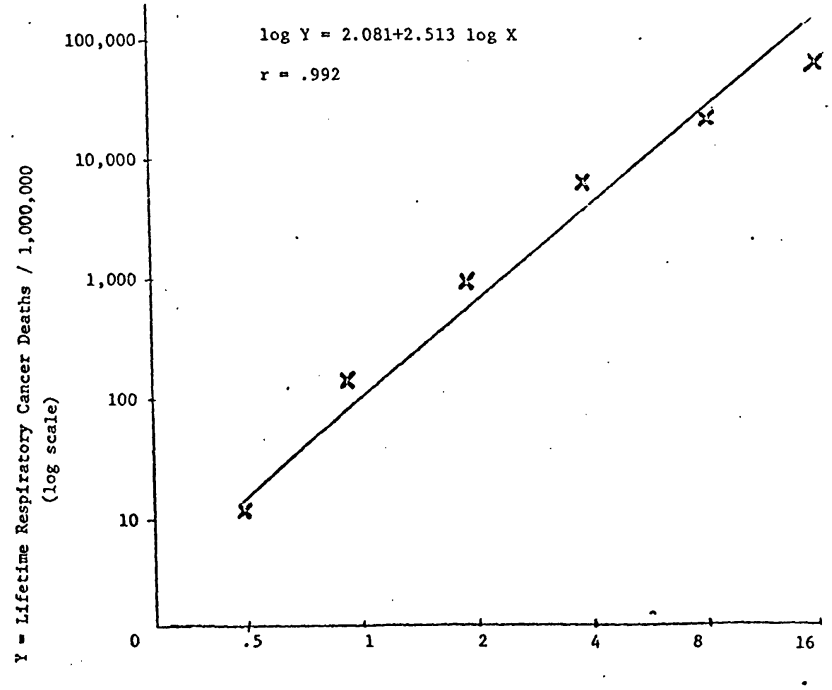
C. Estimates of Exposure Levels of Consumer Users of Wall Taping Compounds

Rohl (1975) measured peak fiber concentrations of ten drywall taping compounds during sanding, dry mixing, and floor sweeping. The average peaks were as high as 47 f/cc with the highest individual peak of 59 f/cc. Based on these peaks the 8-hour time weighted average was estimated as 10 f/cc. Taken with assumption 6 that high intermittent exposure was estimated to have doubled the effect of continuous exposure, this estimate was increased to 20 f/cc. If there are four uses projected per year, the estimate of yearly equivalent is

$$\frac{20\ \text{f/cc/day} \times 4\ \text{days}}{200\ \text{days/year}} = .4\ \text{f/cc/day for 1 year}$$

FIGURE 1. Response vs. Dose for Low Level Asbestos

Exposure. Asbestos Induced Respiratory Cancer Deaths per Million Lifetime vs. Daily Exposure (f/cc) for 1 Year.
Estimates Based on the Model



SGP 0009337

TABLE 1. Lifetime (40 Year) Risk Estimates of Respiratory Cancer Deaths by Dose for a 1 Year Equivalent Exposure. Median Latent Periods and Relative Risks are Included.

8-Hour Avg. Daily Exposure Level-D f/cc	Latent Periods Years $t=98.65(\frac{1}{D})^{1/3}$	Potential Cases/ Million (see text)	Proportion Developed in 40 Years (log normal)	Asbestos Induced Respir. Cancer Deaths/ (3)x(4)	Relative Risk $\frac{(3)+850x40}{850x40}$
.5	124.3	5,072	.0026	13	1.00038
1	98.6	10,145	.0130	132	1.00388
2	78.3	20,290	.0488	990	1.02912
4	62.1	40,578	.1488	6,038	1.17759
8	49.3	81,155	.3030	24,590	1.72324
16	39.1	162,310	.4776	77,519	3.27997
18.75	37.1	190,205	.5727	109,000	4.20588

Thus, based on the results of the model, Table 1, those four uses in 1 year with heavy exposure will cause an additional 10 lifetime respiratory cancer deaths/million. Continued use for five years will, by assumption 5, raise that estimate to 990 (see Table 1) deaths per million.

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