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DEPARTMENT OF LABOR

Occupational Safety and Health Administration

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

[Part 1 of 3]

29 CFR Parts 1910 and 1926

Occupational Explosure to Asbestos, Tremolite, Anthophyllite, and Actinolite

[Docket No. H-033C]

51 FR 22612

**June** 20, 1986

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 peritioneal 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.

**TEXT:**
SUPPLEMENTARY INFORMATION:

I. Introduction

*A. The Format of This Document (the Preamble)*

The preamble accompanying these revised standards is divided into 13 parts, numbered I through XIII. The followeing is a table of contents:

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

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

2. TR.: Transcript date and page number.

*B. Summary*

Pursuant to sections 4(b)(2), 6(b), 6(c), and 8(c) of the Occupational Safety and Health Act of 1970 (the Act) (84 Stat. 1592, 1593, 1596, 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 51085) 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, D.C. More than 55,000 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.

OSHA has decided to issue two separate standards regulating **occupational exposure to asbestos,** tremolite, anthophyllite, and actinolite: One that applies to workplaces in general industry (including maritime) and another covering construction worksites. In promulgating two separate standards for general industry and construction, OSHA is acting in accordance with the recommendations of the Advisory Committee for Construction Safety and Health (CACOSH), which has reviewed and commented on several versions of the new standard in the construction industry, most recently during CACOSH's deliberations on October 17, 1985, in Washington, DC. These standards will be codified at 29 CFR 1910.1001 for general industry and at 29 CFR 1926.58 for the construction industry. OSHA has developed separate standards for these two industry groupings in recognition of the vastly different conditions prevailing in the workplaces covered by general industry and construction standards. As the April 1984 notice pointed out (49 FR 14127 et seq.). OSHA's existing asbestos standard (29 CFR 1910.1001) was more suitable for fixed-site manufacturing workplaces and a workforce composed of long-term employees, rather than for the short-term projects and highly mobile workforce characteristic of the construction industry.

Support for a separate OSHA standard for construction came from all interested parties in this rulemaking, including the Building and Construction Trades Department (BCTD) of the AFL-CIO (Ex 87-2); CACOSH (Ex. 84-424); the Asbestos Information Association (EX. 84-307); the Associated General Contractors of America (AGC) (Ex. 84-457): The Safe State Program, University of Alabama (Ex. 601.X); and the AFL-CIO Steering Committee on Safety and Occupational Health (Ex. 606.X). These commenters supported separate standards for these two industry groupings because employee exposures to asbestos, tremolite, anthophyllite, and actinolite, appropriate methods of controlling exposures, and prevailing workplace conditions are substantially different in workplaces in construction and general industry.

Although the Summary and Explanation section of the preamble for the construction industry (Section XI of the preamble) discusses the record evidence as it applies to specific provisions of the **final rule** for construction, the reasons given by these commenters in support of a separate standards for construction can be summarized briefly as follows:

(1) The construction industry is characterized by non-fixed worksites that are temporary in nature and differ from those in general industry in regard to site conditions, size and scope of tasks, methods of operation, and environmental conditions.

(2) Employees in the construction industry often do not remain in construction or in the employ of the same employer for a long period of time, in contrast to employees in fixed-site manufacturing facilities.

(3) The unique characteristics of construction operations may make it necessary to tailor some of the requirements traditionally included in OSHA health standards to the specific needs of the construction industry.

OSHA finds merit in these arguments, and in response to the nearly unanimous support for separate standards for general industry and construction, the Agency is issuing separate **final rules** covering these respective workplaces. In addition, OSHA has tailored the requirements of the final construction standard to reflect differences in operations of various types within the construction industry itself. The record demonstrated these intra-industry differences in construction exposure and work conditions by pointing to the generally low exposures and well-controlled conditions prevailing in construction operations involving the installation of new asbestos-containing products and comparing them with those typical of major demolition, renovation, and asbestos removal construction operations. In recognition of this wide diversity in construction projects, the Agency has specifically identified in the **final rule** those additional requirements that apply to construction operations involving asbestos abatement activities. Requirements governing these potentially high-hazard operations are grouped separately in the construction standard under a heading clearly labeled "for removal, demolition, and renovation operations." For example, paragraphs (i)(1) through (i)(3) of the standard are grouped under the title "Protective clothing" and apply to all construction operations other than removal, demolition, and renovation operations, while paragraph (i)(4) is titled "Protective clothing *for asbestos removal, demolition, and renovation operations"* and applies only to such operations.Similarly, paragraphs (e)(1) through (e)(5) contain OSHA's requirements for regulated areas on construction projects other than removal, demolition, and renovation operations, while paragraph (e)(6) specifies the more extensive and stringent requirements for the enclosed negative-pressure regulated areas required for removal, demolition, and renovation operations. OSHA believes that tiering the construction standard to reflect differences in workplace conditions within this industry will simultaneously provide appropriate employee protection and encourage voluntary employer compliance with the **final rule.**

In publishing these two revised standards governing **occupational exposure to asbestos,** tremolite, anthophyllite, and actinolite in construction and in general industry, OSHA is acting to regulate a hazard widely recognized by other Federal agencies, health experts, and the general public. The U.S. Environmental Protection Agency (EPA) has promulgated regulations controlling asbestos under the Clean Air Act, the Toxic

Substances Control Act, and the Clean Water Act. Under section 6 of the Toxic Substances Control Act (TSCA), EPA is proposing to prohibit the manufacture, importation, and processing of asbestos-cement pipe and fittings, roofing felts, flooring felts (and felt-backed sheet flooring), vinyl-asbestos floor tile, and asbestos clothing (51 FR 3738-3759). These uses would be prohibited because EPA believes that safer, economically competitive substitutes for these products are available, and that "the manufacture, processing, and use of asbestos products leaves a legacy of asbestos in the ambient air" (51 FR 3739).

In addition, EPA is proposing to establish a permit system to phase out all other asbestos products. Under this system, EPA would permit current miners or importers to mine or import a specific quantity of asbestos. EPA would require this quantity to decline every year until, after 10 years, mining or importation would only be permitted under a specific exemption for those asbestos applications for which no substitutes had been developed. EPA is also considering requiring labeling for all asbestos products that are not banned, including products manufactured pursuant to permits issued by EPA during the phase-down period, or pursuant to an exemption process.

Emissions of asbestos to the ambient air are controlled under section 112 of the Clean Air Act, which establishes National Emissions Standards for Hazardous Air Pollutants. Regulations in 40 CFR Part 61, Subpart M, specify control requirements for most asbestos emissions, including work practices that must be followed to minimize the release of abbestos fibers during the handling of asbestos waste materials. EPA regulations promulgated under the Toxic Substances Control Act (40 CFR Part 763, Subpart F) address the problem of asbestos construction materials used in schools. These regulations require that all schools be inspected to determine the presence and quantity of asbestos-containing materials in school facilities. Corrective actions are left to the discretion of school officials. EPA regulations promulgated under the Clean Water Act set standards for asbestos levels in effluents to navigable waters.

Throughout this rulemaking, OSHA has consulted with the EPA on various regulatory aspects of dealing with the asbestos hazard. EPA has reviewed and critiqued OSHA's quantitative risk assessment for asbestos (Exs. 84-292, 86-6), and both EPA and OSHA belong to the Federal Asbestos Task Force, established in June 1983, to coordinate Federal regulatory actions with regard to asbestos. The Consumer Product Safety Commission is also a member of this task force because of its mandate to protect consumers from health and safety hazards.

*C. State Plan Revisions*

The 25 states and territories with their own OSHA-approved occupational safety and health plans must revise their existing standard within 6 months of this publication date or show OSHA why there is no need for action; for example, because an existing State standard covering this area is already "at least as effective" as the revised Federal standaerds. These states or territories are: Alaska, Arizona, California, Connecticut, Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, New York, North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah Vermont, Virginia, the Virgin Islands, Washington, and Wyoming. (In Connecticut and New York, the plan covers only State and local government employees.)

II. Regulatory History

OSHA has regulated asbestos since 1971. A 12 f/cc permissible exposure limit (PEL) for asbestos was included in the initial promulgation on May 29, 1971 (36 FR 10466) of OSHA standards pursuant to Section 6 (a) of the Act. In Response to a petition by the Industrial Union Department of the AFL-CIO, OSHA issued an ETS on asbestos on December 7, 1971, which established a PEL of 5 f/cc as an 8-hour time-weighted average (TWA) and a peak exposure level of 10 f/cc.

In June 1972, OSHA promulgated a new final standard that established an 8-hour time-weighted average PEL of 5 f/cc and a ceiling limit of 10 f/cc. These limits were intended primarily to protect employees against

asbestosis, and it was hoped that they would provide some incidental degree of protection against asbestos induced forms of cancer. Effective July 1976, OSHA's 8-hour TWA limit was reduced to 2 f/cc and this limit remained in effect up to the present; the **final rules** published today revise the PEL for 8-hour employee exposures to asbestos, tremolite, anthophyllite, and actinolite to a level of 0.2 fiber/cc.

OSHA's 1972 asbestos standards was reviewed by the court and upheld in all major respects; however, the court remanded two issues for OSHA's reconsideration (*IUD* v. *Hodgson,* 449 F.2d 467 (CADC 1974)). These issues were whether the July 1976 effective date for the 2 f/cc standard should be accelerated for some industries and whether the standard's 3-year retention period for employee exposure monitoring records was adequate. In response to the remand, OSHA increased the record retention period to 20 years (41 FR 11504), and the passage of time mooted the acceleration issue.

In October 1975, OSHA published a notice of proposed rulemaking (40 FR 47652) to revise the asbestos standard because the Agency believed that "sufficient medical and scientific evidence has been accumulated to warrant the designation of asbestos as a human carcinogen" and that advances in monitoring and protective technology made reexamination of the standard "desirable." This proposal would have reduced the 8-hourtime-weighted average to 0.5 f/cc and imposed a ceiling limit of 5 f/cc for 15 minutes.

The basis for the 1975 proposal's reduction in the permissible exposure limit to 0.5 f/cc was OSHA's then-current policy for carcinogens that assumed that no safe threshold level was demonstrable and therefore that the Act required the Agency to set the PEL at a level as low as technologically and economically feasible. This policy was rejected by the Supreme Court in the benzene decision (*IUD* v. *API,* 448 U.S. 601 (1980)) (see the discussion of the implications of the benzene decision for OSHA rulemaking in the Significance of Risk section of the preamble, section VI). The 1975 proposal would have applied to all industries except construction. Further, although OSHA announced its intention to develop a separate proposal applicable to the construction industry, no such proposal was published.

In 1976, the National Institute for Occupational Safety and Health (NIOSH), and in 1980 a NIOSH/OSHA task force, recommended that OSHA reduce the permissible exposure limit for asbestos to 0.1 f/cc, based on evidence of the carcinogenicity of asbestos (Ex. 84-320). OSHA has considered these recommendations in determining what regulatory response is necessary to provide exposed employees with effective protection.

On May 24, 1983, OSHA consulted with the Advisory Committee for Construction Safety and Health (referred to as "CACOSH") concerning the applicability of any new asbestos standard to the construction industry. CACOSH endorsed OSHA's position that any new PEL adopted for general industry should also apply to the construction industry (Ex. 84-424). On November 4, 1983, OSHA published an Emergency Temporary Standard (ETS) for asbestos (48 FR 51086). The ETS marked a new regulatory initiative, related to, but not part of, the 1975 proceeding. The ETS was held invalid by the U.S. Circuit Court of Appeals for the Fifth Circuit on March 7, 1984.

Subsequently, OSHA published a notice of proposed rulemaking (49 FR 14116, April 10, 1984) for a standard covering **occupational exposure to asbestos** in all of the industries governed by the Act: maritime, construction, and general industry. Pursuant to Section 6(c) of the Act, the ETS also served as a proposed rule. Public hearings were held in Washington, D.C., from June 19 to July 10, 1984, to provide interested parties and the public with the opportunity to comment on the proposed revisions, pursuant to notice and section 6(b) of the Act (29 U.S.C. 655(b)(3)). The hearings were presided over by Administrative Law Judge Robert G. Mahoney. Post-hearing submissions of data, comments, and briefs were received through November 1, 1984. The entire record, including over 340 exhibits and approximately 55,000 pages of material, was certified by Judge Mahoney on September 27, 1985, in accordance with 29 CFR 1911.17. Copies of materials contained in the record may be obtained from the OSHA Docket Office, Room N3670, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC. 20210. These final standards on **occupational exposure to**

**asbestos** in construction and general industry are based on a thorough consideration of the entire record of this 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. 601, 65 L. Ed. 2d 1010, 100 S. Ct. 2844 (1980), that before the insurance 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 lessened by a change in practices. . . . (448 U.S. at 642, 65 L. Ed. 2d at 1035)

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 finding that a significant risk exists with anything approaching scientific 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 other 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 disases 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. 84-033). 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 25-30 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, colorectal, 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 50 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., Exs. 84-044, 84-122; Weill et al., Ex. 84-123, Finkelstein, Exs. 84-206, 84-240), asbestos mining and milling (Wagner et al., Ex. 2-21; Liddell et al., Ex. 84-059; McDonald et al., Ex. 84-065; Hobbs et al., Ex. 84-072; Nicholson et al. Ex. 84-086; Rubino et al., Ex. 84-086), asbestos textile manufacturing (Doll, Ex. 84-040; Peto et al., Ex. 84-169; Berry et al., Ex. 84-020; 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; Tagnon 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 Enterlines, Ex. 84-048; McDonald and McDonald, Ex. 84-154; Seidman et al., Exs. 84-087, 261-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. 84-321), Organization for Economic Cooperation and Development (OECD) (Ex. 84-337), NIOSH (Exs. 84-338 and 84-320), Advisory Committee of the Health and Safety Commission of the United Kingdom (Ex. 84-216), the Chronic Hazard Advisory Panel on Asbestos (CHAP) (Ex. 84-256), and the U.S. Environmental Protection Agency (Ex. 84-180). All of these groups have concluded that there is a causal relationship between asbestos exposure and the development of cancer and nonmalignant respiratory disease. NIOSH recommenced reducing the permissible exposure limit (PEL) for asbestos to 0.1 fiber per cubic centimeter (0.1 f/cc) in 1976. 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 chrysotile, 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 insurring lung cancer, mesothelioma, gastrointestinal cancer, and nonmalignant 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; 84-87; 84-90; 84-206; 84-240) and mesothelioma (Exs. 84-36; 84-87; 84-90; 84-206; 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; 163-E; 168-A; 168-B; 261-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 sometimes during 1941-1945 at the Patterson insulation facility, which was known to have a deficient ventilation system. Estimates of asbestos exposure at this facility were not available at the time this study was published. Workers were classified as having worked less than 1 month, 2 months, 3-5 months, 6-11 months, 1 year, or 2 or more years. Workers in all of these exposure categories had excessive mortality from lung cancer. This study demonstrates that workers with exposures of relatively short duration are at excess risk of lung cancer.

This mortality study was updated to include both a longer followup period and exposure extimates (Seidman, Ex. 261-A). The updated analysis included an additional 593 cases involving deaths occurring during the period from 5 to 40 years after onset of work. To increase the comparability of this study with others, Seidman re-analyzed the results of the earlier study by using death rates for white males from New Jersey to calculate Standardized Mortality Ratios (SMRs).Cumulative exposure to asbestos was estimated for each worker using work history records and exposure measurements taken in 1967, 1970, and 1971 from two similar amosite

insulation production plants. These exposure data were collected and reported by NIOSH (Ex. 2-12). Workers were progressively assigned to the following cumulative exposure categories during the 35-year followup period: less than 6.0 f/cc-years, 6.0-11.9 f/cc-years, 12.0-24.9 f/cc-years, 25.0-49.9 f/cc-years, 50.0-99.9 f/cc-years 100.0-149.9 f/cc-years, 150.0-249.9 f/cc-years, and 250 or more f/cc-years. The use of expousre data from plants other than that from which the cohort was derived is appropriate in this study since the exposure measurements were from "plants of the same company where the same products were made utilizing the same machinery, fiber and production processes" (Ex. 261-A, p. 5). The investigators indicated that their exposure estimates may be on the high side for two reasons: (1) Dustier areas tend to be sampled more often than other areas, and (2) a concerted effort was made to have respiratory protection used by workers in the plant from which the study cohort was taken. Furthermore, Dr. Morton Corn, former Assistant Secretary for OSHA and testifying on the behalf of the Building and Construction Trades Department, commented that the Tyler, Texas plant, where some of the exposure data were obtained, was ". . . one of the most contaminated asbestos facilities I've ever been in" (Tr. 7/3, p. 67). Therefore, it is likely that the exposure estimates were overestimated, leading to an underestimate of excess risk for workers in each of the cumulative exposure categories.

Overall deaths were significantly (p less than 0.001) elevated (SMR-167), as were deaths from all cancers (SMR-287), from all "asbestos" diseases (SMR-396), from noninfectious lung disease (SMR-489), and from lung cancer (SMR-541). Colorectal cancer mortality was also significantly (p less than 0.05) increased (SMR-185). In addition, 17 deaths from mesothelioma were observed, a finding of great significance given the rarity of ths disease. A strong cumulative dose-response relationship was evident for both lung cancer mortality and mortality from all "asbestos" diseases.

Dement et al. (Exs. 84-036, 84-037) estimated individual cumulative exposures for 768 workers employed at a chrysotile textile plant during 1930-1975. Mean exposure levels were estimated for these workers on the basis of 5,952 industrial hygiene samples. The following exposure categories were defined: less than 1,000 f/cc-days, 1,000-10,000 f/cc-days and 10,000-40,000 f/cc-days. As explained in the November proposal, OSHA calculated that these categories of cumulative exposure are roughly equivalent to the following exposure categories: less than 2.7 f/cc-years; 2.7-27.4 f/cc-years, 27.4-109.6 f/cc-years, 109.6-274 f/cc-years, and greater than 274 f/cc-years. The first three of these exposure categories fall within at or below the lifetime cumulative exposure permitted by the 2-f/cc standard. Fifteen or more years after the onset of exposure, standardized mortality ratios (SMRs) for lung cancer among white males were 140, 279 (p less than 0.05), and 352 (p less than 0.05) in the first three exposure categories, respectively, demonstrating the existence of a dose-response relationship. Dement et al. (Ex. 84-037, p. 432) concluded that: "Based on data from this study, significantly elevated mortality risks are predicted for lung cancer and for asbestosis at cumulative exposures of 100 fibers/cc-years in the textile industry." OSHA considers that these observations of excess risk from low cumulative exposures are well-supported because of the careful estimation of exposure histories for members of the cohort in this study.

Henderson and Enterline (Ex. 84-048) studied the mortality of 1,075 retired asbestos production workers. Mean estimated exposures for the cumulative exposure categories were 62, 182, 352, 606, and 976 mpcf-years. Based on the recommended conversion factor of 1:1.4 for asbestos production (discussed in the November proposal), 62 mpcf-years is roughly equal to 87 f/cc-years, a cumulative exposure permitted by the 2 f/cc standard. An SMR of 197.7 for respiratory cancer was observed for workers in this cumulative exposure category. This observed excess mortality risk is not as high as that observed by Dement et al. (Exs. 84-036, 84-037); however, the authors of the Dement et al. study suggested that this difference may be the result of the fact that Henderson and Enterline studied retirees, which constitute a select group of survivors; only 8 of the 35 lung cancer deaths observed by Dement et al. (Ex. 84-37) occurred among persons 65 or older.

McDonald et al. (Ex. 84-065) studied the mortality of 11,379 workers exposed to chrysotile mining and milling. Based on a conversion factor for these operations of 1:3 for mpcf to f/cc, the exposure classifications

developed by the authors would correspond to the following exposure categories: less than 90 f/cc-years, 90-899 f/cc-years, and 900 or more f/cc-years. Although they did observe an increased incidence of pneumoconiosis (SMRs 298, 1081, and 5400, respectively), McDonald et al. (Ex. 84-065) observed less lung cancer risk for these exposure categories than other investigators (SMRs were 93, 118, and 225, respectively). Rearding the different findings between the studies by McDonald et al. (Ex. 84-065) and Dement et al. (Exs. 84-036, 84-037) on lung cancer risk from low exposures, Dement et al. suggested that differences in the characteristics of airborne fibers, as well as the presence of a competing risk of pneumoconiosis among miners in the McDonald et al., study, could account for the differences in lung cancer mortality reported in these two studies.

Finkelstein (Ex. 84-240) studied the mortality of 339 men who had been employed at an Ontario asbestos cement factory for 9 or more years. Each cohort memberwas classified as having accumulated 8-69 f/cc-years, 70-121 f/cc-years, or 122-420 f/cc-years of asbestos exposure during the 18 years following onset of exposure. Cohort mortality was analyzed by cumulative exposure, starting 20 years after onset of exposure, and was compared to that of non-exposed Ontario men. Approximate relative risks for lung cancer mortality for the three exposure categories were 8.5, 16.3, and 7.4, respectively. Mesothelioma mortality rates per 1000 man-years were 1.9, 4.9, and 11.9, respectively, showing a clear dose-response relationship between asbestos exposure and mesothelioma. Finkelstein suggested several explanations for the unexpected decrease in excess lung cancer mortality in the highest exposure category: he argued that statistical fluctuations caused by the small size of the cohort or the possible confounding effects of smoking may have been responsible for this unexpected result. More likely, lung cancer risk may have been underestimated for the highest exposure category by Finkelstein's exclusion of any lung cancer deaths that might have occurred during the 20 years from onset of exposure to the beginning of the followup period. In addition to showing dose-response relationships between asbestos exposure and the excess risk from lung cancer and mesothelioma, OSHA notes that Finkelstein's study presents evidence that an excess risk for these diseases exists at cumulative exposures that would be permitted by lifetime exposure to the 2-fcc standard.

Rubino et al. (Ex. 84-086) studied the mortality of 952 male Italian chrysotile miners and millers. The mortality experience of the overall cohort was compared with that of nonexposed Italian males. Compared with nonexposed Italians, the overall cohort had statistically significant excesses of mortality from laryngeal cancer, nonmalignant respiratory diseases, and non-asbestos-related causes, but not from lung cancer. However, there were some trends showing increasing lung cancer risk with increasing length of followup and increasing cumulative exposure. Using the methodology presented in Ex. 84-336, OSHA determined that this study had only a 33.5 percent power to detect a 50 percent increase in lung cancer risk among workers with 20 or more years of followup. Canerally, it is considered desirable for studies to have at least an 80 percent power to detect a 50 percent increase in disease.

Weill et al. (Ex. 84-206) studied mortality among 5,645 men having at least 20 years of latency since first exposure in either of two asbestos cement plants. Each worker's cumulative dust exposure during the 20 years after the onset of exposure was estimated in terms of mpcf-years. Based on the conversion factor of 1:1.4 suggested by Hammad et al. Ex. 84-277), the five cumulative exposure categories would be equivalent to 14 or fewer f/cc-years, 15-70 f/cc-years, 71-140 f/cc-years, 141-280 f/cc-years, and 281 or more f/cc-years. Neither respiratory cancer mortality nor any other cause of death was increased among workers in the three lowest exposure categories. Weill et al. noted that the relatively high proportion (25 percent) of the cohort that was lost to followup and assumed to be alive may have led to an underestimation of respiratory cancer risk. The upper limits of the 95 percent confidence intervals of the SMRs for respiratory cancer for the three lowest exposure categories ranged from approximately 115 to 150, indicating, in OSHA's opinion, that the presence of an excess risk of mortality from lung cancer could not be ruled out for the cohorts in these exposure categories.

Berry and Newhouse (Ex. 84-021) studied the mortality of a large cohort of friction material production workers whose asbestos exposures were relatively low (generally less than 1 f/cc to 5 f/cc) and of short

duration. Cumulative exposures for the cohort averaged less than 50 f/cc-years. Only non-significant increases in mortality from lung cancer were observed; however, mortality from mesothelioma was significantly elevated compared with that of controls. Most of the mesothelioma victims had been exposed to asbestos levels exceeding 5 f/cc; their cumulative exposure estimates were not reported. A sizeable portion of the cohort was studied for a relatively short followup period between onset of exposure and the end of the study. For example, the follwoup period for 33 percent of the men was less than 20 years. Because of the short followup period used, OSHA does not believe that the non-significant increases in lung cancer mortality found by these investigators contradict the findings from other studies, which show that low-level exposure to asbestos has resulted in excessive mortality from lung cancer.

Of the few epidemiologic studies submitted to the docket after the publication of the November proposal, four provide additional information on the risk of lung cancer mortality and/or mesothelioma mortality among workers exposed to asbestos. The first (Cantor, Ex. 168-A; Cantor et al., Ex. 168-B) is only an interim report on a proportionate mortality study and has no estimates of cumulative exposure. Two other studies similarly give no estimates of cumulative exposure; one (Nicholson and Selikoff, Ex. 162-C) investigates the risks of recent exposures of limited duration, while another (Zoloth and Michaels, Ex. 163-E) investigates the effects of intermittent asbestos exposure. The fourth study (Seidman, Ex. 261-A) is an update of a previous study (Seidman et al., Ex. 84-087) and was discussed earlier in this section.

Kenneth P. Cantor, of the National Cancer Institute, submitted an interim report (Ex. 168-A; Cantor et al., Ex. 168-B) on his proportionate mortality study of 7,121 deaths identified among members and retirees of the California local of the United Association of Plumbers and Pipefitters. The interim report was based on 6,398 (89.8 percent) of the 7,121 deaths. No specific information was available on cigarette smoking habits or on asbestos exposure levels. Expected numbers of deaths were calculated from cause-specific proportionate mortality rates by 5-year age and 5-year calendar period groups among U.S. white males. For mesothelioma, the expected number of deaths was estimated on the basis of death certificate information for approximately 10 percent of the U.S. population.Further analysis conducted after the interim report confirms the interim report findings (Ex. 168-A).

The most striking finding from this report is that 15 mesothelioma deaths occurred in this group, while only 2 were expected. A significant (p less than 0.05) excess number of lung cancer deaths was also observed (587 observed, 408 expected). Other smoking-related cancer sites had PMRs at or near expected levels. The investigators concluded:

"It is likely that exposure to asbestos is responsible for at least part, if not all, of the excess number of lung cancers in this group:

1. The excessive number of deaths due to lung cancer is consistent with the elevated number of mesothelioma deaths that points to widespread asbestos exposure.

2. If cigarette smoking *had* [emphasis added] played an important role in causing excess lung cancer deaths, we would expect the PMR for bladder cancer, another smoking-related . . . [malignancy] that has not been linked to asbestos exposure, to also be elevated. There were 40 deaths due to bladder cancer whereas 40.4 were expected (PMR=.99), suggesting no increase in risk for cancers of this site." (Ex. 168-A, pp. 3-4.)

This study, although it is an interim report, is significant for two reasons. First, the excess number of deaths from mesothelioma add to the already considerable weight of evidence for a causal relationship between asbestos exposure and an increased mortality risk from this rare cancer. Second, despite the lack of data on smoking habits for the cohort, the study suggests that asbestos exposure, and not smoking, was the principal cause of the observed excess in lung cancer mortality.

Nicholson and Selikoff (Ex. 162-C) investigated mortality among 1,918 male shipyard workers who were employed on January 1, 1967 and who were first employed before January 11, 1957. More than 80 percent of the cohort was employed for less than 20 years. Although no estimates of exposure levels were given, the authors state that: "in terms of time from onset of exposure and duration of exposure, the exposures have been recent and of limited duration. The full manifestation of the effects of shipyard employment would not yet be expected to be present in this group" (Ex. 162-C, p. 1).

In comparison with the mortality observed in white males in Connecticut, the overall mortality for the cohort with 11.5 years of exposure was significantly (p less than 0.05) elevated (356 observed, 316 deaths expected). Mortality from cancer at all sites was also in excess (90 observed, 80 expected).The major sites of cancer increase were the lung (35 observed, 26 expected) and the gastrointestinal tract (19 observed, 15 expected), two cancer sites known to be related to asbestos exposure. These excesses were seen in both production and support workers, whereas office employees from the same shipyards experienced mortality similar to that of the general male population of Connecticut. Finding such excesses in a cohort that had relatively short employment and that had been followed for a relatively short period of time was, in the authors' words' "unexpected" and leads to augmented concern for the next two or three decades" (Ex. 162-C, pp. 3, 4).

The study (Ex. 162-C) provides additional qualitative evidence of the excess risk of lung cancer mortality and GI cancer mortality experienced by asbestos-exposed workers. Although these investigators were surprised to find such excesses following relatively recent asbestos exposure, other authors (Ex. 306-B, Ex. 320) have noted that significant increases in the lung cancer death rate begin to appear 10 to 14 years after the first exposure and peaks between 30 and 35 years after (Ex. 306-B, p. 57).

Zoloth and Michaele (Ex. 163-E) performed a proportionate mortality analysis of 381 deaths that occurred among white males who had been members of a local New York chapter of the Sheet Metalworkers International Association for at least 10 years. Specific estimates of asbestos exposure levels were not given; however, exposure was described as being intermittent and incidental. Half of the local union members were employed in installation of metal ducts. The expected distribution of deaths was based on U.S. white male mortality rates, with adjustments for age and date of death.

There was significant (p less than 0.05) excess mortality from all cancers (PMR-152), lung cancer (PMR-160), colorectal cancer (PMR-232), and non-Hodgkins lymphoma (PMR-236). In addition, three deaths from mesotheloma were observed. The authors calculated standardized mortality odds ratios (SMOR) using arteriosclerotic heart disease as a referent to offset some of the potential biases in PMRs. The calculated SMORs were reported to be virtually identical to the PMRs, indicating the absence of any significant biases in the PMR's for cancer. The authors concluded that this study, with an overall pattern of observed mortality consistent with that found in other populations exposed to asbestos, "strongly suggests the presence of significant asbestos-related illness is [sic] a population with 'secondary' asbestos exposure" (Ex. 163-E, p. 11).

The interpretation of these results is limited by the design of proportionate mortality studies. Although the investigators reported that half of the local union members were employed in installations of metal ducts, and thus were must likely to be exposed intermittently to asbestos, it is not known what proportion of the deceased members were so employed. Moreover, although the observed deaths occurred in a predominantly metropolitan population, the expected distribution of deaths was based on general U.S. mortality rates; the resultant comparison is not ideal because of the generally recognized differences in mortality patterns of urban populations in comparison to those of the overall U.S. population. These investigators did strengthen their study results by calculating SMORs.

2. Evidence of an Excess Risk of Lung Cancer and Mesothelioma at Low Cumulative Exposures of Asbestos

In establishing whether an existing permissible exposure limit is inadequate for protecting workers against the

risk of occupational disease, the Agency relies principally on the findings of quantitative risk assessments and an evaluation of the significance of the risk presented by exposure at the existing PEL. After conducting the quantitative risk assessment for asbestos, OSHA concludes that the 2-f/cc PEL is inadequate for worker protection and that reduction of the PEL is warranted (see Section V, Quantitative Risk Assessment, and Section VI, Significance of Risk). OSHA's finding that the 2 f/cc PEL is inadequate is supported by the observations of excess cancer mortality among workers who have been exposed to cumulative levels of asbestos lower than would be permitted by lifetime exposure to 2 f/cc. These observations, first referred to in the November proposal and discussed above, were derived from the studies by Dement et al. (Exs. 84-36; 84-37), Henderson and Enterline (Ex. 84-48), Finkelstein (Ex. 84-240), and Seidman et al. (Exs. 84-87, 261-A). In addition, a number of studies have recorded cases of mesothelioma among members of the families of asbestos workers (Anderson et al., Exs. 84-16, 84-17; Vianna and Polon, Ex. 84-186); Li et al., Ex. 84-149). Mesothelioma has also been observed among community members living near asbestos mines and factors (Wagner et al., Ex. 2-21; Newhouse and Thompson, Ex-84-70). For example, in 1976, Anderson et al. (Ex. 84-16) reported that 4 cases of pleural mesothelioma had been diagnosed among 626 family contacts of amosite factory workers. Presumably, family contacts received their exposure to asbestos from dust carried home on the worker's clothing, and especially during the laundering of dusty clothes. Although exposure measurements were not taken for family contacts, OSHA considers it very likely that their cumulative exposure was less than the cumulative exposure that would result from lifetime exposure to the 2 f/cc standard. OSHA believes that these findings, as well as the observation in epidemiological studies of excess mortality resulting from low cumulative exposures to asbestos, further support the Agency's finding from the risk assessment that the 2 f/cc PEL is inadequate for protecting workers against the risk from lung cancer and mesothelioma.

3. Experimental Evidence

Several animal studies are contained in the record that show that experimental animals, when administered asbestos fiber by inhalation, injection, or implantation, develop malignant tumors at a rate higher than unexposed animals (Exs. 84-338; 84-320; 84-205; 94-96; 84-197; 84-120; 84-128; 84-240; 84-193; 84-195). No rulemaking participant questioned the causal relationship between asbestos exposure and the development of malignancies in experimental animals. OSHA believes that, while these studies in general support the findings of epidemiology studies, they are more germaine to the issues brought up during the rulemaking regarding the relationship between fiber type and dimension and the carcinogenicity of asbestos. OSHA discusses these experimental studies in a later section that deals with the issues of fiber type and size.

4. Summary of the Evidence of Lung Cancer and Mesothelioma

After reviewing the studies discussed above, OSHA finds that the evidence for establishing a dose-response relationship between asbestos exposure and an excess risk of either lung cancer or mesothelioma is exceptionally strong. The following studies have shown a positive dose-response relationship for an increased risk of lung cancer mortality and/or mesothelioma mortality: Finkelstein (Ex. 84-240), Dement et al. (Ex. 84-036, 84-037), Henderson and Enterline (Ex. 84-048), Seidman (Ex. 261-A), Berry and Newhouse (Ex. 84-021), Weill et al. (Ex. 84-206), Selikoff et al. (Ex. 84-87), and Peto (Ex. 84-169). OSHA has used these studies in its Quantitative Risk Assessment (see Section V) to show that cumulative exposure levels below that permitted by the existing PEL of 2 f/cc presents an excess risk of cancer mortality.

These studies also show that cumulative exposure levels of asbestos below that permitted by lifetime exposure to the 2 f/cc PEL results in excess mortality from lung cancer and mesothelioma. Furthermore, the Seidman update (Ex. 261-A) and the Nicholson and Selikoff study (Ex. 162-C) clearly indicate that workers exposed for a relatively short period of time experienced significant excess mortality from lung cancer and from all asbestos diseases. OSHA believes that the results of Zoloth and Michael's study (Ex. 163-E) of asbestos-exposed sheetmetal workers further suggests that excess mortality can occur from intermittent exposure conditions. In light of the indings of these three new studies (Exs. 162-C, 163-E, 261-A) and the previously

considered evidence, OSHA concludes that well-conducted studies demonstrate a substantially increased rate of lung cancer and mesothelioma mortality among workers having low cumulative exposures to asbestos.

*C. Carcinogenicity of Asbestos for Sites Other than the Lung and Mesothelium*

1. Epidemiological Studies

In the November proposal, OSHA reviewed several epidemiological studies describing the mortality experience of asbestos-exposed occupational cohorts in regard to cancer occurring at sites other than the lung and mesothelium. Seven studies were reviewed that found statistically significant increases in deaths from gastrointestinal cancer among U.S. and Canadian insulation workers (Exs. 84-090, 84-224), Belfast insulation workers (Exs. 84-041, 84-090), asbestos factory workers (Exs. 84-048, 84-330), shipyard workers (Ex. 84-246), and tremolite and anthophyllite-exposed talc miners (Exs. 84-140, 84-141). Of these studies, the most striking is the investigation of 17,800 U.S. and Canadian insulation workers conducted by Selikoff, Hammond, and Seidman (Ex. 84-090). In this study, significant excess mortality was observed from lung cancer (SMR=406), mesothelioma (180 deaths), esophageal cancer (SMR=253), stomach cancer (SMR=126), colo-rectal cancer (SMR=152), laryngeal cancer (SMR=191), pharyngeal and buccal cavity cancer (SMR=159), kidney cncer (SMR=223), prostate cancer (SMR=137), and non-infectious respiratory diseases including asbestosis (SMR=319).

Selikoff, Hammond, and Seidman concluded:

Asbestos insulation workers in the United States and Canada suffer an extraordinary increased risk of death of cancer and asbestosis associated with their employment. This includes increases in deaths from lung cancer, pleural mesothelioma, peritoneal mesothelioma, cancer of the esophagus, colon and rectum, cancer of the larynx, oro-pharynx, kidney, and perhaps stomach. Some increases were seen in cancer of several other sites, as well, but data are inadequate at this time to permit characterization of their significance although attention is called to such wider increase (Ex. 84-090, p. 114).

In addition to the above-mentioned studies, OSHA reviewed five studies that showed non-statistically significant increases in gastrointestinal tract cancer. The occupational cohorts examined in these studies included chrysotile textile plant workers (Ex. 84-090, p. 114), chrysotile miners and millers (Ex. 84-065), amosite insulation production workers (Ex. 84-087), and asbestos factory workers (Exs. 84-251, 84-082). The November 1983 notice also pointed out that several epidemiological studies failed to find any excess of gastrointestinal cancer among friction material production workers; chrysotile, anthrophyllite, and talc miners, chrysotile factory workers, asbestos gas mask workers, asbestos textile workers, and shipyard workers.

In summary, 12 different epidemiological studies of a variety of occupational cohorts exposed to asbestos have found excess mortality from gastrointestinal cancers; of these, 7 studies found statistically significant excesses. OSHA believes that these findings constitute substantial evidence of an association between asbestos exposure and a risk of incurring gastrointestinal cancer.

2. Experimental Studies

In the November proposal, OSHA discussed a number of toxicological studies conducted on animals to determine the carcinogenicity of ingested asbestos. A study conducted by Ward et al. (Ex. 84-200) found that 32 percent of amosite-treated Fischer 344 rats developed colon carcinoma; a fairly high incidence of colon tumors compared with the incidence among historical controls from the same laboratory.

Two studies show evidence of gastrointestinal tumors developing in chrysotile and amosite-treated animals but not in the control animals (Bolton et al. Ex. 84-214; Smith et al., Ex. 84-193). However, these results were

considered questionable by the authors because, in the case of the Smith et al. study (Ex. 84-193), other investigators observed the same types of tumors in the animal strain studied and, in the case of the Bolton et al. study (Ex. 84-214), asbestos fibers were not found in the mesenteric lymphatic tissue of the amosite-treated animals that developed benign tumors.

However, several studies reported no significant increases in tumor incidence after the administration of chrysotile, amosite, tremolite, and crocidolite asbestos orally to laboratory animals; these studies included those conducted by the National Toxicology Program (NTP) (Exs. 84-225, 84-226, 84-227, and 84-228) and by Donham et al. (Ex. 84-222). In the NTP studies doses well below the maximum tolerated dose (1 percent of diet) were administered, and in some of the NTP studies, relatively short fibers were administered. Although the study by Donham et al. failed to show a significant increase in tumorigenesis, the authors believed that their results showed a trend towards increased colon lesions.

Since the November proposal OSHA has reviewed an additional lifetime feeding study of amosite-treated rats. McConnell et al. (Ex. 306) administered amosite asbestos (1 percent of diet) to a group of 250 8-week-old male and female Fischer 344 rats. When animals were examined for tumors, the incidence of gastro-intestinal tumors among treated male and female rats (7/249 and 4/250, respectively) was found to be comparable to that of untreated male and female controls (4/117 and 2/117, respectively). Treated male rats were found to have a significantly higher incidence of C-cell carcinoma (50/246), compared to male controls (11/117), but due to the lack of other significant findings, the authors did not attribute the increase in the incidence of C-cell carcinoma among treated male rats to amosite exposure.

Although OSHA finds that results from ingestion studies are equivocal and inconsistent with respect to the carcinogenic potential of exposure to asbestos via ingestion in animals, OSHA does not believe that the negative findings from these studies negate or diminish the strong evidence from epidemiological studies. OSHA believes that the study of 17,800 insulation workers conducted by Selikoff et al. (Ex. 84-090) carriers considerable weight with respect to the issue of gastrointestinal cancer and asbestos exposure. This study, which found significant excess morthality from gastrointestinal, laryngeal, kidney, and phrayngeal and buccal cavity cancer, had the highest statistical power of all the epidemiological studies reviewed by OSHA.

*D. Epidemiologic Evidence of the Risk of Asbestosis From Exposure at the Existing PEL*

The existing standard of 2 fibers/cc was established primarily on the basis of an excess risk of asbestosis among workers exposed to asbestos. Since 1972 when the PEL was promulgated, a number of studies with more precise exposure data suggest that a significant excess risk of asbestosis still exists at 2 fibers/cc. The purpose of this section is to review this evidence in light of the revised standard.

This section is organized into three parts. In part 1, asbestosis is described and the variability associated with its diagnosis is discussed with regard to the interpretation of epidemiologic data. In part 2, the disease burden associated with asbestosis is discussed, along with the problems related to the underascertainment of cases. Studies that provide data on asbestosis incidence at low exposure levels are critically reviewed in part 3.

1. Introduction

Asbestosis is characterized by diffuse interstitial fibrosis of the lung. It falls into the class of diseases called pneumoconioses and is caused solely by exposure to asbestos. Asbestosis is a progressive disease and, as such, occurs with varying degrees of severity (Berry et al., Ex. 84-20). The signs and symptoms of asbestosis are no different from those of other forms of interstitial fibrosis and, as a result, the diagnosis is subject to differences in interpretation, resulting in both false negative and false positive conclusions.

In unexposed populations, the diagnosis of asbestosis is rare or nonexistent. A history of asbestos exposure is

essential to the diagnosis, which is typically made on the basis of physical examination and x-ray evidence and less often by means of accompanying pulmonary function tests. No single sign or symptom predicts the presence of or progression to asbestosis (Murphy, Ex. 84-314; Berry et al., Ex. 84-20). However, selected combinations of signs and symptoms appear to have high predictive value for the progression to asbestosis. Nonetheless, in some cases, minor fibrosis with considerable respiratory impairment and disability can be present without equivalent X-ray changes. Conversely, extensive radiographic findings may be present with little functional impairment (Exs. 84-2, 84-338).

Symptoms of early disease include a non-productive cough and fatigue. As fibrosis progresses, shortness of breath is apparent, even with minimal exertion. Rales, i.e., crackles heard on inspiration, are often present but are non-specific and thought to be no more prevalent in absestosis than in other fibrotic diseases (Craighead et al., Ex. 84-033). However, in two studies of workers exposed to asbestos, besilar rales occurred with asbestosis in almost all cases (Murphy, Ex. 84-314; Berry et al., Ex. 84-20). Clubbing of the fingers is seen in the late states of the disease but does not appear to be as specific as other signs or symptoms (Murphy, Ex. 84-314). Cyanosis of the tongue and mucous membranes may also occur in the later stages of the disease (Ex. 84-27).

The roentgenologic diagnosis of asbestosis is based on the presence of small irregular and round opacities distributed prominently in the lower lung fields, accompanied by evidence of pleural fibrosis, pleural calcification, or thickening. Specific details regarding the radiographic features associated with the progression and diagnosis of asbestosis are noted elsewhere (Craighead et al., Ex. 84-033). The presence of crepitations and X-ray changes does not indicate directly that health is impaired, in contrast to the presence of diminished lung function. Typical pulmonary function changes associated with asbestosis include diminished FVC and FEV[1] (Murphy, Ex. 84-314; Berry et al., Ex. 84-20), and, as shown in one study (Murphy, Ex. 84-314), reduced total lung capacity. Evidence does not support a direct relationship between obstructive airway disease, such as is caused by cigarette smoking; rather than by obstructing airways, asbestosis diminishes lung function by restricting the ability of the lung to expand and contract.

Asbestosis is a disease that is irreversible and that evolves and progresses even in the absence of continued exposure. It is not known whether removing an individual from exposure after the appearance of early signs and symptoms will reduce the risk of progression to more severe stages. The probability that asbestosis will progress in the absence of continued exposure appears to be subject to individual variation, as pointed out by Dr. Selikoff:

What we don't know . . . is whether people who are removed from exposure have less progression than people who continue exposure. I wish we knew that.

There are very few data on this. . . . You can be exposed, have an abnormal X-ray, and either continue exposure or be removed from exposure, and not progress.

On the other hand, you can be removed from exposure and have progression occur. . . .This is an individual reaction (Tr. 7/2, p. 171).

At present, the only reliable means of preventing the occurrence of asbestosis is to reduce the cumulative exposure incurred by individuals during their working lifetimes to a level below which the risk of disease is very low.

2. Excess Morbidity and Mortality Attributable to Asbestosis

The morbidity and mortality of asbestosis have been studied in workers exposed to asbestos. Excess morbidity is determined from the incidence of disease, which is the rate at which new cases of disease are diagnosed for a given number of person-years of observation. It is important to establish the date at which the disease first

occurs to accurately estimate the incidence of asbestosis. The mortality rate for asbestosis is the number of deaths due to asbestosis for a given number of person-years of observation. The cause and date of death are determined from death certificates. When a disease has a high case fatality rate; i.e., when the interval between diagnosis and death is short, then the mortality rate and incidence will be similar.

To estimate the incidence of asbestosis, periodic examinations of workers is essential to both identify cases and accurately determine the date of diagnosis.Cases with asbestosis will be missed if they are lost to followup, i.e., cannot be located for examination. If the rate of asbestosis among those who are lost to followup is higher than among those who are examined, a relatively high loss rate can bias the estimate of risk.

The diagnosis of asbestosis can be difficult. It is important, therefore, in epidemiological studies to use standardized methods of diagnosis such as those established by the ILOC. The diagnostic criteria used by different investigators very considerably, and this can account for some of the differences in the incidence of asbestosis seen between studies.

When a disease is well defined and the case fatality rate is high, such as occurs with lung cancer, the mortality rate will be similar to the incidence of the disease. In contrast, when the disease is not well defined or is difficult to diagnose and the case fatality rate is not high, the mortality rate will be less than the incidence. Unlike lung cancer, the onset of asbestosis is not always life threatening. As the disease progresses and health deteriorates, a subject may seek medical care. In the interim, however, the victim may die from other more easily recognizable causes and the existence of the asbestosis and its associated morbidity will not have been ascertained, even though asbestosis may have been the underlying or contributing cause of death. This is a special problem with diseases like asbestosis which are virtually absent in populations that are not exposed to asbestos.

Hammond, Selikoff, and Seidman (Ex. 84-47, p. 475) note that "what is recorded on the death certificate is not always [based on] the best available information on the cause of death. For example, in the absence of the patient's physician, the certificate may be signed by a doctor who knows less about the case; or an autopsy may indicate that the tentative diagnosis of cause of death was incorrect." In addition, a review of available evidence, such as from the medicalrecord, may indicate that the patient died of another cause of death. Hammond et al. (84-47) reviewed all available medical information, including the death certificates for all deaths in a cohort of insulators. Two causes of death were established: one based on the death certificate (DC) only; and a second cause based on the best evidence available. Seventy-six cases of asbestosis were identified from the death certificate. On the other hand, 160 cases were identified on the basis of the best evidence. In contrast, 638 deaths due to cardiovascular disease were ascertained from DC, while only 566 were identified from the best evidence. These data are consistent with the work of Dement et al. (84-37) who found a statistically significant excess risk of cardiovascular disease among asbestos textile workers. This is unusual because the SMR for cardiovascular disease in working populations is consistently less than 100, reflecting a "healthy worker effect."

Unlike diseases such as lung cancer and mesothelioma, which have a relatively short interval between diagnosis and death, individuals with asbestosis experience a relatively long and debilitating period of morbidity. Dr. Holstein, a pulmonary physician, described a typical case:

The main symptom of asbestosis is progressive shortness of breath. When this has its onset in its typically insidious and gradual manner, the individual thinks that he is just getting older or getting a little overweight, can't run as fast as he used to, or gets out of breath more easily than he used to; and attributes it to factors such as the ones I mentioned. A little later on, the person begins to notice that in fact, he or she can't do the things that many other people the same age can do. . . . As time goes on, the dependence on younger workers becomes greater and greater, until pretty soon, the individual is experiencing the fact that he or she really can't carry out the job without such dependence. . . . Eventually, in the very severe cases, a person's life consists of sitting in

an armchair on the ground floor with an oxygen tank, and disconnecting it just long enough to get up and go to the bathroom.

Hence, there are limitations to using death certificates to determine the extent of mortality attributable to asbestosis. Cases will be underascertained and the person-years of morbidity, i.e., the period between diagnosis of asbestosis and death, are not considered. For these reasons, risk analyses of mortality caused by asbestosis will understate the true risk of disease.

3. Epidemiologic Studies

A number of studies have shown an excess risk of asbestosis in workers exposes to asbestos. Individual exposure data in units of fibers/cc-years are available from three studies, all of which show substantial excess risks below 100 fibers/cc-years (the cumulative lifetime exposure permitted by the 2-f/cc-standard) (Berry et al., Ex. 84-20; Dement et al., Ex. 84-35; Finkelstein, Ex. 84-44). These studies are critically reviewed below. Individual exposure data were also used in two other studies but were reported in units of mppcf-years (McDonald et al., Ex. 84-065; Enterline et al., Ex. 84-43). Several other studies that show an excess risk of asbestosis are not reviewed here because the exposure measure was expressed only as duration of time exposed (Weiss, Ex. 84-097; Doll, Ex. 84-40; Pearle, Ex. 84-079) rather than as exposure level.

The approach for assigning exposure levels to individuals, the method used for person-years analysis, the case definition, the completeness of case ascertainment, and the length of the followup period are directly related to the estimated risk of asbestosis at a defined exposure level. These factors are particularly relevant to the studies of Berry et al. (Ex. 84-20, Dement et al. (Ex. 84-35), and Finkelstein (Ex. 84-44).

All three of these studies, which assigned individual exposures in units of fibers/cc, used person-years analysis to estimate the risk of asbestosis in groups of workers defined by their cumulative exposure to asbestos.To derive such estimates the number of years that workers are exposed must be summed for all workers exposed at each exposure level. If an individual leaves the workplace, subsequent years of followup are assigned to the final cumulative exposure incurred by the individual. Two methods are typically used to assign person-years of observation to the cumulative exposure levels incurred during employment. The first approach assigns the number of person-years of observation before the disease is diagnosed in each successive cumulative exposure category. For example, four exposure groups are defined in terms of employment: greater than 5 years; 5-9 years; 10-14 years; and 15+ years. An individual with 20 years of employment contributes person-years of exposure to all four exposure groups. The same principle applies if asbestos exposure is defined as cumulative fibers/cc rather than duration of employment. That is, before person-years of exposure can be assigned to any cumulative exposure group, a worker must have first experienced a lower cumulative exposure; the person-years of exposure are then assigned in accordance with the length of time the worker spent at each exposure level. An alternative approach assigns the total number of person-years of observation only to the highest cumulative exposure group, i.e., in our example, to the denominator for the 15- to 20-year cumulative exposure group. Use of the latter method underestimates the disease incidence in the higest exposure groups and overestimates the incidence in the lowest exposure groups. Dement et al. (Ex. 84-35) and Berry et al. (Ex. 84-20) both used the first approach, while Finkelstein (Ex. 84-44) took the second approach.

The completeness of case ascertainment is directly related to the length of the followup period. If a study's followup period is relatively short, then cases will be underascertained and the risk of disease will be underestimated. In addition, if latency is related to cumulative exposure, i.e., if the median latency is short for high-exposure groups and longer for the low-exposure groups, then the rates for each cumulative exposure group will be underestimated differentially, and most significantly for the lowest exposure group.

On the other hand, if the initial period of followup is ignored then the risk for workers with high exposure may be underestimated. In a study of lung cancer in asbestos cement factory workers Finkelstein (84-240) estimated

the relative risk for workers in three exposure groups, starting 20 years after onset of exposure. The highest exposure group had the lowest risk. If a higher exposure causes a shorter disease latency, then proportionately more cases in the highest exposure group would have occurred prior to the beginning of the followup period, i.e., during the first 20 years since onset of exposure, and would have been missed.

It is often stated that cases of asbestosis are rarely seen within 15-20 years of first exposure; however, Berry et al, (Ex. 84-20) have shown that signs associated with interstitial fibrosis are seen less than 10 years from first exposure and clearly within 10-14 years of first exposure. In addition, Dement et al. (Ex. 84-35) have shown an excess mortality due to other non-malignant respiratory diseases within 10-19 years after initial employment. Selikoff et al. (Ex 84-189) have reported on asbestosis death rates in a cohort with the longest period of followup.In this study, the mortality rate began to increase approximately 13 years after first exposure. A decline in the death rate from asbestosis occurred 45 years from first exposure. The authors suggested that competing causes of death, in part due to smoking, may have accounted for this decline. It is not possible, however, to tell whether the incidence of asbestosis, i.e., the occurrence of new cases, would also have begun to decline 45 years after first exposure.

Berry et al. (Ex. 84-20) studied textile factory workers. Two cohorts were defined: Workers first employed between January 1, 1933 and December 31, 1950 who were still employed as of June 30, 1966; and workers employed after June 30, 1966 who had completed at least 10 years of service up to December 31, 1972. The latter cohort is important because measures of dust fiber levels were available beginning in 1951 and the exposure estimates for individuals in the study are likely to be more valid after that time. In addition, proportionately more of the latter cohort was exposed to a lower mean dust level than the former cohort. Although these dust levels are not equivalent to or below the level stipulated by the current PEL, they are closer to it than the mean fiber or dust levels incurred by the earlier cohort. The maximum number of years of followup. 24, was considerably less than the latency for late-onset cases of asbestosis. Among those first employed after 1950, Berry and his colleagues estimated a 1 percent prevalence of crepitations, "possible asbestosis," and "certified asbestosis" at 37, 46, and 63 f/cc-years, respectively, suggesting that an excess risk exists at levels below 100 f/cc-years. Since the average number of years of followup in this study was only 16 years, new cases will have accrued in the subsequent period. It is also noteworthy that when men who had left the factory prior to 1966 were included in the cohort in an effort to reduce the selection bias associated with the risk of asbestosis, the prevalence of signs associated with the disease increased. However, even in the more complete cohort studied, selection factors remained. The overall effect of these methodological problems is that the measures of prevalence in this study are underestimates.

Finkelstein (Ex. 84-44) studied the risk of asbestosis in 157 Ontario cement production workers first exposed to asbestos between 1948 and 1960 and employed for at least 15 years. Because of the nature of the cement production operation, some workers employed at this plant may not have been exposed to asbestos. Workers were followed up until death or up to October 1, 1980. The number of years since first exposure ranged from 18-33 years, with a median of 25 years, Cases were ascertained primarily through annual examinations or by means of death certificates. The author noted that "83 percent of the production workers received an examination for asbestosis within the 3 years prior to the cutoff date or within 3 years of their death" (Ex. 84-44). It is uncertain how the production workers who were lost to followup (17 percent) were handled in this analysis; OSHA assumes that only those person-years of observation up to the time of the last followup examination were included.

The Ontario criteria for certifying asbestosis, which results in an award of disability pensions, are not strictly defined but involve considerations of such factors as history of occupational exposure, dyspnea, crepitations, clubbing of fingers, radiographic signs of pulmonary fibrosis, and abnormal lung function. In general, it can be assumed that, despite the absence of definitive criteria, the certified asbestosis cases included in this study occurred at an advanced stage of the disease, in contrast to other studies that included possible cases of asbestosis in the analysis.

Only two cases in the Finkelstein study were certified before the 20th year after first exposure. A majority of the asbestosis cases were certified between 22 and 28 years of exposure; however, cases continued to accrue in the followup perido, and there is no clear evidence that the incidence of asbestosis declined after more than 28 years since first exposure. Finkelstein calculated incidence as the number of new cases of certified asbestosis per 100 person-years at risk, i.e., the rate at which new cases of asbestosis develop in a given period of time, which results in a direct measure of the risk of developing of disease. The Finkelstein study found the incidence of asbestosis to be 0.5, 3.4, and 6.5 per 100 person-years of exposure for workers receiving 0-49, 50-99, and 100-149 f/cc-years of exposure, respectively, showing a positive dose-response relationship.

Using a life table method, Finkelstein also calculated the cumulative probability of developing a certified case of asbestosis after 32 years of followup and observed that men in the 0-49, 50-99, and 100-149 f/cc-years categories and 10 percent, 55 percent, and 70 percent probabilities, respectively. Although these estimates are somewhat uncertain because of the small number of subjects in each category, especially in the lowest exposure category, the data do indicate that, even for exposures below 50 f/cc-years, there is an excess risk of asbestosis morbidity. In addition, as noted above, there is no evidence to suggest that the risk of asbestosis declines after 32 years from first exposure.

Finkelstein (Ex. 84-044) notes that a selection bias may have been introduced by excluding workers with fewer than 15 years of employment from the cohort, resulting in an overestimation of risk at lower exposure levels. Typically, one assumes that the morbidity and mortality of the excluded group are the same as those of the group included in the study. Finkelstein suggests that if the excluded individuals had been considered, lower estimates of risk might have been obtained for the lower exposure category; however, this could only have occurred if the risk of asbestosis for the same cumulative exposure level among those excluded was less than that of the group studied. OSHA believes that it is not possible to determine the effect of such a selective exclusion.

Finkelstein estimated the cumulative exposure that would result in a 1 percent probability of developing asbestosis by extrapolating from his exposure-response curve. He arrived at a value of 10 f/cc-years, a figure considerably lower than that derived by Berry et al. (Ex. 84-20). One significant factor that may account for the difference in estimates between these two studies in a difference in the length of their followup period (i.e., it is considerably longer in the Finkelstein study). In addition, the workers studied by Finkelstein may have also been exposed to silica which was used in the production process. If there was an excess risk of silicosis from such exposure which was mistaken for asbestosis then the exposure level resulting in a 1 percent probability of developing asbestosis would have been overestimated. There were no data published on the silica exposure levels to determine if this was a possibility.

Dement et al. (Ex. 84-37) studied the risk of asbestosis in 1,261 males employed for one or more months in a chrysotile asbestos textile operation between January 1, 1940 and December 31, 1965. Mortality followup using data from death certificates was from January 1, 1940 to December 31, 1975 and was 98 percent complete. The method used in this study to assign exposure to individuals was described previously (48 FR 51102). There was a total of 33,141 person-years of observation, and 24 deaths were ascribed to "other respiratory diseases" (ICDA 751-527), the category that includes asbestosis. Of the 24 deaths in this category, asbestosis or pulmonary fibrosis was the underlying cause of 17 deaths. Nineteen of these 24 deaths occurred 20 or more years after initial employment. The overall Standardized Mortality Ratio (SMR) for this category was 552.There was also an increased SMR for deaths due to cardiovascular disease, which is consistent with other observations among asbestos workers. The authors note that "a review of death certificates for the 105 deaths found [that] 6 [certificates] mention asbestosis or pulmonary fibrosis as a contributing condition."

In the study by Dement et al. (Ex. 84-37), there was little difference in SMRs for the "other nonmalignant respiratory diseases" by years since initial employment. For the group observed 10-19 years after first

employment, the SMR was 521; for 20-29 years it was 565; and for greater than 30 years, the SMR was 570. It is noteworthy that even in the group observed 30 or more years after first employment, the SMR remains at an elevated level and showed no decline. The authors also derived SMRs for white males with 15 or more years since first exposure. These were 362, 84, and 879 for the exposure categories less than 1,000 f/cc-days (2.7 f/cc-years), 1,000-10,000 f/cc-days (2.7-27.4 f/cc-years), and 10,000-40,000 f/cc-days (27.4-109.6 f/cc-years), respectively. The excesses were statistically significant for both the first and the third exposure categories, and both of these cumulative exposures are below the cumulative exposure that would be caused by 50 years of exposure at the existing PEL of 2 f/cc. Finally, since other investigators (Selikoff, Ex. 84-90; Elmes and Simpson, (Ex. 84-42) who have studied the mortality of asbestos workers have shown that relying only on death certificates for ascertainment of cases causes a significant number of asbestosis deaths to be missed, the risk estimates reporterd by Dement et al. (Ex. 84-37) may be understated.

4. Summary of the Evidence of Asbestosis

OSHA believes that the studies of Berry et al. (Ex. 84-20) and Finkelstein (Ex. 84-44) show a clear does-response relationship between asbestos exposure and asbestosis, and substantial excess risks due to asbestosis close to or below 100 f/cc-years, the cumulative lifetime exposure permitted by the 2 f/cc standard. The risk of mortality due to asbestosis in the work by Dement et al. (Ex. 84-37) was also in excess in workers exposed to less than 100 f/cc-years, despite the problems of underascertainment of cases. Because asbestosis morbidity is a better indication of risk than is mortality, OSHA has included the studies of Berry et al. (Ex., 84-20) and Finkelstein (Ex. 84-44) in a quantitative risk assessment for asbestosis (see Section V).

In his testimony Dr. Weill concluded that asbestosis deaths would be rare to non-existent under a two fiber standard and a disease of the past at a revised standard of 0.5 f/cc. He stated that:

. . . We are able to detect asbestosis with greater sensitivity. It means we are going to be seeing less severe disease. . . .

The asbestosis that is being seen generally now around the country again, I think by wide agreement . . . is at a low level, even now. This is associated with the exposures of the last several decades, when we know certainly in most instances and particularly in end-product use the exposure would still have been relatively uncontrolled.

In response to questions on the same issue Dr. Lewinsohn noted:

I think asbestosis as it was originally described is a vanishing disease, yes. I think that asbestosis is a different disease than we see now, if it still exists. It's much milder. It's less likely to be fatal. It's less likely to produce significant impairment. . . .

I think the levels of exposure have diminished and the changes, the disease itself is different. You don't see the full blown picture of asbestosis with people who die from asbestosis after less than 10 years' exposure with severely damaged lungs, with heart failure. . . .

What you see today is somebody who has pleural changes or somebody who has very minimal radiological features of asbestosis and who probably goes on to live a reasonably normal life span. . . .

The view that asbestosis mortality and severe asbestosis morbidity is on the decline is corroborated by the work of Berry et al. (84-20) who show that as the cumulative does of asbestos decreases, more cases are diagnosed as having crepitations only or as being possible asbestosis, in contrast to certified asbestosis. Nonetheless, severe cases (Barry et al. Ex. 84-20), disabled cases (Finkelstein, Ex. 84-44), and deaths due to asbestosis (Ex. 84-37) have been found to occur in workers with estimated cumulative exposures well below

100 f/cc-years. Although the clinical impressions of Drs. Weill and Lewisohn regarding a shift in the severity of asbestosis as exposures have declined in the past few decades may be correct, their conclusions regarding the eventual absence of disabling asbestosis and death due to asbestosis at the current standard of 2 f/cc are contradicted by the evidence from epidemiological studies mentioned above. In addition, Dr. Selikoff, under cross examination by Ms. Nash, testified that he is still seeing cases of severe asbestosis more than 10 years since the 2-f/cc standard became effective:

Nash: . . . In your clinical observations, are you continuing to see cases of asbestosis?

Selikoff: Oh, yes.

Nash: Are you continuing to see advanced cases of asbestosis --?

Selikoff: . . . We certainly do. We see deaths. But I have not had the experience of seeing what would happen at 0.1 [f/cc]. But I'm also not willing to expose a large number of people to 0.1 as guinea pigs, so that I can come along twenty years later and give you the answer.

Nash: So you would then obviously believe there's a risk of exposing people at higher levels and developing asbestosis . . .?

Selikoff: Oh, no question -- there is no question about that. That we already know from our extrapolation.(Tr. 7/2, p. 173)

Based on this testimony and the epidemiological data discussed above, as well as the results from OSHA's risk assessment (see Section V), OSHA finds that a reduction of the current 2-f/cc PEL will result in a continued decline in asbestosis incidence.

*E. Effects of Cigaretts Smoking and Asbestos Exposure*

This section discusses scientific evidence describing the influence of smoking on the risk from asbestos-related disease. Because several studies (Exs. 2-5; 84-190; 84-47) were cited in the November proposal as evidence of a multiplicative effect of asbestos exposure and cigarette smoking with regard to producing increased lung cancer risk, several commenters, including the Asbestos Information Association (Ex. 328), argued that OSHA overstated the risk of lung cancer in its qualitative risk assessment by failing to distinguish between the lung cancer risks for asbestos-exposed smokers and nonsmokers. While the scientific data are discussed here, Section VI (Significance of Risk) contains OSHA's response to comments dealing with how the lung cancer risk for asbestos-exposed smokers should be evaluated from a regulatory perspective.

Asbestos-Related Malignant Disease

Several studies were cited in the November proposal as evidence that asbestos-exposed workers who smoke have a higher risk of lung cancer mortality than either asbestos-exposed nonsmokers (Selikoff, Churg, and Hammond, Ex. 2-5; Selikoff, Seidman, and Hammond, Ex. 84-190; Hammond, Selikoff, and Seidman, (Ex. 84-047). The reduced ability of smokers to clear particles from their lungs, compared with the ability of non-smokers to do so, as suggested by Cohen et al. (Ex. 84-031), may help to explain the higher lung cancer risk of asbestos workers who smoke. The Agency also determined that there is no evidenc of an association between cigarette smoking and an increased risk of either mesothelioma or gastrointestinal cancer.

To exemplify the multiplicative effect of asbestos exposure and cigarette smoking in producing an increased lung cancer risk, OSHA discussed two studies at length (Hammond et al. Ex. 84-047; Selikoff et al., Ex. 84-190). The Hammond et al. study (Ex. 84-047) examined the smoking histories of 8,220 of 12,051 asbestos

insulation workers with a followup of 20 or more years since initial exposure. In late 1966, 6,841 of these workers were either current or past cigarette smokers, 488 had a history of pipe or cigar smoking, and 891 had never smoked regularly. The mortality of these workers was observed during the period 1967-1976. The comparison population, drawn from the American Cancer Society's long-term prospective study, consisted of 73,763 white men who had no more than a high school education, were not farmers, were alive as of January 1, 1967, and had a history of occupational exposure to dust, fumes, vapors, gases, chemicals, or radiation. The age-standardized lung cancer mortality rate for non-smoking controls (i.e., the baseline rate) was 11.3 deaths per 100,000 man-years; the rate for smoking controls was approximately 11 times higher (122.6 per 100,000 man-years). The lung cancer mortality of non-smoking asbestos workers was 5 times higher (58.4 deaths per 100,000 man-years) than that of non-smoking controls (11.3 deaths per 100,000 man-years). Hammond et al. (Ex. 84-047) found that the lung cancer mortality of asbestos workers who smoked was 601.6 per 100,000 man-years, a value that is also about five times higher than the baseline rate of lung cancer mortality for smoking controls.

Selikoff et al. (Ex. 84-190) examined the effects of cigarette smoking and asbestos exposure among 582 amosite production workers, 567 of whom had smoking histories. As in the study by Hammon et al. (Ex. 84-047), the age and cause-specific mortality rates were compared within each smoking status category defined by the American Cancer Society cohort. Selikoff et al. (Ex. 84-190) concluded as follows:

Here asbestos exposure greatly multiplied the already high risk that would have been present with cigarette smoking alone. . . . This increased risk is very much the same as that seen among asbestos insulation workers [who smoked]. This observation indicates that the increased risk of death from lung cancer among cigarette-smoking asbestos workers is a specific interaction rather than coincidental, and not, for example, the result of other agents in the environment of the construction trades." (Ex. 84-190).

In the November proposal, OSHA presented two ways of calculating the *probability* that any single case of lung cancer in a person with known exposure to asbestos could be attributed to the asbestos exposure. The first way, proposed by Enterline (Ex. 84-126), was based only on relative risk estimates. Using data from Selikoff et al. (Ex. 84-090) on asbestos insulation workers, Enterline estimated that there was a probability of 75 percent that lung cancers were attributable to asbestos exposure; this probability applied both to smoking and non-smoking asbestos workers. However, in the case of asbestos workers who smoked, OSHA deems it inappropriate to dichotomize causation in terms of smoking *or* asbestos exposure because of the synergistic effect between cigarette smoke and asbestos. OSHA therefore presented its method of calculating the probabilities of causation in the November publication (Table 6 and Table 7, 48 FR 51110). Although OSHA's caculations differ from Enterline's calculations of attributable risk by including a factor for synergism, the two probability estimates do not differ by a great extent. According to OSHA's calculations, asbestos exposure contributes to 79.4 percent of lung cancer deaths among asbestos-exposed workers who smoke, and 77.2 percent of lung cancer deaths among nonsmoking asbestos workers.

Lung Disease and Chest X-ray Abnormalities

In the study by Hammond et al. (Ex. 84-047) discussed above, it was also reported that asbestos insulation workers who smoked one or more packs of cigarettes per day had an asbestosis mortality rate 2.4 times higher than that of asbestos insulation workers who had never smoked regularly. Selikoff et al. (Ex. 84-190), however, observed no increased risk of death from asbestosis among amosite production workers who smoked compared to their nonsmoking co-workers.

Weiss (Ex. 84-097) conducted a chest x-ray and questionnaire survey of 100 asbestos textile workers. Chest roentgenograms were examined for evidence of pulmonary fibrosis. Two asbestos exposure groups were defined: those with less than 20 years of exposure and those with 20 or more years of exposure. The age-adjusted prevalence of pulmonary fibrosis among smokers and non-smokers was 40 and 23 percent,

respectively. None of the 11 non-smokers with less than 20 years of asbestos exposure had pulmonary fibrosis, in contrast to 29 percent of the smokers with less than 20 years of exposure to asbestos. The median duration of exposure to asbestos was similar for these two groups. Based on these findings, Weiss (Ex. 84-097) concluded that both asbestos exposure and cigarette smoking were associated with pulmonary fibrosis and that asbestos workers who smoked had a higher prevalence of fibrosis relative to that among nonsmoking asbestos workers. Weiss did not indicate whether the difference in the prevalence of pulmonary fibrosis between smokers and nonsmokers was statistically significant. OSHA tested the significance of the reported difference using a chi-square test of proportions and did not find a significant difference (p greater than 0.1). This study and its findings were criticized by Kilburn (Ex. 84-237) because Weiss used a definition of pulmonary fibrosis that differed from the standard International Labor Office 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 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 30+ 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 rediological 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 18.8 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. 15.3 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 (8.9 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 rediological 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 anthophyllite, which has been observed to induce lung cancer and asbestosis, but not mesothelioma (OSHA/NIOSH, Ex. 84-200; for amosite: Seidman et al., Exs. 84-87, 261-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. 84-59; Nicholson et al., Ex. 84-72; Rubino et al., Ex. 84-86; Dement et al., Ex. 84-37; Acheson and Gardner, Ex. 84-15; and Berry and Newhouse, Ex. 84-21; for crocidolite: Jones et al., Ex. 84-138; Hobbs et al., Ex. 84-132; McDonald and Newhouse, Ex. 163; Berry and Newhouse, Ex. 84-21; and Newhouse et al., Ex. 163; for anthophyllite: Meurman et al., Ex. 84-181; for tremolite and actinolite: Brown et al., Ex. 84-29; for mixed fiber types: Hughes and Weill, Ex. 84-135; Weill et al., Ex. 84-206; Jones et al., Ex. 84-138; Berry et al., Ex. 84-20; Elmes and Simpson, Ex. 84-42; Peto et al., Ex. 84-80; Lacquet et al., Ex. 84-144; Selikoff et al., Ex. 84-89; Robinson et al., Ex. 84-82; and Balselga-Monte and Segarra, Ex. 84-19).

Several investigators and committees have suggested that exposure to crocidolite and amosite is associated with a different carcinogenic potential than is exposure to chrysotile and anthophyllite, primarily with regard to the risk of mesothelioma (Enterline and Henderson, Ex. 84-122; McDonald and McDonald, Ex. 84-154; Weill et al., Ex. 84-206; Acheson and Gardner, Exs. 84-15, 84-216, and 84-243; Muir, Ex. 84-350; and the Advisory Committee on Asbestos, Ex. 84-216). Among the studies reviewed by OSHA, the variation in mesothelioma mortality among cohorts exposed to different fiber types, expressed as a percentage of all deaths attributed to

mesothelioma, is as follows: for crocidolite, 1.26 to 16 percent (McDonald and McDonald, Ex. 84-154; Jones et al., Ex. 84-138; McDonald and Fry, Ex. 84-64; Hobbs et al., Ex. 84-132); for amosite-chrysotile mixtures containing less than 0.1 percent crocidolite, 4 to 7.7 percent (Hammond, Ex. 84-47; Peto, Ex. 84-168; Robinson at al., Ex. 84-82); for amosite, 2.7 percent (Seidman, Ex. 84-87); and for chrysotile, 0 to 0.5 percent (McDonald et al., Ex. 84-65; Nicholson et al., Ex. 84-72; Dement et al., Ex. 84-37; Rubino et al., Ex. 84-86). Mesothelioma has not been found to be a cause of death among miners exposed to anthophyllite (Meurman et al., Ex. 84-256). The Chronic Hazard Advisory Panel (CHAP) (Ex. 84-256) stated that it appeared that peritoneal mesothelioma was most commonly seen among workers exposed to amosite, less often among workers exposed to crocidolite, and rarely or never among workers exposed to chrysotile. However, as the Panel's report points out, large variations in the data describing peritoneal mesothelioma mortality from crocidolite exposure, frequent misdiagnosis of peritoneal mesothelioma, and the lack of risk data expressed in terms of unit exposure level complicate making definitive conclusions regarding the relationship between fiber type and mesothelioma risk.

For lung cancer, OSHA views the epidemiological evidence for differentials in risk by fiber types as being inconclusive and inconsistent. Some studies (Dement et al., Ex. 84-37; McDonald and Fry, Ex. 84-64) have found that workers exposed to chrysotile have approximately the same or higher risks of lung cancer compared to workers exposed to amphibole fibers, while other studies (McDonald and McDonald, Ex. 84-154; Henderson and Enterline, Ex. 84-158) have found that workers exposed to chrysotile have a lower relative risk of lung cancer. After comparing lung cancer risks per unit of cumulative exposure (also known as K[L], the lung cancer potency factor) among cohorts exposed to different fiber types, the CHAP (Ex. 84-256) reported that studies of workers exposed to chrysotile yielded both high and low values of K[L], as did studies of workers exposed to crocidolite or amosite. Therefore, a consistent pattern showing a higher lung cancer risk among workers exposed to chrysotile or amosite did not emerge. Dr. William Nicholson of the Mount Sinai School of Medicine (Ex. 94) agreed that these conflicting values for K[l] demonstrate that no unique lung cancer risk could be attributed to a particular fiber type. OSHA also concluded (48 FR 51115) that some cross-cohort comparison studies failed to control for important variables such as fiber concentration, age distribution, length of followup observation period, and fiber size distribution.

In the November proposal, numerous studies were discussed that demonstrated that chrysotile, amosite, crocidolite, and anthophyllite asbestos fibers are carcinogenic when administered to laboratory animals via inhalation, injection, and implantation (NIOSH, Ex. 84-338; NIOSH/OSHA, Ex. 84-320; Wagner et al., Exs. 84-205, 94-96, 84-197; Davis et al., Ex. 84-120). In general, animal studies that used standardized asbestos samples from the Union Internationale Centre Cancer (UICC) have demonstrated that chrysotile was more fibrogenic and carcinogenic than emphibole asbestos. For example, Davis et al. (Ex. 84-120) showed that UICC reference samples of chrysotile exhibited a greater potential to produce fibrosis in rats via inhalation than the amphiboles; however, treatment of rats with factory samples of these two types of asbestos showed no difference in fibrogenic potential (Ex. 84-120). Bolton et al. (Ex. 236-C) treated SFF Wistar rats by intraperitoneal injection with UICC chrysotile and UICC crocidolite asbestos and found that chrysotile produced a higher incidence of mesothelioma than crocidolite over a dose range of 0.01 mg to 25 mg per rat. Although, in another study, UICC reference samples of chrysotile and amphiboles inhaled by rats showed similar potential to produce fibrosis and lung tumors (Ex. 84-96), the NIOSH/OHSA Asbestos Work Group commented that, based on the amount of dust deposited and retained in the lung, this study, in fact, showed that chrysotile was more fibrogenic and carcinogenic than the amphiboles (Ex. 84-320, p. 15). Both Canadian and Rhodesian chrysotile prouced lower incidences of mesothelioma than crocidolite, amosite, or anthophyllite when these forms of asbestos were administered intrapleurally to laboratory animals (Ex. 84-197), but no differences in mesothelioma incidences among animals treated with these asbestos types were apparent in another study (Ex. 84-338). These latter studies (Exs. 84-197, 84-338) illustrate the conflicting findings of earlier animal experiments where UICC reference asbestos samples were not not used.

At the informal hearing, Dr. John M. G. Davis of the Institute of Occupational Medicine, Edinburgh, Scotland,

described recent animal experiments that he conducted to examine the relationship between fiber type and the development of asbestos-related disease (Tr. 7/9, pp. 3-79). In one rat inhalation study (Tr. 7/9, p. 16), 10 percent of the lung tissue taken from rats exposed to 10 f/cc UICC chrysotile showed evidence of scarring; only 1.5 and 2.5 percent of lung tissue taken from rats exposed to the same fiber concentration of crocidolite and amosite, respectively, were scarred. The same trend was observed for the incidence of malignant tumors found in exposed rats. Dr. Davis also discussed injection studies on rats (Tr. 7/9, p. 29) that showed, at doses ranging from 0.01 to 15 mg, that chrysotile produced the greatest number of mesothelioma tumors at every does tested. Dr. Davis concluded from these studies that ". . . both by fiber number and by fiber mass, chrysotile appeared to be the most dangerous" (Tr. 7/9, p. 15).

The animal studies reviewed by OSHA and the work described by Dr. Davis suggest that chrysotile has a greater fibrogenic and carcinogenic potency than the amphiboles, a finding that contrasts with the findings of human epidemiological studies that suggest that the amphiboles have a greater potential for producing mesothelioma. Several explanations for these conflicting results were offered into the record. Dr. Davis testified that part of the reason for the different findings between animal and human studies is that . . . 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. 84-171, 84-175, 84-178, 84-202, 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 sued by the workers were chrysotile, 86 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 amphilboles 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. 84-256, 99, Tr. 7/9, p. 39) expressed the opinion that chrysotile fibers, which are composed of several hundered 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, facilitiating 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 one fiber 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. 7/9,X, pp. 38-39).

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. Rulemarking 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. 65)

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 V) and in the establishment of a permissible exposure limit (see Section X) 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. 84-193, 84-195) 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 8 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 and Kuschner (Ex. 84-210) injected asbestos intratracheally into guinea pigs and found fibrosis only with fibers longer than 10 um (Ex. 84-128). NIOSH (Platek et al., Ex. 84-240) conducted inhalation studies in rats with chrysotile fibers less than 5 um in length and did not find increased incidences of pulmonary fibrosis or tumors compared with incidences in controls. Since NIOSH has difficulty in generating fibers with an aspect ratio of 3:1 or greater by the ball milling method and in counting the fibers, the NIOSH results only suggest that short fibers do not induce pulmonary fibrosis or tumors. However, studies conducted by Koler (1982) and Pott et al. (1972, 1976), as discussed by the National Research Council (Ex. 321, p. 182), suggest that amorphous asbestos and fibers shorter than 5 um can induce mesothelioma in rodents, a finding that contrasts with the findings of other animal studies reviewed above.

One problem with the studies conducted by Stanton et al. (Exs. 84-193, 84-195) was the difficulty in generating asbestos samples with fibers of uniform lengths; because of this difficulty, and authors could not conclude that short asbestos fibers were safe despite the finding that exposure to shorter fibers were associated with lower tumor incidences in animals. At the hearing, Dr. Davis (Tr. 7/9, pp. 20-28) discussed some of his findings from rat inhalation and injection studies that used carefully prepared long-and short-fiber samples of amosite. For the inhalation experiment, rats were exposed for 12 months by inhalation to amosite samples of varying fiber lengths. Animals were observed for their full lifespans. Davis observed 12 tumors, as well as extensive lung scarring, in 40 animals exposed to the long-fiber dust. No tumors or scarring was found among animals exposed to the short-fiber dust (Tr. 7/9 p. 26). The amosite samples were also injected into the peritoneal cavities of groups of 25 rats. The long-fiber sample produced mesotheliomas in 95 percent of the animals treated, while the short-fiber sample produced only one mesothelioma tumor. Dr. Davis concluded from these studies that short asbestos fibers were ". . . unable to damage tissues" (Tr. 7/9, p. 28).

Researchers (Ex. 86-4) have also found that a significantly higher percentage of long fibers (greater than 5 um) are retained in the lungs of mesothelioma and asbestosis victims. Morgan (Ex. 86-3) showed that authophyllite fibers less than 5 um in length were more easily cleared from rat lung than larger fibers. It has been well established that shorter fibers are readily engulfed by lung macrophages and transported to the mucociliary escalator or to the lymph system (Exs. 83-3, 86-4, 236-A, 321, Tr. 7/9, pp/ 5-6).

Several researchers (Exs. 86-3, 84-210, 86-4, 236-A, 321, Tr. 7/9, pp. 5-6) have theorized that the greater biological activity of longer fibers may be due to the inability of the macrophage to completely engulf the fiber. This may lead to the release of lysosomal enzymes and oxygen-free radicals from the macrophage, damaging alveolar epithelial cells and initiating fibrosis. In addition, the fibers may disrupt the normal proliferation and differentiation of lung fibroblasts either by directly interacting with the fibroblast or as a result of macrophage secretions.

Since the November proposal, OSHA has received much comment and testimony regarding the relative importance of fiber size, aspect ratio, and surface chemistry of the fiber to carcinogenic potential. Most of these commenters expressed the view that the surface chemistry of the fiber is an important determinant of disease. Dr. Dunnigan of the Universite de Sherbrooke (Ex. 91-15, p. 391, attachment) cited studies and comments of several investigators that were presented at the World Symposium on Asbestos (1982) that point to chemical factors rather than geometric or physical factors as the important determinant of asbestos fiber effects on the cell membrane. They postulated that asbestos fiber interaction with cell membrances causes cell homolysis. Mossman et al. (Ex. 321, p. 39) found asbestos-induced cell damage to be initiated by the reaction of the fiber with the plasma membrane, which causes cell lysis or phagocytosis. The National Research Council, National Academy of Sciences (Ex. 321) cites the work of Wilkinson (1976) and Stossel (1972), who found that

recognition of the asbestos fiber by phagocytes and their subsequent ingestion of the fiber may be due to physicochemical affinities between the fiber and the phagocyte. A study done by Light and Wei (Ex. 91-15) stated that "fiber dimensions are important in determining whether asbestos fibers are able to reach sites where critical cellular interactions take place, and thus could govern whether the potential biological activity of fibers due to their surface charge is displayed" (Ex. 91-15, p. 391, attachment).

Dr. Dunnigan (Ex. 91-15-2, p. 393) contended that, in view of studies suggesting that modification of the fiber structure affects the biological reactivity of the fiber, the "Stanton Hypothesis" (see Exs. 84-193, 84-195) should be reassessed. He argues (Ex. 227-A, Table 4) that this hypothesis assumes that all comminution methods merely reduce the dimensions of the fibers without altering other fiber characteristics. To illustrate that this may not be the case, Dr. Dunnigan (Ex. 91-15-2) cites a 1978 study by Arthur Langer that showed that "ball milling of experimental [asbestos] samples results in important changes in the structural and surface characteristics of asbestos fibers, and reduces their effects on all membranes" (Ex. 19-15-2, p. 393). He also cites a 1980 report done by Dr. Spurny in Germany that concluded that "milling procedures change not only the size distribution, but also the shape and crystal structure of asbestos fibers" (Ex. 19-15-2, p. 393).

In a further elaboration of the evidence against the fiber size theory, Dr. Dunnigan cited a study done by Poole et al. (1983) that shows erionite fibers (in a concentration of 150 f/ug mineral) of the "pathogenic" size range are more reactive than a larger number (1.6X10<5> f/ug) of similarly sized crocidolite fibers (Ex. 227-A-4, p. 12). Studies by Suzuki (1980), Wagner, (1982), and Maltoni et al. (1982) were also cited by Dr. Dunnigan (Ex. 227-A-4) as evidence that fibrous erionite is the most powerful mesothelioma-producing agent, suggesting that theese fibers may display disruptive or catalytic properties not shared equally by other types of fiber.

OSHA believes that the animal studies discussed above, in particular the recent work by Dr. Davis, point to a clear relationship between fiber dimension and disease potential. The finding in these studies that thin fibers (i.e., having an aspect ratio of at least 3:1) greater than 5 um in length are associated with elevated incidences of cancer and lung fibrosis is also consistent with current knowledge regarding lung clearance mechanisms, i.e., that shorter fibers are easily phagocytized and removed from lung tissue. OSHA also acknowledges recent findings that interactions between fibers and cell surfaces, in part, may also determine the course of asbestos-related disease. However, the mechanisms of fibercell interactions and their role in disease causation are not clearly understood at this time.

Some chemists have also suggested that the biochemically active sites or the electrical charge of the chemical groups on the asbestos fiber surface can be modified to reduce the hazardous potential of the fiber (Ex. 84-333). *In vitro* tests of modified asbestos fibers have shown decreased toxicity compared to the untreated asbestos fibers. Drs. Lemen and Groth of NIOSH (Tr. 6/21, pp. 189-191) testified at the public hearings that, to date, *in vitro* studies do not show with any degree of certainty that modification of asbestos fibers can prevent adverse health effects. They contend that the *in vitro* studies did not measure the fiber sizes of the modified asbestos fibers to determine whether the treatment shortened the fiber. Dr. Groth cited a study by Monchaux (Ex. 84-438) that showed that acid leaching leaching of chrysotile decreased its mesothelioma toxicity in rats; however, the treatment also shortened the fiber. He was not certain, therefore, whether the reduced toxicity derived from the treatment or the shortening of the fiber. In addition, no evidence was presented in the record to indicate that modified fibers are incapable of causing adverse effects after administration into laboratory animals. Mr. Warren, of the SNA, in his testimony agreed with this position. When asked whether OSHA should deregulate modified chrysotile, Mr. Warren responded:

SNA's position is not that it wants [modified fibers] to be exempted from regulation. Indeed, it expects to be covered. And that is certainly true because the in vivo testing is not completed. There is no present basis available for making any biological distinction [between modified and unmodified fibers]. (Tr. 7/5, p. 49)

Based on these considerations, OSHA has decided that it is prudent from a public health viewpoint to continue

to include chemically treated asbestos in the Agency's definition of asbestos (See Section IX. Summary and Explanation for General Industry).

*G. Tremolite and Anthophyllite*

In the November, 1984 notice, OSHA reviewed a number of epidemiological studies that suggested that the talc miners and millers are at excess risk of mortality from lung cancer, mesothelioma, and non-malignant respiratory disease (Exs. 84-025, 84-141, 84-181, 84-211), and have a high prevalence of pleural thickening and calcification, decreased pulmonary function, and lung fibrosis (Ex. 84-181). It is known that many, but not all, commercial talc deposits contain serpentine and amphibole asbestos and the minerals tremolite and anthophyllite, which may be found in amorphous, fibrous, or asbestiform habits (Ex. 84-039). At the time, based largely on epidemiological studies conducted by NIOSH (Ex. 84-029, 84-181), OSHA concluded that

"Talcs containing asbestos minerals . . . appear to pose a significant health risk to exposed workers, and talc workers exposed to asbestos should receive the protection afforded by the asbestos standard" (48 FR 51120).

Specifically, Brown et al. (Ex. 84-029) of NIOSH conducted a historical prospective study of talc miners and millers employed at a New York State talc facility operated by the R.T. Vanderbilt Company. Although the company reported that the talc at this facility contained no asbestos, NIOSH (Exs. 84-39, 84-181) reported finding asbestiform tremolite and anthophyllite following analysis of personal and bulk samples by election microscopy and x-ray diffraction techniques. As measured by optical microscopy, average air concentrations of fibers greater than 5 um in length ranged from 1.7 f/cc to 9.8 f/cc as an 8-hour TWA in the mine. In the mill, average 8-hour TWA exposures for these fibers ranged from 1.5 f/cc to 8.4 f/cc.

The cohort studied by Brown et al. (Ex. 84-029) consisted of 398 workers employed between 1947 and 1959. Cause-specific mortality rates were compared to those of U.S. white males, adjusted for age and calendar period. Brown et al. reported significantly elevated increases in cancer mortality (9 observed vs. 3.3 expected) and non-malignant respiratory disease mortality (8 observed vs. 2.9, expected). One death from mesothelioma was reported, but the death could not be specifically attributed to exposure to tremolite or anthopyllite. Of the 10 individuals who died of cancer, 3 worked previously for other New York State talc companies.

Gamble et al. (Ex. 84-181) of NIOSH aso conducted a cross-sectional morbidity study of the same facility. Of 156 male miners, 121 participated in a survey consisting of a respiratory questionnaire, chest X-ray, and spirometric testing. The morbidity experience of this cohort was compared to that of coal miners, potash miners, chrysotile asbestos workers, and synthetic wool textile workers. Coal and potash miners were used as comparison groups because they were likely to be similar to talc miners in many non-occupational respects that affect respiratory morbidity. Gamble et al. (Ex. 84-181) found that, compared to coal and potash miners, talc miners with no previous work history at other talc mines had a signficantly elevated prevalence of pleural thickening and calcification. When all talc workers were combined, with or without prior talc exposure, the researchers found increased prevalences of cough, phlegm production, dyspnea, and x-ray abnormalities. Talc workers also had significantly decreased pulmonary function, which was associated with duration and intensity of exposure.

OSHA also reviewed a third study that presented conflicting findings in workers at the same facility (48 FR 51118). Stille and Tabershaw (Ex. 84-196) studied all male workers employed sometime between 1948 and 1977 at this facility. Vital status and information on control variables were determined for 655 men. Cause-specific mortality rates were compared to U.S. white males, adjusted for age and calendar period. Non-significant excesses of mortality from lung cancer and non-malignant respiratory disease were observed in the cohort; these excesses were attributed to a "smoking effect," rather than to an effect from occupational exposure (Ex. 84-196).

As a further analysis, Stille and Tabershaw (Ex. 84-196) separately analyzed the mortality experience of cohort members with a history of any prior work experience and cohort members with no prior work experience. Among the subcohort of 540 males with prior work experience, significant elevatious were found for mortality from all cancers, liver cancer, lung cancer, lymphopietic cancer, and non-malignant respiratory diseases. No elevated causes of death were found for the subcohort of 115 males with no prior work experience. Stille and Tabershaw concluded that "Since the cancers and lung diseases typically have long latencies, the possibility exists that exposures prior to work at the . . . study mine and mill were responsible for at least some of . . . [the increased incidences of diseases observed]" Ex. 84-196, p. 482). They also concluded that "workers with 'exclusive' . . . . [study mine and mill] employment seem to be at no considerable risk of . . . lung cancer. . . ." (Ex. 84-196, p. 483).

OSHA also presented comments by Brown et al. on the Stille and Tabershaw study (48 FR 51119). To summarize, Brown et al. (Ex. 84-218, pp. 178-179) commented that Stille and Tabershaw failed to analyze mortality by length of followup period. The analysis of subcohort with or without prior work history was "not likely to be very informative," because of the small size and young age of the cohort that had no prior work history. Because of these and other concerns about the Stille and Tabershaw study, Brown et al. concluded as follows:

"[the Stille and Tabershaw] report fails to address adequately the question of whether or not there is an inceased risk from lung cancer specifically associated with working at . . . [this particular facility]. In fact, at this time, it is not possible to answer this question based on epidemiologic data alone, because the population available for study is small, the follow up period is relatively short . . ., data on smoking are lacking, and previous exposures in other neighboring talc mines and mills represents a confounding factor" (Ex. 84-218, p. 179).

Tabershaw and Thompson (Ex. 84-219, pp. 179-180) responded to the criticism of Brown et al., and disagreed with NIOSH's conclusion that the talc from the facility contained asbestiform minerals. They cited other studies in which analysis of talc from the facility failed to find any asbestiform fibers, and took exception to NIOSH claiming that asbestos was present based on only 10 atmospheric samples taken during the grinding of a single ore sample. In addition, Tabershaw and Thompson pointed out that, of the nine individuals reported by NIOSH to have died from lung cancer, 4 were employed for less than one year at the facility making it doubtful that exposure to talc at the facility was the likely cause of lung cancer mortality for those 4 workers.

As part of their post-hearing submission, Organization Resources Counselors, Inc., submitted a publication by the R.T. Vanderbilt Company, in which Dr. Selikoff offered opinion on these epidemiology studies. In this publication, Dr. Selikoff is quoted as follows:

"[Vanderbilt] . . . employees in many cases had worked in other New York State mines. Therefore, in the analysis of studies, a question could be raised whether . . . sufficient latency had existed . . . to determine that people who worked only with Vanderbilt talc has excessive cancers. The data can be looked at in various ways. It does create a problem because the ones with the longest latency were also the one who had worked in other mines and mills by definition. . . . I wish we had enough Vanderbilt workers who had begun work 50 years ago, to be able to tell us what happens ultimately to people who inhale Vanderbilt talc. There simply aren't enough such people, if there are any." (Ex. 123-A)

OSHA agrees with Dr. Selikoff's assessment that the epidemiological data are inconclusive with respect to the asbestos-related risk associated with exposure to talc at the Vanderbilt facility. Although the NIOSH studies (Exs. 84-029, 84-181) are suggestive of an increased risk from lung cancer mortality and non-malignant respiratory morbidity among workers at this facility, they are not definitive because of the confounding factor or prior exposure to talc at other facilities. In addition, OSHA agrees with Tabershaw and Thompson (Ex 84-218) that the inclusion in the cohort of workers with less than one year of work experience at the facility

further complicates the analysis. On the other hand, OSHA does not believe that the study by Stille and Tabershaw (Ex. 84-196) indicates a lack of carcinogenic risk among workers at the facility; their analysis of the subcohort with no other prior work experience is inconclusive because of the small size of the cohort and the lack of an adequate follow-up period. An assessment of the implications of these studies is further complicated by the controversy regarding the presence of asbestiform minerals at this facility. OSHA therefore does not find that these studies shed new light on the issue of the carcinogenic or fibrogenic potential of the various forms of tremolite or anthophyllite.

In addition to the epidemology studies discussed above, OSHA described an animal study conducted by Smith et al. (Ex. 84-194) in which the authors administered intrapleural injections of four different tremolitic substances into hamsters. The ore samples tested included fibrous tremolitic talc from New York, tremolite prepared from talc ore at the facility studied by NIOSH, tremolite prepared from Western U.S. talc deposits, and asbestiform tremolite. Tumors and pleural fibrosis were observed only in animals injected with tremolite from western talc or asbestiform tremolite. Smith et al. suggested that the tremolite sample from the facility studied by NIOSH yielded negative results because of the generally short length of the fibers, despite its high tremolite content. They also suggested that the fibrous tremolite sample from New York failed to elicit a carcinogenic response because of the low content of fibrous talc (tremolite constituted only 35 percent of the sample by weight; in addition, only 25 percent of the tremolite was in fibrous form). Smith et al. concluded as follows:

Since [the two samples that yielded positive results] . . . contain at least 5% of material other than tremolite, we cannot be sure that their activity is due wholly, or even in, part, to tremolite. If we assume that their activity is due to tremolite, then the experiments indicate that appropriately high doses of long, thin particles of tremolite induced tumors, whereas high doses of shorter particles did not. This would, of course, be consistent with previous findings by ourselves and others with other materials, such as chrysotile and glass fibers. (Ex. 84-194, p. 338).

In a post-hearing submission (Ex. 306-A), R.T. Vanderbilt Company submitted two additional studies by Smith that contain the same results report by Smith et al. (Ex. 84-194) for the tremolite from New York talc, this submission also contained a report by McConnell et al. (1983) in which F-344 rats were given a diet consisting of one percent tremolite obtained from Vanderbilt's Gouverneur mine. The tremolite had no effect on survival or tumor development compared to that of control rats. OHSA does not find this study noteworthy since, as discussed earlier in this section, several feeding studies of asbestiform minerals known to be carcinogenic by other routes of exposure have failed to show carcinogenic activity by the oral route.

The evidence presented by the R.T. Vanderbilt Company (Exs. 123-A, 306-A), namely the epidemiology study by Stille and Tabershaw and the animal studies conducted by Smith et al. (Exs. 84-194, 306A), would suggest that there was no evidence for asbestos-related disease at their facility, which they maintain contains no asbestiform fiber. Based on these data, and other evidence submitted on the mineralogy of asbestos (Exs. 123-A, 228, 229-A), they have urged OSHA to revise its definition of asbestos to exclude non-asbestiform fibrous tremolite and anthophyllite. As discussed earlier in this section, the finding of asbestos-related disease and the existence of asbestiform minerals at the Vanderbilt site were highly controversial issues during this rulemaking and, as suggested by Dr. Selikoff, cannot be completely resolved at this time. OSHA therefore finds that there is insufficient evidence upon which to state with any degree of certainty that exposure to some forms of fibrous tremolite or anthophyllite is safe. For this and other reasons discussed in Section X of this Preamble (Summary and Explanation), OSHA has not revised its definition of asbestos to exclude certain fibrous forms of these minerals. The Agency believes that this decision comports with prudent public health policy.

V. Quantitative Risk Assessment

*Introduction*

OSHA's determination that currently exposed workers face a significant risk of asbestos-related disease is primarily based on the results of the quantitative risk assessment performed by the Agency, as discussed in the November proposal [48 FR 51122]. OSHA has critically evaluated the scientific evidence concerning the health risk from asbestos exposure. OSHA, as well as other scientific groups, believes that asbestos exposure causes lung disease, respiratory cancer, mesothelioma, and gastrointestinal cancer. OSHA has also examined evidence that indicates that excess disease risk has been observed at cumulative exposures at or below those permitted by the existing OSHA 8-hour permissible exposure limit of 2 f/cc. In addition, OSHA has made risk estimates of the excess mortality from lung cancer, mesothelioma, gastrointestinal cancer, and the incidence of asbestosis using mathematical models that describe the data observed in epidemiologic studies conducted in various industrial populations.

In many cases, the elevated risks seen in worker populations reflect past exposures that were higher than those permitted today.OSHA's quantitative risk assessment entails using the directly observed risks from these past exposures to estimate risk at lower exposure levels. OSHA believes this is a scientifically appropriate and valid procedure. In some instances, OSHA estimated risks using studies which actually observed risks at or below cumulative exposures permitted by the existing standard. The range of studies used by OSHA covers many different work situations and exposure levels. Where possible, OSHA has quantified the ranges of uncertainties in the estimates. These numerical estimates, as well as those risks observed at low exposures, were evaluated to determine the significance of the risk and to determine whether the new standards will lead to a substantial reduction in risk.

OSHA's critical evaluation of all relevant animal and epidemiological studies resulted in the selection of eight studies that contain good data for the calculation of the dose-response relationship for lung cancer for this **final rule** [Selikoff et al., 1979, Ex. 84-90; Seidman, 1984, Ex. 261-A; Henderson and Enterline, 1979, Ex. 84-48; Weill et al., 1979, Ex. 84-206; Finkelstein, 1983, Ex. 84-240; Peto, 1980, Ex. 84-169; Dement et al., 1982, Ex. 84-35; Berry and Newhouse, 1983, Ex. 84-21] and six for mesothelioma [Selikoff et al., 1979, Ex. 84-90; Seidman et al., 1984, Ex 261-A; Finkelstein, 1983, Ex. 84-240; Peto, 1980, Ex. 84-169; Weill et al., 1979, Ex. 84-206; and Dement et al., 1982, Ex. 84-35]. In general, studies of human cohorts in the workplace should provide a better basis for quantitative risk assessment than studies of experimental animals because of the similarities in the populations at risk and the populations from which the risk estimates are derived. As Dr. Hans Weill, testifying on behalf of OSHA, noted:

The greatest public confidence in decision-making to reduce an environmental or occupational risk results when the data used are the product of well designed and conducted studies of relevant human populations. . . . When an occupational hazard has been identified, useful epidemiologic study results will determine the quantitative relationship between the dose of exposure to the causative agent and the risk of the adverse health response in the exposed population. The product is the exposure-response relationship, which together with a valid estimate of the size of the exposed population, the extent of that exposure and accurate indicators of the disease outcome, give characterization of the risk [Ex. 99, p. 8].

The potency coefficients for lung cancer and mesothelioma (K[L] and K[M], respectively) used to define the dose-response relationship were calculated for each study so that cancer mortality was estimated for various exposure levels and exposure durations. A number of well-conducted and high quality epidemiologic studies were available that contained sufficient information on which to base a quantitative risk assessment. Some of these studies did not contain exposure data, but could be coupled with exposure information from other sources in order to obtain an estimate of K[L] and K[M].

OSHA chose not to use animal studies to predict quantitative estimates of risk from asbestos exposure because of the many high quality human studies available that were conducted in actual workplace situations. As is often the case with animal studies, laboratory conditions may not precisely parallel actual worksite exposures.

In the case of asbestos, for example, is it not clear in all instances whether laboratory animals have been exposed to fiber size distributions similar to those found in workplaces. In addition, asbestos appears to multiply the underlying lung cancer risk of smoking and nonsmoking workers; laboratory animals generally do not have any underlying risk of lung cancer. Instead of relying on the animal studies to estimate risk, OSHA has supplemented the human data with results from animal studies when evaluating the health information and determining the significance of the risk; OSHA believes that the animal studies can provide valuable qualitative information on asbestos-related disease. For example, the animal studies show that all commercial asbestos types can cause cancer and pulmonary fibrosis. Animal studies also indicate that longer, thinner fibers may have breater carcinogenic potency than short, coarse fibers.

The paragraphs below provide a synopsis of OSHA's quantitative risk estimates derived from mathematical models and a discussion of the comments and testimony submitted regarding the quantitative assessment of risk for asbestos. OSHA's proposed estimates of risk may be found in Ex. 84-392, the emergency temporary standard ["the November proposal", 48 FR 51086], and in the April proposal [49 FR 14116].

I. Estimates of Risk for Lung Cancer

A. *The Model.* As discussed in the November proposal, OSHA chose a linear model to describe the relationship between the excess relative risk of lung cancer and asbestos exposure (dose). Relative risk is defined as the ratio of the mortality rate of exposed persons to the mortality rate of equivalent non-exposed persons. Relative risk is frequently approximated by the standardized mortality ratio (SMR), which is the observed number of deaths in the exposed population divided by the number of deaths that would be expected in the exposed population. The number of expected deaths is usually derived from the specific age, sex, and calendar year mortality rates in the comparison population.

Asbestos exposure is generally measured in terms of total or cumulative dose. Total dose, also referred to as cumulative exposure or cumulative dose, is a measure of the amount of asbestos inhaled; it is the product of the duration of exposure (in years [y]) and the intensity of exposure (which is workplace air concentration in millions of particles per cubic foot [mppcf] or fibers per cubic centimeter [f/cc]). Under this definition of exposure, a person exposed to airborne asbestos at 2 f/cc for 20 years (40 fiber-years/cc [f-y/cc]) has the same total dose as a person who is exposed to asbestos at 4 f/cc for 10 years (40 f-y/cc).

The relative risk model used by OSHA in assessing the risk of developing lung cancer from asbestos exposure is described by the following equation:

(Eq. 1)

R[L]=R[E][1+(K[L] X f X d[t-10])]

where R[L] is the lung cancer mortality resulting from the asbestos exposure, R[E] is the expected mortality in the absence of exposure, f is the intensity of exposure in fibers/cc, d is the duration of exposure in years, t is the time from the onset of asbestos exposure in years (minus 10 years 10 allow for a minimum latent period) and K [L] is the proportionality constant that is a measure of the carcinogenic potency of the asbestos exposure (slope of the dose-response curve).

The equation can be rewritten as

(Eq. 2)

R[L]/R[E])-1=K[L] X f d[t-10]

showing, on the left-hand side, the excess relative risk (excess SMR) as a function of K[L] and total dose (fibers times years). It is this form of the equation that is used to derive the individual K[L]'s for each of the eight studies. These eight K[L]'s are used to derive one overall K[L] for lung cancer. Then the excess risk is computed for each five-year age interval; the overall lung cancer risk is then computed as the sum of the risks in each of the five-year intervals from age 25 to age 70. The excess risk is expressed as the number of additional lung cancer deaths per 1000 workers exposed for a specific time period.

Evidence of the linear dose-response relationship for lung cancer is found in several well-conducted epidemiologic studies that examined lung cancer mortality in relation to cumulative asbestos exposure in the workplace (for example, Henderson and Enterline, 1979, Ex. 84-48; Liddell et al., 1977 Ex. 84-59, and Dement et al., 1982, Ex. 84-35). In the three studies cited above, workplace asbestos air concentrations were available from measurements made in the worksite studied. Although the studies differ in the magnitude of the risk found (discussed later in this section), all three demonstrate a linear relationship over the entire range of observation.

As stated in the November proposal, other scientific and scientific groups who have attempted to estimate risk from asbestos exposure have used the linear model for lung cancer [Crump, Ex. 85-22, British Advisory Committee on Asbestos. Ex. 84-216, Acheson and Gardner, Ex. 84-243, Selikoff, Ex. 82-2, EPA, Ex. 84-180, CHAP, Ex. 84-256, National Research Council/National Academy of Sciences, Ex. 321]. The model is generally accepted and OSHA believes use of the linear model for predicting lung cancer due to asbestos exposure is reasonable and well-supported. Although participants in the rulemaking pointed to the uncertainty associated with the use of the linear model, no one suggested another model for computing the lung cancer risks.

Dr. Hans Weill elaborated on this point:

* * * As regards the shape of the dose-response slope, and operational judgment is based on the conclusion that there is currently no available evidence that convincingly proves that the slope is not linear, crossing the [excess] risk axis at the origin. This assumption (as made in the OSHA risk analysis) is justified from the observations at moderate and high levels of exposure that generally indicate linearity, which when extended downward to levels of exposure below which observations are available, are not inconsistent with linear low dose extrapolation [Ex. 99, p. 13].

And, in his testimony, Dr. Weill concluded:

Now, as far as the shape of the curve for the important malignant consequences of asbestos exposure, I think we are all in agreement so far today, that the evidence does not permit us, nor does concern of public health or prudence permit us for the conditions that we are concerned about, to develop on any basis other than linearity of exposure and response in a no threshold model [Tr. 6/19, p. 154].

Dr. William Nicholson of the Mount Sinai Environmental Sciences Laboratory elaborated on the rationale for the choice of the linear model for lung cancer:

In three studies in which it [the linear dose-response curve] has been demonstrated [see above Exs. 84-48, 84-59, and 84-35] the range of exposures is large, over a tenfold range of exposures, that linearity has been documented over a tenfold range of dose. Further, it has biologic plausibility [Tr. 6/19, p. 75].

This biologic plausibility was also discussed by Dr. Kenny Crump, testifying on behalf of the AIA/NA:

There is a theoretical argument (Crump *et al.,* 1976) that suggests that cancer incidence should vary approximately linearly with dose for low doses particularly when there is an appreciable background of

carcinogenesis in unexposed populations. . . .If asbestos induces cancer through the same mechanism as smoking, then there is reason to believe that the response should be approximately linear at low dose . . . just as assumed in the OSHA model [Ex. 237A, pp. 8, 25].

Though Dr. Crump noted in his testimony that the linear model for lung cancer "is a hypothesis which is by no means proven" [Tr. 7/9, p. 90], he stated during cross-examination that "all of the estimates I have made in the testimony were based upon a linear model for lung cancer" and that the linear model for asbestos and lung cancer "has been widely used" [Tr. 7/9, p. 116].

Thus, OSHA feels confident in its adoption of a linear model to predict the risk of lung cancer from asbestos exposure. The model has wide support because of its scientific plausibility and reasonableness and its prudence for use in public health decision-making.

B. *Data Used in the Calculation of Individual k*[L]'s. In the November proposal [48 FR 51125], an estimate of lung cancer potency (K[L]) was calculated for each of 11 studies using equation 1. For studies with individual exposure data, K[L] was the slope of the regression equation fit to these points; for studies having only an overall risk estimate and average estimate of exposure, this single point was used in the calculation of K[L]. For each study, the best estimate of K[L] is indicated along with a range of uncertainty. The ranges given are the result of uncertainties in estimates of exposure, methodological uncertainties that led to alternate evaluations of risk or exposure, or, in some cases, statistical uncertainties associated with the use of small numbers.

The differences in the K[L]'s among the various studies result from a number of different factors. There do appear to be actual differences in risk depending upon the nature of the asbestos exposure.One potential explanation is that workplaces differ with regard to fiber size distribution (long finer fibers appear to have greater carcinogenic potential than coarse fibers). For example, as several participants in the rulemaking acknowledged, there appears to be a distinct difference in the risk from mining and milling and other processes. As Dr. Nicholson summarized:

I think I stated this morning . . . the possibility that the mining work environment may demonstrate a different pre-unit risk. That is, there's three studies showing somewhat lower risks. At least two of them show, with fairly substantial data, lower risk, that the [lower risk] may be a function of the fiber size distribution in the mining environment.

One may have a much greater number percentage, of long curly fibers, which are readily counted, but are not inspired. And, thus, the fiber counts are proportionately high in that environment relative to the amount of asbestos inspired. If seems to be consistently so for chrysotile and also for amosite. For example, one finds very few cases of mesothelioma associated with amosite mining but a considerable number associated with amosite manufacturing.

And so there is perhaps a difference in the mining environment, where they are working with different type of fiber composition [Tr. 6/19, p. 127].

Thus, where airborne fibers are relatively coarse, the K[L]'s are lower than the K[L] values found in studies of textile operations where fibers are fine.

Differences may also be explained by variations in study design and other factors influencing the ability to define the dose-response relationships. One of these is the limited knowledge of past fiber exposures of those populations whose mortality was later evaluated. Prior to 1970, few measurements were made in facilities using asbestos fibers. Further, those measurements that were done usually quantified all dust present in the workplace air and not just fibers. Current techniques, which involve use of membrane filters and phase contrast

microscopy for the counting of fibers longer than five micrometers, have been utilized in Great Britain and the United States only since 1964 [Ayer et al., 1965, Ex. 84-253] and have been standardized in the United States only since 1972 [Leidel], 1979, Ex. 84-62] and even later in Great Britain. In any case, sampling has occurred only in a few of the worksites studied, and then only occasionally. In addition, variability in work activities and in sampling circumstances add considerable uncertainty to knowledge of dose.

Some of the epidemiologic studies, including those by Dement et al. [Ex. 84-35], Liddell et al. [Ex. 84-59] and Henderson and Enterline [Ex. 84-48], include measured air concentrations at the exposure site and used job histories of the study population to estimate exposure. In these cases the dose-response curve was calculated by estimating total asbestos exposure (in mppcf-years or in fiber-years/cc) according to the time that an individual spent at a job with a measured exposure. A conversion factor for converting from mppcf to f/cc was employed on a study-by-study basis, depending on the data available. Other epidemiological studies, for example those by Selikoff et al. [Ex. 84-90] and Seidman et al. [Ex. 84-87], did not have direct industrial hygiene measurements for the studied worker population. For these studies, exposure estimates were derived from industrial hygiene surveys of similar work operations and processes for which industrial hygiene data were available.

OSHA has evaluated these differences and has dealt with their implications on a study-by-study basis. Uncertainties associated with these measurements constitute much of the range of variability surrounding the K [L]'s. Taken as a whole, the asbestos studies contain data of unusually high quality, which has enabled OSHA to make the risk estimates with a high degree of confidence.

There was considerable discussion during the rulemaking about the individual K[L]'s for many of the studies that went into the estimation of the overall lung cancer risk, particularly the inclusion/exclusion of several of the studies in this calculation. The discussion below deals first with the comments on and adjustments to individual K[L]'s and then discusses the impact of their inclusion in the overall estimate of lung cancer risk.

The Selikoff et al. and Seidman et al. Studies. Several participants in the hearing criticized OSHA for including the results from the Selikoff et al., 1979 [Ex. 84-87] and Seidman et al., 1979 [Ex. 84-90] studies in the calculation of K[L]. The major objection to the use of these studies was the lack of concurrent exposure information on the cohorts. For example, Dr. Crump noted that:

The CPSC (1983) Panel placed these two studies in a separate category because of the weakness of the exposure estimates. The Seidman *et al.* study also involved brief exposures (less than four years) exclusively, which makes it less suitable than other studies for estimating the effect of long term exposures [Ex. 237A, p. 26].

Dr. Weill also expressed reservations about including the Selikoff et al. and Seidman et al. studies in the overall estimation of risk [Tr. 6/19, p. 184].

Though it is true that CHAP did characterize the Selikoff et al. and Seidman et al. studies as having "Level 2 exposure data" (no job histories or industrial hygiene measurements available for the cohort, exposure estimate made from best available sources), CHAP still computed K[L] for these two studies with the information available. And, during cross-examination, Dr. Nicholson, a member of CHAP, indicated that CHAP did not weigh the K[L] values from these two studies differently from those in other studies when deriving estimates of the final potency [Tr. 6/19, p. 148]. Dr. Weill emphatically stated that inclusion of the studies in the risk analysis was "not a fatal flaw [Tr. 6/19, p. 184].

OSHA offered a full description of the exposure data used in these two studies in Exhibit 84-392. Since that time, however, new and more complete information on exposures for the Seidman et al. cohort have come to light which strengthen the case for including the results of the K[L] calculation in the overall estimates of risk.

This new information is discussed below.

Although no new evidence has been brought forward on the Selikoff et al. study of insulation workers, OSHA still believes it is appropriate to include the K[L] from this study in determining the overall level of risk. It is the largest of all the studies (17,800 workers) and also reports the largest number of lung cancer deaths (652) and deaths from mesothelioma (180). Excluding this study would mean exlcuding 45% of all the asbestos-related lung cancer deaths and 84% of all the mesothelioma deaths from the overall analysis. OSHA believes it would be a serious error to eliminate such a large portion of the available data, when appropriate estimates of the exposure levels of these workers are available.

OSHA calculated the K[L] from the Selikoff et al. data based on average values (for duration of exposure, level of exposure and time since onset of exposure) derived from several sources. Although the use of average data and overall (average) levels of risk may not be as desirable as risks broken down by cumulative exposure, nevertheless, the estimates of K[L] from these data are nevertheless valid and reasonable. OSHA predicted a K [L] of 0.02 for the cohort, with an uncertainly band of (0.008 to 0.30). The value 0.02 is only twice the best estimate of an overall K[L] of 0.01 and falls well within the range of overall uncertainlty given for the overall K[L], that is, 0.003 to 0.03. Thus, OSHA has not adjusted the original value of K[L] computed for this cohort.

The Seidman et al Update. During the course of the hearing, the testimony of several witnesses strengthened OSHA's confidence in using results from the Seidman et al. study of 820 insulation manufacturing workers. As discussed in Exhibit 84-392, while no data exist on air concentrations at the time the Paterson factory operated, data do exist on air concentrations in two plants that manufactured the same products with similar fiber and machinery. One of these plants, in Tyler, Texas, opened in 1954 and operated until 1971. The other, in Port Allegheny, Pennsylvania, opened in 1964 and closed in 1972. Similar efforts to control dust in these newer plants were apparently made as were made in the Paterson, New Jersey plant. During 1967, 1970, and 1971, asbestos fiber concentrations in these plants were measured by the U.S. Public Health Service and were published by NIOSH [Ex. 2-12].

Participants in the rulemaking criticized the assumption that these exposure data were representative of the exposure conditions in the Paterson plant. Dr. Crump expressed his concern over the use of these data. He stated:

OSHA thus derived exposure estimates from measurements made 21 to 31 years later in the other plants in Texas and Pennsylvania. The reasonableness of these estimates is open to question. It is certainly plausible that the exposure measurements in these plants made after the dangers associated with asbestos became known were less, and perhaps far less than exposures experienced 21-31 years earlier under wartime conditions [Ex. 237A, p. 13].

Dr. Morton Corn, former Assistant Secretary for OSHA, who appeared at OSHA's hearing on behalf of the Building and Construction Trades Department was hired by the companies who owned the plants to recommend and install control measures in the two plants in the late 1960's.At the hearings he was asked to comment on the reasonableness of using data from Tyler, Texas and Port Allegheny to estimate exposures in the Paterson plant. Dr. Corn responded:

I think the procedure is precisely what we're trying to do in dustrial bygiene. And I would endorse trying to link similar plants where no measurements were available to other plants where measurements are available. There's no question about that.

I would classify Tyler as one of the most contaminated asbestos facilities I've ever been in. I think Tyler would be the high estimator. Port, I would consider typical of asbestos processing that I saw in those years. But Tyler was clearly a very bad facility. . . . So I don't know if averaging them, averaging might put you on the high side

if you have measurements for both. I would pit you towards Tyler. . . . Tyler was a fairly startling facility [Tr. 7/3, p. 67].

Hence, given Dr. Corn's characterization of conditions in the two plants, to the extent the OSHA used data from the Tyler plant, the estimates of exposure would be *overestimated,* which would result in an *underestimate* of the potency factor, K[L].

Since the time of the OSHA proposals, the Seidman et al. study has been updated to include longer followup and an expansion of the findings in terms of the jobs of the workers and estimates of the fiber exposure accumulated by the workers during their work at the amosite asbestos factory. The updated study was presented at the hearings as Exhibit 261-A. The study extended the observation period through December 31, 1982, with a total of 593 deaths. Using the data from the Tyler Texas and Port Allegheny plants, Seidman and colleagues attempted to "assign plausible estimates of the exposures likely to have been associated with particular jobs in the Paterson plant" [Ex. 261-A, p. 6]. Seidman described the process as follows:

With the aid of the expertise of Dr. William Nicholson, I've gone back to the records that were accumulated on the Paterson workers, and in conjunction with fiber counts that were available for 1967 from Port Allegheny Plant and for 1967, 1970, and 1971 for the Tyler, Texas plant, *the same kind of fiber was used, the same kind of equipment was used, the same processes ware used to make the same kinds of products,* we arrived at approximate -- we estimated -- looking at what the men themselves reported as to relative levels of dustiness in the jobs they worked at. We established levels of dustiness, dust index which at first was all I thought we could work with and I realized we had specific jobs that we could even modify this with, we assigned fiber counts per cc and then were able to then, with the aid of our historical data, to make an assignment which we applied to out Paterson plant. Then with the aid of the time that the men worked, we arrived at the total work time they worked at the plant, a total work experience dosage in terms of fibers [Tr. 7/12, p. 289, emphasis added].

As Mr. Seidman pointed out, when using the estimates of Tyler and Port Allegheny to determine exposures at the New Jersey plant, the estimates.

* * * may be somewhat on the high side to the extent that industrial hygienists tend to over-sample the dustiler areas of factories. Also, there was a concerted effort to have the Paterson plant workers use respirator protectors which presumably might have reduced the exposure from inspired air while the protectors were being used. . . . It is important to realize that any overestimation there may be in the fiber counts we have assigned, will serve to underestimate the dose-response relationships associated with asbestos exposure at the Paterson plant [Ex. 216-A, p. 6].

Table 5 of Ex. 261-A shows the estimated exposures for over 30 job categories. During cross-examination, Mr. Seidman further explained:

Table 5 comes from two sources, one is internal and one is external. Internally, we had for about 40 percent of the men, a statement as to the dustiness of their job. We had -- they said what their job was and how dusty it was[:] very dusty, somewhat dusty, or not dusty at all. . . . We had, for a number of jobs, what the counts -- fiber counts -- were for the jobs which, as I say, using the same kind of equipment, and same fiber and same kind of product, were in these plants of the same company. These were the general levels used to assign the jobs at UNARCO [Paterson, N.J.] and then modified them slightly depending on what the internal statement as to dustiness was [Tr. 7/12, p. 298-299].

Dr. Nicholson explained further:

The exposure-response data were generated by assigning each individual in the Paterson plant an exposure as calculated above for the period of time he would have been employed in a job with that given title. The total

exposure in fiber-years/ml for each individual was then calculated summing over all jobs that the individual worked in [Ex. 303].

Table 1 gives cumulative observed and expected deaths for the workers in an amosite factory categorized by estimated fiber-year exposure. As noted in Ex. 84-392, it was believed that the average exposure for this population was approximately 35 f/ml, and this was the value used to calculate the original value of K[L] for this cohort. However, in this updated analysis the average exposure was discovered to be closer to 50 f/ml [Tr. 7/12, p. 291]. Mr. Seidman indicated that the high number resulted when the estimates of fiber counts were "weighted by the kinds of jobs that the Paterson people had, [and] the number of people working at the jobs they had in the Paterson plant" [Tr. 7/12, p. 294]. Seidman went on to testify that "If you look at the historic data, there are ranges which go higher, but not on the averages. There are ranges, there are samples that go into the 200's" [Tr. 7/12, p. 295]. He noted, however, that the estimate of 50 f/cc "seems pretty reasonable and plausible to me" [Tr. 7/12, p. 295].

As was pointed out by Mr. Hardy, representing the AIA/NA, during cross-examination, the dose-response curve appears to cross the y-axis at a level above zero. However, Mr. Seidman was clear that possible underestimation errors in the measurements could not account for such differences. He commented --

To move them [the risk points at each dose level] far enough over so that the point on the straight line from this kind of material is going to come to zero [excess risk] on a straight line fit, they'd have such a cloud of dust, they wouldn't see each other at the next bench. . . . People couldn't work in such [conditions] -- even the people who need a job desperately couldn't work in such an atmosphere [Tr. 7/12, p. 308].

**TABLE 1. -- CUMULATIVE OBSERVED AND EXPECTED DEATHS IN AN AMOSITE ASBESTOS FACTORY, 1941-45, BY ESTIMATED FIBER EXPOSURE -- SEIDMAN. 1984 n1**

| | | Lung cancer | | |
|---|---|---|---|---|
| Cumulative exposure f-y/ml | Midpoint | Observed | Expected | SMR |
| <6 | (3.0) | 14 | 5.31 | n2 264 |
| 6.0 to 11.9 | (9.0) | 12 | 2.89 | n3 415 |
| 12.0 to 22.9 | (18.5) | 15 | 3.39 | n2 442 |
| 25.0 to 49.9 | (37.5) | 12 | 2.78 | n2 432 |
| 50.0 to 99.9 | (75.0) | 17 | 2.38 | n2 714 |
| 100.0 to 149.9 | (125.0) | 9 | 1.49 | n2 604 |
| 150 to 249.9 | (200.0) | 12 | 1.32 | n2 909 |
| 250 plus | (250.0) | 11 | 0.94 | 1,170 |
| Total | | 102 | 20.51 | 49 |

n1 From Table 7, Seidman, 1984, Ex. 261-A.

n2 p<.001.

n3 p<.01.

In its original evaluation of this study, OSHA used overall averages (SMR=4.46, 35 f/cc, 1.46 years) to compute the K[L] [0.068=(4.46-1)/(35X1.46)]. Substituting the overall values from the updated study gives a

slightly smaller value of K[L] [0.054=(4.97-1)/(50X1.46)]. In addition, the updated and expanded data base now provides enough data to perform a dose-response regression for the lung cancer data. The data are found in Table 1. As with other data sets, it may be speculated that there is greater uncertainty in the estimates at lower doses. This may be adjusted for by forcing the curve through the origin. Regressing excess SMR on the midpoints of dose gives an estimate of K[L] of 0.045. Although this value of K[L] is somewhat lower than the originally predicted value of 0.068, OSHA has greater confidence in it as an accurate predictor of the asbestos potency in this production population.

The Henderson and Enterline study. OSHA calculated the value of K[L] based on the mortality experience of 1075 retirees from an asbestos products manufacturing plant [Ex. 84-48] by computing the slope of the dose-response relationship from the linear regression (K[L]=0.0066). Henderson and Enterline had presented exposure data in terms of total dust measured in millions of particles per cubic foot, and hence a factor was needed to convert from particles to fiber count. OSHA employed the value 1.4 f/ml/mppcf, based on the work of Hammad in cement plants, which gives a best estimate of K[L] of 0.0047.

Crump has pointed to what he believes to be "considerable uncertainty in the methods used by OSHA to convert from particles to fibers" [Ex. 237A, p. 14]. Citing the CHAP [Ex. 84-256], he recommends that a conversion factor of 2 should have been employed, giving a K[L] of 0.0033. He also notes that "Enterline himself employed a conversion factor of 3.0 (Enterline 1981) [Ex. 84-127]" [Ex. 237A, p. 15]. However, when Dr. Enterline testified before the Ontario Royal Commission in June of 1981, he expressed considerable doubt about the conversion factor of 3, noting "I don't know how anybody comes up with a number like that anyhow" [Ex. 85-2, p. 53]. Enterline also noted that the conversion factor depended on the operation and that "I think, in asbestos cement, maybe that's [3's] the wrong number" [Ex. 85-2, p. 53]. In addition, in the same footnote [Ex. 84-127] cited by Dr. Crump, Dr. Enterline noted that the British Advisory Committee on Asbestos used conversion factors of 1, 2, and 5 f/cc/mppcf and that "the most conservative estimate of response at low doses in terms of protecting the public would result from assuming a low conversion factor" [p. 42]. Whereas CHAP employed a slightly higher conversion factor, it also noted that --

* * * since follow-up of this group began at age 65, it is essentially a study of a survivor population and as such may have underestimated the maximum relative risk actually experienced by the entire cohort. If this peak relative risk provides the best basis for predicting the long-term experience of individuals exposed at lower levels, then the fitted slope should be increased perhaps by a factor of 2.0 [Ex. 84-256, II-102].

CHAP made such an adjustment in its estimate of the slope to account for these biases (Ex. 84-256, II-100]. Therefore, given the fact that CHAP recommends a value of K[L] considerably higher than that put forth by OSHA in the November and April proposals and since Dr. Crump has suggested a value somewhat lower, OSHA believes that its estimate of 0.0047 for K[L] represents a reasonable median estimate of the potency factor for lung cancer in this study population. As noted in Ex. 84-392, however, "A study of a retiree cohort with these characteristics would understate mortality by as much as 62% relative to the maximum observable risk" [p. 30]. Thus accounting for this possible understimation, and with regard to the variation in possible conversion factors, the range of uncertainty around this value may extend from 0.0022 to 0.0106.

The Finkelstein Study. Finkelstein established a cohort of 241 production and maintenance employees from records of an Ontario asbestos cement factory. OSHA computed a K[L] for this cohort based on an average cumulative 18-years exposure of 112.5 f-y/ml for the production workers alone. This group had an SMR of 850, based on 17 observed lung cancer deaths versus 2 expected. These data produced a summary K[L] of 0.067 (Ex. 84-392, p. 33]. OSHA noted some uncertainties in this estimate, particularly because the two lowest exposure categories show risk increasing steeply with exposure, whereas the highest exposure category showed a cancer rate lower than that of the lowest exposure group. OSHA speculated in the proposal that this inconsistency may be due to the small number of deaths in each category.

Several participants raised the question of the suitability of using this value of K[L] in the overall estimate of K [L]. In particular, Dr. Crump pointed to the lack of a dose-response relationship for lung cancer in this cohort, quoting the CHAP conclusion that "no sensible dose-response for lung cancer can be inferred from these results" [Ex. 237A, p. 28]. CHAP noted that:

* * * possible explanations for these results are incorrect exposure estimates and/or very high competing risks for the heavily exposed persons [Ex. 84-256, p. II-111].

It should be noted that CHAP included Finkelstein's study among those categorized in the Level 1 Exposure category, that is, having job histories and industrial hygiene measurements made at the relevant exposure site. Using the entire cohort (both production and maintenance workers], CHAP computed on SMR of 606 (20 observed versus 3.3 expected). Noting reservations about the exposure levels, CHAP gave a K[L] of 0.048 of this cohort [(6.06-1)/(105)].

Given the same reservations as expressed by CHAP, OSHA believes 0.048 to be a valid expression of the potency of exposure to asbestos in this population of asbestos-cement workers, and has lowered its original estimate of K[L] to reflect some reservations about the data.

The Dement et al. Study. OSHA calculated a lung cancer potency factor from the study of Dement and his colleagues, who investigated the mortality experience of 768 workers in a chrysotile textile products manufacturing plant. Data from impinger measurements to total dust in terms of mppcf were available since 1930 for exposures in a textile plant using chrysotile [Dement et al., 1982, Ex. 84-35]. Using a factor of 3 to convert from mppcf to f/ml (also used by CHAP), OSHA computed K[L] as the slope of the weighted regression of excess SMR on the midpoint of dust levels in f-y/ml. As noted in the November proposal, this produced a value of K[L] of 0.042. Participants in the hearing argued that this K[L] was overestimated because Dement and his colleagues had overestimated the SMR's by using an inappropriate control group for the calculation of the expecteds. As OSHA explained in its preliminary risk assessment, Dement at al. employed U.S. national death rates rather than local county rates for computing expected values. The authors noted that:

The choice of an appropriate comparison population for mortality analyses is difficult and arguments could be made for using rates for a set of counties contiguous to the county in which the plant was located. However, there are serious limitations to this approach which were considered in this study and resulted in rejecting the use of local county rate. First, the county in which the plant was located is the site of a large shipyard industry with peak employment of approx. 29,000 persons in 1943 (Blot *et al.* 1978). Employees for this industry were largely drawn from the local population. Many of these workers are thought to have been exposed to asbestos during ship construction and repair. In an ecological study Blot *et al.* (1978) demonstrated an association between county lung cancer rates and shipyard employment. In a more refined case-control study, Blot *et al.* (1979) demonstrated a summary odds ratio of 1.6 for shipyard employment and lung cancer after adjusting for smoking, other occupations, age, race, and county of residence. These data suggest that lung cancer death rates in the area in which the plant was located are likely to be elevated by local shipyard employment.

A second factor to be considered in choosing local rates for comparison is the effect that the plant being studied might have had on local lung cancer death rates. Because of a lack of an employment record system prior to about 1930, it is difficult to estimate the exact number of persons ever employed at this plant; however, this is likely to exceed 10,000 prior to 1965. Thus [sic] could have a significant impact of local lung cancer death rates, assuming an overall lung cancer SMR of 200 or more for these workers.

The effects of shipyard and asbestos plant employment make the use of local death rates inappropriate for this study [Ex. 84-35, p. 879-880].

In addition, state (South Carolina) mortality rates from lung cancer were similar to those of the United States.

Moreover, "[A]vailable smoking data for this cohort suggest that the observed lung cancer and nonmalignant mortality excess among white males cannot be explained by cigarette smoking independent of asbestos exposure" [Ex. 84-37, p. 430].

Although Crump pointed to the arguments raised by Acheson and Gardner [Ex. 84-243] that local rates should have been preferred, OSHA found these arguments unconvincing. Crump recommended a K[L] of 0.023, approximately half the value of K[L] calculated by OSHA. Crump noted that:

* * * Not only does this modification provide a better fit to the Dement *et al.* data, the estimated background rate agrees closely with the 75% excess of local lung cancer rates over national rates (See Figure 3 of Acheson and Gardner, 1983 [Ex. 84-243]). The lower estimate of K[L] = 0.023 also reduces the discrepancy between this and other studies which show a much smaller K[L].

OSHA believes that a reduction of the K[l] to 0.023 is inconsistent with the available data; First, Dement et al. noted that:

* * * even if rates for contiguous counties had been used . . . the expected lung cancer rates for white males would have been increased by only approx. 15%, not nearly sufficient for the observed excess lung cancer risk [Ex. 84-35, p. 880].

Moreover, as Dement pointed out in 1982:

* * * rates for contiguous counties for black males were approximately 45 percent below U.S. rates; thus, the overall excess among blacks is underestimated by the present study, although the numbers were small [Ex. 84-229, p. 179].

Thus, to some extent, these overall estimates may be underestimated. Hence, OSHA concludes that its original estimate of K[L] for this study, 0.042, is valid and reasonable, and thus has adopted it for the **final rule.**

C. *Calculation of the Overall K*[L]. OSHA's best estimates of K[L] from the proposed rule, and the final determination of K[L] for each study are given in Table 2, along with a range of uncertainty. The ranges listed are the result of estimates of exposure uncertainties (usually a factor of two), methodological uncertainties that led to alternate evaluations of risk or exposure, or, in some cases, statistical uncertainties associated with small numbers. In addition to some controversy over the individual K[L]'s, there was widespread disagreement as to which studies should ultimately be included in the determination of an overall K[L] for lung cancer.

**TABLE 2. -- ESTIMATES OF K[L] FROM PROPOSED RULE AND FINAL DETERMINATION**

| | Pro-posal | Final | Range |
|---|---|---|---|
| Henderson & Enterline | 0.0047 | 0.0047 | (.0022-0.011) |
| Weill et al | 0.0033 | 0.0033 | (0.0016-0.0086) |
| Finkelstein | 0.067 | 0.048 | (0.033-0.13) |
| Peto | 0.0076 | 0.0076 | (0.0009-0.023) |
| Dement et al | 0.042 | 0.042 | (0.23-0.21) |
| Berry and Newhouse | 0.0006 | 0.0006 | (0-0.0008) |
| Seidman et al | 0.068 | 0.045 | (.023-.06) |
| Selikoff et al | 0.020 | 0.020 | (0.008-0.03) |
| Arithmetic Mean | 0.027 | 0.019 | |
| Geometic Mean | 0.0113 | 0.01 | |
| Median | 0.0138 | 0.0138 | |

In its preliminary assessment, OSHA used the eight non-mining-and-milling studies to derive an overall estimate of K[L] of 0.01. As noted in the November proposal:

Considering the industrial processes other than mining and milling, OSHA believes 0.01 to be a reasonable estimate of K[L]. It is the geometric mean and median of the K[L]'s derived from studies of asbestos manufacturing and insulation application processes. The geometric mean had the advantage of minimizing the influence of outlying values and a K[L] of 0.01 is approximately within one order of magnitude of all the estimates of K[L]. In sum, the K[L] of 0.01 is a best estimate which contains appropriate recognition of studies with, higher and lower values of K[L]. It should be noted however, that the uncertainties around this estimate of K[L] are such that an appropriate estimate of K[L] could lie between 0.003 and 0.03 [48 FR 51125].

The distinct nature of mining-milling data (and hence, the estimate of K[L] from these data) has been considered earlier. There is some evidence that risks in the asbestos mining-milling operations are lower than other industrial operations due to differences in fiber size. This differential was discussed by Nicholson [Ex. 303A]. Thus, in determining the best overall value for K[L] for the **final rule,** the data from mining and milling processes were not considered.

OSHA still believes it to be valid to employ the same eight studies it used to derive the estimates for the November and April proposals. As discussed earlier, OSHA modified some of the values of K[L] for the **final rule.** Based upon these revised values, OSHA has determined that the best estimate of K[L] is 0.01, the same value derived for the proposals. The values given under the final estimate column in Table 2 have an arithmetic mean of 0.019 and a geometric mean of 0.01. OSHA believes it has chosen reasonable estimates for the individual K[L]'s and has been responsive to the comments made by participants in the hearing. In some cases, OSHA has lowered its original value of the estimate of K[L] in light of these comments or the addition of new data indicating such a change was warranted. The end result is that these small changes in individual values have little effect on the overall K[L] value. This is most likely due to the Agency's choice of a reasonable K[L] for the proposal.

Some scientists have suggested that some asbestos processes such as asbestos textile manufacturing, may pose a greater hazard than other processes. As noted earlier, while mining and milling appear to pose a lesser carcinogenic hazard than manufacturing processes, when OSHA compared the potency factors for lung cancer (K[L]) among different studies of different processes, no consistent pattern of differential lung cancer risk by process emerged. Therefore, again, the choice of a midpoint unit risk for all industrial processes is a reasonable and justified choice.

In sum, the K[L] of 0.01 is a best estimate which contains appropriate recognition of studies with higher and lower values of K[L]. It should be noted, however, that the uncertainties around this estimate of K[L] are such that an appropriate estimate of K[L] could lie between 0.003 and 0.03.

As discussed earlier, Crump believed that both the Seidman et al. and Selikoff et al. studies should have been excluded from the calculation of K[L]. Along with the other adjustments discussed above, Crump estimated an overall K[L] of 0.0065. As Dr. Crump noted in his testimony:

OSHA has developed what I would term an upper limit assessment of asbestos risk. In dealing with uncertainty, OSHA has, in a number of instances, made assumptions that tend to minimize the possibility of underestimating the risk. In addition, the uncertainties in some of their assumptions appear to be underestimated by OSHA. The three most significant assumptions in OSHA's risk assessment that lead to upper limit estimates of risk are the assumptions of: (1) a linear dose-response relationship; (2) the same potency for all forms of asbestos; and (3) attribution of the lung cancer component of risk caused by smoking to the overall risk of asbestos [Ex. 237A, p. 4-5].

However, in addition to Dr. Crump's recommendations, several commenters noted a number of different ways for incorporating the available data into an overall estimate of risk. For example, in his written testimony, Dr. Marvin Schneiderman, who served as a member of CHAP and who was one of the reviewers of OSHA's November proposal, suggested several other reasonable methods for producing "medium estimates." In addition to approaches taken by OSHA, Dr. Schneiderman suggested that one look only at the four studies (from the proposal) which also had data on mesothelioma (Selikoff et al., Seidman et al., Peto, and Finkelstein). This selection produced an overall estimate of K[L] derived from the individual K[L] values of approximately 0.028. He also noted the K[L] of 0.020 which results from use of the five U.S. studies only (Selikoff et al., Seidman et al., Henderson and Enterline, Weill et al., and Dement et al. proposed values of K [L], Ex. 116, p. 7).

Schneiderman concluded that:

The selection of the value of 0.01 [by OSHA] is based both on the various averages that could be computed and also on the informal or subjective weights given to each of the studies by OSHA. If this value is in error, it is possibly biased *downward* by the inclusion of the miners and millers and the foreign studies. Howeve, any error introduced by an underestimate of K[L] will be relatively small. Because of the changing patterns of cigarette smoking which should soon lead to reduced lung cancer mortality smong younger (working-age) men, an underestimate of K[L] is likely to compensate for possible overestimate of lung cancer mortality in the future [Ex. 116, p., 7-8].

Other possibilities for the calculation of K[L] include: (1) Using studies with concurrent exposure data only (Henderson and Enterline, McDonald et al., Peto, and Dement et al.), which gives estimates of K[L] of 0.014 (arithmetic mean) or 0.006 (geometric mean); (2) using only the upper limits of the uncertainly ranges, which gives a K[L] of 0.059 (arithmetic mean) or 0.02 (geometric mean).

The value of 0.01 falls well within the range of K[L]'s suggested by participants in the rulemaking.It is less than two times larger than the lowest value suggested for K[L] (by Crump). In addition, as OSHA discussed in the proposal, there is a range of uncertainty associated with this value that more than covers all suggested values of K[L]. Thus, OSHA believes the value of 0.01 to be a valid, reasonable estimate of K[L] and has employed it in developing its estimates of risk to support these revised rules.

II. Estimates of Risk for Mesothelioma

A. *The Model.* For the November proposal, OSHA chose an absolute risk model to predict the risk for mesothelioma from exposure to asbestos. Absolute risk is calculated as observed deaths divided by the number of person-years at risk. It is believed that use of SMR's or relative risk is not appropriate for mesothelioma because the expected number of deaths in a cohort would be close to zero due to the rarity of the disease. The use of absolute risk to predict risk of mesothelioma was not questioned by any participant in the hearing.

In addition to using absolute risk rather that relative risk, this model is different from that used for lung cancer because both duration of time since initial exposure and duration of exposure are determinative or risk. The magnitude of the risk increases linearly with intensity of exposure, whereas the risk increases exponentially with duration of exposure and time from onset of exposure. The rationale for such a model describing mesothelioma risk has been discussed by several authors [Armitage and Doll, 1969, Ex. 84-252; Pike, 1966, Ex. 84-385]. Such a model was utilized by Newhouse and Berry [1976, Ex. 84-342] in predicting mesothelioma mortality among a cohort of factory workers in England. Limited data from three studies are also available on the dose-response relationship for mesothelioma [Seidman et al., 1979, Ex. 84-87; Hobbs et al., 1980, Ex. 132, and Jones et al., 1980, Ex. 84-138].

The model used by OSHA to assess the risk and derive the potency factor for mesothelioma, K[M], is given by the following equations:

AR[M]=fxK[M][(t-10)<3>-(t-10-d)<3>]

for t>=10+d

AR[M]=fxK[M](t-10)<3>

for 10+d>t>=10

AR[M]=0

for 10>t

where AR[M] is the excess mortality from mesothelioma, f is the intensity of exposure in fibers/cc, d is the duration of exposure in years, t is time after first exposure in years, and K[M] is the proportionality constant that is a measure of the mesothelioma carcinogenic potency (slope of the dose-response curve) [Ex. 84-392].

Dr. Marvin Schneiderman discussed several aspects of the choice of this model for assessing mesothelioma risks. In his written testimony he stated:

The formula for estimating mesothelioma risk has a somewhat different form [from that of lung cancer] -- in keeping with the fact that the excess risk from mesothelioma is measures as an "absolute" rather than a "proportional" risk. . . .

What these formulas say is, first, no disease will be seen sooner that 10 years after first exposure (induction period effect). Second, if d is relatively short (compared to t) then there will be less disease than if the duration of exposure is long. Finally, the ageat-first exposure effect is subsumed in the exponent 3.

The Consumer Product Safety Commission, in the report mentioned above [Ex. 84-256], also gives this formula. The NRC/NAS report on asbestiform fibers [Ex. 321] notes the great sensitivity of the estimate to the exponent of the (t-10) [and the (t-10-d)] term. Taking the term (t-10)<3> as a base, if t=40, the relative values of the term raised to different exponents are:

| | | |
|---|---|---|
| NRC/NAS "middle" | :(t-10)<3.2> | 1.97X(t-10)<3> |
| Peto, et al. | :(t-10)<3.5> | 5.48X(t-10)<3> |
| Nicholson | :(t-10)<4> | 30.0X(t-10)<3> |

These values are somewhat different if the "delay" term is neglected [Ex. 116, p. 6-7].

In his written testimony, Dr. Crump raised several issue concerning the choice of this expression for the time factor. He stated:

Most studies of mesothelioma predict that the mortality risks are a power of elapsed time since first exposure, as assumed by the OSHA model. However, we cannot be sure that this steep rate of increase extends indefinitely into old age as assumed by OSHA. In the Selikoff cohort, which contains the best information on mesothelioma mortality on old age, the number of mesotheliomas in the oldest group (55 + years since first exposure) is only about 1/2 the number predicted from the OSHA model. Although some of this shortfall may be due to underreporting in old age, it is also possible that the deficit is real. If so, the OSHA model will overestimate risk at oldest ages. None of the cohorts contain information on mesothelioma risk after 30 years

past termination of exposure. OSHA's assumption that the risk will continue to increase represents an assumption which is not presently verifiable [Ex. 237A, p. 34].

In a post-hearing comment, Dr. Crump extended his argument. In addition to the data from the Selikoff cohort discussed above, Dr. Crump also discussed the mesothelioma data from the recently completed follow-up of the Seidman et al. study of amosite workers. He pointed out that for these data, ". . . the mesothelioma rate did not continue to raise with increasing age from first exposure, but dropped off 35-40 years from first exposure to 1.8/1000 person-years, which is about 1/3 of the rate observed for 30-35 years from first exposure" [Ex. 312a, Vol. I, Tab A, p. 7]. Dr. Crump noted that, although the OSHA model assumes "that the mesothelioma mortality rate increases indefinitely as a power of time from first exposure . . . the multistage model does predict an eventual reduction, the timing of which is determined by the number of stages affected and the rate of elimination of fibers from the body" [Ex. 312a, p. 8]. Dr. Crump went on to conclude that "if the reduction is real, then the OSHA model will provide a considerable overestimate of mesothelioma risk from exposures in early life" [Ex. 312a, p. 8].

In addition, Crump performed a statistical analysis which demonstrated that the use of a delay model (such as the one proposed by OSHA) will always result in higher estimates of mortality rates at older ages than use of a model which does not incorporate a delay. He concluded that "Thus, rather than compensating for the reduction in risk, OSHA's use of a model with a delay exacerbates the tendency to overestimate risk at older ages" [Ex. 312a, p. 9].

As pointed out by Drs. Crump and Schneiderman, most studies of mesothelioma risk demonstrate that mortality risks are a power of elapsed time since first exposure, and this formulation has received widespread support. In general, the selection of a power of 3 is a reasonable choice and has been used by other reputable bodies (e.g. CHAP, Ex. 84-256). As noted by Dr. Schneiderman, the choice of a power of 3 will tend to give lower estimates of risk other choices of exponents which are also consistent with the available data. In addition, while Crump raised some doubts about the use of a "delay" model, the model also has widespread support in the scientific community (e.g. NAS/NRC, Ex. 321, CHAP. Ex. 84-256). Moreover, Dr. Crump's multistage model also contains a form of delay.

While there is some indication that these risks are, by no means overestimates, the benzene decision gave OSHA leeway to make assumptions which err on the side of overprotection of workers. Thus, OSHA believes the model it has used in the proposal to predict mesothelioma to be a reasonable consideration of the available data and has not changed it for the **final rule.**

In addition to the selection of the time factor, Dr. Crump also expressed concern over OSHA's assumption that the dose-response relationship was linear.He noted that:

The second assumption, namely a linear dose response, is particularly subject to doubt for mesothelioma because there is virtually no dose response data for this cancer. Finkelstein (1983) [Ex. 84-240] contains a table showing dose-response data for mesothelioma derived from a total of only nine mesotheliomas. The Simpson Report (Health and Safety Executive, 1979 [Ex. 84-216]) contained a table (Table 31X) showing a dose response for mesothelioma derived from a case control analysis of data of McDonald *et al.;* however, the table did not appear in the published paper (McDonald *et al,* 1980) [Ex. 237A, p. 35].

Crump plotted the Finkeltein mesothelioma data with linear, quadratic and cubic dose-response curves and observed that "The linear model appears to fit only slightly better than the quadratic, and even the cubic model falls well within the crude 90% confidence bounds" [Ex. 237A, p. 36]. Crump concluded that:

Consequently, a linear dose response for mesothelioma is an assumption which has not been verified observationally. Since it seems biologically implausible that a dose response for cancer would ever be

supralinear (Crump 1984) the linear assumption appears very unlikely to lead to an underestimate of risk from exposure to low concentrations. However, it could possibly provide an overestimate. There have been two general arguments which suggest that a linear dose response is plausible for many carcinogens. One such argument applies for carcinogens that "act by directly causing a mutation in DNA" (NRC, 1977). However, this argument may not be applicable to the carcinogenic mechanism of asbestos in producing mesotheliomas because asbestos has not been shown to be particularly mutagenic. The other general argument holds for carcinogens that produce cancers by the same mechanism by which background tumors are produced (Peto, 1978). However, since the background rate of mesotheliomas is either zero or -- at most -- very small, this argument is not applicable either [Ex. 237A, p. 36].

In an effort to investigate the effects of the choice of the model for mesothelioma, Crump fit a multistage model to the mesothelioma data used by OSHA. He described the model thus:

The multistage model, in its most detailed and complete form (Day and Brown, 1980 and Crump and Howe, 1984), is derived from the assumptions that cancer is initiated in a single cell only after the cell passes through several stages. Cells compete independently to be the first to produce a tumor. The rate at which a cell passes through a dose-related stage is assumed to be proportional to the instantaneous dose.

The model predicts a linear response at low dose whenever either 1) cancers occur "spontaneously" without a carcinogenic insult, or 2) there is only one dose-related stage; otherwise the model predicts a nonlinear response (Crump *et al.,* 1976). The evidence for spontaneous occurrence of mesotheliomas is lacking; consequently, the only way the multistage model can predict a linear response at low dose is for there to be only one dose-related stage. Since there is essentially no dose-response data for mesothelioma, the number of dose-related stages for mesothelioma is open to question [Ex. 237A, p. 44].

At the hearing, Dr. Nicholson defendant the use of the linear dose-response assumption to predict mortality from mesothelioma, stating that:

There's no indication that mesothelioma develops as a result of asbestos fibers acting separately at different stages in the cancer process, which would be required in the multi-stage model to elicit a nonlinear response.

I know of no mechanistic basis that . . . or no experimental data that indicate that that is the case at all.

The limited data what we have, and it is less than that for lung cancer, suggests that linearity is compatible with the data that exists. The data are sufficiently uncertain that one can't say that absolutely linearity is the case. The fact that it's applicable in the case of lung cancer, [a]nd has plausibility of an asbestos fiber doing something, [a]nd the probability of that something being done would be proportional to the number of fibers available to do it exists, and, thus linearity is a most reasonable choice. . . .

One could envision, for example, that mesothelioma comes from those fibers that manage to penetrate the lung wall and get to the pleura. And that in heavy exposure circumstances, the fibrosis that would be present would limit the number that would cross the wall. Thus, you would have in the heavy exposed circumstances fewer mesotheliomas because fewer fibers can penetrate to the pleura than in lower exposure circumstances, giving you a concave downward dose response relationship.

That's just a speculation, as is the speculation of a multi-fiber action at one site. And I don't think either have sufficiently substantive backing to deviate from the use of the linear dose response relationship, which has stood us in good stead in most other circumstances [Tr. 6/19, p. I-140-142]

**TABLE 3. -- ESTIMATES OF K[M] and Goodness of Fit From Six Studies of Occupational Exposure to Asbestos a**

| | | Selikoff (180) b | Seidman (14) | Finkelstein (11) | Peto (7) | Dement (1) | Weill (2) |
|---|---|---|---|---|---|---|---|
| OSHA c | K[Md]d | 1.0 | 5.7 | 12 | 0.7 | 0.22 | 0.07 |
| | P e | 0.07 | 0.74 | 0.39 | .99 | 0.67 | 0.001 |
| MS1 f | K[M] | 110 | 300 | 7,800 | 40 | 3.6 | |
| | P | 0.76 | 0.12 | 0.97 | 0.99 | 0.32 | 0.037 |
| MS2 g | K[M] | 12 | 100 | 270 | 1.9 | 4.4 | 0.76 |
| | P | 0.62 | 0.39 | 0.99 | 0.99 | 0.39 | 0.39 |
| MS3 h | K[M] | 0.59 | 2.4 | 15 | 0.061 | 3.1 | 0.016 |
| | P | 0.62 | 0.73 | 0.83 | 0.99 | 0.39 | 0.90 |

a Crump (Ex. 237A).

b Number of Mesothelioma Deaths.

c Estimates derived from OSHA model (Ex. 84-392). P values and K[M] for Dement et al. and Weill et al. from Crump (Ex. 237A).

d K[M] X10.<8>

e P Value associated with Chi-squared goodness-of-fit test.

f Estimates derived from multistage model with one dose-related stage.

g Estimates derived from Multistage model with two dose-related stages.

h Estimates derived from Multistage model with three dose-related stages.

Table 3 summarizes the results of the goodness-of-fit tests for OHSA's model and the multistage model with one, two or three stages, for each of the data sets used by OSHA and for two additional sets of data. Consideration of the results in Table 3 show that, in fact, in four of the six cohorts, the best fitting model was linear with asbestos concentration (i.e., either the OSHA model or the multistage model with one stage showed the best fit. For the Finkelstein data, the multistage model with two stages fit only slightly better than the linear model, P=0.99 versus P=0.97). For three of the six data sets, the OSHA model fit as well or better than the multistage model. Although the fit of the OSHA model was adequate for the Finkelstein data, the OSHA model did not fit as well as the multistage model (P=0.39 [OSHA] versus P=0.99 [Crump]). And with regard to the Weill data, the fit of the OSHA model was inadequate (P=0.001) and the three-stage multistaga model provided an excellent fit to the data (P=0.90). Similarly, as reported by Dr. Crump, the fit of the OSHA model to the Selikoff et al. data was "marginal", and the multistage model with one dose-related stage provided a very good fit to the date (P=0.76). Implications of the goodness-of-fit tests on the selection of the individual estimates of K[M] will be discussed in the next section.

On the basis of these results, OSHA believes its choice of a risk model for mesothelioma is scientifically responsible. As discussed above, the model has received support from a large number of regulatory agencies, scientific bodies, and individual experts in risk assessment. Moreover, as will be seen in the next section, estimates of the individual K[M] derived from this model are reasonable (and perhaps low), and represent the best estimate of the mesothelioma risk posed by exposure to asbestos.

B. *Data Used for the Calculation of Individual K[M]'s.* In the November proposal, OSHA used four studies judged by the Agency to have data adequate for the quantification of mesothelioma risk [Selikoff et al., Exs. 84-170, 84-90; Seidman et al., Ex. 84-87, 84-170; Peto et al., Ex. 84-170, and Finkelstein, Ex. 84-240]. As Dr. Nicholson pointed out at the hearings:

These were the four studies that did provide sufficient information that could be utilized.

What is necessary is not simply the number of deaths in a particular study, but one has to know the time of those deaths; because the (fit) that was made involves the matching of the equation that's given there, risk according to time per months of exposure, with data on mesothelioma risk at different times from onset of exposure in a defined population.

We had to know the number of cases per person-years of risk [Tr. 6/19, p. I-121-122].

OSHA believed that these four studies were particularly appropriate studies for inclusion in the calculation of K[M] because of the large numbers of mesothelioma deaths observed in these four studies (180, 14, 7, and 11, respectively). It should be noted that these four studies are the same four studies employed by CHAP in its analysis of mesothelioma risk from asbestos exposure [Ex. 83-256, II-119-120].

OSHA acknowledged in the preamble to the November proposal that its estimates of K[M] were derived from studies with four of the five highest K[L] values. OSHA noted that there may be "some bias in examining the value of K[M] independent of the K[L] in the same studies because it is likely that these K[M] would tend to be slightly higher than those derived from other studies, due to the demonstrated high power of these studies to detect risk" [48 FR 51125]. To account for this bias in its analysis, OSHA arrived at an average K[M] by examining the ratios of K[M] to K[L]. This gave an estimate of K[M] of 1 X 10<-8> rather than the higher central values of 4.98 X 10 <-8> rather than the higher central values of 4.98 X 10<-8> (the arithmetic mean) and 2.91 X 10<-8> (the geometric mean). OSHA believed this adjustment to the K[M] value to be appropriate to avoid serious overestimation of the risk of mesothelioma.

Dr. Crump raised a number of issues regarding the calculation of K[M] from these studies. As he had for the calculation of K[M], Dr. Crump noted that the Seidman et al. and Selikoff et al. studies are "particularly inappropriate for risk assessment because of the lack of exposure data" [Ex. 237A, p. 39]. OSHA's reasons for accepting the data from these two studies and the justification for their use in quantitative risk assessment have already been discussed in Section I. In light of the new data received from the Seidman cohort, OSHA has revised its estimates of K[M]. Using the data in Table 1 [Ex. 267A] and four points of observation, the K[M] from the updated study is 2.4 X 10<-8>, somewhat lower than the value for K[M] put forth in the proposal for the original Seidman study. This is not unexpected, particularly in light of the higher average exposure found upon reexamination of the data.

Dr. Crump's second major objection to the use of these studies relateds to the issue of differential risk by fiber type. At the hearing, Dr. Crump noted that --

* * [T]urning to the risk specifically due to mesothelioma, I feel there is strong evidence that the risk in humans at least is less from chrysotile exposure than from amphibole exposures. OSHA estimated risks from four studies, each of which involved either exclusive or considerable exposures to amphiboles.

Although these estimates were adjusted downwards somewhat by comparing them with lung cancer estimates, they still are considerably larger than estimates made from populations exposed predominantly to chrysotile which I have made [Tr. 7/9, p. 84].

In his written testimony, Dr. Crump elaborated on this position:

* * I believe there is considerable data to indicate that chrysotile is less risky [than the amphiboles]. OSHA has already omitted from its risk calculation data from mining and milling operations, on the grounds that these exposures are not representative of those in the populations of workers OSHA has responsibility to protect. I believe this principle should also be applied to the chrysotile-amphibole question, and that risk to modern day workers, who are exposed almost exclusively to chrysotile, should be estimated from studies in which chrysotile exposures predominate [Ex. 237A, p. 47].

In an effort to expand the data from which to calculate an overall K[M], Dr. Crump calculated K[M]'s for two additional studies "for which exposures were predominantly to chrysotile. Theses are the Dement *et al.* study, where exposures were to only chrysotile, and the Weill *et al.* study, in which 77% of the workers were exposed exclusively to chrysotile" [Ex. 237A, p. 40]. The mesothelioma data for these two studies are found in Tables D and E. The K[M] calculations for various models are found in Table 3.

For the Dement et al. data found in Table 4, the model used by OSHA provided a much better fit to the data (P=0.67) than any of the multistage models, and gave a K[M] of 2.2 X 10<-9>, approximately five times lower than the K[M] of 1 X 10<-8> K given in the proposal. Of the multistage models, all of which allowed showed good fit, the three-stage model gave a K[M] of 3.1 X 10<-8>, more than 10 times larger than that estimated by the OSHA model and three times larger than OSHA's expressed preferred estimate of risk. Dr. Crump calculated the ratio of K[M]/K[L] for the Dement et al. study (K[M]/K[L]=2.2 X 10<-9>/0.042=5.2 X 10<-8>) and concluded that "this indicates that the assumption implicitly made by OSHA of a constant ratio is not universally valid" [Ex. 237A, p. 41]. Using Crump's preferred estimate of risk for K[L] (0.023) gives a ratio of 9.5 X 10<-8>, approximately 10 times smaller than the average K[M]/K[L] used in OSHA's determination of an overall K[M].

**TABLE 4. -- NUMBER OF MESOTHELIOMA DEATHS AND ABSOLUTE RISK BY YEARS FROM FIRST EXPOSURE, DEMENT ET AL. (1983) a**

| Years since first exposure (Avg) | Observed mesothel-iomas | Person-years | Absolute risk b |
|---|---|---|---|
| 10 (5) | 0 | 11,390 | 0 |
| 1020 (15) | 0 | 10,921 | 0 |
| 2030 (25) | 0 | 8,055 | 0 |
| 30 + (35) | 1 | 2,775 | 0.3604 |
| Total | 1 | | |

a From Crump (Ex. 237A, Table 4).

b Absolute risk=(number of deaths/person-years)X1,000.

**TABLE 5. -- Number of Mesothelioma Deaths and Absolute Risk by Years From First Exposure, Weill et al. (1972) a**

| Years since first exposure (Avg) | Observed mesothe-liomas | Person-years | Absolute risk b |
|---|---|---|---|
| 10-15 (12.5) | 0 | 31,180 | 0 |

| | | | |
|---|---|---|---|
| 15-20 (17.5) | 2 | 29,473 | 0.0678 |
| 20-25 (22.5) | 0 | 25,080 | 0 |
| 25-30 (27.5) | 0 | 14,018 | 0 |
| 30-35 (32.5) | 0 | 3,832 | 0 |
| 35 + (37.5) | 0 | 1,565 | 0 |
| Total | 2 | | |

a From Crump (Ex. 237A, Table 4).

b Absolute risk = (number of deaths/person-years)X1,000.

Table 5 gives the results of the calculation of K[M] for the Weill et al. study. Data from the Weill et al. cohort gives, by far, the smallest values of K[M]. The OSHA model shows an inadequate fit to the data (P=0.001) with a K[M] of 7.0 X 10<-10>. The three-stage multistage model showed excellent fit to the data (P=0.90) and gave a K[M] of 1.6 X 10<-10>, almost 100 times smaller than the overall K[M] calculated by OSHA in the proposal.

Dr. Crump pointed to the calculation of K[M] for the six studies, three with mixed exposures (Selikoff et at., Seidman et al., and Finkelstein) and three with predominantly chrysotile exposures (Peto et al., Dement et al., and Weill et al.) and observed that:

What one sees here is a large difference between the potency estimates in the upper three studies involving the mixed exposures and those in the lower three involving exposures primarily to chrysotile. . . . [I]f you look at the geometric mean, there is about a 20-fold difference in the risk. Although there is more uncertainty in the numbers in the lower group because of smaller numbers of mesotheliomas, these values are still not consistent with the ones in the upper group. I feel that, taken together, they do show a pattern of a smaller risk experienced by the workers -- based upon exposure measurements -- workers exposed predominantly to chrysotile.

The value of potency used by OSHA was 1, which is smaller than the estimates for the upper studies, but as you can see, it is considerably greater than the estimates made for populations exposed mainly to chrysotile [Tr. 7/9, p. 87].

However, during questioning, Dr. Crump admitted that --

* * * [T]he chrysotile estimates I was making, I was thinking about exposures which are today predominantly chrysotile. I wasn't thinking of necessarily applying those in situations where the exposures were to mixed fibers in removal operations [Tr. 7/9, p. 119].

Although the asbestos manufacturing industry may confine itself primarily to the use of chrysotile fiber in its products, OSHA believes now, as it did at the time of the proposal, that the major sources of exposure to asbestos workers in the next 20 to 40 years will be in the demolition, renovation, and removal of asbestos products (for example, insulation) which were installed 30 to 40 years ago. These products generally contain amphiboles. This was brought out by Dr. Nicholson during cross-examination, when he noted that:

I should make the point though we are concerned in much of the regulation of the future with exposures that will be to materials that have already been put in place, in the insulation materials, the sprayed on asbestos materials, all these loosely friable [sic] insulation materials that have been applied over the years.

Virtually all of the those exposures to those materials will be of a mixed fiber type. And so I think that's what we have to deal with. You can find in some circumstances, some manufacturing circumstances, pure fiber exposure. I don't know what their risk started at, as the discussion has indicated, because of the variabilities inherent in those studies.

But most of the exposures that we have in the future will be mixed fiber exposures [Tr. 6/19 p. I-144].

Hence, OSHA believes it is wholly correct in using esitmates of K[M] from studies of mixed exposures as well as single-fiber type exposures in determining an overall estimate of mesothelioma risk.

Moreover, in a post-hearing submission, Dr. Nicholson gave some additional analysis of the carcinogenic response to different asbestos fiber types [Ex. 303A]. In an effort to make a broader comparison of mesothelioma according to exposure by mineral type, Dr. Nicholson compared the risk of pleural and peritoneal mesothelioma with that of lung cancer in a variety of studies. After various appropriate adjustments, the ratio of mesothelioma as a percentage of adjusted excess lung cancer was calculated for four studies of interest. This analysis showed reasonable agreement with the analysis done by OSHA. Dr. Nicholson concluded:

In comparing the different ratios of pleural mesothelioma to adjusted lung cancer for all studies in which the major exposure was to one fiber type, one can see that there are roughly comparable ratios for chrysotile, amosite and mixed exposure. Crocidolite has approximately a two-fold greater number of mesotheliomas as percent of excess adjusted lung cancer. However, as noted previously, the untraced individuals in the various crocidolite cohorts may lead to an overestimate of this ratio. Though some greater potency may be considered for crocidolite regarding mesothelioma (a factor of two perhaps), the uncertainty associated with other factors in a given exposure circumstance lead to much greater differences. For example, as was seen in the case of lung cancer, different exposure circumstances with the same fiber led to nearly 100-fold differences. Thus, the suggestion that there are dramatic differences between different asbestos varieties has no basis in fact. Much greater differences would appear to be related to process, to fiber size distribution effects within a single asbestos variety (note the difference between textiles and mining, e.g.), or to methodological differences in cohort studies (e.g., the asbestos cement studies of Weill et al. and of Finkelstein) [Ex. 303A, p. 6].

In addition to the data from occupational cohorts, Nicholson also pointed to some evidence of environmental exposures as supportive evidence. He noted that:

Mesothelioma has been documented in a variety of non-occupational circumstances, including among family contacts of asbestos-exposed individuals. . . . Notable is that family contact cases are seen with exposure to chrysotile, amosite and crocidolite. Relative to the risk at work, there appears to be little difference in the family contact risk by fiber type.

Animal studies substantiate the above analysis and suggest that all varieties of asbestos should be considered equally potent with respect to the production of either lung cancer or mesothelioma. Table 6 [of Ex. 303A] lists the data of Wagner et al. (1974) [Ex. 84-96] from inhalation studies using different forms of asbestos. Canadian chrysotile produced as many mesotheliomas as crocidolite and more than amosite or anthophyllite. Further, it produced lung cancer with a single day's exposure [Ex. 303A, p. 6-7].

The addition of the Weill et al. study and the Dement et al. study to the data base used for the overall calculation of K[M] raises several points. First, the small number of mesothelioma deaths in the two studies makes the estimates of risk much less reliable. Dr. Nicholson discussed the advantage of additional information, but remarked that --

* * * [T]he total number of cases involved in those two studies is three. So it would be a very large uncertainty of any estimates made with those. And when one averaged it with the much higher levels of the four studies, would not substantially after the lower value which was chosen in the OSHA document.

That is, we would now be using an average of six studies rather than four. . . . [I]f those additional two studies were utilized there may not have been the need to artificially lower the average that was obtained using the four studies that were cited here . . . [i]n essence, what I'm saying is that if you take account of all the data, I don't think it would change the estimate of K[M] substantially. And, in fact, the correction that was made to lower the estimate is an appropriate one. It fits most of the data that do exist [Tr. 6/19, p. I-138].

Dr. Crump also noted the added uncertainty associated with the use of studies containing small numbers of deaths [Ex. 7/9, p.87].

OSHA has computed the arithmetic and geometric means of the K[M]'s of the six studies for both the values of K[M] from the OSHA model (including Dement et al. and Weill et al. as computed by Crump) and for the "best fit" model using the K[M] from the multistage model with one, two or three stages. As Dr. Nicholson suggested, the inclusion of the Dement et al. and Weill et al. data may "eliminate the need to artificially lower the average" by looking at the ratio of K[M] to K[L], since these two studies represent the lower end of the mesothelioma risk. Using the data in Table 3, the OSHA model gives an arithmetic mean of the K[M] of 2.73X10<-8>, (almost three times that proposed) and a geometric mean of 0.82 X 10<-8>, approximately equal to OSHA's best estimate of K[M] given in the proposal.

The mean values of the estimates of K[M] from each of the six studies from the multistage model with the best fit are astonishingly high, with an arithmetic mean of 64.26 X 10<-8> to 70.92 X 10<-8>, (up to 70 times larger than OSHA's preferred estimate of K[M]) and a geometric mean of the six K[M]'s of 2.45 X 10<-8> to 7.2 X 10<-8>. Further inspection of Table 3 demonstrates that using several values of K[M] from models with only slightly poorer fit (e.g., .097 vs. 0.99) would produce estimates of risk several orders of magnitude larger. Hence, according to this analysis, OSHA's original choice of a best estimate of K[M] of 1X10<-8> is by no means an overestimate, as Dr. Crump apparently contends; indeed, his own calculations show that 1 X 10<-8> in fact, greatly underestimates the mesothelioma risk which may be experienced by asbestos-exposed workers.

In addition, OSHA has examined several alternate combinations of the data, including computing the best estimate of K[M] from the ratio of K[M]/K[L]. As in the lung cancer data, these calculations produce estimates which bracket the 1X10<-8>.

Dr. Crump's preferred estimate of K[M] of 2X10<-9> [Ex. 237A, p. 48] was based solely on the studies of predominantly chrysotile-exposed workers and was meant to represent the mesothelioma risk of workers exposed predominantly to chrysotile; his preferred estimates was not meant to characterize the risk of mesothelioma faced by workers in a variety of workplaces -- including the major exposures to mixed fibers that will occur in asbestos removal, demolition, and renovation operations [Tr. 7/9, p. 119].

OSHA has therefore determined that Dr. Crump's approach is not adequate to address the question of the total risk posed by asbestos exposure, and the Agency has chosen instead to base its best estimate of risk on the six studies with sufficient data to quantify the excess risk of mesothelioma. Hence, OSHA concludes that its best estimate of K[M] remains at 1X10<-8>, as proposed. The addition of the two studies with small numbers of deaths adds some uncertainty to this estimate but, as indicated, this estimate is likely to represent a substantial underestimate of the risk of mesothelioma actually experienced by asbestos-exposed workers.

III. Estimates of Risk for Other Cancers

As discussed in Section IV, OSHA has concluded that workers exposed to asbestos are likely to be at an

increased risk of gastrointestinal cancer. Though an excess of GI cancer has not been observed consistently in every study of asbestos workers, and while the ratio of gastrointestinal cancer to lung cancer varies considerably from study to study, there appears to be sufficient evidence to roughly estimate the excess gastrointestinal cancer risk in asbestos-exposed populations. A number of submissions to the record recognized the relationship between asbestos and gastrointestinal cancer [see, e.g., Exs. 91-40, 116, 163e, 158, 261A, 277, 297, 321]. In general, the risk ranges from about 5 to 20% of the excess lung cancer risk.

The AIA/NA commented that:

Although excess GI cancers have been found in some heavily exposed worker studies, no such excesses have been found in many other studies. Of the twenty-one studies reviewed by OSHA (in each of which there was a minimum of 10 observed or expected GI cancers), only seven had statistically significant excess GI cancers (Ex. 84-392 at 13) [Ex. 328, p. 1-21].

However, Dr. Nicholson pointed out at the rulemaking hearing that:

* * * [Ex. 84-392] said 21 studies were listed. Twelve demonstrated an excess gastrointestinal cancer, and eight demonstrated a deficit. One was even.

Many of those -- several of those -- actually were studies in which there was also no excess lung cancers. So there were circumstances where the excess risk to be expected was a very low one. And, thus, one would be within the range of statistical fluctuations no matter what the risk was; since the GI cancer . . . risk is never expected to be equal to that of the excess lung cancer risk.

I think, of these 21 studies . . . only 13, if I'm not mistaken, would demonstrate an excess lung cancer risk.

And the ones that do not [demonstrate an excess lung cancer risk] are largely the negative ones [for GI cancer] [Tr. 6/19, p. I-117].

In addition, OSHA believes the finding of a statistically significant excess of GI cancer in seven studies of worker populations to be a substantial body of evidence. As pointed out by Dr. Nicholson, many of the studies in which GI cancer was not observed were unable to detect lung cancer as well. This points perhaps to methodological problems in the studies as well as low exposures.

It was also suggested that the observed excesses could conceivably be due to a misdiagnosis of peritoneal mesothelioma. While OSHA believes it is unreasonable to totally account for these excesses (some as large as 60% of the lung cancer risk) by misdiagnosis, to the extent that the incidence of mesothelioma has been underobserved in these studies, then OSHA's predictions of the risks of mesothelioma are also underestimated.

In an attempt to quantify the risk of gastrointestinal cancer, OSHA considered a simple risk model in which gastrointestinal cancer risk was assumed to be equal to 10% of the lung cancer excess risk. As Dr. Nicholson noted:

Based upon the rough finding and given the fact that there are different dose-response relationships, that overall, considering an increase over lung cancer of 10 percent for gastrointestinal cancer would give an underestimate of possible asbestos-related GI cancers.

One finds that the relationship that I just mentioned, comparing excess GI cancer with excess lung cancer to be such that some studies demonstrated an increase of GI cancer about 50-60 percent that of lung cancer, a very high correlation. Others show, in some cases, dificits, but showed very much lower ratios.

Considering that lung cancer is increasing in recent years, the ratio between excess GI cancer to lung cancer would decrease, a value of 10 percent excess was chosen as a reasonable value. It's a relatively small additional contribution. I think it underestimates what the actual contribution would be [Tr. 6/19, p. I-115-116].

There was some objection to OSHA's quantification of the risk of gastrointestinal cancer (e.g. Ex. 328), the major issue being a lack of an observed dose-response for this type of cancer. Again Dr. Nicholson responded to this objection:

Well, we have limited dose-response data. And it's of two natures. One in terms of increased risk with increased exposure. It would appear that it's a very flat relationship. I've looked at it specifically for insulation workers, and it turns out that within about 10 years, there appears to be an elevated risk 50 percent above that which would be expected, approximately.

And that same elevated risk continues with time among insulators who continue working.

* * * There is a second dose-response relationship that is seen. . . . [I]f one takes those studies in which the number of gastrointestinal cancers either expected of observed exceeds 10, so we're looking at a study that has enough data that it could be -- the results would not be simply statistical variability, and the study shows a statistically significant lung cancer risk so that we're looking at studies that have exposures that are of significance, one finds a fairly reasonable increasing relationship in the risk of, overall risk, of gastrointestinal cancer with the overall risk of access [excess] lung cancer. That is, access [excess] gastrointestinal cancer compared to access [excess] lung cancer correlates reasonable well [Tr. 6/19, p. I-113-114].

And, while Dr. Schneiderman noted "There is no adequate model of digestive cancers", he also stated that "OSHA's estimate [for gastrointestinal cancer risk] appears to be reasonable" [Ex. 116, p. 2]. Even Dr. Weill, who said he would have preferred OSHA not include quantitative estimates of GI cancer risk noted that "it doesn't make a lot of difference in my view in terms of the policy that emerges from such a risk assessment" [Tr. 6/19, p. I-193].

Thus, OSHA feels confident in including estimates of risk from gastrointestinal cancer in the final standard.Though this is still some controversy over the inclusion of these estimates in the risk assessment, OSHA believes there is sufficient evidence to support their inclusion and to suggest that their contribution to the overall estimates of risk may, in fact, be understated. The estimates of risk of gastrointestinal cancer are also given in Table 6 along with estimates of lung cancer and mesothelioma risks.

The incidence of cancers at sites other than the lung, mesothelium, and gastrointestinal tract have been shown to be elevated in some asbestos exposure studies, including laryngeal, kidney, pharyngeal and buccal cavity cancers. To OSHA, it appears that the excess risk for "other cancers" is about the same as for gastrointestinal cancers. OSHA recognizes many uncertainties in quantifying this risk, in view of the inconsistencies in findings among different epidemiologic studies. (Some studies have found excess risk from other cancers, while other studies have not). The sites showing excess risk have also varied among studies. Therefore, OSHA has not made numerical estimates of risks for these other cancers at this time. To the extent that estimates of these cancers are not included in the overall estimates of risk, OSHA has underestimated the total cancer risk posed by exposure to asbestos.

The data indicating gastrointestinal cancer excesses are stronger and more consistent than the data suggesting excesses at these other cancer sites. Thus, OSHA does not feel compelled to quantify the risk of cancer at these other sites at this time. The high quality and well-supported estimates of the excess risk of lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis alone provide sufficient bases upon which to justify this regulatory action.

IV. Estimates of Cancer Mortality

The best estimates of K[L] and K[M] were utilized to estimate the mortality from exposures to varying concentrations of asbestos for different time periods. The calculations are age, intensity and duration specific. Table 6 shows the excess asbestos-related mortality rates from lung cancer, mesothelioma, and gastrointestinal cancer (gastrointestinal cancer excess is assumed to be 10% of the lung cancer excess). Table 6 gives the predicted excess lifetime risk of cancer for exposures of one year, 20 years, and 45 years, assuming first exposure at age 25. In these calculations, Equation 1 and Equation 3 were used with values of K[L] equal to 0.01 and K[M] equal to 1 X 10<-8> and the 1977 U.S. male background lung cancer mortality rates. Because of age-specific increases in lung cancer rates in older men since 1977, estimates based on more recent background rates would be higher. Calculations were done for each 5-year age interval, and then summed to give a total lifetime risk. The calculations performed to give the results in Table 6 assumed that the relative risk increased following ten years after onset of exposure and continued to rise until ten years after cessation of exposure, after which it remained constant.

**Table 6**

**Estimated Asbestos Related Cancer Mortality per 100,000 by Number of Years Exposed and Exposure Level n1**

| Asbestos fiber concentration (f/ml) | Cancer mortality /100,000 exposed: Lung | Mesothelioma | Gastrointestinal n2 | Total |
|---|---|---|---|---|
| | | 1 year exposure | | |
| 0.1 | 7.2 | 6.9 | 0.7 | 14.8 |
| 0.2 | 14.4 | 13.8 | 1.4 | 29.6 |
| 0.5 | 36.1 | 34.6 | 3.6 | 74.3 |
| 2.0 | 144 | 138 | 14.4 | 296.4 |
| 4.0 | 288 | 275 | 28.8 | 591.8 |
| 5.0 | 360 | 344 | 36.0 | 740.0 |
| 10.0 | 715 | 684 | 71.5 | 1470.5 |
| | | 20 year exposure | | |
| 0.1 | 139 | 73 | 13.9 | 225.9 |
| 0.2 | 278 | 146 | 27.8 | 451.8 |
| 0.5 | 692 | 362 | 69.2 | 1123.2 |
| 2.0 | 2713 | 1408 | 271.3 | 4392.3 |
| 4.0 | 5278 | 2706 | 527.8 | 8511.8 |
| 5.0 | 6509 | 3317 | 650.9 | 10476.9 |
| 10.0 | 12177 | 6024 | 1217.7 | 13996.7 |
| | | 45 years exposure | | |
| 0.1 | 231 | 82 | 23.1 | 336.1 |
| 0.2 | 460 | 164 | 46.0 | 670.0 |
| 0.5 | 1143 | 407 | 114.3 | 1664.3 |
| 2.0 | 4416 | 1554 | 441.6 | 6411.6 |
| 4.0 | 8441 | 2924 | 844.1 | 12209.1 |
| 5.0 | 10318 | 3547 | 1031.8 | 14896.8 |
| 10.0 | 18515 | 6141 | 1851.5 | 26507.5 |

n1 Assumes exposure begins at age 25. Risks are calculated using U.S. male lung cancer background rates for 1977.

n2 Estimated as 10% of lung cancer risk rather than calculated using dose-response information.

Several comments should be made regarding the results in Table 6. Though excess relative risk in linear in dose, the excess mortality rates given in Table 6 are not strictly linear in dose. Therefore, for example, the risk at 2 f/cc is not exactly 4 times the risk at 0.5 f/cc, though there is a close approximation. It should also be noted that the risks for longer periods of exposures do not appear to be a straight forward multiplication of the risks of shorter duration. In the longer exposure categories, where exposures will affect older workers, some adjustments have been made for competing risks which are likely to affect the death rate from lung cancer. In addition, when looking at total cancer risks, it must be remembered that these include the risk of mesothelioma, which is related to time in an exponential fashion.

As can be seen from Table 6, the predicted risk from mesothelioma is approximately equal to the lung cancer risk for one year of exposure and to about half of the risk value for lung cancer in the 20-year exposure group. The excess risk of mesothelioma after a lifetime exposure (45 years) to asbestos is approximately one-third the lifetime excess lung cancer risk. These predictions comport with observations in several populations, where mortality from mesothelioma is observed to comprise approximately 50% of the excess mortality from lung cancer.

Using the equations given earlier, and based on the calculations in Table 6, OSHA predicts a lifetime excess risk of total cancer for a lifetime exposure (45 years) to 2 f/cc as 6411 excess deaths per 100,000 workers, or approximately 64 per 1000. Since risk from a 20 year exposure to asbestos may also be of interest, the models predict an excess cancer mortality of 4392 dealths per 100,000 workers exposed at 2 f/cc for 20 years.

Reducing in the PEL from 2 f/cc to 0.2 f/cc reduces the risk from lifetime exposure from 64 per 1000 to 6.7 per 1000. Similarly, for a 20 year exposure, the risk is reduced from 44 per 1000 to 4.5 per 1000, representing a 90% reduction in risk. The lifetime risk from one year of exposure follows a similar course. The risk reduces from 296 per 100,000 at 2 f/cc to 30 per 100,000 at 0.2 f/cc.

Lastly, Table 6 contains risks for levels higher than 2 f/cc because OSHA believes some industrial areas (such as construction) may still be at these higher level. This population of workers would consequently experience a much greater reduction in risk by reducing exposures to 0.2 f/cc or less. Moreover, to the extent that the controls that are installed to meet the new PEL result in exposures below 0.2 f/cc, cancer risks will be reduced to a greater extent than indicated in the table.

V. Quantifying the Excess Risk From Asbestosis

The November proposal included a quantification of the excess risk of asbestosis. Asbestosis is a type of pulmonary fibrosis diagnosed on the basis of a history of exposure to asbestos; it is characterized by radiologic changes to the lung, breathlessness, impaired lung function, and other clinical features of fibrosing lung disease. Asbestosis can be manifested in a range of degrees of severity and can result in disability and death.

An early response by the lung to asbestos exposure is formation of plaques, which are opaque patches visible on chest X-rays. The presence of plaques may indicate an increased risk of future development of asbestosis, but this is not certain. Although the significance of pleural plaques in terms of disease is not clear, the presence of plaques is not normal.

Asbestosis has been known to progress or worsen after cessation of exposure to asbestos, probably due to irreversible injury and/or the retention of asbestos fibers in the lung. In addition to lung function impairment, asbestosis contributes to increased asbestos-related mortality. Increased resistance created by the lung obstruction can lead to heart failure.

As pointed out by Dr. Weill in his written testimony, "Exposure-response relationships have been reported using as the biologic response indicator either a constellation of clinical findings to define asbestosis, or certification by a worker's compensation panel or board" [Ex. 99, p. 24], but such approaches have "varying degrees of limitation" [Ex. 99, p. 12]. Because of the many possible combinations, and therefore "definitions" of asbestosis given by different groups, the quantification of a single risk associated with asbestosis is difficult. As Dr. Weill noted during cross examination:

* * * The problem is with asbestosis, the quantification is not exactly the same as it is with malignant disease, because one is dealing with a different set of rules in the ascertainment of this health effect. And no two studies have exactly the same scheme for making a decision that this individual has asbestosis and this individual doesn't [Tr. 6/19, pp. 205-206].

In his prehearing testimony, Dr. Weill explained further:

Mortality data are not useful in quantifying the risk of asbestos-induced lung fibrosis (asbestosis). Affected workers may die with asbestosis but not of it, in which case it is not likely to appear on the death certificate as the primary cause of death. In contrast, sensitivity of detecting early evidence of asbestosis in a living exposed population has increased substantially in recent years. . . . Since much of the asbestosis being seen now is the result of lower dust levels in the past two decades, the films are likely to be classified in the lower categories of profusion of small opacities (fewer shadows meaning less severe disease). As is frequently the case with biological measurements, it is at these lower limits of disease detection that inter- and intra-observer variability is greatest. Again, it is gratifying to know that in spite of these recognized problems, excellent exposure-response relationships have resulted from the radiographic classification described [Ex. 99, 23].

Quantitative studies exist, primarily for the disabling forms of the disease; specifically, two separate studies provide information to develop a dose-response relationship between asbestos exposure and incidence of asbestosis [Ex. 84-254 and 84-44.] Details of the data were reported at 48 FR 51130. It is clear that material impairment from asbestosis occurs prior to the onset of its disabling stage.

As discussed in the November proposal, Berry et al. [1979, Ex. 84-20] studied a group of 379 men who worked at an asbestos textile factory for at least 10 years. Dust measurements were available and were correlated to each job performed for each year under study. Health effects were correlated to cumulative exposure. Using prevalence data, Berry et al. found a dose-response relationship with cumulative exposure (f-y/cc) for three endpoints, crepitations, possible asbestosis, and certified asbestosis. In addition, these data also support the hypothesis that there is a low, or possibly no, threshold for asbestosis, since there is increased risk at cumulative exposures as low as 37 fiber-years/cc.

Berry and Lewinsohn [1979, Ex. 84-254] have reported the incidence of asbestosis in this same asbestos textile factory. The population was divided into two cohorts: those first employed before 1951 and those employed after 1950. A dose-response relationship is apparent for the incidence data, though it is not quite as consistent as for the prevalence data.

In a second study, Finkelstein [1982, Ex. 84-44] looked at the development of compensable (certified) asbestosis among 201 workers at an asbestoscement factory in Ontario. A dose-response relationship was developed using estimated cumulative exposures based on plant dust measurements and using medical information from the Ontario Workmen's Compensation Board.

As noted by Dr. Weill, "A final complicating aspect in the development of exposure-response information on asbestosis is that it is a slowly progressive disorder which may (and frequently does) continue to worsen after exposure ceases" [Ex. 99, p. 12].

OSHA's original estimates of risk were derived from a simple linear regression of the incidence of asbestosis on the midpoints of the cumulative exposure data of Berry and Lewinsohn and of Finkelstein. A linear relationship was assumed, at least for the point estimation of 0.5 fibers/cc for 45 years (or 22.5 fiber-years/cc). As Dr. Weill stated:

While the shape of the dose-response curve for asbestosis cannot be determined with certainty, it is clear that this fibrotic effect is dose-related, perhaps linearly, and whether a threshold exists may very well depend on the response indicator chosen [Ex. 99, p. 11].

The assumption of risk linearity is consistent with the fact that early stages of the disease are observed at low exposures. This point was reiterated by Howard Ayer on behalf of the Organization Resources Counselors, Inc. when he noted that:

It does appear clear that there is a simple linear relationship between the frequency and degree of asbestosis and the cumulative exposure to asbestos dust. Time is merely a factor in that it takes a certain amount of time -- at least a matter of years -- to develop the effect on the lung [Ex. 91-10-2, pp. 4-5].

A similar conclusion is drawn in the report of the British Advisory Committee on Asbestos, when the committee noted that: "The present authors come down in favor of a dose-response relationship [asbestosis] without a threshold for chrysotile within the range experienced in industry" [Ex. 84-216, volume 2, p. 38]. Based on this recommendation, OSHA did employ a linear model in the prediction of risk from asbestosis, but made no attempt in the proposal to extrapolate the data below the 0.5 f/cc level or above the 10 f/cc level using this model.

Based on the three cohorts discussed above, OSHA calculated estimates of the lifetime incidence of asbestosis for the Finkelstein, Berry and Lewinsohn pre-1951 cohort, and the Berry and Lewinsohn post-1950 cohorts, respectively. The estimates from the three cohorts differ by an approximate factor of three. This may be indicative of some of the methodological differences among the studies. For example, it is possible that the estimates made from Berry and Lewinsohn's data may be underestimates. The maximum duration of follow-up in that study was 23 years, with an average follow-up of 16 years. Observations from Finkelstein's data (his Table 1) demonstrate that only 41% (23/56 cases) of total incidence was experienced in the first 24 years since first exposure. That is, 59% of the asbestosis incidence was not expressed until at least 25 years from onset of exposure. Thus, it is likely that the low incidence rates in the Berry and Lewinsohn studies (and, therefore the low estimates of risk predicted from these data) are reflective of the short follow-up period for this group of workers.

On the other hand, Finkelstein's (1982) observations may overstate the incidence of asbestosis because at autopsy there was histologic evidence of silicosis as well as asbestosis in many men. Finkelstein states that "we have, nevertheless, chosen to call their disease 'asbestosis' as we believe that is the pathologic process of most significance. Most of the parenchymal radiographic abnormalities were small irregular opacities and the mortality pattern among the men was consistent with the toxic effects of asbestos" [Ex. 84-44, p. 500].

More importantly, it is indeed possible that all of these investigators may have understated asbestosis risk by examining only certified disability from asbestosis, which is an advanced stage of the disease. As noted in the November proposal, there was evidence of the early signs of asbestosis at levels as low as 37 f-y/cc (this level produced a 1% prevalence of crepitations) and is consistent with the predictions made above. During the hearings, several witnesses stressed the range of physical and mental disability/impairment which may occur long before even radiologic evidence of disease appears. Typical of these comments were those made by Dr. Irving Selikoff of the Mount Sinai School of Medicine. He stated:

So, what you're seeing on x-ray is always very much less than is really present pathologically. So that, when you see a positive x-ray, there's a fair amount there in the lung . . . I've seen people with comparatively little on x-ray, who can't walk across a room. But by and large, all it means is that there's been scarring [TR. 7/2, p. 170].

While several participants commented in general on the risk of asbestosis, there was little direct comment on OSHA's quantitative estimates of risk. Hence, for these revised rules. OSHA has relied on the models developed for the proposal to predict the risk of asbestosis at the new PEL of 0.2 f/cc. Using OSHA's best estimate of risk, that from the Finkelstein data, OSHA predicted that exposure over a working lifetime to the 2 f/cc level will result in approximately a 5% incidence of asbestosis. Reducing the exposure to 0.2 f/cc would result in a lifetime incidence of asbestosis of 0.5%. While OSHA did not make predictions of risk at levels below 0.5 f/cc in the proposed rules, testimony received during the rulemaking increases OSHA's confidence that the Agency's estimates of risk at 0.2 f/cc are valid and reasonable. This is due primarily to the comments noting the validity of the model in the low dose region. Given the difficulties in accurately diagnosing cases of asbestosis and the fact that OSHA's estimates only take the risk of disabling asbestosis into account, OSHA believes that the Agency's estimates may be underestimates of the true risk of asbestosis to exposed workers.

VI. Significance of Risk

As discussed above in Section III (Pertinent Legal Authority), the Supreme Court in the Benzene case (*Industrial Union Department, AFL-CIO* v. *American Petroleum Institute* 448 U.S. 601 (1980)) ruled that, prior to the issuance of a new or revised standard regulating occupational exposures to toxic materials, OSHA must make a determination that a "significant" health risk exists and that the new standard will reduce or eliminate that risk. OSHA's analytical approach to making a determination that a significant risk of material impairment exists from exposure to hazardous workplace chemicals takes into consideration a number of factors that are consistent with recent court interpretations of the OSH Act and rational, objective policy formulation. As prescribed by Section 6(b)(5) of the Act, OSHA examines the body of "best available evidence" on the toxic effects of hazardous chemicals to determine the nature and extent of possible health consequences resulting from exposure to the hazardous agent in question. Quantitative risk assessments are conducted, where possible, and the results are considered along with other relevant information, such as the nature and severity of the health consequences, to determine whether a hazardous agent poses a significant risk to workers at the current permissible exposure level. The Agency also determines whether a reduction in the permissible exposure level for the hazardous agent will substantially reduce that risk.

The Court gave some general guidance to the Agency for arriving at findings of the significance of an occupational health risk. It recognized that the Agency's determination that a particular level of risk is "significant" will be based largely on policy considerations (*IUD* v. *API,* 448 U.S. 655, 656, n. 62). To illustrate how one may make a determination from quantitative information that a health risk is significant, the Court stated as follows:

It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. 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 (*IUD* v. *API* 448 U.S. at 655).

Although the Court's example is based on a quantitative expression of the risk, the Court indicated that the significant risk determination required of OSHA is not "a mathematical straitjacket," and that "OSHA is not required to support the finding that a significant risk exists with anything approaching scientific certainty." "A reviewing court [is] to give OSHA some leeway where its findings must be made on the frontiers of scientific

knowledge [and] . . . the Agency is free to use conservative assumptions in interpreting the data with respect to carcinogens, risking error on the side of overprotection rather than underprotection" (448 U.S. at 655, 656).

OSHA has followed these guidelines in making a determination that the risk of material health impairment resulting from **occupational exposure to asbestos** is significant. The epidemiological and toxicological evidence and testimony presented in the November notice and in Section IV (Health Effects) of this preamble clearly show that exposure to asbestos is carcinogenic to humans and additionally causes disabling fibrotic lung disease. Lung cancer constitutes the greatest health risk to asbestos workers; in some occupational cohorts, this disease has been responsible for more than half of the excess mortality from asbestos exposure. Malignant mesotheliomas of the pleura and peritoneum, which are extremely rare among non-exposed persons, have been conclusively linked with asbestos exposure. Some studies of asbestos-exposed workers have also shown increases in mortality from gastrointestinal and other types of cancer. It has been known for years that exposure to asbestos is the only known cause of asbestosis, a progressive, fibrotic lung disease causing effects ranging from shortness of breath during exertion to complete disability, respiratory and cardiac failure, and death. OSHA's determination that the health risks from asbestos exposure is significant is based, in part, on the irreversible and ultimately fatal nature of these diseases, particularly of lung cancer and mesothelioma.

The finding that a significant risk exists is primarily supported by OSHA's quantitative risk assessment, which is based on studies of asbestos-exposed worker populations. OSHA's risk assessment (discussed in Section V of this preamble) estimates that 64 excess cancer deaths (including those from lung and gastrointestinal cancer and mesothelioma) will occur among 1,000 workers exposed at the existing permissible exposure limit of 2 f/cc for 45 years, a working lifetime.The estimates of mortality risk from mesothelioma, lung cancer, and gastrointestinal cancer are 16, 44, and 4 excess deaths, respectively, per 1,000 workers exposed for 45 years at 2 f/cc.

OSHA also estimated the risk of lung cancer, mesothelioma, and gastrointestinal cancer for 20-year and 1-year durations of exposure to asbestos at 2 f/cc. From this analysis, OSHA estimates that the risk from all asbestos-related cancers among workers exposed from 20 years to 2 f/cc is 44 excess deaths per 1,000 workers. The estimated cancer risk from all cancers among workers exposed to 2 f/cc for one year is estimated to be 3 excess deaths per 1,000 workers.

Additionally, OSHA estimated the risk (i.e., the predicted incidence) of asbestosis morbidity at the existing permissible exposure level of 2 f/cc. OSHA's best estimate is based on the results of a high-quality study of the incidence of compensable (certified) asbestosis at an asbestos-cement factory (Ex. 84-240). Based on cumulative exposure data and assuming a linear model, OSHA estimates that the incidence of asbestosis is 50 cases per 1,000 workers exposed for 45 years to 2 f/cc.

In the April notice, OSHA characterized the basis for determining that a significant risk exists at the 2 f/cc level as being "particularly strong" (49 FR 14120). This assessment was based on the reliance on occupational epidemiological studies for the quantitative risk assessment, the high quality of the scientific data, the consistent estimates of dose-response among the various studies used, and the appropriateness of the models and methods employed in the risk assessment. Review of the record evidence submitted since publication of the April notice has served to reinforce OSHA's confidence in the data and analysis underlying the determination that a significant risk exists at the existing permissible exposure level for asbestos.

Regarding the quality of the data, several commenters stated that the health evidence for asbestos-related disease is far more convincing, due to the quality and number of human studies available, than are health effects data for any other hazardous substance. This point was emphasized at the informal hearing by Dr. Nicholson under cross-examination by Ms. Seminario of the AFL-CIO:

Seminario: Would you say that [the data for asbestos] . . . is generally better and more complete than . . . [for

other toxic substances]?

Nicholson: I don't even think there's a comparison. The data for asbestos are so much more extensive than those of other toxic substances in the workplace. It's a wide divergence.

Seminario: . . . Basically, you have asbestos with a lot of studies and a lot of information, and a great number of workers included as subjects in those studies . . . compared to less complete data for other toxic substances?

Nicholson: Yes.

Seminario: . . . [I]t really is a much more complete data base for conducting risk assessment and making estimates [of risk] than you would have for any other substance?

Nicholson: Yes, it is

Seminario: . . . [I]n conducting risk assessments, in many cases, those risk assessments will be based not on epidemiologic studies, but, indeed, on animal studies. Is that correct?

Nicholson: Often, that may be our only recourse, in other studies. . . . [I]f one reviews the [International Agency for Research on Cancer Monographs] . . . volumes 1-29 that have evaluated human carcinogens, they have only deemed 18 agents or work processes to have sufficient data for which one could . . . establish carcinogenicity [in humans], let alone provide quantitative risk assessments in hypothetical circumstances. So our human data are very scanty for most agents (Tr. 6/19 pp. 134-135).

Similarly, Dr. Hans Weill commented that ". . . we know of no other occupational disease for which more complete exposure-response data are available from human population studies" (Ex. 99, p. 30). In its post-hearing submission, Organization Resources Counselors, Inc. stated that "[a]sbestos is a proven carcinogen of long standing. Volumes of scientific work attest to the fact that asbestos produces both lung cancer and mesothelioma" (Ex. 127-A, p. 2). These comments, and the evidence contained in the record on health effects from asbestos exposure (see Section IV) reaffirm OSHA's belief that the data used in the quantitative risk assessment are of unusually high quality.

A review of the rulemaking record has also strengthened OSHA's belief that it used the most appropriate models to calculate the risk. To estimate the risk for lung cancer, OSHA used a linear dose-response model based on evidence found in several epidemiologic studies that examined lung cancer mortality in relation to cumulative asbestos exposure (Exs. 84-43, 84-59, 84-35), and on the use of a linear model by several other investigators (Exs. 85-22, 84-216, 84-243, 82-2, 84-180, 84-256, 321). For mesothelioma, OSHA used an absolute risk model, which has been used or suggested by a number of other authors to estimate the risk of mesothelioma (Exs. 84-252, 84-385, 84-342, 84-87, 132, 84-138). In response to record comments submitted after publication of the April notice, OSHA revised the individual potency factors for lung cancer (K[l]) and mesothelioma (K[m]) for some of these epidemiological studies (see Section V of this preamble). These adjustments had little effect on the overall K[l] of 0.01 and K[m] of 1X10<-8> originally proposed by OSHA for the combined data sets. OSHA believes that this finding reflects the reasonableness of the risk estimates for lung cancer and mesothelioma set forth in the April notice.

The first element established by the Supreme Court's Benzene decision (*IUD* v. *API* 448 U.S.) for determining the significance of risk of material impairment -- that a significant risk existed at the existing permissible exposure limit of 2 f/cc -- is thus clearly and decisively established by OSHA's risk assessment and by the insidious nature of asbestos-related disease. In making a determination that this risk is significant, OSHA relies, in part, upon the Supreme Court's indication of when a reasonable person might consider a risk significant and take steps to decrease that risk. OSHA finds, as indicated by the risk assessment, that the

existing standard of 2 f/cc would permit an excess cancer mortality risk of 64 deaths per 1,000 employees and an estimated asbestosis incidence of 50 cases per 1,000 employees exposed for a working lifetime; this excess risk must be considered significant and unacceptable using virtually any reasonable basis for making such a determination. OSHA also finds that the excess risk of cancer mortality resulting from 20 years of exposure to asbestos (44 excess deaths/1,000 workers) is also significant. As pointed out in the April notice (49 FR 14120), the risk from asbestos exposure at the 2 f/cc level has also been acknowledged as being unacceptable by other governments (Exs. 84-378, 84-379). The level of risk estimated by OSHA at the existing permissible exposure limit is also comparable to the estimated risks for other toxic substances that OSHA has regulated or rpoposed to regulate in the past.

In accordance with the second element of the Supreme Court's Benzene decision on the determination of significant risk, OSHA has determined that reducing the permissible exposure limit for asbestos to 0.2 f/cc is reasonably necessary to reduce the cancer mortality risk from exposure to asbestos. OSHA's risk assessment shows that lowering the permissible exposure limit from 2 f/cc to 0.2 f/cc reduces the asbestos related cancer mortality risk from lifetime exposure from 64 deaths per 1,000 workers to 6.7 deaths per 1,000 workers; this corresponds to a 90 percent reduction in the risk. The asbestos-related cancer risk is also reduced by 90 percent, from 44 deaths to 4.5 deaths per 1,000 employees, for a 20-year exposure duration. It is estimated that the incidence of asbestosis for workers exposed for a working lifetime under the new standard will fall by 90 percent, from 50 cases to 5 cases per 1,000 employees. As these figures show, significant risks of asbestos-related cancer mortality and asbestosis morbidity are not eliminated at the exposure level that is permitted under the new standard; however, the reduction in the risk of asbestos-related death and disease brought about by promulgation of the new standard is both significant and dramatic.

The observation that significant risk is not eliminated under the new permissible exposure level of 0.2 f/cc led some rulemaking participants to urge OSHA to promulgate an even lower permissible exposure limit. For example, in its post-hearing brief, the Building and Construction Trades Department of the AFL-CIO agreed with OSHA's findings on the significance of risk:

. . . OSHA's estimates point to two conclusions.First, lowering the PEL from its present level will significantly reduce the risk of mortality from lung cancer, mesothelioma and gastrointestinal cancer. This is especially evident at the BCTD-recommended PEL of 100,000 fibers per cubic meter (0.1 f/cc) where 61 fewer deaths per 1,000 workers will occur. Second, while under . . . [the Benzene decision] it is unnecessary to find the existence of a significant risk at intermediate levels above the *new* PEL . . ., a significant risk exists even at this lowest of potential PEL's. (Ex. 330, p. 11)

OSHA agrees with the BCTD that a signifiant risk of asbestos related disease would exist even under a standard having a permissible exposure limit of 0.1 f/cc. As OSHA explained in the April notice in the Summary and Explanation sections of the preamble to the final standards for asbestos for General Industry and Construction, OSHA's decision to promulgate a permissible exposure limit of 0.2 f/cc is not based on a determination that significant risk is eliminated at this level. Given that a significant risk of harm persists even at very low levels of lifetime exposure to asbestos, OSHA's decision to promulgate a PEL of 0.2 f/cc is based on a determination that this level is the lowest level that can feasibly be attained in operations in workplaces in both general industry and construction.

Some commenters, such as Organization Resources Counselors, Inc. (ORC) (Ex. 123-A) and the Asbestos Information Association of North America, (AIA/NA) (Ex. 328), argued that OSHA overstated the risk of disease from asbestos exposure. Specifically, they objected to the following:

-- OSHA's use of past exposure levels, or the 2 f/cc PEL coupled with the assumption of lifetime exposure duration, as benchmarks for determining risk, rather than the lower exposure levels and shorter durations typically found in industry today.

-- Failure to account for differential risks posed by different types of asbestos fiber.

-- Failure to distinguish between the cancer mortality risk for asbestos-exposed workers who smoke and those who do not.

Regarding the use of past exposure data or the current PEL of 2 f/cc to estimate risk levels, the ORC commented as follows:

. . . ORC recommends that estimates of risk be based on exposures . . . that are relevant to 1984 workplace conditions. It is important to know as accurately as possible what the actual risk is at today's exposure levels, but this is not possible unless we recognize the factors in the risk equation that have changed from 1944 to 1984. (Ex. 123-A, p. 12)

Similarly, the AIA/NA stated:

OSHA further errs toward over-prediction of risk by assuming, without substantiation, that workers will experience exposures at the level of the standard for up to 45 years. In fact, the record evidence indicates [that] exposures will average significantly below any standard. . . . As would be predicted from accepted technological feasibility and industrial hygiene practice control, average workplace exposures to asbestos have been found to be one-fourth or less of a given standard (based on OSHA field monitoring results). . . . More detailed data from the United Kindom confirm that under its former 2 f/cc standard, average exposures in all but textile manufacturing were but one-tenth the PEL, and in textile generation -- the most difficult to control -- exposures averaged one-fourth thd standard. (Ex. 328, pp. 22-23)

ORC and AIA/NA also objected to the use of a 45-year exposure duration for estimating risks. ORC commented that "[t]he majority of 1984 exposure are intermittent, and 4-5 days per month would be on the high side for an industry-wide average" (Ex. 123-A, p. 14). The AIA/NA argued as follows:

OSHA's significant risk findings are also predicated on an assumed 45-year lifetime exposure. Although 45-year exposures are theortically possible, the evidence in the record demonstrates that only a very small minority of workers will be exposed that long. The vast majority of asbestos-exposed workers will experience fewer then 10 years [of] exposure. As Dr. Nicholson notes at the hearing, approximately half of all workers leave an industry within six months, and the remaining half work in a given industry between eight and twelve years. (Ex. 328, p. I-24)

The AIA/NA concluded that the actual risk to workers exposed to asbestos is approximately one-sixteenth that predicted by OSHA, because ". . . average exposures over and average working life will be for one-fourth the time at one-fourth the level of OSHA's lifetime exposure predictions" (Ex. 328, p. I-25). For this reason, the AIA/NA claimed that significant risk would be eliminated at a new PEL of 0.5 f/cc.

OSHA agrees that the record indicates that the actual exposure conditions and employment patterns of many workers today do not conform to the exposure and duration characteristics underlying the lifetime exposure assumption used in the Agency's risk assessment. However, when determining whether a hazardous substances poses a significant risk and that reduction of a PEL is warranted, OSHA must consider what degree of risk would be *permitted* by the existing standard, even though many workers may in fact be at lesser risk because their employers have chosen to reduce their exposures to levels below those required by that standard. It is for this reason that OSHA bases its determinations of significant risk on exposure to a PEL and not on reported exposure conditions. However, it should be noted that OSHA does analyze current exposure conditions in workplaces when assessing the potential benefits of new regulations, as required by Executive Order 12091. For example, in this rulemaking, OSHA has quantified the benefits of the new standard, taking into account

current occupational exposure conditions (see Section VII).

The use of the lifetime exposure (45-year) assumption has also been standard in determining significant risk in previous OSHA rulemakings. OSHA has several reasons for using a lifetime exposure assumption. First, the use of a 45-year lifetime exposure duration is based on guidance given in the OSH Act. As specified in Section 6(b)(5): "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 of functional capacity *even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life"* (emphasis added). OSHA believes that it is reasonable to assume that a person begins work at age 20 and continues until the age of 65, a 45-year span of employment. Under Section 6(b)(5) of the Act, OSHA is compelled to promulgate standards that ensure that employees, even those exposed to the hazardous agent for their entire working lifetime, are at the lowest risk that can feasibly be attained. Therefore, OSHA's determinations of significant risk must take into account the fact that many workers may be exposed throughout their entire working lives, and reflects the view that OSHA is regulating workplace conditions and not specific employees.

A second reason for using an assumption of lifetime exposure is that this method permits comparison of the risks from asbestos exposure to the risks posed by other substances that OSHA has regulated or proposes to regulate. Such comparisons are useful to the Agency in ensuring that a consistent policy underlies OSHA's determinations of significant risk. Because the Agency has determined significance of risk in previous rulemakings based on the lifetime exposure assumption, the use of shorter exposure duration for calculating the risk of asbestos-induced disease would preclude the Agency from making such comparisons. As stated in the April notice (49 FR 14120), the Agency has determined that exposure to asbestos results in an excess disease risk that is many times that found for other hazardous agents that have been regulated by OSHA.

OSHA also believes that the argument made by the AIA/NA, that use of an assumption involving a shorter exposure duration would result in a reduction in risk, is invalid. OSHA's risk assessment shows that the total asbestos-related cancer risk is not linearly related to duration of exposure, and that risk is not reduced proportionally when the exposure durations used are reduced. The reasons for this effect are twofold: First, as the pupulation of asbestos-exposed workers ages, the proportion of this population dying form asbestos decreases because many of these individuals die from other diseases that are related to aging. Second, the relationship between exposure duration and the risk of dying of mesothelioma is not linear. Both of these elements contribute to the non-linearity of the relations between exposure duration and the risk of incurring asbestos-related cancer. The non-linearity of the relationship between risk and duration is illustrated by comparing the total asbestos-related cancer risk for a 45-year exposure duration with that for a 20-year exposure duration. Although there is a 56 percent reduction in exposure duration, there is only a 31 percent reduction in total asbestos-related cancer risk (from 64 to 44 deaths per 1,000 employees). Accordingly, assuming that employees are exposed to asbestos for shorter durations because of employee turnover would actually *increase* the absolute risk among the larger number of workers exposed for less than their working lifetimes, compared with the risk predicted for a constant number of workers exposed for a working lifetime. Such an increase in absolute risk is a result both of the larger number of workers exposed to asbestos for some period of time if turnover is taken into account and the non-linearity of the relationship between exposure duration and asbestos-related cancer risk. This is illustrated in a technical report (Ex. 84-405) submitted to the record by OSHA showing that calculating risks taking employee turnover and less-than-lifetime exposure into consideration results in a larger number of predicted asbestos-related cancer deaths than would be predicted using a model that assumes a lifetime exposure duration and no employee turnover. Therefore, OSHA finds that use of the lifetime exposure assumption does not result in an overstatement of the risk of mortality from asbestos-related cancers.

This concept is particularly relevant to the construction industry, which is characterized by higher employee

turnover as compared to manufacturing industries. One commenter, the Associated General Contractors of America (AGC) argued that OSHA's risk estimates do not apply to the construction industry because of the unique exposure patterns characteristic of that industry:

Many of the studies on the dangers of asbestos have only limited implications for the construction industry. Forty-five years of exposure to 2 f/cc of airborne asbestos may cause sixty-four excess cancer deaths per one-thousand workers, but few if any construction employees will ever experience such exposure. Very few employees will remain in the industry for forty-five years. Very few will even experience more than low level, intermittent exposure to asbestos. (Ex. 84-457, p. 1)

OSHA recognizes that many construction employees are exposed on a less frequent basis than employees in general industry. However, OSHA disagrees with AGC's contention that the health evidence for asbestos has "limited applications" for construction employees. First, there are construction employees, particularly those employed by asbestos abatement and demolition contractors, who have regular exposures to asbestos. Second, as discussed above, OSHA's determination of the significance of risk must be based on the risks that would be permitted by a standard, and not the actual risk of employees who are exposed at a level below that standard. OSHA has no basis for believing that risks posed by exposure to asbestos at the current PEL of 2 f/cc in construction would be any different than the risks to employees exposed to 2 f/cc in general industry.

Another issue raised by the AIA/NA involved the effect of fiber type on OSHA's risk estimate for asbestos-related cancer. By not accounting for the different carcinogenic potencies of the various fiber types, the AIA/NA maintained that the ". . . predicted risk from mesothelioma is likely to be substantially over-estimated" (Ex. 328, p. I-17). The AIA/NA went on to state.

. . . OSHA's sole reliance on four studies where exposures were mixed, and were a large number of mesotheliomas were found, biases its risk assessment to the high side . . . Had OSHA relied on a more representative set of studies showing the highest potencies, their mesothelioma risk estimate would have been reduced by a least half. (Ex. 328, p. I-19)

OSHA discusses the health evidence for different fiber types in Section IV of this preamable. In that section, OSHA concluded that, although epidemilogical studies indicate that exposure to amphiboles is associated with a greater mesothelioma risk than is exposure to chrysotile, animal studies show the opposite effect. Several rulemaking participants suggested a variety of reasons for this discrepancy. OSHA agrees with Dr. Davis (Tr. 7/10, p. 65) that, on a fiber-by-fiber basis, there are no data to show conclusively that amphibole fibers are more potent than chrysotile fibers. For this reason, OSHA did not distinguish among fiber types when conducting the Agency's risk assessment. Furthermore, no evidence was submitted to the record to indicate that such a fiber-type differential exists for lung cancer risk, which constitutes the largest component of the total cancer mortality risk predicted by OSHA's risk assessment. Moreover, even if OSHA agreed with the AIA/NA and used an estimate of mesothelioma risk that was reduced by 50 percent, the risk of dying of asbestos-related cancer continues to be significant even at the new PEL of 0.2 f/cc: reducing the mesothelioma risk by half results in an excess of 5.3 asbestos-related cancer deaths per 1,000 employees, a figure more than 5 times the Supreme Court's guidelines for significant risk. Therefore, OSHA does not agree with that its risk estimates are significantly overstated because they do not differentiate among fibers of different types.

A controversial issue raised during the rulemaking was whether the combined impact of smoking and asbestos exposure on the incidence of asbestos-related disease should lead OSHA to promulgate regulations prohibiting smoking in workplaces in lieu of establishing a lower PEL for asbestos. The epidemiological evidence presented in Section IV (Health Effects) of this preamble does indicate that the combined effect of asbestos exposure and smoking on lung cancer risks is greater than the sum of the individual lung cancer risks for these two hazards. The evidence for the effect of smoking and asbestos exposure on the incidence of asbestosis is equivocal, and there is no known relationship between smoking and mesothelioma risk. Based on this

evidence, the AIA/NA argued that:

By failing to take the smoking factor into account, the OSHA risk assessment attributes a substantial portion of the risk, which is solely a matter of personal habit, to workplace exposure . . . . Section 5(b) of the OSHA Act requires each worker to comply with standards that apply 'to his own actions,' indicating that Congress intended to regulate employee conduct at least where the employer cannot control it . . . . [B]y failing to separate out the substantial portion of the lung cancer risk due to smoking, OSHA has again overestimated the risks of exposure to asbestos. Given that smokers are easily identifiable and that successful programs can be instituted to eliminate or substantially reduce smoking among asbestos workers . . . the risk Assessment fails to provide the necessary scientific basis for assessing risk reduction measures through a revised standars. (Ex. 328, p, I-28)

OSHA believes that the AIA/NA's belief that the Agency's risk assessment does not account for the portion of lung cancer risk caused by smoking is not accurate. OSHA's risk assessment for lung cancer is based on studies that measured the *relative* risk of lung cancer among asbestos-exposed populations, and not the *absolute* risk. In other words, all of the studies on which the Agency's risk assessment is based measured the increase in risk among asbestos-exposed workers over and above that experienced by the general population, which includes somkers. In some of these studies, smoking was a confounding factor that was controlled for. It is unlikely that most of the excess lung cancer deaths found among asbestos-exposed cohorts are attributable solely to smoking, as evidenced by the failure of these studies to observe significant excesses of other smoking-related diseases, such as bladder cancer and heart disease. Therefore, OSHA finds that the lung cancer risk estimates predicted by the quantitative risk assessment cannot be principally attributed to smoking.

This view is also held by Dr. Weill, whose written testimony states that "while it is clear that the extent and prevalence of smoking in a study population, its various exposure groups, and the comparison or control group, can have an extremely important effect on lung cancer exposure-response curves, there is insufficient information available to allow smoking to be used in quantitative risk assessment for asbestos-related lung cancer" (Ex. 99, p. 28). Moreover, OSHA's estimate of the risk of mesothelioma mortality, which is not confounded by smoking is significant in itself (1.64 deaths per 1,000 workers) for lifetime exposure at the new PEL of 0.2 f/cc.

Methodological considerations aside, OSHA find it inappropriate, from a public health viewpoint, to determine the significance of occupational risk for different populations of workers who may have different sensitivities and different lifestyles on the basis of forces that act outside of the workplace. Section 6(b)(5) of the Act makes it clear that OSHA is to promulgate standards that ensure that ". . . no employee will suffer material impairment of health or functional capacity . . ." as a result of exposure to occupational hazards. Although it is true that smoking is associated with a considerable risk of lung cancer mortality, exposure to asbestos substantially increases that risk among workers who smoke. OSHA has consistently maintained that reducing the permissible exposure limit is the approach that "most adequately assures" that employees will not suffer material impairment of health as a result of occupational exposure to toxic substances. OSHA is continuing this policy by choosing not to attempt to make a distinction among exposed worker populations who may have different lifestyles. OSHA's authority to regulate workplace hazards and to reduce their associated risks, even in cases where exposure to the hazard may also occur outside the workplace, was recently reaffirmed by the U.S. Court of Appeals for the Fourth Circuit in its decision upholding OSHA's Hearing Conservation Amendment *(Forging Industry Association* v. *Secretary of Labor):*

[The Forging Industry Association] . . . constructs its first argument that because hearing loss may be sustained as a result of activities which take place outside the workplace . . . OSHA acted beyond its statutory authority by regulating non-occupational conditions or causes. . . . [T]he [hearing Conservation] amendment does nothing more than ensure that a hearing-endangered worker is provided with protection *in the workplace* [emphasis in original] in order to decrease the risk of a hearing impairment. Having identified employee

susceptibility to noise, '[t]he Act does not wait for an employee . . . [to] become injures, authorizes the promulgation of health and safety standards . . . in the hope that these will act to prevent . . . injuries from ever occuring. *Whirlpool Corp.* v. *Marshall,* 445 U.S. 1, 12 (1980). . . .

[That hearing loss sustained outside the workplace may aggravate that sustained within the workplace] . . . is scant reason to characterize the primary risk factor as non-occupational. Breathing automobile exhaust and general air pollution, for example, is damaging to the lungs, whether [the lungs are] healthy or not. The presence of unhealthy lungs in the workplace, however, hardly justifies failure to regulate noxious workplace fumes. Nor would there be logic to characterizing regulation of the fumes as non-occupational because the condition inflicted is aggravated by outside irritants (*IFA* v. *Secretary,* p. 9, 13).

Therefore, OSHA is well within its statutory authority when it regulates asbestos as a workplace carcinogen and applies the revised asbestos standard to all exposed employees, despite the presence of non-occupational factors, such as smoking, that serve to compound the risk of some workers. OSHA believes that, by promulgating this revised standard, it is carrying out its Congressional mandate to reduce serious occupational risks, to the extent feasible, for all American workers exposed to asbestos.

VII: Final Economic Impact and Regulatory Flexibility Analysis

This analysis has been performed in accordance with the requirements of Executive Order 12291 and the Regulatory Flexibility Act of 1980 (5 U.S.C. 601 et seq.). The following paragraphs summarize the economic and other impacts of the **final rule** on those industries most likely to be affected.

*Industries Affected*

The industries affected by the final standard include primary manufacturing, secondary manufacturing, automotive brake and clutch repair, shipbuilding and ship repair, and construction.

Primary Manufacturing

Several industrial processes are used by primary manufacturers to create these diverse product lines, and many potential sources of airborne asbestos fibers can be identified throughout each process. Two particular operations that are common to all processes and that have a high potential for generating airborne asbestos fiber are fiber introduction and product finishing.

The fiber introduction stage includes operations that are necessary for preparing the asbestos fiber for subsequent mixing or blending. Broken bags and spills in the fiber receiving and storage areas account for the releases of airborne fibers during this operation. (It should be noted, however, that such exposures may be reduced through modern packing methods.) Fibers may also become airborne when compacted asbestos fiber is removed from the supplier's sealed containers prior to mixing. Depending on the product line, the compacted fiber may be "willowed" or "fluffed" to facilitate mixing. Asbestos fiber may become airborne due to leakage or spillage during mixing, mixer unloading, or processing operations. In a dry-mix process, fibrous asbestos may become airborne as the batch is weighed and additional materials are added. Once the fiber has been wetted with water or other substances, encapsulated, or bonded with other materials, fiber release is significantly reduced.

In product finishing operations, asbestos fibers become airborne when they are torn loose from the parent product as it is cut, sawed, drilled, texturized, shaped, or otherwise modified to form a finished product. Occupational exposures may also occur after the finishing operation or in the handling and disposing of asbestos-contraining wastes. The number of plants and the number of potentially exposed workers are presented in Table 7.

Secondary Manufacturing

Secondary fabricators are defined as establishments that receive products from primary manufacturers and further process or fabricate these products to produce other intermediate or finished products. Primary asbestos products that undergo significant secondary processing include flat asbestos-cement (A/C) sheet, friction products, gaskets and packings, plastics, and textiles. Secondary processing involves sawing, pressing, slitting or drilling of asbestos-containing materials and, hence, produces some relatively high exposure levels. The number of plants and workers are presented in Table 8.

Service Industries and Construction

In the service sectors two industries are affected: (1) Automotive brake and clutch repair and (2) shipbuilding repair. The number of sites and the number of potentially exposed workers in these sectors are shown in Table 9.

**TABLE 7. -- ANNUAL PRODUCTION AND ESTIMATED NUMBER OF ESTABLISHMENTS, WORKERS EXPOSED, AND EXPOSURE LEVELS FOR PRIMARY MANUFACTURERS OF ASBESTOS PRODUCTS**

| Product line | Annual production | Estimated number of workers exposed | Estimated number of establish-ments | Estimated 8-hour TWA exposure levels (f/cc) |
|---|---|---|---|---|
| A/C pipe | 258,060 tons | 512 | 5 | 0.01-1.21 |
| A/C sheet | 604,310 squares | 203 | 6 | N/D-2.4 |
| Friction materials | 260,000,000 pieces | 5,104 | 51 | N/D-7.9 |
| Textiles | 4,730 tons | 413 | 3 | N/D-3.79 |
| Flooring | 750 (10<6>) ft<2> | 278 | 3 | N/D-0.3 |
| Gaskets | 35.8 (10<6>) ft<2> | 214 | n1 19 | 0.03-2.06 |
| Packings | 51.4 tons | 101 | n1 19 | 0.03-2.06 |
| Paper | 72,324 tons | 387 | 22 | N/D-1.42 |
| Coatings | 177 (10<6>) gallons | 1,327 | 78 | N/D-3.3 |
| Plastics | 8,409 tons | 324 | 4 | N/D-1.11 |
| Total | | 8,861 | 191 | |

n1 The same plants make both gaskets and packings.

N/D=Non-Detectable.

Sources: RTI 1984 Survey Data [Exhibit. 84-473]; OSHA MIS Files; OSHA Hearing; and ICF Inc., Asbestos Products and Their Substitutes, Appendix C, December 1983.

**TABLE 8. -- ESTIMATED NUMBER OF PLANTS, WORKERS EXPOSED, AND EXPOSURE LEVELS FOR SECONDARY FABRICATORS OF ASBESTOS PRODUCTS**

| Product line | Estimated number of establish-ments | Estimated number of workers exposed | Estimated 8-hour TWA exposure levels (f/cc) |
|---|---|---|---|
| Gaskets/packings | 289 | 9,972 | N/D-0.77 |
| Automotive remanufacturing | 181 | 4,750 | N/D-1.6 |
| Plastics | 245 | 2,450 | N/D-0.29 |
| Friction materials | 40 | 1,504 | N/D-0.75 |
| A/C sheet | 23 | 345 | N/D-3.2 |
| Textiles | 51 | 172 | N/D-1.8 |
| Total | 829 | 19,193 | |

N/D=Non-detectable.

Sources: RTI 1984 Survey Data [Exhibit. 84-473]; OSHA MIS Files; OSHA Hearing; and ICF Inc., Asbestos Products and Their Substitutes, Appendix C, December 1983.

**TABLE 9. -- ESTIMATED NUMBER OF ESTABLISHMENTS, WORKERS EXPOSED AND ASBESTOS EXPOSURE LEVELS IN SERVICE AND REPAIR INDUSTRIES**

| Sector | Number of establish-ments | Number of workers exposed | 8-hour TWA exposure levels (f/cc) n1 |
|---|---|---|---|
| Automotive brake and clutch repair | 285,188 | 526,998 | N/D-0.94 |
| Shipbuilding and repair | 400 | 15,000 | n1 N/D-1.42 |
| Total | 285,588 | 541,998 | |

n1 These data do not include nuclear rip-out where wet methods are not permitted. The 8-hour TWA exposures during nuclear rip-out range from 0.2 f/cc to 7.2 f/cc.

N/D=Non-detectable.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, based on RTI 1984 Survey Data, Phase 1 Report, Regulatory Analysis of the Proposed Standard on Asbestos [Exhibit No. 84-473]; Management Information Systems (MIS) Files; OSHA Hearing; ICF Inc., Asbestos Products and Their Substitutes, Appendix C, December 1983.

Asbestos exposures in the construction industry occur during various activities (see Table 10). For example, such exposures occur when installing A/C pipe and sheet, finishing drywall, sanding vinyl-asbestos floor tiles,

installing build-up roofing, removing old insulation, removing or repairing drywall, demolishing buildings containing asbestos products, and removing old built-up roofing. Workers involved in the maintenance and repair of pipes, boilers, or furnaces in a wide variety of buildings are also exposed to asbestos.

*Availability of Substitutes*

The extensive tort litigation in the area of **occupational exposure to asbestos** and the awareness of the health effects associated with asbestos exposure have provided a strong incentive for producers and users of asbestos products to utilize substitutes. For example, approximately 50-75 percent of producers of phenolic molding compounds have substituted other materials such as clay or fiberglass for asbestos. Similar success has been achieved in the production of floor tile, where non-asbestos fibers and petrochemicals are being used, and in friction materials. Fiberglass has been used successfully as a substitute for asbestos fiber in many products. Roofing felts, pipeline felts, and adphalt coatings have all been produced using fiberglass in place of asbestos fibers.

In the past, the price of substitute materials has been much higher than the price of asbestos. The "full price" of using asbestos, which includes the potential cost of control methods, tort litigation, etc., however, has increased significantly in recent years. Consequently, the difference between the cost of using asbestos and the cost of using other substitute materials has diminished greatly and in many instances has disappeared entirey.

**TABLE 10. -- ESTIMATED NUMBER OF WORKERS EXPOSED AND ASBESTOS EXPOSURE LEVELS IN THE CONSTRUCTION INDUSTRY**

| Sector | Estimated No. of workers exposed | Mean 8-hour TWA exposure levels (f/cc) |
|---|---|---|
| New construction | 29,320 | 0.13 |
| Abatement | 81,366 | 1.85 |
| Demolition | 24,455 | 0.61 |
| General building renovation | 133,700 | 2.8 |
| Routine maintenance in commercial and residential buildings | 217,745 | 0.29 |
| Routine maintenance in general industry | 259,643 | 0.51 |
| Total | 746,228 | |

Sources: RTI [Exhibit. 473]; Building owners Survey (Ex. 84-474); Consad Phase 1 Report (Ex. 84-474); and 1982 Census of Construction.

Technological Feasibility

Introduction

This analysis determines the extent to which it is currently feasible to reach a permissible exposure limit (PEL) of 0.2 fibers per cubic centimeter during affected work operations without the use of respirators. The informaltion in the public record provides the basis for OSHA's determination that a PEL of 0.2 f/cc for an 8 hour time-weighed average (TWA) can be achieved, with a few exceptions, across the asbestos-products manufacturing industry. Exposure data indicate that some of the plants in this industry have combined engineering controls and prudent work practices to reach exposure levels below 0.2 f/cc. OSHA recognizes that

some data show the current difficulties of reaching a 0.2 f/cc TWA, but OSHA believes compliance with the new PEL will become increasingly feasible in these operations. In the construction industry, the data show the capability of meeting the PEL in most operations by the conscientious application of engineering and work practice controls.

Based on this analysis, OSHA has determined that compliance with the 0.2 f/cc PEL is feasible in most industries most of the time through the use of wet methods, engineering controls, and good housekeeping practices. There are some operations, however, for which compliance through the use of engineering controls and work practices alone does not appear feasible at this time. These situations are usually due to the inability of the operation to use wet methods (e.g., textiles, nuclear rip-out, building repair, etc.), and the volume of dust generated (e.g., cutting operations for A/C pipe and sanding A/C sheet). During these operations, therefore, respiratory protection must also be used until employers apply current technology more effectively or apply new technology to the control of asbestos dust.

General Considerations

As stated above, OSHA based its conclusion about the technological feasibility of the 0.2 f/cc level on the record evidence and data summarized later in this section. The following discussion sets out the legal and policy framework for making these determinations.

Section 6(b)(5) of the Occupational Safety and Health (OSH) Act provides that OSHA may promulgate standards to the extent that they are economically and technologically feasible. In meeting its statutory mandatee to set "feasible" standards, OSHA is guided by judicial review of 14 years of Agency standards setting.

According to the Supreme Court, requirements may be imposed up to the limits of what is "technologically achievable." [*American Textile Mfgs. Institute et al.,* 452 U.S., fn. 34, 1981 OSHA sec. 25,457.] Accordingly, OSHA may promulgate standards which can be met most of the time by the technologically advanced plants in an industry. [See e.g., *American Iron and Steel Inst.* vs. *OSHA,* 577 F. 2d 825, 932-35 (3d Cir. 1978).] [*Ibid,* 5717 F. 2d at 835.] Current exposure levels in such technologically advanced plants may meet the PEL only one some measured days, yet that level may be considered feasible [*Ibid;* 577 F.2d at 835]. In addition, in cases where data show the current industry exposure levels are in excess of the new PEL, the new PEL is, nevertheless, determined to be technologically feasible if substantial evidence exists to show that companies acting in good faith can develop the necessary technology to reach the new PEL [*United Steelworkers,* 647 F.2d at 1269, 1272].

The D.C. Circuit has explained that the purpose served by OSHA's industrywide feasibility determination is to create "a general presumption of feasibility for an industry . . . [is] that industry can meet the PEL without relying on respirators" [647 F. 2d at 1296]. In the case of asbestos, OSHA has determined based on this rulemaking record and guided by this body of decisions that most industry sectors in most operations most of the time will be able to meet a time weighted average PEL of 0.2 f/cc primarily through the application of currently available engineering and work practice controls. Supplemental respirator use will be needed only occasionally. (Later, in this section OSHA discusses on an industry sector basis more detailed reasons and evidence supporting these feasibility determinations.)

Claims about technological feasibility made by participants in the rulemaking supported all exposure levels considered in the proposal, from 0.1 f/cc to 0.5 f/cc. Participants advanced policy arguments and evidence in support of their positions. For example, the AFL-CIO stated that the evidence showed that 0.1 f/cc was feasible for general industry to achieve primarily through engineering and work practice controls [see, for example, Exhibits 143 and 335]. However, as detailed in the specific industry sector discussions, the evidence indicates that the 0.1 f/cc level is not currently feasible in most dry operations in manufacturing and secondary

processing of asbestos products. In the construction activities of renovation and major abatement, a proponent of a 0.1 f/cc level for construction agrees with OSHA that supplemental respirator use will be necessary to meet that lower level [see Exhibit 330]. Therefore, OSHA has determined that a 0.1 f/cc may not be achievable in most operations without routine respirator use.

In contrast, other participants contended that a 0.2 f/cc level was technologically infeasible in most manufacturing industries and, therefore, that a 0.5 f/cc should be designated as the PEL. Proponents of a 0.5 f/cc PEL did not dispute reports of the levels of exposure currently being achieved in such industries. In fact, the major proponent of the 0.5 f/cc level, the Asbestos Information Association of North America (AIA/NA) agreed that "OSHA's proposed PEL of 0.2 f/cc is close to the center of the best achievable exposure range for most manufacturing workplaces [see Exhibit 312 A]. Additionally, AIA projects that the incentive effect of a new reduced PEL will result in "long term average exposures to typical asbestos product manufacturing workers . . . in the neighborhood of 0.1 f/cc or below." AIA further projects that "[e]ven employees in the most difficult to control industry workplaces would not experience average exposure levels above 0.2 f/cc" [Exhibit 312 A].

AIA objected to finding the 0.2 f/cc level technologically feasible for two reasons. First, AIA defined a "feasible" exposure level as one in which an employer will have a 95 percent level of confidence that exposures on any day will not exceed the PEL.Therefore, according to AIA, because airborne asbestos exposure levels fluctuate from day to day, setting a 0.5 f/cc PEL would be necessary to assure that employers will not be subject to citation on unrepresentative "high" days. The second reason given by AIA is that because the measurement and analytical method for assessing asbestos exposures is uncertain at lower levels, imposing a 0.2 f/cc PEL will not allow employers to ascertain whether they are in compliande [Exhibit 328, p. 7].

Day-to-Day Variability of Exposure Levels

To demonstrate day-to-day variability, AIA submitted evidence of recent exposure levels at plants identified as well controlled in various industry sectors. AIA stated that these data showed that the airborne asbestos exposures varied significantly from day to day at the same work station due to factors beyond the employer's control [Exhibit 312, Table H].

OSHA believes that AIA's data in fact supports the Agency's conclusion that 0.2 f/cc is feasible. AIA's data from three asbestos-cement pipe plants show that all operations in these plants would be able to meet a 0.2 f/cc PEL more than 50 percent of the time. These data also show that most operations in the asbestos-cement pipe industry could be expected to do significantly better. Approximately 80 percent of the measurements in the fiber introduction area and approximately 90 percent of the measurements in the pipe formation and lathe finishing area could be expected to read under 0.2 f/cc [Exhibit 312A, Table III] based on AIA's own calculations. In addition, OSHA disagrees with AIA's contention that since little can be done about the sources of variability and a conscientious employer must keep his average exposures far below the PEL, so that he will not inadvertently be cited on a "high day" [Exhibit 312A, Tab H, p. 4]. AIA listed the factors that influence variability, including changes in internal airflows such as fans being turned off or blocked, inoperative or blocked ventilation systems, or changes in individual work practices.

OSHA has observed in its enforcement experience that proper inspection and maintenance of ventilation systems can greatly increase their effectiveness and reduce the variability resulting from inefficient operation of such control systems [see also Exhibit No. 335]. OSHA also believes that variation in work practices may be minimized by supervision and training programs. While OSHA agrees with AIA that there is a day-to-day variability in exposure, OSHA believes that many of the major sources of day to day variability can be moderated by diligent employer control.

OSHA also disagrees with AIA's contention that the appropriate legal test for technological feasibility would

assure that all employers may be 95 percent confident that an OSHA inspector will not measure an over exposure based on one day's sampling. There is nothing in the Act that would support such a test. No court that has reviewed OSHA's feasibility determinations has suggested such a test. In fact, the District of Columbia Court of Appeals has stated in pre-enforcement review that the court would not expect OSHA to prove the standard *certainly* feasible for *all* firms at *all* times in *all* jobs. [United Steel workers supra, 647 F. 2d 1270]. However, applying AIA's definition of feasibility would require a feasibility level that would give employers virtually that level of assurance (i.e., 95 percent versus 100 percent). The Agency's experience in promulgating and enforcing the former asbestos standard and other health standards provides additional policy reasons to reject AIA's test for determining industrywide feasibility.

AIA's test for feasibility depends upon a static picture of exposure levels and patterns. But as stated above, all feasibility determinations are projections of future control results. OSHA appropriately has decided that higher levels will fall as experience in applying controls increases. OSHA also has projected that the mix of circumstances under which those measurements were derived will change under the new standard. The mere lowering of the PEL creates its own incentive effect of decreased exposures and will reduce exposure variability.

Other policy reasons argue against AIA's statistical formulation of feasibility. Most importantly, to give a 95-percent level of assurance to employers that an OSHA inspector will not find a measurement above the PEL would require OSHA to deny to *employees* the assurance that they will be protected by exposure levels that are achievable. For example, it can be calculated that a plant that exceeds the PEL 70 percent of the time has a 35 percent chance that OSHA will not sample above the PEL during a visit in which a single 8-hour TWA sample was taken. AIA's data showed that *all* operations in the asbestos cement pipe industry can achieve 0.2 f/cc more than 50 percent of the time. Setting a level above 0.2 f/cc would mean that employees would unnecessarily be allowed to be exposed to higher levels than are *now* being achieved, simply to increase the level of assurance that an OSHA inspector will not obtain a high sample on a one day inspection. Such a result would undermine employee protection and would be inconsistent with the policies of the OSH Act.

OSHA believes that employers can increase their assurance of not being unreasonably cited by implementing measures that would not expose employees to such increased risk. The employer can reduce the chances of citation by exercising diligence in applying available controls, by supervising the work habits and practices of employees, and by inspecting and maintaining systems in optimum condition. All of these measures will not only reduce employees' average exposures, but also will reduce their high exposures, and thus lower the probability of OSHA issuing a citation. Based on OSHA's experience in regulating other substances with notable day-to-day variability, such as coke oven emissions, OSHA is confident that employers can control a significant portion of such exposure changes.

Due to the nature of asbestos fibers, in some workplace operations, OSHA may measure on a day when exposures are above the PEL due to random exposure variations, even though the employers have installed and maintained engineering controls, instituted available work practices and conscientiously applied housekeeping measures that maintain exposures below the PEL most of the time. Therefore, where an employer can show, based on a series of measurements made pursuant to the sampling and analytic protocols set out in this standard, that the OSHA one-day measurement may be unrepresentatively high, OSHA may reinspect the workplace and measure the employees' exposure or may decide not to issue a citation, unless OSHA has reason to believe that there are circumstances within the employer's control to account for the high exposure measurement.

OSHA is not setting out specific "rebuttal" criteria in the standard that would bind OSHA always to reinspect and that would deny an employer the opportunity to contest citation only when certain specified criteria are met. One reason is that OSHA believes the informed judgment of the OSHA inspector is superior to a rule that would be based only on the number and result of the employer's measurements. Such a rule would not

accommodate the OSHA inspector's observations about the quality of the employer's sampling and analytic program and the asbestos control, housekeeping, and training programs which OSHA believes are equally important in showing *why* fluctuations occur.

OSHA believes, however, that an employer's demonstration that an inspector's one-day sample is unrepresentative, in most cases, should consist of a series of full-shift measurements of the exposure of the employee under consideration. These measurements should consist of all valid measurements of the employee under consideration taken within the last year and should show that on only relatively rare occasions could random fluctuations result in measured TWA concentrations above the PEL.

Where the OSHA inspection or other information shows that the employer's exposure control programs and equipment are broken or are poorly maintained, where housekeeping programs have not been instituted or are inadequate, or where training programs do not exist or do not meet the standard, it is likely that OSHA's one-day measurements accurately reflect high exposure conditions that are not due to random exposure fluctuations but that are the result of the inadequacies of the employer's protective program. Consequently, citation is appropriate in such circumstances and no reinspection will be performed regardless of the employer's past measurements results.

It should be noted that the calculations of probable overexposures referred to in the above discussion are based on data from measurements taken in 1983 and earlier. Evidence in the record shows a gradual decline in asbestos levels over the last 5 years although the same technology is being used (e.g., compare data on the fiber receiving process in Exhibit 84-442 against the more recent data in Exhibit. 225). OSHA anticipates that, in general, exposure levels and the probability of overexposures will decline as employers more conscientiously apply all the available controls and adopt whatever new technology may become available. In this regard OSHA points to a new technique for reducing dust during abatement activity. The details of which were submitted to OSHA after the record was closed (see CACOSH, Exhibit. 344-18). OSHA believes that even minor refinements of existing technology will help employers achieve lower asbestos dust levels and will demonstrate that the concern for possible unfair citations due to day to day variability is illusory.

Based on all these considerations, OSHA believes that AIA's concerns about the issuance of citations due to occasional excursions above the PEL, are greatly overstated.

Sampling Error

The second contention made by AIA is that the sampling and analytic method for monitoring asbestos is so imprecise at lower levels that employers cannot with confidence evaluate whether they are in compliance. As discussed in great detail in the measurement section, OSHA has determined that the revised phase contrast method set out in this standard can reliably measure asbestos exposures below the action level of 0.1 f/cc if the procedures and protocols set out in the appendix are conscientiously followed.

OSHA acknowledges, however, that this sampling and analytic method for measuring asbestos has the potential for error. OSHA, therefore, will add a value that is equivalent to the sampling and analytical error (SAE) of the method to the exposure level measured by an OSHA inspector and will not cite for overexposure unless the measurement exceeds the PEL plus the SAE. As discussed in the section on method of measurement, OSHA believes that the record supports retaining the former SAE of 25 percent [OSHA Industrial Hygiene Technical Manual, 1984, p. A-240; see discussion in method of measurement section, *infra*]. OSHA, therefore, will not cite an employer for overexposure unless the measured one-day's overexposure exceeds 0.25 f/cc -- that is, the PEL of 0.2 f/cc plus the SAE of 0.05 f/cc. Since the sampling and analytical error potential can also result in measurements that are *lower* than the actual concentrations, the application of the SAE always will give the benefit of the doubt to the employer and assume that actual concentrations are less by 25 percent of the measured results. OSHA believes this additional margin will add to

the assurance an employer has about his capability for compliance and will further reduce the possibility that he will be unfairly vulnerable to an OSHA citation.

OSHA has also required a number of practices that will standardize sample analysis. These include specifications of a procedure for analysis and laboratory quality control programs.

Summary

In summary, OSHA has determined that the 0.2 f/cc PEL is technologically feasible and will not result in an unfair issuance of a citation to the conscientious employer. OSHA's analysis of each affected industry sector is presented below. In this analysis, OSHA concentrated on the revised PEL of 0.2 f/cc. As stated above, most the comments received by the Agency agree that 0.5 f/cc is feasible. Some comments, including those of the AFL-CIO [Exhibit No.335], argued that a PEL of 0.1 f/cc is feasible, but most of the "best" plant exposure data indicate that average exposures at many stations (e.g., most dry mechanical operations) are in excess of 0.1 f/cc and cannot be reduced using current controls and practices.

Tables 11 and 12 summarize OSHA's findings concerning the feasibility of reducing worker exposures to below the 0.2 f/cc PEL. They show that over 99 percent of the affected employees in general industry are expected to be below the PEL. Exposures for over one-half of the affected employees in construction sectors could be reduced to that level. OSHA, therefore, has determined that it is feasible for most industry sectors to comply with the 0.2 f/cc PEL most of the time.

**TABLE 11. -- FEASIBILITY SUMMARY TABLE FOR GENERAL INDUSTRY: PROJECTION OF WORKERS EXPOSED BELOW AND ABOVE 0.2 F/CC FOLLOWING THE PROMULGATION OF THE STANDARD AND THE ADOPTION OF ENGINEERING CONTROLS AND WORK PRACTICES**

| Industry sector | Total No. of asbestos-exposed workers | Projected No. of workers exposed to asbestos levels below 0.2 f/cc | Projected No. of workers exposed to asbestos levels above 0.2 f/cc * |
|---|---|---|---|
| Primary manufacturing: | | | |
| A/C pipe | 512 | 409 | 103 |
| A/C sheet | 203 | 150 | 53 |
| Textiles | 414 | 123 | 290 |
| Floor tile | 276 | 276 | 0 |
| Coatings | 1,327 | 1,327 | 0 |
| Friction | 5,104 | 4,777 | 327 |
| Paper | 387 | 387 | 0 |
| Gaskets | 315 | 315 | 0 |
| Plastics | 324 | 278 | 46 |
| Subtotal | 8,861 | 8,042 | 819 |
| Secondary manufacturing: | | | |
| A/C sheet | 345 | 230 | 115 |

| | | | |
|---|---|---|---|
| Textiles | 172 | 143 | 29 |
| Friction | 1,504 | 1,003 | 501 |
| Gaskets | 9,972 | 9,972 | 0 |
| Plastics | 2,450 | 2,450 | 0 |
| Auto remanufacturing | 4,750 | 4,750 | 0 |
| Subtotal | 19,193 | 18,548 | 645 |
| Service and repair: | | | |
| Ship repair | 15,000 | 12,434 | 2,566 |
| Auto repair | 526,998 | 526,996 | 0 |
| Subtotal | 541,998 | 539,434 | 2,566 |
| Grand totals | 570,052 | 566,022 | 4,030 |

* Estimates derived from RTI survey data presented in Appendix D of the Final Regulatory Impact and Regulatory Flexibility Analysis (RIA).

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

**TABLE 12. -- FEASIBILITY SUMMARY TABLE FOR CONSTRUCTION: PROJECTION OF WORKERS EXPOSED BELOW AND ABOVE 0.2 F/CC FOLLOWING THE PROMULGATION OF THE STANDARD AND THE ADOPTION OF ENGINEERING CONTROLS AND WORK PRACTICES**

| Industry sector | Total No. of asbestos-exposed workers n1 | Projected No. of workers exposed to asbestos levels below 0.2 f/cc | Projected No. of workers exposed to asbestos levels above 0.2 f/cc n1 |
|---|---|---|---|
| New construction | 29,320 | 27,115 | 2,205 |
| Abatement | 81,365 | 13,560 | 67,805 |
| Demolition | 24,455 | 3,980 | 20,475 |
| Renovation | 133,700 | 51,300 | 82,400 |
| Routine maintenance in commercial/ residential building | 217,745 | 124,155 | 93,590 |
| Routine maintenance in general industry | 259,643 | 175,053 | 84,590 |
| Total | 764,228 | 395,163 | 351,065 |

n1 Excludes small short duration jobs with negligible exposures.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

Primary Manufacturing

The production of the primary asbestos products can be divided into receiving (unloading, transporting, and storing the raw asbestos fiber), fiber introduction, and processing (mixing, drying, and finishing). The best available control technology consists of a combination of extensive local exhaust ventilation and a diligently enforced, comprehensive program of work practices and housekeeping. The automatic bag opening equipment, which is used in some sectors, is an example of the technology currently available to minimize asbestos exposures during fiber introduction. In several sectors, some finishing processes are completed with the use of water spray to reduce airborne levels of asbestos.

Two manufacturing steps that all primary asbestos product manufacturers have in common are the receipt of asbestos shipments and the introduction of asbestos fiber into the process. Due to the universal use of these steps throughout the industry as well as the large potential for release of asbestos fibers, a qualitative discussion of these steps is presented below.

Raw asbestos is shipped to manufacturers via railcar or truck. Manufacturers usually receive from 25 to 50 bundles of 100-pound bags of raw asbestos fibers.The packaging of the asbestos varies, but loose fibers or fibers pressed into bricks are usually wrapped in plastic or Kraft (TM) paper bags. These bags are transported on pallets that are constructed with high shear-resistant glue to prevent movement during shipping and handling. The entire bundle of asbestos bags is often shrink-wrapped with plastic to further reduce the potential for fiber release.

When trucks or railcars arrive at the plant, they are opened and examined for damaged bags. If any major damage is found, the entire shipment is returned to the supplier. Any minor damage is repaired by vacuuming the spilled fiber and sealing the broken bag with tape. The pallets are removed from the railcar or trailer by forklift and are stacked in the storage area [Exhibit 335].

Due to prudent work practices and recent improvements in the packaging of asbestos fibers, OSHA has determined that it is feasible for primary manufacturers of asbestos products to receive and store shipments of asbestos without experiencing exposures above the PEL of 0.2 f/cc. According to Marsden Hutchins of Quin-T Corporation:

. . . fiber as now received lends itself to dust-free storage. Care in handling to avoid and/or clean up after accidental bag breaks makes this a relatively trouble-free area. [Exhibit 91-16, Section J, p. 17.]

Data provided in Dr. Gordon Bragg's feasibility report [Exhibit 235-A, Table III] indicate that an A/C pipe manufacturing plant with the best available technology and stringent work practices experienced a mean TWA of 0.03 f/cc during the reception and storage of asbestos shipments.

In addition to receiving and storing asbestos fibers, all primary manufacturers of asbestos products share the fiber introduction step. OSHA has concluded that it is feasible for this processing step to be completed with exposures below 0.2 f/cc. This conclusion is supported by data presented by the Research Triangle Institute (RTI) from its 1984 industry survey. In the RTI survey, exposures during fiber introduction ranged from 0.07 f/cc to 0.2 f/cc [see Appendix C of the Regulatory Impact Analysis].

The introduction of asbestos fibers to the manufacturing process begins with the transportation by forklift of the pallets of asbestos bags to the head of the production line. There, depending on the product line, the bags

are sent either unopened to the mixing stage or are cut open and the asbestos is dumped onto a conveyor to be carried to the mixing stage. When unopened bags of asbestos enter into the process, exposure levels are not a problem in the introduction step. In written testimony, Mr. Hutchins indicated that only 5 percent of Quin-T Corporation's production of asbestos paper and gaskets required the asbestos paper bags to be opened prior to mixing [Exhibit No. 91-16, Section J, p. 5]. Asbestos bags packaged in polyethylene are not always opened in the production of asbestos/vinyl flooring or asbestos-reinforced plastics.

When the bags must be opened, either automated or manual debagging operations are used. Exposures at automated debagging stations have been measured to be less than 0.2 f/cc [Exhibit 235A, p. 101]. It has also been demonstrated that manual debagging operations have had exposures below the proposed PEL of 0.2 f/cc. Dr. Bragg reported an 8-hour exposure of 0.07 f/cc for the operator at a manual debagging station. He also cited an article by First and Love in which exposures at a manual debagging operation were measured to be 0.047 f/cc or lower for seven samples [Exhibit 235-A, p. 101]. Thus, OSHA has determined that it is feasible for both manual and automated debagging operations to reach exposures below the proposed PEL.

Asbestos-Cement Pipe

Data submitted to the record indicate the ability of most work stations at well-controlled A/C pipe plants to reach levels below the PEL of 0.2 f/cc except during the coupling cut-off operations. The basic steps in the manufacture of A/C pipe are fiber introduction, materials mixing, pipe forming, curing, and finishing. To reduce exposures throughout the A/C pipe manufacturing process, work practices and engineering controls have been applied to work stations as described below.

Following fiber introduction, the asbestos is carried through various processing steps by conveyor belt. The use of pneumatic conveying systems kept under negative pressure, along with local hood exhaust dust-control systems, has virtually eliminated the possibility of exposure at this stage of processing.

While being conveyed through the processing steps, the fiber is fluffed and blended and then thoroughly mixed with specific amounts of Portland cement, silica sand, and reprocessed scrap. The processing and dry mixing of the ingredients take place automatically in closed blending tanks which are maintained under a slight negative pressure by local exhaust ventilation to minimize worker exposure.

Following the dry mixing process, water is added and the resultant slurry is processed through a pipe making machine known as a "wet machine." The wet machine deposits a homogeneous mixture of the slurry in the form of a thin lamination onto a conveyor. The layer of wet asbestos cement is then conveyed to the press section of the wet machine where it is continuously wrapped around a long steel cylinder until the proper size of pipe of formed. This continuous wrapping process is carried out under high pressure which forces each new lamination to bind with the previously wrapped layer.

After the wrapping process is complete, the formed pipe is removed from the press section of the wet machine and processed through primary curing ovens to allow the cement to attain an initial set. Later, the semi-hardened pipe is placed in an autoclave where it is subjected to a high-pressure steam environment which forces the cement and silica in the pipe to undergo an accelerated cure. At this stage of the process, the asbestos fibers in the pipe become bound in a cement mixture [Exhibit 91-16, Section H].

After autoclaving, cured pipe sections are cut to uniform lengths, machined in a variety of ways (sawing, lathing, drilling), and outfitted with a coupling. The finished pipe is inspected and each section of pipe to be used for conveying water under pressure is tested hydrostatically.

A/C pipe coupling is also produced in these plants. The coupling is manufactured and then cut into smaller sections for use in pipe connection. The repetitious cutting of the coupling lengths causes high asbestos

exposures. For this cutoff operation and other finishing processes like lathing and drilling, the use of custom-engineered hoods, local exhaust systems, wet sawing, and special single-point cutting tools has reduced exposure levels. Exhaust air is filtered into baghouses and the collected dust is typically removed in closed containers for recycling or disposal.

As a good housekeeping practice, measures are taken during the pipe formation process to clean up spills of slurry that could dry and become a source of emissions. These housekeeping practices include the use of wet vacuum machines and squeegees instead of brooms for cleaning floors.

The exposure data for A/C pipe used in OSHA's feasibility determination are summarized in Table 13. The average exposures at all of the processes are less than 0.15 f/cc. Among the highest exposures are those for dry mechanical operations; however, these also average less than 0.15 f/cc. Other data submitted show that some dry material operations may have difficulty achieving the new PEL some of the time. For example, the data presented by Dr. Bragg show that exposures at coupling cutoff operations in an A/C pipe plant are the highest, averaging 0.369 f/cc [Exhibit 312-A, Section H, Table II]. The high exposures during the coupling cutoff operation are also consistent with data submitted by the International Brotherhood of Boilermakers, Iron Ship Builders, Blacksmiths, Forgers and Helpers (AFL-CIO). These data show that out of 82 exposure readings taken at a CertainTeed Corporation plant, only 2 (both for coupling cutoff operations) exceeded 0.2 f/cc [Exhibit 225]. OSHA believes, however, that most dry mechanical operations can achieve the new PEL.

**TABLE 13. -- EXPOSURES FOR A/C PIPE MANUFACTURERS**

| Job classification/process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | No. of observations | Source of data |
|---|---|---|---|---|
| Fiber introduction | 0.136 | 0.089 | 83 | OHSA MIS n1 |
| Wet mechanical | .097 | .094 | 87 | Do. n1 |
| Dry mechanical | .134 | .145 | 124 | Do. n1 |
| Other operations | .077 | .100 | 240 | Do. n1 |

n1 Unpublished compliance data from the Management Information System data base for 1979-1984.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

Based on these data, OSHA concludes that the 0.2 f/cc PEL is feasible for all operations at A/C pipe plants using current technology except for coupling cutoff where respirators will have to be used to supplement engineering controls. OSHA, therefore, considers is feasible for the other operations, particularly mixing and conveying of materials within the plant, to reach exposures below 0.2 f/cc.

A/C Sheet

The manufacturing process of A/C sheet is similar in many aspects to that of A/C pipe. Unlike A/C pipe manufacturing, however, OSHA was unable to find data to indicate that exposures at even the "best controlled" A/C sheet plants are below 0.2 f/cc. The mean exposures at most stations, for which OSHA has data, are approximately 0.5 f/cc. Based on the analysis by Dr. Bragg, however, OSHA believes that A/C sheet manufacturers are not using the best available techniques to control asbestos dust.

The data indicate that fiber is less well-controlled in the sheet manufacturing environment than the cement pipe

operation. For example, we would expect that it is possible to control exposures at the fiber introduction stage to values similar to those found in asbestos cement pipe. As a result, the data . . . does not represent the best available technology in our opinion and the improved use of local exhaust ventilation, wet processing and good housekeeping should be capable of reducing exposure levels to values typical of the A/C pipe industry. However, the sanding operation is unique to sheet and there may be a serious control problem for this operation at a PEL of 0.5 "f/c.c." or lower. [Exhibit 235-A, pp. 65-69.]

In addition, the AFL-CIO attributed the higher exposure levels in the asbestos-sheet industry to the failure of this industry to use available controls to reduce exposures [Exhibit 335, p. 39]. Thus, OSHA has determined that by using the same control technology that is currently being used in the A/C pipe sector, it will be feasible for the A/C sheet sector to comply with a 0.2 f/cc PEL. However, in sanding, which is unique to A/C sheet, achieving the new PEL will require the use of respirators.

As previously described, OSHA has determined that fiber introduction for all primary manufacturing processes, including A/C sheet, can be performed with exposures below the PEL of 0.2 f/cc. The dry and wet mixing slages of A/C sheet production are virtually the same as the mixing steps of A/C pipe production. With the use of conveying systems kept under negative pressure, local exhaust systems, and fully enclosed exhaust mixers, it is possible for exposures to be kept under 0.2 f/cc during this phase of production.

The advanced processing steps of A/C sheet manufacture are also similar to those of A/C pipe. Following wet mixing, the slurry flows into vats and is deposited on rotating cylinder molds where the appropriate thickness is formed. The sheet is passed under embossing rolls or hydraulic presses and is then removed from the press for curing by heated air or steam-heated autoclaves. After curing, the A/C sheet undergoes a variety of finishing operations. The highest and most difficult exposures to control occur during these mechanical finishing operations, which is also true for A/C pipe manufacturing. It is possible to reduce worker exposures to below 0.2 f/cc in finishing operations with the use of local exhaust ventilation and tools equipped with exhaust systems or wet spray devices. OSHA, however, has found no evidence indicating it is feasible to lower exposure levels to below 0.2 f/cc during the sanding of A/C sheet without the use of respirators.

As in A/C pipe production, OSHA recognizes that it is difficult to reduce exposures during the cutting operation to below 2.0 f/cc. Technological improvements demonstrated in construction activities, however, have led to reduced exposures during cutting to below the PEL of 0.2 f/cc. OSHA believes that there is a strong likelihood that similar developments will occur in the manufacture of A/C pipe and sheet and in the production of other primary asbestos products. Other innovations, such as shrouded tools used in field cutting, might be applied on a larger scale to current cutting practices in factories. As suggested by Dr. Bragg, the local exhaust ventilation and good housekeeping used in the processing steps of A/C pipe could be successfully applied to A/C sheet processing. Mr. Alfred Netter of Supradur Manufacturing Corporation recognized in his written testimony the importance of good househeeping when he stated the following:

Work practices -- merely keeping the floors clean -- reduce greatly the amount of dust in the air created by the movement of equipment. When used properly, this and other housekeeping chores can provide very effective dust control. [Exhibit 91-16, Section I, p. 9].

OSHA, therefore, concludes that with a combination of engineering controls and work practices it will be feasible for this sector to comply with the 0.2 f/cc PEL for all operations except sanding, where supplemental respiratory protection will be used to achieve the PEL.

Friction Products

Asbestos friction products include drum brake linings, disc brake pads, and clutch facings for automobiles, as well as materials for industrial and commercial applications where motion must be controlled. Although each

of these products is manufactured by a unique process, the basic order is fiber introduction, wet or dry mixing of the asbestos with other ingredients, and production forming, curing, and finishing.

OSHA has determined that it is feasible to achieve exposure levels below 0.2 f/cc during all operations except grinding, by using engineering controls and work practices. For grinding, supplemental respiratory protection will be required.

Friction products are molded using a wet-mix or a dry-mix process or a combination of the two methods. Dry-mixing is generally used for disc brake pads and brake blocks, whereas wet-mixing generally is used to mold drum brake linings and clutch plates used in automatic transmissions. Compared with the slurry processing for drum brake linings, exposures tend to be higher during the processing of the more friable dry-mix used to make disc brake pads. Both dry-mix and wet-mix processes are used in the manufacture of clutch facing.These steps of fiber introduction and mixing closely resemble those of other primary manufacturing processes (e.g., A/C pipe).

Following mixing, the dry mix is fed through a compression molder and the wet mix through an extruder. Then, formed strips are cut and bent into various widths and lengths. Dry-mixed formulations are transferred to pressing molds where slabs are formed, sometimes after a pre-heating step. The slabs are hot pressed, are sawed into specific parts, and are then sent to a curing oven. Following curing, the parts undergo finishing steps to produce the final product.These steps include sawing, grinding, drilling, tapping, and boring.

In the friction products industry, finishing operations generate the greatest quantity of emissions, with as much as 30 percent of the asbestos in the products being ground away as dust. The Friction Materials Standards Institute claimed that a 0.2 f/cc TWA PEL is not feasible [Exhibit 90-180]. OSHA has determined, however, that although there are some operations for which the 0.2 f/cc PEL is not yet feasible, it is feasible for most operations to comply with the 0.2 f/cc PEL using engineering controls and work practices. This feasibility determination is based on exposure data obtained during an RTI site visit to the Raymark plant in Stratford, Connecticut [see Appendix B of RIA].

The Raymark plant is a primary producer of friction materials and sheet gasketing and is the second largest producer of friction products of the plants in the RTI survey. The exposure data reveal that most of the workers involved in the manufacturing of friction products are exposed to less than 0.2 f/cc of asbestos. Exposures for the 15 employees involved with fiber introduction for asbestos friction materials ranged from 0.03 f/cc to 0.21 f/cc, which is similar to the exposure data in A/C pipe manufacture. OSHA, therefore, believes that 0.2 f/cc PEL is feasible for fiber introduction. Exposures for the 28 workers involved in wet mechanical operations, in which the various products are prepared for curing, ranged from non-detectable to 0.3 f/cc, with most appearing to be below 0.2 f/cc. OSHA, therefore, concludes that it is also feasible for those activities to comply with a 0.2 f/cc PEL. This determination agrees with the hearing testimony by Dr. Franklin Mirer of the United Auto Workers (UAW) who ascertained that current technology has the ability to lower exposure levels for these practices to below 0.2 f/cc [Hearing Transcript of July 2, 1984, p. 94].

Exposures for the employees involved in dry mechanical operations, however, ranged from 0.07 f/cc to 1.7 f/cc. About one-third of the workers were regularly exposed to levels above 0.2 f/cc during the grinding of drum brake linings and the pressing and machining of clutch facings. The difficulty of controlling exposures for these dry mechanical operations is consistent with data presented by Dr. Bragg which show that exposures at many of the dry mechanical operations average between 0.3 f/cc and 0.7 f/cc. Dr. Bragg referred to the impracticality of using wet methods during these particular practices because of their detrimental effect on the final friction product [Exhibit 235-A, p. 79]. Dr. Mirer of UAW acknowledged the high exposures during the manufacture of friction products and suggested the use of substitute materials [July 2, 1984, Transcript, p. 92]. The AFL-CIO also has stated that the production of asbestos friction products is a problem area in terms of exposures [Exhibit 335, p. 44]. Thus, it appears that supplemental respiratory protection will be required to

comply with the 0.2 f/cc PEL during grinding operations.

Textiles

Asbestos textiles are manufactured by either wet or dry processing. Not all asbestos textile products can be made by the wet process because chemicals used in the wet process alter the characteristics of the fiber making it undesirable for some applications. Likewise, although some operations of the conventional "dry" method could be run using dampened fibers, some fiber qualities required by the final textile product exclude the use of dampening techniques.

In the dry process, the asbestos fiber is debagged and dry blended. Cotton, rayon, or other natural or synthetic fibers can be added to impart strength and other characteristics. Following the standard textile processes, the carding operation, which is one of the problem areas for exposures, combs the fiber mix into a web of parallel fibers which is then divided into strips known as roving. The roving is spun and twisted to produce single or plied asbestos yarns. Due to the high velocity of the spinning operation, this processing step has been a source of high exposures. The roving can be dampened by wet rollers or mist spray prior to spinning to lower the exposures. During the spinning and other processing steps, however, the strands often break and release asbestos dust as the ends whip around the spindles. Yarns are coated to produce thread, and are braided into cord, rope, or tubing. Depending on the characteristics of the final product, a damp or dry loom can be used during weaving operations.

In the wet process, the asbestos fibers are mixed with water and chemicals. The resulting slurry is extruded directly into strands.This method eliminates the carding operation, a major source of emissions during the conventional process. The strands are then spun and go through the subsequent processing steps which are similar to those of the conventional method. According to some of the developers of wet processing equipment, the balance of the processing steps are performed wet or with the fibers bound, thereby reducing exposures [Exhibit 323].

Local exhaust ventilation is the primary engineering control used to reduce levels of asbestos dust in plants using dry methods to produce asbestos textiles. It is normally provided at the bag opening and fiber introduction stages, and during the willowing and blending, carding, and winding operations. Dust control measures are particularly stringent in plants that blend cotton into the fabric, due to health hazards associated with exposure to cotton dust.

As none of the four post-1980 studies on wet operations at primary textile plants (2 from RTI survey and 2 from OSHA MIS files) show exposures in excess of 0.1 f/cc, OSHA has determined that it is feasible for these operations to comply with a 0.2 f/cc PEL. Other data submitted by the Amalgamated Clothing and Textile Workers Union (ACTWU) [Exhibit 260-A] and obtained by RTI during a site visit to a Raymark Corp. plant [Appendix B of the RIA] show that exposures during dry operations generally exceed 0.2 f/cc. Consequently, OSHA does not believe it is feasible for the dry operations of carding and spinning to comply with the 0.2 f/cc without the supplemental use of respirators. Data in the Bragg report [Exhibit 235-A, Tables VI and XVII] also indicate that these operations will have difficulty achieving average exposures below 0.2 f/cc without the use of respiratory protection.

The AFL-CIO also believes that using dry methods in the manufacture of asbestos textiles is a problem area and that some operations will have difficulty in achieving the PEL [Exhibit 335, pp. 44]. The AFL-CIO has stated that it is feasible for the textile industry to comply by switching to wet processing. OSHA has determined, however, that this is not a viable option in most cases because wet processing changes the nature of the textile. RM Industrial Products Company, Inc., one of the suppliers of wet processing technology, acknowledges that the wet process "is not a complete substitute for conventionally prepared asbestos yarn products" [Exhibit 323, p. 3].

OSHA's experience with cotton textile operations has shown that a careful work practice and housekeeping program is effective in reducing cotton dust levels in the plant. Dry cotton textiles operations are similar to asbestos yarn manufacturing and OSHA believes the adoption of the controls developed for cotton dust, such as frequent vacuuming of floors and machine parts, can be used successfully in asbestos textile manufactures. OSHA expects that dry asbestos textile manufacturing will use the latest control strategies available, and should be able to reduce worker exposure to below current levels. For carding and spinning operations in dry mechanical asbestos textile manufacturing, respirators will be used to achieve the PEL.

Vinyl/Asbestos Floor Tile

During the manufacture of vinyl/asbestos floor tile, opened paper or unopened plastic bags of raw asbestos fibers are dumped into a mixer along with other dry ingredients. The mixer combines the ingredients into a hot plastic mass that binds the asbestos fibers, thus reducing the potential for exposure. The hot mix is dumped onto a conveyor and transported under negative pressure to a two-roll mill. The mill presses the plastic into a continuous slab which is passed through a series of calender rolls to achieve the desired thickness. The warm sheet next passes through an embosser which imparts a surface design if desired. After cooling and waxing, the sheet is cut to size, inspected, and packaged for shipment. Cutting scraps are returned to the mixer for recovery.

Local exhaust ventilation is provided at stations such as fiber introduction and cutting which potentially may have high exposures. Mottling granulation and scrap grinding may be isolated in enclosed rooms. Housekeeping is performed continuously to clean up spilled dry material.

Table 14 summarizes the exposure data that forms the basis for OSHA's feasibility determination for vinyl/asbestos floor tile. As shown in the table, the reported exposures at each of the three jobs were less than 0.2 f/cc. OSHA, therefore, has determined that it is feasible for this sector to comply with the 0.2 f/cc PEL. This determination is consistent with 1984 data submitted by Dr. Bragg, which showed that for operations other than fiber introduction, exposures range from 0.01 f/cc to 0.2 f/cc.

**TABLE 14. -- EXPOSURE DATA FOR THE MANUFACTURER OF VINYL ASBESTOS FLOOR TILE**

| Job classification/ process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | No. of observa-tions |
|---|---|---|---|
| Fiber introduction n1 | n2 0.014 | 0.022 | 14 |
| Dry mechanical n3 | n4 .105 | n4 .095 | n5 15 |
| Other n3 | n4 .105 | n4 .095 | n5 72 |

n1 OSHA MIS period 1979-1984.

n2 All observations were less than 0.1 f/cc.

n3 RTI site visit to Amtico Flooring, Lawrenceville, N.J.

n4 Ranges were from 0.01 f/cc to 0.2 f/cc, and the means and standard deviations were based on an assumption of a symmetrical distribution.

n5 Number of employees who were represented by the average exposures was used as data on the number of samples were not provided.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

Gasket and Packings

Asbestos-based gaskets and packings are used to prevent the leakage of fluids in process equipment. Asbestos is an effective sealant because it generally does not react with machine fluids and is heat resistant. In the manufacture of gaskets, raw asbestos fibers are either introduced by emptying bags or are added in unopened pulpable bags. The fibers are them mixed wet or dry, with fillers and bonding materials. During mixing, the raw fibers are encapsulated by binders and solvents which reduce the potential for fiber release throughout the rest of the manufacturing process. The mixture is rolled into sheets which may be further processed on-site or may be packaged for shipment to secondary fabricators or to suppliers of replacement parts for industrial equipment.

Asbestos-based packings can be manufactured by a number of processes. The most common production method involves the impregnation of dry yarn with a lubricant. The coated yarns then are braided into continuous lengths and calendered to specific sizes and shapes.

Exposure data upon which OSHA based its feasibility determination were obrtained by RTI during a site visit to the Stratford, Connecticut, plant of the Raymark Corp. [see Appendix B of the RIA] and from two facilities responding to the RTI survey [see Appendix C of the RIA]. All three plants reported exposures at various work stations (e.g., wet mechanical, dry mechanical, etc.), other than those involved in fiber introduction and milling, to be at levels below 0.2 f/cc. The level of exposure during braiding and twisting of treated asbestos yarn is controlled by local exhaust ventilation and is supplemented by general control measures, including dilution ventilation and systematic cleaning. In addition to wet mixing operations, sheet and gasket cutting causes very little generation of airborne fibers. Thus, OSHA has determined that it is feasible for these operations to comply with the 0.2 f/cc PEL.

Fiber introduction levels at these plants were reported to be in excess of 0.2 f/cc, with exposures at two of the plants reported to be in excess of 0.75 f/cc. OSHA, however, believes that these plants did not utilize the best available technology and that it is feasible for fiber introduction stations to comply with the 0.2 f/cc PEL. This determination of feasibility was made because the fiber introduction process in the gasket and packing industry is similar to that in other primary manufacturing industries where exposures are currently below 0.2 f/cc (e.g., A/C pipe and floor tile).

Asbestos Paper

In the manufacture of asbestos paper, raw asbestos fiber is most often introduced in unopened pulpable bags, although for some types of paper the fiber is dumped from the bags. In order to decrease exposures in cases where the fiber is dumped from the bags, asbestos may be obtained in noncompressed pulpable paper bags so that bags may merely be slit and added directly to the mixer, where it is immediately wetted. The use of batch sizes requiring whole bags of asbestos (rather than 1/3 or 1/2 bags) can further minimize asbestos handling and the potential for dust generation. As in other manufacturing processes, the asbestos fiber is carried under negative pressure by conveyor to a mixer. There, the fiber is wet-mixed with paper stock, binder, and other ingredients. The stock slurry flows into the papermaking machine and forms a sheet with a solids content of less than 5 percent. Although the moisture content is reduced greatly during transit through the paper machine, the wet nature of the material largely precludes the release of airborne asbestos.

The steam-heated rolls in the drying section typically have canopy hoods and exhausts to remove water vapor

and heat. This type of hooding and exhaust augments the general ventilation in the area and aids in removing asbestos particulates released during the drying operation.

Local exhausts, area hoods, and central exhaust collection systems represent the normal control measures used to minimize asbestos exposure at the slitting and calendering stages. Housekeeping is also critical here.

The rewinding step involves the bulk packaging of paper products on spools, reels, or beams from larger rolls. The operation is dry, and the hoods and local exhaust may be used as dust-control measures during these operations.

Although airborne asbestos fibers are generated throughout the entire manufacturing process, exposure levels vary widely depending on the asbestos content of the product. If comparable control systems are used, airborne fiber levels at a plant producing a gasket paper containing 90 percent asbestos are normally higher than levels at a plant producing specialty papers, or beverage or pharmaceutical filters containing 10 percent asbestos. Emissions also can vary depending on the physical process itself. Some plants perform fiber introduction and stock preparation (i.e., wet-mixing) as separate operations and others combine these into a single operation.

Housekeeping in the stock preparation area represents a crucial control measure for minimizing operator exposure to asbestos. Central vacuum-cleaning systems and mechanical floor-sweeper-vacuum units often are used during these operations.

OSHA based its feasibility determination on data provided by the Quin-T Corporation's plant in Tilton, New Hampshire. As shown it Table 15, these data are the most recent and comprehensive available for asbestos paper production. The mean exposures for all areas were less than the 0.2 f/cc PEL. OSHA concludes that it is feasible for this industry to comply with the 0.2 f/cc PEL. This position is consistent with RTI's findings for two paper firms that responded to their survey [see Appendix C of the RIA].

**TABLE 15. -- WORKER EXPOSURE FOR ASBESTOS PAPER MANUFACTURE**

| Job classification process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | No. of observa-tions |
|---|---|---|---|
| Fiber introduction n1 | 0.05 | 0.04 | 6 |
| Wet mechanical | .09 | .10 | 22 |
| Day mechanical n2 | .14 | .12 | 17 |
| Other | .08 | .11 | 25 |

n1 These data omit one outlier of 0.56 f/cc. As all of other data were 0.1 f/cc or below, OSHA assumed that this observation was due to an equipment problem.

n2 These data omit one outlier of 1.3 f/cc. As all of the other observations were 3.4 f/cc or below, OSHA assumed that this observation was also due to an equipment problem.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

Coating and Sealants

Many types of coatings and sealants have asbestos added as a reinforcing agent and property modifier. In most instances, the final product is asphalt-based and is used for roof coatings and automobile undercoatings.

The production processes for surface coatings and sealants are similar. In the production of these products, the fibers must be opened, or fluffed, as much as possible. Thus, a fluffing operation to agitate the fibers follows the fiber introduction stage. Dry ingredients are then mixed with the opened fibers followed by the addition of the asphalt or coal tar and solvents. After mixing, the fiber is encapsulated and little asbestos dust is generated. The coatings and sealant blends are then packaged and prepared for shipment.

For this industry, the major potential sources of airborne fibrous exposures precede the mixing operation due to accidential spills during fiber receiving and storing, and from emissions during fiber introduction. As in the manufacture of other asbestos products, OSHA has determined that it is feasible to perform these tasks with with exposures below 0.2 f/cc. The fluffing and mixing operations are kept under negative pressure, and housekeeping around these operations is continuous.

OSHA based its feasibility determination on data provided by the Monsey Products Company for the firm's Indianapolis, Garland, and Rockhill plants. These data, which are the most comprehensive available on coating facilities that have good work practices, n1 are summarized in Table 16.

n1 Data provided indicate that the four other plants did not appear to have the same quality of control technology [Exhibit 312A, Section L].

**TABLE 16. -- WORKER EXPOSURES DURING THE MANUFACTURE OF ASBESTOS COATINGS AND SEALANTS**

| Job classification process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | No. of observations |
|---|---|---|---|
| Fiber introduction * | 0.13 | 0.15 | 34 |
| Other | .04 | .05 | 13 |

* These data omit one outlier of 1.03 f/cc. As all of the other observations were below 0.7 f/cc, with most below 0.15 f/cc, OSHA assumed that this observation was due to an equipment problems.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

As indicated in the table, the average exposures for these work stations were less than 0.2 f/cc at both stations in the coating plants. OSHA, therefore, has determined that it is feasible for this industry sector to comply with the 0.2 f/cc PEL. This feasibility determination is consistent with the limited 1983 exposure data submitted by Dr. Bragg as well as with the position of the AFL-CIO [Exhibit 335, p. 41].

Asbestos-Reinforced Plastics

Due to their heat-resistant qualities, asbestos-reinforced plastics are used in the electrical, electronic, automotive, and printing industries. In the manufacture of these plastics, raw asbestos fiber is introduced and

dry mixed with catalysts and other additives. The mixture is heated into a resin in the form of pellet or powder preform. The preform may be further processed onsite or packaged and sold to other manufacturers. Based on the information provided in a 1984 report prepared by Versar for EPA [Exhibit 333], primary manufacturers process only 30 percent of the preform that they produce. The remaining 70 percent is shipped to secondary manufacturers who shape and finish the asbestos-based plastic resin. In the shaping process of the final plastic product, the preform is rolled, stamped, pressed, or molded.The product is then cured in an isolated area with a ventilation system. The strength and stiffness characteristics of the final product are partially controlled by the time and temperature conditions during curing.

OSHA's feasibility determination for asbestos reinforced plastics is based in part upon data obtained from two plants surveyed by RTI and from three OSHA MIS reports. These data are summarized in Table 17.

**TABLE 17. -- WORKER EXPOSURES DURING THE MANUFACTURE OF ASBESTOS-REINFORCED PLASTICS**

| Job classification/process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | No. of observations | Source of data. |
|---|---|---|---|---|
| Introduction | 0.1 | N/A | n1 34 | RTI survey. n2 |
| Introduction | N/D | 0.001 | 3 | OSHA MIS. |
| Wet mechanical | 0.01 | N/A | n1 2 | RTI survey. n3 |
| Dry mechanical | 0.14-0.57 | N/A | n1 35 | RTI survey. n4 |
| Other | 0.04 | 0.047 | 13 | OSHA MIS. |

n1 Number of employees who were represented by average exposure was used since data on the number of samples were not given.

n2 Identified as plant "1."

n3 Identified as plant "g."

n4 Identified as plant "h."

N/A=Not available.

N/D=Not detectable.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

Since these data, especially the MIS data, do not represent plants using the best controls, OSHA's determination is also based upon the technologies currently available in the other primary sectors.

The data indicate that exposures at the fiber introduction and wet mechanical processes in this industry are below 0.2 f/cc and that the problem exposure areas during the manufacture of the plastics appear to be in dry finishing operations. These operations are similar to dry mechanical operations in other asbestos products manufacturing industries and include grinding and sanding, which OSHA has determined may not be feasible to achieve exposure levels below 0.2 f/cc without the use of respirators. Thus, OSHA believes it is

technologically feasible for most operations to achieve a 0.2 f/cc TWA, but that respirators will be required during grinding and sanding.

Secondary Manufacturing

Secondary manufacturers modify or fabricate primary asbestos products to yield final products (e.g., impregnated roofing felt) or intermediate products (e.g., asbestos textiles made into fire-resistant clothing). Receiving and handling these primary products do not pose exposure problems. Compared with the primary processing steps of fiber introduction, mixing, and conveying loose fibers, secondary fabrication takes place in a more controllable environment. Exposures occur in this sector when stable asbestos products are altered by dry mechanical operations that release encapsulated fibers into the air. As supported by data, exposures resulting from these dry mechanical finishing operations can be controlled by shrouded tools and by wet methods in some cases. As with primary manufacturing, OSHA has determined that it is feasible for these industries to comply with the 0.2 f/cc PEL in all operations with the exception of some maintenance activities (e.g., repairing or servicing the controls that protect the other workers) and a limited number of dry mechanical operations. The basis for this determination is presented below.

A/C Sheet

The secondary manufacturing of A/C sheet prepares the product for specific installation requirements. This fabrication requires the same dry mechanical processes that were described for primary manufacturing processes, such as sawing, drilling, routing, beveling, and sanding. Some of the firms that responded to RTI's survey reported using wet spray during sawing and routing. As in other processes, tools are equipped with local exhaust systems. High exposures are likely to remain a problem during sanding, which is unique to A/C sheet production.

OSHA's determination of feasibility in this sector is based on data obtained in response to the RTI survey (see Table 18). As all of the exposures shown in the table are below 0.15 f/cc, and because the 1983 data [Exhibit 235-A, Table XXII] for a secondary user of A/C sheet are also all below 0.15 f/cc, OSHA has determined that it is feasible for this sector to comply with the 0.2 f/cc PEL, except for sanding, where respirators will be required.

**TABLE 18. -- WORKER EXPOSURES DURING SECONDARY MANUFACTURE OF ASBESTOS CEMENT SHEET**

| Plant designation | Annual production | Job classification | 8-hr TWA exposure levels (f/cc) | No. of workers at oper-ations |
|---|---|---|---|---|
| m | >1 million lbs | Dry mechanical | N/D 0.14 | 20 |
| p | 7,000 sq yds | Wet mechanical | 0.10 | 1 |
| l | N/A | Wet mechanical | N/D | 5 |
| q | 21,000 sq yds | Other | <0.10 | 15 |

N/D=Non-detectable.

N/A=Not available.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, based on RTI survey [Appendix C

of the RIA].

Friction Products

In this sector, manufacturers assemble automatic transmission parts, disk and drum brakes, and automotive clutches. Asbestos products undergo a final forming process which may include grinding. The product is then assembled by means of a riveting operation. An example of this secondary fabrication of friction products is the assembly of disc brakes. The asbestos brake pad received from the primary manufacturer is prepared prior to attachment to the metal brake shoe. This preparation might involve drilling holes or grinding to fit a shoe. The pad is then riveted to the metal shoe. Despite the use of local exhaust, grinding generates high volumes of asbestos dust. Thus, grinding results in problem exposures as it does in primary manufacturing.

OSHA's determination of feasibility in this sector is also based on data obtained in response to the RTI survey. These data, which were obtained from four plants, are summarized in Table 19. As the average exposures shown were well below 0.2 f/cc, OSHA has determined that it is feasible for this sector to comply with the 0.2 f/cc PEL, except for grinding operations, where respirators will be used.

Gaskets and Packing

The report prepared by Versar [Exhibit 333] indicated that 95 percent of asbestos gaskets and packings undergo secondary manufacturing. Secondary fabrications cut the gaskets from paper sheets using metal die stamping or pressing machinery. Sawing and drilling are sometimes performed in the finishing of the gaskets.

The greatest potential for exposure in the secondary fabrication of packings occurs during slitting and braiding operations. Wet methods are sometimes used in the braiding of asbestos yarns. Local exhaust systems are used along with housekeeping practices to minimize exposures.

**TABLE 19. -- WORKER EXPOSURES DURING THE SECONDARY FABRICATION FRICTION PRODUCTS**

| Job classification/process | Mean 8-hr TWA exposure (f/cc) | Standard deviation | Number of observations |
|---|---|---|---|
| Dry mechanical n1 | 0.07 | 0.04 | n2 66 |
| Other n3 | 0.04 | 0.03 | n2 152 |

n1 Data obtained from plants designated as "ee," "hh," "mm," and "nn."

n2 Four plants reported average values. This number presents the employment at the plants in this job category.

n3 Data obtained from plants designated as "hh," "nn," and "qq" in the RTI survey.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, as derived from RTI survey.

OSHA's feasibility analysis for this sector is based on 70 observations obtained from the OSHA MIS compliance data for the years 1979 through 1984. These observations ranged from non-detectable to 0.43 f/cc, with a mean value of 0.06 f/cc and a standard deviation of 0.1 f/cc. Based on these data which do not represent the best controlled plants, OSHA has determined that it is feasible for this sector to comply with the 0.2 f/cc

PEL.

Textiles

Secondary manufacturers produce fire-resistant and heat-resistant materials and electrical insulation from asbestos cloth and yarns. Data from OSHA MIS data and RTI surveys [See Appendix C of the RIA] indicate that the cutting of asbestos fibers and the sewing of these materials with asbestos thread result in exposures above 0.2 f/cc PEL. OSHA's feasiblity determination that this sector may have difficulty meeting the PEL is based on data obtained from two plants in response to the RTI survey and from an OSHA inspection report. These data are summarized in Table 20.

As it may not be feasible for these operations to be performed with exposures below 0.2 f/cc, respirators may have to achieve the PEL. This determination is consistent with the data provided by Raymark [Appendix B of the RIA] and with the position of the AFL-CIO [Exhibit 335, p. 44] that this is a problem sector. OSHA, however, expects that plants in this sector would utilize controls used by other asbestos processors (e.g., local exhaust ventilation, vacuums, etc.). These controls are currently available and their implementation should reduce exposures.

**TABLE 20. -- WORKER EXPOSURES DURING THE SECONDARY MANUFACTURE OF ASBESTOS TEXTILES**

| Job classification/ process | Mean 8-hr TWA exposures (f/cc) | No. of observations | Source of data |
|---|---|---|---|
| Sewing and cutting of fabric. | 0.6 | 3 | OSHA MIS. |
| Sewing and cutting of fabric. | 1.5-1.8 | n1 8 | RTI Survey. n2 |
| Other | 0.185 | 2 | OSHA MIS. |
| Other | 1 | n1 12 | RTI Survey. n3 |

n1 Number of samples was not reported. These data represent the number of workers represented by the readings.

n2 Plant designated as "ss" (see Appendix C of the RIA).

n3 Plant designated as "rr" (see Appendix C of the RIA).

Source: U.S. Department of Labor, OSHA Office of Regulatory Analysis.

Plastics

The secondary manufacture of asbestos-reinforced plastics involves the forming and finishing of preform plastics received from primary manufacturers. The process steps are the same as these for primary manufacturing. The preform is received and then remelted. It is then rolled, stamped, pressed, or molded as in primary manufacturing. The product is cured in an enclosed area which is furnished with local ventilation.

When curing is complete, the product is finished through operations that may include grinding, drilling, or sanding. Hand and portable tools are equipped with shrouded exhaust/collection systems. Larger finishing machines use local exhaust systems near the surface being finished.

The dry mechanical operations performed in this industry are similar to the finishing steps of primary manufacturing where exposures have been shown to exceed the 0.2 f/cc PEL. There were no comments submitted to the OSHA record, however, that indicated that a 0.2 f/cc TWA would not be feasible for this sector. Consequently, although the Agency recognizes that some dry finishing operations may cause high exposures for short periods of time, OSHA believes it is technologically feasible to reach a 0.2 f/cc TWA. This determination is based on seven OSHA compliance reports which indicated an average exposure of 0.1 f/cc.

Automotive Brake and Clutch Remanufacturing

This type of remanufacturing is a salvage operation that rebuilds worn brakes and clutches. Worn brake pads and clutch facings are stripped from their metal supports and are replaced with new pads and linings. The stripping of the old asbestos pad is a potential source of high exposures. To remove the entire used pad, the operation may require abrasive action which causes dust to be generated. Once the metal back of the old pad has been cleaned, the process is identical to the assembly procedure described earlier for the fabrication of secondary friction products. OSHA based its feasibility determinations on data obtained from the OSHA MIS data base and from responses to the RTI survey. These data are summarized in Table 21. As the mean exposures for this industry are 0.12 f/cc or below, OSHA has determined that it is feasible for this sector to comply with the 0.2 f/cc PEL.

**TABLE 21. -- WORKER EXPOSURE DATA FOR AUTOMOTIVE BRAKE AND CLUTCH REMANUFACTURING**

| Job classification/process | Mean 8-hour TWA exposure (f/cc) | Standard deviation | Number of observations | Source of data |
|---|---|---|---|---|
| Dry mechanical | 0.05 | 0.06 | n1 112 | RTI survey. n2 |
| Dry mechanical | .12 | .11 | n3 23 | OSHA MIS. |
| Other | .08 | .10 | n4 56 | Do. |

n1 Data on the number of samples were not provided. This figure is the number of workers represented by the data.

n2 Plants designated as "tt," "uu," "vv," "xx," "zz," "GH," and "KL."

n3 Data on 24 observations were available for the years 1979 through 1984. One outlier was omitted (1.6 f/cc) since all of the other observations were 0.5 f/cc or below.

n4 Data on 58 observations were available. Two outliers were omitted (1.1 f/cc and 1.0 f/cc) since all of the other observations were 0.4 f/cc or below.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, as derived from RTI survey.

Service Industries

*Automotive Brake and Clutch Repair.* Workers who repair brakes and clutches made with asbestos may be exposed because brakes and clutches deteriorate with wear, thereby resulting in friable asbestos. Asbestos dust present on these automotive parts is easily disturbed and becomes airborne during the repair and removal of the linings.Exposures above 0.2 f/cc are particularly prevalent when compressed air is used to clean the linings. These exposures can be significantly reduced, however, by using solvent mists on the linings and then wiping them off, or by using vacuums to remove the dust.

OSHA determined that it is feasible for this industry to meet the 0.2 f/cc. This determination is based primarily on data obtained from the OSHA MIS compliance data base and from a November 22, 1982, study by the National Institute for Occupational Safety and Health (NIOSH) [Report No. 32.4]. The OSHA data contained 47 observations from the period 1979 through 1984, with a mean 8-hour TWA exposure of 0.03 f/cc and a standard deviation of 0.14 f/cc.In addition, the NIOSH study demonstrated that average exposures were below 0.1 f/cc when using either the solvent mist or the high-efficiency particulate air (HEPA) vacuum systems. Thus, OSHA determined that the 0.2 f/cc is feasible in this sector.

*Shipbuilding and Repair.* Current shipbuilding activities should not generate any worker exposure to asbestos because the use of asbestos has been phased out of this type of construction. The greatest potential for asbestos exposure is during the removal, or "rip-out," of old asbestos material. Rip-out often requires sawing, tearing, cutting, and scraping to remove existing asbestos materials, and these activities frequently occur in confined spaces. Additional sources of asbestos exposure for a small number of shipyard workers occur during operations such as gasket cutting. OSHA believes that these additional exposures can be kept below the PEL of 0.2 f/cc through the use of ventilation and wet methods, which have been used successfully in other industries.

OSHA, however, anticipates problems in controlling exposures during major rip-out operations. These operations involve the removal of asbestos from large areas such as machinery rooms or engine rooms. The particular constraints of the shipbuilding/repair work environment limit the use of traditional engineering controls. Safety rules restrict the number of hoses, pipes, and other equipment that can pass through certain bulkhead openings below deck. The confined spaces in ships impede the use of even portable ventilation equipment in certain areas. In addition, wetting agents are not permitted for rip-out activity in nuclear reactor compartments because of the fear of contamination.

For example, in testimony at the formal hearings, Mr. James R. Thorton of the Newport News Shipbuilding Drydock Co. presented exposure data collected during major rip-outs of reactor compartments where the use of water and saturating agents was restricted. These data show that 41 percent of the exposures were greater than 2.0 f/cc, and another 32 percent were between 0.5 f/cc and 2.0 f/cc [Hearing Transcript of June 25, 1984, p. 79]. The Federal Employees Metal Trades Council [Exhibit 158-6] submitted to the record other monitoring results of major asbestos rip-outs in the reactor compartment of nuclear submarines. These data showed similar exposure levels, with 40 percent of the exposures greater than 2.0 f/cc and 10 percent between 0.5 f/cc and 2.0 f/cc. Thus, OSHA concludes that the 0.2 f/cc PEL is not feasible during asbestos rip-outs of nuclear components without the use of respirators.

According to Mr. Thorton, the exposure results for major asbestos rip-outs of non-nuclear components (where wetting agents can be used) show that 5 percent of the exposures are greater than 2.0 f/cc, and 28 percent are between 0.5 f/cc and 2.0 f/cc. One of the respondents ("QR") to the RTI survey reported exposures ranging from less than 0.02 f/cc to 0.5 f/cc for the wet removal of pipe wrap, wallboard, and gasket materials. The respondent stated that PEL of 0.2 f/cc can be attained during these small-scale or "minor rip-out" operations by using wet removal practices. OSHA has thus determined that the 0.2 f/cc PEL is feasible for certain minor rip-outs in non-nuclear vessels, but that respirators will be needed during major rip-outs in non-nuclear vessels.

Construction

*New Construction.* Although concerns about the potential health hazards of asbestos exposure have curtailed its use substantially in recent years, a number of asbestos materials are still used in new construction. These products include A/C pipe and sheet, vinyl/asbestos floor tile, and asphalt roofing felts and coatings.

*A/C Pipe.* In a study [Exhibit 84-279] performed in 1977 for the A/C Pipe Producers Association, Equitable Environmental Health, Inc., (EEH) collected short-term personal samples to evaluate exposure during various operations that might be performed in the field on A/C pipe, using different types of equipment. For example, while unloading pipe at the site and laying pipe in the trench, the highest TWA concentrations reported were 0.03 f/cc and 0.02 f/cc, respectively. These data suggest that there is little potential for exposure in these operations and that no specific controls are necessary to keep exposures below the 0.2 f/cc PEL.

When installing A/C pipe, however, it may be necessary to cut, machine, or tap the pipe at the work site, which may expose workers to airborne asbestos fibers. Although the current trend is for more of these activities to be performed by the manufacturer rather than in the field [Exhibit 333, Sections G,O,Q], cutting and machining are associated with potentially high exposures. Joe Jackson of the Association of A/C Pipe Producers (AACPP) noted, however, the feasibility of installing A/C pipe with exposures below the PEL of 0.2 f/cc. In pre-hearing written testimony he stated as follows:

Workers following AACPP's recommended work practices could almost always ensure that they would avoid peak exposures in excess of 0.75 f/cc over 15 minutes, while eight-hour time-weighted average exposures would remain at 0.1 f/cc or below. [Exhibit 91-16, Section 0, p. 12.]

Based on the EEH study, OSHA has determined that these exposures can be controlled to levels under 0.1 f/cc through the use of shrouded or doty tools. Thus, the Agency has determined that it is feasible to comply with the 0.2 f/c PEL during the installation of A/C pipe.

*A/C Sheet.* In new construction activities, the installation of A/C sheet may require sawing, drilling, or sanding operations, Much of this activity, however, is performed by primary and secondary manufacturers, thereby reducing the need for additional fabrication in the field.

For on-site fabrication that does occur, the use of tools fitted with local exhaust shrouds connected to a HEPA vacuum have been demonstrated to reduce concentations significantly [Exhibits 312-A and 298]. TWA exposures during the installation of A/C sheet have been reported to be below 0.2 f/cc, even for drilling and cutting [Exhibit 84-474, Appendix A]. In fact, some studies reported only from 40 percent to 50 percent of the measurements above concentrations of 0.1 f/cc [Exhibits 308 and 333, Section R]. Thus, OSHA has determined that it is feasible to meet a PEL of 0.2 f/cc through the use of engineering controls during the installation of A/C sheet.

*Vinyl/Asbestos Floor Tile.* In four studies [Exhibit 84-474, p. 314] performed for the Resilient Floor Covering Institute, personal breathing zone samples were collected to evaluate worker exposures during various installation and removal operations for both sheet vinyl floor covering and vinyl-asbestos floor tile. The results indicated that TWA airborne fiber concentrations ranged from below detectable (less than 0.01 f/cc) to 0.10 f/cc during the installation of sheet vinyl, and from below detectable to 0.03 f/cc during the installation of vinyl-asbestos floor tile. In another study, Dunnigan and Lebel [Exhibit 84-474, p. 3.14] reported TWA concentrations below detectable levels for the installation of vinyl-asbestos floor tile.

When installing a new floor, it is often necessary to first remove the old tile or sheet vinyl floor covering. The data obtained [Exhibit 84-474, p. 314] indicate that when the recommendations of the Resilient Floor Covering Institute (e.g., wet sweeping and handling, and prohibiting powersanding and blowing asbestos dust) were followed, average TWA airborne fiber concentrations were below the 0.2 f/cc PEL during the removal of the

old floor. Thus, OSHA determined that it is feasible to comply with the 0.2 f/cc PEL during the removal and installation of vinyl/asbestos flooring.

*Asphalt Roofing Felts and Coatings.* Asbestos roofing felts are composed of approximately 85 percent chrysotile asbestos, saturated with tar or asphalt. During installation, the roofing felts are cut to length with knives and are attached to the roof with nails. Asphalt is then applied over the felts. The removal of roofing felts generally requires chopping (with an axe) or sawing (with a circular mounting on wheels) the existing roof membrane into pieces that can be pried or scraped from the the deck. Because the asbestos fibers are encapsulated with tar or asphalt during the production of the felt, the fiber release during installation and removal is expected to be relatively low.

In written testimony, Eric Wormser of Gibson-Homans emphasized that during the "tear-off" of an old roof, "there still is no asbestos exposure since asbestos fibers in any old coating or cement are encapsulated in the product" [Exhibit 91-16, Section K, p. 6]. Nevertheless, as the condition of the roof deteriorates due to age and exposure to the elements, the quantity of asbestos fibers released will increase. This is clearly shown in studies conducted by Johns-Manville, and reported by GCA Corporation [Exhibit 84-474, p. 3.17]. Personal breathing zone and area samples were collected at 11 separate construction sites to evaluate worker exposure to asbestos during the removal and subsequent replacement of old roofing. The results indicated TWA airborne fiber concentrations as high as 0.60 f/cc during the installation of roofing felts, with a mean concentration of 0.22 f/cc. Thus, engineering controls and work practices may not reduce exposures below the 0.2 f/cc PEL in all cases and respirators will be required during some roofing projects.

*Asbestos Abatement.* Because of the concerns about potential health hazards, many building owners and managers, as well as industrial firms, are performing asbestos abatement projects to prevent or reduce the potential for fiber release. Generally, these involve either removal (with or without replacement using a non-asbestos substitute), encapsulation with a polymeric coating, or enclosure. In recent years, many contracting firms have been formed that specialize in asbestos abatement.

In general, asbestos removal involves one of two categories of products: (1) Spray-on or trowel-applied fireproofing or acoustical plasters; and (2) insulation of pipes, boilers, or process equipment. In removing asbestos, a widely used practice is to wet the material to be removed, usually with water having a surfactant added to enhance penetration [Exhibit 84-474, p. 3.22]. The use of vacuums equipped with HEPA filters, or wet mopping are the preferred methods of clean up.

In written testimony, Suzanne Kossan of the International Brotherhood of Teamsters gave evidence to support the effectiveness of wet methods, when she stated the following:

Of over 7,000 air samples gathered [in 1983] at Maryland construction sites, approximately one-half of the samples showed asbestos exposure levels less than 0.1 f/cc, 8-hour TWA. [Exhibit 223, p. 3].

The data by T. Joel Loving of the University of Virginia [Exhibit 84-474, p. 3.23] show that although wet methods are effective in reducing exposures to below the current PEL of 2.0 f/cc during asbestos removal, 47 percent of the observations exceeded 0.5 f/cc, and a total of 59 percent exceeded the 0.2 f/cc PEL. The Loving report also summarized similar data from other investigators.

The data from Clayton Environmental Consultants, Inc. [Exhibit 84-474, p. 3.27] for the removal of fireproofing and acoustical plastics using both wet methods and a HEPA vacuum, for example, show eight short-term exposures ranging from below detectable to 170 f/cc. In fact, of 255 personal samples collected, 79 percent exceeded the 0.2 f/cc PEL. Joseph Durst, Jr., of United Brotherhood of Carpenters and Joiners of America, acknowledged the difficulty of reducing exposure levels during abatement projects and stated as follows:

Although exposures could be brought down to the level of 500,000 to one million fibers/m<3> [through the use of wet methods and engineering controls], exposures below 100,000 fibers/m<3> may be difficult to achieve in some cases. In those cases personal protective equipment will be necessary and would be the only feasible way to reduce exposures to below safe levels. [Exhibit 143, p. 4.]

Thus, on the basis of these data, OSHA has determined that engineering controls cannot routinely reduce exposures below the 0.2 f/cc PEL during major asbestos removal projects and that the supplemental use of respirators may be required.

For minor removal projects, where small amounts of asbestos are removed, OSHA has determined that the 0.2 f/cc PEL is feasible. For example, data supplied by Clayton Environmental Consultants, Inc., indicate that 8-hour TWA exposures during the removal of preformed pipe insulation from process pipe at petroleum refineries using wet methods, range from less than 0.01 f/cc to 0.57 f/cc with a geometric mean value of 0.09 f/cc [Exhibit 84-474, Table 3.10]. OSHA assumes that smaller jobs would be associated with such lower TWAs (due to the shorter duration of exposure). In addition, "glove bags" are available for certain types of jobs. In 15 area samples collected during the removal of asbestos from steam pipes while using glove bags [Exhibit 84-474, Table A-2], TWA concentrations ranged from below detectable (less than 0.1 f/cc) to 0.02 f/cc. These data demonstrate that glove bags can reduce airborne fiber concentrations to below the 0.2 f/cc PEL.

Encapsulants are still being used in many asbestos abatement projects. Encapsulants are water-soluble latex products that are sprayed on to asbestos materials to bind and prevent the release of asbestos fibers. An encapsulant may either be a bridge, which forms a film over the surface of the insulation material, or a penetrant, which soaks at least partially through the fiber matrix. By its nature, encapsulation, when applied by an experienced professional, does not normally involve high fiber release. In personal samples collected by Clayton Environmental during the application of both bridging and penetrating encapsulants, TWA concentrations, however, ranged from 0.03 f/cc to 0.28 f/cc, with a geometric mean of 0.17 f/cc. Thus, with the majority of samples below 0.2 f/cc, OSHA believes that it is generally feasible for this sector to comply with the 0.2 f/cc PEL during encapsulation work, although respirators may be needed on some projects.

*Renovation/Remodeling of Existing Structures.* Asbestos has been used widely in construction until the mid-1970s when certain applications were curtailed by the Environmental Protection Agency (EPA). As a result, substantial amounts of asbestos materials are present in numerous buildings that were constructed in earlier years.

In addition to the uses in new construction described above, materials containing asbestos are used for pipe and boiler insulation, fireproofing, drywall tape and spackling, and acoustical plasters. Consequently, such materials are present in office buildings, schools, hospitals, residential buildings, industrial facilities, power plants, etc. that were built in earlier years.

In renovation projects, workers indirectly involved with asbestos products may be exposed inadvertently by disturbing these materials [Exhibit 207]. For example, in multistory buildings where beams and/or decking are covered with asbestos fireproofing, electricians, pipefitters, telephone installers, or workers who repair heating ventilation and air-conditioners may be exposed to appreciable concentrations of asbestos fibers when working above suspended ceilings. This exposure may result from direct contact with the fireproofing, or from the disturbance of settled fibers from various surfaces above the ceiling (i.e., existing pipe, ductwork, or drop ceiling tiles).

In personal samples collected in office buildings and schools, [Exhibit 84-474, p. 3.31] Clayton Environmental Consultants measured TWA exposures ranging from 0.02 f/cc to 1.4 f/cc, with a geometric mean of 0.14 f/cc,

while workers were removing drop ceiling tiles from the ceiling tract. The results of the samples collected in the breathing zones of electricians, pipefitters, and heating, ventilation, and air-conditioning (HVAC) workers indicated geometric mean TWA concentrations of 0.11 f/cc, 0.12 f/cc, and 0.14 f/cc, respectively [Exhibit 84-474, Table A-12]. The highest value measured was 2.8 f/cc for an HVAC worker. In each case, wet methods were employed for any direct contact with asbestos material, and HEPA vacuums were used for clean-up. These values are consistent with OSHA inspection data [Exhibit 84-474, Table A-11].

A variety of other activities may also involve the disturbance of asbestos materials and the subsequent exposure of renovation workers. For example, carpenters and drywallers may install new walls which, if attached to beams covered with fireproofing, may result in exposure. The results of samples collected by Clayton Environmental Consultants, Inc., indicate geometric mean TWA concentrations of 0.16 f/cc for carpenters and 0.41 f/cc for drywallers. Personal samples taken by the Argonne National Laboratory during similar activities showed TWA concentrations ranging from 0.35 f/cc to 0.87 f/cc using wet methods and HEPA vacuums [Exhibit 84-474].

OSHA has determined that engineering controls (such as negative-pressure enclosures and vacuums) are generally effective in limiting exposures after asbestos containing materials have been disturbed, but that workers who actively disturb these materials will probably require respiratory protection to comply with the 0.2 f/cc PEL.

*Routine Facility Maintenance.* Routine maintenance and repair activities may also involve the disturbance of asbestos materials and products, as described in the industry profile.Such activities include the repair of leaking steam pipes in buildings and the adjustment of HVAC equipment above suspended ceilings.

TWA exposures ranging from 0.02 f/cc to 1.4 f/cc have been measured in personal samples collected during the removal of drop ceiling tiles. In data reported by Paik and coworkers [Exhibit 207], the average concentrations during routine maintenance activities ranged from 0.9 f/cc to 1.4 f/cc.

In samples collected by Clayton Environmental during the inspection and repair of HVAC equipment near asbestos insulation materials, TWA concentrations ranged from 0.04 f/cc to 0.9 f/cc, with a geometric mean of 0.21 f/cc [Exhibit 308, Table A-14]. Results consistent with these findings were also reported by Argonne National Laboratory during maintenance activities where wet handling was used, when possible, and where HEPA vacuums were used [Exhibit 298].

These data demonstrate a potential for exposure of maintenance personnel to concentrations exceeding 0.5 f/cc. With the exception of wet handling, which is feasible in only very limited situations due to problems such as electrical wiring, and the use of HEPA vacuums for the clean-up of any debris generated during maintenance activities, OSHA believes that there does not appear to be any feasible engineering controls or work practices available to reduce these potential exposures to levels below the 0.2 f/cc PEL and that respirators will be required to comply with the 0.2 f/cc PEL.

*Demolition.* Demolition of all or part of a building or industrial facility that contains asbestos would also be likely to cause a disturbance of asbestos materials.

Under current EPA regulations (40 CFR Part 61, Subpart M, National Emission Standard for Asbestos), demolition is defined as the "wrecking to taking out [SIC] of any load-supporting structural member of a facility together with any related handling operations." EPA requires that friable asbestos materials be removed from buildings or industrial facilities prior to wrecking or dismantling the structures. Presuming compliance with the EPA regulation, the only potential for exposure would be during the removal of such materials before demolition. The feasibility of compliance with the 0.2 f/cc PEL for asbestos removal was discussed previously. The demolition project at the National Press Building in Washington, D.C., further illustrates this feasibility.

During this project, work practices were so effective in limiting exposure levels that asbestos levels were higher outside the building than inside where the demolition was occurring. Although no personal samples were taken, areas samples in work activity zones revealed average exposure levels below 0.1 f/cc [Exhibit 268].

Conclusion

OSHA has determined that compliance with the 0.2 f/cc PEL is feasible in most industries most of the time through the use of wet methods, engineering controls, and good housekeeping practices. There are some operations, however, for which compliance through the use of engineering controls and work practices alone does not appear achievable at this time. These situations are usually due to the inability of the operation to use wet methods (e.g., some textile operations, nuclear rip-out, some building repair etc.), or due to space limitations, (e.g., maintenance and major rip-outs on ships) and the volume of dust generated (e.g., cutting coupling operations for A/C pipe and sanding A/C sheet). During these operations, therefore, respiratory protection must also be used to comply with the 0.2 f/cc PEL. Finally, engineering controls are needed even when immediate exposures exceed 0.2 f/cc, however, because they protect workers in neighboring areas from being exposed over the PEL.

*Benefits*

The inhalation of asbestos fiber has been clearly associated with three clinical conditions: asbestosis, mesothelioma (a cancer of the lining of the chest or abdomen), and lung cancer. Many studies have also observed increased gastrointestinal cancer risk. Risk from cancer at other sites, such as the larynx, pharynx, and kidneys, is also suspected.

Initial exposure limits for asbestos were based on efforts to reduce asbestosis which was known to be associated with asbestos exposure. The reduction in the number of cases of asbestosis, however, resulted in workers living long enough to develop cancers that are now recognized as associated with asbestos exposure. The following discussion of the benefits associated with a reduction in exposures, therefore, focuses on the number of cancer cases avoided within the exposed work force. The results are expresed in terms of deaths avoided because these cancers almost always result in death.

The benefits of a reduction in the PEL depend upon current exposure levels, the number of workers exposed, and the risk associated with each exposure level. The current ambient aid levels estimated by OSHA and the estimated number of workers exposed to asbestos are presented in Tables 22 through 23. Based on the Agency's economic and feasibility analyses, OSHA estimated the new exposure and employment levels that would resut from the promulgation of the revised 0.2 f/cc PEL. These are also presented in Tables 22 and 23. The lifetime risk of three kinds of cancer (lung cancer, mesothelioma, and gastrointestinal cancer) was estimated by OSHA for 1 year of exposure and is presented in Section VI of this preamble.

**TABLE 22. -- ESTIMATES OF OCCUPATIONAL EXPOSURE TO ASBESTOS IN GENERAL INDUSTRY FOR 1984**

| Industry segment | Current 2.0 f/cc No. of exposed workers | Current 2.0 f/cc Level of exposure (f/cc) | proposed 0.2 f/cc No. of exposed workers | proposed 0.2 f/cc Level of exposure (f/cc) |
|---|---|---|---|---|
| Primary manufacturing: | | | | |
| Asbestos/cement pipe | 512 | 0.12 | 512 | 0.02 |
| Asbestos/cement sheet | 203 | .69 | 159 | .13 |
| Friction materials | 5,104 | .68 | 4,801 | .11 |

| | | | | |
|---|---|---|---|---|
| Textiles | 413 | .37 | 405 | .03 |
| Floor tile | 276 | .06 | 276 | .06 |
| Gaskets and packings | 315 | .37 | 306 | .07 |
| Paper | 387 | .13 | 380 | .03 |
| Coating and sealants | 1,327 | .31 | 1,327 | .06 |
| Plastics | 324 | .28 | 322 | .05 |
| Secondary manufacturing: | | | | |
| Asbestos/cement sheet | 345 | .45 | 345 | .07 |
| Friction products | 1,504 | .27 | 1,458 | .10 |
| Gaskets and packings | 9,972 | .08 | 8,741 | .02 |
| Textiles | 172 | .59 | 170 | .05 |
| Plastics | 2,450 | .10 | 2,420 | .04 |
| Automotive remanufacturing | 4,750 | .19 | 4,669 | .03 |
| Services: | | | | |
| Automotive repair | 526,998 | .06 | 526,998 | .01 |
| Shipbuilding and repair | 15,000 | .27 | 15,000 | .02 |

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis. Based on the analysis presented in Appendix G of the RIA.

**TABLE 23. -- ESTIMATES OF EXPOSURE TO ASBESTOS IN THE CONSTRUCTION INDUSTRY: 1984 n1**

| | Current 2.0 f/cc | | Proposed 0.2 f/cc | |
|---|---|---|---|---|
| Industry segment | No. of full-time equiva-lent workers | Level of exposure (f/cc) n2 | No. of full-time equiva-lent workers | Level of exposure (f/cc) |
| New construction: | | | | |
| Asbestos/cement pipe | 1,415 | 0.035 | 1,415 | 0.035 |
| Asbestos/cement sheet | 1,225 | .130 | 1,225 | .10 |
| Built-up roofing installation | 1,375 | .220 | 1,375 | .022 |
| Asbestos abatement: | | | | |
| Asbestos removal | 3,820 | .140 | 3,820 | .021 |
| Asbestos encapsulation | 453 | .220 | 453 | .022 |
| Demolition | 3,163 | 0.61 | 3,163 | .001 |
| General building renovation: | | | | |
| Drywall demolition | 51,300 | .340 | 51,300 | .003 |
| Built-up roofing | 10,990 | .120 | 10,990 | .012 |
| Routine maintenance in commercial and residential buildings: | | | | |
| Repair/replace ceiling files | 895 | .450 | 895 | .045 |
| Repair/adjust ventilation/lighting | 2,688 | .310 | 2,688 | .006 |
| Other work above drop ceiling | 385 | .310 | 385 | .006 |
| Repair plumbing/boiler | 2,854 | .180 | 2,854 | .018 |
| Repair roofing | 3,073 | .120 | 3,073 | .012 |
| Repair drywall | 4,618 | .750 | 4,618 | .075 |
| Repair flooring | 18,430 | .020 | 18,430 | .020 |
| Routine maintenance in general industry: | | | | |
| Gasket removal and installation | 768 | .090 | 768 | .080 |

| | | | | |
|---|---|---|---|---|
| Removal of pipe and boiler insulation | 653 | .123 | 653 | .025 |
| Miscellaneous activities | 612 | .294 | 612 | .029 |

n1 Based on the determination that there is a large group of construction workers who are exposed to asbestos infrequently throughout the year. This analysis converts the number of workers to the full-time equivalents (i.e., the number of workers that would be exposed for the full 1-year period).

n2 These exposure levels were estimated based on the assumption that the least costly respirator will be used. If supplied-air respirators are used, as is assument in the cost analysis, then the exposures will be lower.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.Based on the analysis presented in Appendix G of the RIA.

Based on these risk assessments, OSHA estimated the deaths resulting from these three types of cancer, given current exposures. n2 These estimates are presented in Table 24. OSHA estimates that by reducing the PEL from the current 0.2 f/cc level to 0.5 f/cc, approximately 33 cancer deaths per year will be prevented, and by reducing the PEL to 0.2 f/cc, approximately 75 cancer deaths per year will be prevented. Estimates of the number of cancer deaths avoidable by reducing exposures to the 0.2 f/cc PEL in each major industry sector are presented in Table 25. These estimates were based on the revised employment and exposure estimates presented in Tables 22 and 23. The estimated 75 cancer deaths avoided by reducing the PEL from 2.0 f/cc to 0.2 f/cc understates the true benefits of the revised standard because these benefits do not include the reduced incidences of asbestosis-related disabilities nor the reduced incidence of asbestos-related diseases in groups indirectly exposed in the workplace.

n2 Given the nature of the construction industry, many workers are exposed intermittently throughout year. In order to estimate the cancer deaths, full-time equivalents were used -- that is, two workers exposed for one-half year each would total one full-time equivalent.

Based on the analysis of existing studies, which are summarized in the Health Effects Section of this Notice, OSHA estimates that reducing the PEL to 0.2 f/cc would prevent 30 cases of disabling asbestosis. As these cases represent disabilities and not deaths, they were not included in the total estimated benefits. As such cases would result in potential costs to society (e.g., health care, lost worker productivity, and a decline in the quality of life to the affected individual), their prevention does have a positive value.

**TABLE 24. -- EXPECTED DEATHS ATTRIBUTABLE TO 1 YEAR OF OCCUPATIONAL ASBESTOS EXPOSURES AT 1984 LEVELS**

| Industry | Total cancer deaths |
|---|---|
| Primary manufacturing: | |
| A/C pipe | 0.07 |
| A/C sheet | .16 |
| Textiles | 4.00 |
| Floor tile | .18 |
| Gaskets and packings | .02 |

| | |
|---|---|
| Paper | .13 |
| Coatings and sealants | .06 |
| Plastics | .48 |
| Secondary manufacturing: | |
| A/C sheet | .18 |
| Friction materials | .65 |
| Gaskets and packings | .88 |
| Textiles | .12 |
| Plastics | .29 |
| Automotive remanufacturing | .90 |
| Services: | |
| Automotive repair | 39.25 |
| Ship repair | 4.61 |
| Construction: | |
| New construction | .61 |
| Asbestos abatement | .76 |
| Demolition | .23 |
| Building renovation | 22.49 |
| Routing maintenance in commercial and residential building | 11.23 |
| Routine maintenance in general industry | .39 |
| Total | 87.80 |

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis.

**TABLE 25. -- EXCESS CANCER DEATHS AVOIDED DUE TO REDUCING THE PERMISSIBLE EXPOSURE LIMIT TO 0.2 F/CC FOR 1 YEAR**

| | **Total** |
|---|---|