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Occupational Safety and Health Administration

Occupational Exposure to Asbestos; Emergency Temporary Standard

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DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Part 1910

[Docket No. H-0330]

Occupational Exposure to Asbestos

AGENCY: Occupation Safety and Health Administration (OSHA), Labor.

ACTION: Emergency temporary standard.

SUMMARY: OSHA is issuing an emergency temporary standard (ETS) to reduce the permissible exposure limit (PEL) for asbestos from 2 fibers (longer than 5 micrometers) per cubic centimeter (2 f/cc) as an eight-hour time-weighted average, to 0.5 f/cc. During the period of the emergency standard, employers may use all practicable control methods, such as engineering controls, work practices and personal protective equipment to meet the new limit of 0.5 fibers per cubic centimeter (0.5 f/cc). Training programs are also required to be instituted within 30 days.

The basis for this ETS is OSHA's determination that continued employee exposure to asbestos under current conditions that exceed 0.5 f/cc presents a grave danger of developing asbestos-induced cancer and asbestosis to exposed employees, and that an emergency standard is necessary to protect them. The ETS serves also as a proposal to revise the current asbestos standard pursuant to section 6(b) and 6(c) of the Act.

This notice also requests comments on the appropriateness of including the provisions of the ETS as permanent revisions to the asbestos standard. In addition, OSHA will soon publish a separate notice of proposed rulemaking that further explains the issues under consideration for the permanent standard and which raises some additional issues regarding the application of certain provisions of the asbestos standard to the construction industry. That notice will invite public comments and will schedule a rulemaking hearing pursuant to Section 6(b) of the Act concerning the proposed permanent revision to the asbestos standard.

DATES: The effective date for this ETS is November 4, 1983. Comments and evidence concerning the proposed revisions to the permanent standard made by the ETS must be received on or before January 3, 1984. As noted, OSHA will publish a notice of proposed rulemaking shortly that will set due dates for submissions to the asbestos docket for the issues raised therein.

ADDRESSES: Written comments should be submitted to the Docket Officer, Docket No. H-033C, Room S-6212, U.S. Department of Labor, Third Street and Constitution Avenue, N.W., Washington, D.C. 20210.

FOR FURTHER INFORMATION CONTACT: 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.

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I. Introduction

This is an emergency temporary standard (ETS) issued pursuant to section 6(c) of the Occupational Safety and Health Act of 1970 (the Act) (84 Stat. 1596; 29 U.S.C. 655), the Construction Safety Act (Pub. L. 91-54; 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. This emergency standard consists of revisions to the existing standard covering occupational exposure to asbestos, 29 CFR 1910.1001, in order to protect employees from a grave danger from continued exposure to asbestos at current exposure levels.

OSHA has determined that workers exposed to asbestos under exposure conditions existing under the current standard face a grave danger of developing incurable cancer and asbestosis. Further, OSHA has found that an immediate reduction in exposure levels to 0.5 f/cc or below is necessary to protect such employees from this grave danger. Therefore OSHA is issuing this ETS to compel reduced exposures to respond to this emergency situation.

OSHA's determination that a grave danger currently exists is predicated upon quantitative risk estimates in this record which point to a large number of excess deaths from cancer (and asbestosis) among currently exposed workers which are attributable to continued exposures under present working conditions. OSHA estimated the number of cancer deaths avoided through lowering of the exposure to 0.5 f/cc, based on 6 months, 1-year, 20-year and 45-year periods of exposures at current levels.* The predicted cancer deaths avoided that result from these periods of exposure are respectively 210, 426, 5725 and 7815. A more detailed discussion of these estimates is found in the sections, Quantitative Risk Analysis (Section V), Grave Danger (Section III A), and Need for an ETS (Section III B). Risks of this magnitude at permitted and actual exposure levels defined a situation which demanded immediate regulatory action. Evaluation of the relevant scientific data, policy considerations and exposure patterns of workers has convinced OSHA that continued exposure at the current PEL and under actual workplace conditions constitutes a "grave danger" to employees, and that an ETS is needed to protect them.

The ETS requires that employees may not be exposed to concentrations of asbestos exceeding 0.5 f/cc on an 8-hour time-weighed average basis, and permits the employer to choose among engineering controls, work practices and respirators to reduce exposures to the new PEL. However, the requirement in § 1910.1001(c) to utilize feasible engineering controls and work practices to reduce exposure levels to 2 f/cc remains in effect under this temporary standard. The ETS also requires employers to institute a training program within thirty days of the effective date of this emergency standard. The training progam will include instruction on respirator fitting and use, handling of asbestos, medical information, the relationship between smoking and lung cancer, and a review of the asbestos standard. The ETS applies to all industries covered by the Act, including "general industry", construction and maritime industries.

* OSHA is aware, of course, that Section 6(c) of the Act limits the effective time of an ETS to 6 months and OSHA concludes that a grave danger exists and an ETS is necessary even if OSHA focuses exclusively on this six month period. However, the Agency believes it is appropriate to calculate benefits deriving from an ETS using lifetime risks from 20 and 45 years of exposure to the PEL of 0.5 f/cc established by the ETS. Although the ETS expires within 6 months, Section 6(c) requires that rulemaking on a permanent standard also be completed within 6 months, so that there will be no gap in protection for exposed employees. In OSHA's experience and judgment, complying with this statutory directive and completing rulemaking for a permanent standard within 6 months of an ETS has and can be done.

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As provided in 29 CFR 1953.22, the 24 States with their own OSHA-approved occupational safety and health plans are expected to adopt a comparable standard within 30 days of this publication date. These States are: Alaska, Arizona, California, Connecticut (for State and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, and Wyoming.

Also, in response to the emergency conditions faced by exposed workers, OSHA is stepping up its enforcement activities in targeting workplaces where asbestos is handled.

The temporary provisions that are being added to the existing asbestos standard prescribes the major components of an occupational safety and health standard. In general, most of the current requirements remain unaffected by the emergency temporary standard. However, compliance with some requirements will be triggered by the new exposure limit of 0.5 f/cc instead of the former PEL of 2 f/cc. For example, requirements such as change rooms remain unaffected by the ETS and the trigger level for change rooms remains the former PEL of 2 f/cc (§ 1910.1001(d)(4)). However, the ETS requires that where concentrations may exceed the new PEL, the employer must post signs indicating such locations (§ 1910.1001(k)(6)).

OSHA based its decisions concerning appropriate provisions for the ETS on its determinations of the kind and degree of protective action needed to protect against a grave danger and the feasibility of instituting these provisions during the period of the ETS.

Under section 6(b) of the Act, OSHA will shortly schedule an informal rulemaking hearing on the proposed permanent changes to the standard. OSHA will also propose other revisions to the asbestos standard that will be explained in the separate notice of proposed rulemaking. Application of certain provisions of the asbestos standard to the construction industry will be raised at that time. OSHA anticipates that this notice will be published shortly. OSHA is now accepting written comments on those issues raised in the ETS which are relevant to revising the permanent rule. The Secretary must promulgate a section 6(b) standard no later than six months after publication of the emergency standard.

II. Legal Authority and Background

A. Legal Authority

Section 6(c) of the Act provides for immediately effective emergency temporary standards in certain circumstances. The Secretary has the authority to issue such a standard, without rulemaking, "if he determines (A) that employees are exposed to grave danger from exposure to substances or agents determined to be toxic or physically harmful or from new hazards, and (B) that such emergency standard is necessary to protect employees from such danger." 29 U.S.C. 655(c)(1). Thus, the danger must be "from" worker exposure and it must be "grave," not merely significant and the regulation must be "necessary" to address "such danger," not merely reasonably necessary or appropriate to provide safe employment. In addition, as in section 6(b) permanent standards, OSHA may impose requirements in the emergency standard only to the extent that they are "feasible" within the meaning of section 6(b)(5) of the Act.

The Fifth Circuit has emphasized the importance of the severity of health effects in OSHA's consideration of whether an ETS is appropriate:

> ". . . the danger of incurable, permanent, or fatal consequences to workers, as opposed to easily curable and fleeting effects on their health, becomes important in the consideration of the necessity for emergency measures to meet a grave danger." *Florida Peach Growers Association, Inc.* v. *United States Department of Labor*, 4898 F.2d, 132 (CAS).

OSHA also has interpreted relevant judicial decisions to require that its evaluation of the gravity of the danger and the necessity for emergency action must be made in the context of actual workplace conditions.

B. Background

1. *Events Leading to the ETS.* In June, 1983, OSHA received a petition for an ETS from the International Association of Machinists and Aerospace Workers (Exs. 84–244). Subsequently letters supporting this petition were received from 16 other unions (Ex. 84–284 to 84–294, 84–387 to 84–391). The petition requested an ETS to reduce the PEL to 0.1 f/cc, to modify some existing work practice requirements, and to require other protective provisions regardless of exposure level. The main reasons set forth in the petition and supporting letters are that continued exposure under current workplace conditions constitutes a grave danger and that delaying remedial action until a permanent standard is promulgated would cost large numbers of currently-exposed workers their lives. OSHA shares the genuine concern expressed by the petition and letters from unions representing thousands of employees who are directly faced with the threat of asbestos-related disease from continued exposure. And OSHA concludes that failure to issue this ETS may cost a number of exposed workers their lives.

In August 1983, OSHA completed a comprehensive risk assessment based on numerous human studies which estimated the number of excess deaths from the three major asbestos-related cancers, i.e., lung cancer, mesothelioma and gastrointestinal cancer, at the current permissible exposure level of 2 f/cc and at various reduced exposure levels. OSHA placed this document, "Quantitative Risk Assessment for Asbestos-Related Cancers", in the Asbestos docket in August 1983 (Ex. 84–349). OSHA subsequently revised this document (See Ex. 84–392).

2. *History of the Asbestos Standard.* OSHA has regulated asbestos since 1971. A 12 f/cc limit for asbestos was included in the initial promulgation of OSHA standards pursuant to section 6(a) of the Act, on May 29, 1971 (36 FR 10466). 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 on a time-weighted average basis and peak exposures of 10 f/cc.

The current standard, promulgated in June 1972, first established an 8-hour time-weighted average PEL of 5 f/cc and a ceiling limit of 10 f/cc. The limits were intended primarily to protect employees against asbestosis, and it was hoped that they would provide some incidental degree of protection against cancer. Effective July 1976, the TWA limit was reduced by the standard to 2 f/cc. This limit has remained in effect since that time. The standard also includes provisions covering methods of compliance, monitoring, medical surveillance and housekeeping.

Court review of this standard upheld all major provisions, but remanded two issues for OSHA's reconsideration. *IUD* v. *Hodgson*, 499 F. 2d 467 (CADC 1974). These issues were whether the July 1976 date for the 2 f/cc standard should be accelerated for some industries and the adequacy of the 3 year retention period for exposure monitoring records. Subsequently, OSHA increased this retention period to 20 years (41 FR 11504) and the acceleration issue became moot.

After reviewing the then available scientific data, in October 1975 OSHA published a notice of proposed

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rulemaking to revise the asbestos standard because OSHA believed that "sufficient medical and scientific evidence had 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 time-weighted average to 0.5 f/cc and imposed a ceiling limit of 5 f/cc for 15 minutes (40 FR 47652).

The basis for the 1975 proposal's reduced permissible level of 0.5 f/cc was OSHA's then current policy for carcinogens that no safe threshold level was demonstrable and, therefore, the Act required OSHA to set the PEL as low as technologically and economically feasible. This policy was rejected by the Supreme Court in the benzene decision (*IUD* vs. *API*, 448 U.S. 601 (1980)). OSHA limited the proposed revisions in the 1975 notice to all industries except construction. No hearing was scheduled on the 1975 proposal. Also, although OSHA announced its intention to develop a separate proposed revision, applicable to the construction industry, no such proposal has been published to date.

OSHA is basing its present decision to issue an ETS, and to propose revisions to the permanent standard covering all employees, on information and analyses which postdate the 1975 proposal. Therefore, this ETS marks a new regulatory initiative, related to, but not part of the 1975 proceeding. On May 24, 1983, OSHA consulted the Construction Advisory Committee for Occupational Safety and Health (CACOSH) concerning applicability of a new asbestos standard for the construction industry. CACOSH endorsed OSHA's position that changes in the PEL made for general industry should also apply to the construction industry. OSHA is also including the comments and data received in response to the 1975 notice in the record of this proceeding, with the understanding that some commenters may have changed their positions based on intervening scientific developments and policy shifts.

In the decision to issue an ETS, the Agency has reviewed this regulatory history of asbestos. However, the Agency bases the decision to issue an ETS on the actual emergency conditions which now confront exposed workers, on epidemiologic studies that provide data to make numerical estimates of risks and on interpretations of these studies using quantitative risk analysis.

3. *The Rulemaking to Revise the Permanent Standard.* The 6(b) rulemaking initiated by this proposal is intended to be limited to the revisions made by the ETS and the additional proposed amendments raised in the notice of proposed rulemaking to be published soon. The major subjects of the 6(b) rulemaking will be: reducing the permissible exposure limit, revising the definitions of asbestos and asbestos fibers, reassessing the methods of compliance to achieve such limits, revising the provisions regarding respirator selection, revising the sampling and analytical method to improve reliability, and adding a training requirement. OSHA will also raise issues regarding the application of the permanent standard to the construction industry. As previously stated, OSHA will soon publish a separate notice of proposed rulemaking to further explain these issues.

III. OSHA's Rationale for the ETS

OSHA has determined that prevailing conditions involving worker exposure to airborne asbestos dust justify the promulgation of an emergency temporary standard. OSHA estimates that approximately 375,000 workers are exposed to asbestos at various levels (Table 1), ranging from a high value of 20 f/cc to below 0.5 f/cc. OSHA has estimated that under current exposure conditions asbestos-exposed workers face an extraordinarily high risk of contracting asbestos-related cancer whether the risk is computed over a working lifetime of exposure or for exposure periods as short as 6 months. The average excess cancer risks for all workers exposed above 0.5 f/cc using available exposure data and relying on the risk assessment are estimated as approximately 196 excess cancer deaths per 1000 workers for 45 years of exposure, 139 deaths per 1000 workers for 20 years, 10 per 1000 workers for 1 year, and 6 per 1000 workers for 6 months of exposure.

OSHA believes that risks of these magnitudes, taking into account all relevant considerations such as total numbers of workers at risk and quality of supporting data, constitute an emergency situation which requires immediate response by the agency.

The Act states that when certain statutory criteria are met, OSHA is authorized to respond to an emergency situation by issuing an ETS. The two-pronged statutory test for an ETS is that (1) employees must be exposed to a grave danger from exposure to substances or agents determined to be toxic or physicially harmful, and that (2) an emergency standard is necessary to protect employees from such dangers.

After evaluating all the evidence available to the agency concerning the severity and magnitude of the risk of asbestos-related disease to the current asbestos-exposed working population, comparing these risks to other occupational risks, applying relevant policy considerations, and reviewing all relevant judicial decisions for guidance, the agency has determined that both prongs of the statutory test are met and that an ETS should be promulgated.

For purposes of clarity, the discussion is divided into two parts, "Grave Danger" and "The Need for an ETS." OSHA believes, however, that the factors which indicate that a substance constitutes a grave danger are related to and overlap those which determine that an ETS is necessary.

A. Grave Danger

OSHA has determined that the risk to workers from exposures to asbestos at conditions that exist in the workplace pose a grave danger of death from cancer and of severe disability from the lung disease, asbestosis. In making a "grave danger" determination, the severity of the disease produced by exposure to the regulated substance and the magnitude of the predicted risks of disease must be considered. In addition, the Supreme Court has suggested that a determination of "grave danger" indicates a situation where the risk is more than "significant" (*IUD* v. *API*, *supra* n. 45).

OSHA has applied that analytic approach endorsed by the Supreme Court for "significant risk" determinations in evaluating the gravity of the danger faced by asbestos-exposed workers. The Supreme Court gave some general guidance as to the process to be followed. It recognized that while the Agency must support its finding that a certain level of risk exists with substantial evidence it also recognized that its 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).

OSHA believes, therefore, that its determinations regarding the magnitude of the risk faced by employees should, to the extent possible, rely on quantitative expressions of that risk, utilizing the best available data.

The Court stated that the significant risk determination required by the OSH Act is "not a mathematical straitjacket," and "OSHA is not required to support its 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 that] *** the Agency is free to use conservative assumptions in interpreting the data

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with respect to carcinogens, risking error on the side of overprotection rather than underprotection" [488 US at 655, 656].

In the case of asbestos, the data available are of unusual breadth and high quality. However, because risk assessment itself involves many uncertainties, OSHA made certain assumptions in its analysis and evaluation of these data. In assessing the risk for asbestos-exposed workers, OSHA has attempted to use realistic assumptions, although the court stated that the Agency was free to use "conservative assumptions" in interpreting data. OSHA, in many cases, has indicated where different assumptions may produce different results. In addition OSHA cautions that because the risk figures finally derived are the products of a process which, as the Supreme Court acknowledged, is "on the frontiers of science," they should be viewed as approximations of the degree of risk faced by asbestos-exposed workers and not as precise fixed predictions of the number of workers who will actually develop disease.

OSHA has evaluated the kinds of dangers presented by asbestos exposure, the quantification of those dangers under present asbestos exposure conditions, the quality of the data on which risk estimates are based, a comparison of asbestos risks to other occupational risks, and relevant policy and legal considerations in concluding that workers are exposed to a grave danger from asbestos.

1. *Nature of the Diseases.* As stated above, the nature of the disease associated with exposure to a toxic substance is one of the most important elements OSHA evaluates in determining whether a grave danger exists. This factor was discussed in *Florida Peach Growers Association, Inc. v United States Department of Labor, supra.* The court, in overruling OSHA's organophosphate pesticide ETS, observed:

> We reject any suggestion that deaths must occur before health and safety standards may be adopted. Nevertheless, *the danger of incurable, permanent, or fatal consequences to workers, as opposed to easily curable and fleeting effects on their health, becomes important in the consideration of the necessity for emergency measures to meet a grave danger.* 489 F 2nd at 132 (emphasis added)

OSHA is aware of no instances 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 exposures are in large part life-threatening or disabling. Among these diseases are lung cancer, cancer of the mesothelial lining of the pleura and peritoneum, and asbestosis. In addition, workers exposed to asbestos are at increased risk of gastrointestinal cancer, as shown by epidemiologic studies. Although colo-rectal cancer may be curable if detected in an early stage, other gastrointestinal cancers are usually fatal. OSHA also believes that asbestos might induce cancers at other sites, which are also often fatal.

Of these, lung cancer constitutes the greatest health risk for American asbestos workers and has accounted for more than half of excess mortality in some occupational cohorts. About 90% of lung cancer patients die within 5 years of diagnosis. Mesothelioma is an incurable cancer which is usually fatal within a year after diagnosis. It is epidemiologically linked to asbestos exposure, and occurs very rarely, if at all, in persons never exposed to asbestos. Asbestosis, a type of pulmonary fibrosis, is usually non-reversible, its advanced stages are disabling, and can be fatal. OSHA concludes that all these diseases are very serious, and that the excess mortality from such severe diseases must be considered an important factor for making a grave danger determination.

2. *Degree of Risk of Developing Dangerous Disease.* OSHA based its calculations of extent of risk faced by workers under current exposure conditions primarily on the results of a quantitative analysis which derived numerical estimates of cancer risk at various cumulative exposures corresponding to levels at which workers are exposed (Ex. 84–392).

Although 2 f/cc is the current PEL for asbestos exposure, actual exposure conditions vary widely, mostly by industry segment. As explained later in this document and as set forth in Table 1, average ambient exposure levels in various industries include high exposure levels such as 20 f/cc in drywall removal, renovation and demolition; 5 f/cc in shipbuilding and repair; mid-range exposure levels such as 2 f/cc in secondary fabricating of cement sheet, packing and gaskets and paper products and rebuilding and refacing brakes; 1.5 f/cc for dry processing of textiles; and lower exposure levels such as 0.5 f/cc and 0.2 f/cc in the manufacture of floor tile.

Because OSHA is required to consider the actual danger faced by workers in assessing whether exposure to a substance presents a "grave danger", OSHA looked at the risk of developing disease not only at the 2 f/cc permissible level but at all exposure levels which workers currently face. Most of the results of these calculations for cancer are presented in Table 11 in the risk assessment section of this document.

The table sets forth predicted excess lifetime cancer risks for exposures of one year, 20 years and 45 years. Risks for exposures of 6 months are closely approximated by one-half the risks for exposures of one year. Although average exposures in demolition and renovation are estimated at 20 f/cc, the table presents risks only for selected exposure levels up to 10 f/cc.

These calculations show that the risks of asbestos-related disease are alarmingly high at current occupational exposure levels. For example, an estimated total cancer risk of 265 excess deaths per 1000 workers exists for workers exposed for a 45-year lifetime at 10 f/cc, a level which currently exists on some construction sites. At 5 f/cc, the exposure levels which are considered average in shipbuilding and repair, the risk of developing asbestos-related cancer for a 45-year exposure period is 149 excess deaths per 1000 workers. At the current permissible level of 2 f/cc which also represents actual exposure levels in such industries as secondary fabricating of cement sheet, packing gaskets and paper products and rebuilding and refacing brakes, risk is estimated as 64 excess cancer deaths per 1000 workers for a 45-year exposure period.

These risks remain very high when the period of exposure for which calculations are done is shortened to 20 years, which OSHA believes is another appropriate point for examination. The period of 20 years is the approximate midpoint between 1 year and 45 years of exposure; also many workers receive 20 years of exposure. Counterpart risk calculations using a 20-year exposure period are: for workers exposed to 10 f/cc, 140 excess cancer deaths per 1000 workers; for exposures to 5 f/cc, 105 excess cancer deaths per 1000 workers and for exposures to 2 f/cc, 44 excess cancer deaths per 1000 workers.

OSHA also estimated risks of developing cancer for a one year period of exposure at various levels to which employees are exposed. The counterpart risks for exposures to 10 f/cc for one year are: 15 excess cancer deaths per 1000 workers; to 5 f/cc, 7 excess cancer deaths per 1000 workers and to 2 f/cc, 3 excess cancer deaths per 1000 workers.

Even at current workplace exposure levels which are less than the current PEL, extraordinarily high risks of disease exist. At 0.5 f/cc, 17 excess cancer deaths per 1000 workers are

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predicted for a 45-year lifetime exposure, and 11 excess cancer deaths per 1000 workers for a 20-year exposure period.

OSHA notes that the above calculations are for cancer risk only. In addition, asbestos-exposed workers face a high risk of developing asbestosis, a disabling and often fatal disease. Predictions concerning the estimated magnitude of the asbestosis risk have been performed by OSHA and are discussed in the risk assessment section of this document. Accordingly, OSHA estimates that at 2 f/cc, 50 workers per 1000 exposed to asbestos for 45 years will develop disabling asbestosis. At 0.5 f/cc for 45 years, it is estimated that 12 workers per 1000 will develop disabling asbestosis. Asbestosis risks can also be estimated for exposures for durations shorter than 45 years. For example, the risk of disabling asbestosis from exposure to 1.0 f/cc for 22.5 years is 12 cases per 1000 workers. OSHA's estimates of the magnitude of the asbestosis risk are based on sound data from good epidemiological studies. OSHA believes, however, that the confidence which can be placed in predictions of asbestosis risk is not as great as for the predictions of cancer risk. This is because the cancer risk estimates are based on a larger and more varied data base and are derived from dose-extrapolation models that are better established. Because OSHA has determined that the risks for cancer alone indicate a grave danger, the additional risks of developing asbestosis are not necessary to justify this ETS. However, OSHA has considered that the additional and independent risk of developing asbestosis increases the danger faced by exposed workers and underscores the gravity of the health threats to employees posed by asbestos.

3. *Quality of Data on Which Risk Estimates are Based.* The underlying data upon which the quantitative risk assessments for asbestos are based are high quality epidemiologic studies, conducted in occupational environments. OSHA emphasizes that the data bases for asbestos are of unusual quality and size. Unlike most potential occupational carcinogens, asbestos has been studied often and thoroughly for evaluation of its effects on occupational populations.

In deriving these quantitative estimates for cancer risk, OSHA utilized eleven studies for the calculation of the lung cancer risk, four of which were also used to calculate the mesothelioma risk. Investigations involved "cohort" studies where the frequencies of various types of cancers in workers exposed to asbestos were compared to those in "control" groups not exposed to asbestos or to those of general populations such as U.S. males. Studies of such design are able to provide direct estimates of excess risk.

The studies used by OSHA in deriving dose-response relationships for its risk assessment covered a variety of work situations and industrial processes. This variety improves the predictive value of the risk assessment because it lessens or eliminates the possibility that the results were unique to any one occupational situation or were in fact aberrational. The occupational settings studied were: workers exposed at a chrysotile textile plant from 1930–1975 (Dement *et al.* Exs. 84–036 and 84–037); Canadian workers at an asbestos cement facility (Finkelstein Ex. 84–240); Italian chrysotile miners and millers who worked during 1930–1965 (Rubino *et al.* Ex. 84–86); workers in an asbestos cement pipe plant (Weill *et al.* Ex. 84–206); workers in an asbestos production plant and asbestos cement pipe factory (Henderson and Enterline Ex. 84–48); British workers manufacturing asbestos textile products (Peto Ex. 84–169); asbestos miners and millers in Quebec, Canada (Liddell *et al.* Ex. 84–59); and in the Thetford Mines, Canada (Nicholson *et al.* Ex. 84–72); and workers manufacturing asbestos friction materials (Berry and Newhouse Ex. 84–21).

"Well-conducted epidemiologic studies that show a positive association between an agent and a disease are accepted as the most convincing evidence about human risk" (Risk Assessment in the Federal Government: Managing the Process, National Research Council, 1983, p. 21, Ex. 84–322).

No extrapolation from animal data to human data is necessary in order to show carcinogenicity of asbestos. For most substances, OSHA must infer human health effects, such as carcinogenicity, from animal data.

The results of this risk assessment performed by OSHA agree well with other recent risk assessments performed by other governmental and outside scientists (see Acheson and Gardner) (Ex. 84–216 and 84–243); EPA (Ex. 84–180); Kang and Chu (Ex. 84–001); Selikoff *et al.* (84–002); and CHAP (Ex. 84–256).

4. *Comparative Analysis.* Insight into the magnitude of the risk associated with asbestos exposure can be gained by reviewing other occupational risks. OSHA believes it is instructive to compare asbestos risks with other workplace hazards agreed on as presenting an unusually high degree of hazard, where the data are considered both available and reliable.

The risk of excess mortality estimated as a result of exposure to asbestos at the conditions in the workplace today appears to be substantially higher than other risks experienced by workers from occupational injury hazards. The National Safety Council (NSC) has reported the annual death rates in 1981 from work accidents in a variety of industries (Ex. 84–339). Using the NSC data OSHA has reviewed the annual mortality from work accidents per 1000 workers in several industries in light of the excess cancer mortality from a single year of exposure to asbestos per 1000 workers. For example, in the high risk occupations of agriculture and mining-quarrying, the annual mortality rates from work accidents were 0.54 and 0.55 per 1000 workers respectively in 1981 (Ex. 84–339). In contrast, the death rate from work accidents for all industries combined was 0.12 per 1000 workers in 1981.

OSHA has estimated that the lifetime risk for one year of exposure to 2 f/cc of asbestos is about 3 excess cancer deaths per 1000 exposed workers during the remainder of the workers' lifetimes (Ex. 4–349). Thus, asbestos workers' risk of excess cancer mortality from a single year of exposure to 2 f/cc is roughly 5 times higher than the risk of accidental occupational fatalities from one year of employment in agriculture and mining-quarrying.

As shown in Table 1, OSHA estimates that many workers are exposed to asbestos in the vicinity of 2 f/cc. In addition, OSHA calculated the average excess cancer risk to workers exposed at conditions that exist in the workplace today (for those above 0.5 f/cc and using the scenario described in Table 3). OSHA estimates that 10 excess cancer deaths will occur per 1000 workers for 1 year exposure; thus the average risk to workers (exposed above 0.5 f/cc) in the workplace today is approximately 20 times the annual fatal accident rates in agriculture and mining-quarrying.

These comparisons are striking. They show that the estimated risk of dying of cancer from asbestos exposure at levels existing at the workplace today far exceeds the accidental death rate in the riskiest of industries. Although the estimated mortality rates for cancer due to asbestos exposure are not completely comparable to the total actual accidental fatalities, the review is clearly useful in showing that the magnitude of the asbestos risk is grave.

One example of predicted cancer risk as a result of occupational exposure is the following cancer risk estimated from

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occupational exposure to ionizing radiation. The estimated excess cancer fatality rate from 47 years of exposure to the maximum permissible occupational exposure to ionizing radiation (5 rems) is 17 to 29 per 1000 workers (Committee on the Biological Effects of Ionizing Radiation (BEIR) III predictions, see 48 FR 1902). However, most radiation standards (unlike OSHA standards) require that exposure limits be reduced to the lowest level reasonably achievable below the exposure limit (the ALARA principle). Approximately 95 percent of radiation workers have exposures less than one-tenth the maximum permitted limit. The excess cancer deaths at one-tenth the permitted level are 1.7 to 2.9 per 1000 workers exposed 47 years. Asbestos exposures of 45 years to 2 f/cc are predicted by OSHA to result in 64 excess cancer deaths per 1000 workers beginning work at age 25 (Ex. 84–392). OSHA's calculation for the average excess cancer risk to worker exposed at conditions that exist in the workplace today (for those above 0.5 f/cc) for a 45-year exposure, is 196 excess cancer deaths per 1000 workers. This figure was calculated by taking the number of cancer deaths estimated from exposure to existing conditions for 45 years for those workers exposed to greater than 0.5 f/cc of asbestos and dividing by the number of workers exposed to asbestos greater than 0.5 f/cc (multiplied by 1000).

Therefore, the excess cancer risk at 2 f/cc for asbestos workers is estimated as more than twice as high as the maximum permitted radiation cancer risk and about 25 times higher than the estimated cancer risk of 95 percent of the workers exposed to radiation. At existing conditions, asbestos workers' excess cancer risks are estimated to be 85 times higher than the cancer risk faced by 95 percent of the workers exposed to radiation. The risk of asbestosis further increases the significance of the risk from asbestos exposure.

At 0.5 f/cc, OSHA estimates that 17 excess cancer deaths will occur in 1000 workers exposed 45 years. This risk is approximately 7 times higher than the cancer risk faced by 95 percent of the workers exposed to radiation. OSHA finds that these comparative risks strongly support OSHA's finding that workers exposed to air concentrations above 0.5 f/cc are far above the point of significant risk and are at grave danger of dying from cancer.

5. *Conclusion.* OSHA's finding of "grave danger" is based on evidentiary and policy considerations. OSHA's determination that the magnitude of the estimated risk to exposed workers is alarmingly high constitutes the major component of the "grave danger" finding. The overall extraordinary degree of risk, the extent that very high risk is found in many asbestos using industries, and the unusually high quality of the data utilized to make these assessments present a very strong evidentiary basis for a "grave danger" finding. Just as importantly, the unique gravity of asbestos-caused diseases, in particular cancer, such as mesothelioma which is linked almost exclusively to asbestos exposure, strongly supports OSHA's finding of grave danger. Also OSHA's comparison of the risk of asbestos-related disease to other industrial risks underscores the extraordinarily high risk estimated for asbestos exposure. OSHA has also noted the concerns of workers about current workplace conditions and the numerous petitions for an ETS from unions representing many exposed workers. Finally OSHA has relied on its experience in evaluating and regulating workplace hazards in recognizing the extraordinary degree of risk currently faced by asbestos workers and in determining that such risk constitutes a grave danger to those workers.

B. Need for an ETS

OSHA has determined that this ETS is necessary to protect employees from grave danger, the second prong of the Act's test of OSHA's exercise of its ETS authority (Section 6(c) of the Act). As explained in detail, the effect of this ETS is to save many lives which would otherwise be lost to asbestos-related disease if current working conditions were not changed. OSHA believes that employees can be adequately protected against this grave danger only by issuing an ETS. This is because no other Agency action and no other foreseeable event would result in sufficiently reduced asbestos exposures that would alleviate the grave danger. Further, the provisions of the ETS are tailored to effect the necessary exposure reductions expeditiously.

1. *Lives Saved by Issuing an ETS.* OSHA has estimated the number of deaths avoided as a result of an ETS which would reduce the PEL to 0.5 f/cc (see Tables 2 and 3). For cancer only, based on continuing exposures under currently existing conditions for 6 months, the potential number of lives saved is estimated as approximately 210. Based on continuing exposures at currently existing conditions for 1 year, the potential number of lives saved is estimated at approximately 426. Also, OSHA has estimated that the promulgation of an ETS setting a 0.5 f/cc PEL may avoid 5725 cancer deaths assuming 20 years exposure to asbestos of the current workforce at current conditions and 7815 cancer deaths assuming 45 years exposure.

OSHA is aware, of course, that Section 6(c) of the Act limits the effective time of an ETS to 6 months, and OSHA concludes that a grave danger exists and an ETS is necessary even if OSHA focuses exclusively on this six month period. However, the Agency believes it is appropriate to calculate benefits deriving from an ETS using lifetime risks from 20 and 45 years of exposure to the PEL of 0.5 f/cc established by the ETS. Although the ETS expires within 6 months, Section 6(c) requires that rulemaking on a permanent standard also be completed within 6 months, so that there will be no gap in protection for exposed employees. In OSHA's experience and judgment, complying with this statutory directive and completing rulemaking for a permanent standard within 6 months of an ETS has and can be done.

OSHA also believes, based on its experience, that it is very likely that the PEL established after 6(b) rulemaking will be no higher than 0.5 f/cc, the ETS limit. Therefore, OSHA believes that the ETS will result in a reduced lifetime worker exposures of 0.5 f/cc or lower for 20 or 45 years, and that the benefits derived from these exposure reductions for these time periods are appropriately attributed to OSHA's promulgation of this emergency standard.

a. *Employee Exposures.* To derive these estimates of numbers of lives saved, OSHA depended on its knowledge of the following factors: (1) The employee exposure levels from the ambient asbestos air concentrations in the workplace; (2) the number of workers exposed at the various asbestos levels; (3) the duration of the exposure; and (4) the probability of the disease (or the risk) associated with the cumulative exposure.

Employee exposure levels are conventionally measured in terms of the number of asbestos fibers that are 5 microns or more in length in one cubic centimeter of air, f/cc. In these terms, an ambient concentration may seem to be a small amount of asbestos. However, in physical terms, 2.0 f/cc equals 2,000,000 fibers per cubic meter (f/m^3). Humans inhale about one cubic meter of air per hour, depending on degree of physical activity. Thus, at this concentration, a worker would inhale roughly 16,000,000 fibers, 5 microns or more in length, over an eight hour workday.

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Note.—Depending on the industrial process, up to 784,000,000 additional asbestos fibers less than 5 microns in length may also be inhaled, assuming that 98% of airborne asbestos fibers are less than 5 microns.

OSHA continues to use the term, f/cc, for convenience but cautions that the numerical estimates of air concentrations given in these terms are only one way of viewing asbestos concentrations and should not be evaluated without interpreting the meaning of the units.

OSHA used existing information to estimate worker asbestos exposure in each affected industry. Data sources included government contractor reports (Ex. 84–002 and Ex. 84–009), various studies reported in the literature, NIOSH Health Hazard Evaluations, and OSHA compliance data (Ex. 84–355). OSHA reviewed the information, and decided to use the 1980 Research Triangle Institute (RTI) report (Ex. 84–009) as the primary basis for exposure estimates because the RTI estimates appear to OSHA to be the most comprehensive. In addition, RTI used a large number of different data sources to make their exposure estimates, including interviews, existing documentation, and industrial hygiene surveys of worksites.

Using the RTI report as the primary reference, OSHA compared RTI data with other exposure information available for each industry. For example, specific reports were found for asbestos cement manufacturing (Ex. 84–248), textile manufacturing (Ex. 84–267), removal of sprayed asbestos material from buildings (Ex. 84–262), and brake repair (Exs. 84–263, 84–298). The Asbestos Information Association provided data concerning exposures during field fabrication and installation of asbestos cement pipe and sheet in controlled conditions (Ex. 84–295). The Environmental Sciences Laboratory (Ex. 84–002) also made best estimates of worker exposure in various industries for 1975.

In addition, OSHA used its own field inspection experience to estimate exposures. OSHA reviewed selected case files and obtained information that listed OSHA asbestos measurements during compliance inspections from 6/1/79 to 5/31/83 (Exs. 84–354, 84–355).

OSHA adjusted the RTI estimate as appropriate, based upon a qualitative judgment as to which data best represent existing exposure conditions. OSHA's exposure estimates are based, therefore, upon a substantial data base and upon considerable experience in enforcing the existing asbestos standard.

OSHA used the employee exposure levels in each industry to calculate the number of deaths avoided by reducing the exposure from current levels to the emergency PEL. Alternative exposure distributions used for sensitivity analysis will be provided in the Preliminary Regulatory Impact Analysis.

OSHA believes that the exposure estimates are relatively good, given the state of the art of worker exposure estimation techniques and data available today. OSHA is not aware of any other available current exposure estimates.

Industrial hygiene reports of operations in the same industry, but at different work sites, invariably report different asbestos exposures among the workers. OSHA reviewed these reports and made decisions regarding typical industry practices. For some operations, such as brake relining, several reports were available with complete descriptions of the working environment and OSHA was able to use these to make direct exposure estimates. Other reports, such as "Removal of Pre-formed Asbestos Insulation" (Ex. 84–296), described careful processes for asbestos handling that did not appear representative of the methods used throughout industry, since the reported air concentrations were not consistent with other reports showing higher exposures (Exs. 84–306, 84–262).

Table 1 shows OSHA's estimates of employee exposure to asbestos by industry segment.

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Table 1

OSHA Estimate of Employee Exposure to Asbestos

Industry Segment	Number[a] of Exposed Workers	Number[a] of Plants	Current Exposure level f/cc
Primary Manufacturing			
Asbestos/Cement Pipe & Sheet	900	14	1.0
Friction Materials	3677	31	1.5
Floor Tile			
Receiving, introductions mixing	1187	14	0.5
Rest of plant	3559		0.2
Asbestos Paper			
Receiving, introduction mixing & preparation	474	22	0.75
Rest of plant	1423		0.2

Table 1 (Cont.)

OSHA Estimate of Employee Exposure to Asbestos

Industry Segment	Number[a] of Exposed Workers	Number[a] of Plants	Current Exposure level f/cc
Gaskets, Seals and Packings			
Sheet formation, cutting and packaging	219	26	0.2
Rest of plant	657		0.75
Paintings, Coatings and Sealants			
Mixing, compounding and packaging	34	5	0.75
Rest of plant	101		0.0
Textiles-Wet Process	375	2	0.75
-Dry Process	750	3	1.5
Secondary Fabricators[b]			
Cement Sheets	4301	1076	2.0
Paper Products	4301	1076	2.0
Packings and Gaskets	4301	1076	2.0
Textiles	4301	1076	1.0
Automotive Aftermarket			
Rebuilding and Refacing	4091	140	2.0
Brake Repair			
Garages and Brake Shops	176355	68089	0.15
Gasoline Stations	84961	77896	less than 0.1
Shipbuilding and Repair[c]	1522	38	
Shipbuilding	304		0.5
Ship Repair	1218		5.0
Construction[d]			
Installation of New Materials			
Asbestos Cement Pipe	5458	546	0.75
Asbestos Cement Sheet	1765	177	2.0
Roofing Felts	2499	250	0.15

Table I (Cont.)

OSHA Estimate of Employee Exposure to Asbestos

Industry Segment	Number[a] of Exposed Workers	Number[a] of Plants	Current Exposure level f/cc
Repair and Maintenance			
Drywall removal, renovation, and demolition[e]			
	12955	1296	20.0
	38866	3887	0.2
Repair and maintenance involving asbestos removal	14845	1485	0.15
Maintenance workers in schools, chemical plants electrical generating plants and foundries	N/A	N/A	0.15
TOTAL WORKERS EXPOSED ABOVE 0.5 f/cc[f]			48,644
TOTAL EXPOSED WORKERS			375,399

[a] Population and facility estimates were based on the RTI report (Ex. 84-009) and updated to 1983 employment levels by JRB Associates.

[b] The number of plants in the SIC codes making up Secondary Fabricators (2451, 3357, 3523, 3531, and 3842) was derived by JRB Associates by taking the number of establishments with 20 or more employees, as listed in the Economic Information Systems data base for 1983, and expanding that number by the percent of establishments with fewer than 20 employees, as listed in County Business Patterns, 1980. Not knowing exactly how the 17,204 Secondary Fabrication (SF) workers and 4,304 SF plants were distributed among the 4 types of SF facilities, OSHA assumed that the workers and plants were distributed equally among the 4 types of SF facilities.

[c] Assumed that 20% of 3045 shipbuilding/repair workers are not in compliance with current respirator requirements. Hence, the number of workers was adjusted to reflect the 20% of exposed workers.

[d] Assumed average firm employs 9-10 workers as is true of SIC 17 (construction, special trade contractors).

[e] Assumed that 25% of 51820 drywall removal, renovation, and demolition workers are not in compliance with current respirator requirements. Hence, the number of workers was adjusted to reflect the 25% of exposed workers.

[f] This figure excludes segments that are in compliance with 0.5 PEL such as floor tile, brake repair, roofing felts, and repair and maintenance.

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Data from OSHA's Management Information System (MIS) includes a list of all asbestos samples reported by OSHA field staff during June 1, 1979 to May 31, 1983 (Ex. 84–355). During this period, 949 eight-hour time-weighted average (TWA) samples were collected in many different industries. Of the 949 samples, 731(77%) were below 0.5 f/cc 156 (16.4%) fell between 0.5 f/cc and 2.0 f/cc; and 62 (6.5%) were above 2 f/cc. OSHA also reviewed the MIS summary report where the measurement data were grouped within Standard Industrial Code (SIC) classifications (Ex. 84–354). All these data contributed to the development of OSHA's best exposure estimates.

In conclusion, OSHA has used its experience in enforcing the existing asbestos standard to determine the best estimates of worker exposure in the industries. On the whole, in comparison to other toxic substances present in the workplace, asbestos exposure information is well reported. Given that OSHA experience is considerable, OSHA believes its estimates are reasonable and appropriate and provide a satisfactory basis for judgments regarding the extent of risk existing in workplaces.

OSHA acknowledges, however, that the exposure information concerning asbestos removal and renovation in the construction industry is less certain than the other estimates. Despite uncertainities in qualifying exposure in the demolition/removal category, OSHA feels that this best estimate is based upon evidence that is both reasonable and the best available. Furthermore, OSHA believes that any changes made to the estimate as a result of the uncertainities would not be of a sufficient magnitude to warrant a change in its basic findings.

2. *Calculation of Lives Saved and Disease Avoided.* The benefits provided by this ETS consist of cancer deaths and disabling asbestosis avoided. The estimates of deaths avoided by lowering of the exposure limit by the emergency temporary standard are based on the mortality rates developed in the risk assessment discussion. To generate the expected number of excess deaths attributable to asbestos in the U.S. workforce, the expected rate of mortality at each exposure level was multiplied by the population exposed at that level. The expected mortality rate from each exposure level was derived from OSHA's quantitative risk analysis (Ex. 84–392). The expected number of deaths at the reduced PEL was then subtracted from the expected number of deaths at the current worker exposure levels to determine the estimated number of deaths avoided by reducing the permissible exposure limit. The benefits are expressed in terms of estimated deaths rather than disability because the types of cancer associated with asbestos exposure have a very poor survival rate.

An example of such a calculation follows. To calculate the lung cancer deaths avoided during one year of exposure to a worker population in the construction industry who are engaged in the installation of asbestos cement sheet:

Population = 1765 (Table 1).
Estimated Current Exposure Level = 2.0 f/cc (Table 1)
Estimated lung cancer risk for one year at 2 f/cc = 144/100,000 (Table 11)

1. Calculate expected deaths for installers of A/C sheet having one year of exposure at 2 f/cc exposure level:

Population × risk = expected deaths
(1765) (144/100,000) = 2.54 or approximately three deaths among installers.

2. Calculate expected deaths for installers of A/C sheet at PEL of 0.5/cc for one year of exposure:

Estimated risk at 0.5 f/cc = 36.1 per 100,000 (Table 17)
Population × risk = expected deaths
(1765) (36.1/100,000) = 0.64 or approximately one death among installers

3. Calculate lung cancer deaths averted or lives saved for A/C sheet installers by reducing exposure to 0.5: subtract expected deaths at 0.5 f/cc from those expected at 2/f/cc
(2.54) − (0.64) = 1.9 or approximately two lives saved.

The estimated benefits derived from exposure reductions to several PEL's which OSHA is considering for a final standard are presented in Table 2. These benefits represent the estimated number of cancer deaths avoided from those expected due to 20 years exposure at the estimated current exposure levels assuming worker exposure began at age 25. The benefits were calculated using a base of 20 years exposure because, as stated earlier, 20 years is an estimate of typical lifetime exposures for some workers. Exposures of 6 months, one year and 45 years duration are also used to show the grave danger and the need for the standard, based upon a 0.5 f/cc PEL (see Table 3).

For Table 2, OSHA bases its respirator assumptions in the construction industry and shipbuilding industry on its enforcement experience, on its familiarity with the industries, trade unions, and industrial hygienists, and on available studies on this subject. OSHA believes that these estimates may overstate the degree of respirator use. This would lead to underestimation of benefits of the ETS for the following reason: The fewer workers in compliance with the current requirements for respirator use for ambient exposures exceeding 2 f/cc, the greater the potential benefits from an ETS mandating that workers be trained regarding the significant risks from asbestos exposure and the importance of using respirators. Also, workers currently using respirators will benefit by further reductions from 2 f/cc to 0.5 f/cc. However, if respirators mandated by the ETS are not used properly or consistently, then the number of cancer deaths avoided is an overestimate.

To test the results OSHA also calculated the benefits assuming that all shipbuilding/repair and construction/renovation/demolition operations comply with the current standard. All of the shipbuilding/repair workers would thus be protected by respirators which would reduce exposures to 0.5 f/cc. The construction workers would have an exposure of 2 f/cc. The benefits of reducing the PEL to 0.5 f/cc would, therefore be calculated as the difference between expected deaths at 2 f/cc and the expected deaths at 0.5 f/cc for all workers in these segments. Table 4 shows these benefit estimates.

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Table 2

Estimates of All Cancer Deaths Avoided at Three PELs Assuming 20 Years of Exposure

Industry Segment	PEL (f/cc) 0.5	0.2	0.1
Primary Manufacturing	117	172	200
Secondary Manufacturing	471	586	624
Automotive Aftermarket	134	162	370
Shipbuilding/Repair[a]	29	39	43
Construction[b]	4974	5109	5175
Totals	5725	6068	6412

[a] OSHA assumes that 80% of workers in the shipbuilding/repair sector are protected by air-line respirators and only 20 percent may be exposed to the estimated ambient air level.

[b] OSHA assumes that only 25% of construction workers may be exposed to the estimated ambient air levels and these exposures are reduced from 20 f/cc to 0.5 f/cc as a result of the ETS.

Table 3

ESTIMATES OF CANCER DEATHS AVOIDED

Based upon lifetime risk estimates for exposures [a]

Industry Sector	PEL of 0.5 f/cc 6 Months	1 Year	20 years	45 years
Primary Manufacturing	2	8	117	174
Secondary Manufacturing	17	31	471	684
Automotive Aftermarket	4	8	134	194
Shipbuilding/Repair[b]	0	1	29	41
Construction [c]	187	378	4974	6722
TOTALS	210	426	5725	7815

[a] This table assumes exposures are reduced only to 0.5 f/cc. The number of lives saved by promulgation of the ETS may be understated in that respirators may reduce exposures below 0.5 in some industrial situations. However, the number of lives saved may be overstated if the respirators are not fully protective, or are not consistently used.

[b] OSHA assumes that 80% of workers in the shipbuilding/repair sector are protected by air-line respirators and only 20% may be exposed to the estimated ambient air level.

[c] OSHA assumes that only 25% of construction workers may be exposed to the estimated ambient air levels and these exposures are reduced from 20 f/cc to 0.5 f/cc as a result of the ETS.

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Although the results in Table 4 show fewer benefits OSHA believes the number of deaths avoided under this scenario, 80 for six months, 162 for 1 year, 2416 for 20 years, and 3513 for 45 years indicate a grave danger and necessitate this ETS action.

Table 4

Cancer Deaths Avoided with a 0.5 f/cc ETS

Assuming Compliance with the 2 f/cc Standard

Industry Sector	6 months	1 year	20 years	45 years
Primary manufacturing	2	8	117	174
Secondary manufacturing	17	31	471	684
Automotive Aftermarket	4	8	134	194
Shipbuilding/Repair*	0	0	0	0
Construction**	57	115	1694	2461
TOTAL	80	162	2416	3513

*OSHA assumes that the entire industry sector complies with the 2 f/cc standard through use of controls or respirators capable of reducing exposure to 0.5 f/cc.

**OSHA assumes that the renovation and demolition industry sectors are protected to 2 f/cc by use of half mask air purifying respirators.

3. *No Other Agency Action Is Adequate To Protect Employees Against This Grave Danger.* OSHA believes that Congress intended that the ETS authority may be exercised when OSHA determines that a grave danger is presented and when the provisions of the ETS are crafted to be an immediately effective means of protecting employees against such danger.

OSHA, however, has evalauted the potential capability of other possible actions to reduce the risk from asbestos exposure. OSHA has determined that no other agency action short of issuing this ETS which compels an immediate four-fold exposure reduction will sufficiently protect employees against the grave danger of developing asbestos related disease. In making this determination OSHA considered the usefulness of stepping up enforcement of the current asbestos standard, and initiating a 6(b) rulemaking proceeding to permanently reduce exposure levels. These actions will be discussed in turn. OSHA notes that these actions are not mutually exclusive, and in fact, OSHA is both stepping up its enforcement activities and embarking on a required 6(b) rulemaking as coordinated activities in addition to issuing an ETS.

(a) OSHA first concludes that merely increasing its enforcement of the current asbestos standard would not sufficiently reduce the grave risk of asbestos-related disease to exposed workers. Even if such increased enforcement resulted in immediately uniform compliance with the current 2 f/cc standard in all industries, risks to asbestos-exposed workers would remain unacceptably high. As stated above, OSHA has estimated that at a 2 f/cc level employees exposed over a working lifetime of 45 years are predicted to have an excess risk of dying from cancer of 84 in 1000 and of contracting disabling asbestosis of 50 in 1000; employees exposed over 20 years are predicted to have excess cancer risk of 44 in 1000 and a disabling asbestosis risk of approximately 22 in 1000. Even at exposures lasting one year, an estimated 3 employees out of 1000 are predicted to die of asbestos related cancer, and there is also additional risk of developing asbestosis. Risks at the 2 f/cc level have been acknowledged as unacceptable by other governments which have reduced their permissible levels below 2 f/cc.

To estimate the number of lives at stake if only the current standard were immediately enforced instead of compelling a 0.5 f/cc level through this ETS, OSHA made other calculations using certain assumptions about actual compliance levels.

For this analysis, OSHA assumed that all construction, renovation and demolition operations comply with the current standard so that workers in this industry would have exposures of 2 f/cc. The difference in the number of deaths avoided by compliance with the 2 f/cc standard compared with those avoided from compliance with the 0.5 f/cc ETS is still very high and shows that asbestos exposure even at 2 f/cc represents a grave danger to such exposed employees. Table 4 sets forth the results of these calculations. Thus, for a 20 year period of exposure, OSHA estimated that merely ensuring compliance with the current 2 f/cc limit may cost 2416 employees their lives. For one year of exposure, OSHA estimated that 162 employees may die if exposures are not reduced to 0.5 f/cc, assuming full compliance with the current 2 f/cc limit.

OSHA believes that these benefits (i.e. lives saved) represent the lower bound of those that would result from the ETS for two reasons. Half mask respirators which may be used at air concentrations of 20 f/cc to comply with the current standard may not effectively reduce employee exposure below 2 f/cc. Further under the ETS, half mask respirators may only be used in workplaces where concentrations do not exceed 5 f/cc. Employees in workplaces between 5 and 20 f/cc must use more protective respirators under the ETS; hence, it is likely that their actual

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exposures may be reduced to below 0.5 f/cc and the benefits of the ETS will be correspondingly increased. Further, an ETS by nature of the action itself, and the accompanying enforcement program, will undoubtedly boost the incentives to comply with all protective provisions of the asbestos standard.

(b) OSHA rejected relying on merely beginning Section 6(b) rulemaking proceedings to revise the standard to reduce the PEL as an inadequate response to the grave danger faced by asbestos-exposed workers. Beginning rulemaking proceedings results in no immediate workplace changes. Employees would still continue to be exposed to those conditions which define a grave danger for at least the pendency of the rulemaking. In OSHA's experience, completing 6(b) rulemakings not initated by an ETS concerning hazardous substances can take many years. For example, the coke oven emission standard took approximately 3½ years, the lead standard, more than 6 years and the cotton dust standard, more than 4 years. These periods do not include any of the additional delays in the effective dates of OSHA standards that were due to judicially imposed stays, which have resulted in delays lasting several years. Under the most favorable circumstances, however, OSHA believes that it is possible that a section 6(b) rulemaking limited to the issues raised herein might be completed in approximately one year, absent an ETS.

As shown above, the estimated risks of developing asbestos-related cancer due to exposure for one year under current conditions, are still extraordinarily high. The additional risks of developing asbestosis due to one year's exposure under current conditions, although quantified with less certainty, are also more than significant. OSHA also believes that the risks of six months exposure, approximated by taking over half of the one year risks under current conditions, also are unacceptably high. OSHA emphasizes as stated above, that OSHA's experience shows that without an ETS, proceedings leading to a permanent health standard are unlikely to be completed within a six month period. The explanation of OSHA's capability to produce a standard within 6 months of an ETS lies in the urgency generated by OSHA's finding of a grave danger, the existence of a specific statutory deadline to complete a rulemaking within 6 months and the need to prevent a gap in protection between the expiration of the ETS and the imposition of the permanent standard for a substance already determined to present a grave danger.

OSHA also believes that it is helpful to evaulate the extent of risk resulting from lifetime exposure periods of 20 and 45 years in the absence of this ETS. As shown in Tables 10 and 11, these risks are extraordinarily high.

4. *Other Factors Indicating a Need for an ETS.* Although worker exposure to asbestos has been declining over the years, OSHA believes that exposure conditions will continue to present grave danger in the near future unless an emergency standard is promulgated. OSHA bases this finding on its evaluation of exposure information, asbestos use statistics, consideration of the nature of industrial exposure today, and the degree of compliance with the current standard.

Although OSHA anticipates a decline in use of asbestos in products, this decline will not materially affect asbestos use in the near future. The extensive tort litigation regarding asbestos and the awareness of health effects associated with asbestos exposure provides strong inducements for producers and users of products to switch to substitutes for asbestos. In fact, asbestos consumption has declined over the years as shown:

Year	U.S Consumption Pattern (1000 tons)
1975	552
1976	659
1977	672
1978	619
1979	561
1980	360
1981	349
1982	248

Worker asbestos exposures will continue, however, for many years in the future because substitutes for some products do not appear immediately available.

For other products, old replacement parts will be needed for many years. For example, automobile manufacturers may switch to non-asbestos brakes and clutch facing in new vehicles in the next several years. Old vehicles, containing asbestos parts, will remain in use for many years thereafter. Paik et al. found over half of all multistory buildings surveyed (68 out of 127) contained sprayed on asbestos material (Ex. 84-262). The potential for asbestos exposure exists as long as asbestos remains in these buildings. Renovation work in the building such as relocating walls and electrical wiring, will result in continued exposure for carpenters, painters, electricians, laborers, sheet metal workers and others.

Therefore, OSHA does not believe that major exposure level reductions are about to occur in the near future. The trend towards reduction in asbestos use and asbestos exposure has been a slow gradual process with the exception of instances where the government intervened. For example in 1973 the EPA banned use of sprayed on insulation containing more than 1 percent asbestos. Without OSHA acting at this time, OSHA predicts that the grave danger conditions will persist, certainly during the period required for OSHA's issuance of a 6(b) standard, and probably for many years thereafter.

5. *The Need for the Specific Provisions of the ETS.* The provisions of the ETS require the employer to use any method of control to reduce employee exposure to 0.5 f/cc and to train workers concerning the hazards associated with asbestos and procedures for reducing the risk. These provisions must be implemented immediately. OSHA believes they are greatly needed to afford employees meaningful protection. As the benefit section explained in detail, compelling exposure reductions through an ETS will result in a great number of lives saved.

In addition, as stated earlier OSHA believes that its estimates of the degree of compliance with respirator requirements in the construction and shipbuilding industry may overstate the degree of present respirator use. Thus, OSHA may be understating the benefits to be derived from an ETS, and hence the need for such an ETS to be promulgated. In addition, OSHA's estimates of exposures in demolition and renovation segments of the construction industry are approximately 20 f/cc. Because under the ETS the PEL is lowered to 0.5 f/cc, the airborne concentration at which more protective

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respirators (with a protection factor exceeding 10) must be worn drops from 20 f/cc (10 times the former PEL of 2 f/cc) to 5 f/cc (10 times the new PEL of 0.5 f/cc). Therefore OSHA believes that more workers will wear more protective respirators under the ETS than under the current permanent standard, that their actual exposure levels will be reduced to below 0.5 f/cc and consequently that their risks will be reduced even more than the calculations indicate.

OSHA finds also that requiring a training program to be instituted as quickly as possible is one of the most effective methods of maximizing the beneficial impact of the exposure reduction and of all the protective provisions of the current standard. Thus, the content of this ETS has been chosen as the most effective short-term strategy to reduce asbestos risks which will be accepted and implemented.

6. *Conclusion.* OSHA finds that workers exposed to asbestos in the workplace at existing exposure conditions need this emergency temporary standard to protect them from the grave danger presented by these conditions. OSHA finds that by compelling a reduction in exposure to 0.5 f/cc for those employees presently exposed over that level, many lives will be saved. Training imposed by the ETS will enhance the risk reductions, although quantification of that additional reduction cannot be calculated. 0.5 f/cc is the lowest feasible level achievable through this emergency action, where short-term implementation of the controls is required.

Only by issuing an ETS compelling reductions in exposure levels below the current permissible limit of 2 f/cc can OSHA bring about adequate reductions in risks. The administrative action of stepping up enforcement of the current standard is an inadequate response to OSHA's finding that current conditions present a grave danger.

OSHA does not believe that any significant reduction will occur within an acceptable time period without this emergency standard action. OSHA has observed a gradual reduction in asbestos use, but, in the Agency's experience, significant and rapid exposure reductions usually occur in response to standards. The provisions of the ETS will significantly reduce the risk and reduce it quickly. Therefore OSHA has determined that the ETS is necessary to reduce the grave danger.

IV. Occupational Health Data

A. Introduction

1. *Asbestos-related Diseases.* Asbestos exposure can cause a number of disabling and fatal diseases. Among these diseases are lung cancer, cancer of the mesothelial lining of the pleura and peritoneum, and asbestosis. It is also likely that asbestos increases the risk of gastrointestinal cancers. Of all the diseases caused by asbestos, death from lung cancer constitutes the greatest health risk for American asbestos workers. Lung cancer has been responsible for over half of the excess mortality from asbestos exposure in some occupational cohorts.

The relationship between lung cancer and asbestos exposure has been established by numerous epidemiologic studies of diverse groups. Asbestos-induced lung cancer usually has a latency period in excess of 20 years and may be diagnosed at an earlier age than for non-exposed persons (Craighead et al., 1982; Ex. 84–033). Few cases of lung cancer are curable despite advances in medical and surgical oncology. Only 9% of lung cancer patients survive five or more years after diagnosis (American Cancer Society, 1983; Ex. 84–160). Asbestos exposure acts synergistically with cigarette smoke to multiply the risk of developing lung cancer.

Mesothelioma also has been conclusively shown to be associated with asbestos by many studies. In some asbestos-exposed occupational groups, 10%–18% 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 to 30 years is required in order to observe mesotheliomas in an occupational cohort. Some victims of mesothelioma have had a latency period exceeding 40 years since their initial exposure to asbestos (Craighead et al., 1982; Ex. 84–033). This form of cancer is rarely curable and is usually fatal within a year after diagnosis. There is no evidence for a relationship between cigarette smoking and mesothelioma risk.

Asbestos exposure can cause pleural and/or other pulmonary disease. Pleural plaques are one of the markers of exposure and may develop within 10 to 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 commercial types of asbestos induce plaques. Plaques can occur even when fibrosis is absent 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., 1982; Ex. 84–033).

Asbestosis is pulmonary fibrosis caused by the accumulation of asbestos fibers in the lungs. Adverse effects of asbestosis range from shortness of breath upon exertion to cyanosis, effusions of serous fluid, respiratory failure, cardiac decompensation, and death. Often, asbestosis is a progressive disease, even in the absence of continued exposure. Symptoms of 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 upon the presence of characteristic radiologic changes, symptoms, rales, other clinical features of fibrosing lung disease and a history of exposure to asbestos. Cigarette-smoking asbestos workers may have an increased risk of asbestosis relative to non-smoking asbestos workers. (Craighead et al., 1982; Ex. 84–033).

Some epidemiologic studies have observed increases in esophageal, stomach, colo-rectal, kidney, laryngeal, pharyngeal, and buccal cavity cancers. While the magnitude of increased cancer risk for these sites is not as great as for lung cancer and mesothelioma, the increased risk is nevertheless of considerable importance because of the high background rates of some of these tumors in the general population. A 50% increase in a common cancer such as colo-rectal cancer results in many more deaths than a 50% increase in a rare cancer. Colo-rectal cancer, if detected and treated in an early localized stage, has a five year survival rate of about 70% (American Cancer Society 1983; Ex. 84–160). Surgical and medical treatment is less successful for the other sites listed above.

Adverse effects from exposure to asbestos have been observed in workers involved in asbestos cement pipes and shingles manufacturing (Enterline et al., 1973a, 1973b; Weill et al., 1979; Finkelstein, 1982, 1983) (Exhibits 84–122, 84–123, 84–206, 84–044, 84–240), asbestos mining and milling (Wagner et al. 1960; Liddell et al., 1977; McDonald et al., 1980; Hobbs et al., 1980; Nicholson et al., 1979; Rubino et al., 1979) (Exhibits 2–21, 84–059, 84–065, 84–132, 84–072, 84–086), asbestos textile manufacturing (Doll, 1955; Peto et al., 1980; Berry et al., 1979;

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Dement et al., 1983) (Exhibits 84–040, 84–169, 84–020, 84–037), insulation work (Selikoff et al., 1979) (Exhibit 84–090), shipbuilding (Selikoff et al., 1979; Blot et al., 1980; Tagnon et al., 1980) (Exhibits 84–091, 84–109, 84–182), and in a variety of asbestos products manufacturing industries (Jones et al., 1980; Henderson and Enterline, 1979; McDonald and McDonald, 1978; Seidman et al., 1979; Robinson et al., 1979; Acheson et al., 1981) (Exhibits 84–138, 84–048, 84–154, 84–087, 84–082, 84–103).

It should be noted that 2 fibers per cubic centimeter, which is the current OSHA standard, is equivalent to 2 million fibers per cubic meter of air. Because humans breathe in about a cubic meter of air every hour, depending on physical exertion, the current OSHA PEL for asbestos allows workers to inhale 2 million asbestos fibers per hour during an 8-hour work day. For the sake of brevity, subsequent discussion in this preamble will express exposure in terms of fibers per cubic centimeter (f/cc) rather than fibers per cubic meter.

Note.—The current OSHA standard includes asbestos fibers 5 micrometers or more in length, thereby excluding shorter fibers. Since up to 98% of airborne asbestos fibers are shorter than 5 micrometers, workers may inhale up to 100 million asbestos fibers per hour during an 8-hour work day.

Since OSHA's publication of a notice of proposed rulemaking in 1975, additional studies have confirmed that asbestos exposure causes a high risk of cancer. In addition, much more complete data on the nature of dose-response relationships for asbestos-induced diseases are now available. These studies generally indicate that the PEL set by OSHA in 1972 is inadequate to protect asbestos workers from either lung disease or cancer.

The following agencies and organizations have reviewed the health data for asbestos: International Agency for Research on Cancer (IARC) (1977, Ex. 84–321), Organization for Economic Cooperation and Development (OECD) (1979, Ex. 84–337), NIOSH (1976, 1980, Exhibits 84–338 and 84–320), Advisory Committee of the Health and Safety Commission of the United Kingdom (1979, Ex. 84–216), the Chronic Hazard Advisory Panel on Asbestos (CHAP) (1983, Ex. 84–256), and the U.S. Environmental Protection Agency (1982, Ex. 84–180). All of these groups have concluded that there is a causal relationship between asbestos exposure and the development of cancer and non-malignant respiratory disease. NIOSH recommended reduction of the PEL for asbestos to 0.1 fibers 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 fibers per cubic centimeter (0.1 f/cc), based on the limitations of current technologies of measuring air concentrations of asbestos. The 1979 report of the Advisory Committee of the Health and Safety Commission of the United Kingdom, hereafter referred to in this section 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. Currently, it appears that the United Kingdom may lower the PEL for chrysotile to 0.5.

2. *Evaluation of Risk.* OSHA's first step in analysis of risk of disease from exposure to a potentially hazardous agent is a qualitative evaluation of scientific data. This evaluation involves reviewing human and experimental studies to consider such factors as overall study design, methods of data collection, biologic plausibility of findings, consistency of findings from different studies, temporal correctness of the association, and other factors as well as general scientific judgment.

Subsequently, after a specific agent has been judged to be hazardous, the quantitative exposure-response relationships between the agent and disease can be investigated. The available data on air concentrations of the substance or biological indices of exposure, such as fiber contents within lungs, can be reviewed for cohorts of workers demonstrated to have an increased risk of disease. If workers with an observed excess risk of disease have received cumulative exposures permitted by the current OSHA permissible exposure limit, then a potential significant health risk from exposure to the PEL has been established. If the workers with observed excess risk received cumulative exposures above those permitted by the current PEL, then risk from the current PEL may be estimated from risk observed at higher levels by using dose-response extrapolation models.

The section, *Epidemiologic Evidence on Risk from Exposure at the Current PEL*, will discuss the extent to which excess risk has been *observed* from low exposures to asbestos. Section V., *Quantitative Risk Analysis*, will discuss the *prediction* of excess risk from low asbestos exposures using dose-extrapolation models based on studies observing excess risk in humans. OSHA considers that both risks *observed* by studies and risks *predicted* by dose-extrapolation models are valid indicators of the existence of significant health risks.

Exposure data frequently are not available for workers exposed before 1970. Where historical exposure data are available, the data often have such limitations as having been collected and analyzed using industrial hygiene techniques no longer in use or having been collected in only some areas of the worksite or having been collected on only a few occasions. Therefore, of necessity, estimates of dose-response based on epidemiologic studies will have a fairly broad range of uncertainty. OSHA must examine the best available data on exposure-response to arrive at a determination of significance of risk, despite inherent and inevitable uncertainties in the data.

The current 8-hour time-weighted average PEL for asbestos of 2 fibers per cubic centimeter (2 f/cc) envisages that workers will not receive a cumulative exposure exceeding 100 f/cc-years (=2 f/cc × 50 years of occupational exposure). For asbestos, OSHA believes that a number of studies suggest that increased risk of lung cancer, asbestosis, and mesothelioma have occurred from cumulative exposures estimated as close to or below 100 f/cc-years.

Note.—OSHA typically uses 45 years as the period of a full working lifetime for purposes of quantifying risk from exposure to toxic agents. For asbestos, many scientists have used 50 years to represent a full working lifetime. Thus, both 45 years of exposure and 50 years of exposure are used in this document for the purpose of analyzing dose-response relationships for asbestos.

In these studies, the cumulative exposures resulted from exposure *levels* greater than the current OSHA PEL of 2 f/cc for an 8-hour day. For example, workers who accumulated 100 f/cc-years could have been exposed to an average level of 5 f/cc for a period of 20 years. Section B.(3), below, discusses the epidemiologic evidence for risk from low exposures in more detail. OSHA believes that a significant health risk has been observed for cumulative exposures that could be accumulated by workers exposed to no more than the present PEL of 2 fibers per cubic centimeter (2 f/cc).

Estimates of cumulative exposure are approximations of total dose received by a worker during the period of employment involving exposure to asbestos. Cumulative exposures generally are estimated by multiplying the varying intensities of exposure, such as the 8-hour time-weighted averages, by the number of year's exposed. Most theories of the mechanism of

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carcinogenesis assume that the risk of transformation of target cells to cancer cells increases with increases in the total dose. Exposures received after such a transformation has taken place may aid the development of cancer, but have not caused the cancer. Thus, in some instances, using total cumulative exposure may overstate the exposures sufficient to produce increased cancer risk. For this reason, some epidemiologists omit the later years of exposure to a carcinogen in analyses of mortality in relation to cumulative exposure.

In addition to cumulative exposure, *intensity* of exposure can be examined in relation to disease. Intensity of exposure is often approximated by 8-hour time-weighted averages (TWA's). The OSHA PEL of 2 fibers per cubic centimeter (2 f/cc) as an 8-hour TWA has only been in effect since 1976. At this time, it does not appear possible to determine whether intensity of exposure has an effect on disease risk separate from that of cumulative exposure. This is because the current OSHA PEL has been in effect only since 1976, which is an insufficient period to observe asbestos-related diseases, which characteristically have long latency periods (in excess of 25 years). As discussed in the section, *Quantitative Risk Analysis*, it appears that duration of exposure may have an independent effect on mesothelioma risk.

The following sections analyze recent epidemiologic and experimental studies and discuss important aspects of the occupational health data concerning asbestos.

B. Epidemiologic Evidence on Risk from Exposure at the Current PEL

1. *Conversion of Particle Counts to Fiber Counts.* Currently, personal asbestos samples of exposed workers are collected with a membrane filter, and fibers are counted using a phase contrast microscope equipped with an eyepiece graticule. In the past, particles were counted rather than fibers, area samples were taken rather than personal samples, and samples were collected using thermal precipitators or midget impingers. These past industrial hygiene measurements were expressed in millions of particles per cubic foot (abbreviated as mppcf or mpcf), whereas current measurements are expressed as fibers per cubic centimeter or per milliliter (f/cc or f/ml).

Conversion of measurements from mpcf to f/cc is not a simple matter of applying a single multiplicative factor due to the differing work environments in which samples were taken and differing sampling methodologies. The factor for converting mpcf to f/cc has been suggested as ranging from 1:1 to 1:5 depending on the industry studied by the scientists (Kang and Chu, Ex. 84–1; Hammad et al., Ex. 84–277; McDonald, Liddell, Gibbs, Eyssen, and McDonald, Ex. 84–065). For example, using conversion factors of 1:1 to 1:5 a cumulative exposure of 20 mpcf-years could range from 20 f/cc-years to 100 f/cc-years.

The British Occupational Hygiene Society Committee on Asbestos conducted a study of dust concentrations measured by current methods and historical methods (Ex. 84–024). Conversion factors for historical methods to current methods were 1: 0.07, 1: 2.2, and 1:2, depending on the historical method.

For asbestos mining and milling, this section will use the midpoint of the range of conversion factors: 1 mpcf = 3 f/cc. For asbestos production and asbestos cement production this section will use Hammad et al.'s suggested conversion factor of 1:1.4 for mpcf to f/cc.

2. *Epidemiologic Studies.* The epidemiologic studies that OSHA interprets as suggesting that significant health risk has been observed at low asbestos exposures fall into 3 categories: (1) Studies of household contacts of asbestos workers, (2) studies of workers whose exposures were short-term, and (3) studies of workers with cumulative exposures estimated to be close to or below 100 f/cc-years. This section summarizes these studies and presents OSHA's analysis of these studies.

A number of studies have observed mesotheliomas and an increased prevalence of chest X-ray abnormalities among families of asbestos workers (Anderson et al., Exhibits 84–016 and 84–017; Vianna and Polan, Ex. 84–186, Li et al., Ex. 84–149). In addition, mesotheliomas have been observed in community members living near asbestos mines and factories (Wagner et al., Ex. 2–21; Newhouse and Thompson, Ex. 84–070).

Anderson et al. (Ex. 84–016) observed a 35.9 percent prevalence of chest X-ray abnormalities, including pleural thickening, plaques, pleural calcification, and irregular opacities, among 626 household contacts of amosite factory workers compared to a 4.6 percent prevalence of chest X-ray abnormalities among 326 controls drawn from the same community as the amosite workers. Controls were matched to the cases by age and sex. As of 1976, 4 cases of mesothelioma had been diagnosed among the 626 family contacts of the amosite workers. Presumably, family contacts received their exposure to asbestos from dust carried home on the worker's clothing, especially during the laundering of dus clothes. About a 10-fold increase in prevalence of pleural thickening compared to controls was observed in family contacts of workers with only one year of exposure within the amosit factory.

Estimated asbestos exposure levels o family contacts and community members observed to be at risk of asbestos related disease have not been reported. OSHA considers it very likely that the cumulative exposures of the family contacts and community members were less than 100 f/cc-years.

Seidman, Selikoff, and Hammond (Ex. 84–087) studied the mortality of 820 amosite production workers employed sometime during 1941–45. Seidman et al. reported that dust concentrations had never been measured in this plant and that the plant was known to have deficient ventilation systems. 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 categories of length of employment had excessive mortality from lung cancer. For example, men employed less than one month had a lung cancer standardized mortality ratio (SMR) of 267, based on supplemental autopsy, clinical, and surgical information. [The standardized mortality ratio is calculated as the observed number of deaths in the exposed population divided by the number of deaths that would be expected in the exposed population, based on mortality rates of an appropriate comparison population. The SMR is frequently used as an approximation to the relative risk.] Hence, OSHA considers that Seidman et al. demonstrated that excess lung cancer risk could be experienced even by workers with exposures of relatively short duration.

Berry, Gilson, Holmes, Lewinsohn, and Roach (Ex. 84–020) studied the workers of an asbestos textile factory in the United Kingdom. Berry et al. reported in 1979 that the average exposure level was 5 f/cc and that 6.6 percent of men employed after 1950 had "possible asbestosis." Among men employed after 1950, Berry et al. observed 1 percent prevalences of crepitations, "possible asbestosis," or "certified asbestosis" at 37, 46, and 63 f/cc-years respectively.

Berry et al. concluded: "In view of these findings there is no room for complacency about the 2 f/cm³ standard

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and efforts should be continued to reduce asbestos dust to as low a level as possible. At this stage it is impossible to state definitely that the standard is inadequate, because its introduction is so recent, and it is essential to follow up groups exposed to low levels in order to improve the data necessary for the formulation of better standards" (Ex. 84–020, p. 109).

Finkelstein (Ex. 84–044) studied the development of compensable (certified) asbestosis among 157 Ontario asbestos cement production workers in relation to cumulative exposures (f/cc-years). All workers in the study cohort had at least 15 years of exposure. Ontario criteria for certifying asbestosis, which results in the award of disability pensions, are not inflexible and involve consideration of such factors as history of occupational exposures, dyspnea, crepitations, clubbing of fingers, radiographic signs of pulmonary fibrosis, and abnormal lung function. Certification of asbestosis hence would occur at an advanced stage of disease. Each production worker was classified as having received 0–49, 50–99, 100–149, 150–199, or 200–249 f/cc-years of cumulative exposure within the 18 years following initial exposure.

Finkelstein calculated incidence rates consisting of number of new cases of certified asbestosis per 100 person-years at risk. [Incidence is the rate at which new cases of asbestosis develop in a given period of time. It is a direct measure of the risk of developing the disease.] Incidence rates were 0.5, 3.4, and 6.5 for workers who received 0–49, 50–99, and 100–149 f/cc-years respectively. Finkelstein also calculated the cumulative probability of having developed certified asbestosis by the end of 32 years of latency, and observed that men in the 0–49, 50–99, and 100–149 f/cc-years categories had about 10 percent, 55 percent, and 70 percent probabilities respectively. Due to small numbers of men in each category, these estimated probabilities had much statistical uncertainty.

Finkelstein stated that the uncertainties in exposure assignments and the selection bias arising from exclusion from the study of workers with less than 15 years of employment may have led to overestimation of risk at low exposures, which was "to some extent balanced by the fact that the end point we studied was certified disability, an advanced stage of asbestosis" (Ex. 84–044, p. 501). OSHA considers that Finkelstein's findings of excess risk from low cumulative exposures are very important because the outcome, certified asbestosis, was based upon substantial medical evidence of severe disability from the disease.

Dement and colleagues conducted a detailed study of plant processes and dust control methods at a chrysotile textile plant during 1930–1975 (Ex. 84–036, 84–037). Exposure histories were constructed for each worker and estimates of individual cumulative exposures in terms of f/cc-days were made. After 1940, exposure levels usually were in the range of 5–10 f/cc. Workers were categorized as receiving exposures of less than 1000; 1000–10,000; 10,000–40,000; 40,000–100,000; and greater than 100,000 f/cc-days. Because Dement et al. included holidays and weekends in their estimates of f/cc-days, their estimates of cumulative exposure are likely to be overstated. OSHA calculated that Dement et al.'s categories of f/cc-days are roughly equivalent to less than 2.7; 2.7–27.4; 27.4–109.6; 109.6–274; and greater than 274 f/cc-years of occupational exposure.

Note.—Because Dement et al. counted holidays and weekends for their calculations of f/cc-days, OSHA divided f/cc-days by 365 to arrive at estimates of f/cc-years.

Dement et al.'s first 3 exposure categories fall within the range of cumulative exposures permitted by the current OSHA asbestos standard.

Among white males with 15 or more years of latency, lung cancer SMR's were 140, 279, and 352 in the categories of less than 1000, 1000–10,000, and 10,000–40,000 f/cc-days respectively. The latter 2 categories' excesses of lung cancer were statistically significant. For other non-malignant respiratory disease (excluding both infectious respiratory diseases and bronchitis), the following SMR's were observed for white males with 15 or more years of latency: 362 for men with less than 1000 f/cc-days, 84 for men with 1000–10,000 f/cc-days, and 879 for men with 10,000–40,000 f/cc-days. The excesses were statistically significant for both the first and third category of cumulative exposure.

Dement et al. concluded: "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" (Ex. 84–037, p. 432).

OSHA considers that Dement et al.'s observations of excess risk from low cumulative exposures are well-supported because of the careful estimation of exposure histories for the cohort.

Henderson and Enterline (Ex. 84–048) studied the mortality of 1075 asbestos production workers who retired during 1941–1967. Mortality was observed during the period 1941–1973. Cumulative exposures, expressed in millions of particles per cubic foot of air times years exposed (mpcf-years), were estimated for each man included in the study cohort. Mean exposures for 6 cumulative exposure categories were 62, 182, 352, 606, and 976 mpcf-years.

Hammad et al. (Ex 84–277) suggested a mpcf to f/cc conversion factor of 1:1.4 for a cement plant. Because cement and other mineral particles have been extensively used in asbestos products manufacturing, using a conversion factor of 1:1.4 does not appear to be unreasonable for Henderson and Enterline's study cohort. Using a factor of 1:1.4, 62 mpcf-years is roughly equal to 87 f/cc-years.

A respiratory cancer SMR of 197.9 was observed in the cumulative exposure category with a mean exposure of 62 mpcf-years. Thus, OSHA views this study as having observed excess risk of dying from respiratory cancer among men receiving cumulative exposures permitted by the current OSHA PEL. Using a conversion factor of 1:3 would result in an estimate of cumulative exposure for the low exposure category that is about 90% higher than that envisaged by the current OSHA PEL.

Dement et al. commented on the marked differences between the studies of Dement et al. and Henderson and Enterline with regard to risk observed from low cumulative exposures (Ex. 84–037). Dement et al. suggested that these differences may be attributable to the fact that Henderson and Enterline studied retirees 65 years or older, with these retirees constituting a select group of survivors. Only 8 of the 35 lung cancer deaths observed by Dement et al. occurred in persons 65 or older.

McDonald, Liddell, Gibbs, Eyssen, and McDonald (Ex. 84–065) studied the mortality of 11,379 workers exposed to chrysotile mining and milling born during 1891–1920. Mortality was observed during the period 1910–1975. Each worker was classified as having accumulated less than 30, 30–299, or 300 or more mpcf-years by age 45. Using a conversion factor of 1:3 for mpcf to f/cc these groupings would correspond to less than 90, 90–899, and 900 or more f/cc-years. Lung cancer SMR's were 93, 118 and 225 for the 3 categories starting with less than 30 mpcf-years respectively. SMR's for pneumoconiosis were 298, 1081, and 5400 for cumulative exposure categories of less than 30, 30–299, and 300 or more mpcf-years. Hence, McDonald et al. did not observe much lung cancer risk but did observe an

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increase in pneumoconiosis risk in the low exposure category.

A case-control analysis used similar exposure categories; however, the analysis was intrinsically incapable of observing an excess risk in the less than 30 mpcf-year category because the controls had also been chrysotile miners and millers.

Regarding the different findings on lung cancer risk from low exposures between the studies by McDonald et al. and Dement et al., Dement et al. suggested imprecision of exposure estimates and differences in airborne fiber characteristics as possible reasons for the differences. Dement and Harris previously had found that textile processing produced a higher proportion of long thin fibers falling within the ranges delineated as carcinogenic by the animal studies of Stanton and colleagues (See Section E(3) below, *Experimental Data).*

Finkelstein (Ex. 84–240) also studied mortality from lung cancer, mesothelioma, and other diseases among workers at an Ontario asbestos cement factory established in 1948. The study cohort consisted of 339 men hired before 1960 who had been employed at the factory for 9 years or more. Each cohort member was classified as having accumulated 8–69, 70–121, or 122–420 f/cc-years of asbestos exposure within the 18 years following initial exposure. These exposure categories, hereafter referred to as Groups A, B, and C, had the following mean cumulative exposures respectively: 44, 92, and 180 f/cc-years. Groups A and B are low exposure groups.

Mortality by cumulative exposure was analyzed starting 20 years after initial exposure, so that the calendar period during which deaths were observed spanned 1968–1980. Cohort mortality was compared to that of Ontario men during 1970–1974. Age-standardized mortality rates per 1000 man-years for specific causes of death were computed for Groups A, B, and C and for Ontario men. Thus, this study did not have the biases from confounding effects of age that can occur when SMR's are being compared among different groups of workers (See Ex. 84–335). One potential problem is that lung cancer mortality may have risen in Ontario during 1975–1980; this would result in some underestimation of comparison values and hence some overestimation of lung cancer risk. However, this overestimation of risk likely would be slight.

Mesothelioma mortality rates per 1000 man-years for Groups A, B, and C were 1.9, 4.9, and 11.9 respectively. Lung cancer mortality rates per 1000 man-years were 13.6, 26.1, 11.9, and 1.6 for Groups A, B, and C and for Ontario men respectively. Approximate relative risks for Groups A, B, and C, as compared to Ontario men, are 8.5, 16.3, and 7.4 for lung cancer mortality. Gastrointestinal cancer mortality was also elevated in Groups B and C relative to Ontario men.

The lung cancer mortality rates did not consistently increase with increasing estimates of cumulative exposure in that Group C had the lowest lung cancer excess. Finkelstein suggested several potential explanations, including inaccuracy of exposure classifications, statistical fluctuations resulting from the small size of the cohorts and confounding effects of smoking if there were differences in smoking habits among Groups A, B, and C. Because a consistent dose-response was observed both for mesothelioma mortality and asbestosis in the study group, Finkelstein suggested that the exposure classifications may have been correct.

Finkelstein concluded that lung cancer mortality rates "may be raised several-fold", (Ex. 84–240, p. 143) at cumulative exposures of 100 f/cc-years. With regard to gastrointestinal cancer, Finkelstein judged that no firm conclusions could be drawn due to the small number of deaths, although there was a trend of increasing risk with increasing cumulative exposure. Mesothelioma death rates were considered by Finkelstein to be related to cumulative exposure.

OSHA believes that Finkelstein's study presents evidence of excess lung cancer and mesothelioma risk from relatively low cumulative exposures, namely 44 and 92 f/cc-years. Lung cancer risk may have been somewhat understated by Finkelstein's exclusion of lung cancer deaths which occurred before 20 years of follow-up.

Rubino, Piolatto, Newhouse, Scansetti, Aresini, and Murray (Ex. 84–086) studied the mortality of 952 male Italian chrysotile miners and millers during 1946–1975. Mortality from respiratory disease and other causes of death, but not from lung cancer, was excessive in this cohort. Criteria for inclusion in the study cohort were survival until January 1, 1946, and at least one month of employment during 1930–1965. Comparison of mortality was made to age, calendar period, and cause-specific mortality rates of Italian males. In addition, mortality rates were compared among the cohort members using a case-control analysis and a historical prospective analysis. The cumulative fiber exposure was estimated for each worker in terms of f/cc-years. The investigators simulated past working conditions and measured dust concentrations during these simulations in order to make more accurate estimates of exposure during periods (pre-1969) in which exposures were not measured. Mean concentrations of asbestos were estimated to range up to 50 f/cc before 1950.

Compared to Italian males, the overall cohort had statistically significant excesses of mortality from laryngeal cancer, non-malignant respiratory diseases, tuberculosis, cardiovascular diseases, cirrhosis of the liver, and accidents. Lung cancer mortality was elevated only slightly (SMR = 106); however, there was some trend of increasing lung cancer risk with increasing length of follow-up. For example, the SMR for lung cancer was 206 during 1971–1975.

Because all analyses of mortality in relation to cumulative exposure consisted of comparisons among the cohort members, rather than between the various exposure subgroups and Italian males, these analyses were incapable of detecting excess risk in the lowest cumulative exposure category. These analyses were capable only of detecting trends toward increased risk with increased exposure. Although the excess lung cancer risk (odds ratio = 2.89) in the high exposure group was not statistically significant. Rubino et al. considered it "likely that the increasing mortality truly reflects the effects of higher exposure" (Ex. 84–086, p. 193). [The odds ratio is an estimate of the relative risk defined as the ratio of the odds of dying from cancer in the exposed population to the odds of dying from cancer in an unexposed population.]

Rubino et al.'s study is unusual in not finding a higher lung cancer risk among workers exposed to relatively high levels of asbestos. OSHA believes that the trends of increasing lung cancer risk with both increasing length of follow-up and increasing exposure are consistent with an asbestos etiology, however. OSHA also believes that the high risks of mortality from other causes such as non-malignant respiratory disease might have obscured lung cancer risk, especially since these high risks appeared within the first 19 years following exposure and asbestos-related lung cancer generally has a longer latency period. Also, OSHA calculated that this study only had 33.5% power to detect a 50% increase in lung cancer risk among workers with 20 or more years of follow-up.

Note.—Statistical power quantifies the ability of a study to detect a true increased risk of a specified magnitude and refers to the

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probability of not missing a true risk. Generally, it is considered desirable for studies to have at least 80% power. Because lung cancer is a relatively common cancer, epidemiologic studies should have at least 80% power to detect a 50% increase in risk of lung cancer. See Ex. 84–336 for a description of OSHA's method of calculating power.

Weill, Hughes, and Waggenspack (Ex. 84–206) investigated exposure-response relationships between respiratory cancer risk and exposure in an asbestos cement plant. Weill et al. did not observe excess risk among men with low cumulative exposures. A total of 5,645 men with at least 20 years of latency since first exposure in either of 2 asbestos cement plants were studied. All men had at least one month of employment before 1970 and their vital status was determined as of December 31, 1974.

Each worker's cumulative dust exposure during the 20 years from initial exposure was estimated in terms of mpcf-years. Men were classified in one of 5 different cumulative exposure categories: 10 or fewer, 11–50, 51–100, 101–200, and 201 or more mpcf-years. Using the conversion factor of 1:1.4 suggested by Hammad et al., the 5 cumulative exposure categories would be equivalent to 14 or fewer, 15–70, 71–140, 141–280, and 281 or more f/cc-years. Respiratory cancer SMR's were as follows, starting with the lowest category: 77, 70, 26, 290, and 226. None of the other causes of death were in excess for workers in the 3 lowest categories.

Concerning their failure to detect excess respiratory cancer mortality in their lowest categories of exposure, Weill et al. stated: "Such findings are not necessarily incompatible with a linear response curve at low doses because of the relative insensitivity of currently used epidemiologic methods in detecting slight increases in risk when compared to background. They do indicate, however, that any excess risks at low degrees of exposure are small" (Ex. 84–206, p. 353).

Weill et al. noted that the relatively high proportion (25%) of the cohort who were lost to follow-up and assumed alive through 1974 may have led to underestimation of respiratory cancer risk. The upper limits of the 95% confidence intervals of the respiratory cancer SMR's for the 3 lowest exposure categories ranged from about 115 to 150, indicating, in OSHA's opinion, that excess risk could not be ruled out for these categories.

Berry and Newhouse (Ex. 84–021) studied mortality during 1941–1976 of a cohort of friction material production workers whose exposures were relatively low. Levels of exposure ranged from less than 1 f/cc to 5 f/cc after 1931, and cumulative exposures for the cohort averaged less than 50 f/cc-years. Although excessive mortality from mesothelioma was observed, there appeared to be little excess mortality from lung cancer. Most of the mesothelioma cases had been exposed to asbestos levels exceeding 5 f/cc. The cumulative exposures to asbestos of the mesothelioma cases were not reported by the authors.

Unexpectedly, this study observed excessive mesothelioma mortality but only a non-significant excess of lung cancer mortality. A sizable portion of the study cohort had a short follow-up period between their initial exposure and the study cut-off date. For example, 33% of the men had follow-up periods of less than 20 years. Lung cancer risk may be expected to increase in this cohort as the members are followed for a longer period.

3. *Summary.* A number of epidemiologic studies have examined exposure-response relationships for asbestos and asbestos-related diseases. OSHA recognizes that there are many inevitable uncertainties associated with epidemiologic studies of exposure-response. Rarely, if ever, are personal samples of asbestos concentrations for exposed workers during the entire period of exposure available. Investigators typically have developed individual indices of exposures from reconstructed occupational histories, recent industrial hygiene data, and assumptions about past working conditions for which exposures were not measured. Another question concerns the factors for conversion from historical methods of measuring dust concentrations to current methods of measuring fiber concentrations. The ratio of millions of particles per cubic foot (mpcf) to fibers per cubic centimeter (f/cc) has been suggested as ranging from 1:1 to 1:5. Conversion factors of 1:3 for mpcf: f/cc in asbestos mining and milling and 1:1.4 in asbestos production and asbestos cement operations appear reasonable to OSHA.

OSHA also recognizes the statistical variation associated with estimation of Standardized Mortality Ratios (SMR's). Such statistical variation, as well as differences in study design, statistical power, and length of follow-up, may account for some of the divergent findings among studies.

Because the present OSHA PEL of 2 f/cc was effective in 1976, there are few, if any, occupational cohorts exposed solely to 2 f/cc or less who have follow-up intervals sufficient for the appearance of diseases related to asbestos exposure. At present, OSHA is not aware of any evidence suggesting that *intensity* of exposure will affect excess risk in a manner different from *cumulative exposure*, which encompasses both duration of exposure and intensity of exposure. Most epidemiologic studies have observed increasing risk with increasing cumulative exposure. *Duration* of exposure may have an independent effect on mesothelioma risk (see Section V., Quantitative Risk Analysis).

A worker exposed to the OSHA PEL of 2 f/cc for 50 years would have a cumulative exposure of 100 f/cc-years. Hence, studies of workers estimated to have received close to or less than 100 f/cc-years provide evidence concerning risk from exposure to the OSHA standard, even though the past intensities of exposure may have exceeded 2 f/cc.

OSHA considers that the following studies have observed increased risk close to or below 100 f/cc-years. Berry et al. (Ex. 84–020) observed a 1% prevalence of crepitations, possible asbestosis, and certified asbestosis at 37, 46, and 63 f/cc-years respectively among asbestos textile workers. Finkelstein (Ex. 84–044) observed a 10% probability of having certified asbestosis among asbestos cement production workers with cumulative exposures of less than 50 f/cc-years who had been observed 32 years since their initial exposures. Finkelstein (Ex. 84–240) also observed excessive lung cancer mortality among workers with average cumulative exposures of 44 and 92 f/cc-years.

Among asbestos textile workers, Dement et al. (Ex. 84–036) observed excessive mortality from lung cancer and non-malignant respiratory diseases at cumulative exposures of less than 1000 f/cc-days and 1000–10,000 f/cc-days (equivalent to less than 2.7 and 2.7–27.4 f/cc-years). Henderson and Enterline (Ex. 84–048) observed about a 2-fold increase in lung cancer mortality among retired asbestos production workers receiving an average cumulative exposure of 62 mpcf-years (equivalent to 87 f/cc-years using a conversion factor of 1:1.4).

At relatively low cumulative exposures, the following studies did not observe excess lung cancer risk or observed only a small increase in lung cancer risk. Berry and Newhouse (Ex. 84–021) observed little excess lung cancer mortality among friction material production workers whose cumulative exposures averaged less than 50 f/cc-years. Mesothelioma cases were observed by Berry and Newhouse;

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however, their cumulative exposures were not reported. Weill et al. (Ex. 84–206) did not find increased mortality from any cause of death below 101 mpcf-years (equivalent to 141 f/cc-years using a conversion factor of 1:1.4) in a cohort of asbestos cement workers. Rubino et al. (Ex. 84–086) observed only a slight excess of lung cancer mortality (and a large excess of mortality from other causes, such as non-malignant respiratory disease) despite high exposure levels. Data were not analyzed by Rubino et al. in such a way as to permit estimation of risk from low cumulative exposures.

McDonald et al. (Ex. 84–065) observed little or no excess lung cancer mortality among asbestos miners and millers receiving low cumulative exposures. Excess mortality from pneumoconiosis was observed in the low exposure group, however. Workers receiving less than 30 mpcf-years by age 45 had an SMR of 298 for pneumoconiosis (30 mpcf-years equals 90 f/cc-years using a conversion factor of 1:3).

As is commonly observed among epidemiologic studies of etiologic agents for disease, there are some inconsistencies in the findings of different studies of workers accumulating relatively low exposures. Statistical variation, differences in the size distribution of airborne fibers, imprecision of exposure estimates, and competition among different causes of death might explain some of the inconsistencies. Nonetheless, OSHA considers that many well-conducted studies observed substantially increased risk of death from lung cancer and non-malignant respiratory disease among workers receiving cumulative exposures permitted by the current OSHA standard. OSHA concludes that these study results provide evidence of grave danger from low cumulative exposures to asbestos.

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

1. *Introduction.* A number of studies of asbestos workers have observed excesses of cancer at sites other than the lung and mesothelium. These sites include colon and rectum, esophagus, stomach, larynx, pharynx, buccal cavity, kidneys, and ovaries. Based on these studies, OSHA has concluded that gastrointestinal malignancies appear to have been produced by asbestos inhaled in the workplace.

A variety of community-based epidemiology studies have investigated the effects of ingestion of asbestos in drinking water. One study suggested that asbestos in drinking water increased cancer incidence in San Francisco and Oakland (Kanarek et al.), while other studies did not observe a relationship between asbestos in drinking water and cancer incidence (Harrington et al., Meigs et al., Levy et al.) (Kang. Ex. 84–139). OSHA is aware that ecological studies generally have certain limitations for determining the effects of long-term environmental exposure to specific substances, including confounding variables such as migration into and from communities and multiple exposures to other carcinogens and toxic chemicals. For example, increased cancer mortality in a particular community may result from occupational exposures rather than from carcinogens in the drinking water or ambient air. Because studies of occupational cohorts do not have as many limitations as ecological studies, occupational studies have the potential to be more determinative concerning carcinogenic risk to humans, depending on size of the cohort, length of observation of the cohort, and other pertinent factors. Therefore, because well-conducted epidemiologic studies of asbestos workers are available and because inhalation rather than ingestion is the primary route of workplace exposure, OSHA has based its conclusions on the potential carcinogenicity of asbestos for sites other than the lung and mesothelium on epidemiologic studies of asbestos workers.

2. *Epidemiology Studies.* Elmes and Simpson studied the mortality of Belfast insulators employed during 1940, mostly in shipyards (1971, 1977) (Exhibits 84–041 and 84–042). Vital status was observed during the period 1940–1975. Excess mortality was observed from asbestosis, lung cancer, mesothelioma, and gastrointestinal cancers (stomach, jejunum, pancreas, colon and rectum).

Elmes and Simpson stated that "both in respiratory and in gastrointestinal cancers there is the difficulty of differentiating between mesothelial and epithelial tumours" (Ex. 84–042, p. 176). Of the total 122 deaths in this cohort, either autopsy information or clinical information with biopsies and/or radiographs supplemented the death certificates for all but 22 deaths. By 1966, 13 deaths had been coded as gastrointestinal cancers on death certificates, 12 of which continued to be classified as gastrointestinal cancers after supplemental information had been obtained. Using the expected value of 5.16 deaths for all non-respiratory cancers that were reported by Elmes and Simpson, OSHA calculated that the gastrointestinal cancer excess was statistically significant at the 0.05 level (one-tailed Poisson test).

Elmes and Simpson concluded in their 1971 paper: "Cancer of the lung and mesotheliomas do not account for all the excess of deaths; a significant excess of other cancers remain . . . and most of these appear to be in the gastrointestinal tract" (Ex. 84–041, p. 235). In their 1977 paper, Elmes and Simpson reported a decline in both asbestosis and gastrointestinal cancer as major causes of mortality among the survivors followed from 1967–1975.

Selikoff, Hammond, and Seidman studied a cohort of 17,800 U.S. and Canadian insulation workers (Ex. 84–090). Comparison of insulators' mortality during 1967–1976 was made to age and calendar period-specific mortality rates of U.S. white males. The large size of this cohort resulted in very high statistical power to detect increased mortality from specific causes. Therefore, OSHA considers that this study carries much weight with regard to the question of asbestos-induced malignancies. The investigators sought supplemental clinical, surgical, and autopsy information in order to determine the extent of misclassification of cancers.

Based on death certificate information alone, significant excess mortality was observed from lung cancer (SMR=406), mesothelioma (104 deaths), esophageal cancer (SMR=253), stomach cancer (SMR=126), colon-rectal cancer (SMR=152), laryngeal cancer (SMR=191), pharyngeal and buccal cavity cancer (SMR=159), kidney cancer (SMR=223), all other cancers (SMR=191), and non-infectious respiratory diseases (SMR=319), including 78 deaths from asbestosis. With the exception of deaths listed as "all other cancers", reclassification of deaths based on supplemental clinical, autopsy, and surgical data resulted in slightly higher SMR's for the causes of death listed above.

OSHA believes that Selikoff et al.'s reclassification of causes of death is justifiable because misdiagnosis of mesothelioma and asbestosis can occur due to these conditions' resemblance to more common diseases. Furthermore, it is possible that metastases from primary lung cancers could be misdiagnosed as primary cancers of other sites, and vice-versa. One disadvantage of reclassification of deaths, however, is that the extent of misclassification in the comparison population of U.S. white males remains unknown. Nonetheless, OSHA believes that, for this particular cohort, the advantage of reclassification in terms of improving the certainty about the causes of death outweighs the disadvantage.

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In the category listed as "all other cancers," 252 deaths were observed compared to 131.8 deaths expected. The excess in this category was mostly due to increased mortality from pancreatic cancer (SMR=281), liver and biliary tract cancer (SMR=265), prostate cancer (SMR=137), and brain cancer (SMR=163). When reclassified according to supplemental information, the pancreatic cancer excess declined greatly and the liver and biliary tract cancer excess disappeared.

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 death from lung cancer, pleural mesothelioma, peritoneal mesothelioma, cancer of the esophagus, colon and rectum, cancer of the larynx, oropharynx, 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, 114).

Dement and collegues (Ex. 84–037) observed a statistically non-significant increase in digestive cancer mortality (SMR=131). When Dement et al. confined their analysis to white males with latency intervals of 15 or more years and examined exposure-response relationships for digestive system cancer, SMR's increased with increasing cumulative exposure, ranging up to 390. Because of the small numbers of deaths, none of the excesses of digestive cancer in any of the exposure categories were statistically significant.

McDonald and colleagues (Ex. 84–065) observed some increases (not statistically significant) in mortality from esophageal and stomach cancer and from colo-rectal cancer among chrysotile miners and millers in Quebec. Gastrointestinal cancer SMR's increased with heavier cumulative exposures.

Seidman, Selikoff, and Hammond (Ex. 84–087) studied a cohort of 820 amosite insulation production workers employed at a New Jersey plant during 1941–1945. A statistically non-significant excess of gastrointestinal cancer (esophagus, stomach, and colon-rectum) was observed (SMR=121). Henderson and Enterline (Ex. 84–048) studied the mortality during 1941–1973 of 1348 retired asbestos factory workers. Fifty-five deaths from digestive system cancer were observed compared to 39.9 deaths expected (SMR=137.8) (OSHA calculated that this excess was statistically significant at the 0.05 level, using a one-tailed Poisson test). Nicholson, Selikoff, Seidman, and Hammond (Ex. 84–251) also studied a cohort of U.S. asbestos factory workers and observed a statistically non-significant 1.5-fold excess of gastrointestinal cancer. Newhouse and Berry (Ex. 84–330) observed a gastrointestinal cancer SMR of 136 in a cohort of London asbestos factory workers (60 deaths observed versus 44.2 expected) (OSHA calculated that this excess was statistically significant at the 0.05 level using a one-tailed Poisson test). Puntoni et al. (Ex. 84–246) studied the mortality of shipyard workers exposed to asbestos in Genoa, Italy. These workers were also exposed to other toxic substances, including silica, benzene, carbon tetrachloride, and polycyclic aromatic hydrocarbons. Compared to the age-specific mortality rates of male Genoans, the shipyard workers had statistically significant increases in mortality from colon cancer (relative risk=1.81). All of the studies listed in this paragraph also observed excessive mortality from lung cancer.

In addition, other investigators have observed excesses of digestive sytem cancer. Robinson et al. (Ex. 84–082) observed an SMR for digestive system cancer of 121 (not a statistically significant excess) among asbestos production workers. Kleinfeld et al. (Ex. 84–140, 84–141) observed an SMR of 400 (statistically significant) for digestive system cancer among tremolite and anthophyllite exposed workers mining talc. Mancuso and Coulter (Ex. 84–224) also observed a significantly elevated digestive system cancer SMR among insulation workers. Finkelstein observed a statistically non-significant 2-fold increase in mortality from digestive system cancer among asbestos cement workers (Ex. 84–240).

A number of studies have not observed excessive gastrointestinal cancer mortality among workers exposed to asbestos. Nicholson, Selikoff, Seidman, Lilis, and Formby (Ex. 84–072), who studied the mortality of 544 chrysotile miners and millers from Quebec, observed a gastrointestinal cancer SMR of only 105. Weill, Hughes, and Waggenspack (Ex. 84–206), who studied a cohort of 5,645 asbestos cement production workers, also did not observe an excess of deaths from gastrointestinal cancer. Other investigators who did not observe an increase in gastrointestinal cancer mortality include Berry and Newhouse (friction materials production workers), Rubino et al. (chrysotile miners), Meurman et al. (anthophyllite miners), Brown et al. (talc workers exposed to asbestos). Weiss (chrysotile factory workers), McDonald and McDonald (asbestos gas mask workers), Peto et al. (asbestos textile workers), Thomas et al. (asbestos cement workers), Rossiter and Coles (shipyard workers), and Jones et al. (asbestos gas mask workers) (Ex. 84–256).

When non-gastrointestinal cancers at sites other than the lung and mesothelium are considered, at least 4 studies observed excesses at these other sites: Selikoff et al., Shettigara and Morgan, Puntoni et al., and Stell and McGill. Other studies have not observed excesses of cancers at other sites.

One issue which the Agency had to address was how to weigh the positive epidemiologic data versus the non-positive epidemiologic data. One consideration is statistical power, the ability to detect a true risk if such a risk exists. The Chronic Hazard Advisory Panel on Asbestos (CHAP) convened by the U.S. Consumer Product Safety Commission did not find a consistent relationship between having a higher degree of statistical power and finding excessive mortality from these other cancers (Ex. 84–256). It should be noted that the study that had the highest statistical power by virtue of studying 17,800 workers, namely that of Selikoff et al., observed excesses of gastrointestinal, laryngeal, kidney, and pharyngeal and buccal cavity cancer.

Another consideration is the relationship between the magnitude of excess lung cancer risk and excess gastrointestinal cancer risk. CHAP observed that studies with high lung cancer excesses were also likely to have found gastrointestinal cancer excesses. Because high lung cancer risks would be expected to have resulted from relatively high exposures, this observation suggests that gastrointestinal cancer excesses are found where exposures are greater, which increases the plausibility of there being a true association between asbestos and gastrointestinal cancer.

In summary, at least 12 different occupational cohorts exposed to asbestos have been observed to have excesses of mortality from gastrointestinal cancer, 7 of which were statistically significant. OSHA considers that these findings constitute substantial evidence for an association between asbestos exposure and gastrointestinal cancer risk. The evidence for a relationship between asbestos exposure and cancer at other sites is noteworthy yet not as substantial as that regarding gastrointestinal cancer.

Goldsmith (Ex. 84–235) reviewed the evidence for a causal relationship between asbestos exposure and non-

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pulmonary cancer in 8 studies of 11 occupational cohorts followed for at least 20 years since initial exposure. He concluded that the data "cast doubt on whether there is site-specificity of asbestos-related cancer" and that "a systemic carcinogenic role is more likely [for asbestos]" (Ex. 84–235, pp. 346–347).

3. *Toxicology Studies.* A number of toxicology studies have been conducted to determine the carcinogenicity of ingested asbestos. Inhaled asbestos is thought to enter the digestive tract when asbestos fibers caught in the mucous lining of the lung are brought up to the throat and swallowed. Also, fibers caught in the nose may travel down to the pharynx (Kang, Ex. 84–139). Evans et al. (Ex. 84–236) conducted an inhalation study in rats of radio-labelled crocidolite. The radio-labelled crocidolite traveled to the larynx, esophagus, and gastrointestinal tract immediately after exposure, and was largely excreted in the feces within the 30 days following inhalation.

The National Toxicology Program (NTP) has administered chrysotile, amosite, tremolite, and crocidolite in feed (1% of diet) to laboratory animals. Increased incidences of tumors have not been reported in any of the NTP studies (Exhibits 84–225, 84–226, 84–227, 84–228). In some of the studies, relatively short fibers were administered. The asbestos dose of 1% in diet was not the maximally-tolerated dose (MTD), unlike most carcinogenesis bioassays.

Ward, Frank, Wenk, Devor, and Tarone (Ex. 84–200) examined the effect of oral exposure to amosite or chrysotile on gastrointestinal (GI) carcinogenesis among F344 rats who had been injected with azoxymethane. Azoxymethane is a well-recognized intestinal carcinogen for laboratory animals. Rats were also exposed to amosite alone and azoxymethane alone. The exposure period was 10 weeks and the rats were observed for 95 weeks or more (some rats did not survive as long as 95 weeks). Although amosite and chrysotile exposure did not significantly increase the incidence of GI cancer among rats given azoxymethane injections, the 49 rats receiving amosite alone had an unusually high incidence (32%) of colon carcinoma compared to the incidence among historical control F344 rats maintained in the same laboratory.

Ward et al. commented on these findings: "The results of these experiments suggest that oral asbestos exposure may have caused an increased incidence of intestinal tumors in male F344 rats . . Although our findings did not conclusively demonstrate the co-carcinogenic or carcinogenic effect of asbestos for the intestinal tract, our results suggest that additional sensitive animal experiments are needed" (Ex. 84–200, p. 311).

Bolton, Davis, and Lamb (Ex. 84–214) administered amosite, crocidolite, and chrysotile in diet supplements to Wistar rats for periods of up to 25 months. Malignant tumors, including GI tumors, were not increased in the asbestos-exposed animals compared to the controls; however, chrysotile-treated rats had a statistically significant excess of benign tumors. The excess of benign tumors in chrysotile-treated rats was largely due to an excess of mesenteric hemangiomas. Bolton et al. thought that the observed excess of benign tumors in chrysotile-treated animals was not likely to be due to asbestos because they did not observe asbestos fibers in the mesenteric lymphatic tissues of the animals.

Donham, Berg, Will, and Leininger (Ex. 84–222) fed F344 rats a diet consisting of 10% chrysotile. A total of 189 asbestos-fed rats and 312 control rats were studied. Regarding their findings, Donham et al. stated: "Although the risk differences for development of colon tumors specifically is not statistically significant at the 5% level, we feel there is suggestive evidence that ingested asbestos may have some role in colon carcinogenesis . . ." (Ex. 84–222, p. 1080).

Smith, Hubert, Sobel, Peters, and Doerfler (Ex. 84–193) administered amosite asbestos in drinking water to Syrian hamsters of the Lak: LVG strain for periods ranging up to 22 months. There was some clustering of malignant tumors, including a peritoneal mesothelioma, a pulmonary carcinoma, and 2 squamous cell carcinomas of the forestomach, in the hamsters exposed to amosite (4 of 180 animals). None of the control animals developed these types of malignant tumors. Smith et al. did not consider the clustering of malignant tumors in amosite-exposed animals to be related to their ingestion of amosite because these types of tumors have been observed in control Syrian hamsters of the Lak: LVG strain by other investigators.

OSHA considers that there is some evidence that oral ingestion of asbestos is carcinogenic to laboratory animals; however, this evidence is rather inconsistent. The generalizability of the non-positive NTP studies is somewhat limited by the low doses and short fibers that were administered to the animals.

4. *Summary.* In summary, at least 12 epidemiologic studies have observed increased mortality from gastrointestinal cancers among workers exposed to asbestos, 7 of which were statistically significant. Also, at least 4 epidemiologic studies have observed excesses of cancer at sites other than the respiratory tract, mesothelium, and gastrointestinal tract. A number of other studies have not observed increases in mortality from cancers at sites other than the lung and mesothelium. It is possible for physicians to misdiagnose peritoneal mesothelioma as gastrointestinal or other cancers; however, the excess of gastrointestinal cancer persisted even after Elmes and Simpson and Selikoff et al. reclassified causes of death using supplemental autopsy, surgical, and clinical information. OSHA believes that the results of these reclassifications constitute additional evidence for an association between asbestos exposure and development of gastrointestinal cancer.

OSHA regards the numerous epidemiologic studies indicating increased risk from gastrointestinal cancer as outweighing non-positive epidemiologic studies and non-positive and equivocal findings in animals ingesting asbestos. Dose-response relationships for gastrointestinal cancer are characterized less well than for respiratory system cancers. Nonetheless, OSHA concludes that gastrointestinal malignancies should be included in quantitative analyses of excess cancer risk from asbestos exposure because such cancers have made substantial contributions to the increased mortality of many cohorts of asbestos workers. Otherwise, excess cancer risk from asbestos exposure would be understated.

The excesses of malignancies at sites other than the lung, mesothelium, and gastrointestinal tract observed by Selikoff et al. are particularly noteworthy because of the large size of Selikoff et al.'s cohort. The large size of their cohort resulted in a high degree of statistical power and a high degree of stability for the observed SMR's. OSHA considers that asbestos might induce cancer at sites other than the lung, mesothelium, and gastrointestinal tract based on the studies finding cancer excesses at these other sites, especially the study of Selikoff et al. OSHA will not attempt to quantify the excess risk at these other sites in relation to exposure. OSHA views the evidence for asbestos inducing gastrointestinal tract cancer as stronger and more consistent than the evidence for asbestos inducing cancer at these other sites.

D. Effects of Cigarette Smoking

1. *Introduction.* A multiplicative effect of asbestos exposure and cigarette smoking with regard to producing

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increased lung cancer risk was shown in a 1968 paper by Selikoff, Churg, and Hammond (Ex. 2–5). Subsequently, other studies of occupational cohorts confirmed this finding (Selikoff, Seidman, and Hammond, Ex. 84–190; Hammond, Selikoff, and Seidman, Ex. 84–047). Cohen et al. (Ex. 84–031) observed poorer particle clearance from the lungs of smokers than from the lungs of non-smokers. This finding may help to explain the higher lung cancer risk of smoking asbestos workers. In addition, smoking asbestos workers have been reported to be at higher risk of asbestosis and chest X-ray abnormalities, including pleural plaques (Hammond et al., Ex. 84–047; Weiss, Ex. 84–097; Weiss, Levin and Goodman, Ex. 84–099). There is no evidence for an association between cigarette smoking and either mesothelioma risk or gastrointestinal cancer risk (Hammond et al., Ex. 84–047).

2. *Respiratory Cancer.* Hammond et al. (Ex. 84–047) collected smoking histories from 8220 of the 12051 insulation workers with a follow-up period of 20 or more years since initial exposure who had been studied by Selikoff and colleagues. The mortality experience of these workers was observed during 1967–1976. Of the 8220 workers who answered the questionnaire on smoking habits in late 1966, 6841 were either current or past cigarette smokers, 488 had a history of pipe or cigar smoking, and 891 had never smoked regularly. The comparison population was drawn from the American Cancer Society's long-term prospective epidemiologic study conducted by volunteers, and 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 major advantage of this comparison population was the availability of age-specific mortality rates by smoking status. Also, men with the above described education and occupational histories likely would resemble the insulation workers more than the general U.S. white male population.

Age-standardized lung cancer mortality rates for controls and for asbestos workers are given in Table 5. As shown in Table 5, non-smoking asbestos workers had a mortality rate from lung cancer that was 5 times higher than that of non-smoking controls. The lung cancer mortality of smoking asbestos workers was also 5 times higher than the controls with a history of cigarette smoking. Thus, for both smoking and non-smoking asbestos workers, the relative risk of death from lung cancer was about 5-fold. Hence, the relationship between cigarette smoking and asbestos exposure can best be described as multiplicative in nature.

Table 5

Age-Standardized Lung Cancer Death Rates by Smoking Status and Occupational History of Asbestos Exposure

Group	Exposure to Asbestos?	History of Cigarette Smoking?	Death Rate*	Mortality Difference	Mortality Ratio
Controls	No	No	11.3	0.0	1.00
Asbestos Workers	Yes	No	58.4	+ 47.1	5.17
Controls	No	Yes	122.0	+111.3	10.85
Asbestos Workers	Yes	Yes	601.6	+590.3	53.24

*Rate per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers (based on death certificate information) From Hammond et al., Ex. 84 047

Regarding these data, the NIOSH/OSHA Asbestos Work Group stated:

"The combined effect of smoking and asbestos exposure appears to be more than simple addition. If the combined effect were additive, one would expect death rates of 169.7 per 100,000 man-years among asbestos workers who smoked. This rate was derived from the sum of the baseline rate (11.3) plus the excess over that baseline due to asbestos (58.4–11.3=47.1) plus the excess due to smoking (122.6–11.3=111.3). The data seem rather to satisfy a multiplicative model. It was shown that smoking alone increased the death rate about 11 times, and asbestos alone increased it 5 times. Therefore, for a multiplicative model, the mortality ratio for those exposed to both asbestos and smoking would be 55 (5 times 11) times greater than those who were exposed neither to asbestos nor to smoking. The mortality ratio for those exposed to asbestos and to cigarettes was actually 53.24" (Ex. 84–320, p. 27).

Selikoff, Seidman, and Hammond examined the effects of cigarette smoking and asbestos exposure among 582 amosite production workers, 567 of whom had smoking histories (Ex. 84–190). As in Hammond et al.'s study, comparison was made to age and cause-specific mortality rates within each smoking status category of the American Cancer Society (ACS) cohort. Non-smoking amosite workers had a greater than 5-fold increase in lung cancer risk compared to non-smokers in the ACS cohort, while smoking amosite workers had an almost 5-fold increase compared to their smoking ACS counterparts. Based on the excess lung cancer risk in non-smokers, Selikoff et al. stated that asbestos exposure alone produced an increased risk of lung cancer, although the increase among non-smokers was limited in terms of total numbers of additional deaths. For cigarette smoking asbestos workers, Selikoff et al. described the increased numbers of lung cancer deaths as "devastating." Selikoff et al. observed no increased risk of death from mesothelioma.

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gastrointenstinal cancer, and asbestosis ong smoking amosite workers pared to non-smoking amosite workers.

3. *Lung Disease and Chest X-ray Abnormalities.* Hammond et al. reported that asbestosis workers who smoked one or more packs of cigarettes per day had an asbestos mortality rate 2.8 times higher than that of asbestos workers who had never smoked regularly (Ex. 84–047). Hammond et al. also reported that ex-smokers who were asbestos workers had substantially lower death rates than asbestos workers who had not quit smoking.

Weiss (Ex. 84–097) conducted a chest X-ray and questionnaire survey of 100 asbestos textile workers. Weiss reported that smoking workers had a 40% prevalence of pulmonary fibrosis and non-smoking workers had a 24% prevalence of pulmonary fibrosis. A gradient in risk of fibrosis was observed for both increasing age and increasing duration of exposure. The age distributions of the smokers and non-smokers were similar, as were the median durations of exposure to asbestos. Weiss concluded that both asbestos exposure and cigarette smoking were associated with lmonary fibrosis and that smoking bestos workers had a higher prevalence of fibrosis relative to non-smoking asbestos workers.

Weiss did not indicate whether the difference in pulmonary fibrosis prevalence between smokers and non-smokers was statistically significant. OSHA tested the significance of the difference in prevalence of pulmonary fibrosis between smokers and non-smokers using a chi-squared test of proportions and did not find a significant difference (p greater than 0.1).

In 1981, Weiss, Levin, and Goodman (Ex. 84–099) reported the results of a survey of 45 men aged 40 or more who had worked 5 or more years in an asbestos manufacturing plant. The prevalence of pleural plaques was greater in cigarette smokers; however, there was some confounding of this relationship by cumulative asbestos exposure, which also seemed to influence the prevalence of pleural plaques. Weiss et al. stated:

"Conclusions are restrained by the small number of workers in this investigation. A clear-cut answer to the question as to whether the association between plaques and smoking is spurious will require a much larger research effort, preferably in a cohort study" (Ex. 84–099, p. 429).

Pearle (Ex. 84–079) surveyed 131 asbestos-exposed shipyard workers to determine the relative contribution of asbestos exposure and smoking to lung function decrements and chest X-ray abnormalities. Pearle found that both cigarette smoking and asbestos exposure reduced FEV1 (forced expiratory volume in 1 second) and FVC (forced vital capacity), with combined exposure having a "cumulative or possibly synergistic effect" (Ex. 84–079, p. 39). Pearle also reported that both smoking and asbestos exposure produced pleural and interstitial abnormalities and that smoking appeared to be the primary factor in airways obstruction and diffusion impairment.

Berry, Gilson, Holmes, Lewinsohn, and Roach (Ex. 84–020) studied 379 men employed at an asbestos textile mill as of June 30, 1966. Smoking histories were available for 376 men. Men were classified as having never smoked, as ex-smokers, or as current smokers (light, medium, or heavy cigarette consumption). The mean cumulative exposures (fibers/cubic centimeter-years) were similar among the smoking status groups, and age adjustments were made to account for the younger ages of non-smokers. Significantly greater prevalances of crepitations and small radiological opacities were observed in heavy smokers and ex-smokers compared to non-smokers and light smokers. For example, 16% of heavy smokers employed after 1950 had small radiological opacities compared to 4.8% of never-smokers employed after 1950.

Kilburn (Ex. 84–237) has reviewed the studies of the relationship between cigarette smoking and X-ray evidence of pulmonary fibrosis. Kilburn criticized Weiss's use of a definition for pulmonary fibrosis other than that of the ILO in his 1971 study, as well as the small number of workers reported on in Weiss's 1971 paper and his other papers. A study by Samet et al. which included a relatively large number of subjects and which did not find an effect of cigarette smoking on radiologic abnormalities characteristic of asbestosis was cited. Kilburn concluded that cigarette smoking neither produced X-ray appearance of pulmonary fibrosis nor contributed to fibrosis resulting from asbestos exposure.

In summary, there is some evidence that smoking asbestos workers have a higher risk of mortality from asbestosis, as well as a higher prevalence of crepitations, lung function decrements, and small radiological opacities. This evidence is inconclusive.

4. *Attributing Probable Etiologies to Lung Cancer Among Individual Asbestos Workers.* Enterline (Ex. 84–126) analyzed the probability that any single case of lung cancer in a person with known exposure to asbestos could be attributed to the asbestos exposure. His paper emphasized that it cannot be stated with *certainty* that a lung cancer in an individual worker was due to asbestos exposure; rather, statements can only be made concerning *probabilities* of cause and effect. He converted observed relative risks into probabilities using the following formula:

$$\text{Individual Probability of Lung Cancer being Due to Asbestos Exposure} = \frac{(\text{Relative Risk} - 1)}{\text{Relative Risk}} \times 100$$

Using Selikoff et al.'s data on insulators, where a relative risk of about 4.5 was observed for smoking asbestos workers and non-smoking asbestos workers alike, Enterline estimated a probability of 75% that lung cancers were attributable to asbestos exposure for both smoking and non-smoking asbestos workers ($3.5/4.5 \times 100$ is about 0.75).

Enterline's paper is important in exploring the extent to which asbestos can be considered the etiologic agent for lung cancer in exposed workers. However, in the case of smoking asbestos workers, dichotomizing causation as either due to smoking or due to asbestos does not seem appropriate to OSHA, because of the factor of synergism between cigarette smoke and asbestos. Using Selikoff et al.'s data, probabilities of causation were estimated by OSHA for each etiologic factor (See Tables 6 and 7).

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Table 6

LUNG CANCER MORTALITY BY SMOKING STATUS[1/]

Lung Cancer Rate per 100,000 per Year	
Smoking Asbestos Insulators:	362.0
Non-Smoking Asbestos Insulators:	40.4
Smoking U.S. Males:	74.4
Non-Smoking U.S. Males	9.2

[1/] From Selikoff et al., 1979 (Ex. 84-090)

Table 7

PERCENT ATTRIBUTABLE RISK FROM ASBESTOS EXPOSURE AND SMOKING

Lung Cancer Risk Attributable To:	Formula	% Attributable Risk for Asbestos Workers: Smoking	Non-Smoking
Background	9.2	2.5%	22.8%
Asbestos alone	40.4 - 9.2= 31.2	8.6%	77.2%
Smoking alone	74.4 - 9.2= 65.2	18.0%	
Interaction of Smoking and Asbestos	362 - 9.2 - 31.2 - 65.2 = 256.4	70.8%	
		99.9%	100%

While OSHA's calculations differ from Enterline's calculations of attributable risk by including the synergism factor, the probability estimates do not differ much. According to OSHA's calculations, asbestos exposure contributes to 79.4% and 77.2% of lung cancer deaths among smoking asbestos workers and non-smoking asbestos workers respectively.

5. *Summary.* OSHA considers that asbestos exposure by itself can cause lung cancer, asbestosis, mesothelioma, and gastrointestinal malignancies, thereby presenting a significant health risk to exposed workers and a grave danger under current exposure conditions. Cigarette smoking by itself can cause lung disease and lung cancer. Cigarette smoke and asbestos exposure appear to have a multiplicative relationship for causation of lung cancer. Mortality from asbestosis has been reported to be increased in cigarette-smoking asbestos workers compared to non-smoking asbestos workers, although this evidence is inconclusive. Pleural plaques and the radiologic abnormalities characteristic of pulmonary fibrosis have also been reported to be more prevalent in cigarette-smoking asbestos workers; this evidence is also inconclusive. Ex-smoking asbestos workers have been reported to have decreased lung cancer risk relative to smoking asbestos workers. There is no evidence for any relationship between cigarette smoking and induction of mesotheliomas and gastrointestinal malignancies.

E. Relative Carcinogenicity and Toxicity of Different Fibers

1. *Introduction.* OSHA has reviewed the data concerning the relative carcinogenicity and toxicity of different asbestos fiber types and has decided no to make distinctions in this emergency temporary standard by asbestos fiber type with regard to regulatory provisions designed to protect workers from the harmful effects of asbestos exposure. Mainly, this is because lung cancer is the leading cause of death associated with asbestos exposure and there do not appear to be differentials i lung cancer risk by fiber type.

Some investigators and committees have suggested that different fiber type of asbestos have differences in their carcinogenic potency, including Enterline and Henderson (Ex. 84–122), McDonald and McDonald (Ex. 84–154), Weill et al. (Ex. 84–206), Acheson and Gardner (Exs. 84–015 and 84–243), Mui (Ex. 84–350), and the Advisory Committee on Asbestos (Ex. 84–216). These scientists generally believe that crocidolite and amosite are more carcinogenic than chrysotile and anthophyllite. Based on these scientis recommendations, both the United Kingdom and the province of Ontario, Canada have promulgated the followi standards for asbestos:

0.2 fibers per cubic centimeter (0.2 f/c for crocidolite

0.5 fibers per cubic centimeter (0.5 f/c for amosite

1 fiber per cubic centimeter (1 f/cc) fc chrysotile and all other forms of asbestos

Recently, the United Kingdom has announced reductions in the limits fc chrysotile and amosite. It is importar note that the question of differentials potency by fiber type primarily conc induction of mesotheliomas, not asbestosis and lung cancer. For mesothelioma, Acheson and Gardne 1979 described "a powerful case. . . that crocidolite has been more dangerous than chrysotile and anthophyllite" (Ex. 84–216, p. 11), whereas they characterized evidenc

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fiber type differentials in risk from lung cancer and asbestosis as "inconclusive" and "slight" respectively (Ex. 84–216, p. 11).

In 1983, Acheson and Gardner stated: "[P]eritoneal mesothelioma has an almost [e]xclusive relationship with exposure to the amphiboles crocidolite and amosite" (Ex. 84–243, p. 8). Concerning lung cancer, they noted that although one study (Weill et al., Ex. 84–206) had found a higher risk among workers exposed to crocidolite, no "clear distinction" of risk by fiber type could be found when the slopes of dose-response curves were compared. Hence, they concluded that available evidence did not support the assumption that chrysotile was a less potent lung carcinogen (Ex. 84–243, p. 8).

In 1983, the Chronic Hazard Advisory Panel on Asbestos (CHAP) convened by the U.S. Consumer Product Safety Commission also reviewed the evidence for fiber type differentials in carcinogenic risk (Ex. 84–256). They concluded that there were inconsistent and inconclusive findings with regard to lung cancer differentials by fiber type. For peritoneal mesothelioma, CHAP concluded that epidemiologic studies suggested that this disease was most common in amosite workers, less common in crocidolite workers and rare or non-existent in chrysotile workers. Evidence for a fiber type differential in pleural mesothelioma risk was not [c]onsidered by CHAP to be as [s]tantial as for peritoneal mesothelioma.

Some health scientists who believe that evidence links crocidolite exposure to a substantial increase in mesothelioma risk have suggested separate regulatory treatment for crocidolite (Ex. 84–216). OSHA has been urged to consider requiring that workers exposed to crocidolite or asbestos mixtures containing crocidolite wear appropriate respirators, irrespective of airborne concentrations of asbestos and irrespective of compliance with the permissible exposure limit. Furthermore, OSHA has been urged to consider requiring that employees exposed to crocidolite be informed of the potential greater risks associated with crocidolite and the reasons for requiring respirator usage whenever crocidolite exposures occur. OSHA invites comments on this suggestion and on other alternative approaches to separate regulatory treatment of crocidolite.

There are also scientific questions concerning the relationship between fiber dimensions and ability to cause disease. OSHA believes that asbestos fibers longer than 5 micrometers (μm) [c]ause lung disease and cancer, provided [t]at the ratio of fiber length to fiber width is 3:1 or greater. Evidence for risk from fibers less than 5 μm in length is inconclusive.

A critical analysis of the evidence concerning risk differentials by fiber type and fiber dimension follows.

2. *Epidemiologic Data—Introduction:* Commercial asbestos fiber types including amosite, chrysotile, and crocidolite, have been observed to be associated with elevated risks of asbestosis, lung cancer, and mesothelioma whether exposures occurred to a single fiber type or to various combinations of fiber types (NIOSH/OSHA, 1980, Ex. 84–320). Excess lung cancer risk and asbestosis have been observed in anthophyllite asbestos workers; however, no cases of mesothelioma have been reported among anthophyllite workers.

For amosite, an association between exposure and disease has been observed by the following investigators: Seidman et al. (Ex. 84–087), Anderson et al. (Ex. 84–017), and Murphy et al. (Ex. 84–311). For chrysotile, the following investigators have obtained positive findings: McDonald et al. (Ex. 84–065), McDonald and Fry (Ex. 84–064), Liddell et al. (Ex. 84–059), Nicholson et al. (Ex. 84–072), Rubino et al. (Ex. 84–086), Dement et al. (Ex. 84–037), and Acheson and Gardner (Ex. 84–015). For crocidolite, positive findings were observed by the following investigators: Jones et al. (Ex. 84–138), Hobbs et al. (Ex. 84–132) and McDonald and McDonald (Ex. 84–154). Meurman et al. (Ex. 84–181) observed an excess lung cancer risk among anthophyllite miners in Finland. In addition, numerous studies have observed asbestosis, lung cancer, and mesothelioma among workers exposed to 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. 84–020; Elmes and Simpson, Ex. 84–042; Peto et al., 84–80; Lacquet et al., Ex. 84–144; Selikoff et al., Ex. 84–089; Robinson et al., Ex. 84–082; and Balselga-Monte and Segarra, Ex. 84–019). Also, several studies of talc miners and millers, where the talc contained tremolite and anthophyllite, have observed excesses of lung cancer and lung disease, and reductions in pulmonary function (Kleinfeld et al., Ex. 84–140, 84–141; Brown et al., Ex. 84–025; Gamble et al., Ex. 84–181). In addition, zeolite, an asbestiform mineral found in the soil and water in two villages in Turkey, may be responsible for those villages' high mortality rates from mesothelioma (Artvinli and Baris, Ex. 84–018; Baris et al., Ex. 84–110).

OSHA is not aware of any epidemiologic studies which compare workers exposed to different fiber sizes within the same industrial process. Undoubtedly, this is due to the scarcity of such occupational groups since most occupational populations have been exposed to a mixture of long and short fibers. Therefore, most of the data on differential risk by fiber size are from experimental studies rather than epidemiologic studies and will be discussed in section (D)(3) *Experimental Data.*

Evaluating the relative carcinogenicity of the different asbestos fiber types involves comparison of results from different epidemiologic studies. OSHA believes that it is important to note the potential difficulties of comparing different occupational cohorts. Standardized Mortality Ratios (SMR's) are risk measures that are dependent on the age distribution (as well as other factors) of the particular study population. This is because the values used for comparison, referred to as the expected values, are derived from mortality rates specific for age, race, sex, and calendar period in the comparison population. Cancer risk rises with increasing age, so that an older population would have higher expected values for cancer mortality.

Misleading results may be obtained when comparing risk measures for cancer among study populations with different underlying age distributions. An example of the misleading results that may be obtained in such an instance is given in Ex. 84–335, which illustrates that SMR's of diverse study populations may be the same while the actual excess risk of mortality may vary greatly because of differences in age distribution. Sex and calendar period of observation also affect expected values, because women have lower mortality rates from lung cancer and lung cancer has risen greatly since the 1940's. Length of follow-up is another pivotal variable because of the long latency periods necessary for development of lung cancer and especially mesothelioma.

Epidemiologic Studies of Lung Cancer, Mesothelioma, and Absestosis: The following studies examined the question of differential lung cancer risk by asbestos fiber type by comparing the mortality experiences of different occupational cohorts: McDonald and McDonald (Ex. 84–154), Henderson and Enterline (Ex. 84–048), Weill et al. (Ex. 84–206), McDonald and Fry (Ex. 84–064), and Acheson et al. (Ex. 84–015). Some of these studies also addressed the question of differential mesothelioma risk by fiber type. Some additional studies of workers exposed to a single fiber type are also pertinent to evaluation of risk by fiber type.

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including McDonald et al. (Ex. 84–065), Hobbs et al. (Ex. 84–132), Weiss (Ex. 84–098), Dement et al. (Ex. 84–037), and Berry and Newhouse (Ex. 84–021).

McDonald and McDonald (Ex. 84–154) suggested that crocidolite was more carcinogenic than chrysotile based on their comparison of the mortality of Canadian chrysotile miners and millers with that of Canadian workers manufacturing crocidolite-containing gas masks. For gas mask workers, 13% and 16% of the observed deaths were from lung cancer and mesothelioma respectively. In contrast, 6% and 0.26% of the observed deaths among chrysotile miners and millers were from lung cancer and mesothelioma respectively. One potential problem was that the personnel records of the crocidolite workers were incomplete, which could result in biases toward observing greater or lesser risk in this cohort. An unpublished report by Dr. Han K. Kang (Ex. 84–139) commented as follows on this comparison:

(1) Two major potential confounding factors, fiber size distribution and fiber concentration, were not analyzed by the authors. If the gas mask plants had higher fiber concentrations and/or a higher proportion of long thin fibers than the mines, a higher cancer risk would be expected in the plants.

(2) Comparison of risk measures such as Standardized Mortality Ratios (SMR's) and Proportionate Mortality Ratios (PMR's) might not be appropriate between the two occupational cohorts because of potential differences in age distribution and length of follow-up period.

Henderson and Enterline (Ex. 84–048) compared mortality of retired workers exposed to different asbestos fiber types. After adjusting for differences in cumulative dust exposures, Henderson and Enterline reported the following respiratory system cancer SMR's for workers in an asbestos factory and workers manufacturing asbestos cement: 247 for workers receiving chrysotile exposures only, 364 for workers receiving amosite exposures only, and 461 for workers receiving combined exposures to chrysotile and crocidolite. Asbestos cement pipe workers had a very high lung cancer SMR of 522. Henderson and Enterline stated: "Men who worked in the production of asbestos cement pipe exhibited a higher risk of respiratory cancer, as did men with some crocidolite asbestos exposure. Because these two groups overlap, we could not be certain that crocidolite asbestos was responsible for the increased risk" (Ex. 84–084).

Concerning Henderson and Enterline's findings, OSHA notes that the asbestos cement pipe exposures may have been dustier than earlier estimates indicated and that some of the workers thought to have been exposed only to chrysotile may have had indirect exposure to crocidolite. Also, workers in the asbestos cement pipe operation probably were exposed mostly to chrysotile (Kang, Ex. 84–139). Another potential confounding factor was that exposure levels were uncertain for each group of workers.

Weill, Hughes, and Waggenspack (Ex. 84–206) studies the mortality of asbestos cement pipe workers in two plants. In the first plant, workers were exposed to both crocidolite (3% of product) and chrysotile. In the second plant, workers were exposed to chrysotile, amosite (1% of product), and crocidolite (infrequently).

Weill et al. reported respiratory system cancer SMR's as follows for workers receiving cumulative asbestos exposures greater than 200 million particles per cubic foot-months (mpcf-months) during the first 20 years of employment:

No crocidolite exposure	SMR = 182
Intermittent exposure to crocidolite	SMR = 357
Steady exposure to crocidolite	SMR = 241

OSHA notes that the apparent differences in lung cancer risk by fiber type observed by Weill et al. are based on relatively small numbers of deaths. Hence, statistical variation may be considered a possible explanation for the observed differences. It is not known if the 75% follow-up rate may have contributed to these apparent differences.

Weill, Ziskind, Waggenspack, and Rossiter (Ex. 84–207) suggested that crocidolite exposed workers had poorer pulmonary function than workers exposed to other forms of asbestos. They reported that crocidolite workers had smaller lung volumes, a higher prevalence of X-ray changes, lower FEV1's, and reduced diffusing capacity.

Note.—FEV1 refers to the volume of air forcibly expired in one second, and diffusing capacity refers to the ability of oxygen to leave the alveoli and enter the pulmonary bloodstream. Reductions in FEV1 and diffusing capacity are indicative of impaired lung function.

A comparison between Canadian chrysotile miners studied by McDonald et al. (Ex. 84–065) and Australian crocidolite miners studied by Hobbs et al. (Ex. 84–132) has been made by Kang (Ex. 84–139). The Australian miners with one or more years of employment and at least 15 years of follow-up had a lung cancer SMR that was approximately twice as high as that of Quebec miners with one or more years of employment and at least 20 years of follow-up. Comparison of these two groups of miners is problematic because of potential differences in age distribution, fiber concentrations, and length of employment (Kang, Ex. 84–139).

Acheson, Gardner, Pippard, and Grime (Ex. 84–015) studied mortality of female workers manufacturing mostly chrysotile-containing gas masks in one plant (Plant 1) during 1939 and female workers manufacturing mostly crocidolite-containing gas masks and some chrysotile-containing gas masks in a second plant in 1939 (Plant 2). Plant 2 had a larger excess of mortality than Plant 1. Ages at initial employment were very similar at the 2 plants. At Plant 1, 7 lung cancer deaths were observed versus 4.8 expected (SMR = 145, statistically non-significant). One death from pleural mesothelioma was observed in a female worker from Plant 1.

At Plant 2, 15 lung cancer deaths were observed versus 6.2 expected (SMR = 241, p less 0.01). Mesothelioma was mentioned on 5 women's death certificates, 3 of which coded mesothelioma as the underlying cause of death. A statistically significant increase in ovarian cancer mortality was also observed at Plant 2 (12 deaths observed versus 4.4 expected, SMR = 275, p less than 0.01). Possibly, the deaths certified as due to ovarian cancer may have actually been misclassified peritoneal mesotheliomas.

Acheson et al. concluded that "the pattern of mortality is consistent with the view that mesothelioma (and possibly ovarian cancer) is particularly associated with exposure to crocidolite" (Ex. 84–015, p. 347).

Acheson et al.'s study has 2 advantages relative to other studies of populations exposed to different asbestos fiber types: both the age distributions and lengths of follow-up were similar for Plant 1 and Plant 2. Neither air concentrations nor durations of employment were reported for the 2 plants, however. Because Plant 2 had been operating longer than Plant 1, Plant 2 workers may have had a greater median length of employment. Manual manufacturing processes were employed at Plant 2, while mechanical processes were employed at Plant 1. Perhaps the manual process led to higher dust exposures for the individual workers at Plant 2. Yet it is also possible that the mechanical process at Plant 1 reduced

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the diameters of the fibers, rendering them more hazardous. OSHA considers that this study provides suggestive evidence for a fiber type differential in mesothelioma risk; however, this evidence is not conclusive because of the lack of information on the air concentrations and durations of employment.

Both plants had excesses of lung cancer mortality, although only the Plant 2 excess was statistically significant. OSHA calculated that the statistical power to detect a 50% increase in lung cancer risk was only 19% at Plant 1. Given the low power to detect increased risk at Plant 1, OSHA believes the statistically non-significant excess of lung cancer at Plant 1 should not be disregarded. Also, Acheson et al. calculated 95% confidence intervals for lung cancer mortality as 135–397 for Plant 2 and 58–298 for Plant 1. In OSHA's view, these overlapping confidence intervals for the 2 plants indicate that the observed differences in lung cancer mortality cannot be considered statistically different. Hence, OSHA concludes that Acheson et al.'s study does not establish the existence of a differential in lung cancer risk by fiber type in these 2 occupational cohorts.

Weiss (Ex. 84–098) studied a cohort of 264 production workers in a paper and millboard factory. This factory had used only chrysotile asbestos. No increased mortality from lung cancer was observed among these workers (4 lung cancer deaths observed versus 4.32 expected). Two men died of abestosis.

OSHA noted limitations of Weiss's study that include lack of asbestos exposure data and very low statistical power. OSHA calculated that this study had only 20.8% power to detect a 50% increase in lung cancer risk. Hence, OSHA does not consider Weiss's study as reliable evidence of low cancer risk among chrysotile workers.

Dement et al. (Ex. 84–037) observed a high lung cancer risk among chrysotile textile workers (35 deaths observed versus 11.10 expected, SMR=315). A high risk of mortality from non-malignant respiratory diseases, including asbestosis, was also observed in this cohort (28 deaths observed versus 9.53 expected, SMR=294). One peritoneal mesothelioma was confirmed by autopsy. Several other death certificates listed "cancer of the abdomen," but the authors were unable to obtain additional data to determine whether these particular cancers were actually mesotheliomas. Expected values for mortality were derived from U.S. mortality rates.

Dement et al. suggested that the higher risk of respiratory cancer mortality in their chrysotile exposed cohort compared to cohorts of chrysotile miners and millers may have been due to different fiber size distributions. Using expected values of lung cancer mortality derived from the South Carolina county in which the chrysotile textile plant was located, instead of U.S. mortality rates, would have reduced the lung cancer SMR's for the cohort because lung cancer mortality rates were 75% higher in the county than in the United States as a whole. Dement et al. considered use of the county rates to be inappropriate because of the probable contribution of a nearby shipyard as well as the study plant to the elevated lung cancer rates in the county. State (South Carolina) mortality rates from lung cancer were similar to those of the United States.

Dement et al. also addressed the possibility of confounding effects from smoking. Questionnaires administered in 1964 and 1971 to workers employed at the plant did not indicate that the study cohort had a higher prevalence of smoking compared to the U.S. population.

OSHA regards Dement et al.'s study as strong evidence of the potential for high lung cancer risk from exposure to chrysotile.

McDonald and Fry (Ex. 84–064) published a preliminary report on their study of 2 asbestos factories (Plants A and B) where chrysotile was used exclusively and 1 factory (Plant C) where chrysotile, amosite, and crocidolite were used. Plant A was the study plant reported on by Dement and colleagues. Plant C had previously been studied by Robinson and colleagues. Respiratory system cancer mortality was in excess at all 3 plants. Expected values for each plant were derived from the age, sex, and cause-specific mortality rates of the states in which the plants were located. SMR's were calculated beginning 20 years after initial employment at the plants. Air concentrations were highest in Plant C, next to highest in Plant A, and lowest in Plant B. Criteria for inclusion in the study cohorts were: (1) One month or more of employment by January 1, 1959, and (2) having any Social Security Administration files. Vital status was determined as of December 31, 1977.

The overall respiratory system cancer SMR was 285 at Plant A, confirming the high lung cancer risk observed at the same plant by Dement and colleagues. Furthermore, McDonald and Fry noted that their inclusion of clerical workers and workers with 1–5 months of exposure in the cohort, unlike Dement et al., probably reduced observed respiratory cancer risk. Among male workers employed 1–4 years at Plants A, B, and C, respiratory system cancer SMR's were 225, 149, and 41 respectively. Among male workers employed 20 or more years at Plants A, B, and C, respiratory system cancer SMR's were 315, 168, and 140 respectively. The lower respiratory cancer risk in men in Plant C was unexpected, given the higher air concentrations in Plant C.

A different pattern of respiratory system cancer risk was apparent for female workers compared to male workers; however, the female mortality rates were not stable because of the small number of deaths. Female workers in Plants A, B, and C had respiratory system cancer SMR's of 234, 348, and 52 respectively.

McDonald and Fry characterized the differences in the distribution of mesothelioma deaths among the 3 plants as "striking." Plant A had 1 death from mesothelioma (=0.1% of all deaths) and Plant B had no deaths from mesothelioma. Plant C had 18 deaths from mesothelioma (1.26% of all deaths). Possibly, an even greater number of deaths from mesothelioma occurrred at Plant C, due to unrecognized cases being coded as abdominal cancers or other/unspecified cancers.

Robinson et al. (Ex. 84–082) had also observed a high mesothelioma risk among workers at Plant C. McDonald and Fry considered the probability of missing true cases of mesothelioma at Plants A and B to be low because of good cancer case ascertainment in the areas where these plants were located. McDonald and Fry stated that exposure-response relationships at the 3 plants might be clarified after the individual subjects were classified by exposure levels.

OSHA considers the following points important for interpreting the findings of McDonald and Fry. Although McDonald and Fry stratified by duration of exposure for their inter-plant comparison of lung cancer mortality, other important variables were not controlled for, including age at initial employment, fiber concentration, and length of follow-up. Plant C, which had the greatest number of deaths from mesothelioma, also had the highest exposure levels. Hence, OSHA considers that the differentials in mesothelioma risk among the 3 plants are suggestive, but do not conclusively demonstrate increased mesothelioma risk from crocidolite relative to other asbestos fiber types. Of interest is the finding of higher lung cancer risks at the chrysotile plants (A and B) relative to plant C. Again, the variables of fiber

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concentration, age distribution, and length of follow-up need to be controlled for when comparing lung cancer risk of different cohorts.

Berry and Newhouse (Ex. 84–021) and Newhouse, Berry, and Skidmore (Ex. 84–163) studies the mortality of a cohort of workers manufacturing friction materials. Exposure was to chrysotile exclusively, except during 1929–1933 and 1939–1944 when a small percentage of the workforce was exposed to crocidolite. Asbestos exposures were low in the plant; only 5% of the male workers accumulated as much as 100 f/cc-years. The only cause of death in excess was mesothelioma. To examine etiologic factors for mesothelioma, a matched case-control study was conducted for 10 mesothelioma cases. For each case, 4 controls were selected from the plant. Controls were matched on year of hire at the factory, sex, age, and duration of employment. Definite crocidolite exposure was discovered in the occupational histories for 8 of the 10 mesothelioma cases and for 3 of the 40 controls. About 90% of the cases and 25% of the controls had been exposed to levels of chrysotile exceeding 5 f/cc. When Berry and Newhouse limited their comparison to those cases and matched controls who had been exposed to over 5 f/cc of chrysotile, a statistically significant greater number of cases had an occupational history of exposure to crocidolite as well.

Matching the controls for the mesothelioma cases on year of hire controlled for the important factor of length of follow-up. OSHA considers that both crocidolite and chrysotile are implicated in the causation of mesothelioma by this study: chrysotile because 90% of the cases received high chrysotile exposures compared to 25% of the controls, and crocidolite because 80% of the cases compared to 7.5% of the controls had been exposed to crocidolite.

The Chronic Hazard Advisory Panel on Asbestos (CHAP) compared lung cancer risk per unit of cumulative exposure (also known as K_L, the lung cancer potency factor) among the cohorts exposed to different fiber types. CHAP reported that studies of chrysotile workers yielded both low and high values of K_L, as well as studies of crocidolite workers and amosite workers. Hence, CHAP considered that evidence for a differential in lung cancer risk was mixed and inconclusive (Ex. 84–256).

Pathology Studies: Wagner, Berry, and Pooley (Ex. 84–202) examined the asbestos contents of tissue samples of lungs of asbestos textile workers who died from mesothelioma and other causes. Wagner et al. did not find either higher chrysotile or higher crocidolite contents in the lungs of the mesothelioma cases compared to the lungs of asbestos workers who died from other causes.

McDonald, McDonald, and Pooley (Ex. 84–175) conducted a study of the mineral fiber contents of lung tissues for 99 autopsied mesothelioma cases and 99 autopsied controls. Controls matched for sex and age were selected for each mesothelioma case. All controls had been diagnosed as having pulmonary metastases from a primary cancer other than lung cancer. Lung tissue specimens were examined for mineral fiber content using an electron miscroscope and X-ray energy-dispersive analyzer.

Chrysotile, amosite, crocidolite, anthophyllite, and tremolite were detected in the lung specimens of the cases and controls. For amosite and crocidolite, about 3 times as many mesothelioma cases as controls had more than 1 million fibers per gram of dried lung tissue. For anthophyllite, twice as many cases as controls had more than 1 million f/gram. For chrysotile and tremolite, little difference in fiber contents was observed between cases and controls.

McDonald, McDonald, and Pooley stated: "The equal quantities of chrysotile fibers found in lung tissue from cases and controls fail to support any association between this mineral type of asbestos and mesothelial tumors. However, the lung chrysotile fibre content at death must be interpreted cautiously, as these fibres probably disappear in the course of time" (Ex. 84–175, p. 420).

There are several points to consider concerning McDonald et al.'s study. First, if the fibers responsible for inducing mesothelioma were also responsible for inducing cancers at sites other than the lung and mesothelium, then a true effect of a fiber on mesothelioma genesis could fail to be detected because of the stipulation that all controls had to have pulmonary metastases. Gastrointestinal cancers sometimes metastasize to the lung, and some studies have observed excesses of gastrointestinal cancer among asbestos workers. Second, since chrysotile constitutes up to 90% of commercially used asbestos and since asbestos exposure is ubiquitous, one would expect controls to have chrysotile fibers in their lungs. The widespread exposure to chrysotile could obscure a true relationship between chrysotile and mesothelioma.

McDonald et al. suggested that chrysotile may be cleared faster from the lungs than other asbestos fibers, so that chrysotile retention in the lungs may not be the best way to estimate chrysotile exposure. Rowlands, Gibbs, and McDonald (Ex. 84–178) suggested a tendency for chrysotile to be removed from the lungs because of their observation that the quantity of tremolite in the lungs of Quebec miners and millers was similar to that of chrysotile, despite the miners' much greater exposure to chrysotile. In general, OSHA believes that the results of autopsy studies of fiber content of lungs cannot serve as direct measures of risk differentials by fiber type.

Mesothelioma Findings: As mentioned in the introduction to this section, the primary basis for the hypothesis that crocidolite is more carcinogenic than other forms of asbestos is the observed variation in mesothelioma mortality among cohorts exposed to different fiber types. A summary of the percentage of deaths from mesothelioma among cohorts exposed to different fiber types follows.

Meurman et al. (Ex. 84–256), who observed excess lung cancer mortality among anthophyllite miners, did not find any deaths from mesothelioma in their cohort. In Seidman et al.'s cohort of amosite insulation production workers, a total of 14 mesotheliomas were identified by death certificate or pathological reports (Ex. 84–087). These 14 deaths from mesothelioma constituted 2.7% of total deaths in this cohort.

Cohorts of chrysotile workers had relatively low percentages of deaths from mesothelioma:

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Table 8

MESOTHELIOMA DEATHS AMONG COHORTS OF CHRYSOTILE WORKERS

Study	% of deaths from mesothelioma
McDonald et al. (Ex. 84-065) - miners	0.3
Nicholson et al. (Ex. 84-072) - miners	0.5
Dement et al. (Ex. 84-037) - textile workers	0.5
Rubino et al. (Ex. 84-086) - miners	0

Hammond et al., however, observed a higher percentage of deaths from mesothelioma (Ex. 84-047). Hammond et al. studied insulation workers exposed predominantly to chrysotile and to some amosite, but not to crocidolite. From death certificates and other clinical material, 175 deaths (7.7%) were attributed to mesothelioma out of a total of 2271 deaths. Peto (Ex. 84-168) studied a factory which used mostly chrysotile and some crocidolite, and found a high mesothelioma risk. Robinson et al. (Ex. 84-082) observed 4% of deaths from mesothelioma in a cohort where the factory used the following percentages of asbestos fiber types: chrysotile—99%, amosite—0.9% and crocidolite—0.07%.

Among crocidolite workers, McDonald and McDonald (Ex. 84-154) observed 16% of deaths from mesothelioma, Jones et al. observed 10% of deaths from mesothelioma (Ex. 84-138), McDonald and Fry (Ex. 84-064) observed 1.26% of deaths from mesothelioma, and Hobbs et al. (Ex. 84-132) observed 9% of deaths from mesothelioma.

The Chronic Hazard Advisory Panel on Asbestos (CHAP) stated that peritoneal mesothelioma "appears to be most common in workers exposed to amosite, less often to crocidolite, and rarely or never to chrysotile" (Ex. 84-256, p. 127). However, CHAP noted the large variation in peritoneal mesothelioma mortality among crocidolite workers, the lack of risk data expressed in terms of unit exposure, and the frequent misdiagnosis of peritoneal mesothelioma. Consequently, CHAP suggested that factors other than fiber type, such as fiber dimension, may be important for induction of peritoneal mesothelioma. For pleural mesothelioma, the differences in mortality among cohorts were not as marked as for peritoneal mesothelioma, according to CHAP (Ex. 84-256, p. 126).

Summary of Epidemiologic Data: In summary, there is evidence that all commercial asbestos fiber types, except for anthophyllite, cause asbestosis, mesothelioma, and lung cancer in humans. Anthophyllite has been shown to cause lung cancer and asbestosis, but no mesotheliomas have been reported among workers exposed to anthophyllite. For lung cancer, OSHA views the evidence for differentials in risk by fiber type as inconclusive and inconsistent. Some studies have found that chrysotile workers have roughly the same or higher risk of lung cancer compared to workers exposed to amphibole fibers, while other studies have found that chrysotile workers had somewhat lower relative risks of lung cancer. As discussed by CHAP with regard to their comparison of lung cancer risk per unit of cumulative exposure, consistent patterns of higher lung cancer risk among workers exposed to crocidolite and amosite did not emerge. With some exceptions, comparisons of lung cancer risk among occupational cohorts exposed to different fibers generally have failed to control for the following variables: Fiber concentrations, age distribution, and length of observation. These variables are sufficiently important determinants of asbestos-induced lung cancer risk such that OSHA believes these particular cross-cohort comparisons provided inconclusive evidence concerning differences in carcinogenic potency of the various fibers. Other important variables that may confound cross-cohort comparisons are fiber size distributions, hygienic measures instituted by the companies, and respirator use.

The evidence for a differential in mesothelioma risk, particularly peritoneal mesothelioma, by fiber type is more consistent than for lung cancer. Nonetheless, this evidence appears not to be conclusive because of failure by most investigators to control for important variables when comparing different occupational cohorts. Mesothelioma risk is affected by the same variables that determine the degree of lung cancer risk. Furthermore, mesothelioma has a longer latency period than asbestos-induced lung cancer and rises exponentially with increasing time since initial exposure, such that length of observation is a variable that must be controlled for in cross-cohort comparisons.

Chrysotile is a lung carcinogen for humans. Even if chrysotile could be definitively shown to induce fewer mesotheliomas than the other forms of asbestos, OSHA believes that epidemiologic studies indicate that chrysotile poses a significant health risk to humans by inducing lung cancer and asbestosis.

OSHA has concluded that fiber type is not an important determinant of lung cancer mortality arising from asbestos exposure. Because lung cancer is the major cause of excess mortality among asbestos workers, OSHA does not deem it appropriate to permit higher levels of exposure to chrysotile than to other asbestos fiber types on the basis of the possibility that chrysotile may induce fewer mesotheliomas than the amphiboles (amosite, crocidolite).

3. *Experimental Data.* OSHA believes that numerous studies of laboratory animals exposed to asbestos have found that the dimensions of fibers rather than types of fibers are major determinants of risk for lung disease and cancer (Harington, Ex. 84-131; Pott, Ex. 84-173; Stanton, Ex. 84-93, 84-195; Wagner et al., Ex. 84-198; Wright and Kuschner, Ex. 84-210). Aspect ratios (i.e. length to width ratios) of fibers have also been suggested as factors influencing carcinogenicity (Bertrand and Pezerat, Ex. 84-114). Because fiber size distribution appears to affect disease-causing potency, the source of the asbestos used for laboratory experiments and the methods used to produce the asbestos "clouds" should be considered when interpreting the results of laboratory experiments.

Chrysotile, amosite, and crocidolite have all induced cancer in animals upon administration by inhalation, injection and implantation (NIOSH, Ex. 84-338; NIOSH/OSHA, Ex. 84-320; Wagner et al., Ex. 84-205; Davis et al., Ex. 84-120). Davis et al. found that UICC chrysotile

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produced more fibrosis in rats via inhalation than did UICC amosite. Factory samples of chrysotile, however, produced a similar degree of fibrosis via inhalation compared to factory samples of amosite.

Wagner et al. conducted inhalation studies of 5 UICC asbestos samples (2 chrysotile, 3 amphibole) in C/D Wistar rats, and found similar degrees of pulmonary fibrosis and lung tumor incidences for all exposed groups (Ex. 84–096). [The Union Internationale Centre Cancer (UICC) was the source for the asbestos fiber samples used in the study.] Animals exposed to the 2 chrysotile samples retained much less dust in their lungs at the end of the exposure periods than the 3 other exposed groups. Concerning this study, the NIOSH/OSHA Asbestos Work Group commented: "In terms of degree of response related to the quantity of dust deposited and retained in the lungs of rats, chrysotile appears to be much more fibrogenic and carcinogenic than the amphiboles" (Ex. 84–320, p. 15). Of interest were the mesotheliomas occurring after only one day of exposure to amosite and crocidolite.

Intrapleural administration of various forms of asbestos produced the following incidence of mesotheliomas: crocidolite—61%, amosite—36%, anthophyllite—34%, Canadian chrysotile—30%, and Rhodesian chrysotile—19% (Wagner et al. Ex. 84–197). In contrast, Stanton and Wrench (Ex. 84–338) did not observe differences in mesothelioma incidence by fiber type with intrapleural implantation.

Chrysotile may act as a co-carcinogen in addition to acting as a primary carcinogen. Kung-Vosamae and Vinkmann (Ex. 84–143) observed a strong interaction between chrysotile administered intratracheally and N-nitrosodiethylamine administered orally with regard to production of lung tumors in hamsters. N-nitrosodiethylamine has been demonstrated to be carcinogenic to many different species by many different routes of administration.

In 1977, Stanton, Layard, Tegeris, Miller, May, and Kent published the results of tests of 17 fibrous glasses of varying types and dimensions (Ex. 84–093). These fibrous glasses were implanted in the pleurae of female Osborne-Mendel rats, and surviving animals were sacrificed after 2 years. Statistical analyses indicated that the fibers less than or equal to 1.5 micrometers (μm) in diameter and longer than 8 μm produced the highest incidence of pleural sarcomas. Fibers less than 8 μm in length appeared to be inactivated by phagocytosis. (Phagocytosis is the engulfing of foreign particles by phagocytes, which are cells that usually digest these particles in order to protect the body from them.) Stanton et al. stated: "Since neoplastic response to a variety of types of durable fibers, particularly asbestos fibers, was similar, our experiments reinforce the idea that the carcinogenicity of fibers depends on dimension and durability rather than physicochemical properties and emphasize that all respirable fibers should be viewed with caution" (Ex. 84–093, p. 587).

OSHA believes that Stanton et al.'s study has important implications for the question of fiber type differentials in risk. The study suggests that any observed differences in risk by fiber type may be due to differences in the fiber size distribution within the workplaces being compared rather than inherent chemical properties of the particular fibers. Stanton et al.'s study suggests that fibrous materials besides asbestos can produce a carcinogenic response in both the pleura and peritoneum, provided that these materials possess carcinogenic dimensions. Among the fibrous materials demonstrated by Stanton et al. to produce malignant tumors following implantation are: All forms of asbestos, including amosite, anthophyllite, chrysotile, crocidolite, and tremolite; borosilicate; glass; aluminum silicate glass; mineral wool; aluminum oxide; potassium titanate; silicon carbide; sodium aluminum carbonate; wollastonite; and attapulgite. It should be noted that Stanton et al.'s study did not rule out carcinogenicity of fibers outside of the dimensions shown to be carcinogenic.

Stanton et al. (1981) continued their implantation experiments, greatly expanding the number of durable minerals, including asbestos, tested via implantation into the pleurae of female Osborne-Mendel rats (Ex. 84–195). Stanton et al. observed that the most carcinogenic fibers were those 0.25 μm or less in diameter and greater than 8 μm in length; however, high correlations with carcinogenicity were also observed for fibers 1.5 μm or less in diameter and longer than 4 μm.

Wright and Kuschner (Ex. 84–210) studied the production of fibrosis by intratracheal injection of asbestos fibers in guinea pigs. They observed fibrosis only from asbestos fibers longer than 10 μm. Gibbs and Hwang (Ex. 84–128) pointed out that industrial processes using asbestos may reduce the percentage of shorter fibers in the air.

NIOSH (Platek, Groth, Finnell, Stoll, and Ulrich) conducted a study of the chronic effects of inhalation of short asbestos fibers, with short fibers defined as those less than 5 micrometers (μm) in length (Ex. 84–230). Both rats and monkeys were exposed to a chrysotile asbestos aerosol for 18 months. The surviving rats were observed for 6 months following the cessation of exposure. Neither pulmonary fibrosis nor tumors were increased in the exposed rats compared to the control rats. Monkeys are being maintained for an indefinite period following exposure to determine the chronic effects of exposure.

The NIOSH investigators encountered some difficulties in trying to generate short fibers. The ball milling method was used to generate fibers, resulting in asbestos balls that could not meet the desired 3:1 aspect ratio. Some problems also occurred with regard to the methods of counting asbestos fibers and determining aerodynamic diameters (Ex. 84–230). Consequently, OSHA considers that Platek et al.'s study provides suggestive but not conclusive evidence that short asbestos fibers do not induce pulmonary fibrosis or tumors.

Some chemists have posited that asbestos fibers have biochemically active sites on their surfaces that can be modified so as to reduce the hazardous potential of asbestos fibers (Ex. 84–333). The electrical charge of chemical groups on the surface of asbestos fibers has been hypothesized to influence toxicity of fibers. For example, one chemical process removes the magnesium hydroxide groups on the surface of chrysotile and replaces them with silanol groups, producing a form of asbestos known as silanized asbestos. In vitro tests (tests conducted on cells in test-tube simulations of living systems) have been conducted for normal asbestos fibers and chemically-treated asbestos fibers. Decreased toxicity in chemically treated asbestos fibers compared to normal asbestos fibers has been reported (Ex. 84–333). OSHA considers that the hypothesis that biochemically active sites on the surface of asbestos fibers determine the degree of carcinogenicity of the fibers is not well supported at this time. This is because many in vivo studies, especially those conducted by Stanton et al., have found that fiber dimensions rather than chemical properties appeared to be the primary determinant of fiber carcinogenicity. Stanton et al. found that a variety of non-asbestos fibers could induce cancer, if they were milled to specific dimensions. The in vitro studies of chemically treated asbestos are not as determinative of health risk as the in vivo studies of fiber dimension and hence do not refute the findings of Stanton et al. and other investigators

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concerning the overriding importance of fiber dimension. Therefore, OSHA will continue to include chemically-treated asbestos longer than 5 micrometers with a 3:1 or greater aspect ratio in the scope of the asbestos standard, on the basis that chemically-treated asbestos poses a health risk similar to that of untreated asbestos.

In summary, OSHA regards experimental studies of animals as suggesting that all asbestos fiber types possess similar carcinogenic potency. Animal studies also suggest that fiber dimensions rather than chemical properties or fiber type may be the strongest determinant of carcinogenicity, in OSHA's view. Extrapolation of the results of experimental studies in animals to the human experience necessitates an assumption that the fiber dimensions used for experimental studies resemble fiber dimensions in workplaces. This assumption might not be valid in all instances.

4. *Other Factors.* The various types of asbestos fibers may differ in the percentage of fibers available for inhalation and the length of time that dust clouds remain airborne (Rowe, Ex. 84–085). Crocidolite might stay airborne for a longer period of time and have a greater number of respirable fibers compared to amosite, which in turn may exceed anthophyllite in regard to these two characteristics (Muir, Ex. 84–350). The preparation method for chrysotile will determine these two characteristics for a laboratory sample of chrysotile or for a particular workplace with chrysotile exposure (Rowe, Ex. 84–085). Because thicker fibers might drop to the ground faster and crocidolite is generally finer than the other fibers, crocidolite use might lead to dustier work environments and hence a greater health risk, even if crocidolite is not more dangerous on a fiber-for-fiber basis (Muir, Ex. 84–350). However, most respirable fibers, which are the fibers likely to cause disease, will tend to remain airborne for long periods of time.

Another point to consider is whether, for different fiber types, the ratio between fibers visible under a microscope and ultra-fine submicroscopic fibers varies. This would confound comparisons between cohorts apparently exposed to the same levels of different visible fibers under the light microscope, if indeed fibers not counted by the present optical microscopy method pose a hazard to health (Muir, Ex. 84–350).

OSHA believes that thicker fibers are found in mining and milling and that subsequent manufacturing processes tend to break fibers apart and reduce their diameter. Based on experimental findings implicating long thin fibers as being the most carcinogenic fibers, these subsequent processes may have a greater carcinogenic hazard compared to the initial mining and milling operations (Nicholson, 1981, Ex. 84–071).

OSHA examined the question of differentials in carcinogenic risk among manufacturing processes which fall under OSHA's jurisdiction. As is the case for studies of fiber type, cross-cohort comparisons of processes should control for the following variables: fiber concentrations, duration of follow-up, duration of exposure, and age distribution. To account for the variable of fiber concentration, OSHA compared the potency factors for lung cancer (K_L) of different manufacturing studies because potency factors are based on risk per unit dose. As discussed in Section V, *Quantitative Risk Analysis*, the potency factors for lung cancer were not consistently higher for any particular manufacturing process. Because potency factors for mesothelioma (K_m) could be calculated for only 4 studies, OSHA could not compare K_M's for different manufacturing processes. In summary, there does not appear to be a consistent pattern of differential lung cancer risk by manufacturing process. Therefore, OSHA deems it appropriate to continue to apply a single PEL to all segments of industry covered by the Agency.

Relatively thick respirable fibers tend to lodge in the lung while thinner fibers can travel to the periphery of the lung, and penetrate and then lodge in the pleura. These fine fibers lodged in the pleura might be the inducers of mesothelioma (Nicholson, 1981, Ex. 84–071). Bignon et al. (Ex. 84–105) studied the lungs and pleurae of shipyard workers, and found larger fibers, especially amphiboles, in the lungs. In the pleurae, fine and small fibers, usually chrysotile, were found.

In summary, OSHA recognizes that the fiber types may differ with regard to their tendencies to break into fine fibers. Crocidolite appears to divide into finer fibers more readily than other asbestos fiber types, which may render it more hazardous. Manufacturing processes could increase the carcinogenicity of asbestos by generating fibers that are thinner in diameter relative to mining and milling processes. While these factors are interesting and contribute to the formation of hypotheses regarding the mechanisms of asbestos carcinogenesis, OSHA believes that these factors do not provide a definitive answer to the question of possible differentials in risk by fiber type. OSHA views the data from epidemiologic and experimental studies, which have observed excess cancer risk from exposure to all asbestos fiber types, as being the most important with regard to the question of differential health risk by fiber types.

5. *Tremolite and Anthophyllite.* Some but not all commercial talc deposits contain serpentine asbestos and fibrous amphibole asbestos, including chrysotile, tremolite, and anthophyllite (Dement and Zumwalde, Ex. 84–039). Kleinfeld et al. (1967, Ex 84–181) studied the proportionate mortality of 220 talc miners and millers in New York State who had been exposed to asbestos contained in talc. All men were employed during 1940 and had 15 or more years of exposure during 1940–1965. Of 91 deaths in the study cohort, 10 (11%) were from respiratory system cancer and 28 (31%) were from pneumoconiosis or complications of pneumoconiosis. Kleinfeld et al. calculated that only 2.9 (3.2%) deaths from respiratory system cancer would have been expected, resulting in a greater than 3-fold risk of respiratory cancer. A follow-up study by Kleinfeld et al. (1974, Ex. 84–141) included 260 workers exposed to asbestos in talc. Of the 108 deaths observed, 13 (12%) were from respiratory cancer compared to 4 (3.7%) expected. The high proportion of deaths from pneumoconiosis may have reduced the number of deaths from lung cancer.

Kiviluoto et al. (Ex. 84–181) and Nurminen et al. (Ex. 84–181) observed pneumoconiosis and excess lung cancer mortality among Finnish worker exposed to talcs containing fibrous anthophyllite and fibrous tremolite. No deaths from mesothelioma were reported by these investigators.

Yazicioglu, Ilcayto, Balcj, Sayli, and Yorulmaz (Ex. 84–211) reported a high prevalance of pleural calcification and thickening and a high mortality rate from mesothelioma among residents of Cermik, a town in Turkey in which there are many deposits of asbestiform minerals which are used as construction materials. The construction materials have been shown to contain fibrous tremolite, antigorite, lizardite, chlorite, and talc. Yazicioglu et al. also reported an excess of lung cancer in this population.

Brown, Dement, and Wagoner of NIOSH (Ex. 84–025) conducted a historical prospective study of talc miners and millers of one company in New York State reported by the company to be mining talcs not containing asbestos minerals. NIOSH (Dement and Zumwalde), however, had reported asbestos exposure in the talc

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mine and mill. NIOSH conducted an industrial hygiene survey of the study talc mine and a neighboring talc mine known to contain asbestos fibers. Silica exposures were found to be very low, well below NIOSH's recommended PEL for silica. Radon daughter measurements made by the Mine Enforcement Safety Administration (MESA) showed only nil to trace levels. Dement and Zumwalde found that exposure characteristics between the study mine and neighboring mine were substantially similar:

"In fact, the airborne dust samples from the mine and mill studied by NIOSH and maintained by the company to be asbestos free were found to contain a *higher* proportion of positively identified asbestiform amphiboles largely due to a higher tremolite content. All other fiber characteristics, such a[s] median length, diameter, aspect ratio, and proportion less than 5 θm in length, were not statistically different at the 0.5 level" (Ex. 84–181, p. 10).

The NIOSH study cohort (Brown et al.) consisted of all white males employed sometime during 1947–1959. Vital status of the 398 cohort members was determined as of June 30, 1975. Comparision was made to age, calendar period, and cause-specific mortality rates of U.S. white males. Significant increases in lung cancer mortality (9 observed deaths versus 3.3 expected) and non-malignant respiratory disease mortality (8 observed versus 2.9 expected) were observed. One death from mesothelioma occurred. Since the individual who died from mesothelioma had previously worked in the construction industry, his death could not be definitely ascribed to his exposure to tremolite or anthophyllite. Of the 10 individuals who died from respiratory system cancer, 3 had previously worked for other New York State talc companies.

NIOSH investigators also addressed the potential confounding effects of cigarette smoking. In their opinion, a cohort of heavy smokers would have no more than a 49% increase in lung cancer risk in relation to all U.S. white males. Because they observed a greater increase in lung cancer risk, almost a 3-fold risk, they judged that cigarette smoking was unlikely to account for the observed excess lung cancer risk among these talc miners and millers exposed to asbestos. The cross-sectional morbidity survey conducted by NIOSH in 1975 found a 48% prevalence of smoking, a prevalence similar to that of U.S. males. Brown, Dement, and Wagoner stated that "exposures to asbestiform tremolite and anthophyllite stand out as the prime suspected etiologic factors" associated with the observed excess risks of lung cancer and respiratory disease. They concluded that "exposures to talcs from the Gouverneur mining area are associated with an increased risk of bronchogenic cancer and non-malignant diseases of the respiratory system" (Ex. 84–181).

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 time-weighted average for 6 different job titles in the mine. In the mill, average air concentrations for such fibers ranged from 1.5 f/cc to 8.4 f/cc as an 8- hour time-weighted average for 18 different job titles. (Ex. 84–181, pp. 7–10).

In addition to the excess mortality from lung cancer and non-malignant respiratory disease observed by Kleinfeld et al. and Brown et al., numerous studies of talc miners and millers exposed to asbestos contained in talcs have established that these workers have a high prevalence of pleural thickening, pleural calcification, decrements in pulmonary function, and fibrosing lung disease (Dreessen, 1933; Dreessen and Dalla Valle, 1935; Siegel et al., 1942; Schepers and Durkan, 1955; Messite et al., 1959; Kleinfeld et al., 1963, 1964, 1964, 1964, 1965, 1965, 1973; Meurman et al., 1974; Kiviluoto et al., 1964; Ahlman et al., 1972; Porro et al., 1942; Ex. 84–181). Many of these studies were conducted in the same geographic area as the studies by Brown et al. and Kleinfeld et al.

A cross-sectional morbidity study of the same company whose mortality experience was studied by Brown and colleagues was performed by NIOSH investigators (Gamble, Fellner, and DeMeo, Ex. 84–181). As discussed above, NIOSH considered that this company's workforce was exposed to asbestos contained within talc. NIOSH observed markers of asbestos exposure in the lungs of these workers in addition to respiratory symptoms and lung function decrements. Of 156 male miners and millers, 121 participated in the survey. Respiratory questionnaires, chest X-rays, and spirometric testing were administered to participating workers. Comparison of respiratory morbidity was made to 9347 coal miners, 1097 potash miners, chrysotile asbestos workers and synthetic wool textile workers. OSHA considers that one of the major strengths of Gamble et al.'s cross-sectional morbidity study was the choice of comparison populations. Because talc, coal, and potash miners are likely to be similar in many non-occupational factors that may affect respiratory morbidity, the likelihood of observed differences in respiratory morbidity being due to specific workplace exposures of the talc workers rather than other risk factors for lung disease is greatly increased. Comparisons with the coal and potash miners were stratified by age, height, smoking status, and duration of employment in mining.

Compared to coal miners and potash miners, the talc workers with no previous occupational exposure within other talc mines and mills had statistically significant increases in pleural thickening and a higher prevalence of pleural calcification. When all talc workers were combined without regard to previous occupational exposure to talc, increased prevalences of cough, phlegm production, dyspnea, pleural thickening, pleural calcification, and irregular opacities on the X-rays were observed in talc workers compared to one or both mining control groups.

In addition, talc workers had significantly decreased pulmonary function (FEV1 and FVC). Decreased lung function was associated with increased cumulative exposures and lengths of exposure.

Talc workers had a similar prevalence of respiratory symptoms when compared to chrysotile asbestos workers and a much higher prevalence of symptoms when compared to synthetic wool textile workers. In contrast, pleural thickening was four times as common in talc workers compared to the chrysotile workers. Smoking was not found to be associated with the observed radiographic changes in talc workers.

Regarding their studies of morbidity and mortality of workers exposed to talc containing asbestos, NIOSH concluded:

"A thorough review of the available literature demonstrated that findings of the present studies are in agreement with those of other studies of occupational groups exposed to the same or similar minerals or mineral mixtures. This is especially true for occupational exposures to anthophyllite asbestos. These findings make it imperative that workers from the mine and mill studied, herein, be routinely observed using medical surveillance criteria established in the OSHA and MSHA asbestos standard. Furthermore, all provisions of these standards should be followed during the production and subsequent use of these talcs" (Ex. 84–181, p. 33).

Stille and Tabershaw (Ex. 84–196) of Tabershaw Occupational Medicine Associates studied all male workers employed sometime during 1948–1977 at the talc mine and mill studied by NIOSH. A total of 708 men were eligible for the study, and vital status as of December 12, 1978 was ascertained for 672 of the men. Of the 708 men, 53

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workers were excluded because of lack of information on specific variables, leaving a cohort of 655 for analysis. Comparison was made to age, calendar period, and cause-specific mortality rates of U.S. white males.

For the overall cohort, excesses in mortality that were not statistically significant were observed for lung cancer and non-malignant respiratory disease. Stille and Tabershaw judged that the non-significant excess of lung cancer was "consistent with a smoking effect" rather than an effect from occupational exposure (Ex. 84–196, p. 481).

Stille and Tabershaw also separately analyzed the mortality of the talc workers with a history of any work experience before employment at the study mine and mill. This prior work experience included all previous jobs, not only jobs at other talc mines and mills. For this subcohort of 540 white males, mortality from the following causes of death was significantly elevated: all cancers (SMR=192), liver cancer (SMR=1013), respiratory system cancer (SMR=228), lymphopoietic cancer (SMR=374), and non-malignant respiratory diseases (SMR=307). Stille and Tabershaw stated: "Since the cancers and lung diseases typically have long latencies, the possibility exists that exposures prior to work at the TMX study mine and mill were responsible for at least some of these diseases" (Ex. 84–196, p. 482).

When the mortality of the subcohort with no work experience prior to employment at the study mine and mill was analyzed, no causes of death were in excess. In fact, the SMR for all causes was significantly decreased (SMR=50). As would be expected, this cohort was generally younger than the cohort which had previous work experience. Stille and Tabershaw characterized these findings as follows: (1) "Workers with 'exclusive' TMX [study mine and mill] employment seem to be at no considerable risk of having lung cancer develop" and (2) "exposures at TMX seem to be noncarcinogenic" (Ex. 84–196, p. 483).

Brown, Beaumont, and Dement of NIOSH commented on Stille and Tabershaw's study (Ex. 84–218, p. 178; Ex. 84–231), listing several problems in the analysis by Stille and Tabershaw that could account for the different conclusions of the two sets of investigators. First, Stille and Tabershaw failed to analyze mortality by length of followup latency interval). Brown et al. thought such an analysis was particularly important given that recently hired workers were allowed to enter the cohort as late as one year before the study cut-off date for vital status determination.

Second, Brown et al. commented that Stille and Tabershaw's division of the study group into subcohorts with and without work experience prior to their employment at the study facility resulted in "selection biases inherent in the definition of the subcohorts" (Ex. 84–218, p. 179). The selection biases which tended to lessen the statistical power of the subcohort without previous work experience included the short length of follow-up, short durations of exposure, and small size of the subcohort. Brown et al. stated: "Any mortality analysis based on such a small cohort with generally short latency is not likely to be very informative" (Ex. 84–218, p. 179).

Third, Brown et al. criticized Stille and Tabershaw's analyses concerning the duration of employment of the lung cancer cases. These analyses examined the observed lung cancer deaths without calculating the expected number of deaths for each latency category and duration of employment category. In addition, Brown et al. pointed out that Stille and Tabershaw's conclusions had not adequately acknowledged that many study facility workers had been employed by neighboring talc companies with exposures similar to those of the study facility.

Brown et al. concluded that Stille and Tabershaw's report did not adequately address the question of increased lung cancer risk from working at the study facility. In addition to the points discussed above, NIOSH also commented that death certificates should have been coded according to the rules of the ICDA in effect at the time of death and then converted to either the 7th or 8th revision of the ICDA for analysis, rather than coding the deaths directly to the 8th revision. NIOSH also had questions concerning the 53 workers eliminated from the cohort and how workers lost to follow-up were treated in the analysis.

Tabershaw and Thompson responded to the NIOSH critique with the following comments (Ex. 84–218). First, they cited mineral scientists and laboratories who disagreed with NIOSH's method of identifying asbestos in silicate mineral mixtures and who did not find significant asbestos concentrations in the talc processed at the mine and mill. Second, they stated that talc ore at the study facility was not similar to that of other talc mines of upstate New York. Third, Tabershaw and Thompson reiterated that many of the lung cancer deaths occurred in short-term workers employed for less than one year. In their opinion, this lessened the likelihood of the lung cancers being attributable to occupational exposure at the study plant. Tabershaw and Thompson also pointed out that only one talc miller developed lung cancer, despite the historically higher exposures of talc millers. In conclusion, Tabershaw and Thompson stated that application of the asbestos standard to the Vanderbilt workforce was unwarranted based on current evidence.

OSHA calculated a statistical power of 20% to detect a 50% increase in risk of lung cancer in Stille and Tabershaw's subcohort of workers with no previous work experience. (See Ex. 84–336 for a description of how OSHA calculated statistical power.) OSHA considers that this very low statistical power supports NIOSH's criticism of the sensitivity of Stille and Tabershaw's study design. The statistical power would have been even lower if it had been calculated for the group of workers with at least 20 years of latency.

As discussed earlier, cross-cohort comparison may be problematic if the age distributions, durations of exposure, and lengths of follow-up differ among the cohorts being compared. OSHA believes that this appears to be the case with TOMA's comparison of the subcohorts with and without previous work experience.

Several points are notable with regard to the question of the short-term workers who developed lung cancer. First, short durations of exposure may result in high cumulative doses depending upon the intensity of the exposure. Second, the phenomenon of short-term asbestos exposure and subsequent disease has been observed in other epidemiologic studies. Third, if the majority of person-years-at-risk were contributed by short-term workers, then finding most of the lung cancer deaths among these workers would not be unusual. Also, Tabershaw and Thompson did not discuss the expected number of lung cancer deaths when they mentioned the single lung cancer death among the study facility talc millers.

In 1983, Consultants in Epidemiology and Occupational Health (CEOH) prepared another analysis of the mortality of Stille and Tabershaw's study cohort (Ex. 84–257). NIOSH reviewed CEOH's analysis and stated:

"Although the findings of these analyses appear to support their [CEOH's] hypothesis that the talc is non-carcinogenic, the resulting statistical analyses are based on *assumptions, small numbers,* and *short latency.* Therefore, the CEOH conclusions are based on analyses that are inherently deficient in being able to

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detect a true risk in an exposed population" (Ex. 84–375, p. 3).

Smith, Hubert, Sobel, and Marquet (Ex. 84–194) studied health effects of talc containing asbestos in laboratory animals. Smith et al. administered intrapleural injections of 4 different tremolitic substances into hamsters, including fibrous tremolitic talc from New York state, tremolite prepared from tremolitic talc ore of the facility studied by NIOSH, tremolite prepared from western U.S. tremolitic talc ore, and asbestiform tremolite. Periods of observation ranged from 350 days to 600 days after injection. Tumors and pleural fibrosis were observed only in animals injected with asbestiform tremolite and tremolite prepared from western U.S. tremolitic talc ore. Smith et al. found that the samples of asbestiform tremolite had greater proportions of long thin fibers than the sample of tremolite from the facility studied by NIOSH.

The sample of fibrous tremolitic talc from New York state also contained long thin fibers and Smith et al. suggested that the negative results from this particular sample were due to its much lower tremolite content relative to the carcinogenic samples. Smith et al. suggested that the sample of tremolite from the facility studied by NIOSH may have failed to induce tumors because of the shorter length of its fibers, despite its high tremolite content. Commenting on their results and on the fact that the carcinogenic samples contained material other than tremolite. Smith et al. stated:

> "* * * we cannot be sure that their activity is due wholly, or even in part to tremolite * * * consideration must be given, not merely to the amount of tremolite, but also to other factors, such as the morphologic characteristics of the mineral" (Ex. 84–194, p. 338).

OSHA considers that interpretation of Smith et al.'s findings is limited by the small number of animals studied, short survival times, and short periods of observation.

In summary, OSHA concludes that NIOSH studies have shown that exposure to asbestos when it is present in talc appears to have resulted in excess mortality from lung cancer and non-malignant respiratory disease and excess risk of pleural thickening and lung function decrements. OSHA is cognizant of the fact that talc mining and milling in the absence of asbestos exposure can result in pneumoconiosis. Furthermore, NIOSH's findings on mortality are consistent with those of Kleinfeld et al., Kiviluoto et al., Nurminen et al., and Yazicioglu et al. NIOSH's morbidity findings are consistent with those of numerous other studies. OSHA believes that the epidemiologic study of the talc company conducted by Stille and Tabershaw of Tabershaw Occupational Medicine Associates has serious limitations such that it does not refute the hypothesis that talc containing asbestos poses a lung cancer hazard to exposed workers. Smith et al.'s experimental study observed positive findings of tumorigenicity for asbestiform talc and non-positive findings for the talc from the facility studied by NIOSH; however, OSHA thinks that limitations of Smith et al.'s study render its findings inconclusive for talc from the facility studied by NIOSH. In any event, the positive human findings outweigh the non-positive animal findings. Talcs containing asbestos minerals, therefore, 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. OSHA notes that the broader issue as to which mineral fibers are to be included in the definition of asbestos will be part of the rulemaking for the permanent standard. This ETS action does not change the definition of asbestos as found in 1910.1001.

6. *Summary.* The evidence presently before the Agency indicates that all asbestos fiber types, except for anthophyllite, cause lung cancer, mesothelioma, and asbestosis. Anthophyllite causes lung cancer and asbestosis. Mesothelioma has not been reported in anthophyllite workers.

For lung cancer, OSHA notes that inconsistent and inconclusive findings with regard to differential risk by fiber type have been observed by different investigators. Some studies have observed the same or higher lung cancer risk in chrysotile workers compared to workers exposed to amphibole fibers; other studies observe lower risk of lung cancer in chrysotile workers. For example, high respiratory cancer risk was observed by Dement and colleagues among chrysotile textile workers. As discussed by CHAP, consistent patterns of higher lung cancer risk by fiber type did not emerge. With some exceptions, investigators generally failed to control for variables affecting lung cancer risk when comparing the mortality of different occupational cohorts. Among the important determinants of lung cancer risk, which were not consistently controlled for, are fiber concentration, length of observation, and age distribution.

OSHA views evidence for differential mesothelioma risk by fiber type as suggestive yet not conclusive. Occupational cohorts exposed to crocidolite and amosite appear to have had a higher proportion of deaths from mesothelioma, particularly peritoneal mesothelioma, compared to occupational cohorts exposed to chrysotile. Nonetheless, OSHA is unable to characterize this evidence as conclusive because most investigators failed to control for fiber concentration, length of observation, and age distribution when making cross-cohort comparisons of mortality from mesothelioma. Controlling for length of observation is particularly important because mesothelioma usually has a latency period of 30 years or more and the risk rises exponentially with increasing time since initial exposure.

Lung cancer is the major cause of asbestos-induced occupational mortality. Mesothelioma and asbestosis also are significant causes of excess mortality among asbestos workers. OSHA has concluded that there is a grave risk for lung cancer and asbestosis among workers exposed to all asbestos fiber types and that workers need to be protected from these risks by this ETS, which sets the lowest PEL that is feasible at this time. As a practical regulatory endpoint, therefore, it is inconsequential whether the mesothelioma risk is greater for certain fiber types than for other types.

Experimental studies of animals have found that chrysotile is as least as potent a carcinogen as other asbestos fiber types. One study found that chrysotile was more carcinogenic than other asbestos fibers when administered by inhalation. Many experimental studies have reported that fiber dimension is a very important determinant of carcinogenicity in laboratory animals. Fibers longer than micrometers (um) and with diameters 1.5 um or less have been implicated as the most carcinogenic fibers. A variety of different natural and synthetic substances having fibers longer than 4 um with widths of 1.5 um or less have induced cancers via implantation in laboratory animals, suggesting that the chemical properties of asbestos (such fiber type) and non-asbestos fibers may not affect carcinogenicity but that fib dimensions may determine carcinogenicity. Thus, in OSHA's vie animal studies do not support the hypothesis that there are differences carcinogenic potency among the vari fiber types.

Fiber types may differ with regard the fineness of fibers used in industr processes. Finer fibers may be more carcinogenic, according to animal studies, and crocidolite appears to b divided more readily into fine fibers

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than other asbestos fiber types. Also, manufacturing processes may break fibers apart to generate fibers thinner in diameter than those found in the mining and milling processes.

NIOSH scientists (Brown, Dement, and Wagoner) observed excessive mortality from lung cancer and non-malignant respiratory disease among workers in one plant exposed to talc containing asbestos. A morbidity survey of the same plant found increased risk of pleural thickening and lung function decrements. NIOSH concluded that increased risk of lung cancer and non-malignant respiratory disease was apparent in workers exposed to talc containing asbestos. Kleinfeld and colleagues also observed excess lung cancer and respiratory disease mortality among workers exposed to talc containing asbestos, and numerous morbidity surveys have documented respiratory disease excesses among talc miners and millers exposed to asbestos. Stille and Tabershaw conducted a study which they interpreted as contradicting NIOSH's findings regarding excessive mortality among talc workers exposed to asbestos. Stille and Tabershaw's study had serious methodologic limitations such that NIOSH's conclusions still appear to be valid. While not all talc products contain asbestos, OSHA finds that when asbestos minerals are contained in talc products, there appears to be a health risk to exposed workers.

Practical administrative considerations also are pertinent to the question of making regulatory distinctions by fiber type. Several types of asbestos may be present in the same workplace, as is the case for insulation, creating considerable measurement difficulties. Determining asbestos type(s) within each workplace might complicate compliance and exposure monitoring. However, OSHA is aware that the United Kingdom has been able to overcome such complications.

In summary, OSHA concludes that on the basis of the epidemiologic and experimental studies reviewed above, it is not appropriate at this time to make distinctions by fiber type for regulatory purposes. Accordingly, quantitative analyses of risk should base estimates of excess risk on asbestos fibers as a whole rather than on separate fiber types. Provisons of OSHA's standard for asbestos, including the permissible exposure limit, should also be the same for all commerically used asbestos fiber types.

F. Conclusions

Asbestos poses a grave danger to the health of exposed workers, having caused excess mortality and disability in epidemic proportions among some groups of exposed workers. Asbestos causes non-malignant respiratory disease, which can result in complete disability and death. Asbestos also causes malignant mesothelioma, which is usually rapidly fatal, and lung cancer, which is usually fatal. The causal relationship between asbestos exposure and disease has been established by a multitude of epidemiologic studies conducted throughout the world, including the United States, United Kingdom, Canada, Australia, Italy, and Finland. Furthermore, asbestos has been shown to cause excess disease in many different occupational environments, starting with mining and milling of asbestos and continuing to cause disease in various manfacturing processes such as asbestos textile production and asbestos cement production. Asbestos also has been shown to be extremely hazardous when used as a product by insulation workers and shipbuilding workers. Numerous scientific organizations and agencies have reviewed the health data for asbestos and have concluded that asbestos exposure causes lung cancer mesothelioma, and asbestosis.

To determine the potential of a hazardous agent to cause disease at low exposures, OSHA has examined the results of studies of workers receiving low exposures, where such studies are available and have sufficient sensitivity to detect excess risk of disease. OSHA has also attempted to predict risk at low exposures from risk observed at high exposures by using dose-extrapolation models (see section V). Both of these methods of evaluating risk at low exposures are valid.

Studies are not available of workers exposed solely to asbestos levels of 2 f/cc which have sufficient cohort size and long enough follow-up periods to permit observation of increases in cancer and lung disease mortality. Studies are available of workers with low cumulative exposures to asbestos, however. Workers exposed 50 years to the OSHA PEL of 2 f/cc would have a cumulative exposure of 100 f/cc-years. Hence, studies of workers with cumulative exposures close to or below 100 f/cc-years provide some evidence of risk from working lifetime exposures to OSHA's PEL. Studies finding adverse pulmonary effects or excessive mortality from lung disease from exposure close to or below 100 f/cc-years were conducted by Berry et al., McDonald et al., Dement et al., Berry and Newhouse, and Finkelstein. Dement et al., Finkelstein, and Henderson and Enterline also observed excess mortality from lung cancer among workers receiving relatively low cumulative exposures. Weill et al. and McDonald et al. did not observe increased lung cancer mortality among workers receiving relatively low cumulative exposures. Despite some inconsistencies in findings regarding risk from low cumulative exposures, OSHA considers that the many well-conducted studies which observed substantially increased risk of morbidity and mortality among workers receiving low cumulative exposures are evidence of significant hazard at exposures allowed by the existing PEL. Section V., *Quantitative Risk Analysis,* will discuss *predicted* risk from exposure to OSHA's PEL.

A number of investigators have suggested that different fiber types of asbestos have differences in their ability to induce mesothelioma. Specifically, crocidolite has been hypothesized to be the most hazardous type of asbestos because of suggestive evidence that indicates that the mesothelioma risk may be higher for crocidolite exposure than for other asbestos types. All commercially used fiber types have induced lung cancer, mesothelioma, and asbestosis in exposed workers, except for anthophyllite, which has induced lung cancer and asbestosis. OSHA regards evidence for differentials by fiber type in lung cancer risk and asbestosis risk as being inconclusive and inconsistent. However, evidence for fiber type differentials in risk with regard to induction of mesothelioma, particularly peritoneal mesothelioma, appears to be more consistent, based on epidemiologic studies of occupational cohorts exposed to different fiber types. Because variables that affect the sensitivity of studies for detection of risk of mesothelioma have not been controlled for by most investigators comparing different occupational cohorts, OSHA views the evidence for fiber type differentials in mesothelioma risk as inconclusive. Among the variables that influence the appearance of mesothelioma are length of observation, duration of exposure, fiber concentration, and age distribution.

It is possible that mesothelioma risk may be higher from exposure to finer fibers, and that operations using crocidolite may tend to break fibers apart to make them more carcinogenic. Studies in laboratory animals have not consistently observed differential risk by fiber type with regard to induction of pulmonary fibrosis and cancers; in fact, some studies in animals have found chrysotile to be the most carcinogenic among the various asbestos fibers tested.

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Lung cancer is the major cause of asbestos-associated occupational mortality. The very high risk of lung cancer from exposure to all asbestos fiber types supports choosing the same PEL for all fiber types.

Several epidemiologic studies, including one performed by NIOSH, have found that workers exposed to talc containing asbestos have excess mortality from lung cancer and non-malignant respiratory disease. Many other studies have observed excess morbidity from respiratory disease, chest X-ray abnormalities, and lung function decrements among such workers. One non-positive study conducted by Stille and Tabershaw of talc workers has methodologic limitations such that it does not refute studies finding excess mortality among workers exposed to talc containing asbestos.

A number of studies in laboratory animals have implicated long thin asbestos fibers in the etiology of cancer and lung fibrosis. Stanton et al. found that fibers greater than 4 μm in length and 1.5 μm or less in diameter were the most carcinogenic; however, OSHA believes that Stanton et al.'s studies do not exclude the possibility of carcinogenicity of fibers shorter or thicker than these dimensions.

At least 12 studies have observed excess mortality from gastrointestinal cancer (stomach, esophagus, colon and rectum) among asbestos exposed workers, 7 of which found statistically significant excesses. Other cancer sites have also been observed to be increased by different investigators. OSHA believes that the positive human studies finding gastrointestinal cancer excesses outweigh the non-positive and equivocal animal studies of ingested asbestos, as well as the non-positive human studies. Therefore, OSHA concludes that excess mortality from gastrointestinal cancer should be included in quantitative risk analyses for asbestos.

Cigarette smoke and asbestos have a multiplicative (synergistic) effect with regard to production of lung cancer. Asbestosis also might be increased in cigarette smoking asbestos workers relative to non-smoking asbestos workers. OSHA is not aware of any evidence for an effect of cigarette smoking on induction of mesotheliomas or gastrointestinal malignancies among asbestos workers. Hammond et al. found that both smoking and non-smoking asbestos insulation workers had relative risks for lung cancer of about 5.

V. Quantitative Risk Analysis

A. Introduction

As explained above, OSHA's determination that currently exposed workers face a grave risk of asbestos-related disease is primarily based on the results of quantitative risk assessment performed by the Agency. The process of assessing the risk from asbestos exposure includes several steps. 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 (and discussed in the section entitled *Epidemiologic Evidence on Risk from Exposures at Current the PEL*. Section IV B.) 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. In addition, OSHA has made risk estimates of excess mortality from lung cancer, mesothelioma, gastrointestinal cancer and 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 current standard. The range of studies covers many different work situations and exposure levels upon which to base the extrapolations. 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 standard will lead to a substantial reduction in risk.

The sections below provide a brief synopsis of OSHA's quantitative risk estimates derived from mathematical models. A more detailed description of OSHA's calculations for asbestos-related cancers is in the public docket and is available upon request (Ex. 84-392).

B. Dose-Response Model for Lung Cancer

Dose-response information for lung cancer based on the epidemiological studies indicates that the risk of asbestos-induced lung cancer increases linearly with dose. Therefore, OSHA believes it is appropriate to use a mathematical model which assumes that the excess risk is linearly proportional to dose to estimate the excess mortality which is associated with asbestos exposure. This excess risk is expressed as the number of additional lung cancer deaths per 1000 workers exposed for a specific time period. Excess risk equals the cumulative dose multiplied by a potency coefficient (slope of the dose-response curve) estimated from the studies. [Note: 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) and the intensity of exposure (which is workplace air concentration in mppcf or fibers/cc). Under this definition of exposure, a person exposed to airborne asbestos at 2 fibers/cc for 20 years (40 fibers/cc-years) has the same total dose as a person who is exposed to asbestos at 4 fibers/cc for 10 years (40 fibers/cc-years)].

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 the 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 the section), all three demonstrate a linear relationship over the entire range of observation.

Other scientists 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). 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.

Relative risk is defined as the ratio of the mortality rate of exposed persons to the mortality rate of equivalent non-

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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.

Based upon the considerations listed above and on the data, the relative risk model used by OSHA in assessing the risk of developing lung cancer from asbestos exposure is described by the following equation:

$$R_L = R_E [1 + (K_L \times f \times d_{t-10})] \quad \text{(Eq. 1)}$$

That is,

$$(R_L/R_E) - 1 = K_L \times f \times d_{t-10} \quad \text{(Eq. 2)}$$

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 the asbestos exposure in years (minus 10 years to 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). (For a more detailed rationale for the choice of this model, see Ex. 84-392). This calculation was performed 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 concept of direct proportionality of the risk of lung cancer to the dose can be demonstrated with a simple example. Using Equation 2, if does is increased by a factor of three, such as increasing the duration of exposure from two years to six years, the excess relative risk [$(R_L/R_E) - 1$] risk increases by a factor of three. Also, reducing dose by reducing air concentrations, such as from 4 fiber/cc to 1 fiber/cc, reduces the excess relative risk by a factor of four. [It should be noted that the estimates of risk given in Tables 10 and 11 are estimates of excess risk, not excess relative risk. Therefore, direct proportionality of dose and risk as described above may not be observed. Further explanation is given in Section E.]

C. Dose-Response Model for Mesothelioma

OSHA believes that the risk of mesothelioma is best estimated by an absolute risk model. Absolute risk is calculated as (observed deaths/person-years at risk). 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. In addition to using absolute risk rather than 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 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 for 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 are also available from three studies 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 data indicate that mortality from mesothelioma begins to increase only after a minimum of ten years following the initial exposure and begins to decrease after 45 years from onset of exposure (Selikoff et al., 1979, Ex. 84-90 and Nicholson et al., 1983, Ex. 84-251). The mortality from mesothelioma can be described by the following equations (Equations 3).

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$$AR_M = f \times K_M [(t - 10)^3 - (t-10-d)^3] \quad \text{for } t \geq 10+d$$

$$AR_M = f \times k_M (t-10)^3 \quad \text{for } 10+d > t \geq 10$$

$$AR_M = 0 \quad \text{for } 10 > t$$

where AR_M is the 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 a dose-response curve) (Ex. 84-392).

D. OSHA's Quantitative Risk Assessment

OSHA's critical evaluation of all relevant animal and epidemiological studies resulted in selection of eleven epidemiological studies for calculation of the K_L for lung cancer (Selikoff et al., 1979, Ex. 84–90; Seidman et al., 1979, Ex. 84–87; 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; Liddell et al., 1977, Ex. 84–65; Nicholson et al., 1979, Ex. 84–72; and Rubino et al., 1979, Ex. 84–86) and four for K_M for mesothelioma (Selikoff et al., 1979, Ex. 84–90; Seidman et al., 1979, Ex. 84–87; Finkelstein et. al., 1983, Ex. 84–240; and Peto, 1980, Ex. 84–169). In general, studies of human cohorts in the workplace should provide a better basis for quantitative risk populations at risk and the populations from which the risk estimates are derived. In determining the potency coefficients K_L and K_M, Equations 1 and 3 were used to define the dose-response relationship 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 due to the many high quality human studies that exist that were conducted in actual workplace situations. Rather OSHA has supplemented the human data with results from animal studies in the evaluation of the health information and in the determination of the significance of risk because OSHA believes that the animal studies provide valuable qualitative information on asbestos-related disease. It is 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 for developing lung cancer. However, the animal studies do show that all commercial asbestos types can cause cancer and pulmonary fibrosis. Animal studies also indicate that longer, thinner fibers may have greater carcinogenic potency than short coarse fibers.

The range of estimates of risk from the eleven epidemiologic studies is rather large. The differences in results among the studies can be explained in several ways. There appears 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 distributions (longer finer fibers appear to have greater carcinogenic potential than the coarse fibers). The observed K_L values for studies of mining and milling operations, where airborne fibers are relatively coarse, are lower than the K_L values found in studies of textile operations where fibers are fine.

Differences may also be explained by the variations in study design and other factors influencing the ability to detect excess risks. 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 for few worksites and then only occasionally. OSHA has evaluated these differences and have dealt with their implications on a study by study basis, as explained in the quantitative risk assessment (Ex. 84–392). OSHA notes that, despite these apparent limitations, 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.

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), McDonald et al. (Ex. 84–48), and Henderson and Enterline (Ex. 84–48), have 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/cc-years) according to the time that an individual spent at a job with a measured exposure value. A conversion factor for converting from mppcf to f/cc was employed on a study by study basis depending upon the data available. Other epidemiological studies, for example those by Selikoff et al. (Ex. 84–90), and Seidman et al. (Ex. 84–87), had neither job histories nor direct industria 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. The study by Seidman et al., however, contained good information regarding duration of exposure (which can often be examined as a surrogate fc dose in establishing the shape of the exposure-response relationship).

As discussed in Section IV, OSHA ha concluded that workers exposed to

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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. In general, the risk ranges from about 5 to 20% of the excess lung cancer risk. (A detailed table of the risk from gastrointestinal cancer observed in 27 studies is given in Ex. 84–392). In an attempt to quantify the risk of gastrointestinal cancer, OSHA considers that a simple risk model in which the lung cancer excess is multiplied by 0.1 (10%) is appropriate for estimating the risk from gastrointestinal cancer. The estimates of risk from gastrointestinal cancer are also given in Tables 10 and 11.

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

E. Estimates of Cancer Mortality

A best estimate of K_L was calculated for each of the eleven epidemiologic studies and an estimate of K_M was calculated from four of these studies (see Table 5) (Ex. 84–392). For each study, the best estimate for K_L and K_M is indicated along with a range of uncertainty. The ranges listed are those are the result of estimates of exposure uncertainties (usually a factor of 2), methodological uncertainties that led to alternate evaluations of risk or exposure or in some cases, statistical uncertainties associated with small numbers. Detailed derivation of each range of uncertainty is discussed in Ex. 84–392.

The distinct nature of mining-milling data (and hence, the estimates of K_L from these data) have 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. Thus, in determining the best overall value for K_L from the eleven studies, the data were examined both with and without the K_L calculated from the studies of mining-milling processes.

The range of individual values for K_L covers two orders of magnitude, from 0.0006 to 0.068. The arithmetic mean of the eleven values of K_L (unit risk per f-y/cc) is 0.0201, and 0.0267 when the K_L's from mining and milling are excluded. The geometric mean of the data is 0.007; when the estimates of K_L from mining-milling operations are excluded, the geometric mean of the K_L's is 0.0113. The K_L's have a median of 0.0051 with the mining-milling processes and a median of 0.0138 when the mining-milling processes are excluded.

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 has 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.

The estimates of K_M given in Table 15, are derived from studies with four of the five highest K_L values. That is, there is 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. The arithmetic mean of the K_M's is 4.98×10^{-4}, and the geometric mean of the K_M's is 2.91×10^{-4}.

To account for some of this bias when estimating K_M, it is useful to examine the ratio of K_M to K_L. For the four studies for which K_M was calculated, the range of the ratios of K_M to K_L is only two-fold (from 0.75×10^{-4} to 1.79×10^{-4}). Both the arithmetic and geometric means of these ratios are 1×10^{-4}. Thus, 1×10^{-4} is an appropriate choice as the best estimate of K_M/K_L. Using this estimate of the ratio K_M/K_L and the preferred estimate of K_L (0.01), the preferred estimate of K_M would be 1×10^{-6} ($K_M=1\times10^{-2}\times1\times10^{-4}$). A range of 3×10^{-6} to 3×10^{-5} for K_M would appropriately represent most exposure situations.

There is no evidence in this analysis that would suggest that a special lung cancer potency is ascribable to a specific type of fiber. Some of the highest and lowest values for K_L are obtained from pure chrysotile exposures (for example, K_L calculated from data of Dement et al. is 0.042; using data from Peto et al. gives a K_L of 0.0076). Exposures involving a mixture of fibers, including amosite and crocidolite, also span a large range of values for K_L. Wide differences also occur in the results of separate epidemiological studies of similar work conditions.

Some scientists have suggested that some asbestos processes, such as asbestos textile manufacturing, may pose a greater hazard than other processes. For example, mining and milling appears to pose a lesser carcinogenic hazard than manufacturing processes. OSHA compared the potency factors for lung cancer (K_L) among different epidemiology studies of manufacturing processes because the potency factors are based on risk per unit dose. No consistent pattern of differential lung cancer risk (i.e., higher K_L's) by process emerged. One study of asbestos textile workers found a very high risk while another found a much lower risk, and the same was true for the two studies of asbestos production workers and the two studies of asbestos cement workers. Therefore, the choice of a midpoint unit risk for all industrial processes ($K_L=0.01$) is a reasonable and justified choice.

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 beginning at different ages. The calculations are age, intensity and duration specific. Tables 10 and 11 show the excess asbestos-related mortality rates from lung cancer, mesothelioma, and gastrointestinal cancer (gastrointestinal cancer excess is assumed to be 10 percent of the lung cancer excess). In these calculations, Equation 1 and Equation 3 were used with values of K_L equal to 0.01 and K_M equal to $1\times10-^{5}$ 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,

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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 Tables 10 and 11 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 10 gives estimates of risk for one year of exposure to asbestos at various concentrations for workers beginning exposure at ages 20, 30, 40, 50 and 60. It should be noted that employees exposed at earlier ages show higher risk of all cancers due to the long period of time during which it will be possible for disease to develop. One year of exposure to asbestos at 2 f/cc starting at age 20 may result in a total excess cancer risk of 345 per 100,000 workers.

Table 11 gives the predicted excess lifetime risk of cancer for exposures of one year, 20 years, and 45 years assuming first exposure at age 25.

Several comments should be made regarding the results in Tables 10 and 11. Though excess relative risk is linear in dose, the excess mortality rates given in Tables 10 and 11 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 exposures categories, where exposure 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 the 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 11, the predicted risk from mesothelioma is approximately equal to the lung cancer risk for one year of exposure and, about one-half 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 percent of the excess mortality from lung cancer.

Using the equations given earlier, and based on the calculations in Table 11, OSHA predicts a lifetime excess risk of total cancer for a lifetime exposure (45 years) to 2 f/cc as 6,411 excess deaths per 100,000 workers, or approximately 64 per 1,000. Recognizing that a 20 year exposure to asbestos may be another approximation of actual worker experience of interest, the models predict an excess cancer mortality of 4,392 deaths per 100,000 workers.

Reduction in the PEL from 2 f/cc to 0.5 f/cc reduces the risk from lifetime exposure from 64 per 1,000 to 17 per 1,000. Similarly, for a 20 year exposure, the risk is reduced from 44 per 1,000, to 11 per 1,000, representing a 75% 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 74 per 100,000 at 0.5 f/cc. As discussed above, this implies that the lifetime excess risk from a six-month exposure to asbestos would be approximately 198 per 100,000 at 2 f/cc, and 37 per 100,000 at 0.5 f/cc.

Lastly, Table 11 contains the risks for levels higher than 2 f/cc, because OSHA believes some industrial areas (such as construction) may be at these higher levels. This population of workers would consequently experience a much greater reduction in risk by exposures to 0.5 f/cc, or less.

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Table 9

Estimates of the Slope Parameters for Lung Cancer (K_L and Mesothelioma (K_M), by Process

Study	K_L 1/	$K_M \times 10^8$	K_M/K_L ($\times 10^6$)
MANUFACTURING			
Asbestos Products			
Henderson & Enterline (1979)	0.0047 (0.0026 - 0.0066)		
Asbestos Cement Products			
Weill et al. (1979)	0.0033 (0.0016 - 0.0086)		
Finkelstein (1983)	0.067 (0.033 - 0.13)	12 (4-30)	1.79
Textile Products			
Peto (1980)	0.0076 (0.0009 - 0.023)	0.7 (0.3-2)	0.92
Dement et al. (1982)	.042 (0.023 - 0.21)		
Friction Products			
Berry & Newhouse (1983)	0.0006 (0 - 0.008)		
Insulation Products			
Seidman et al. (1979)	0.068 (0.0049 - 0.14)	5.7 (3-11)	0.84
INSULATION APPLICATION			
Selikoff et al. (1979)	0.020 (0.008 - 0.030)	1.5 (0.5 - 2.5)	.75
MINING-MILLING			
Liddell et al. (1977)	0.00065 (0.0002 - 0.0011)		
Nicholson et al. (1979)	0.0023 (0.001 - 0.007)		
Rubino et al. (1979)	0.0051 (0 - 0.009)		

1 Values in parentheses represent the range of uncertainty around the estimates of K_L and K_M. These are computed on a study by study basis and calculations for each study are discussed in the text.

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Table 10

Estimated asbestos related cancer mortality from a one year exposure to various fiber concentrations

Asbestos fiber concentration (f/ml)	Cancer mortality /100,000 exposed			
	Lung	Mesothelioma	Gastrointestinal[1]	Total
Age at first exposure: 20				
0.1	7.2	9.5	.7	16.2
0.2	14.3	18.9	1.4	34.6
0.5	35.8	47.3	3.6	86.7
2.0	142.8	188.7	14.3	345.8
Age at first exposure: 30				
0.1	7.3	4.9	.7	12.9
0.2	14.5	9.8	1.4	25.7
0.5	36.3	24.4	3.6	64.3
2.0	144.9	97.4	14.5	256.8
Age at first exposure: 40				
0.1	7.1	2.1	.7	9.9
0.2	14.1	4.3	1.4	19.8
0.5	35.3	10.7	3.5	49.5
2.0	140.9	42.6	14.1	197.6
Age at first exposure: 50				
0.1	6.1	0.7	.6	7.4
0.2	12.2	1.5	1.2	14.9
0.5	30.4	3.7	3.0	37.0
2.0	121.7	14.7	12.2	148.6
Age at first exposure : 60				
0.1	4.0	0.2	.4	4.6
0.2	8.0	0.3	.8	9.1
0.5	19.9	0.9	2.0	22.8
2.0	79.6	3.4	8.0	91.0

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

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Table II

Estimated Asbestos Related Cancer Mortality per 100,000 by Number of Years Exposed and Exposure Level [1]

Asbestos fiber concentration (f/ml)	Cancer mortality /100,000 exposed			
	Lung	Mesothelioma	Gastrointestinal[2]	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

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

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

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F. Quantifying the Excess Risk From Asbestosis

Many studies have found the nonmalignant lung disease, asbestosis among asbestos-exposed workers. In fact, early occupational health standards governing asbestos exposure primarily sought to reduce the incidence 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 might indicate increased risk of future development of asbestosis, but this is not certain. Although the disease significance of pleural plaques is not clear, the presence of the plaques is not normal.

Berry et al. (1979, Ex. 84–20) defines "possible asbestosis" as a lung condition based upon signs of early asbestosis. He classifies "possible asbestosis" on the finding of basal rales or crepitations, radiological changes of varying degree, a falling gas transfer factor, and restrictive changes in lung volume or ventilatory capacity. The definition of asbestosis varies among different investigators. Berry et al.'s definition of possible asbestosis, for example, is not sufficient for the victim to qualify for British Disablement Benefit.

Asbestosis can progress to a disabling condition where active work is no longer possible. Although no single definition of disability exists, various governments have adopted definitions of asbestos disability to administer their worker's compensation laws. Finkelstein explains the system used by the Ontario Workmen's Compensation Board:

> Workers submitting claims to the Compensation Board are examined by the Advisory Committee, which recommends whether or not a Disability Pension be granted. There are no strict criteria for the awarding of a Disability Pension for asbestosis, but some of the factors considered include a history of "adequate" exposure, a history of dyspnea, the presence of crepitations or clubbing, radiographic signs of pulmonary fibrosis and abnormal pulmonary function results (Ex. 84–44, p. 496).

Asbestosis has been known to progress or worsen after cessation of exposure, 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.

Because of the many possible definitions of asbestosis given by different groups, the quantification of a single risk associated with asbestosis is difficult. It is clear that material impairment from asbestosis occurs prior to the onset of its disabling stage. 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. 84–20 and 84–44).

Two definitions will be helpful in interpreting the data concerning asbestosis. *Incidence* is the rate at which new cases of asbestosis develop in a given period of time. It is a direct measure of the risk of developing the disease. On the other hand, *prevalence* measures the number of cases alive in a population at a given period of time. Numerically, it equals the sum of all the incidence cases in the past minus all the deaths that have occurred in people who had developed the disease. Prevalence can be reflective of existing risk for asbestosis in a population; prevalence however, can be high for other reasons, such as increased survival. Moreover, incidence establishes a time sequence whereas prevalence looks at both cause and effect simultaneously. The best estimates of risk of asbestosis have been calculated from incidence data of two studies. Berry and Lewinsohn (Ex. 84–254) and Finkelstein (Ex. 84–44). OSHA has also examined prevalence rates to support a quantitative assessment of risk for asbestos, and these data will be discussed first.

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. Table 12 shows the observed prevalence of crepitations, "possible asbestosis", and certified asbestos for data from Berry et al., as taken from the fitted curve in Figure 4 of their paper. Possible asbestosis was diagnosed by the factory medical officer if he thought that a man was developing signs or symptoms of early asbestosis; 50% of the men diagnosed with possible asbestosis received certification within the 3.5 years following this diagnosis. The results in Table 12 represent a group of workers employed after 1950, who had a relatively short duration of follow-up (maximum interval from first exposure was 23 years). A higher percentage of asbestosis would most likely have been observed if the follow-up period in the study had been extended.

The observations of Berry et al. presented in Table 12 are probably underestimates of risk. First, the risk shown in Table 12 does not give the probability of developing disease after exposure has ended, but rather, reports the disease found at one point in time. Second, the data includes some workers who may not have been followed long enough for asbestosis to appear.

TABLE 12

PREVALENCE OF CREPITATIONS, POSSIBLE ASBESTOSIS AND CERTIFIED ASBESTOSIS FOR CUMULATIVE EXPOSURES TO ASBESTOS FROM BERRY et al. (1979)

	Cumulative exposure, fiber/cc - years		
Percent with condition	Crepitations	Possible Asbestosis	Certified Asbestosis
1	37	46	63
5	65	84	100
10	86	118	130
15	100	134	148

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These data of Berry et al. demonstrate several features of the nature of the asbestosis risk. The data show a continuum of clinical response over the range of doses; that is, these clinical observations support the existence of a dose-response relationship. Second, these observations also support the existence of a low, or possibly no, threshold for asbestosis, since there is increased risk at cumulative exposures as low as 37 fiber/cc-years.

Berry and Lewinsohn (1979, Ex. 84–254) have reported the incidence of asbestosis in this same asbestos textile factory. These data are presented in Table 13. A dose-response relationship is apparent for the incidence data, but it is not quite as consistent for the prevalence data.

Table 13
Incidence of Possible Asbestosis by Cumulative Exposure
Berry and Lewinsohn

Cumulative Exposure (fiber/cc-years)	% Incidence First employment Before 1951	After 1950
0-49	-	0.4
50-99	3	1
100-149	2.6	2
150-199	3.9	
200-249	6.2	
300-349	4.6	

Finkelstein (1982, Ex. 84–44) looked at the development of compensable (certified) asbestosis among 201 workers at an asbestos-cement 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. Table 14 shows the incidence of certified asbestosis cases as a function of cumulative exposure from the Finkelstein study.

TABLE 14

INCIDENCE OF CERTIFIED ASBESTOSIS AS A FUNCTION OF CUMULATIVE EXPOSURE FINKELSTEIN (1982)

Cumulative exposure fiber-years/cc	% Incidence
0-49	0.5
50-99	3.4
100-149	6.5
150-199	7.9
200-249	14.3

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." On the other hand, Finkelstein's study may have understated asbestosis risk by examining only certified disability from asbestosis, which is an advanced stage of asbestosis.

OSHA's estimates of risk were derived from a simple linear regression of the incidence on the midpoints of the cumulative exposure data of Berry and Lewinsohn (Table 13) and Finkelstein (Table 14). A linear relationship was assumed, at least to the point estimation of 0.5 fibers/cc for 45 years (or 22.5 fiber years/cc). This assumption is consistent with the fact that early stages of diseases are observed at low exposures. A similar conclusion is drawn in the report of the British Advisory Committee on Asbestos (Ex. 84–216, volume 2, p. 38). "The present authors come down in favor of a dose response relationship without a threshold for chrysotile within the range experienced in industry."

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TABLE 15

ESTIMATES OF LIFETIME ASBESTOSIS INCIDENCES

Exposure level, fiber/cc	% Incidence		
		Berry: first employed:	
	Finkelstein	Before 1951	After 1950
0.5	1.24	0.45	0.35
1	2.49	0.89	0.69
2	4.97	1.79	1.38
5	12.43	4.46	1.38
10	24.86	8.93	6.93
Slope	0.055	0.020	0.015
R^2	0.975	.901	.994

The results of each of the regression analyses and predictions of incidence for several dose concentrations are given in Table 15. At this time OSHA makes no attempt to extrapolate the data using this model below the 0.5 fiber/cc level or above 10 fiber/cc level. The estimates from the 3 cohorts differ by an approximate factor of 3. This may be indicative of some of the methodological issues raised earlier. 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 first exposure. Thus, it is likely that the low incidence rates in the Berry and Lewinsohn study (and therefore, the low estimate predicted by OSHA) are reflective of the short follow-up period for this group of workers.

OSHA believes that the best estimates of the incidence of asbestosis are those derived from the Finkelstein data. They indicate that among workers exposed for a lifetime exposure to 2 f/cc of asbestos, approximately 5% will develop asbestosis. Reducing this level to 0.5 f/cc would reduce incidence to 1.24%. It should be noted that these risk estimates represent incidence of disabling asbestosis. First signs of adverse pulmonary effects are reported to occur at lower levels.

G. Other Quantitative Risk Assessments

Since 1979, several scientists and scientific committees have estimated risk associated with asbestos exposure. (Exs. 84–1, 84–2, 84–216, 84–256.) The risk assessments are in approximate agreement. They all use epidemiological studies conducted in the occupational environment to generate quantitative estimates. Animal studies are used to only support and justify methodological procedures and assumptions qualitatively. For lung cancer, scientists generally accept the linear model relating increased asbestos exposure to risk and generally accept the lack of a threshold. As stated by the British Advisory Committee on Asbestos: "For lung cancer, the available data in man, all of which are derived from industry, show an increase in risk with increasing dose of dust, and we find no evidence within the range of dust levels studied for a threshold of dose below which there is no evidence of risk" (Ex. 84–216, p. 55). In general, all the risk assessments use cumulative exposure as the measure of exposure for all cancer risk estimates (cumulative exposure equaling intensity times duration of exposure).

Given the uncertainties inherent in quantitative risk assessment, as well as the inevitable variations in findings among the many epidemiologic studies, OSHA believes that the different quantitative risk estimates agree relatively well. The variations that do exist can be explained by the assumptions made or by simple methodological differences. Some variations may be due to differences in the work environment used in the assessments.

For example, Crump's assessment, given as testimony to the Ontario Royal Commission on Asbestos in August 1981, contains quantitative estimates of risk for several studies, including smoking-specific risk estimates. Crump does not give a best estimate of risk or an overall risk estimate. His estimates of risk are based upon an assumption that worker exposure that would result from a standard set at a 2 fiber/cc limit is actually much lower than 2 fibers/cc. That is, his risk estimate under a 2.0 fibers/cc standard assumes that average worker exposure would be only 1.0 fibers/cc (p. 49). He reduces this level by a factor of two to account for differences between personal and static sampling (p. 50). Thus, Crump's estimates for a 2 fiber/cc standard will be four times lower than those estimates made by OSHA for average worker exposures of 2 fiber/cc. Such differences in assumptions should be kept in mind when comparing risk assessments by different authors (Crump's estimate at 2 f/cc would be compared to OSHA's estimate at 0.5 f/cc). OSHA has presented the risk as if the working population were exposed to an average concentration of 2, 1, 0.5 and 0.1 fibers/cc reflecting OSHA's belief that a standard of 2 f/cc does not preclude worker exposure at that level; in fact exposures may even exceed 2 fibers/cc for short periods and still produce an 8-hour TWA below 2 f/cc.

The report of the British Advisory Committee on Asbestos contains one of the first quantitative risk assessments performed for asbestos. This was updated in 1983 by Acheson and Gardner (Ex. 84–243). The report describes risks for lung cancer, mesothelioma, other asbestos related cancers and asbestosis and it contains a rather thorough description of the health hazards associated with asbestos exposure. The British report's risk estimates for lung cancer do not differ in a major way from OSHA's estimates. For example, with regard to exposure to chrysotile, the Acheson and Gardner update states that "We concluded that, for example, an excess mortality from asbestos-related disease of 2 percent might be associated with any point in a range of from 5 fibers/ml to 0.4 fibers/ml and that bearing in the mind the very considerable uncertainties a figure towards the lower end of the array [0.4 f/ml] might represent an appropriate compromise." (Ex. 84–243, p. 14). As a comparison from Table 11, OSHA's best estimate of risk is that an exposure to 0.5 fibers/cc (ml) would result in a 1.2 percent increase in deaths from lung cancer (or 1143 excess deaths per 100,000).

VI. Technical and Economic Feasibility

Based on an evaluation of evidence contained in the record, OSHA finds that the provisions required by the ETS are technically and economically feasible. OSHA has examined the various industries and work operations impacted by the standard and their ability to comply with the provisions of the ETS. Because the ETS requires prompt reduction of risk, OSHA

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assessed the industry's ability to implement the required controls immediately.

The ETS allows considerable flexibility in achieving the PEL. As a result, three options are available to lower the asbestos fiber concentrations to which workers are exposed: (1) Engineering controls such as automatic bag opening devices, specialized vacuum equipment and increased ventilation; (2) work practices, such as wet treatment of the asbestos material and increased clean-up of the work place, and (3) use of approved respirators. Due to the emergency nature of this action which requires immediate response to reduce worker exposure, OSHA assumes that respirators will be the initial method used to comply with the ETS. A full discussion of the technological and economic feasibility of the alternative methods for each industry and for the various PELs under consideration for revising the permanent standard will accompany the proposal which will be published separately.

OSHA believes that, consistent with the estimates of current exposure levels, engineering controls are currently in place and work practices, in operation which, if applied conscientiously, would immediately result in concentrations at least as low as 0.5 in many industries (Ex. 84–262; Ex. 84–263; Ex. 84–9 and Ex. 84–295). For purposes of assessing the technological and economic feasibility, however, OSHA assumed a worst-case scenario in which each industry segment would have to implement a respirator program in order to achieve immediate reduction in worker exposure levels below the estimated current concentrations. Furthermore, for purposes of worst-case analysis, OSHA assumed that none of these industries has any respirator program except for the shipbuilding and construction segments. OSHA makes this assumption because OSHA estimates that most workers in industries other than shipbuilding and construction are exposed to eight-hour time-weighted averages less than 2 f/cc, and the OSHA standard issued in 1972 only requires a respirator program when engineering controls and work practices cannot bring exposures to 2 f/cc. To the extent that some firms do have an existing respirator program, the costs are overestimated.

A. Technical Feasibility

The following table presents the assumptions OSHA made regarding the respirator program elements required by each industry to obtain a PEL of 0.5 f/cc. The types of respirators needed for each industry sector were determined using the respirator selection table in the ETS with reference to the estimated current exposure conditions. OSHA assumed that the least costly approved respirator would be selected. For example, where industries have exposures less than ten times the PEL, OSHA anticipated that disposable respirators would be purchased, because of their lower short-term costs. When exposures exceeded ten times the PEL, OSHA assumed that some plants would either use air line respirators, or full facepiece respirators, depending upon the operation. To the extent that firms choose a higher-cost respirator to increase the protection factor or durability, respirator costs may be understated. Furthermore, OSHA has not included in the cost analysis a consideration for lost worker productivity due to wearing respirators. Costs may be understated by whatever amount productivity is reduced. Other anticipated respirator program elements required to determine costs for the ETS are listed below. These elements are derived from the existing provisions found in the Asbestos Standard, 29 CFR 1910.1001 and the standards for respirators, 29 CFR 1910.134.

All of the required respirators and filters are readily available and can be purchased through local distributors. Since the program relies mostly on disposable respirators, OSHA considers that there will be no supply constraints. As the worst-case (or high) estimate, OSHA assumes that approximately 50,000 workers will wear respirators because of the ETS who did not previously wear respirators. OSHA has concluded that the use of respirators will be effective in providing improved worker protection during the period of the ETS. In addition to encouraging generally more widespread use of respiratory protective measures, the ETS will stimulate a heightened understanding of the health hazards from asbestos exposure and will result in more effective use programs and strategies. Issues involving the appropriateness of respirator use as a long-term solution to controlling asbestos exposures are raised in the section 6(b) rulemaking proceeding (see Ex. 84–345, 84–346, 84–347, 84–348).

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TABLE 16
Anticipated Respirator Program Elements Required to Meet PEL

Industry Segment	Anticipated Type of Respirators Used to Meet the PEL	Other Program Elements
Primary Manufacturing		
A/C Pipe & Sheet	1 disposable/workday/worker	Full admin. costs[b]
Friction Materials	2 air-line/plant (for high concentration situations)[a]	Fit Test[c] Training[d]
Asbestos Paper Gaskets	1 disposable/worker/day for portions exposed at 0.75	50% admin. costs Fit Test Training
Floor Tile	(Nothing for PEL of 0.5)	
Paints, Coatings and Sealants	1 disposable/worker/day for 75% of workers	75% admin. costs Fit Test Training
Textiles		
Wet Process	1 disposable/worker/day	Full admin. costs Fit Test Training
Dry Process	1 disposable/worker/day	Full admin. costs Fit Test Training
Secondary Fabricators		
Cement Sheets, Paper Products, Packing and Gaskets	1 disposable/worker/day 2 air-line/plant for high concentration situations	1% admin. costs Fit Test Training
Textiles	1 disposable/worker/day	1% admin. costs Fit Test Training
Automotive Aftermarket		
Rebuilding and Refacing	1 disposable/worker/day 1 air-line/plant	1% admin. costs Fit Test Training

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Respirator Program Requirements (Cont.)

Industry Segment	Anticipated Type of Respirators Used to Meet the PEL	Other Program Elements
Brake Repair	Nothing	
Gasoline Stations	Nothing	
Shipbuilding/Repair		20% of admin. costs
Shipbuilding	Nothing	Fit Test
Ship Repair	1 disposable/worker/day for 20% of total workforce	Training
Construction		10% admin. costs
Installation	1 disposable/worker/day	Fit Test
A/C Pipe		Training
A/C Sheet		
Roofing Felts	Nothing	
Demolition/Renovation	1 HEPA full face for 25% of the workforce	25% admin. costs Fit Test Training
Repair & Maintenance	Nothing	

[a]OSHA assumes that certain jobs such as cleaning of vacuum equipment will produce exposure levels greater than 10 times the PEL. Consequently, OSHA assumes 2 air line respirators will be required per plant except for small operations which will require only one air-line respirator per plant.

[b]Administrative costs represent the salary of one full time professional at $30,000 per annum and one full time clerk at $15,000 per annum. OSHA has adjusted administrative costs to represent expected reasonable time spent administering a respirator program in each industry. In the secondary fabrication and automotive aftermarket sectors, for example, OSHA anticipates that supervisors will spend 15 minutes a day distributing and monitoring the use of disposable respirators. These sectors have far less of a management burden than large firms, and thus, administrative costs are calculated at 1 percent of the full administrative amount. OSHA believes that administrative costs have been overstated in most segments in order to present a worst case scenerio.

[c]OSHA assumes that every employee must be fit tested for respirator use at a cost of $21 per employee.

[d]OSHA has allowed 3 hours for respirator and asbestos training. OSHA considers that this is very liberal, especially in industry segments characterized by small plants and by the use of disposable respirators. Costs for this element are therefore overstated to present the worst-case scenario.

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OSHA also examined the feasibility of analytical methods for measuring asbestos air concentrations at the 0.5 f/cc limit established by the ETS. OSHA's existing standard requires that all measurements of airborne concentrations of asbestos fibers be made by the membrane filter method at 400–450 × (magnification) (4 millimeter objective) with phase contrast illumination (29 CFR 1910.1001(e)). After reviewing the relevant evidence made available since the 1975 proposal, OSHA finds that phase contrast microscopy is a feasible and effective method for measuring airborne asbestos fibers to determine compliance with the permissible exposure levels set by the Emergency Temporary Standard.

The most important issue raised by comments to the 1975 notice is whether phase contrast microscopy analysis is capable of reliably measuring airborne concentrations of 0.5 fibers/cc and less. OSHA acknowledges that asbestos airborne measurement procedures using phase contrast microscopy, as with any industrial hygiene analytical procedure, inherently contains several error sources. These errors have been statistically evaluated by Leidel et al. (Ex. 84–62), and, in 1979, by the Air Monitoring Committee of the Asbestos Information Association/North America, referred to herein as AIA (Ex. 86–002) both using round-robin sample exchange data. Chatfield also examined this question (Ex. 84–319). In the Leidel et al. and the AIA evaluations, the error, measured as a coefficient of variation (CV), was found to be related to the number of particles counted from the filter. For 100 fibers counted Leidel, et al. found a CV of 0.12, whereas the AIA report found a CV of 0.35 [1] or errors associated with interlaboratory-intrafilter variability.

Based upon these studies, taken at their face value, it appears that the phase contrast microscope analysis is capable of reasonably reliable measurements at 0.5 fibers/cc. As stated in the AIA report (Ex. 86–002, p. AB–2), "The calculated results indicate that the 95% confidence limits on a measured 8-hour TWA can be relatively constant with a wide, but usable, range down to concentrations approaching 0.5 fibers/cc [less than] 5M."

The AIA report shows a higher error than does the Leidel et al. report. One possible reason for this difference may be that the AIA report assessed the variability in measurements as they are being made today by the many laboratories who are making the measurements. For example, only 27 of the 46 laboratories participated in the PAT program and no counting guidelines were given, whereas, the Leidel et al. report included only a small number of laboratories operated by Johns Mansville Company, that probably used very similar procedures and conducted similar training.

In late 1982, Chatfield prepared a report entitled "Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres" for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario (Ex. 84–319). Chatfield analyzed intra-and inter-laboratory variability and arrived at conclusions somewhat similar to those of AIA and Leidel. Chatfield also recommended methods by which the accuracy and precision of phase contrast microscopic analytical techniques could be improved. Significantly, he noted "in view of the number and frequency of measurements required, there is currently no fully developed alternative method [to phase contrast microscopy] which could be immediately implemented."

OSHA notes that the authorities cited above believe that it may be possible to reduce phase contrast microscopy errors if improved and standardized procedures are followed, perhaps by adding requirements to the standard. It does not appear, however, that improvements of this nature can be quickly made in the immediate format of this ETS. Therefore, based on the evidence before it at the time of issuance of this emergency standard, OSHA believes that it is generally not possible to measure asbestos concentrations below 0.5 f/cc reliably and reproducibly using phase contrast microscopy under current laboratory practices. OSHA finds that the phase contrast microscopy method can be feasibly used to measure asbestos air concentrations down to 0.5 f/cc.

[1] The AIA report also reports a separate CV for overall variability in the sample evaluation step plus "random variability in the sample collection step." This "random variability of sample collection" as described in this study may be a measure of the true difference in air concentrations from one location to the next and may not be a measure of random variability. A more carefully designed sampling strategy with precise control of instrumentation placement, air currents, and dust generation is necessary to eliminate differences in airborne dust concentrations from one location to another

B. Economic Feasibility

The industry costs based on the program elements described in Table 16 are presented in Table 17. For the ETS, the costs of asbestos training are included with respirator training. Costs for warning signs are not included, since, for the purposes of the ETS, these signs could be hand-made at very low costs. OSHA did not analyze costs associated with the alternate benefits scenario (See Table 4) since that scenario was constructed to show a lower range of benefits but does not represent an estimate of current industry practice.

The costs are overstated to the extent that some firms already have a current respirator program and to the extent that careful application of existing engineering controls and work practices would reduce concentrations to the PEL in some firms and thereby make respirator use unnecessary. Furthermore, the costs assume that dust masks and filters will have to be replaced every 8-hour day. Some of this cost (i.e., the disposable respirators, filters and administrative overhead) can be in fact spread over the period during which the ETS is in effect.

Finally, the costs assigned to the Shipbuilding/Repair and Construction-Demolition/Renovation segments represent cost which reflect increased compliance with current obligations (e.g. increased respirator use to meet the current standard as a result of the ETS training requirements) and are not directly attributable to the ETS.

Note.—OSHA anticipates that the ETS will spur many employes who previously were not in compliance to expend the necessary resources in order to come into compliance with the ETS.

The total cost is estimated at $35,565,402 for 6 months. This translates into an average cost per employee of $708. Average 6 month costs per worker presented on an industry basis in Table 17 range from $251 in the automotive aftermarket segment to $973 in the construction segment. These costs are not a large portion of industry shipments as presented in Table 18. Moreover, firms in these industries will be able to pass the costs forward because asbestos substitutes in most industries are not immediately available. For all these reasons, OSHA finds that the ETS is economically feasible.

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Table 17

Estimated Costs of Respirator Program for ETS with PEL of 0.5

Industry Segment	Total Costs ($) 1 - year	6 - months	6-mo. average Cost/employee
Primary Manufacturing	6,404.965	4,143,242	481
Secondary Fabricators	13,547,224	9,435,532	548
Automotive Aftermarket	1,620,197	1,027,864	251
Shipbuilding/repair	626,286	324,526	533
Construction	36,329,740	19,634,238	973
Total	58,528,412	34,565,402	708

Table 18

ETS Compliance Costs Compared to Sales by Industry Segment

Industry Segment	Cost/Sales
Primary Manufacturing	.003
A/C Pipe & Sheet	.002
Frictin Materials	.008
Asbestos Paper	.003
Paints, Coatings and Sealants	.005
Gaskets, Seals and Packings	.001
Textiles	.014
Secondary Fabricators	.011
Cement Sheets	.072
Paper Products	.011
Packings and Gaskets	.010
Textiles	.014
Automotive Aftermarket	
Rebuilding and Refacing	.0004
Shipbuilding/Repair	.0001
Construction	.0004

Note: Sales values are for 1977 and 1978. Values for Primary Manufacturing and Secondary Fabricators were taken from 84-003; values for the Automotive Aftermarket were taken from U.S. Industrial Outlook, 1983; and values for Shipbuilding/Repair and Construction were taken from the 1982/83 Statistical Abstract. The value for Construction was adjusted by substracting the value for Highway & Streets and Residential and adding the value for Demolition/Wrecking.

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VII. Environmental Impact, Requirements of Executive Order 12291, and the Regulatory Flexibility Act

The National Environmental Policy Act (NEPA) of 1969 (U.S.C. 4321 et seq.), as implemented by the guidelines (40 CFR Part 1500) of the Council on Environmental Quality (CEQ), requires that federal agencies assess their regulatory actions to determine if there is a potential for a significant impact on the quality of the human environment and, if necessary, to prepare an environmental impact statement.

In accordance with these requirements and DOL NEPA regulations (29 CFR Part 11 [Subpart B, 11.10 (a) (4)]) OSHA had determined that because of the emergency nature of this standard, no environmental impact statement will be perpared for the ETS. The courts have held that NEPA does not require advance preparation of an environmental impact statement for an OSHA ETS (*Dry Color Manufacturing Assocation* v. *U.S. Department of Labor* 486 F.2d 98, 107 (3rd Cir. 1973)). OSHA will assess, however, the environmental effects of the proposed permanent egulation of asbestos. The results of this study will be available for review and comment prior to the hearings on the proposed permanent standard and will be an appropriate issue for discussion at the public hearing.

In the interim, OSHA welcomes any comments on any environmental effects that might occur as a result of promulgation of a rule on asbetos.

Pursuant to the authority of Section 8(a)(1) of Executive Order 12291 OSHA has not prepared a Regulatory Impact Analysis of this ETS. Preparation of such an analysis was not practicable for OSHA to perform in time to issue this ETS to respond to the grave dangers faced by asbestos exposed workers. OSHA, however, is completing a Preliminary Regulatory Impact Analysis of the proposal to revise the permanent standard which will be made public at the time the proposal is published.

The Regulatory Flexibility Act requires an agency to prepare a Regulatory Flexibility Analysis only for those rules for which a notice of proposed rulemaking is published. OSHA's issuance of an ETS therefore is not covered by the Regulatory Flexibility Act because the OSHA Act provides that ETS's be issued without regard to notice, public comment and other requirements in the Administrative Procedure Act. The proposal to revise the permanent asbestos standard, however is subject to the requirements of the Regulatory Flexibility Act and OSHA will evaluate the proposal to ascertain whether analysis under the Regulatory Flexibility Act is required.

VIII. Summary and Explanation of the ETS

The requirements of this emergency temporary standard are set out in a new paragraph, § 1910.1001(k). They are limited to additional provisions to the existing asbestos standard which OSHA considers essential and feasible to protect employees from the grave danger resulting from asbestos exposure until OSHA can promulgate more comprehensive revisions in accordance with section 6(b) of the Act. The major changes in the standard made by the ETS are: (1) The new permissible exposure level; (2) methods of compliance permitted to achieve the new level; and (3) a requirement for the establishment of training programs within 30 days. The following section discusses the major provisions of the ETS, the necessity for including these provisions in the ETS, and some additional provisions to trigger certain requirements at the reduced permissible level of 0.5 f/cc.

1. *Paragraph (k)(1) Scope.* As part of the ETS, OSHA is adding a paragraph on the scope of the standard. The paragraph is intended to make clear that the emergency standard applies to all workplaces where employees may be exposed to asbestos in all industries covered by the current asbestos standard *ie.* general industry, construction and maritime.

2. *Paragraph (k)(2) Permissible level of exposure.* As part of the ETS, OSHA is adding paragraph (k)(2) which sets a new PEL, effective immediately, of 0.5 f/cc on an 8 hour time weighted average basis. This reduced level may be achieved by any feasible combination of engineering controls, work practices and respiratory protection in order to allow employers to institute effective measures to reduce employee exposures immediately.

OSHA chose 0.5 f/cc as the permissible exposure level primarily because it believes that reducing employee exposures to 0.5 f/cc in all industries will greatly reduce the risk of developing asbestos induced cancers, primarily lung cancer, mesothelioma and gastrointestinal cancer. As set out in the discussion on "grave danger" and "benefits", the number of lives OSHA believes may be saved through an immediate reduction of exposure to 0.5 f/cc is substantial.

OSHA also believes that the 0.5 f/cc level is appropriate for several other reasons. First, because an emergency standard must be feasible, and employers must be able to comply with the standard in a short period of time, OSHA has set a level which is likely to be achieved immediately, using equipment and control techniques that are currently available.

Second, OSHA believes, based on the data generated by OSHA's contractor, Research Triangle Institute, that some workplaces, especially in the manufacturing sector, may be close to achieving a 0.5 f/cc level through the more rigorous use of engineering controls, work practices and housekeeping methods which are now in place. OSHA encourages employers to continue their efforts to implement these methods in order to assure that, for the long term, the most comprehensive and effective program of protection from asbestos exposure will be provided.

Third, OSHA believes that reliability of the currently required asbestos measurement methods to measure asbestos exposures less than 0.5 f/cc should be open for discussion during a rulemaking hearing, rather than imposed through an ETS. OSHA has therefore not adopted the 0.1 f/cc level petitioned by the unions but instead is considering adding provisions to the asbestos standard that may improve the reliability of both sampling and analysis in the 6(b) rulemaking for the permanent standard and thus allow lower levels to be reliably measured.

3. *Paragraph (k)(3) Methods of compliance.* The ETS adds a new paragraph (k)(3), which permits employers to reduce the permissible exposure limit from the current 2 f/cc limit to the 0.5 f/cc limit by any feasible combination of engineering controls, work practices and respiratory protection. The current requirement in paragraphs (c)(1) and (c)(2) to first utilize engineering controls and work practices to reduce exposure levels to 2.0 f/cc remains unaffected by this ETS.

Flexibility in choosing compliance strategies for the period of an ETS has been incorporated in most other previously issued emergency standards. The policy reflects OSHA's determination to craft emergency standards that are truly responsive to emergency conditions and which afford immediately available protection to affected workers.

4. *Paragraph (k)(4) Employee information and training.* The ETS adds a paragraph to the asbestos standard requiring the employer to provide a training program within 30 days of publication of the emergency standard for all employees whose exposures are reasonably expected to exceed the PEL of 0.5 f/cc, without regard to the use of

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respirators, and to assure that all such employees participate in the program and are informed of specified categories of information. OSHA considers this provision to be "necessary" within the meaning of section 6(c) of the Act, to reduce the grave danger faced by asbestos exposed employees. The absence of a training program requirement in the asbestos standard has been pointed out as one of the serious deficiencies of the current standard. OSHA believes that participation in an adequate training program is essential for the protection of employees because most protective provisions enlist the employee as an active participant. For example, many employees handling asbestos depend on effective work practices. Without training in applying these work practices, employee protection would be inadequate. Where the employee plays a more passive role in his protection such as where engineering controls are relied on, training is also essential, because the employee must know the sources of workplace asbestos contamination, and the health hazards of asbestos exposure, in order to assess his own exposure situation and to help recognize when engineering controls are not operating properly. Certainly where housekeeping plays an important role in control, instruction about housekeeping methods, for example, frequent vacuuming, is essential. Perhaps most importanly, where employee protection depends upon respirator use, OSHA's experience shows that training employees in the use, fitting and limitations of respirators is critical to the effectiveness of respirator protection. Accordingly this requirement applies where airborne concentrations are reasonably expected to exceed 0.5 f/cc, even if employees use respirators to reduce breathing zone concentrations and thereby comply with the ETS.

As set forth in paragraph (k)(4) the employer must inform the employee of the health effects of asbestos exposure; the relationship between asbestos and smoking in producing lung cancer; the operations exposing employees to asbestos fibers and necessary protective steps to minimize exposure; the purpose, proper use, fitting instructions and limitations of respirators, and the contents of all the provisions of the Asbestos Standard at 1910.1001.

5. *Paragraph (k)(5) Respiratory protection during the ETS.* The ETS adds a new paragraph (k)(5) which includes a table which ties respirator selection to the 0.5 f/cc PEL. Under the ETS, the concentration cut-offs for various kinds of respirators are multiples of the reduced PEL of 0.5 f/cc, rather than multiples of the previous 2 f/cc permissible limit. For example, approved air purifying respirators may be used only where asbestos concentrations are not expected to exceed 5 f/cc (10 x the PEL). Before the ETS, because the PEL was 2 f/cc, such respirators could be used where asbestos concentrations would not have exceeded 20 f/cc (10 x the PEL of 2 f/cc).

Similarly, powered air purifying respirators may be used where asbestos concentrations do not exceed 100 times the PEL, which at the new level of 0.5 f/cc is 50 f/cc. Previously, employers could have used such respirators at concentrations up to 200 f/cc.

It is likely that the main impact of the reduced PEL on respirator choice will be in operations and industries where exposure levels are between 5 f/cc and 20 f/cc. Formerly, employees exposed in this range could use half-mask air purifying respirators; now they must be protected by a powered air purifying respirator or a full facepiece respirator, or they may use a supplied air respirator.

6. *Paragraph (k)(6). Warning signs during the ETS.* The ETS requires that legible signs warning of the health hazards of asbestos be displayed at locations where airborne concentrations of asbestos fibers exceed the reduced exposure limit of 0.5 f/cc. No specific legend is required signs for newly posted during the ETS. OSHA wishes to make as practicable as possible the rapid posting of signs, especially in workplaces where there has been previous non-compliance and in areas where asbestos concentrations were formerly below the 2.0 f/cc PEL.

XI. Public Participation

Interested persons are invited to submit written data, views and arguments with respect to the revisions to the asbestos standard made by the ETS. These comments must be postmarked on or before January 3, 1984 and sent to the Docket Officer, Docket No. H–033C, Occupational Safety & Health Administration, U.S. Department of Labor, 200 Constitution Avenue, NW., Room S–6212, Washington, D.C. 20210.

The data, views and arguments that are submitted will be available for public inspection and copying at the above address. All timely written submissions will be made a part of the record of the proceeding.

List of Subjects in 29 CFR Part 1910

Occupational safety and health, Asbestos, Health, Emergency temporary standard, Cancer.

Authority and Signature

This document was prepared under the direction of Thorne G. Auchter, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue, NW, Washington, DC 20210. Pursuant to Sections 6(b), 6(c), 8(c) and 8(g) of the Act, 29 CFR 1910.1001 is amended by adding a new paragraph (k) as set forth below.

(Secs. 6(b), 6(c), 8(c) and 8(g), Pub. L. 91–596, 84 Stat. 1593, 1596, 1599, 1600; 29 U.S.C. 655, 657; Sec. 107, Pub. L. 91–54, 83 Stat. 96 (40 U.S.C. 333); 29 CFR Part 1911; Secretary of Labor's Order No. 9–83 (48 FR 35736))

Signed at Washington, D.C., this 2nd day of November 1983.

Thorne G. Auchter,

Assistant Secretary of Labor.

PART 1910—[AMENDED]

Section 1910.1001 of Part 1910 of Title 29 of the Code of Federal Regulations is hereby amended by adding a new paragraph (k) reading as follows:

§ 1910.1001 Asbestos.

* * * * *

(k) *Emergency temporary standard effective November 4, 1983.*—(1) *Scope.* This emergency temporary standard is issued pursuant to section 6(c) of the Act and applies to all workplaces where employees may be exposed to asbestos in all industries covered by the Act, including, general industry, construction and maritime. Except to the extent modified by this emergency temporary standard all provisions of § 1910.1001 remain in effect.

(2) *Permissible levels of exposure.* The 8-hour time-weighted average airborne concentration of asbestos fibers to which any employee may be exposed shall not exceed one-half (0.5) fiber, longer than 5 micrometers, per cubic centimeter of air, as determined by the method prescribed in paragraph (e) of this section.

(3) *Methods of compliance with the emergency temporary standard.* Notwithstanding any other requirements of this section, compliance with the reduced exposure limit of 0.5 f/cc shall be achieved by any feasible combination of engineering controls, work practices, and personal protective equipment and devices.

(4) *Employee information and training.*—(i) As soon as possible, but not later than thirty (30) days from the effective date of this emergency temporary standard, the employer shall institute a training program for all employees exposed to airborne concentrations of asbestos in excess of

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5 f/cc, without regard to the use of respirators and shall assure their participation in the program during the effective period of this emergency temporary standard.

(ii) The employer shall assure that each such employee is informed of the following:

(A) The health effects associated with asbestos exposure;

(B) The relationship between asbestos and smoking in producing lung cancer;

(C) The nature of operations which could result in exposure to asbestos and necessary protective steps to minimize exposure including, as applicable, engineering controls, work practices, respirators, housekeeping and protective clothing;

(D) The purpose, proper use, fitting instructions and limitations of respirators permitted by the standard; and

(E) A review of all the provisions contained in 1910.1001.

(5) *Respiratory protection during the ETS.* Notwithstanding any other requirement of this section, where respirators are used to achieve the permissible exposure limit of 0.5 f/cc they shall be selected according to Table 1.

(6) *Warning signs during the ETS.* In addition to the requirements of paragraph (g)(1) of this section, legible signs warning of the health hazards of asbestos shall be provided and displayed at each location where airborne concentrations of asbestos fibers may exceed 0.5 f/cc.

TABLE 1

Respiratory Protection

For Airborne Concentrations of Asbestos

Airborne Concentration of Asbestos (TWA)	Required Respirator[1]
Not in excess of 5 f/cc (10 X PEL)	Reusable or single use air purifying respirator
Not in excess of 50 f/cc (100 X PEL)	Full facepiece air purifying respirator, or a powered air purifying respirator
Greater than 50 f/cc	A type "C" continuous flow or pressure demand, supplied air respirator.

[1] Respirators specified for high concentrations may be used at lower concentrations of asbestos.

(Secs. 6(b), 6(c), 8(c) and 8(g), Pub. L. 91-596, 84 Stat. 1593, 1596, 1599, 1600; 29 U.S.C. 655, 657; Sec. 107, Pub. L. 91-54, 83 Stat. 96 (40 U.S.C. 333); 29 CFR Part 1911, Secretary of Labor's Order No. 9-83 (48 FR 35736))

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