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Occupational Safety and Health Administration

29 CFR Parts 1910 and 1926

Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite; Final Rules

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Parts 1910 and 1926

[Docket No. H-033C]

Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite

AGENCY: Occupational Safety and Health Administration, U.S. Department of Labor.

ACTION: Final rules.

SUMMARY: In these final standards the Occupational Safety and Health Administration (OSHA) amends its present standard (29 CFR 1910.1001) regulating occupational exposure to asbestos. The standards published today establish a permissible exposure limit of 0.2 fiber per cubic centimeter of air (f/cc), determined as an 8-hour time-weighted average airborne concentration. The standards apply to all industries covered by the Occupational Safety and Health Act, including the construction and maritime industries and general industry. Separate standards and separate statements of reasons (Summary and Explanation sections) have been developed to apply to general industry (including maritime) and to construction, because the differences in exposure and workplace conditions in general industry and construction worksites warrant separate treatment. The standards will be codified in 29 CFR Parts 1910 and 1926, OSHA's General Industry and Construction standards, respectively. The basis for promulgation of these regulations is a determination by the Assistant Secretary that employees exposed to asbestos, tremolite, anthophyllite, and actinolite face a significant risk to their health and that these final standards will substantially reduce that risk. The record in this rulemaking demonstrates that employees occupationally exposed to asbestos are at risk of developing such chronic diseases as asbestosis, lung cancer, pleural and peritoneal mesothelioma, and gastrointestinal cancer.

The standards also provide for requirements for methods of compliance, personal protective equipment, employee monitoring, medical surveillance, communication of hazards to employees, regulated areas, housekeeping procedures, and recordkeeping. An "action" level of 0.1 f/cc as an 8-hour time-weighted average is established as the level above which employers must initiate certain compliance activities, such as employee training and medical surveillance. Where the employer can demonstrate, by means of exposure monitoring results or historical data, that the exposures of his or her employees do not exceed the action level, the employer is not obligated to comply with many of the standard's requirements. The 0.2 f/cc 8-hour limit reduces significant risk from exposure and is considered by OSHA, based upon substantial evidence in the record as a whole, to be the lowest level feasible.

EFFECTIVE DATE: The amended standards published today take effect July 21, 1986, except the following paragraphs which contain information collection requirements which are under review at the Office of Management and Budget: 29 CFR 1910.1001 (d)(2), (d)(3), (d)(5), (d)(7), (f)(2), (g)(3)(i), (j)(5), (l), and (m); 29 CFR 1926.58 (f)(2), (f)(3), (f)(6), (h)(3)(i), (k)(3), (k)(4), (m), and (n).

ADDRESS: For additional copies of these final standards, contact: OSHA Office of Publications, U.S. Department of Labor, Room S-4203, 200 Constitution Avenue, NW., Washington, DC 20210, Telephone (202) 523-9667.

FOR FURTHER INFORMATION CONTACT: Mr. James F. Foster, Director, Office of Information and Consumer Affairs, OSHA, U.S. Department of Labor, Room N-3637, 200 Constitution Avenue, NW., Washington, DC 20210, Telephone (202) 523-8151.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. The Format of This Document (the Preamble)

The preamble accompanying these revised standards is divided into 23 parts, numbered I through XIII. The following is a table of contents:

References to the rulemaking record are in the text of the preamble, and the following abbreviations have been used:

1. Ex: Exhibit number in Docket H-033C. Docket H-033C is located in Room N3670 at the Department of Labor.

2. TR: Transcript date and page number.

B. Summary

Pursuant to sections 4(b)(2), 6(b), 8(c), and 8(g) of the Occupational Safety and Health Act of 1970 (the Act) (84 Stat. 1592, 1593, 1598, 1599; 29 U.S.C. 653, 655, 657), the Construction Safety Act (40 U.S.C. 333), the Longshoremen's and Harbor Workers' Compensation Act (33 U.S.C. 941), the Secretary of Labor's Order No. 9-83 (48 FR 35736), and 29 CFR Part 1911, these final standards hereby amend and revise the current asbestos standard, 29 CFR 1910.1001.

This action follows publication of proposed notices on November 4, 1983 (48 FR 51086) and on April 10, 1984 (49 FR 14116) and the holding of a public hearing to provide the public with an opportunity to comment on these proposed revisions. The hearings were held from June 19 to July 10, 1984, in Washington, DC. More than 25,000 pages of testimony and comments were received into the record of this rulemaking and have been analyzed by the Agency in developing these final standards. Based on this record, OSHA has determined that employees exposed to asbestos, tremolite, anthophyllite, and actinolite at the existing permissible exposure limit (PEL) of 2 fibers per cubic centimeter of air (2 f/cc) at worksites in the construction and maritime industries and in general industry workplaces face a significant risk to their health and that these final standards will substantially reduce that risk. Evidence in the record of this proceeding has shown that employees exposed at the revised standards' PEL of 0.2 fiber/cc remain at significant risk of incurring a chronic exposure-related disease, but considerations of feasibility have constrained OSHA to set the revised PEL at the 0.2 fiber/cc level.

The standard issued in 1971 defined asbestos as chrysotile, crocidolite, amosite, tremolite, anthophyllite, and actinolite. All of these minerals represent a hazard to workers, and the revised standard continues to regulate all of them. However, some forms of these minerals are no longer included in the definition of the word "asbestos". The regulatory text clearly specifies that the standards apply to occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. In the preamble, however, where the word "asbestos" is used this should be interpreted as applying to tremolite, anthophyllite, and actinolite as well.

proceeding, including materials discussed or relied on in the November 1983 and April 1984 notices, the record of the informal hearing, and all written comments and exhibits received.

III. Pertinent Legal Authority

The primary purpose of the Occupational Safety and Health Act (29 U.S.C. 651 et seq.) (the Act) is to assure, so far as possible, safe and healthful working conditions for every American worker over the period of his or her working lifetime. One means prescribed by the Congress to achieve this goal is the mandate given to, and the concomitant authority vested in, the Secretary of Labor to set mandatory safety and health standards. The Congress specifically mandated that:

> The Secretary, in promulgating standards dealing with toxic materials or harmful physical agents under this subsection, shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of standards, and experience gained under this and other health and safety laws. (Section 6(b)(5))

Where appropriate, OSHA standards are required to include provisions for labels or other appropriate forms of warning to apprise employees of hazards, suitable protective equipment, exposure control procedures, monitoring and measuring of employee exposure, employee access to the results of monitoring, appropriate medical examinations, and training and education. Moreover, where a standard prescribes medical examinations or other tests, they must be available at no cost to the employee (Section 6(b)(7)). Standards may also prescribe recordkeeping requirements where necessary or appropriate for the enforcement of the Act or for developing information regarding occupational accidents and illnesses (Section 8(c)).

In vacating OSHA's revision to its benzene standard, the Supreme Court required in *Industrial Union Department, AFL–CIO v. American Petroleum Institute*, 448 U.S. 607, 65 L. Ed. 2d 1010, 100 S. Ct. 2844 (1980), that before the issuance of a new or revised standard pursuant to section 6(b)(5) of the Act, OSHA must make two threshold findings. OSHA must find that a significant risk exists under the current standard and that the issuance of a new standard would reduce or eliminate that risk. The Court stated:

> We agree . . . that subsection 3(8) requires the Secretary to find, as a threshold matter, that the toxic substance in question poses a significant health risk in the workplace and that a new, lower standard is therefore "reasonably necessary or appropriate to provide safe and healthful employment and places of employment." 448 U.S. 607 at 614–15, 65 L. Ed. 2d 1010 at 1018–19.

The Court also stated:

> . . . Before he can promulgate any permanent health or safety standard, the Secretary [of Labor] is required to make a threshold finding that a place of employment is unsafe—in the sense that significant risks are present and can be eliminated or reduced by a change in practices. . . . [448 U.S. at 642, 65 L. Ed. 2d at 1033]

The decision, although it recognized the uncertainties involved, indicated that the determination of "significant risk" should, if at all possible, be established on the basis of an analysis of the best available evidence through such means as quantitative risk assessments. However, in making that determination, the Supreme Court in its general guidance for the future noted that:

> . . . The requirement that a "significant" risk be identified is not a mathematical straitjacket. It is the Agency's responsibility to determine, in the first instance, what it considers to be a "significant risk." [448 U.S. at 655, 65 L. Ed. 2d at 1043]

It pointed out that while OSHA:

> . . . must support its findings that a certain level of risk exists by substantial evidence, we recognize that its determination that a particular level of risk is "significant" will be based largely on policy considerations. [448 U.S. at 656, 65 L. Ed. 2d at 1043, n. 62]

Finally, the Court pointed out that:

> . . . OSHA is not required to support its findings that a significant risk exists with mathematical precision or certainty.
>
> Although the Agency's findings must be supported by substantial evidence . . . OSHA [has] some leeway where its findings must be made on the frontiers of scientific knowledge. [448 U.S. at 656, 65 L. Ed. 2d at 1043]

In the only concrete example of significance, the Court stated:

> Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal, a reasonable person might well consider the risk significant and take appropriate steps to decrease or eliminate it. [Id. at 655, 656 L. Ed. 2d at 1043]

After OSHA has determined that a significant risk exists and that such risk can be reduced or eliminated by the proposed standard, it must set the standard "which most adequately assures, to the extent feasible on the basis of the best available evidence, that no employees will suffer material impairment of health . . ." (section 6(b)(5) of the Act). The Supreme Court has interpreted this section to mean that OSHA must enact the most protective standard possible to eliminate a significant risk of material health impairment, subject only to the constraints of technological and economic feasibility. (*American Textile Manufacturers Institute, Inc. v. Donovan*, 452 U.S. 490 (1981)).

Moreover, section 4(b)(2) of the Act provides for OSHA standards to apply to construction, maritime, and other workplaces where the Secretary determines that these standards are more effective than the existing standards that would otherwise apply to these workplaces. The Secretary so finds, and these standards will therefore apply to all workplaces where the Secretary has authority to regulate.

IV. Health Effects

A. Overview of Asbestos-Related Diseases

OSHA is aware of no instance in which exposure to a toxic substance has more clearly demonstrated detrimental health effects on humans than has asbestos exposure. The diseases caused by asbestos exposure are life-threatening or disabling. Among these diseases are lung cancer, cancer of the mesothelial lining of the pleura and peritoneum, asbestosis, and gastrointestinal cancer. Of all of the diseases caused by asbestos, lung cancer constitutes the greatest health risk for American asbestos workers. Lung cancer has been responsible for more than half of the excess mortality from asbestos exposure in some occupational cohorts.

The relationship between lung cancer and asbestos exposure has been established in numerous epidemiologic studies of diverse groups. Asbestos-induced lung cancer usually has a latency period in excess of 20 years, and this cancer may be manifested at a younger age than is true for lung cancer victims who are not exposed to asbestos (Craighead et al., Ex. 84–003). Few cases of lung cancer are curable, despite advances in medical and surgical

oncology. Only 9 percent of lung cancer patients survive for 5 or more years after diagnosis (American Cancer Society, Ex. 84-160). Asbestos exposure acts synergistically with cigarette smoking to multiply the risk of developing lung cancer.

Many studies have also shown conclusively that mesothelioma is associated with asbestos exposure. In some asbestos-exposed occupational groups, 10-18 percent of deaths have been attributable to malignant mesotheliomas. Malignant mesotheliomas of the pleura and peritoneum are extremely rare in persons not exposed to asbestos. Generally, a latency period of at least 15-30 years is required before mesotheliomas are observed in an occupational cohort, although some victims of mesothelioma have had latency periods exceeding 40 years (Craighead et al., Ex. 84-033). This form of cancer is rarely curable and is usually fatal within a year after diagnosis.

Some epidemiologic studies of asbestos-exposed persons have shown increases in esophageal, stomach, colo-rectal, kidney, laryngeal, pharyngeal, and buccal cavity cancers. Although the increased risk of incurring cancers at these sites is not as great as the increased risk of lung cancer and mesothelioma, the increase is of considerable importance because of the high background rates, and therefore the large number of victims, associated with some of these tumors in the general population. For example, a 50 percent increase in a common cancer such as colo-rectal cancer results in many more deaths than a 10 percent increase in a rare cancer.

Asbestosis is pulmonary fibrosis caused by the accumulation of asbestos fibers in the lungs. The adverse effects of asbestosis range from shortness of breath during exertion to cyanosis, effusions of serous fluid, respiratory failure, cardiac decompensation, and death. Asbestosis is often a progressive disease, even in the absence of continued exposure. The symptoms of the disease are shortness of breath, cough, fatigue, and vague feelings of sickness. When the fibrosis worsens, shortness of breath occurs even at rest. One clinical feature of early asbestosis as well as other lung diseases is end-inspiratory crackles (rales). Diagnosis of asbestosis is based on the presence of characteristic radiologic changes, symptoms, rales, other clinical features of fibrosing lung disease, and a history of exposure to asbestos.

Asbestos exposure can cause pleural and/or other pulmonary disease. Pleural plaques are one of the markers of asbestos exposure and may develop within 10-20 years after the initial exposure. Plaques are opaque patches visible on chest X rays that consist of dense strands of collagen (connective tissue protein) lined by mesothelial cells. All commercially used types of asbestos induce plaques. Plaques can occur without fibrosis and do not seem to reflect the severity of pulmonary parenchymal disease. Pleural calcification is also commonly found in persons who have been exposed to asbestos (Craighead et al., Ex. 84-033).

The adverse effects of exposure to asbestos have been observed in workers involved in the manufacture of asbestos cement pipes and shingles (Enterline et al., Ex. 84-044, 84-122; Weill et al., Ex. 84-123, Finkelstein, Ex. 84-206, 84-240), asbestos mining and milling (Wagner et al., Ex. 84-22; Liddell et al., Ex. 84-059; McDonald et al., Ex. 84-063; Hobbs et al., Ex. 84-072; Nicholson et al., Ex. 84-056; Rubino et al., Ex. 84-086), asbestos textile manufacturing (Doll, Ex. 84-040; Peto et al., Ex. 84-169; Berry et al., Ex. 84-120; Dement et al., Ex. 84-037), insulation work (Selikoff et al., Ex. 84-109), shipbuilding (Selikoff et al., Ex. 84-091; Blot et al., ex. 84-109; Talmon et al., Ex. 84-182), talc mining and milling (Brown et al., Ex. 84-29) and in a variety of asbestos products manufacturing industries (Jones et al., Ex. 84-138; Henderson and Enterline, Ex. 84-048; McDonald and McDonald, Ex. 84-134; Seidman et al., Exs. 84-087, 231-A; Robinson et al., Ex. 84-082; Acheson et al., Ex. 84-103).

The conclusions just expressed are widely accepted both in the U.S. and abroad. The following agencies and organizations have reviewed the health data for asbestos: International Agency for Research on Cancer (IARC) (Ex. 84-201); Organization for Economic Cooperation and Development (OECD) (Ex. 84-207), NIOSH (Exs. 84-208 and 84-220), Advisory Committee of the Health and Safety Commission of the United Kingdom (Ex. 84-215), the Chronic Hazard Advisory Panel on Asbestos (CHAP) (Ex. 84-256), and the U.S. Environmental Protection Agency (Ex. 84-150). All of these groups have concluded that there is a causal relationship between asbestos exposure and the development of cancer and non-malignant respiratory disease. NIOSH recommended reducing the permissible exposure limit (PEL) for asbestos to 0.1 fiber per cubic centimeter (0.1 f/cc) in 1978. In 1980, a joint NIOSH-OSHA Asbestos Work Group stated that there was no level of exposure to asbestos below which clinical effects did not occur and recommended a PEL of 0.1 fiber per cubic centimeter (0.1 f/cc), based on the limitations of current technologies for measuring airborne concentrations of asbestos. The 1979 report of the Advisory Committee of the Health and Safety Commission of the United Kingdom (hereafter referred to as the U.K. Committee) led to the reduction of the British standard for asbestos to 1 f/cc for crysotile, 0.5 f/cc for amosite, and 0.2 f/cc for crocidolite.

The following sections describe the record evidence that demonstrates the causal relationship between asbestos exposure and increased risks of incurring lung cancer, mesothelioma, gastrointestinal cancer, and non-malignant respiratory diseases such as asbestosis. In addition, evidence is presented pertaining to the relationship between exposure to various types and sizes of asbestos fiber and the risks of asbestos-related disease; evidence concerning the synergistic effect of smoking and asbestos exposure on the risks of developing lung cancer is also presented. Most of the health effects evidence was previously presented in OSHA's November proposal (48 FR 51099-51122). The current publication summarizes the evidence contained in that Federal Register notice and presents in detail new evidence obtained during and after the public hearing.

B. Epidemiologic Evidence of Risk of Lung Cancer and Mesothelioma Mortality

1. Epidemiologic Studies

The epidemiologic studies of greatest interest are those that show a correlation between the intensity and duration of asbestos exposure and an observed excess in lung cancer and mesothelioma. In the November proposal, OSHA reviewed several studies that provided information on exposure level and incidence of lung cancer (Exs. 84-21; 84-36; 84-37; 84-48; 84-57; 84-90; 84-205; 84-240) and mesothelioma (Exs. 84-36; 84-57; 84-90; 84-205; 84-240). These studies, which provide the basis for OSHA's Quantitative Risk Assessment are briefly reviewed here, along with a number of more recent investigations (Exs. 162-C, 162-E, 188-A; 188-B; 231-A) that were submitted to the record after publication of the November proposal.

Seidman et al. (Ex. 84-087) studied cause specific mortality among 820 amosite insulation manufacturing workers employed sometime during 1941-1945 at the Patterson insulation facility, which was known to have a deficient ventilation system. Estimates of asbestos exposure at this facility

criterion. Citing a study by Samet et al. which used a relatively large cohort, Kilburn argued that smoking neither produced the x-ray appearance of pulmonary fibrosis nor contributed to fibrosis resulting from asbestos exposure.

Pearle (Ex. 84–079) studied 141 shipyard workers who were referred for medical exams because of suspected asbestos-related lung disease. The shortest duration of exposure in this group was 7 years. Chest x-rays were taken on all subjects and pulmonary function data were collected, including FVC, FEV_1, and diffusion capacity. X-rays were examined for pleural thickening and interstitial abnormalities consistent with asbestosis. Smoking groups were defined in terms of nonsmokers, light smokers, moderate smokers, and heavy smokers. Three asbestos exposure groups were also defined as being mild, moderate, or heavy, based on the duration of exposure (0–14 years, 15–19 years, and 20+ years, respectively). Three percent of the nonsmokers had interstitial disease, all of whom were concentrated in a heavy exposure group. By contrast, 8–12 percent of the smokers had significant interstitial disease, with the highest prevalence in the mild and moderate asbestos exposure groups. These differences between nonsmokers and smokers, however, were not statistically significant. The prevalence of pleural disease in heavy smokers was 25 percent, compared with 9 percent in nonsmokers. This difference was statistically significant. The prevalence of pleural disease among the light and moderate smoking groups was similar to that in heavy smokers. The largest difference in the prevalence of pleural disease between heavy smokers and nonsmokers is found in the group with mild asbestos exposure. These prevalence measures were not adjusted for age, however, and it cannot be concluded definitively that the statistically significant difference in prevalence between heavy smokers and nonsmokers is attributable to smoking history alone.

Berry et al. (Ex. 84–020) studied 379 men employed in an asbestos textile mill. Two cohorts were defined: those first employed before 1951 and those employed on or after 1951. The mean cumulative exposure for the earlier cohort was approximately twice that of the more recent cohort. Smoking histories were available for 376 men. Five smoking groups were defined: Never smoked, 1–4 cigarettes per day, 5–14 cigarettes per day, 15+ cigarettes per day, and ex-smokers. In the most recent cohort, the prevalence of crepitations, possible asbestosis, certified asbestosis, and small radiological opacities was higher among heavy and ex-smokers compared with light smokers (1–4 cigarettes per day) and nonsmokers. For example, 15 percent of heavy smokers had certified asbestosis versus none in the nonsmoking and light smoking groups. By contrast, there were no apparent differences in the prevalence of asbestosis or other conditions among the five smoking groups from the earlier cohort, which incurred a higher mean cumulative exposure, was older, and had a longer period of followup than the more recent cohort. This study suggests that, although there may be differences in the prevalence of asbestosis among smokers and nonsmokers who have been exposed recently to asbestos, the prevalence of asbestosis among smokers and nonsmokers tends to be more similar as the latency period increases or at higher levels of exposure to asbestos.

One additional study received since the November proposal is pertinent to this issue. Nicholson and his colleagues obtained chest x-rays and administered pulmonary function tests to 916 brake line repair and maintenance workers and approximately 205 nonexposed blue collar workers (Ex. 172–B). Chest x-ray abnormalities were defined to include parenchymal changes of 1/0 or greater, pleural thickening, pleural plaques, and pleural calcification. Predicted values for spirometry were based on the revised analysis by Miller et al. (1980) of the 1971 data of Morris, Kuski and Johnson (Ex. 172–B).

The percentage of workers with any evidence of chest x-ray abnormality among those with garage employment was 24.2 percent compared with 18.6 percent among workers with no stated asbestos exposure or garage employment (Ex. 172–B). This overall difference between the two groups is accounted for by differences in the prevalence of parenchymal abnormalities (19.0 percent vs. 13.3 percent) rather than pleural abnormalities (8.4 percent vs. 8.9 percent). However, significant differences existed in the percentages of pleural abnormalities among those employed in work having direct asbestos exposure (22.2 percent) or shipyard employment (25.2 percent) and those employed only in garage work (8.4 percent) or having no asbestos exposure (6.9 percent).

These results were interpreted by the authors to mean that "pleural abnormalities often appear from relatively low asbestos exposures and can exceed parenchymal abnormalities in prevalence at long times from onset of exposure" (Ex. 172–B, p. 29). Similar results were obtained after standardizing for age and smoking history.

The pulmonary function test data, when standardized for smoking, indicated virtually identical results for the unexposed controls, the brake repair workers, and individuals exposed or possibly exposed to asbestos (Ex. 172–B). The investigators note that these findings are not surprising because "forced vital capacity is usually a less sensitive determination of asbestos-related changes than the presence of x-ray abnormalities and forced expiratory volume in 1 second relates to exposures other than asbestos" (Ex. 172–B, p. 46). Although this study (Ex. 172–B) provides evidence that asbestos causes chest x-ray abnormalities over and above those that may be caused by smoking, the data were not sufficient to show that asbestos-exposed workers who smoke suffered more lung impairment than either asbestos-exposed nonsmokers or non-exposed smokers (Ex. 172–B).

In summary, OSHA finds that there is limited though conflicting evidence that asbestos workers who smoke have a higher risk of dying from asbestosis, as well as a higher prevalence of crepitations, lung function decrements, and small radiological opacities than their nonsmoking co-workers.

F. Relationship of Fiber Size and Type of Risks from Asbestos-Related Disease

1. Evidence for a Differential Risk by Fiber Type

In the November proposal (48 FR 51110), OSHA reviewed numerous epidemiological studies concerning the toxicity and carcinogenicity of different asbestos fiber types. OSHA concluded that all fiber types, alone or in combination, have been observed in studies to induce lung cancer, mesothelioma, and asbestosis in exposed workers, with the exception of antophyllite, which has been observed to induce lung cancer and asbestosis, but not mesothelioma (OSHA/NIOSH, Ex. 84–200; for amosite: Seidman et al., Exs. 84–67, 231–A; Anderson et al., Ex. 84–17; and Murphy et al., Ex. 84–311; for chrysotile: McDonald et al., Ex. 84–65; McDonald and Fry, Ex. 84–64; Liddell et al., Ex. 84–39; Nicholson et al., Ex. 84–72; Rubino et al., Ex. 84–86; Dement et al., Ex. 84–37; Acheson and Gardner, Ex. 84–13; and Berry and Newhouse, Ex. 84–21; for crocidolite: Jones et al., Ex. 84–139; Hobbs et al., Ex. 84–132; McDonald and Newhouse, Ex. 182; Berry and

. . . it is much easier to generate dust clouds from amphiboles (than from chrysotile).

So. . . . people who were exposed to amphiboles in the past almost certainly were exposed to very high levels (compared to the levels of chrysotile to which people were exposed) (Tr. 7/9, p. 35).

Using a similar line of argument, Dr. Hans Weill of Tulane University suggested that epidemiologic studies show a fiber-specific risk differential because "It is likely . . . that a cloud of asbestos dust contains a higher proportion of respirable 'carcinogenic' fibers if crocidolite is present . . . Crocidolite might therefore be more likely to be deposited in the deep portion of the lung and migrate more easily to the pleural surfaces" (Ex. 99, pp. 17–18).

Although the higher levels of amphiboles to which workers were exposed in the past may partly explain the different findings between epidemiologic and animal studies, physical differences between chrysotile and the amphiboles that affect the ability of the lung to clear fiber particles may also have led to these different findings. A number of studies have shown that chrysotile is more rapidly cleared from the lung than are the amphiboles (Exs. 64–171, 64–173, 64–173, 64–202, 312). For example, Glyseth et al. (Ex. 312) examined the asbestos content of lung tissue samples taken from asbestos cement workers who had died of pleural mesothelioma or lung cancer. Although more than 90 percent of the fibers used by the workers were chrysotile, 65 to 99 percent of the fibers found in the lung tissues were amosite, crocidolite, and anthophyllite. The differential lung retention of various fiber types has also been demonstrated in animals. Castleman (Ex. 121) discussed a study by Wagner (1982) that found that animals exposed to chrysotile fibers developed lung cancer even though a smaller amount of chrysotile was retained in the lung compared to similar tests with amphiboles. He suggested that "chrysotile fibers engaged in a process that led to cancer before removal and decomposition of . . . [the fibers] occurred" (Ex. 121, p. 2). Dr. Weill believed that "these differences in tissue persistence may wholly or partially explain the observations [that exposure to amphiboles are associated with a higher prevalence of mesothelioma] in human . . . populations . . . Non-confirmation of fiber type differences in animal experiments may be related to the much shorter life span . . . [of experimental animals, which would not allow] the effects of varying tissue persistence to be expressed" (Ex. 99, p. 18).

Dr. Davis also testified that the differential lung retention of chrysotile and the amphiboles may account for the conflicting results of human and animal studies, albeit by a different mechanism. He explained this view as follows:

[I suggest] that chrysotile or sufficient chrysotile is able to remain in the lung tissue for two or three years. Enough of it [to induce cancer] will stay for the [entire] life span of the rat. That means it can exert its maximum effect in the rat, and it means that the rat results showing chrysotile as being [more] hazardous are genuine.

I believe that chrysotile is largely removed from human lung tissue during the much longer 20, 30, [or] 40-year tumor induction period that you have got to have in human beings. I think that if that wasn't the case, then all the epidemiological evidence would be showing that chrysotile was the nastiest of the dusts" (Tr. 7/9, p. 36).

Several rulemaking participants (Exs. 64–236, 99, Tr. 7/9, p. 39) expressed the opinion that chrysotile fibers, which are composed of several hundred smaller fibrils, are easily broken apart in the lung as the result of magnesium leaching from the fibers. The magnesium loss reduces the structural strength and length of the fiber, facilitating removal of the fiber by phagocytosis. This process occurs to a lesser extent with the amphiboles, which contain a smaller quantity of magnesium. Although this may explain why chrysotile is more easily cleared from the lung, it also effectively increases the dose, in terms of the number of fibers, that reaches the lung. Dr. Davis explained this possibility:

"Now I believe what happens—and we have evidence of this—is that chrysotile deposited in lung tissue quite rapidly separates out into its individual fibrils. So if you think you have deposited a cell 10 in the lung tissue, six weeks later you have actually got 100, which potentially at least are the same length, but are very, very much thinner.

Now I think this certainly explains some of the very high harmful potential of chrysotile in our animal experiments. We are actually giving the animals . . . many more fibers even when we are trying to use equal doses [of chrysotile and amphiboles]" (Tr. 7, S.N. pp. 35–39).

To summarize the data on risk differential by asbestos fiber type, human epidemiological studies have suggested that occupational exposure to amphiboles is associated with a greater risk of mesothelioma than is exposure to chrysotile. No clear risk differential for lung cancer or other asbestos-related disease has been demonstrated by epidemiological studies. Animal experiments, however, have indicated that chrysotile is a more potent carcinogen than amphiboles when administered by inhalation or intrapleural injection, thus conflicting with the findings of human epidemiology studies. Rulemaking participants have suggested several reasons for the discrepancy: (1) Exposures to amphiboles in the past were much higher than exposures to chrysotile, (2) chrysotile fibers break up and are more easily cleared from the lung than are amphiboles, effectively reducing the residence time of chrysotile in the human lung, and (3) the break-up of chrysotile fibers into individual fibrils occurs more readily than for amphibole fibers, thus increasing the effective dose of chrysotile in animals. Dr. Davis explained at the hearing that the net effect of these biological mechanisms is unknown:

". . . Is one fiber . . . of amphibole more dangerous than one fiber . . . of chrysotile? There I . . . [have] to point out that our evidence cannot answer this with certainty. On the one side, you have almost certainly the greater harmful potential of chrysotile and the greater durability of the amphiboles. . . . I could imagine that one fiber of each in human beings will end up roughly the same harmfulness, or that might not be the case. It may be that the greater durability of amphiboles will still give a little bit of an edge. I have no definite data on this, and nobody else has." (Tr. 7/9, p. 63)

OSHA agrees with Dr. Davis that epidemiological and animal evidence taken together, fail to establish a definitive risk differential for the various types of asbestos fiber. Accordingly, OSHA has, in its Quantitative Risk Assessment (see Section VI) and in the establishment of a permissible exposure limit (see Section XI) recognized that all types of asbestos fiber have the same fibrogenic and carcinogenic potential.

Evidence for a Differential Risk by Fiber Size and Aspect Ratio. Several studies contained in the rulemaking docket suggest that fiber dimension is an important determinant in asbestos-related disease development. Stanton et al. (Exs. 64–153, 64–155) studied the effects of various sizes of fibrous materials, including all forms of asbestos, implanted in the pleura of rats and found that some fibrous glasses and all asbestos fiber types produced malignant tumors. The most carcinogenic fibers were 0.25 um or less in diameter and greater than 8 um in length. Fibers less than 5 um in length appeared to be engulfed and digested by phagocytes. However, fibers that were 1.5 um or less in diameter and longer than 4 um (an aspect ratio of approximately 3) also showed a higher correlation with carcinogenicity. Wright