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# WORKPLACE EXPOSURE TO ASBESTOS
## Review and Recommendations

DHHS (NIOSH) Publication No. 81-103

NIOSH-OSHA
Asbestos Work Group
April 1980

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES**
**Public Health Service**
**Centers for Disease Control**
**National Institute for Occupational Safety and Health**

**U.S. DEPARTMENT OF LABOR**
**Occupational Safety and Health Administration**

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**MEMORANDUM FOR:** *Dr. Eula Bingham*
*Assistant Secretary for Occupational Safety and Health*
*Dr. Anthony Robbins*
*Director, National Institute for Occupational Safety and Health*

**FROM:** *Asbestos Work Group*

**SUBJECT:** *The Updated Scientific Information on Asbestos and Recommended Occupational Standard for Asbestos Exposure*

In the fall of 1979, a NIOSH/OSHA committee was formed at the direction of Dr. Eula Bingham, Assistant Secretary of Labor for Occupational Safety and Health, and Dr. Anthony Robbins, Director of the National Institute for Occupational Safety and Health (NIOSH), to review the scientific information concerning asbestos-related disease and assess the adequacy of the current OSHA occupational health standard of 2,000,000 fibers per cubic meter greater than 5 μm in length (2Mf/m$^3$). Since the 1972 promulgation of this 2,000,000 f/m$^3$ standard, OSHA, in 1975, proposed lowering the standard to 500,000 f/m$^3$; NIOSH, in 1976, recommended lowering the standard to 100,000 f/m$^3$; and the British Advisory Committee on Asbestos, in 1979, recommended lowering its occupational exposure standards. The NIOSH/OSHA committee has reviewed the most recent scientific information, including documents concerning the above developments and the 1977 International Agency for Research on Cancer (IARC) review of the carcinogenicity hazards of asbestos, and presents the following major conclusions and recommendations. A detailed updating of significant scientific literature since the 1976 NIOSH Criteria Document and the 1977 IARC Monograph is attached.



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*1. Definition of Asbestos.* Having considered the many factors involved in specifying which substances should be regulated as asbestos, the committee recommends the following definition:

> Asbestos is defined to be chrysotile, crocidolite, and fibrous cummingtonite-grunerite including amosite, fibrous tremolite, fibrous actinolite, and fibrous anthophyllite. The fibrosity of the above minerals is ascertained on a microscopic level with fibers defined to be particles with an aspect ratio of 3 to 1 or larger.

*2. Sampling and Analysis of Airborne Asbestos.* The committee concludes that the membrane filter-phase contrast microscopy method represents the only technique available that can reasonably be used for routine monitoring of occupational exposures and sampling for compliance purposes. However, the committee recognizes the lack of specificity of this method for fiber identification, and recommends the use of supplementary methods such as electron microscopy for fiber identification in cases of mixed fiber exposures. In recommending the primary use of light microscopy, the committee also wants to stress the inability of this method to detect short asbestos fibers to which workers are exposed. The toxicity of asbestos fibers shorter than the 5-micrometer detection limit of light microscopy cannot be dismissed on the basis of current scientific information.

*3. Biologic Effects of Exposure to Asbestos.* Animal studies demonstrate that all commercial forms and several noncommercial forms of asbestos produce pulmonary fibrosis, mesothelioma, and lung neoplasms. Chrysotile is as likely as crocidolite and other amphiboles to induce mesotheliomas after intrapleural injection, and also as likely to induce lung neoplasms after inhalation exposures.

Human occupational exposures to all commercial asbestos fiber types, both individually and in various combinations, have been associated with high rates of asbestosis, lung cancer, and mesothelioma. While significant excesses of cancer of several other sites have been observed in exposed workers, presently available information is insufficient to determine the role of specific fiber types.

On the basis of available information, the committee concludes that there is no scientific basis for differentiating between asbestos fiber types for regulatory purposes. Accordingly, the committee recommends that a single occupational health standard be established and applied to all asbestos fiber types.



Available data show that the lower the exposure, the lower the risk of developing asbestosis and cancer. Excessive cancer risks, however, have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure. Accordingly, the committee recommends that, to the extent uses of asbestos cannot be eliminated or less toxic materials substituted for asbestos, worker exposures to asbestos must be controlled to the maximum extent possible.

*4. Inadequacy of Current 2,000,000-Fiber Occupational Standard.* The committee concluded that a variety of factors demonstrates that the current 2,000,000-fiber standard is grossly inadequate to protect American workers from asbestos-related disease. First, the 2,000,000-fiber standard was designed in 1969 by the British Occupational Hygiene Society (BOHS) for the limited purpose of minimizing asbestosis. Disease prevalence data from the BOHS study population collected subsequent to 1969 strongly suggest that this standard is insufficient to prevent a large incidence of asbestosis. Second, all levels of asbestos exposure studied to date have demonstrated asbestos-related disease, and a linear relationship appears to best describe the shape of the dose-response curve. These considerations led the committee to conclude that there is no level of exposure below which clinical effects do not occur. Third, the absence of a threshold is further indicated by the dramatic evidence of asbestos-related disease in members of asbestos-worker households and in persons living near asbestos-contaminated areas. These household and community contacts involved low level and/or intermittent casual exposure to asbestos. Studies of duration of exposure suggest that even at very short exposure periods (1 day to 3 months) significant disease can occur.

Although various models can be and have been fashioned to postulate possible dose-response relationships involving asbestos, the committee believes that the limited current data preclude the creation of any one empirical curve to describe *the exact* dose-response relationship. Over the last three decades, measurement techniques for asbestos have changed in several crucial respects, and there have been no suitable methods available to date to compare the results of prior techniques to current methods.



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In addition, no adequate epidemiological information is available on the disease experience of workers exposed below the current standard and followed for a sufficient period to identify long latent effects. Consequently, the committee cannot present a precise dose-response relationship for the variety of asbestos-related diseases. However, the committee firmly believes that compelling evidence demonstrates that prevention of asbestos-related diseases requires that an occupational standard minimize all asbestos exposures, and definitely be set far below the current 2,000,000-fiber standard.

*5. Recommended Occupational Standard for Asbestos Exposure.* Given the inadequacy of the current 2,000,000-fiber standard, the committee urges that a new occupational standard be promulgated which is designed to eliminate non-essential asbestos exposures, and which requires the substitution of less hazardous and suitable alternatives where they exist. Where asbestos exposures cannot be eliminated, they must be controlled to the lowest level possible. A significant consideration in establishing a permissible exposure limit should be the lowest level of exposure detectable using currently available analytical techniques. At present this level would be 100,000 fibers greater than 5 μm in length per cubic meter averaged over an 8-hour workday. Regardless of the choice of a permissible exposure limit, the best engineering controls and work practices should be instituted, and protective clothing and hygiene facilities should be provided and their use required of all workers exposed to asbestos. Respirators are not a suitable substitute for these control measures. The committee also reiterates its judgment that even where exposure is controlled to levels below 100,000 fibers, there is no scientific basis for concluding that all asbestos-related cancers would be prevented.

*6. Medical Surveillance Program.* Appropriate medical surveillance is crucial to detect and minimize the progression of some asbestos-related diseases. Considerable emphasis should be placed on baseline medical examinations for all workers potentially exposed or who have been exposed to asbestos at any level. These examinations should include the following: (1) a 14″ x 17″ postero-anterior chest X-ray; (2) spirometry including forced vital capacity (FVC) and forced expiratory volume in one second ($FEV_1$); (3) a physical examination of the chest including auscultation for the presence or absence of rales, rhonchi,



and wheezing; (4) an assessment of the presence or absence of finger clubbing; and (5) a history of respiratory symptoms and conditions including tobacco smoking.

An occupational history should include a history of exposure to asbestos and exposure to other substances of real or potential medical significance. Performance criteria for these procedures, including the periodicity of subsequent medical surveillance, should be developed by NIOSH in consultation with OSHA and professional societies and organizations concerned with the diagnosis and prevention of respiratory diseases. The committee does not recommend comprehensive annual medical examinations as presently required. Sputum cytology should be evaluated in the development of an improved medical surveillance program. The committee believes that sputum cytology may prove to be a valuable supplement to X-ray evaluation.

It is also crucial that all required medical surveillance be promptly evaluated and the results reported to the employee. Furthermore, the standard should provide for periodic reporting of aggregate medical information concerning an employer's entire workforce. Results at a minimum should be displayed in a non-identifiable, aggregate format so that the employer, employees, and OSHA can see the prevalence of abnormalities possibly associated with asbestos-related disease, and also see how this prevalence has changed over time.

The committee recognizes that OSHA's recent lead standard contains a multiple physician review mechanism whereby workers can get independent medical evaluations by physicians of their choice. The lead standard also contains a medical removal protection program whereby workers can obtain special health protection where necessary, accompanied by appropriate economic protection. The committee feels that these programs are relevant to asbestos workers and should be considered as part of a new occupational asbestos standard.

Medical records generated due to the standard's medical surveillance program should be maintained for at least 40 years or for 20 years after termination of employment, whichever is longer.

*7. Other Recommendations.* The committee further recommends the following: (1) Due to the widespread current and past uses of asbestos products in the maritime and construction in-



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dustries, it is vital that any new asbestos standard address these industry sectors as well as other workplaces with employees exposed to asbestos. Regulation of these industries should be structured around the principle that where work must be done using asbestos, only those employees needed to do this work should be present, and only for the minimum period of time needed to complete this work.

(2) Due to the sampling and analytical difficulties concerning asbestos, manufacturers of asbestos-containing products such as construction materials should perform detailed monitoring of exposures which could result from all foreseeable uses of their products, including misuse. This monitoring should include electron microscopy to identify fiber type mix and exposures to fibers less than 5 μm in length. This monitoring data should accompany these products downstream so the users not only know that asbestos exposures may occur, but also know the nature of potential exposures. This monitoring data could, if appropriate, avoid the need for small employers who use asbestos-containing products to have to conduct monitoring on their own.

(3) Due to the fact that other agencies regulate occupational exposures to asbestos (such as the Mine Safety and Health Administration), these agencies should be urged to participate in the development of a new standard and adopt this new standard.

(4) Because cigarette smoking enhances the carcinogenic effect of asbestos exposure on the lung, particular emphasis should be placed on this in any educational program developed under a new standard.

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# I. ASBESTOS NOMENCLATURE/DEFINITIONS.

## Review

There is considerable controversy as to which mineral particles should be considered "asbestos" insofar as demonstrated health effects are concerned (Campbell et al., 1977; Ampian, 1978; Zoltai, 1978; Langer, 1979). Until recently, most environmental and epidemiological studies concerning mineral fibers were focused on occupational cohorts exposed to asbestos fibers from commercial deposits. However, there is currently much interest in exposures to other minerals such as submicroscopic amphibole fibers and cleavage fragments and related health effects. Mineralogists have voiced concern that mineralogical terms have been used improperly, potentially classifying many non-asbestos materials as asbestos (Ampian, 1978; Campbell et al., 1977; Zoltai, 1978).

An important problem which exists is the basic definition of what minerals should be called "asbestos." Various definitions have been proposed or used:

*National Academy of Sciences (1977):*
"The name for a group of naturally occurring hydrated silicate minerals possessing fibrous morphology and commercial utility."

*Bureau of Mines (1977):*
"(1) A collective mineralogical term encompassing the asbestiform varieties of various minerals; (2) an industrial product obtained by mining and processing asbestiform minerals." Asbestiform minerals were further defined to be "a specific type of mineral fibrosity in which the fibers and fibrils possess high tensile strength and flexibility."

*IARC (1977):*
"Asbestos is the generic name used for a group of naturally occurring mineral silicate fibers of the serpentine and amphibole series."

*Zoltai (1978):*
"... a collective term referring to the unusual crystallization of certain minerals in the form of long, strong, and flexible fibers, aggregated in parallel or radiating bundles from which fibers can *easily* be separated."



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The above definitions demonstrate an important problem. That is, the condition of fibers in nature as a result of crystal growth is the only criteria for distinguishing asbestos from other silicates (Langer, 1979). Most properties mentioned above can only be measured on bulk samples (megascopic properties). However, airborne fibers in the occupational setting are only observable on the microscopic level, thus not allowing measurements of such properties as tensile strength and flexibility. Langer et al. (1979) have pointed out that, using strict mineralogical nomenclature, isolated submicroscopic single fibers derived from known asbestos sources could not be termed "asbestos." In fact, among the many minerals demonstrating a fibrous habit in nature, only six minerals are commercially exploited and thus considered "asbestos." These include the serpentine mineral chrysotile and the amphiboles cummingtonite-grunerite including amosite, anthophyllite asbestos, tremolite asbestos, actinolite asbestos, and crocidolite.

In addition to problems relative to exposures to mineral fibers other than one of the six mentioned above, acicular cleavage fragments are frequently indistinguishable from mineral fibers derived from commercial asbestos sources, especially on the submicroscopic scale. Cleavage plays an important role during commutation for some amphibole minerals. Submicroscopic amphibole mineral fragments often demonstrate structural and chemical properties indistinguishable from asbestos homologues. Airborne size characteristics such as length and diameter are often similar to asbestos.

## General Definition

The foregoing considerations present a fundamental question of how broad a new or revised regulation should be. Arguments can be made for inclusion of all fibrous minerals posing risks comparable to commercially exploited fibrous minerals called asbestos. On the other hand, the fibrous minerals generally called asbestos appear to form the most pressing hazards to the largest number of current workers. This effort has not attempted to recommend coverage of all fibrous minerals or analogs, but has focused on commercial materials generally considered asbestos and asbestiform contaminants common to commercially exploited materials. We recommended the following definition for regulatory purposes, pending thorough and complete regulation of the hazards of all fibrous materials.



NIOSH Def.

1. Asbestos is defined to be chrysotile, crocidolite, and fibrous cummingtonite-grunerite including amosite, fibrous temolite, fibrous actinolite, and fibrous anthophyllite.

2. The fibrosity of the above minerals is ascertained on a microscopic level with fibers defined to be particles with an aspect ratio of 3 to 1 or larger.

We also expressly adopt the following approach articulated by the recent British Advisory Committee on Asbestos (Vol. 1, p. 11):

> *Asbestos* is a generic term for the fibrous forms of several mineral silicates. These occur naturally in seams or veins, generally between about 1 and 20 millimetres (mm) in width in many igneous or metamorphic rocks and belong to one of two large groups of rock-forming minerals: the serpentines and amphiboles.
>
> We recognize the mineralogical complexities associated with the definition and identification of asbestos and asbestos fibre, but for the purposes of this report we concentrate on the fibre types with which people are most likely to come into contact as a result of their use in industry. The serpentine group contains the type of asbestos known as chrysotile ('white asbestos'), which is the only asbestiform member of this group of minerals and by far the commonest and commercially the most important type of asbestos. The amphibole group contains crocidolite ('blue asbestos'), amosite ('brown asbestos'), anthophyllite, actinolite and tremolite. Amosite is an acronym for Asbestos Mines of South Africa and is mineralogically known as cummingtonite–grunerite asbestos. Tremolite may occur as a contaminant with chrysotile and with other minerals such as talc. Crocidolite, amosite and anthophyllite have all been exploited commercially, although anthophyllite is no longer in significant quantities.

The above definitions of asbestos should not be taken to mean that fibers or mineral fragments of other minerals are without biological significance (IARC, 1977). Although epidemiologic data for other "mineral fibers" are limited at this time, prudence dictates that such substances be handled with caution.



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# II. ASBESTOS SAMPLING AND ANALYSIS

## Update on New Methods

Since the NIOSH revised recommended asbestos standard was published in December, 1976, there have been several new developments in the area of sampling and laboratory analysis. Kim et al. (1979) have developed a quick screening test for chrysotile, crocidolite, and amosite which can be used for bulk material samples. The test is based upon the formation of color complexes with $Mg^{+2}$ and $Fe^{+2}$ released from asbestos upon acid digestion. The $Mg^{+2}$ from chrysotile is complexed with p-nitrobenzenazo-s-naphthol. The $Fe^{+2}$ from crocidolite and amosite is complexed with 1,10-phenathroline. A positive test is indicated by formation of colored complex for $Mg^{+2}$ and/or $Fe^{+2}$. The test by Kim et al. is not specific for asbestos; however, the detection limit is reasonable for bulk samples with 1-2 mg being detectable in any given sample. Of 70 samples tested, 52 were correctly classified as containing asbestos or not, 18 samples gave false positives, and there were no false negatives. The method has little promise for airborne samples.

Lange and Haartz (1979) have developed a method for chrysotile asbestos determinations by X-ray diffraction. The method for membrane filters involves ashing followed by redeposition on silver membrane filters. The 7.33Å peak for chrysotile is primarily used in an integrated mode. Normalization using reflections from the silver membrane is employed along with X-ray absorption corrections. The lower limit of detection is reported to be 2 μg on a filter with good linear response to over 200 μg per filter. Minerals such as antigorite, lizardite, kaolinite, and possibly chlorite are potential interferences with chrysotile. The method has not been adapted for amphibole determinations.

Lilienfeld and Elterman (1977) and Lilienfeld et al. (1979) have developed a portable monitor capable of real-time determinations of airborne fiber concentrations. The monitor is based upon rotation of elongated particles by means of a rotating electric field of large voltage gradient. Fibers of various lengths are then detected by synchronous detection of modulated light scattered from a continuous-wave helium-neon laser beam with modulation generated by the rotating particles. Concentrations between 0.001 and 30 fibers/cc are reported to be detectable. At a concentration of 1 fiber/cc, a relative standard deviation of 10% is reported. The minimum detectable fiber length and diameter are estimated to be 2 μm and 0.2 μm, respectively. The instrument is not specific for asbestos as other elongated particles align within the electric field, and the instrument cannot be easily used for obtaining "breathing zone" samples.



Gale and Timbrell (1979) have reported progress in development of an automated method for determining fiber density on membrane filters. The method involves first clearing the membrane filter by conventional methods followed by aligning fibers on the filter in a strong magnetic field. The sample is then placed in a specially designed microscope on a motor driven stage. Fiber density is determined by measuring light scattered from the rotating fibers. The method is not yet commercially available, but is predicted to have a lower limit of detection of about 0.1 fiber/cc based on a 4-hour sampling period.

## Optical Microscopy

The phase contrast method recommended by NIOSH for compliance sampling in the occupational setting was reviewed in the 1976 NIOSH document. Since that review, Leidel et al. (1979) have reported studies to better define precision of the method at lower levels. Minor changes in fiber counting methods have also been recommended by NIOSH to correct a potential statistical bias.

Based on the most recent data available, Leidel et al. (1979) estimated the coefficient of variation for the membrane filter sampling–phase contrast counting method to be 0.11 to 0.15, given a total count of at least 100 fibers. With a reduced fiber count of 10 fibers in the analysis, the coefficient of variation is estimated to be 0.41. Statistical tests based upon these estimates of precision are recommended by NIOSH for determining compliance or non-compliance with regulatory standards (Leidel et al., 1979). Procedures are available for single full shift samples, multiple samples covering the workshift, or short "grab samples."

The phase contrast method is clearly capable of measuring airborne fiber levels down to 0.1 fibers/cc (fibers longer than 5 μm) given that due consideration is given to inherently high variability at such levels. The method is highly sensitive for detection of fibers longer than 5 μm; however, specificity of the



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method for identifying asbestos fibers may be a serious problem under certain circumstances. Fiber identification is based only upon fiber length and aspect ratio; therefore, the method is not specific in situations where a mixture of asbestos and non-asbestos fibers occur or where large numbers of other elongated particles are present. The lack of specificity becomes more serious at lower fiber concentrations, and alternate methods for identification are likely to be required. The most likely choice for fiber identification in airborne dust samples is electron microscopy where both electron diffraction and microchemical analyses may be used to identify fibers (NIOSH, 1976). The fraction of asbestos fibers determined by these methods could then be multiplied by phase contrast determinations to arrive at asbestos fiber levels. It seems reasonable that such determinations only need be made for a statistically determined sample and not for each airborne dust sample, with subsequent determinations made only upon process or product modifications. The statistical confidence of the airborne asbestos fraction determinations should be taken into account in determinations of compliance or non-compliance.

In addition to the problem of lack of specificity for fiber identification, only a fraction of all airborne asbestos fibers are actually accounted for by the phase contrast method, which considers only fibers longer than 5 μm. The phase contrast method, therefore, can only be considered an "index" measure of fiber exposure. In fact, the fraction of airborne fibers longer than 5 μm is extremely variable, ranging from 1 to 50%, depending on fiber type and industrial operation (Dement et al., 1976). In addition to determinations of fiber identification by electron microscopy, it may also be desirable to determine airborne fiber size and specifically the fraction of airborne fibers longer than 5 μm.

## III. BIOLOGIC EFFECTS OF EXPOSURE TO ASBESTOS IN ANIMALS

*In Vivo*

Animal studies reported since 1976, in which several types of asbestos have been utilized, further support the findings published in the NIOSH Revised Recommended Asbestos Standard. In that publication, reference was made to research which adequately demonstrated that all commercial forms and several other types of asbestos can produce mesotheliomas and primary bronchogenic neoplasms in animals.



Although mesotheliomas were most readily produced by intrapleural injections, they were also produced by inhalation exposures (Wagner et al., 1974). Since then, additional studies by Wagner et al. (1979) have shown that a commercial grade, predominantly short fiber Canadian chrysotile, which is used primarily for paint and plastic tile fillers, can induce mesotheliomas when injected intrapleurally into rats, and induce primary lung neoplasms when the animals are exposed by inhalation.

Not only is chrysotile as potent as crocidolite and other amphiboles in inducing mesotheliomas after intrapleural injections (Wagner et al., 1973), but also equally potent in inducing pulmonary neoplasms after inhalation exposures (Wagner et al., 1974). 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 (Wagner et al., 1974). The concentration of dust in the lungs of rats exposed to Canadian chrysotile was only 1.8-2.2% of the dust concentration in the lungs of animals exposed to amphiboles (after 24 months of inhalation exposures). Yet the lung tumor incidences and degrees of pulmonary fibrosis were similar in all groups. The reasons for higher incidences of lung cancer and mesotheliomas in workers exposed to amphiboles is, therefore, probably related to higher concentrations of respirable fibers during their exposures.

Research to this day has not been able to establish a fiber length below which there exists no carcinogenic potential by inhalation, the most common route of occupational exposure. This is true because of the unavailability of specifically sized fibers (Pott, 1979).

Not only were naturally occurring fibers carcinogenic, but synthetic fibers were carcinogenic as well. Pylev (1979) obtained mesotheliomas in 54% of rats injected intrapleurally with a milled synthetic hydroxy-amphibole, and primary lung neoplasms in 23% of hamsters injected intratracheally with a synthetic chrysotile. Mesotheliomas were also induced in 9/60 hamsters injected intrapleurally with glass fibers, 82% of which were greater than 20 μm in length (Smith et al, 1979).

Further experimentation with fibers of differing diameters and lengths supports the previous observation that long, thin fibers are much more carcinogenic than short or thick fibers. Utilizing 16 preparations of fiberglass of differing fiber lengths



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and diameters, Stanton et al. (1977) were able to show that glass fibers with diameters less than 1.5 μm and lengths greater than 8 μm were carcinogenic in the pleura of rats, and that fibers shorter or wider than those were much less carcinogenic. For example, one preparation in which 60% of the fibers were less than 1.5 μm in diameter induced pleural sarcomas in 64% of rats, whereas another preparation in which only 16% of the fibers were less than 1.5 μm in diameter induced pleural sarcomas in only 14% of rats. Ninety-five percent of the fibers in both preparations were greater than 8 μm in length.

Besides the corroborating evidence for the carcinogenic potential of asbestos, recent results indicate a strong co-carcinogenic effect. Kung-Vosamae and Vinkmann (1979) reported a strong synergism between nitroso-diethylamine (NDEA) administered orally and chrysotile given intratracheally. NDEA given orally alone induced lung tumors in only 2% of the hamsters, whereas NDEA administered orally plus Canadian chrysotile given intratracheally induced lung neoplams in 40%. Chrysotile alone induced no lung tumors.

Additional research on the transport of fibers into tissues has confirmed that fibers reach the lymphatics shortly after oral administration (Masse et al., 1979). In view of the ability of intratracheally administered chrysotile to act synergistically with at least one nitrosamine, it is possible that ingested asbestos could act synergistically with orally administered nitrosamines to induce cancer in the gastrointestinal tract.

*In Vitro*

In vitro studies of all commercial forms of asbestos have been inconsistent when repeated or performed at different laboratories. Their correlation with in vivo studies has also been inconsistent and, thus, their value in studying the etiology of asbestos induced diseases is unclear at present.

## IV. BIOLOGIC EFFECTS OF EXPOSURE TO ASBESTOS IN HUMANS

### Amosite

Seidman et al. (1979) have extended their study of amosite asbestos workers with short-term exposures. The study group consisted of 820 men first employed between June, 1941 and December, 1945 in the production of asbestos insulation and



who were alive in 1961. Followup was through 1977, with expected deaths adjusted for age and calendar time estimated using death rates for white males in the general population of New Jersey.

Among the cohort studied by Seidman et al., 83 lung cancers were observed according to death certificate information, whereas 23.1 were expected. Among 61 men employed less than 1 month, 3 lung cancers were observed versus 1.3 expected. Although based on small numbers, excess mortality from lung cancer showed an increasing trend with employment duration. Cancer latency periods were progressively shortened with increasing employment duration. Four mesotheliomas were reported on death certificates in contrast to 14 which were identified on autopsy and other tissue diagnoses. Three in the group had less than 1 year of exposure. Although no environmental data are available for this plant, dust counts were made in another plant using the same fiber type and production process. Seidman et al. reported average exposure at this plant to be 23 fibers/cc. Further data available for this comparison plant were published by NIOSH (1972) showing mean exposures to range from 14 to 75 fibers/cc. At such concentrations, a lung cancer relative risk of 2.3 could be calculated with employment less than 1 month.

Anderson et al. (1979) evaluated the risks of non-malignant and malignant disease associated with household exposure to work-derived amosite dust. Four mesothelioma cases were reported among household contacts of former workers at a plant manufacturing amosite insulation products in Paterson, New Jersey. Anderson et al. also reported a 35.9% prevalence of radiographic abnormalities among household contacts of former employees at this same amosite plant, compared with a 4.6% prevalence among a control group. These radiographic abnormalities included pleural thickening, pleural calcification, pleural plagues, and irregular opacities. These studies raise the specter of non-occupational hazards associated with casual or low-level exposures to amosite.

Murphy et al. (1978) reported a followup to their first report (1971) of shipyard pipe coverers exposed predominantly to amosite asbestos. Workers in the original Murphy report of 1971, with "asbestosis" diagnosed by multiple criteria, had a poor prognosis as reported in the 1978 longitudinal survey.



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**Chrysotile**

Robinson et al. (1979) reported an additional 8 years of observation and 385 deaths to the Wagoner et al. (1973) study of mortality patterns among workers at one facility manufacturing asbestos textile, friction, and packing products. Chrysotile constituted over 99% of the total quantity of asbestos processed per year, except for 3 years during World War II. During these 3 years, amosite was selectively used to a limited extent because of U.S. Naval specifications, and accounted for approximately 5% of the total asbestos used per year. Crocidolite and amosite for the other years accounted for less than 1% of total usage in very selected areas. Exposures to these other two types may have played some role in the etiology of disease; however, due to the overwhelming exposure of the cohort to chrysotile, it is likely that the other exposures played a minor role in the overall mortality patterns. Robinson et al. confirmed Wagoner et al.'s observations of statistically significantly excess deaths due to bronchogenic cancer, suicide, heart disease, and nonmalignant respiratory disease including asbestosis and a statistically non-significant excess of digestive cancer and lymphoma. Robinson et al. described 17 mesotheliomas whereas no mesotheliomas were detected in the Wagoner et al. study where observation of mortality ceased in 1967. The appearance of 17 mesothelioma in the updated study reflects latency periods of 24 to 53 years since onset of first exposure. Further analysis indicated 14 of 17 mesothelioma deaths occurred after the original study period. This observation confirms other findings that mesotheliomas are characterized by very long latency periods. Chovil and Stewart (1979) also reported latencies of 6 to 44 years, with a mean of 26.9 years.

Weiss (1977) reported no unusual mortality experience over a 30-year period for a cohort of workers employed in a paper and millboard plant stated to be using only chrysotile. The author concluded that the study results were suggestive of a minimal hazard from chrysotile. This conclusion must be viewed in light of the limitations inherent in the study. The study population was small (n= 264) and only 66 workers had died at the time of analyses. Two of these workers died of asbestosis. Moreover, the unusually low Standard Mortality Ratio (SMR) for many causes of death in the Weiss et al. paper suggests the possibility of a selection bias greater than that usually seen when contrasting industrial populations with the general population.



McDonald et al. (1973, 1974) reported an increased risk of lung cancer among men employed in Quebec chrysotile mines and mills. The risk of lung cancer among those workers most heavily exposed was 5 times greater than those least exposed. Liddell et al. (1977) further analyzed the mortality experience of the cohort of chrysotile asbestos miners and millers previously studied by McDonald et al. and found excesses of respiratory cancer, asbestosis, and mesothelioma. These same chrysotile miners and millers of Quebec, as of 1977, had experienced nine confirmed and two suspected mesotheliomas (McDonald, 1978). The author concluded for the seven cases observed at Thetford mines that "There is therefore no good reason to doubt chrysotile exposures as the cause."

A recent study by Nicholson et al. (1979) examined the mortality of 544 Quebec chrysotile mine and mill employees which corresponded closely in terms of duration of exposure and periods of observation to cohorts of mixed fiber asbestos factory workers and insulation workers established in other studies. Among this cohort of 544 men with at least 20 years of employment in chrysotile mining and milling at Thetford Mines, Canada, 16% of the deaths were from lung cancer and 15% from asbestosis. The risk of death for asbestosis, at equal times from onset of exposure, was very similar in the miners and millers to that found in the factory workers and insulators. Lung cancer was similar among the miners and millers and in the factory workers but higher in the insulators. One death from mesothelioma was reported in this study.

Selikoff (1977*b*) surveyed 485 current employees of a chrysotile mine in Baire-Verte, Newfoundland, which had been in operation since 1963. Fifty employees (10%) had one or more radiographic abnormalities of the type commonly associated with asbestos exposure. Parenchymal abnormalities were most common, and pleural changes were detected in only 3% of the individuals surveyed. For those individuals employed less than 5 years the prevalence of abnormalities was 5%, and this increased with duration of employment. Changes occurred most commonly in those with the most intense exposures. This study was designed only to assess asbestos-related disease under more modern conditions than have previous studies (Kogan et al., 1972; Rossiter et al., 1972); thus, assessments of the effects of short duration of exposure and long latency could not be made. The interpretation of these data is further complicated



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by the lack of a control population and environmental measurements. The study does demonstrate the prevalence of chest X-ray changes in an appreciable proportion of employed workers, despite a short period since initial exposure.

Rubino et al. (1979) reported nine asbestosis deaths among chrysotile asbestos miners in northern Italy. Excess lung cancer (7 vs. 3.4) was seen only during the last quinquennium of observation, 1971-1975, that period of time after greatest latency. Also, one mesothelioma was reported in this latest period.

Studies examining lung tissue of mesothelioma cases and comparison groups have shown equivocal results as to the possible relationship of chrysotile in lung tissue and mesothelioma. Jones et al. (1979) found no evidence to indict chrysotile, while Acheson and Gardner (1979) estimated a 6-fold relative risk of mesothelioma for persons with only chrysotile in lung tissue as compared with controls with no asbestos fiber in their lungs.

Boutin et al. (1979) reported on a study of chest film abnormalities among chrysotile miners and millers in Corsica. They studied 166 ex-workers of the mines and mill closed in 1965, and compared them with 156 controls without asbestos exposure and with similar demographic variables. Chest films were read according to the ILO U/C Classification system. Compared with controls, chrysotile workers had a prevalence of all parenchymal abnormalities 2.4 times that of controls. For those with a profusion of 1/2 or more, the prevalence ratio was approximately twice the controls. Pleural changes were twice as prevalent in chrysotile workers as in controls. Exposures among this cohort were reported to have been very high, with exposure levels ranging from 85 to 267 millian parts per cubic foot (mppcf).

## Crocidolite

Jones et al. (1976) reported a high incidence of mesothelioma among women who worked predominantly with crocidolite in a factory producing gas mask canisters during World War II, and have recently extended observations on this population (Jones et al., 1979). Among this group of 1,088 workers exposed only between 1940 and 1945, 22 pleural and 7 peritoneal mesotheliomas were observed. This is likely an underestimate since 373 women were lost to observation. A linear dose-response



relationship with length of employment was observed for mesothelioma, with three mesotheliomas observed among those exposed 5-10 months.

McDonald and McDonald (1978) have also studied mortality of 199 workers exposed to crocidolite during gas mask manufacture in Canada during 1939 to 1942. This cohort was followed through 1975, and 56 deaths occurred. Out of these 56 deaths, 4 (7%) were from mesothelioma and 8 from lung cancer. It should be pointed out that an additional five mesotheliomas not reported on death certificates were diagnosed on review of pathology or autopsy material.

## Mixed Fiber Types

Weill et al. (1979) and Hughes and Weill (1979) reported on the mortality experience of a cohort of 5,645 men employed in production of asbestos cement products and who had at least 20 years since first exposure. These workers were exposed largely to chrysotile, with some crocidolite and amosite. Among this group, 601 persons were identified as deceased by the Social Security Administration. Those with unknown vital status (25%) by this source were assumed to be alive, thus likely resulting in underestimation of the true risk. Death certificates were obtained for 91% of the known deaths. Dust exposures were estimated using each worker's employment history in conjunction with historical industrial hygiene data.

Weill et al. (1979) observed increased respiratory cancer mortality only among those with exposure in excess of 100 mppcf/year, where 23 cases were observed versus the 9.3 expected. The unusually low SMR for all causes in the low exposure groups suggests the possibility of a selection bias, and any interpretation of risks at low exposures should be done with caution. Two pleural mesotheliomas were reported. Separating the cohort by type of fiber exposure, the authors concluded that the addition of crocidolite to chrysotile enhanced the risk for respiratory malignancy; however, an excess risk (8 observed vs. 4.4 expected) was observed among those not exposed to crocidolite, with cumulative exposures in excess of 200 mppcf-months (16.6 mppcf-years). Both average concentration of exposure and duration of exposure were found to be related to cancer risk.

Jones et al. (1979) studied the progression of radiographic abnormalities and lung function among asbestos cement work-



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ers. Chest films taken in 1970 and 1976 on 204 workers were read independently by two readers according to the ILO U/C 1971 Classification scheme. These films were read side-by-side in known order and ranked according to progression. Spirometric measurements were made in 1973 and 1976. The major findings of the Jones et al. study were: (1) the progression of small opacities was dependent upon both average and cumulative exposure; (2) significant declines in lung function were shown to result from both smoking and cumulative exposure; and (3) pleural abnormalities progressed as a function of time with little association to additional exposure. No estimates were made of the incidence of various respiratory abnormalities in relation to exposure.

Peto (1979) reported on the mortality experience of a cohort of asbestos textile workers previously studied by Doll (1955), Knox et al. (1968), and Peto et al. (1977). Data from this factory had previously been used by the British Occupational Hygiene Society (BOHS, 1968) in establishing occupational exposure standards and was subsequently studied by Lewinsohn (1972). Routine dust measurements in this factory were first made in 1951. Among the 255 males first employed after 1951, 12 lung cancers were observed, whereas only 4.65 were expected, based on national death rates. Among those with 20 or more years since initial employment, 8 lung cancers were observed versus 1.62 expected. Fiber exposures were estimated to be 32.4 fibers/cc in 1951, decreasing to 1.1 fibers/cc in 1974. These estimates are 2.4 times previously estimated values for this plant (Peto et al., 1977). Peto estimated the relative risk for cumulative exposures of 200-300 fibers/cc-year to be between 2 and 3. The cohort is too small and followup too short to estimate cancer risks at lower exposures. No mesotheliomas were observed in the cohort first employed after 1951; however, the followup period is insufficient to address this question.

Berry et al. (1979) extended their 1968 observations concerning asbestosis by including persons completing 10 or more years employment by 1972. Persons who left after June 30, 1966, were also contacted and encouraged to participate, with 68 of 113 persons eventually participating. Outcome measures studied included chest radiographs, medical examination including assessment of basal crepitations, and pulmonary function (FEV, FVC, FRC, TLC, RF, TL, $PaCO_2$). Chest films were read by four readers by the ILO/UC 1971 Classification system



with readings being averaged. Dust exposures were estimated for each person using available hygiene data and estimates of control effectiveness.

In this study, "possible asbestosis" was diagnosed based on one or more combinations of basal rales or crepitations, radiological changes, a falling transfer factor, and restrictive lung function changes. Among these 379 men, 60 cases of possible asbestosis were diagnosed by the factory medical officer, whereas 85 cases were diagnosed by an independent clinician. Collaboration by these investigators subsequently resulted in 82 men with crepitations, 58 with "possible asbestosis," and 34 with certified asbestosis. Using the exposure data, these authors estimated the cumulative dose necessary for a 1% incidence for crepitations, possible asbestosis, and certified asbestosis to be 43, 55, and 72 fibers/cc-year, respectively. These authors pointed out limitations of the cumulative dose concept and acknowledged the imprecision of their exposure estimates. Two cases of certified asbestosis were observed among non-smokers and nine among ex-smokers. There were, in general, fewer respiratory symptoms and signs in non-smokers and light smokers than in heavy and ex-smokers.

Elmes and Simpson (1977) have extended their earlier (1971) report to include deaths occurring since 1965 through 1975. The mortality trend has shifted from a preponderance of asbestosis and gastrointestinal cancer deaths to malignancies of the lung and mesothelioma, diseases associated with longer latent periods. These authors stated that their findings would suggest any standard based "on the prevention of asbestosis may not provide adequate protection against neoplasia."

Morbidity and mortality analysis by Lacquet et al. (1979) of workers in a Belgian asbestos cement factory revealed a strong dose-response relationship for asbestosis, and pleural and parenchymal lung changes. Pleural thickening and adhesions began occurring in the lowest dose category (0-49 fibers/cc-year). Parenchymal lung changes occurred less frequently. No cases of asbestosis were recognized in workers with less than 100 fiber-years of exposure. Asbestosis occurs more frequently and with shorter latency periods (as the exposure levels increase) and adverse mortality tends to occur at longer latency periods (as the dose decreases) (Seidman et al., 1979). Because the observation period of the Lacquet et al. study was only 15 years, it cannot be assumed that the absence of asbestosis in the low dose categories currently observed will not occur



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in these low dose categories after a longer latent period, or that pleural and parenchymal lung changes are not indicators of early lung change that can or will progress to asbestosis. The mortality portion of the study revealed asbestosis and an excess of digestive cancer, but not excesses for lung cancer or mesothelioma. This, again, is not surprising since lung cancer and mesothelioma tend to develop after latent periods greater than 15 years.

Baselga-Monte and Segarra's (1978) examination of 1,262 workers employed in four factories in the Barcelona area demonstrated a dose-response relationship based upon radiologic images. The authors demonstrated a quick response for pleural radiological changes at individual cumulative doses as low as 5 fibers/cc-years, while the pulmonary and pleuropulmonary responses tend to appear later, but not at statistically different doses. The authors were reluctant to draw conclusions because of the design of the epidemiologic evaluation, which considered only active employees. Other epidemiologic studies of worker populations would indicate that evaluation of only active employees tends to underestimate the health risk since diseased workers oftentimes tend to self-select out of the active workforce (Fox and Collier, 1976; Enterline et al., 1972; Borow et al., 1973). Baselga-Monte and Segarra concluded that "the present worldwide trend to establish more exigent hygienic criteria for exposure to asbest(os) is confirmed." Based on their working model, this level for a 50-year working life should be 0.07-0.10 fiber/cc, "(taking into account protection levels of 89 and 95%)."

**Malignant Neoplasms other than Mesothelioma and Cancer of the Lung.**

A number of epidemiological studies indicate less striking associations of excess risks of other types of cancers (in addition to bronchial and mesothelial) and occupational asbestos exposure. Selikoff (1977*a*) reported increased rates for cancer of the stomach and esophagus (20 observed vs. 6.46 expected) and cancer of colon (23 observed vs. 7.64 expected) among 632 asbestos insulation workers in the New York and New Jersey area. Selikoff et al. (1979) made similar observations among 17,800 asbestos insulation workers in the United States and Canada. They reported increased mortality from cancer of the esophagus (18 observed vs. 7.1 expected), stomach (18 observed vs. 14.2 expected), and colon and rectum (58 observed vs. 38.1 expected) among this study cohort. Similar observations



have been reported by others (Elmes and Simpson, 1971; Kogan et al., 1972).

Cook and Olson (1979) have recently shown that sediment in human urine contains amphibole fibers, thus providing the first evidence that mineral fibers pass through the human gastrointestinal mucosa under normal conditions of the alimentary canal.

Stell and McGill (1973) found that of 100 men with squamous-cell carcinomas of the larynx, 31 had known exposure to asbestos, compared with only 3 in matched controls. Similar associations have been reported by Morgan and Shettigara (1976); Shettigara and Morgan (1975); Rubino et al. (1979); and Selikoff et al. (1979*a*). Newhouse et al. (1979), however, utilizing an interview of patients at the Royal National Throat, Nose, and Ear Hospital in London, found that asbestos exposure was not more common among cases as compared to controls.

Significant increases in cancer of the buccal cavity and of the pharynx have been reported by Selikoff et al. (1979*a*). Among 17,800 asbestos insulation workers they observed 16 deaths due to cancer of these sites whereas 10.1 deaths would have been expected based on U.S. white male rates.

Robinson et al. (1979) reported an excess of deaths due to lymphosarcoma and malignant lymphoma among white males employed in an asbestos textile, friction, and packing products manufacturing facility. There were 7 deaths due to cancer of these sites, while 3.28 cases were expected.

## V. SMOKING AND ASBESTOS

Hammond et al. (1979) recently reported the results of their 10-year followup study (January 1, 1967-December 31, 1976) of 12,051 asbestos insulation workers who had 20 or more years of work experience. They were able to obtain complete smoking histories of a large number of study subjects (8,220 workers) and compare their lung cancer mortality with that of a control population with a known smoking history. As a control population, 73,763 men in the American Cancer Society's prospective cancer prevention study were selected. These men were similar to asbestos workers in many respects. They were white males; non-farmers; had no more than a high school education; had a history of occupational exposure to dust, fumes, vapors, gases, chemicals, or radiation; and were alive as of January 1, 1967. Most of all, their smoking habits were also known.



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Age-standardized lung cancer mortality rates for control and asbestos workers were as follows:

| Groups | Exposure to asbestos | History of cigarette smoking | Death* rate | Mortality ratio |
|---|---|---|---|---|
| Control | No | No | 11.3 | 1.00 |
| Asbestos workers | Yes | No | 58.4 | 5.17 |
| Control | No | Yes | 122.6 | 10.85 |
| Asbestos workers | Yes | Yes | 601.6 | 53.24 |

*Rates per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers. Number of lung cancer deaths based on death certificate information.

Asbestos workers who did not smoke showed about a 5 times greater risk of dying of lung cancer when compared to the non-smoking control population. Asbestos workers who did smoke also had a 5 times greater risk of dying of lung cancer as compared to the controls who smoked. This means the relative risk associated with asbestos is about 5-fold for smokers and non-smokers alike. Therefore, the probability that their lung cancer was due to asbestos exposure is about 80% in both smokers and non-smokers.

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.

Liddell et al. (1977) further analyzed the mortality experience of a cohort of chrysotile asbestos miners and millers previously studied by McDonald et al. (1973, 1974) and McDonald and McDonald (1976). This cohort of 10,951 men born between 1891 and 1920, and who had at least 1 month of employment, was followed through December 31, 1973. Cause of death was ascertained for 97% of the 4,037 known deaths, whereas 1,117 (10%) were lost to followup. Smoking habits were ascertained through a questionnaire administered to those living or to relatives of deceased workers who died after 1951. Unlike previous reports on this cohort, person-years were accumulated by 5-year age groups and 5-year periods of calendar time, with expected deaths by cause calculated using mortality rates for males in the Province of Quebec.

For this cohort, the SMR for all causes was 107, and a SMR of 125 was observed for cancer of the lung and pleura. There also were 40 pneumonoconiosis deaths. Using the whole cohort as the referent population, an excess of respiratory cancer was observed only after cumulative exposures of 300 mppcf-years (relative risk = 1.39). However, only 15 of the 40 pneumonoconiosis deaths occurred with exposures greater than 300 mppcf-years. When available smoking data were taken into account, lung cancer SMRs of 48 and 46 were calculated for non-smokers and ex-smokers, increasing to 206 for heavy smokers. There were seven mesothelioma deaths among the cohort.

The Liddell et al. study suffers in that an "unexposed" group is not used for dose-response analyses of lung cancer; thus, risk at low doses could not be estimated. Secondly, smoking-specific death rates were not used for calculation of expected lung cancer deaths, thus underestimating risks among non-smokers.

There is little or no evidence that cigarette smoking is related to increased risk of pleural or peritoneal mesothelioma (Hammond et al., 1972, 1979).

Data from two studies suggest that cigarette smoking may contribute to the risk of asbestosis. Hammond et al. (1979) reported that the asbestosis death rate of asbestos workers who smoked was 2.8 times as high as that of non-smoking



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asbestos workers. Weiss (1971) reported a prevalence of pulmonary fibrosis of 40% (30/75) among asbestos workers who smoked in contrast to a prevalence of 24% (6/25) among non-smoking asbestos workers.

A small experimental study indicated that the particle clearance in the smokers was considerably slower than in the non-smokers. Cohen et al. (1979) reported that after a year 50 percent of magnetic dust ($Fe_3O_4$) originally deposited remained in the lungs of the smokers while only 10 percent remained in the lungs of the non-smokers. The authors suggested that smoking may impair the clearance of other dusts, including those that are toxic. This may help to explain the higher incidence of lung disease in smokers.

In summary, both asbestos and smoking are independently capable of increasing the risk of lung cancer mortality. When exposure to both occurs, the combined effect with respect to lung cancer appears to be multiplicative rather than additive. From the evidence presented, we may conclude that asbestos is a carcinogen capable of causing, independent of smoking, lung cancer and mesothelioma.

## VI. EXPOSURE TO ASBESTIFORM MINERALS OTHER THAN COMMERCIALLY MINED ASBESTOS

Gillam et al. (1976) reported a threefold excess risk of mortality from respiratory cancer and a twofold excess of non-malignant respiratory disease (excluding influenza and pneumonia) among miners exposed to amphibole fibers in the cummingtonite-grunerite ore series at concentrations less than 2.0 fibers/cm$^3$. A large majority of the airborne fibers was shorter than 5 μm in length. McDonald et al. (1978), in a subsequent study of the same mine, examined the mortality experience of persons with at least 21 years of employment at the mine and mill. This study showed significant excesses of pneumoconiosis (mainly silicosis), tuberculosis, and heart disease. No overall excess of malignant diseases was found. However, when the population was broken down by estimated exposure, respiratory cancer was in excess in the highest exposure group. The findings of McDonald et al. do not negate those reported by Gillam et al., but, rather, tend to strengthen them in that McDonald et al. used a rigid survival criteria (inclusion only of



those employed 21 or more years), and further diluted the underground exposed effect by including persons never exposed underground.

Commercial talc deposits are sometimes found to contain serpentines (chrysotile, antigorite, and lizardite) and fibrous and non-fibrous amphiboles. One important deposit studied has been mined in the Gouveneur Talc District of upper New York State. Talcs in this area contained less than 1-2% silica, but have been shown to contain tremolite and anthophyllite, resulting in elevated miner and miller exposures to these fibers (Dement and Zumwalde, 1979).

Kleinfeld et al. (1967, 1974) demonstrated elevated proportionate mortality due to lung cancer and respiratory disease among talc miners and millers in New York State. Brown et al. (1979) conducted a retrospective cohort mortality study among workers of one company in this area. The study cohort consisted of all white males initially employed sometime between January 1, 1947 and December 31, 1959, with followup through June 30, 1975. Expected cause-specific deaths were calculated using age, calendar time, and cause-specific mortality rates of the U.S. Among this cohort, 10 respiratory cancers were observed, whereas only 3.5 were expected. Excess mortality was also observed for non-malignant respiratory diseases. One case of mesothelioma was observed; however, this worker was known to have had prior employment with unknown exposures in the construction industry.

Gamble et al. (1979) studied respiratory disease morbidity among a cohort employed at the same mine and mill studied by Brown et al. (1979). A total of 121 currently employed workers were given a respiratory questionnaire, PA and lateral chest films, and spirometry tests. Talc workers with greater than 15 years employment were found to have an increased prevalence of pleural abnormalities compared to coal miners, potash miners, and chrysotile asbestos workers. $FEV_1$ and FVC reductions demonstrated significant association with particulate and fiber exposure.

Mesothelioma of the pleura and peritoneum have been detected in two villages in Turkey: Tuzkoy and Karain (Artvinli and Baris, 1979 and Baris et al., 1978). Mineralogical analysis of samples from ore and water has revealed the asbestiform mineral zeolite but no asbestos. These fibers were usually 1-2 μm in diameter and 30-40 μm in length. Annual rates of



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malignant pleural mesothelioma in Tuzkoy were found to be 22 cases per 10,000 people, and 182 cases per 10,000 in Karain, while studies in Pennsylvania (Lieben and Pistawka, 1967), Finland (Nurminen, 1975), and England, Wales, and Scotland (Greenberg and Davies, 1974) reported annual incidences of 1 to 2.3 cases per 1,000,000 persons.

## VII. NON-OCCUPATIONAL EXPOSURE TO COMMERCIAL SOURCES OF ASBESTOS

Anderson et al. (1979) reported on the occurrence of X-ray abnormalities among household contacts of workers in an amosite asbestos factory. The study cohort consisted of 679 household contacts who had lived in the household of an actively employed amosite asbestos factory worker and who themselves had not had occupational exposure to asbestos or other fibrogenic dust. These individuals were given a PA chest film which was read by five readers according to the ILO/UC Classification of 1971. For comparison, 325 controls living in the same community as the study subjects, matched by age and sex, were examined.

Among the study population, there was a 17% prevalence of small opacities, versus 3% for the controls. Pleural calcifications were present in 8% of the household contacts and were seen in none of the controls. There were 35% with one or more radiological abnormalities among household contacts in contrast to 5% among controls. An increasing prevalence of all abnormalities with duration of exposure was observed.

Churg et al. (1978) reported a case of mesothelioma of the pericardium in a man treated 15 years earlier for angina pectoris by dusting of the pericardial cavity with a mixture of fibrous dusts including anthophyllite asbestos, tremolite asbestos, and fibrous glass.

## VIII. DOSE-RESPONSE RELATIONSHIPS

Evidence available to date indicates that a large dose of asbestos will produce a bigger health hazard than a small dose. Seidman et al. (1979), using the length of time worked in an amosite asbestos factory as a measure of the dosage of asbestos, reported an increased risk of dying from lung cancer with increasing duration of employment. Henderson and Enterline (1979), using cumulative dust exposure as an estimate of dose,



reported that the dose-response relationship for lung cancer is more likely linear. They predicted the relationship to be SMR=100+0.658 times cumulative dust exposure (mppcf-years). Liddell et al. (1977) also reported a similar relationship, i.e., a tendency for the mortality for lung cancer to increase with the dose.

Berry et al. (1979) reported that the occurrence of crepitations, possible asbestosis, and certified asbestosis was related to the cumulative dose.

Newhouse and Berry (1973) suggested that the risk of dying from mesothelioma increases with increasing dose. Jones et al. (1979) reported a linear relationship between the mesothelioma rate and length of exposure. In a study of the women workers in a wartime gas mask factory, they found that women having a long employment period had a higher proportion of death due to mesothelioma than those who had a short period of employment.

Although there appears to be little dispute that a larger dose of asbestos will pose a greater health risk, the exact nature of the dose-response relationships may be subject to considerable debate. This is so primarily because of problems of exposure estimation. Methods of measuring dust levels have changed over time with respect to sampling instrument (thermal precipitation vs. midget impinger vs. membrane filter), location of sampling (personal vs. area), and dust counting (particles vs. actual fibers) and/or evaluation techniques (whole fields vs. eyepiece graticule). As a result, conversion of dust levels obtained by one method to levels comparable to another method is far from simple, and is subject to considerable error. Another factor which may lead to differences of opinion on the exact shape of the dose-response curve is the measure of the dose. The commonly used measures of exposure are the cumulative dose and the duration of employment. Since using cumulative dose as a measure of exposure gives equal weight to the concentrations of dust experienced in each year of exposure, exposure of many years ago is considered as important as recent exposure. This practice is unrealistic for the chronic diseases having a long latency period. Duration of employment has also been used as a measure of exposure under the assumption that increasing the work time approximates increasing the dose. This procedure has the same problem as using the cumulative dose. Fur-



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thermore, in the absence of reliable past exposure data, the duration of employment may not equal the total dose of asbestos.

With regard to the linear hypothesis, the British Advisory Committee on Asbestos stated the following in 1979:

> Our reasons for preferring a linear hypothesis are: (1) It fits the data for occupational exposures; (2) it is the simplest hypothesis and the one most readily used for extrapolation to the probable effects of low doses; (3) it is likely to lead to an overestimate rather than underestimate of risks at very low doses. (Final Report, Vol. 2, p. 14).

Data available to date provide no evidence for the existence of a threshold level. Virtually all levels of asbestos exposure studied to date demonstrated an excess of asbestos-related disease.

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