Federal Register

FRIDAY, JULY 29, 1977

PART V



CONSUMER PRODUCT SAFETY COMMISSION

■

RESPIRABLE FREE-FORM ASBESTOS

Proposed Rulemaking

8001 0068

SCF-ALLF-06630

HFM - 003366

"incombustible". There are six basic varieties of asbestos minerals which are found in fiber form: chrysotile (the most common variety, and that ordinarily found in asbestos-containing products), amosite, crocidolite, actinolite asbestos, tremolite asbestos, and anthophyllite asbestos. Most of the world supply of commercial asbestos is chrysotile, the fibrous form of serpentine.

Asbestos minerals all have nonfibrous 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. (June 3, 1977, CPSC/Health Sciences, (7).)

Asbestiform minerals are generally characterized by the following properties: their ability to be broken down into smaller fibrils; great elongation; heat resistance; resistance to biotransformation; flexibility; high-tensile strength, and spinnability.

DISEASES RELATED TO ASBESTOS EXPOSURE

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 or are encapsulated in the product composition. A potential health risk occurs when asbestos fibers become airborne, such as by mixing, sanding, or cleanup operations when using patching compounds. A single individual engaged in such a process may be exposed to potential asbestos fiber inhalation risk and may also cause exposure of other individuals in the vicinity.

Certain malignancies are related to exposure to 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 (IARC, 22).

Some conclusions from a Review (Huff, 21) of the major literature on asbestos are:

1. Under experimental conditions, asbestos is pathogenic in animals. Asbestos is known to be carcinogenic in humans, causing lung cancer, mesotheliomas, and pleural lesions.

2. The mechanisms of pathogenicity are poorly understood. The etiological significance of fiber size or type is controversial, and the physiochemical properties of various asbestos types as related to biological effects are incompletely defined.

3. 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.

4. Animal models necessary to accurately predict the potential effects of asbestos in humans have not been developed.

5. 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.

6. Malignancies can arise 20 years or more after occupational exposure; however, they also reportedly result from indirect, non-occupational exposure.

7. Available data indicate that asbestos is a widespread environmental pollutant.

The most common diseases that might result from asbestos exposure are:

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. No report of this with frequency detected.

Asbestosis may be evidenced, in its advanced stages, by characteristic manifestations 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.

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 occurring 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, or rectum usually do not become clinically evident until more than 20 years have passed from onset of exposure. The long latency period 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 20 years or more from initial exposure. 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.

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, (1)).

In humans, the latency period for chemical carcinogens may well extend from 20 to 40 years or more. Analogous latency periods have also been demonstrated in animal studies. This means that the disease may undergo a long period of development before a tumor is medically detected.

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.

CANCERS ASSOCIATED WITH ASBESTOS

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 (41); Enterline, de Coufle and Henderson, 1972 (12)). As expected, the high risk of bronchogenic carcinoma and mesothelioma persisted among groups of factory workers. Moreover, these later studies confirmed the excess gastrointestinal cancer that had been suggested earlier, and extended the spectrum of asbestos related cancers.

(1) 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 (42); and Nicholson, 1976 (35)), and among users of these products (Selikoff, Hammond and Seidman, 1973 (41)). The exact percentage varies with circumstances of exposure, age of the workers, duration of the workers' exposure 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 and Hammond

8001 0069

HFM - 003367

(39) 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 (17)) where, again, it was found that nonsmoking 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 aprproximately eight times the risk of developing lung cancer compared to other cigarette smokers who are not occupationally exposed to asbestos.

(2) Pleural and peritoneal mesotheliomas. In 1960 Wagner, Sleggs and Marchand (47) 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, (13) 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 records of cases from hospitals near one large asbestos factory indicate that it must be very common (Barrow, Conston, Livornese and Schalet, 1967, (3)). 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, (17); Selikoff, Hammond and Seidman, 1973, (41)). 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, 1976, (30)).

A recent study of mesotheliomas diagnosed in a hospital located in the vicinity of a shipyard reported an increased incidence of diffuse malignant mesothelioma since World War II, not only among exposed shipyard workers, but also among individuals with no occupational exposure. (Hasan. (20)).

(3) 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, (41), Elmes and Simpson, 1971 (9)). 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, 1972 (45); Newhouse and Berry, 1975 (30)), and of the esophagus (Selikoff, Hammond, and Seidman, 1973 (41)). 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.

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, 31)).

In 1930, the British Home Office Survey by Merewethier and Price (28), 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. The objective of the Regulations, 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, (35)).

By 1960, through epidemiological studies, an association between asbestos exposure and mesothelima had been demonstrated (Wagner (47)) and was soon substantiated by additional epidemiological studies (Newhouse (31), Selikoff (37)) 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 implementation 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 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, 1975, (6).)

The National Institute of Occupational Safety and Health (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 OSHA permanent asbestos standard in 1972, results of a new evaluation of the earlier British factory worker population showed many abnormal X-ray findings of the lungs and pleura.

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 (1).

It is also reported that clinical data are becoming available concerning asbestos lung scarring in individuals exposed at much lower than occupational levels. (Anderson, (1)).

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. (Anderson, (1)).

Non-occupational, indirect occupational exposure, or intermittent or brief exposures

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

In inhalation exposure experiments, rats exposed to asbestos dust were reported to have developed tumors, including mesotheliomas, from as little as a single 7 hours exposure (48).

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 (16); Webster, 1973, (49)). In such circumstances, it appears that the lifetime exposure was less than 100 fibers-years/ml. The same discrepancy between present projected exposures and the risks of asbestos cancer exists when considering cases of mesothelioma resulting from household contact with asbestos among members of families or asbestos workers (Lillington, 1974 (25)), 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 workmates. In 1968, Harries of

Note time factor.

8001 0070

HFM - 003368

the Royal Navy (18) reported cases of mesothelioma among shipyard workers at the Royal Navy dockyard in Devenport, in trades which did not directly involves 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 (Whitewell and Rawcliffe, 1970 (50)); McEwan et al, 1970 (27)); Stumphius, 1971 (46); Greenberg and Davies, 1974, (16). 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 (44); Fletcher, 1972, (15)).

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 mesotheliomas. (Fletcher, 1972 (15); Edge, 1975 (6)). 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, (35)).

It is extremely difficult, if not impossible, to document brief exposures reported to have occurred 20 or more years before the onset of symptoms. 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.

(Much of the foregoing information on asbestos-related diseases was prepared by the Occupational Safety and Health Administration (OSHA) and published at 40 FR 47651, October 9, 1975. "Occupational Exposure to Asbestos" has been helpful in preparing this proposal (6)).

CONSUMER EXPOSURE

Reports of the hazard, or potential for hazard, from exposure to asbestos resulting from use of asbestos-containing consumer products are very limited. The only known quantitative study of asbestos levels in products regulated by this Commission was reported by Rohl, et al (1975 (36)), 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, and sanding and cleanup operations) may expose the user and other members of the household to * * * significant concentrations of asbestos".

Asbestos in the household presents a great risk due to the presence in the household of persons, such as children, who may be particularly vulnerable to carcinogens. It is generally observed that, because of the long latency period, exposure to inhalable asbestos in the some can be life-shortening for children. In this connection, Dr. Paul Kotin of the Johns-Manville Company, stated to an OSHA Advisory Committee that, in his laboratory experience, cancer can be most readily induced in the young (14). This point was further clarified at a briefing before the Commission on June 9, 1977 during which Dr. Kotin stated his belief that the young are doubly vulnerable to carcinogens—first, because of a long lifetime ahead and secondly because young children grow at a more rapid rate than adolescents. Thus, he explained, "A rapidly dividing cell, a cell which has a turnover time shorter when compared to another cell, is perhaps more likely to be subject to the action of a carcinogen."

The Commission concludes that exposure to asbestos from either patching compounds or artificial emberizing materials presents an unreasonable risk of injury sufficient that either product standing alone should be banned. The Commission also notes that consumers are exposed to asbestos from sources other than the products proposed for banning. The asbestos fibers are present in the general environment has been demonstrated by studies such as those by Huff (21), Selikoff, and Harwood (38). Consumers who are exposed to asbestos fibers from artificial embers and ash and patching compounds thus receive additional doses of asbestos and can be presumed to face a greater risk than members of the population who are not so exposed, and a greater cumulative risk than if no asbestos were present in the general environment.

A bibliography of additional scientific and medical literature reviewed by the Commission is noted below and an annotated bibliography is available for review in the Office of the Secretary.

"THRESHOLD" LIMIT

Because of the variability of individual response to carcinogens and other factors, the concept of a "no effect" or "threshold level" of exposure 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 (35).

In the Proceedings of the Nineteenth Meeting of the Interagency Collaborative Group on Environmental Carcinogenesis devoted to Threshold Evaluated held at the National Institute on August 14, 1975 (23), Dr. H. F. Kraybill, Chairman, states that:

> Investigators in the field of carcinogenesis have doubted the existence of a threshold for a carcinogen or at least have maintained that there are no acceptable procedures for assessment of a safe-dose for a carcinogen (i.e. threshold). Thus, it has been concluded that it is not feasible to prove or disprove the presence or absence of a threshold-dose through downward extrapolation from the dose-response curve in regions of detectably significant dosage. The conservatism in this direction is prompted by certain realizations as to the self-replicating nature of the cancer cell, the possible existence of irreversible effects, the aspects of lasting alteration induced by a tumor-producing event, the perpetuation of the neoplastic lesion after the original inducer has disappeared from the milieu, and finally, the possibility of cancer induction from a mutation in a somatic cell.

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 respirable free-form asbestos would be considered safe.

OTHER FEDERAL AGENCY ACTIONS TO REGULATE EXPOSURE TO ASBESTOS

The Occupational Safety and Health Administration of the Department of Labor (OSHA), the Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA) each has a responsibility to regulate exposure to asbestos.

OSHA regulates exposure to asbestos by workers in all industrial and commercial settings except agriculture. The regulation (29 CFR 1910.1001) states that the 8-hour time-weighted average airborne concentrations of asbestos fibers to which any employee may be exposed shall not exceed two fibers, longer than 5 micrometers, per cubic centimeter of air. In 1975, OSHA proposed an amendment to lower exposure to .5 fibers per cubic centimeter of breathing air. An OSHA proposal to regulate exposure by workers in the building construction industries is expected to be ready for publication within the next few months.

FDA has banned the use of asbestos in filters used in the formulation of parenteral drugs (21 CFR 211.40(j)) and, prior to the establishment of the Consumer Product Safety Commission, banned the use of asbestos in fabric used for general wearing apparel. (16 CFR 1500.17(a)(7)).

EPA regulates asbestos which is released into the air and into the water. These regulations (40 CFR 61, 427, 436) do not set standards for allowable amounts of asbestos in the breathing air but rather minimize or eliminate emissions from industrial locations. These regulations specifically prohibit the presence of asbestos in emissions from plants manufacturing materials for use in building roadways and in manufacturing items such as cloth, textiles, cement, fireproofing and friction products, paper, felt, floor materials, paints, adhesives,

8001 007]

HFM - 003369

sealants, caulks, and a variety of other items.

ASBESTOS REGULATIONS AFFECTING CONSUMER PRODUCTS IN OTHER COUNTRIES

In June, 1976, Canada banned the sale and importation of asbestos-containing products for use by children. In April, 1976, England introduced a voluntary labeling scheme for products containing asbestos.

In Sweden, the use of asbestos-cement products for new work was prohibited as of July, 1976, and for maintenance in existing structures will be discontinued by the end of 1977. Manufacturing of these products is not prohibited. According to the Swedish Standard for Occupational Exposure:

"The TLV of 2f/ml was lowered to 1f/ml as of January, 1977, and is scheduled to be further reduced to 0.5 f/ml by 1 January, 1978." (June 27, 1977, Asbestos Information Association (2).)

PART B. PATCHING COMPOUNDS

Asbestos fibers are used in patching compounds to reinforce a material as it cures, to control shrinkage and cracking, and to reinforce the material as the compound is alternately exposed to heat, cold and moisture after curing. Asbestos also provides a measure of sound and heat insulation to the material, and enhances the workability of the compound, particularly during troweling. The properties considered by industry to be most valuable in the preparation of patching compounds are: asbestos is not affected by fungus, mildew, vapor, etc. and it has low density; good adsorption qualities; high resistance to electricity, fire and alkaline substances; low magnetic permeability; and it is not a smooth fiber.

Patching compounds are mixtures of talcs, pigments, clays, casein, ground marble, mica or other similar materials with water and a binding material such as asbestos. Patching compounds are available in dry form (to be mixed with water by the user) or a ready-mix paste form. The product is used to cover, seal or mask cracks, joints, holes and similar openings in the trim, walls, and ceilings, etc. of building interiors.

In patching compounds, asbestos is released when using the dry form and mixing it with water, or by sanding or scraping the dried patching compound in the process of finishing and smoothing the surface as in remodeling and in cleanup operations. Such activities may present a hazard to consumers when the activity is performed either by a consumer or a professional user of these products.

Patching compounds covered by this ban are those that are customarily produced or distributed for sale to or for the personal use, consumption or enjoyment of consumers in or around a permanent or temporary household or residence, a school, in recreation or otherwise. Patching compounds that are labeled as, marketed, and sold solely for industrial use would not be within the scope of this ban. Information available to the Commission indicates that most patching compounds for industrial use are distributed in such ways that consumers have access to these products. Moreover, merely labeling a patching compound for industrial or professional use only, would not exclude such articles from the ban. If the manufacturer, distributor or retailer fosters or facilitates the product's sale to or use by consumers, the product would be considered a consumer product within the scope of this ban.

Recent court cases have examined the statutory definition of consumer product and indicate that the presence of a product in a consumer environment would be considered a primary determinant of whether that product is a consumer product under the CPSA.[1] In considering the statutory definition and its legislative history in light of these cases, it appears to the Commission that substantially all patching compounds are consumer products under the CPSA.

EVALUATION OF THE RISK

In assessing the degree and nature of the risk of injury to consumers, the Commission has reviewed experimental data and human experience information. In addition, on the basis of data by Rohl, et al. (36), the Commission's Health Sciences staff has calculated an assessment of the risk of consumer exposure which is available at the Office of the Secretary. The calculations are based on the application of a theoretical model (modification of that by Enterline and Henderson, 1976, (11), to epidemiological data cited in the literature. (Skeikoff, Hammond and Seidman, 1973; (41). For purposes of this assessment, the Commission considered the use of patching compounds by a consumer, for six hours a day four times a year, to be a high yet reasonably foreseeable yearly exposure. The increased risk of death from respiratory cancer induced by this yearly exposure is estimated at between 10 and 2,000 per million. For five years of exposure at these levels, the risk increases geometrically and is estimated at between 1,000 and 12,000 per million. Based on current information, the Commission estimates that the lower estimate of 10 per million is closer to the actual risk for a one year exposure. Nevertheless, in view of the seriousness of the injury and the cumulative effects of asbestos exposure, even this minimum figure represents an unacceptable risk. The Commission believes that reducing exposure to respirable free-form asbestos in the home represents a substantial decrease in risk to consumers, since, for many people, the major exposure to inhalable asbestos is in the home.

INDUSTRY RESOURCE CONSIDERATIONS

ASSESSMENT OF SUBSTITUTES

The elimination of asbestos from patching compounds would necessitate the use or development of substitutes which are similar to asbestos, or which impart similar qualities to the product. Several substitutes for asbestos in patching compounds are in current use. The primary economic concerns focus on the effect of substitutions on the utility derived from the product (mainly in terms of performance characteristics) and on the prices of patching compounds.

Asbestos possesses a unique combination of strength, pliability, and heat-cold-, and moisture-resistant qualities; substitutes which have been tested in these product categories often do not last as long, are less workable, and may take a long time to perfect (asbestos-free spackling compounds were at least five years in development). Some products not readily reformulable may be dropped from the market. Availability of substitutes may also be limited temporarily on a regional basis, depending on the paint and building materials industries' ability to reformulate asbestos-containing products. Many producers are, however, either presently marketing asbestos-free materials or expect to in the near future.

Substitution may also lead to higher costs of production and prices for some of these products. The total cost to most patching compound manufacturers of chemically reformulating products is not expected to be large relative to sales; it is, however, expected that some cost increases will occur which will likely be passed on in the form of higher prices (this may be 10 to 15 percent). (Preliminary Economic draft study—subsection on Spackling/Joint Compounds, OSHA, (7).)

Inventories of existing products in distribution channels may vary over a wide range. Similarly, inventory turnover cycles at various points in distribution may vary considerably.

These conditions may be further affected by OSHA's course of action on asbestos. Should a commercial-industrial fiber limit standard be imposed, the industry would probably eliminate asbestos from paints and other building materials as well as from patching compounds.

ENVIRONMENTAL ISSUES

The Commission has studied the environmental effects of this proposal as required by the National Environmental Policy Act. An environmental assessment is on file at the Office of the Secretary of the Commission and indicates, by negative declaration, that no significant effects on the environment are foreseen by the proposals issued below.

Asbestos is an ubiquitous mineral fiber that has been used for many years in the U.S. in occupational settings and in building construction. This has led to low-level, but widespread exposure of the public. Asbestos is found in the air, water, and land (in minute concentrations relative to those found in or near asbestos mines or processing facilities).

The elimination of asbestos from patching compounds available to or used or enjoyed by consumers will significantly reduce household fiber release from mixing, sanding, and clean-up. It will not, however, significantly reduce fiber

[1] *United States of America* v. *The Anaconda Co.*, et al., Misc. No. 77-0024 (D.D.C.), June 15, 1977. *Kaiser Aluminum & Chemical Corp.* v. *CPSC*, Civ. No. 76-44 (D.Dela.) March 11, 1977.

8001 0072

HFM - 003370